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 origin increased by > 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)
process. A hypoplastic portal system may not be able to adapt to the increased blood flow at the same rate as the constriction rate of the device; therefore, subacute or chronic portal hypertension can develop.1,2,3,6

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.4,5

Whichever technique (ie, ligation, ameroid constrictor, or cellophane banding) is used for attenuation of an extrahepatic CPSS, the clinical outcome remains unpredictable.6,8,11,12,15-21,23 because presently the severity of portal vein hypoplasia cannot be determined either pre- or intraoperatively.1,14,30,11,19 Use of histopathologic changes in the liver,1,10 portal pressure,7,9,18 partial versus complete ligation,1,9,12,15,17,21 age of the dogs,8,24 and portographic images7 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 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 food15 combined with direct visualization of the shunt via transabdominal ultrasonography. Spleno-caval (9/17), right gastric-caval (3/17), and spleno-azygous (3/17) shunts were found.

Technique—The CPSS of each dog was attenuated surgically with polyester suture material by a single surgeon (FJS) by use of a reported technique.2 After premedication (0.5 mL/kg [0.14 mL/lb] with the combination of droperidol and fentanyl,2 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 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. 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 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.25 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;2 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 the time-averaged mean velocity.

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.

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 postsystemic collaterals (APSCs) were diagnosed ultrasonographically, when a wide left gonadal vein was found, as a result of splenorenal collaterals entering the left renal vein from the caudal direction.10,23 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.24,31,32,33

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 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 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 poor category because the preligation hepatopetal (i.e., 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 6 dogs with complete shunt ligation (portal vein thrombosis) and in 3 of the 10 dogs with partial shunt ligation (developed APSCs and 1 died).

Portal hemodynamics during surgery—Changes in right atrial pressure were reflected in the velocity spec-
right gastric-caval shunts, hepatopetal portal flow was
detected cranial to the shunt origin before shunt atten-
uation; 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 hepatofugal flow.

Although the portal vein cranial to the gastroduo-
denal 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 col-
lapse 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 diam-
eter 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 seg-
ment 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 preattenua-
tion 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 hepatofu-
gal, the shunt flow also remained hepatofugal. These 2
dogs developed APSCs postoperatively, and hyperam-
monemia persisted; ultrasound-guided liver biopsies

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
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 por-
tosystemic 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 atten-
uation; 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 atten-
uation. Positive values indicate hepatopetal blood flow; negative
values indicate hepatofugal blood flow.

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 sud-
denly.
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).

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**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.
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 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. 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 liver. However, if the resistance towards the liver is higher (because of aplastic or severely hypoplastic portal branches) than towards the portal vein, the blood from the portal vein flows towards the shunt, resulting in hepatofugal portal flow. If the resistance is lower towards the liver, the gastrroduodenal blood flows towards the liver, resulting in hepatopetal portal flow. When the resistance is approximately equal cranial and caudal to the gastrroduodenal vein, the gastrroduodenal blood is divided, causing hepatopetal portal flow cranial to the gastrroduodenal 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 gastrroduodenal 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, 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 ligature. This finding might explain why establishing a safe threshold value for postattenuation portal pressure in dogs has failed. 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. The postoperative history and clinical signs of the dog that died abruptly at home 2 days postoperatively were identical with the postoperative signs of the dog that died abruptly at home 2 days postoperatively, 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.

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. Surgical ligation of extrahepatic CPSSs guided by intraoperative Doppler ultrasound is an excellent method for safe and effective shunt closure.

References


