Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in 14 dogs

V. SZATMÁRI, J. ROTHUIZEN, F. J. VAN SLUIJS, T. S. G. A. M. VAN DEN INGH, G. VOORHOUT

Doppler ultrasonography was used to evaluate the portal vein in 14 dogs before, immediately after and four weeks after a partial ligation of a congenital extrahepatic portocaval shunt. By four weeks after the operation, the hepatofugal or zero flow in the portal vein segment cranial to the shunt origin had become a hepatopetal flow in 13 of the dogs, which became clinically healthy. The other dog continued to have a hepatofugal flow in the portal vein cranial to the origin of the shunt and continued to show clinical signs of hepatic encephalopathy. The shunt remained functional in six of the dogs, and three of them developed portosystemic collaterals in addition. In the other eight dogs the patent shunt was non-functional, because a hepatopetal flow was detected in the shunt adjacent to the portal vein. This flow was the result of the splenic vein entering the shunt, and the splenic blood dividing; some flowed via the shunt towards the portal vein, preventing the portal blood from shunting, and the rest flowed via the attenuated shunt segment to the caudal vena cava. Shunting of the splenic venous blood was clinically insignificant.

PORTOSYSTEMIC shunting occurs when anomalous veins allow the portal blood to enter the systemic veins directly without first flowing through the hepatic sinusoids (van den Ingh and others 1995); it can be acquired or congenital. Acquired portosystemic collaterals develop as the result of sustained hepatic or prehepatic portal hypertension, by the enlargement of rudimentary extrahepatic vessels through which no blood normally passes (Vitums 1959, Johnson 1987). The formation of collaterals is a compensatory mechanism to maintain normal portal pressure by allowing the portal blood to be drained into the lower-pressure systemic veins. Congenital portosystemic shunting is the result of a single or double anomalous vein being present without concurrent portal hypertension (van den Ingh and others 1995). The definitive treatment of a congenital portosystemic shunt involves the occlusion of the shunting vessel as close as possible to the systemic venous circulation (van Vechten and others 1994, Wolschrijn and others 2000). Attenuating the shunt forces blood to flow through the portal branches, which are often hypoplastic, resulting in the development of portal hypertension (Rothuizen and others 1982, van den Ingh and others 1995). Hypoplasia of the portal vein may be either primary or secondary to portal hypoperfusion (van den Ingh and others 1995). The degree of portal hypertension that develops during the attenuation of a congenital portosystemic shunt depends on the severity of the hypoplasia of the portal vein, that is, the capacity of the portal branches to absorb the increased blood flow, and on the degree of attenuation of the shunt. If severe portal hypertension develops after the shunt has been ligated the patient may die during or shortly after the surgery as a result of circulatory collapse or thrombosis of the portal vein (Scavelli 1989, Roy and others 1992, van Vechten and others 1994). If these acute complications do not occur, portosystemic collaterals may develop as a result of sustained portal hypertension (van Vechten and others 1994).

It has been suggested that the complete occlusion of a congenital portosystemic shunt might provide a better outcome than a partial ligation (Johnson and others 1987, Swalec and Smeak 1990, Hottinger and others 1995, Harvey and Erb 1998, Hunt and Hughes 1999, Howe and Boothe 2002), but some studies have observed no difference between the clinical outcomes in dogs treated by either partial or complete ligation of the shunt (Mathews and Gofton 1988, Lawrence and others 1992, van Vechten and others 1994, Komtebedde and others 1995, Smith and others 1995). In most of the studies, the underlying reasons for the poor outcomes were not determined. It has been suggested that if the complete occlusion of a shunt is not feasible during an operation, because it would result in the development of a fatal portal hypertension, a second operation should be performed later to attempt to occlude the shunt completely (Hottinger and others 1995, Howe and Boothe 2002). The underlying idea is that the initial partial ligation would allow the portal system to adapt to an increased blood flow and the portal branches would gradually become wider before the second operation.

A second operation should only be considered when the portosystemic shunting persists exclusively through the congenital shunt. When shunting occurs through acquired collaterals the further attenuation of the congenital shunt is contraindicated. To determine whether the portosystemic shunting occurs via congenital or acquired portosystemic shunts, or both, mesenteric portography can be used (Vogt and others 1996). 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 (Thurmon and others 1996). Scintigraphy can be used to detect and quantify portosystemic shunting (van Vechten and others 1994, Meyer and others 1999), but the technique cannot distinguish between congenital and acquired shunting. Doppler ultrasonography provides a non-invasive method for examining the abdominal blood vessels in unsedated dogs, and the direction and velocity of the blood flow can easily be determined (Szatmári and others 2001). However, no descriptions of the ultrasonographic findings in dogs undergoing a partial ligation of a congenital portosystemic shunt have been found in the literature.

The objectives of this study were to describe the haemodynamic changes in the portal system of dogs undergoing a partial attenuation of a congenital shunt, and to suggest ultrasonographic criteria for deciding whether or not a dog should undergo a second operation to ligate the shunt completely. It was also hoped that evidence might be obtained to help to explain, at the level of portal haemodynamics, why some dogs with a partially attenuated congenital portosystemic shunt have excellent clinical outcomes, but others have poor outcomes.

Veterinary Record (2004) **155,** 448-456

V. Szatmári, DVM, PhD, **G. Voorhout,** DVM, PhD, DipECVDI, Division of Diagnostic Imaging, **J. Rothuizen,** DVM, PhD, DipECVIM, **F. J. van Sluijs,** DVM, PhD, DipECVS, Department of Clinical Sciences of Companion Animals, **T. S. G. A. M. van den Ingh,** DVM, PhD, DipECVP, Department of Pathology, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 8, 3584 CM Utrecht, The Netherlands

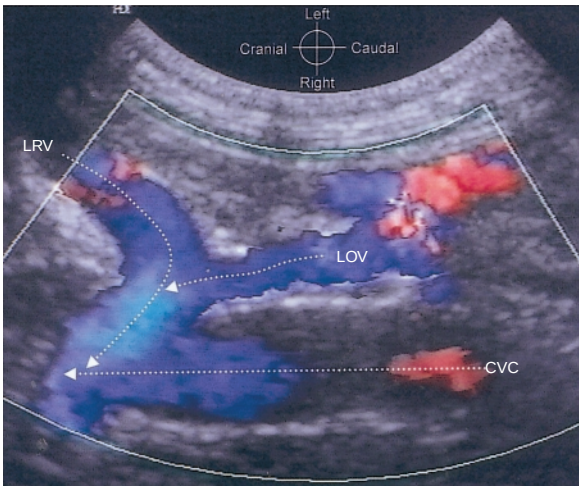


FIG 1: Dilated left ovarian vein (LOV) as a result of acquired splenorenal collaterals in a miniature schnauzer four weeks after the partial attenuation of a congenital extrahepatic splenocaval shunt. The colour Doppler ultrasound image was made via the left flank with the dog in right lateral recumbency. Arrows indicate the direction of blood flow. LRV Left renal vein, CVC Caudal vena cava

MATERIALS AND METHODS

The 14 dogs that were selected for the study had undergone surgical ligation of an extrahepatic congenital portosystemic shunt at the Companion Animal Clinic of Utrecht University between April 2001 and April 2003. They had also fulfilled the following criteria: the shunt was partially attenuated; the owners returned with their dogs for a re-examination four weeks after surgery; and ultrasound examinations were performed before and during the operation.

Thirteen of them were toy breed dogs, three of which had extrahepatic right gastric-caval shunts and 10 had extrahepatic splenocaval shunts; the other dog, a rottweiler, had an extrahepatic splenocaval shunt. The age of the dogs at surgery ranged between four months and five-and-a-half years. The postattenuation diameter of the shunt ranged from 1·5 to 5·0 mm.

The diagnosis was established preoperatively in the unsedated animals by transabdominal ultrasonography, using a HDI 3000 ultrasound system (ATL) with a 7·4 MHz phased array transducer and an 8·5 MHz curved linear array transducer, by direct visualisation of the shunt after having measured a 12-hour fasting blood ammonia level above the normal reference range of 24 to 45 µmol/litre.

The shunts were attenuated by one surgeon (F. J. v. S.) using the method described by Wolschrijn and others (2000), combined with intraoperative Doppler ultrasonography (Szatmári and others 2003), to the narrowest diameter that did not result in cyanosis of the intestines. If signs of portal hypertension, including intestinal cyanosis, were seen during the surgery, then the ligature was removed and another with a larger diameter was placed around the shunting vessel. The pressure in the portal vein was not measured. Before and after placing the ligature around the shunt, the direction of blood flow was determined in the shunt and in the portal vein cranial and caudal to the origin of the shunt by colour Doppler ultrasonography. The results of the ultrasonography after the shunt had been ligated did not influence the surgeon's decision about the degree of attenuation of the shunt. The pre-, intra- and postoperative ultrasound examinations were made by one ultrasonographer (V. S.).

When the dogs were re-examined four weeks later, 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 for identifying portal hypertension), and to the size and symmetry of the liver, as an indicator of portal venous perfusion of the organ. Acquired portosystemic collaterals were diagnosed ultrasonographically when the left gonadal vein was dilated (Fig 1) (Szatmári and others 2004a). Special efforts were made to image the right and left branches of the portal vein. Colour Doppler ultrasonography was used to determine the direction of flow in the shunt, the portal branches and the portal vein 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 a uniform insonation method, was used to measure the velocity of blood flow in the portal vein caudal to the origin of the shunt (Szatmári and others 2001). The time-averaged mean portal velocity was calculated by a built-in automatic spectrum analyser (HDI 3000 HighQ; ATL).

The ultrasonographic results obtained in each dog before surgery, after the shunt had been attenuated during surgery and four weeks later were compared.

On the basis of the ultrasonographic results four weeks after surgery, four categories of outcome were defined: excellent, when the direction of the flow in the shunt and throughout the portal vein were hepatopetal (towards the liver), and no acquired portosystemic collaterals were found; good, when the direction of flow in the entire portal vein was hepatopetal, but hepatofugal (away from the liver) in the shunt, and no collaterals were found; fair, when the direction of flow was hepatopetal throughout the portal vein, hepatofugal in the shunt, but collaterals were found; and poor, when the direction of flow was hepatofugal in the shunt and in the portal vein cranial to the origin of the shunt, but no collaterals were found.

When the dogs were examined four weeks after surgery, ultrasound-guided core biopsies of the liver were taken for histopathological examination if the whole or a part of the liver was small, despite the flow throughout the portal vein being hepatopetal, and if their blood ammonia concentration was above the normal range.

RESULTS

Right gastric-caval shunts

Three dogs had right gastric-caval shunts; the directions of blood flow recorded before, immediately after and four weeks after the shunts were attenuated are shown in Table 1. Before the operation the left and right portal branches and the portal vein cranial to the origin of the shunt could not be visualised.

Four weeks postoperatively the owners of all three dogs reported that the clinical signs had resolved completely. The blood ammonia levels were normal and there were no ultrasonographic signs of portal hypertension. The liver was normal in size and symmetrical. The left and right portal branches were easily recognisable. Colour Doppler ultrasonography revealed that the direction of blood flow throughout the portal vein and in the portal branches was hepatopetal in each of the three dogs, whereas the direction of flow in the shunt was hepatopetal in two (Fig 2) and hepatofugal in one. The time-averaged mean blood velocity in the portal vein caudal to the origin of the shunt ranged from 13·0 to 21·3 cm/second.

Splenocaval shunts

The other 11 dogs had splenocaval shunts and the directions of blood flow before, after and four weeks after the shunts were attenuated are shown in Table 1. Before the operation the ultrasonographic findings were identical in all of the 11 dogs; the liver was small, the left and right portal branches could not be identified, and the directions of flow in the shunt and in the segment of portal vein between the origin of the shunt and the point of entry of the gastroduodenal vein were hepatofugal (Fig 3).

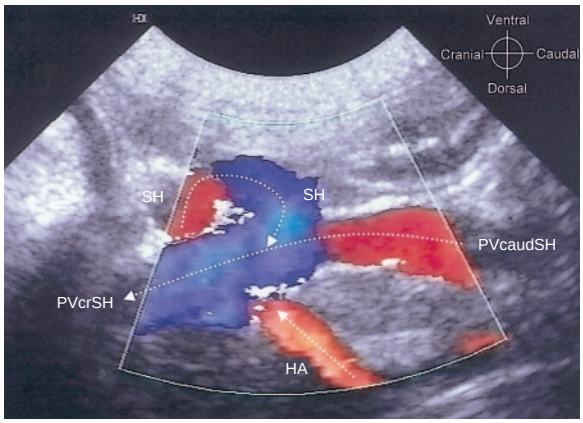


FIG 2: Colour Doppler ultrasound image, made via the left flank of a Maltese dog in right lateral recumbency, of a congenital extrahepatic right gastric-caval shunt, four weeks after partial surgical attenuation. The image shows the origin of the cranial shunt-loop and arrows indicate the direction of blood flow. The directions of flow in the shunt (SH) and throughout the portal vein are hepatopetal. The portal vein cranial to the shunt origin (PVcrSH) has the same diameter as the portal vein caudal to the origin (PVcaudSH). HA Hepatic artery

TABLE 1: Directions of blood flow before, immediately after and four weeks after the partial surgical attenuation of a congenital extrahepatic portosystemic shunt, and the clinical outcomes in three dogs with right gastric-caval shunts and 11 dogs with splenocaval shunts*

Outcome	Number of dogs	Before attenuation	Immediately after attenuation	Four weeks after attenuation
Right gastric-caval shunts	3			
Excellent	2	–↓← Small liver	←↑←	←↑← (Fig 2) Normal liver
Good	1	–↓← Small liver	←↓←	←↓← Normal liver
Splenocaval shunts	11			
Excellent	5	→↓← (Fig 3) Small liver	←↑← (Fig 4a)	←↑← (Figs 4b, c) Normal liver
Excellent	1	→↓← (Fig 3) Small liver	←↓← (Fig 5a)	←↑← (Figs 4b, c) Normal liver
Good	1	→↓← (Fig 3) Small liver	←↓← (Fig 5a)	←↓← (Figs 5b, c) Normal liver
Fair	2	→↓← (Fig 3) Small liver	→↓← (Fig 6a)	←↓← ≈ (Figs 1, 5b) Asymmetric liver
Fair	1	→↓← (Fig 3) Small liver	←↑– (Fig 8)	←↓← ≈ (Figs 1, 5b) Normal liver
Poor	1	→↓← (Fig 3) Small liver	→↓← (Fig 6a)	→↓← Small liver

* Outcome categories were defined on the basis of the portal haemodynamics four weeks postoperatively. The arrows indicate the directions of blood flow as shown on Fig 4a: the first horizontal arrow represents the portal vein cranial to the origin of the shunt, 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 origin of the shunt
←and ↑ Hepatopetal flow , → and ↓ Hepatofugal flow , – Zero flow, ≈ Acquired portosystemic collaterals

In the 11 dogs with splenocaval shunts, the time-averaged mean blood velocity in the portal vein caudal to the origin of the shunt ranged from 10·7 to 25·3 cm/second four weeks after surgery.

Dogs with an excellent outcome Immediately after the attenuation of the shunt the flows throughout the portal vein and in the shunt adjacent to the portal vein of five dogs were hepatopetal (Fig 4a). In one other dog the flow throughout the shunt was hepatofugal (Table 1).

Four weeks postoperatively the owners of all six of these dogs reported that the clinical signs had resolved completely. The blood ammonia levels were normal. The liver was symmetrical and it was estimated to be larger than before surgery. There were no ultrasonographic signs of portal hypertension. The left and right portal branches were easily recognisable. The direction of blood flow throughout the portal vein, in the portal branches and in the shunt adjacent to the portal vein was hepatopetal (Fig 4b). In the segment of shunt between the splenic vein and the portal vein the direction of flow was hepatopetal, and in the segment between the splenic vein and the caudal vena cava it was hepatofugal, some of the blood from the splenic vein flowing via the shunting vessel to the portal vein and the rest flowing via the attenuated segment of shunt to the caudal vena cava (Fig 4c).

Dog with a good outcome In one dog, immediately after the shunt had been attenuated the flow of blood throughout the portal vein was hepatopetal and the flow in the shunt adjacent to the portal vein was hepatofugal (Fig 5a).

Four weeks postoperatively the owner of the dog reported that the clinical signs had resolved completely. Its blood ammonia level was normal and there were no ultrasonographic signs of portal hypertension. The liver was normal in size and symmetrical. The left and right portal branches were easily recognisable. The direction of blood flow throughout the portal vein (Fig 5b) and in the portal branches was hepatopetal, and throughout the shunt it was hepatofugal (Fig 5c).

Dogs with a fair outcome In two of the three dogs (a four-month-old cairn terrier and an eight-month-old miniature schnauzer) classified as having a fair outcome, the flow of blood in the shunt and in the portal vein cranial to the origin of the shunt immediately after the shunt had been attenuated was hepatofugal (Fig 6a), but by four weeks after surgery the direction of flow became hepatopetal throughout the portal vein (Fig 5b, Table 1). In one of the dogs the change of the direction of flow in the portal vein cranial to the origin of the shunt was detected one day after surgery, during an unscheduled ultrasound examination (Fig 6b). After four weeks the livers of both dogs were markedly asymmetrical, the left half being very small and the right half enlarged. The diameters of the portal branches corresponded, the left branch being narrower in both cases than the right, whereas normally the left branch is wider. A histopathological examination of liver biopsies from the small left half of the livers revealed portal vein hypoplasia (Fig 7a), whereas biopsies taken from the enlarged right half had normal hepatic architecture (Fig 7b).

In the third dog with a fair outcome (a nine-month-old Yorkshire terrier), the flow of blood in both the shunt and the portal vein cranial to the origin of the shunt was hepatopetal immediately after the attenuation of the shunt, but zero in the portal vein caudal to the origin of the shunt (Fig 8). However, four weeks after surgery the direction of flow in the shunt was

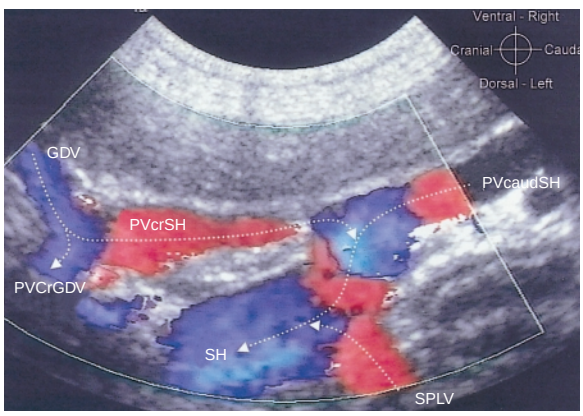
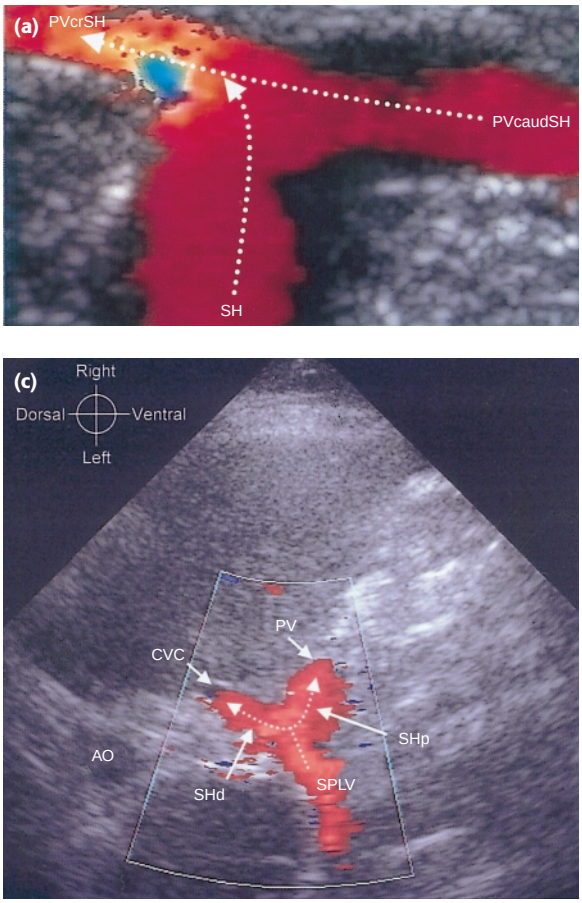


FIG 3: Transabdominal colour Doppler ultrasound image of a congenital extrahepatic splenocaval shunt before attenuation. Arrows indicate the direction of blood flow. The direction of flow is hepatofugal in the segment of portal vein between the origin of the shunt (SH) and the point of entry of the gastroduodenal vein (GDV) because most of the blood from the GDV flows caudally to the shunt. The blood from the intestines and from the spleen is shunting. PVcaudSH Portal vein caudal to the shunt origin, PVcrSH Portal vein segment between the shunt origin and the point of entry of the gastroduodenal vein, PVcrGDV Portal vein cranial to the point of entry of the gastroduodenal vein, SPLV Splenic vein



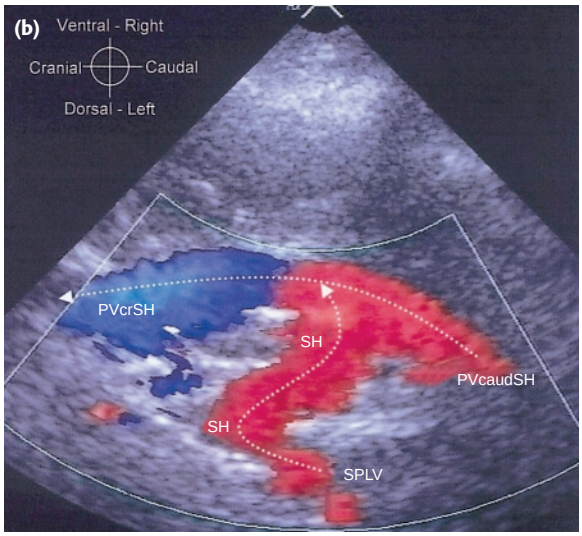


FIG 4: Changes in blood flow induced by the partial attenuation of a splenocaval shunt with an excellent outcome; the direction of the blood flow is indicated by the dotted arrows. (a) Longitudinal intraoperative colour Doppler ultrasound image made immediately after partial attenuation of the shunt showing hepatopetal flow in the shunt (SH) and in the portal vein cranial (PVcrSH) and caudal (PVcaudSH) to the origin of the shunt. PVcrSH has the smallest diameter and the highest flow velocity. (b) Longitudinal transabdominal colour Doppler ultrasound image via the right flank, four weeks after partial attenuation of the shunt. The blood from the splenic vein (SPLV) flows via the SH to the portal vein, preventing the portal blood from shunting. The direction of flow throughout the portal vein is hepatopetal (compare with Fig 3). (c) Transverse transabdominal colour Doppler ultrasound image via a right intercostal space, four weeks after partial attenuation of the shunt. The SPLV enters the middle of the shunting vessel. Some of the splenic venous blood flows to the portal vein (PV) via the proximal segment of the shunt (SHp) and the rest flow to the caudal vena cava (CVC) via the distal segment of the shunt (SHd); only splenic blood is shunting. AO Aorta

hepatofugal (Fig 5c) and the flow throughout the portal vein was hepatopetal (Fig 5b). The liver was symmetrical and it was estimated to be larger than before surgery (Table 1).

Four weeks postoperatively the owners of all three dogs reported that the clinical signs had resolved completely; however, the dogs' blood ammonia levels were high. The left and right portal branches were easily recognisable. The blood flow throughout the portal vein (Fig 5b) and in the portal branches was hepatopetal, but it was hepatofugal throughout the shunt (Fig 5c). In addition to the functional congenital shunt, there were acquired collaterals in each of the three dogs (Fig 1).

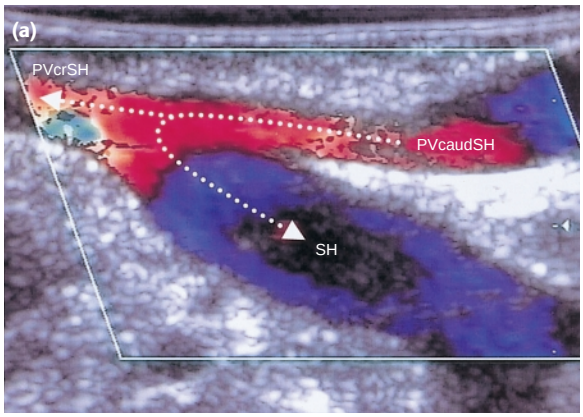
Dog with a poor outcome In one dog (a four-month-old rottweiler) the results of the ultrasonographic examinations during and after the surgery did not differ from each other (Table 1, Fig 6a). Four weeks postoperatively the owner had observed no clinical improvement. The dog's blood ammonia level was high. Its liver was small and the left and right portal branches could not be identified. There were no ultrasonographic signs of portal hypertension. The flow of blood in both the shunt and the portal vein cranial to the origin of the shunt was hepatofugal.

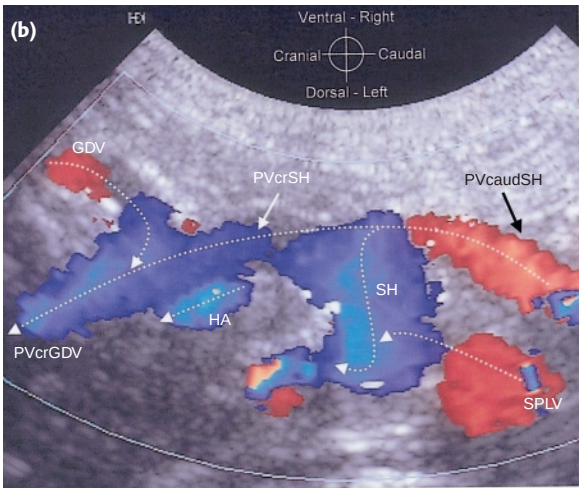
DISCUSSION

A partially attenuated and patent portosystemic shunt is generally assumed to be functional and therefore clinically significant (Swalec and Smeak 1990, Hottinger and others 1995, Komtebedde and others 1995, Harvey and Erb 1998, Howe and Boothe 2002), otherwise whether a shunt has been partially or completely occluded would not be an issue, and an attempt to complete the occlusion would not be considered. However, there is a substantial difference between the anatomy, and therefore the postoperative evaluation, of intrahepatic and extrahepatic shunts. An intrahepatic shunt is the direct continuation of the trunk of the portal vein via the left or the right portal branch, and if there is blood flow via the attenuated segment of the shunt the toxin-rich blood in the portal vein keeps shunting into the caudal vena cava, and can be responsible for hyperammonaemia. In contrast, in an extrahepatic shunt there is a T-shaped junction at the point of origin of the shunt because the portal vein bifurcates into the shunting vessel as well as continuing to the liver; furthermore, the splenic vein or the gastric veins are in direct connection with the shunting vessel. As a result, if blood is shunting via the attenuated part of an extrahepatic shunt the toxin-rich blood of the portal vein is not necessarily being shunted, because the blood may have originated from a portal tributary.

When a portosystemic shunt is completely occluded during surgery, hyperammonaemia can be the result of either the development of acquired portosystemic collaterals, or the opening of the shunt by the degradation of the suture material, or the presence of a second, previously unrecognised portosystemic shunt.

When a shunt is only partially occluded, hyperammonaemia can be the result of persistent shunting via the attenuated shunt, either with or without the development of acquired portosystemic collaterals. It is essential to differentiate between these two conditions because the development





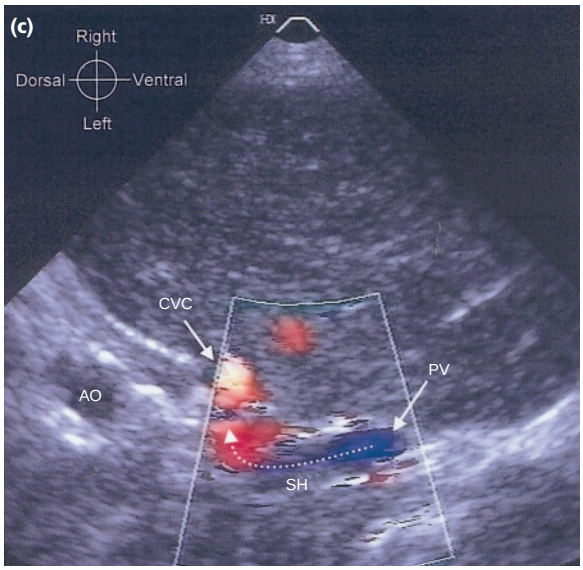


FIG 5: Changes in blood flow induced by the partial attenuation of a splenocaval shunt with a good outcome; the direction of the blood flow is indicated by dotted arrows. (a) Longitudinal intraoperative colour Doppler ultrasound image made immediately after partial attenuation of the shunt showing hepatofugal flow in the shunt (SH) and hepatopetal flow in the portal vein both cranial (PVcrSH) and caudal (PVcaudSH) to the origin of the shunt (compare with Fig 4a). (b) Longitudinal transabdominal colour Doppler ultrasound image via the right flank, four weeks after partial attenuation of the shunt. The direction of flow throughout the portal vein is hepatopetal and in the SH it is hepatofugal. The blood in the splenic vein (SPLV) and in the portal vein caudal to the origin of the shunt (PVcaudSH) is shunting (compare with Figs 3 and 4b). HA Hepatic artery, GDV gastroduodenal vein, PVcrSH Portal vein between the origin of the shunt and the point of entry of the GDV, PVcrGDV Portal vein cranial to the point of entry of the GDV. (c) Transverse transabdominal colour Doppler ultrasound image made via a right intercostal space, four weeks after partial attenuation of the shunt. The direction of the blood flow throughout the SH is hepatofugal, so that the toxin-rich blood from the portal vein (PV) flows to the caudal vena cava (CVC). The stenosis of the SH caused by the ligature is clearly visible next to the CVC. In the lumen of the CVC the orange colour signal indicates a rapid blood flow (poststenotic jet). AO Aorta

of acquired portosystemic collaterals makes it impossible to occlude the shunt further.

The progressive remission of portosystemic shunting has been observed in partially attenuated intra- and extrahepatic portosystemic shunts, probably as a result of the spontaneous mechanical closure of the shunt by thrombosis and scar formation (van Vechten and others 1994, Meyer and others 1999, Szatmári and others 2003). However, the results of this study show that a patent extrahepatic portosystemic shunt 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 (van Vechten and others 1994, Meyer and others 1999). Scintigraphy cannot differentiate between portosystemic shunting through congenital shunts and acquired collaterals, and is therefore of little value for the follow-up evaluation of attenuated congenital shunts, because it cannot help in the decision whether to undertake a second operation. Angiography can determine whether the shunting occurs via the attenuated congenital shunt or through acquired collaterals, but requires the use of ionising radiation and anaesthesia. The most commonly used angiographic techniques for evaluating portosystemic shunting are intraoperative mesenteric portography and splenoportography. The advantage of mesenteric portography is that a jejunal or mesenteric vein is catheterised, so that the route of the toxin-rich blood can be followed; however, the procedure involves a laparotomy. When mesenteric portography detects acquired collaterals a high anaesthetic risk has been taken merely for diagnostic purposes.

Splenoportography can be performed without a laparotomy by the ultrasound-guided injection of contrast medium into a parenchymal splenic vein, but the results can be misleading. As was shown by colour Doppler ultrasonography in the present study, the splenic vein plays a central role in the portal haemodynamics of partially attenuated extrahepatic congenital portosystemic shunts. This pressure-balancing role of the splenic venous blood is the result of the fact that the splenic vein enters the shunt halfway between the portal vein and the caudal vena cava. In a non-functional shunt, some of the splenic blood keeps flowing through the attenuated shunt to the caudal vena cava and the rest 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, and shunting of the splenic blood therefore 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. However, splenoportography cannot differentiate between blood shunted from the splenic or the mesenteric vein, and it therefore also indicates portosystemic shunting when only the blood of the splenic vein flows through the shunt, and may result in false-positive findings. This phenomenon explains the observations of van Vechten and others (1994) that despite evidence that a shunt was

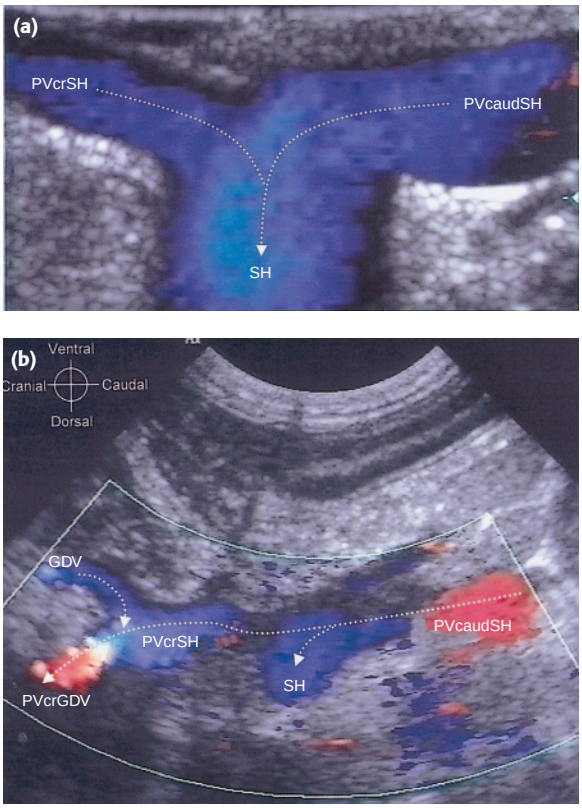


FIG 6: Changes in blood flow induced by the partial attenuation of a splenocaval shunt with only a fair outcome owing to primary hypoplasia of the portal vein. Arrows indicate the direction of blood flow. (a) Longitudinal intraoperative colour Doppler ultrasound image of a partially ligated congenital extrahepatic splenocaval shunt immediately after partial attenuation of the shunt. The flow direction is hepatofugal in the shunt (SH) and in the portal vein cranial to the origin of the shunt (PVcrSH). There is normal flow in the portal vein caudal to the origin of the shunt (PVcaudSH). (b) Longitudinal transabdominal colour Doppler ultrasound image via the right flank, one day after partial attenuation of the shunt. The flow throughout the portal vein has become hepatopetal. The lumen of the segment of portal vein cranial to the point of entry of the gastroduodenal vein (PVcrGDV) is very narrow and the velocity of flow is therefore very high (aliasing artefact)

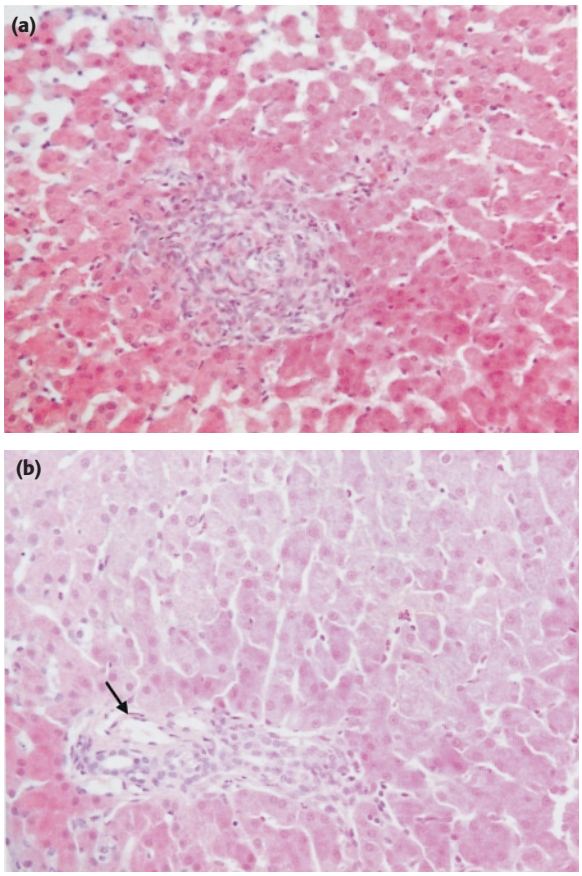


FIG 7: Photomicrographs of liver biopsy specimens taken from a cairn terrier four weeks after the partial attenuation of a congenital extrahepatic splenocaval shunt. (a) Portal vein hypoplasia in the small left liver lobes, showing unrecognisable portal branches and arterial proliferation in the portal area. (b) Normal hepatic architecture in the enlarged right liver lobes; the portal vein is easily recognisable (arrow). Haematoxylin and eosin. x **20**

patent, the shunt-values determined by scintigraphy were within the reference range.

In the present study the classification of the outcome was based on the haemodynamics determined ultrasonographically. If telephone interviews or questionnaires had been used to evaluate the surgical outcome, as in other studies (Johnson and others 1987, Hunt and Hughes 1999), only the rottweiler would have had an unfavourable outcome. In fact, the rottweiler was not considered to have a completely unfavourable outcome because there was no evidence of the development of acquired portosystemic collaterals, and further attenuation of its congenital shunt could have resulted in a favourable outcome. The outcome was considered to be only fair when acquired portosystemic collaterals developed because they represent a type of portosystemic shunting that cannot be further corrected and which may later cause the clinical signs to recur. However, all the dogs with acquired portosystemic collaterals were clinically healthy when examined four weeks after surgery. The results showed that the clinical signs ceased once the blood from the portal vein reached the liver, that is, hepatopetal flow was detected in the portal vein cranial to the origin of the shunt, whether or not there were acquired collaterals or a functional congenital shunt. Hepatopetal portal flow cranial to the origin of the shunt means that the blood from all the portal tributaries flows via the portal branches to the hepatic sinusoids, a great improvement to the circulation of the liver. Preoperatively, none or only some of the blood from the gastroduodenal vein reaches the liver of dogs with an extrahepatic portosystemic shunt, and this is insufficient for normal hepatic development and function. The improved postoperative portal circulation of the liver results in enlargement of the lobes of the liver and blood flow in the right and left portal branches.

A major aetiological difference in the development of acquired portosystemic collaterals was observed in the three dogs that developed sustained portal hypertension. In the two dogs with a primary portal vein hypoplasia, the direction of the blood flow in the portal vein segment cranial to the origin of the shunt could not be changed from hepatofugal to hepatopetal even by a temporary total occlusion of the shunt. In contrast, in the third dog an exaggerated attenuation of the shunt was the probable cause of the development of acquired portosystemic collaterals. If a slight attenuation of a shunt results in hepatopetal flow in the portal vein segment cranial to the origin of the shunt and in the shunt, further attenuation is unnecessary because it does not reduce the shunting of the portal blood, but increases the level of portal hypertension because a larger proportion of the splenic venous blood is forced to flow to the narrow segment of portal vein that is cranial to the origin of the shunt instead of to the caudal vena cava. The absence of flow in the portal vein caudal to the origin of the shunt is an indication of portal hypertension. Subsequently, the sustained portal hypertension caused the development of acquired portosystemic collaterals and the resumption of hepatofugal flow in the shunt. If the congenital shunt had been attenuated to a smaller extent, then the direction of flow in the shunt might have remained hepatopetal and an excellent rather than a fair outcome might have been achieved. During surgical attenuation of an extrahepatic congenital shunt, ultrasonography should be used to determine the least attenuation that maintains a hepatopetal flow in the shunt and throughout the portal vein. Further attenuation would increase the level of portal hypertension without reducing the volume of blood shunted or improving the circulation of the liver. When the flow in the shunt and in the portal vein cranial to the origin of the shunt remains

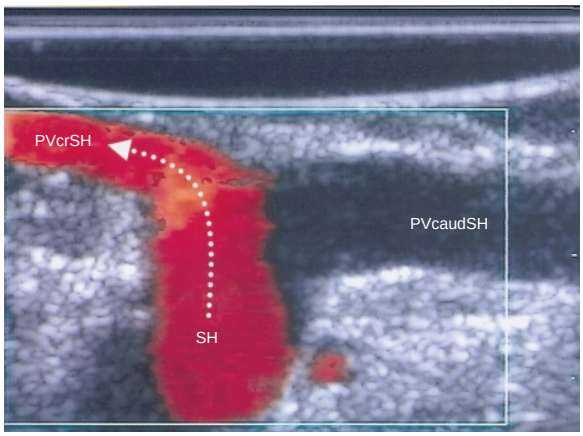


FIG 8: Colour Doppler ultrasound image of a splenocaval shunt in a Yorkshire terrier taken during surgery to attenuate the shunt. The outcome was only fair, probably owing to the attenuation being too severe. The direction of flow in the shunt (SH) and in the portal vein cranial to the origin of the shunt (PVcrSH) (arrow) is hepatopetal. No flow can be detected in the portal vein caudal to the origin of the shunt (PVcaudSH), an indication of portal hypertension (compare with Figs 4a and 6a)

hepatofugal during a temporary total occlusion of the shunt, the shunt should be attenuated to the smallest diameter that ensures continuous blood flow in the portal vein caudal to the origin of the shunt (Szatmári and others 2004b). An excellent outcome can be obtained whether the shunt is partially or completely occluded, and the degree of attenuation has to be determined in each individual dog on the basis of the presence and direction of blood flow in the portal vein and the shunt.

In four of the dogs the ultrasonographic findings were different immediately after the shunt had been attenuated and four weeks after surgery, indicating that clinically relevant haemodynamic changes may develop, which may lead to clinical improvement or deterioration. However, in most cases, the postoperative haemodynamic changes in the portal vein may be predicted on the basis of the intraoperative ultrasound findings, especially in dogs with a fair outcome.

In one dog with an excellent outcome the direction of flow in the shunt changed from hepatofugal to hepatopetal. Local inflammation around the ligature may have further reduced the diameter of the shunt, and the resistance towards the liver may have been reduced by the dilation of the small portal branches. As a result, the attenuated segment of the shunt could not accommodate the total flow of splenic venous blood, and some of it was forced to flow to the portal vein.

In two dogs with a fair outcome (a cairn terrier and a miniature schnauzer) the direction of flow in the portal vein cranial to the origin of the shunt changed from hepatofugal to hepatopetal after the operation. It is possible that the ligation of the shunt resulted in the development of severe portal hypertension, and the congested portal blood caudal to the origin of the shunt was forced to flow to every possible direction to reach the lower pressure systemic venous system, that is, towards the attenuated shunt, towards the hepatic sinusoids, and towards tiny rudimentary vessels which eventually became acquired portosystemic collaterals.

Finally, in one dog with a fair outcome (a Yorkshire terrier) the direction of flow in the shunt changed from hepatopetal to hepatofugal after surgery. Presumably, the shunt had been attenuated too severely, causing the development of severe portal hypertension. The portal vein cranial to the origin of the shunt was unable to accommodate the blood from both the splenic vein and the mesenteric vein. The vascular bed of the splenic vein appears to have a lower reserve capacity to dilate than that of the jejunal veins, because during the operation there was continuous hepatopetal flow in the dog's splenic vein and shunt, but no flow was detectable in the portal vein caudal to the origin of the shunt immediately after the shunt had been attenuated. The congested portal blood was presumably forced to flow to every possible direction to reach the lower pressure systemic veins, as in the cairn terrier and miniature schnauzer. As a result, normal portal flow caudal to the origin of the shunt was restored, but the flow in the shunt became hepatofugal, which means that the shunt became functional again, and acquired portosystemic collaterals developed to alleviate the severe portal hypertension.

On the basis of these observations the following conclusion can be drawn: when the direction of flow in a partially attenuated extrahepatic portosystemic shunt is hepatopetal adjacent to the portal vein, there is no portosystemic shunting, either congenital or acquired.

The mean portal blood flow velocities measured four weeks postoperatively in the 14 dogs were within the reference range (18·1±7·6 cm/second) that was established in normal dogs by Nyland and Fisher (1990). Apparently, the first priority of the portal circulation after the onset of portal hypertension due to the ligation of a shunt is to restore and maintain the physiological blood flow in the portal vein. In this study the velocity of flow in the portal vein caudal to the origin of the shunt did not give any additional information relevant to the presence of portal hypertension or the level of portal perfusion. However, a hepatopetal direction of flow in the portal vein cranial to the origin of the shunt four weeks after the surgery was correlated with a favourable clinical outcome. Although the dogs with acquired portosystemic collaterals or functional congenital shunts were clinically healthy four weeks after surgery, they might have had an increased risk of the clinical signs recurring.

Primary and secondary portal vein hypoplasia cannot be differentiated by the histopathological examination of the liver (van den Ingh and others 1995). A possible unfavourable outcome in dogs with a coexisting primary portal vein hypoplasia cannot therefore be predicted before a congenital portosystemic shunt is attenuated, because secondary portal vein hypoplasia, which is the result of hypoperfusion of the liver, is always present in dogs with such a shunt. Once the portal circulation has become normal postoperatively, that is, there is hepatopetal flow throughout the portal vein, the histopathological signs of secondary portal vein hypoplasia disappear. In contrast, primary portal vein hypoplasia cannot be resolved by restoring normal portal flow. In two dogs with a fair outcome evidence of primary portal vein hypoplasia was found both ultrasonographically and histologically when they were examined four weeks after surgery. In both dogs the abnormality affected only the left portal branch, which supplies the larger part of the liver (four lobes), whereas the right portal branch supplies only the right lateral lobe and the caudate lobe. A hypoplastic right portal branch may have no clinical significance, if it exists at all. It is not possible to diagnose primary portal vein hypoplasia preoperatively in dogs with a congenital portosystemic shunt, but the knowledge would not affect the decision of whether a dog with an extrahepatic shunt should undergo surgery. Observations from the present study suggest that every dog under the age of six years with a shunt should undergo surgery because, even if the dog has primary portal vein hypoplasia and the attenuation of the shunt would cause the development of acquired portosystemic collaterals, normal portal circulation can be restored by changing the hepatofugal or zero flow in the portal vein cranial to the origin of the shunt to hepatopetal, which would ensure normal hepatic development and function. The clinical signs of hepatic encephalopathy also usually disappear once the flow becomes hepatopetal throughout the portal

vein because the amount of mesenteric blood that used to shunt has markedly decreased, and the acquired portosystemic collaterals probably divert predominantly the splenic venous blood, because splenorenal collaterals are the most common type in dogs (Vitums 1959).

None of the dogs in this study was more than six years old, because an earlier study had recorded significantly worse clinical outcomes in older animals (Wolschrijn and others 2000); the reasons are unknown, however, because Doppler ultrasonography was not part of the protocol in that study. A prospective study, including older dogs and applying ultrasonography during and after surgery, should help to diagnose the portal haemodynamic reasons for the less favourable outcomes in older animals.

When a dog has acquired portosystemic collaterals or the blood flow in the shunt adjacent to the portal vein is hepatopetal, no second surgery for further attenuation of a congenital shunt should be carried out. However, when the direction of flow in the portal vein cranial to the origin of the shunt is hepatofugal, additional surgery is recommended. When the flow is hepatopetal throughout the portal vein and hepatofugal in the shunt, further attentuation of the shunt should be considered, depending on the dog's clinical signs.

Ultrasonography is a reliable method for evaluating the haemodynamics of the portal vein, and helpful in deciding whether further surgery is required after the partial attenuation of a congenital portosystemic shunt. Doppler ultrasonography gives more information in a non-invasive way than angiography or diagnostic laparotomy. The minimum information required from an ultrasound examination after surgery is the direction of blood flow in the portal vein cranial to the origin of the shunt, the direction of flow in the shunt adjacent to the portal vein, and the width of the left gonadal vein as an indication of the presence of acquired portosystemic collaterals.

ACKNOWLEDGEMENTS

Dr Szatmári was supported by the Hungarian State Eötvös Scholarship. Mr Aart van der Woude helped in preparing the illustrations.

References

HARVEY, J. & ERB, H. N. (1998) Complete ligation of extrahepatic congenital portosystemic shunts in nonencephalic dogs. *Veterinary Surgery* **27,** 413-416

HOTTINGER, H. A., WALSHAW, R. & HAUPTMAN, J. G. (1995) Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. *Veterinary Surgery* **24,** 331-336

HOWE, L. M. & BOOTHE, H. W. (2002) Diagnosing and treating portosystemic shunts in dogs and cats. *Veterinary Medicine* **97,** 448-459

HUNT, G. B. & HUGHES, J. (1999) Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. *Australian Veterinary Journal* **77,** 303-307

JOHNSON, C. A., ARMSTRONG, P. J. & HAUPTMAN, J. G. (1987) Congenital portosystemic shunts in dogs: 46 cases (1979-1986). *Journal of the American Veterinary Medical Association* **191,** 1478-1483

JOHNSON, S. E. (1987) Portal hypertension. Part I. Pathophysiology and clinical consequences. *Compendium on Continuing Education for the Practicing Veterinarian* **9,** 741-748

KOMTEBEDDE, J., KOBLIK, P. D., BREZNOCK, E. M., HARB, M. & GAROW, L. A. (1995) Long-term clinical outcome after partial ligation of single extrahepatic vascular anomalies in 20 dogs. *Veterinary Surgery* **24,** 379-383

LAWRENCE, D., BELLAH, J. R. & DIAZ, R. (1992) Results of surgical management of portosystemic shunts in dogs: 20 cases (1985-1990). *Journal of the American Veterinary Medical Association* **201,** 1750-1753

MATHEWS, K. & GOFTON, N. (1988) Congenital extrahepatic portosystemic shunt occlusion in the dog: gross observations during surgical correction. *Journal of the American Animal Hospital Association* **24,** 387-394

MEYER, H. P., ROTHUIZEN, J., VAN SLUIJS, F. J., VOORHOUT, G. & VAN DEN BROM, W. E. (1999) Progressive remission of portosystemic shunting in 23 dogs after partial closure of congenital portosystemic shunts. *Veterinary Record* **144,** 333-337

NYLAND, T. G. & FISHER, P. E. (1990) Evaluation of experimentally induced canine hepatic cirrhosis using duplex Doppler ultrasound. *Veterinary Radiology* **31,** 189-194

ROTHUIZEN, J., VAN DEN INGH, T. S. G. A. M., VOORHOUT, G., VAN DER LUER, R. J. T. & WOUDA, W. (1982) Congenital porto-systemic shunts in sixteen dogs and three cats. *Journal of Small Animal Practice* **23,** 67-81

ROY, R. G., POST, G. S., WATERS, D. J. & HARDY, R. M. (1992) Portal vein thrombosis as a complication of portosystemic shunt ligation in two dogs. *Journal of the American Animal Hospital Association* **28,** 53-58

SCAVELLI, T. D. (1989) Complications associated with the diagnostic, medical, and surgical management of portosystemic shunts. *Problems in Veterinary Medicine* **1,** 147-158

SMITH, K. R., BAUER, M. & MONNET, E. (1995) Portosystemic communications: follow-up of 32 cases. *Journal of Small Animal Practice* **36,** 435-440

SWALEC, K. M. & SMEAK, D. D. (1990) Partial versus complete attenuation of single portosystemic shunts. *Veterinary Surgery* **19,** 406-411

SZATMÁRI, V., ROTHUIZEN, J., VAN DEN INGH, T. S. G. A. M., VAN SLUIJS, F. J. & VOORHOUT, G. (2004a) Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). *Journal of the American Veterinary Medical Association* **224,** 717-727

SZATMÁRI, V., SÓTONYI, P. & VÖRÖS, K. (2001) Normal duplex Doppler waveforms of the major abdominal blood vessels in dogs: a review. *Veterinary Radiology & Ultrasound* **42,** 93-107

SZATMÁRI, V., VAN SLUIJS, F. J., ROTHUIZEN, J. & VOORHOUT, G. (2003) Intraoperative ultrasonography of the portal vein during attenuation of intrahepatic portocaval shunts in dogs. *Journal of the American Veterinary Medical Association* **222,** 1086-1092

SZATMÁRI, V., VAN SLUIJS, F. J., ROTHUIZEN, J. & VOORHOUT, G. (2004b) Ultrasonographic assessment of hemodynamic changes in the portal vein during surgical attention of congenital extrahepatic portosystemic shunts in dogs. *Journal of the American Veterinary Medical Association* **224,** 395-402

THURMON, J. C., TRANQUILLI, W. J. & BENSON, G. J. (1996) Considerations for general anesthesia. In Lumb & Jones' Veterinary Anaesthesia. Eds J. C. Thurmon, W. J. Tranquilli, G. J. Benson. Baltimore, Lea & Febiger. pp 5-34

VAN DEN INGH, T. S. G. A. M., ROTHUIZEN, J. & MEYER, H. P. (1995) Circulatory disorders of the liver in dogs and cats. *Veterinary Quarterly* **17,** 70-76

VAN VECHTEN, B. J., KOMTEBEDDE, J. & KOBLIK, P. D. (1994) Use of transcolonic portal scintigraphy to monitor blood flow and progressive postoperative attenuation of partially ligated single extrahepatic portosystemic shunts in dogs. *Journal of the American Veterinary Medical Association* **204,** 1770-1774

VITUMS, A. (1959) Portosystemic communications in the dog. *Acta Anatomica (Basel)* **39,** 271-299

VOGT, J. C., KRAHWINKEL, D. J., BRIGHT, R. M., DANIEL, G. B., TOAL, L. R. & ROHRBACH, B. (1996) Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using the ameroid constrictor. *Veterinary Surgery* **25,** 495-502

WOLSCHRIJN, C. F., MAHAPOKAI, W., ROTHUIZEN, J., MEYER, H. P. & VAN SLUIJS, F. J. (2000) Gauged attenuation of congenital portosystemic shunts: results in 160 dogs and 15 cats. *Veterinary Quarterly* **22,** 94-98

Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in 14 dogs

V. Szatmári, G. Voorhout, J. Rothuizen, et al.

Veterinary Record 2004 155: 448-456
doi: 10.1136/vr.155.15.448