# Ultrasonographic assessment of hemodynamic changes in the portal vein during surgical attenuation of congenital extrahepatic portosystemic shunts in dogs

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**Objective**—To determine portal hemodynamic changes associated with surgical shunt ligation and establish ultrasonographic criteria for determining the optimal degree of shunt narrowing and predicting outcome.

**Design**—Case series.

**Animals**—17 dogs, each with a single congenital extrahepatic portosystemic shunt.

**Procedure**—Pre- and postligation flow velocities and flow directions were determined by Doppler ultrasonography intraoperatively in the shunt and in the portal vein cranial and caudal to the shunt origin. Outcome was evaluated 1 month after surgery by measuring blood ammonia concentration and performing abdominal ultrasonography.

**Results**—Hepatofugal flow was detected in 9 of 17 dogs before shunt attenuation in the portal segment that was between the shunt origin and the entering point of the gastroduodenal vein. If hepatofugal flow became hepatopetal after shunt ligation, hyperammonemia resolved. Hepatofugal portal flow was caused by blood that flowed from the gastroduodenal vein toward the shunt. Shunt attenuation converted hepatofugal flow to hepatopetal in the shunt in 12 of 17 dogs. Chronic portal hypertension developed or perioperative death occurred when the portal congestion index caudal to the shunt origin increased by > 3.6 times.

**Conclusions and Clinical Relevance**—After hepatopetal flow in the cranial portal vein and the shunt is established, further shunt narrowing is contraindicated. Increase of the portal congestion index caudal to the shunt > 3.5 times should be avoided. Poor outcome because of severe hypoplasia of the portal branches can be expected if the flow direction remains hepatofugal after shunt occlusion cranial to the shunt origin. (*J Am Vet Med Assoc* 2004;224:395–402)

Portosystemic shunts are abnormal macroscopic vascular connections that allow the portal blood to directly enter the systemic venous circulation, bypassing the hepatic sinusoids, and can be classified according to their anatomy as intra- or extrahepatic.[1-9] An extrahepatic portosystemic shunt is thought to be congenital if a single (rarely double) anomalous vein is present without concurrent portal hypertension.[1] Extrahepatic **congenital portosystemic shunts (CPSSs)** can result in clinical signs of hepatic encephalopathy in young dogs, but may also remain clinically inapparent until old age.[3,5,8]

The definitive therapy for CPSSs would ideally be complete occlusion of the shunt.[5,7,8] However, in most dogs, underdeveloped (ie, hypoplastic) portal branches allow only partial shunt ligation to be performed.[5-9] Because shunt attenuation forces the blood to flow through the portal branches, which are frequently hypoplastic because of hypoperfusion or as a primary defect,[1,6,10,11] postligation portal hypertension usually develops.[1,5-7,9,11] A greater degree of attenuation is associated with development of more severe portal hypertension.[5-7] Some dogs tolerate complete ligation without developing clinical signs of portal hypertension. Complete shunt occlusion can only be performed if the portal system is well developed and postligation portal hypertension remains mild.[4,5-8]

During surgical ligation of a CPSS, the narrowest possible shunt diameter is determined in steps by assessing portal hypertension.[8] To avoid acute fatal portal hypertension, 2 methods have been recommended for use during surgical shunt ligation, either separately or in combination. Measuring portal pressure by direct catheterization of a portal tributary has been generally used for direct quantitative assessment of portal hypertension.[4,5,12-19] The other method is based on monitoring qualitative signs (color changes of the intestines) and indirect quantitative variables (magnitude of change in mean systemic arterial blood pressure and heart rate) to determine the degree of postligation portal hypertension that is acceptable.[4-6,8,11,20] Both methods allow acute portal hypertension to be successfully avoided; however, development of chronic portal hypertension remains a frequent complication.[4,5,7,9,19]

For several years, ameroid constrictors have been used in surgeries for CPSS to cause gradual attenuation of the shunting vessel, resulting in complete shunt occlusion in 1 to several weeks.[17,21,22] In theory, gradual shunt attenuation allows the underdeveloped portal branches to become adapted to the increased blood flow. Although using an ameroid constrictor does not require intraoperative assessment of portal hypertension, shunt attenuation becomes an uncontrollable

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process. A hypoplastic portal system may not be able to adapt to the increased blood flow at the same rate as the contraction rate of the device; therefore, subacute or chronic portal hypertension can develop.[21]

Cellophane banding of extrahepatic CPSSs is another method that has been used to create gradual shunt occlusion. Its application requires initial narrowing of the shunt, so intraoperative assessment of portal hypertension is necessary.[22,23]

Whichever technique (ie, ligation, ameroid constrictor, or cellophane banding) is used for attenuation of an extrahepatic CPSS, the clinical outcome remains unpredictable[4,5,8,11,12,15-19,21,23] because presently the severity of portal vein hypoplasia cannot be determined either pre- or intraoperatively.[1,6-8,10,11,19] Use of histopathologic changes in the liver,[1,10] portal pressure,[7,9,18] partial versus complete shunt ligation,[7,9,12,18,20,24] age of the dogs,[8,24] and portographic images[7,9] has not yielded satisfactory results.

The purpose of the study reported here was to determine portal hemodynamic changes associated with surgical shunt ligation and establish ultrasonographic criteria for determining the optimal degree of shunt narrowing and predicting outcome.

## Materials and Methods

**Dogs**—Between March 2001 and March 2002, 23 client-owned dogs underwent gauged surgical attenuation of a single extrahepatic CPSS at the Utrecht University Clinic for Companion Animals because of clinical signs of hepatic encephalopathy. The first 6 dogs were not enrolled in this study but were used to establish the examination protocol.

Mean age of the 17 dogs was 18.0 months (range, 4 to 66 months), mean weight was 4.6 kg (10.1 lb; range, 1.3 to 8.1 kg [2.9 to 17.8 lb]), and 10 of 17 were female. A diagnosis of extrahepatic CPSS was made on the basis of high venous blood ammonia concentration (> 45 µmol/L) after 12-hour withholding of food[3,6] combined with direct visualization of the shunt via transabdominal ultrasonography. Spleno-caval (9/17), right gastric-caval (5/17), and spleno-azygos (3/17) shunts were found.

**Technique**—The CPSS of each dog was attenuated surgically with polyester suture material[a] by a single surgeon (FJvS) by use of a reported technique.[8] After premedication (0.3 mL/kg [0.14 mL/lb] with the combination of droperidol and fentanyl,[b] IM; 1 mL contains 2.50 mg of droperidol and 0.05 mg of fentanyl), anesthesia was induced with propofol (3 to 5 mg/kg [1.4 to 2.3 mg/lb], IV) and maintained with inhalation of isoflurane (0.8% to 1.0%) vaporized in oxygen. Intraoperative analgesia was provided with a continuous IV infusion of sufentanil (1 µg/kg/h [0.45 µg/lb/h]). All dogs were ventilated mechanically. Systemic arterial blood pressure was recorded continuously after catheterization of a femoral artery.

For intraoperative ultrasonography, a high-definition ultrasound system[c] was used, equipped with a 26-mm-long, 5- to 10-MHz intraoperative linear-array transducer. The keyboard and transducer were covered with sterile material.[d] All examinations were performed by a single ultrasonographer (VS) in the operating suite and recorded on videotape for further analyses and documentation.

A midline celiotomy was performed, and all organs were left in the abdominal cavity. The descending duodenum was temporarily retracted towards the midline to expose the portal vein and was then released. The ultrasound transducer was placed directly on the portal vein at the point of the shunt origin to obtain a B-mode longitudinal image and a color Doppler image of the portal vein and the shunt. Three consecutive Doppler spectra were obtained in the pulsed-wave Doppler mode[25] from the shunt and the portal vein segments cranial and caudal to the shunt origin. A uniform insonation method was used for the portal vein; for the shunt, the sample volume was adjusted to approximately two-thirds of the shunt diameter.[26,27] Because a linear transducer was used, the Doppler ultrasound beam was directed as needed (beam steering) to obtain good-quality spectra. Time-averaged mean velocities were obtained on frozen pulsed-wave Doppler images by the built-in automatic spectrum analyzer,[e] and the mean of 3 measurements was calculated.

After automatic analysis of at least 3 good-quality Doppler spectra, the transducer was rotated 90° to obtain a cross-sectional B-mode image of the portal vein at the same points where velocity measurements had been taken. The cross-sectional area of the portal vein was determined by a continuous trace method.[c] The congestion index of the portal vein was calculated from the cross-sectional area divided by the time-averaged mean velocity.[28]

The first series of measurements was performed immediately after celiotomy before any manipulation of the shunt, and the second series at least 5 minutes after the gauged shunt attenuation (ie, just before abdominal closure). Ultrasonographic measurements did not influence the surgeon in decision-making about how narrow the shunts would be attenuated. The surgeon determined in steps the narrowest possible shunt diameter that did not cause signs of serious portal hypertension; that is, the intestines remained acyanotic, the heart rate did not increase > 15%, and the systemic mean arterial blood pressure did not decrease > 15%, compared with values recorded at the beginning of surgery.[8]

**Follow-up**—The outcome was assessed 1 month postoperatively by measuring venous blood ammonia concentration (after 12-hour withholding of food) and performing a transabdominal ultrasound examination. Flow directions in the portal vein and shunt were revealed via color Doppler ultrasonography. **Acquired portosystemic collaterals (APSCs)** were diagnosed ultrasonographically, when a wide left gonadal vein was found,[f] as a result of splenorenal collaterals entering the left renal vein from the caudal direction.[10,29] From dogs with hyperammonemia, ultrasound-guided liver biopsy specimens were taken for histopathologic examination.

Five outcome categories were established on the basis of the clinical findings, blood ammonia concentrations, and ultrasonographic results of the 1-month follow-up. The outcome was considered excellent if the dog was healthy, blood ammonia concentration was within reference range, flow direction in the CPSS was hepatopetal, and no APSCs were detected; outcome was considered good if the dog was healthy, blood ammonia concentration was within reference range, flow direction in the CPSS was hepatofugal, and no APSCs were detected; outcome was considered fair if the dog was healthy, blood ammonia concentration was increased, flow direction in the CPSS was hepatofugal, and APSCs were detected; outcome was considered poor if the CPSS could not be attenuated; and outcome was considered fatal if the dog died within 5 days after surgery. The causes of poor, fair, and fatal outcomes are severe portal vein hypoplasia or aplasia, exaggerated shunt attenuation, or both.[1,5,6,8,17,19,21]

**Data analyses**—The intraoperative ultrasonographic findings were not used during the surgeries, but were later evaluated together with the results of the 1-month follow-up. To evaluate the effect of surgical shunt attenuation on outcome, a graph was made[g] of the magnitude of increase of the congestion indices (ie, the postligation congestion index divided by the preligation congestion index) measured in the portal vein caudal to the shunt origin in the 16 dogs in which CPSSs had been attenuated. Dogs with fair and fatal out-

comes were in the poor category, and dogs with excellent and good outcomes were in the good category.

To evaluate association between developmental level of the portal branches and outcome, graphs were made[g] of the preligation portal velocity values measured cranial and caudal to the shunt origin. Dogs with excellent and good outcomes were in the good category, and the dogs with fair, poor, and fatal outcomes were in the poor category; however, 1 dog that developed postligation portal vein thrombosis was placed in the good category because the preligation hepatopetal (ie, physiologic) blood flow direction in the portal vein cranial to the shunt origin excluded a diagnosis of portal vein hypoplasia or aplasia. Because the good and poor groups overlapped in both graphs, the portal flow velocity values cranial and caudal to the shunt origin were combined, creating a **differential portal velocity (DPV)**. The DPV was obtained by subtracting the preattenuation time-averaged mean portal velocity measured cranial to the shunt origin from the preattenuation time-averaged mean portal velocity measured caudal to the shunt origin. Differential portal velocity takes into consideration the direction and velocity of flow in the portal vein cranial and caudal to the shunt origin. Hepatopetal flow was coded with a positive sign and hepatofugal with a negative sign.

## Results

Mean duration of ultrasonography during each surgery was 22 minutes (range, 14 to 35 minutes). Mean duration between the pre- and postligation ultrasonographic measurements was 37 minutes (range, 16 to 60 minutes).

Intraoperative ultrasonography was essential in localizing the shunt in 2 dogs with peritoneal adhesions. Intraoperative clinical observations of the abdominal viscera were similar in each dog (slight discoloration but no obvious cyanosis) after the shunts had been narrowed to the narrowest possible diameter, although the intraoperative ultrasonographic findings and the outcomes were different.

**Outcome**—All dogs recovered from anesthesia; however, 2 of 17 died unexpectedly. Both were alert and ate well from the first postoperative day until sudden collapse 2 days after surgery. In 1 dog, transabdominal ultrasonography revealed that the entire portal vein and the CPSS were filled with a thrombus, whereas the other dog was not returned to the clinic for examination. The dog with portal vein thrombosis was euthanatized. The owners of both dogs refused necropsy.

The shunt could not be attenuated in 1 dog because of aplasia of the portal vein segment cranial to the entering point of the gastroduodenal vein. In the remaining 14 dogs, owners reported complete resolution of clinical signs 1 month postoperatively, although in 2 of these dogs, hyperammonemia persisted because of persistent shunting and collateral vessel formation.

Complete shunt ligation was performed in 6 dogs and partial ligation in 10 dogs. The smallest diameter of the shunt after gauged partial attenuation ranged from 1.5 to 2.5 mm. Complications developed in 1 of the 6 dogs with complete shunt ligation (portal vein thrombosis) and in 3 of the 10 dogs with partial shunt ligation (2 developed APSCs and 1 died).

**Portal hemodynamics during surgery**—Changes in right atrial pressure were reflected in the velocity spectra of dogs with short shunts, although this periodicity was not or hardly recognizable in dogs with long shunts. Shunt attenuation damped or ceased the periodicity.

**Hemodynamics in the shunt**—All dogs had continuous hepatofugal flow in the shunt before shunt ligation (**Fig 1 and 2**). This became hepatopetal in 12 of 16 dogs after shunt attenuation (6 of 6 with complete and 6 of 10 with partial attenuation; **Fig 3 and 4**), and in 4 of 16, the flow remained hepatofugal with decreased flow velocity (all with partial attenuation; **Fig 5**).

**Portal hemodynamics cranial to the shunt origin before shunt ligation**—In 4 of the 5 dogs that had

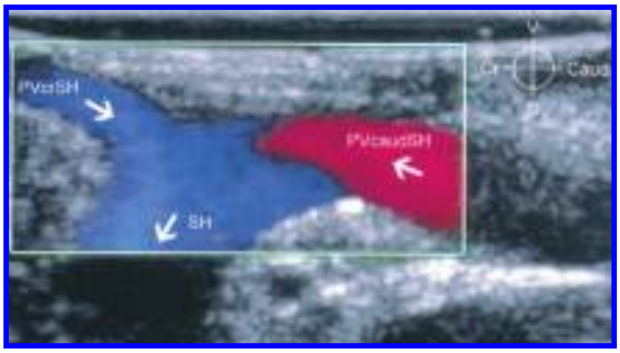


Figure 1—Intraoperative color Doppler ultrasound image of a dog with a congenital extrahepatic portosystemic shunt before shunt attenuation. The portal vein is imaged at the point where the spleno-caval shunt originates. Cranial to the shunt, the portal flow is hepatofugal. Notice that the portal vein is narrower cranial to the shunt origin than caudal to it. Red indicates flow toward the transducer and blue indicates flow away from the transducer. Arrows indicate flow directions. Cr = Cranial. Caud = Caudal. V = Ventral. D = Dorsal. SH = Congenital extrahepatic spleno-caval shunt. PVcrSH = Portal vein segment cranial to the origin of the shunt. PVcaudSH = Portal vein segment caudal to the origin of the shunt.

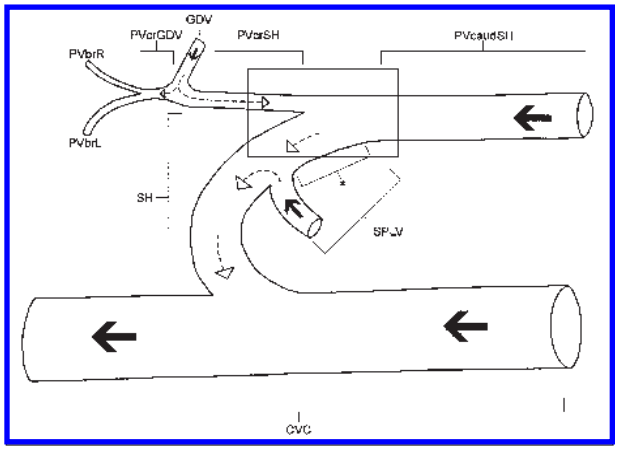


Figure 2—Schematic drawing of a congenital extrahepatic portosystemic shunt before shunt attenuation in a dog. The framed region is illustrated in Fig 1. Gastroduodenal blood is responsible for the hepatofugal portal flow between the shunt origin and the gastroduodenal vein. Black arrows indicate the flow that will not change subsequent to shunt attenuation; open interrupted arrows indicate the flow that may change after shunt attenuation. It appears that the splenic vein enters the shunting vessel and the shunt itself originates from the portal vein. In reality, the shunting vessel originates from the splenic vein, and the shunting blood of the portal vein results in dilation of this segment of the splenic vein and generates hepatofugal flow in it (*). PVbrL = Left portal branch. PVbrR = Right portal branch. GDV = Gastroduodenal vein. PVcrGDV = Portal vein segment cranial to the point where the gastroduodenal vein enters the portal vein. SPLV = Splenic vein. CVC = Caudal vena cava. *See* Figure 1 for remainder of key.

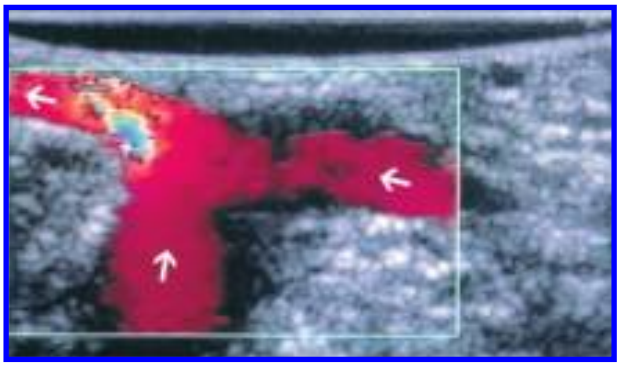

Figure 3—Intraoperative color Doppler ultrasound image of a dog with a congenital extrahepatic portosystemic shunt immediately after partial shunt attenuation. The portal vein is imaged at the point where the spleno-caval shunt originates. After partial shunt attenuation, the flow direction became hepatopetal in the shunt and the portal segment cranial to the shunt origin. Portal flow velocity caudal to the shunt origin is decreased. *See* Fig 1 for key.

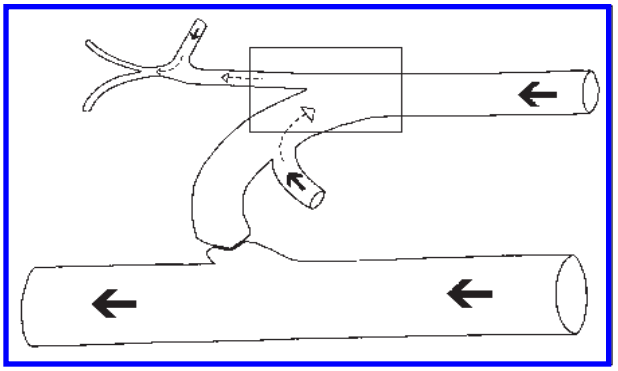

Figure 4—Schematic drawing of a congenital extrahepatic portosystemic shunt after partial shunt attenuation in a dog. The portal vein is imaged at the point where the spleno-caval shunt originates. The framed region is illustrated in Fig 3. Solid arrows indicate the flow that did not change subsequent to shunt attenuation; open, interrupted arrows indicate the flow that changed after shunt attenuation.

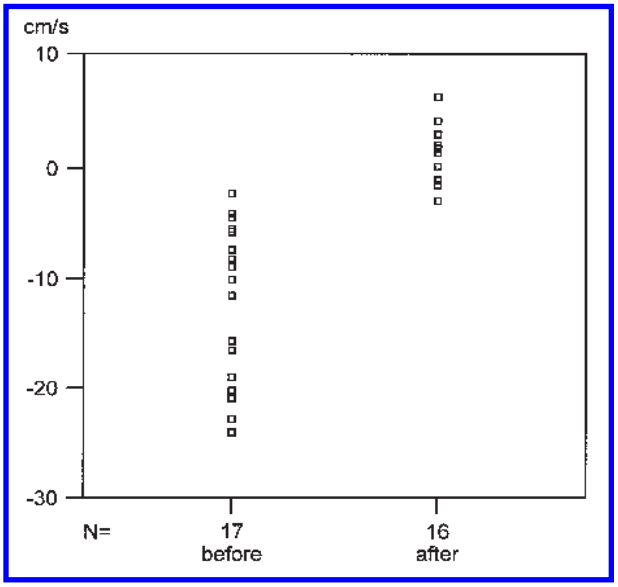


Figure 5—Comparison of time-averaged mean velocities in a congenital extrahepatic portosystemic shunt measured adjacent to the portal vein before (n = 17 dogs) and after (16) shunt attenuation. Positive values indicate hepatopetal blood flow; negative values indicate hepatofugal blood flow.

right gastric-caval shunts, hepatopetal portal flow was detected cranial to the shunt origin before shunt attenuation; in the remaining dog, this portal segment could not be imaged.

In 9 of the 12 dogs in which the shunt originated from the splenic vein, hepatofugal portal flow was found between the shunt origin and the entering point of the gastroduodenal vein before shunt attenuation (**Fig** 1, 2, and **6**). Of the remaining 3 dogs in this group, 2 had to-and-fro flow and 1 had hepatopetal flow.

Although the portal vein cranial to the gastroduodenal vein was macroscopically visible before shunt attenuation, it could not be ultrasonographically imaged in most of the dogs in which the shunt arose from the splenic vein. This resulted from luminal collapse attributable to lack of perfusion. If this portal vein segment was imaged, very slow hepatopetal flow was detected, even if hepatofugal portal flow was observed caudal to the gastroduodenal vein. The diameter of the portal vein gradually decreased from caudal to cranial; the widest segment was that caudal to the shunt origin, the segment between the shunt origin and the gastroduodenal vein was thinner, and the segment between the gastroduodenal vein and the portal bifurcation was the thinnest (Fig 2).

**Portal hemodynamics cranial to the shunt origin after shunt ligation**—After attenuation, the preattenuation hepatopetal flow direction remained unchanged, but increased in velocity. In the 2 dogs with to-and-fro flow and 6 of the 8 dogs with hepatofugal flow, in which shunts were attenuated, flow became hepatopetal immediately after attenuation (Fig 3, 4, and 6). In the 2 dogs in which portal flow direction remained hepatofugal, the shunt flow also remained hepatofugal. These 2 dogs developed APSCs postoperatively, and hyperammonemia persisted; ultrasound-guided liver biopsies

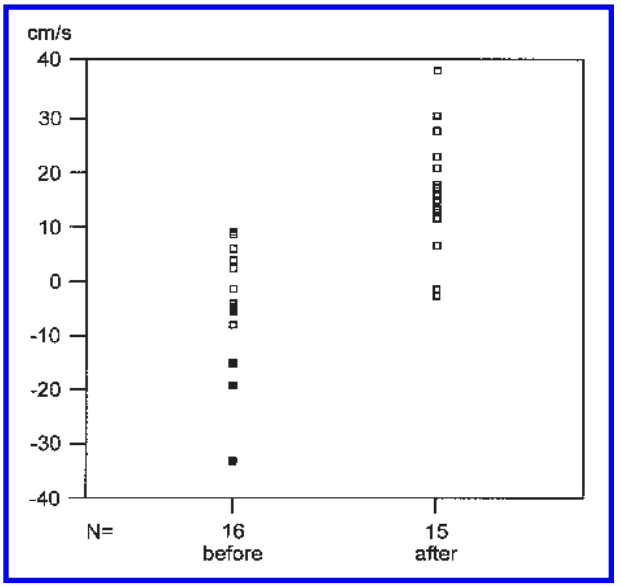


Figure 6—Comparison of time-averaged mean velocities in the PVcrSH before (n = 16) and after (15) shunt attenuation. Solid squares indicate preligation values of 2 dogs that developed acquired portosystemic collaterals 1 month after surgery, a dog with aplasia of the cranial portal vein, and a dog that died suddenly.

performed 1 month postoperatively revealed portal vein hypoplasia in biopsy specimens. When the shunt of the dog with aplasia of the cranial portal vein was temporarily attenuated, both the shunt flow and the portal flow cranial to the shunt remained hepatofugal, while severe visceral cyanosis developed.

The portal flow cranial to the shunt consistently became hepatopetal in dogs in which shunt flow became hepatopetal. However, the reverse did not happen; the shunt flow remained hepatofugal in 2 of the 6 dogs in which hepatofugal portal flow became hepatopetal cranial to the shunt.

**Portal hemodynamics caudal to the shunt origin**—Before shunt attenuation, hepatopetal portal flow was detected caudal to the shunt; this remained hepatopetal after attenuation in all dogs, although the velocity was decreased (**Fig** 1, 3, and 7). After shunt attenuation, a narrow range of velocities was observed; portal flow velocity therefore corresponded to the intestinal color that indicated an acceptable degree of portal hypertension when the shunt was attenuated to the narrowest possible diameter.

**Prediction of outcome**—Differences were found between the variables of dogs with good outcomes and those with poor outcomes regarding intraoperative portal flow directions and velocities. The congestion index of the portal vein measured caudal to the shunt increased > 3.6 times subsequent to shunt attenuation in the 4 dogs with poor outcome. The increase was < 3.6 times in the 12 dogs with good outcomes (**Fig 8**).

When hepatopetal portal flow was detected cranial to the shunt immediately after attenuation, blood ammonia concentration measured 1 month postoperatively had returned to reference range, even when the shunt flow remained hepatofugal (2/16 dogs). One exception was the dog that developed portal vein thrombosis; however, in that dog, the portal congestion index caudal to the shunt increased by 7.4 times. Abdominal ultrasonography did not reveal signs of portal hypertension in the dogs in which blood ammonia concentrations were within reference range.

The hepatofugal portal flow directions remained unchanged cranial to the shunt origin after shunt attenuation only in the 2 dogs that later developed APSCs. However, transabdominal ultrasonography on the sixth postoperative day revealed that the intraoperatively detected postligation hepatofugal flow had subsequently become hepatopetal in both dogs. The flow direction in the shunt vessel, however, remained hepatofugal.

Blood ammonia concentrations 1 month after surgery were within reference range in all dogs that had hepatopetal or to-and-fro portal flow cranial to the shunt before shunt attenuation (4 dogs with right gastric-caval, 2 with spleno-caval, and 1 with spleno-azygos shunts), except for the dog that developed portal thrombosis.

In the 2 dogs that developed APSCs, examination of liver biopsy specimens obtained 1 month after surgery revealed portal vein hypoplasia. In these dogs and the dog with aplasia of the cranial portal vein, the hepatofugal portal flow velocities cranial to the shunt origin and the portal flow velocities caudal to the shunt origin before shunt attenuation were among the highest of the 17 dogs (Fig 6 and 7); however, there were overlaps among the values of the group with good outcomes and those of the group with poor outcomes. If DPV was > +28, complications developed (formation of APSCs, inability to attenuate the shunt because of aplasia of the cranial portal vein, and sudden death); if DPV was < +28, the outcome was good (**Fig 9**).

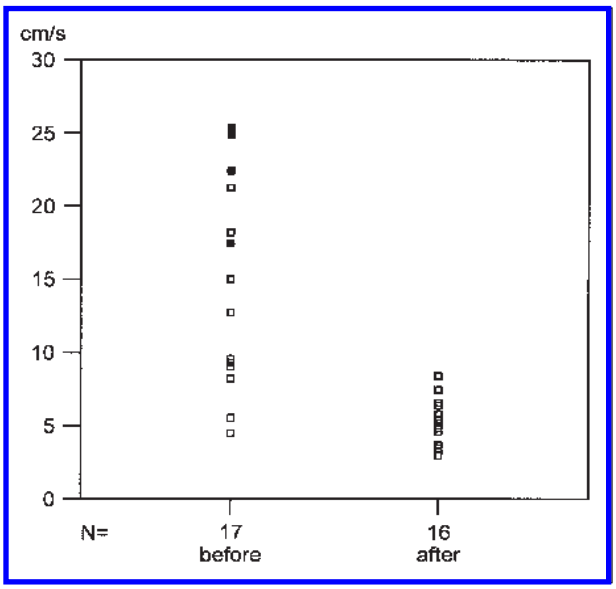


Figure 7—Comparison of time-averaged mean velocities in the PVcaudSH before (n = 17 dogs) and after (16) shunt attenuation. Solid squares indicate preligation values of 2 dogs that developed acquired portosystemic collaterals 1 month after surgery, a dog with aplasia of the cranial portal vein, and a dog that died suddenly.

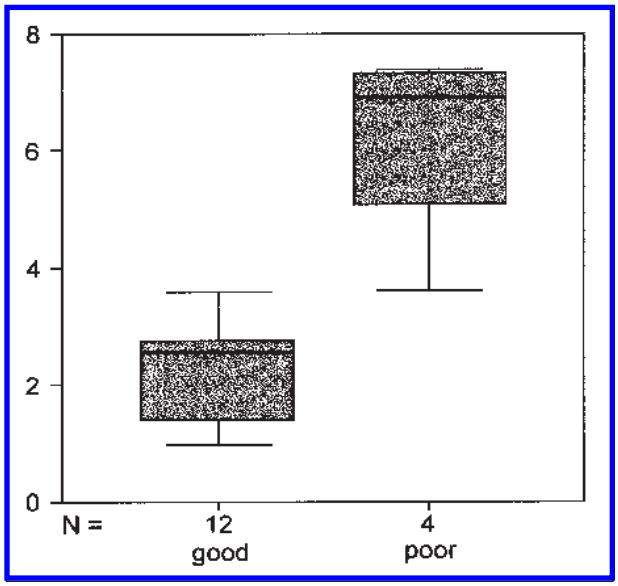


Figure 8—Comparison of the magnitude of increase of portal congestion indices (0 to 8) caudal to a shunt in dogs with a congenital extrahepatic portosystemic shunt with good outcomes (n = 12) and poor outcomes (4). The box represents the 25th to 75th percentiles, the line within the box represents the median, and the whiskers represent the range.

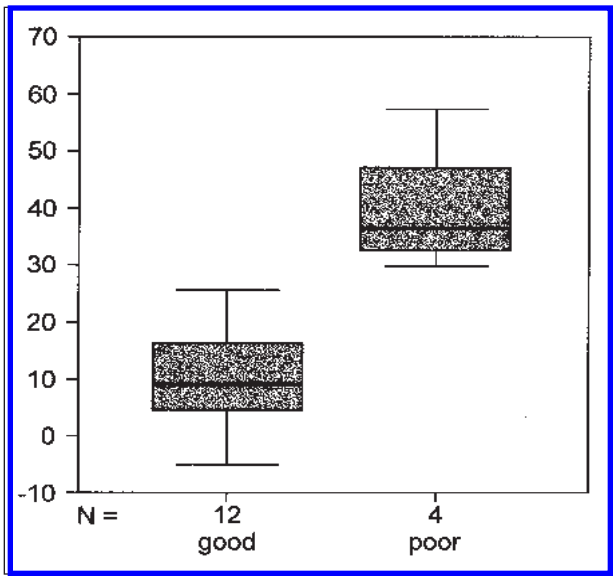


Figure 9—Comparison of differential portal velocities (cm/s [–10 to 70]) in dogs with a congenital extrahepatic portosystemic shunt with good outcomes (n = 12) and poor outcomes (4).

## Discussion

Use of intraoperative Doppler ultrasonography allowed us to make several novel observations because the entire portal system was easily accessible, unlike transabdominal ultrasonography in which the rib cage and gastrointestinal gas may hinder examination. In addition to the flow information, intraoperative ultrasonography proved to be useful to localize the shunt when adhesions from an earlier laparotomy hindered direct visualization of the vessels. Hepatopetal postattenuation flow in the shunting vessel has not been previously described to our knowledge, presumably because scintigraphy or portography has been predominantly used for follow-up studies[6,9] and is able to reveal only hepatofugal flow.

It is recommended that portosystemic shunts be ligated as far from their origin as possible because this is technically easier and portal tributaries may enter the shunting vessel.[8,9] Ligatures placed between the portal vein and the point where a portal tributary enters the shunting vessel could still allow shunting by the tributary. However, if the ligature is placed between the caudal vena cava and the point where the portal tributary enters the shunt and a complete shunt ligation is performed, the blood of this tributary must flow via the shunt to the portal vein, resulting in hepatopetal flow in the shunt. After partial attenuation, hepatopetal shunt flow can still develop if the resistance towards the portal vein is lower than towards the ligature. If the resistance is higher towards the portal vein, the shunt remains functional. In cases of splenocaval or spleno-azygos shunts, the tributary that enters the shunt is the splenic vein, and in cases of right gastric-caval and right gastric-azygos shunts, the tributary that enters the shunt is probably the left gastric vein via the anastomosis between the left and right gastric veins. When the shunt flow becomes hepatopetal after partial shunt attenuation, a portion of the blood of the portal tributary that enters the shunt may still be shunting, but it is unimportant because the blood of the splenic vein does not contain more toxins than any systemic vein, and the encephalopathic toxin content of the gastric veins is negligible, compared with that of the portal vein. The most important fact is that hepatopetal flow in the shunt prevents the blood of the portal vein from shunting, and the toxin-rich venous blood that comes from the small and large intestines has to flow to the liver through the sinusoids. When the shunt flow remained hepatofugal and the portal flow cranial to the shunt origin became hepatopetal, the amount of shunting blood was probably so small that the blood ammonia concentration could return to reference range.

Hepatofugal portal flow, well-known in humans with cirrhosis, is reportedly caused by development of intrahepatic arterio-portal communications attributable to the disorganized hepatic architecture.[30-32] However, hepatofugal portal flow associated with an extrahepatic CPSS has only been described in 1 dog, and no explanation was given.[2] We found that hepatofugal portal flow cranial to the shunt origin was actually limited to the portal vein segment that was between the origin of the shunt and the entrance point of the gastroduodenal vein. Because either slow hepatopetal or no flow was detected cranial to the entering point of the gastroduodenal vein, the blood of the gastroduodenal vein was found to be responsible for the hepatofugal portal flow. If the resistance towards the liver is higher (because of aplastic or severely hypoplastic portal branches) than towards the shunt, the blood from the gastroduodenal vein flows towards the shunt, resulting in hepatofugal portal flow. If the resistance is lower towards the liver, the gastroduodenal blood flows towards the liver, resulting in hepatopetal portal flow. When the resistance is approximately equal cranial and caudal to the gastroduodenal vein, the gastroduodenal blood is divided, causing hepatopetal portal flow cranial to the gastroduodenal vein and hepatofugal portal flow caudal to it. When to-and-fro portal flow is observed cranial to the shunt, the portal blood can intermittently reach the liver. Continuous hepatofugal portal flow, however, prevents this.

We believe that preattenuation hepatopetal and to-and-fro portal flow directions cranial to the shunt origin indicate that the resistance of the portal branches is lower than that in dogs with hepatofugal flow because part of the portal blood flows spontaneously through the hepatic sinusoids, not only through the shunt as in dogs with hepatofugal portal flow. When hepatofugal portal flow became hepatopetal immediately after shunt attenuation, the outcome was excellent or good. Shunt attenuation increases resistance towards the shunt. If this resistance exceeds the resistance towards the portal branches, the portal blood flows towards the liver and the preattenuation hepatofugal portal flow becomes hepatopetal. If the resistance towards the attenuated shunt remains lower than towards the liver, the gastroduodenal blood continues to flow hepatofugally. Although further shunt attenuation could further increase resistance towards the shunt, the degree of portal hypertension will also increase, causing unacceptably severe portal hypertension. In cases of aplasia

or severe hypoplasia of the portal vein cranial to the entering point of the gastroduodenal vein, the resistance towards the liver will always exceed the resistance that could be reached towards the shunt by shunt narrowing.

Severe portal vein hypoplasia must be suspected if the direction of portal flow cranial to the shunt remains hepatofugal when the shunt is temporarily attenuated to a diameter that is the same as the diameter of the portal vein cranial to the shunt origin. Hepatofugal portal flow has not been commonly identified in dogs, probably because most veterinarians have used portography or scintigraphy for diagnosis of CPSS, and the recommended ultrasonographic approach, the transverse right intercostal view,[2] does not allow evaluation of portal flow directions.

Despite the similar postattenuation portal flow velocities caudal to the shunt origin and the acyanotic intestinal colors during surgery, the clinical outcome was variable in the 16 dogs that underwent a shunt ligation. This finding might explain why establishing a safe threshold value for postattenuation portal pressure in dogs has failed.[4,18,24] All 3 variables (portal pressure, portal flow velocity caudal to the shunt, and intestinal color) are related and correspond to the portal hemodynamics caudal to the shunt; however, the portal flow directions cranial to the shunt also influence the clinical outcome, as we determined. Moreover, measuring flow velocity by Doppler ultrasound has advantages over direct portal pressure measurement and intestinal color assessment because it is both noninvasive and quantitative. The depth of anesthesia was unlikely to affect our measurements because the cardiovascular state of the patients was monitored continuously, and the mean arterial blood pressure was maintained higher than 60 mm Hg.

Portal vein thrombosis is a rare but fatal iatrogenic complication and is probably a result of exaggerated shunt ligation.[33] The postoperative history and clinical signs of the dog that died abruptly at home 2 days postoperatively were identical with the postoperative course of the dog that had ultrasonographically proven postligation portal vein thrombosis and of the dogs that have been described with postligation portal vein thrombosis in the literature (uncomplicated recovery and sudden collapse several days after surgery).[33]

Results of intraoperative observation of the abdominal viscera did not differ between dogs that developed APSCs or portal vein thrombosis as a result of exaggerated shunt closure and dogs with excellent or good outcomes. Retrospective analysis of the portal flow variables revealed that the magnitude of increase of portal congestion index measured caudal to the shunt origin was different between the 2 groups. Poor outcome can be the result of underlying severe portal vein hypoplasia, but can also happen in dogs with well-developed portal branches if the shunt attenuation is exaggerated.

Preattenuation portal flow velocities were high in dogs with complications, except for 1 dog with portal vein thrombosis. We hypothesize that the severely hypoplastic portal branches prevented portal blood to flow towards the sinusoids. As blood flows through the sinusoids, the portal velocity is relatively slow.[2] Another possible explanation for slow preligation portal flow velocities that were associated with good outcomes could be that if the preligation velocity is already low, the magnitude of decrease in postligation velocity (as well as increase in pressure) is small, requiring only mild adaptation from the portal system and reducing the risk of complications. The DPV would be an especially useful variable if it were able to differentiate between severely and mildly hypoplastic portal systems even before surgery, although this has not been investigated. Acquired portosystemic collaterals were diagnosed with ultrasound on the basis of our experience that dilatation of the left gonadal vein in dogs is a very sensitive and 100% specific sign of spleno-renal collateral vessels, the most consistently observed route of portosystemic collateral circulation.[10,29]

To determine the optimal degree of shunt narrowing in dogs, the largest possible shunt diameter that ensures hepatopetal flow in the shunt and in the portal vein cranial to the shunt should be found. After this stage is reached, further shunt attenuation is contraindicated. Regardless of the flow directions in the cranial portal vein and the shunt, hepatopetal portal flow caudal to the shunt should always be maintained with a minimum time-averaged mean velocity of 3 cm/s, and a > 3.5-times increase in the portal congestion index caudal to the shunt should be prevented. Because these recommended values and the threshold DPV value were derived retrospectively from our case series, their overall validity should be tested prospectively.

Intraoperative ultrasonography can reveal the hemodynamic features of the portal and shunt flow quickly, noninvasively, and without radiation exposure next to the operating table. Portal flow direction is more closely associated with clinical outcome than other variables that have been studied.[1,6-12,18,24,34-36] Surgical ligation of extrahepatic CPSSs guided by intraoperative Doppler ultrasound is an excellent method for safe and effective shunt closure.

[a]Ethibond Excel 2/0, Ethicon, Johnson & Johnson International, Hamburg, Germany. Available in the United States from: Ethicon, Johnson & Johnson, San Angelo, Tex.

[b]Thalamonal, Janssen-Cilag BV, Tilburg, The Netherlands. Available in the United States as Innovar-vet from: Pitman-Moore Inc, Mundelien, Ill.

[c]ATL HDI 3000, high-definition ultrasound system, Philips, Woerden, The Netherlands. Available in the United States from: Philips Medical Systems, ATL Ultrasound, Bothell, Wash.

[d]Surgi-Tip Intraoperative Transducer Cover & Intraoperative Polyethylene Ultrasound System Drape Kit, CIVCO Medical Instruments, Kalona, Iowa.

[e]High Q of the ATL HDI 3000, Philips, Woerden, The Netherlands. Available in the United States from: Philips Medical Systems, ATL Ultrasound, Bothell, Wash.

[f]Szatmári V, Rothuizen J. How can you tell with ultrasound that a patient with high blood ammonia has a congenital or acquired portosystemic shunt or no shunt at all? (abstr), in *Proceedings*. 27th Cong World Small Anim Vet Assoc 2002;42.

[g]SPSS 11.0 for Windows, SPSS Inc, Chicago, Ill.

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