CHAPTER 6

Intraoperative ultrasonography of the portal vein during gauged attenuation of intrahepatic portocaval shunts in dogs

based on the article by

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Summary

A method for intraoperative measurement of portal blood flow velocity with duplex Doppler ultrasonography in 7 dogs with congenital intrahepatic portosystemic shunts was described. Our aims were to determine whether intraoperative ultrasonography was an acceptable alternative to mesenteric portography in such dogs and to identify quantitative portal hemodynamic variables that might correlate with clinical outcome better than portal pressure does. Ultrasonographic measurements did not influence decision-making by the surgeon, who attenuated the shunt on the basis of appearance of the viscera and change in mean systemic arterial blood pressure.

All dogs recovered without complications, and surgery was considered to be successful in all 7. Intraoperative B-mode ultrasonography provided real-time information about the anatomy of the shunt and the portal branches, suggesting that it may be a useful alternative to mesenteric portography. The time-averaged mean portal blood velocity ranged from 6.5 to 33.7 cm/s before shunt attenuation and from 5.0 to 9.5 cm/s after shunt attenuation. This narrow range of postligation velocities suggested that intraoperative ultrasonography may be an alternative to intraoperative portal pressure measurement.

Introduction

In dogs, an intrahepatic portosystemic shunt is a congenital, typically single, abnormal macroscopic vascular connection between the left or right portal branch and the caudal vena cava or ipsilateral hepatic vein.[1,2] Most affected dogs develop signs of hepatic encephalopathy between 2 months and 1 year of age.[3] Definitive treatment of intrahepatic portosystemic shunting involves complete occlusion of the shunt vessel.[1-5] However, attenuating the shunt vessel forces blood to flow through the portal branches, which are frequently hypoplastic because of hypoperfusion, resulting in portal hypertension. The degree of portal hypertension that develops during attenuation of a shunt vessel depends on the capacity of the portal branches to absorb the increased bloodflow.[1] Therefore, the shunt can be completely occluded only in those dogs in which the portal system is well developed. In most dogs, however, hypoplasia of the portal branches allow only partial shunt occlusion to be performed,[2-5] and the narrowest possible shunt diameter is determined by assessing the degree of portal hypertension during gradual shunt attenuation.[2-5]

Two methods have been recommended for avoiding acute fatal portal hypertension during shunt attenuation in dogs with portosystemic shunting. The first involves measuring portal pressure by direct catheterization of a portal tributary to obtain a direct quantitative assessment of the degree of portal hypertension.[3,5-7] However, several factors can make the interpretation of pressure changes unreliable[2,8-11]; moreover, fatal hemorrhage may occur as a complication of this technique.[8,12] The other method involves monitoring qualitative signs (eg, color of the intestines) and indirect quantitative variables (eg, magnitude of the change in the mean systemic arterial blood pressure and heart rate) to determine an acceptable degree of postligation portal hypertension.[4,8,13]

Some years ago, ameroid constrictors, which gradually attenuate the shunt vessel, resulting in complete shunt occlusion in 1 to several weeks' time, were introduced.[10,14-17] The underlying idea behind using ameroid constrictors for occlusion of portosystemic shunts was that the gradual shunt attenuation would allow underdeveloped portal branches to adapt to the increased blood flow. However, although applying an ameroid constrictor does not require intraoperative assessment of portal hypertension, shunt attenuation becomes an uncontrollable process with this method. 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 would force it to; therefore, acute or chronic portal hypertension can develop.[14,15] Moreover, ameroid constrictors have routinely been used only on extrahepatic portosystemic shunts, because their application on intrahepatic shunts involves risks, such as perforation of the shunt because of the increased dissection needed and development of acute portal hypertension because of kinking of the shunt.[15,16] To reduce these risks, a technically challenging procedure has been recommended, namely placement of an ameroid constrictor on an extrahepatic portocaval venograft after complete ligation of the intrahepatic shunt.[15]

Regardless of the technique used for shunt attenuation and for assessing postligation portal hypertension, the clinical outcome remains unpredictable,[2-5,8-15] largely because there is no method currently available to reliably evaluate the capacity of the hypoperfused portal branches to accept the increased blood flow that results from shunt attenuation (ie, the degree of portal vein hypoplasia).[1,4,12,18-20] The purposes of the present report were to describe a noninvasive technique for assessing postligation portal flow in dogs with intrahepatic portosystemic shunts, identify a quantitative portal hemodynamic

variable that might correlate with clinical outcome better than portal pressure does, and describe use of intraoperative ultrasonography as an alternative to mesenteric portography.

Description of the Technique

Intrahepatic portosystemic shunts were attenuated surgically by a single surgeon (FJvS), using the technique described by Wolschrijn et al.[4] For surgery, dogs were premedicated with atropine and a mixture of droperidol and fentanyl (Thalamonal (0.3 mL/kg [0.14 ml/lb] of a solution containing 2.50 mg of droperidol/mL and 0.05 mg of fentanyl/mL, IM), Janssen-Cilag BV, Tilburg, The Netherlands). Anesthesia was induced with propofol, IV, and maintained with isoflurane vaporized in oxygen. Intraoperative analgesia was provided with a continuous IV infusion of sufentanil (1 µg/kg/h [0.45 µg/lb/h]). Dogs were mechanically ventilated during surgery.

For intraoperative ultrasonography, a commercially available high-definition ultrasound system (ATL HDI 3000, Philips Medical Systems, Advanced Technical Laboratories) was used (**Fig 1**). This was equipped with a 26-mm-long, 5 to 10-MHz, intraoperative linear array transducer (**Fig 2**). Both the keyboard and the transducer were covered with sterile material (Surgi-Tip intraoperative transducer cover and intraoperative polyethylene ultrasound system drape kit, CIVCO Medical instruments, Kalona, Iowa, USA). All examinations were performed by a single ultrasonographer (VS) in the operating suite and were recorded on videotape for further analyses and documentation.

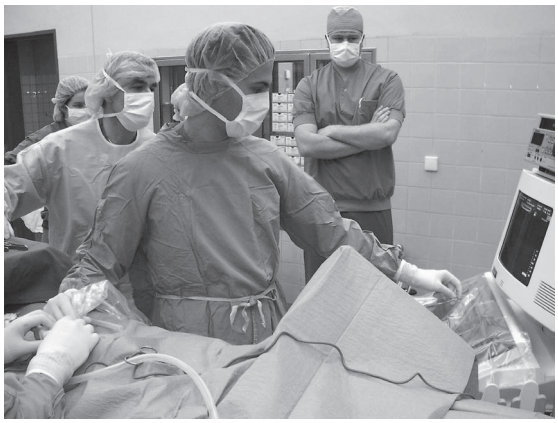

Figure 1.
Intraoperative ultrasonography during a surgical attenuation of a congenital portosystemic shunt.

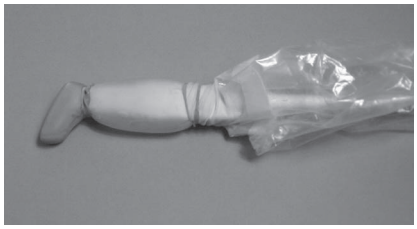

Figure 2.
An intraoperative linear array transducer (5-10 MHz) with sterile cover.

A midline celiotomy was performed, and all organs were left in the abdominal cavity. The descending duodenum was temporarily retracted toward the midline to expose the portal vein and was then released. The ultrasound transducer was placed directly on the trunk of the portal vein between the splenic and gastroduodenal veins, first to obtain a B-mode longitudinal image and then to obtain a color Doppler image. Three consecutive Doppler spectra were obtained in the pulsed-wave Doppler mode by placing the sample

volume in the portal vein. The size of the sample volume was adjusted so that it would overlap the walls of the vessel (uniform insonation method) without including any adjacent vessels.[21-23] Because a linear transducer was used, the Doppler ultrasound beam was directed as needed (beam steering) to obtain good-quality spectra (**Fig 3**).[23]

A specific region of the Doppler spectrum was set on a frozen image to make the built-in spectrum analyzer (HighQ, ATL HDI 3000) perform automatic Doppler display calculations (**Fig 3B**). Artifacts and poor-quality parts of the tracing were avoided when a region was specified. Using the highest scrolling speed possible (5 cm/s), the longest spectrum that was displayed on the screen and, thus, could be specified was 3.44 seconds. Peak and minimum velocities, as well as time-averaged peak and time-averaged mean velocities, were obtained.

After automatic analysis of at least 3 good-quality Doppler spectra, the transducer was rotated by 90^{o} to obtain a cross-sectional B-mode image of the vessel at the same point where velocity measurements had been taken.[24] Cross-sectional area was measured by use of the continuous trace method, performed with a built-in program that allows the lumen to be traced with a trackball and cursor.

The first series of measurements was performed immediately after celiotomy, before any manipulation of the shunt. Afterwards, B-mode ultrasonographic scanning of the portal vein was continued cranially to localize the intrahepatic shunt and identify its course. Attention was paid to the presence of a sinus-like dilatation of the shunt and the type and position of veins that connected with the shunting vessel. To differentiate hepatic veins from portal branches, morphologic features such as width, course, and wall appearance were evaluated with B-mode ultrasonography, and hemodynamic features such as velocity and direction of flow were evaluated with color Doppler ultrasonography.[23] The second series of measurements was performed at least 5 minutes after shunt attenuation, before abdominal closure. B-mode ultrasonographic scanning of the portal vein cranial to the measuring point was again performed to confirm that the ligature was placed at the best point possible.

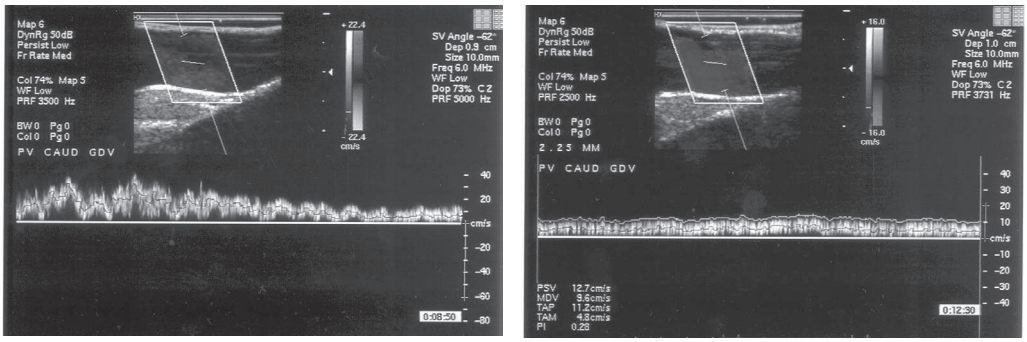

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Figure 3.

Intraoperative spectral Doppler ultrasonography of the portal vein in a golden retriever with an intrahepatic portocaval shunt. The color and pulsed-wave Doppler beams are steered, the sample volume incorporates the entire vessel (uniform insonation method).

A. Before surgical ligation the portal flow velocity is variable due to the right atrial pressure changes.

B. After partial attenuation of the shunt the portal flow velocity is decreased and the variability in the velocity spectrum became damped.

The optimal degree of shunt attenuation, that is the narrowest shunt diameter that could be achieved without causing severe portal hypertension, was determined in steps, according to the criteria of Wolschrijn et al.[4] In brief, the degree of portal hypertension was considered to be acceptable when the pancreas and intestines were not cyanotic, the colors of the jejunal arteries and veins did not differ markedly from each other, and the heart rate did not increase more than 15% and the mean systemic arterial blood pressure did not decrease more than 15%, compared with values recorded at the beginning of surgery. For shunt ligation, polyester suture material (Ethibond Excel 2-0, Ethicon, Johnson & Johnson Intl, Hamburg, Germany) was used. The systemic arterial blood pressure was measured by direct catheterization of a femoral artery and was recorded continuously.

From the measured ultrasound values, portal flow (mL/s) was calculated by multiplying the time-averaged mean velocity (cm/s) by the cross-sectional area of the vessel (cm^2).[22,24,25] Mean portal flow (mL/min/kg) was calculated by dividing portal flow by body weight (kg) and multiplying by 60.[21,22] The congestion index was calculated as the cross-sectional area of the portal vein (cm^2) divided by the time-averaged mean velocity (cm/s).[26] Relative size of the portal vein was obtained by dividing the cross-sectional area of the portal vein (cm^2) by the body weight of the dog (kg) and by comparing the cross-sectional area of the portal vein to that of the caudal vena cava measured at the level of the right adrenal gland.

The outcome of shunt attenuation was assessed 1 month after surgery by measuring blood ammonia concentration after food had been withheld for 12 hours and by performing transabdominal ultrasonography with a 4 to 7-MHz sector transducer, paying special attention to the size of the liver, the presence of free abdominal fluid and acquired portosystemic collaterals, and the width and course of the portal vein, as well as the attenuated and non-affected portal branches. Color Doppler ultrasonography was used to determine whether the attenuated portosystemic shunt was still patent and to examine blood flow in the portal vein and left and right portal branches. If the ammonia concentration was high at the time of this first examination, a second re-examination was scheduled for 3 months after surgery. Dogs were not sedated or anesthetized for these transabdominal ultrasound examinations. During ultrasonography, the size of the liver was assessed subjectively by examining the position of the stomach in relation to the diaphragm on the left side and the length of the portion of the right kidney that was covered by the caudate liver lobe on the right side. Acquired portosystemic collaterals were diagnosed ultrasonographically when a wide left gonadal vein was seen entering the left renal vein caudally (resulting from splenorenal collateral vessels)[27,28] or when a vessel that originated from the portal trunk or from a portal tributary was seen with a flow direction away from the portal system.

The outcome was considered to be favorable if, after surgery, the dog no longer had signs of hepatic encephalopathy and did not have ultrasonographic evidence of sustained portal hypertension at the time of re-check examinations.

Results

Nine client-owned dogs underwent surgical attenuation of an intrahepatic portosystemic shunt at the Utrecht University Clinic for Companion Animals during 2001 with this technique. Results for the first 2 are not included in the present report, because the

ultrasonographic scanning technique was not yet consistent. However, the experience gained with these dogs was used to establish the protocol applied in the remaining dogs.

The 7 dogs in which the technique was used consisted of 2 Labrador Retrievers, a Golden Retriever, an Old English Sheepdog, a Giant Schnauzer, a Münsterländer, and an Irish Wolfhound mix. Mean age at the time of surgery was 6.5 months (range, 3 to 15 months); mean weight was 18 kg (40 lb; range, 10 to 29 kg [22 to 64 lb]). Four were male, and 3 were female.

In all 7 dogs, portosystemic shunting was suspected on the basis of history, results of a physical examination, and blood ammonia and bile acids concentrations measured after food had been withheld for 12 hours. In all dogs, transabdominal ultrasonography, performed with the dogs awake, was used to confirm the diagnosis and assess the size of the liver. In each dog, a shunt could be visualized. The shunt originated from the left portal branch in 4 dogs and from the right portal branch in 3.

Complete shunt ligation was not possible in any of the 7 dogs. The smallest diameter of the shunt after attenuation ranged from 2 to 5 mm, which represented a mean reduction in cross-sectional area of the shunt of 84% (range, 60 to 95%; **Fig 4**). For this calculation, the cross-sectional area of the shunt prior to attenuation was assumed to be equal to the cross-sectional area of the portal vein before shunt attenuation, as it was not possible to examine the cross-sectional area of the entire length of the shunt to determine the smallest preligation cross-sectional area.

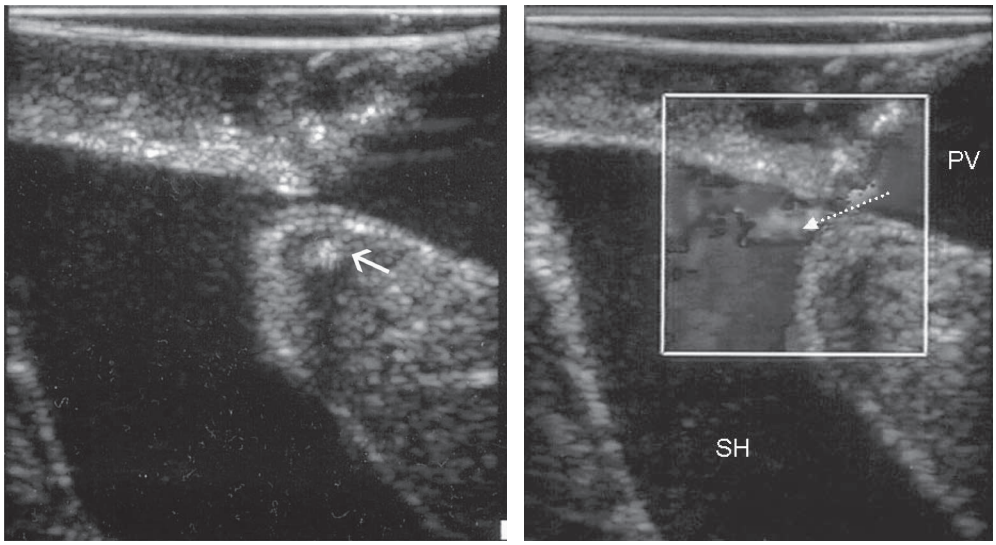

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Figure 4.

Intraoperative ultrasonogram of a partially attenuated intrahepatic portocaval shunt in a dog with a persistent ductus venosus. The diameter of the narrowed part is 2.25 mm.

A. On the B-mode image the cross-section of the suture material can be seen as a hyperechoic spot (arrow) with distal shadowing.

B. On the color Doppler image the presence and direction (arrow) of blood flow can be seen. PV portal vein, SH shunt. (**Color** version on page 181.)

All 7 patients survived surgery without complications and were discharged from the hospital on the second postoperative day. One month after surgery, owners of all 7 dogs reported clinical improvement or a complete absence of clinical abnormalities. Blood ammonia concentration was within reference limits (reference range, 24 to 45 μmol/L) in 2 dogs, slightly high in 3 dogs (67, 76, and 78 μmol/L), and high in the remaining 2 dogs (142 and 210 μmol/L). Transabdominal color Doppler ultrasonography revealed blood flow through the attenuated shunt in 6 dogs; blood flow was slow in dogs with normal and slightly high blood ammonia concentrations and fast in dogs with high blood ammonia concentrations (**Fig 5**). In 1 dog, color Doppler ultrasonography could not be performed because the shunt was located too far from the transducer and the dog was constantly panting. Quantification of blood flow through the attenuated shunt with pulsed-wave Doppler ultrasonography was not feasible because the anatomy of the shunt and the location of the ligature.[23] In all 7 dogs, the liver was estimated, on the basis of ultrasonographic findings, to be larger 1 month after surgery than it had been before surgery. No ultrasonographic signs of sustained portal hypertension, such as free abdominal fluid, acquired portosystemic collaterals, or dilatation of the portal vein with slow blood flow,[21,29] were detected in any of the dogs at the 1-month re-examination.

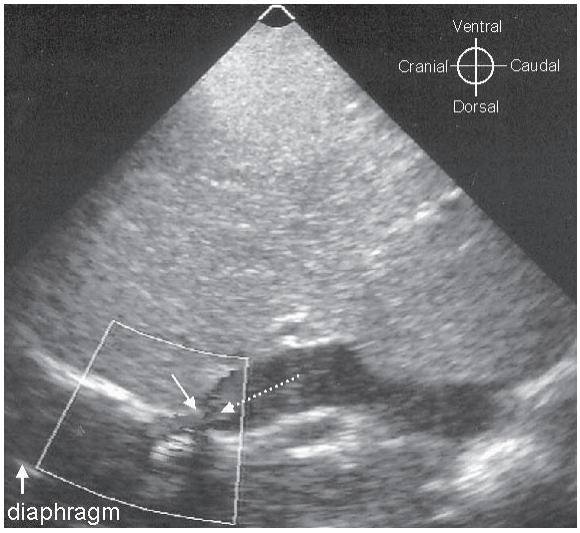

Figure 5.
Transabdominal color Doppler ultrasonogram of a partially ligated left-divisional intrahepatic portocaval shunt 1 month after surgery. The narrowing of the vessel caused by the ligature is clearly seen (arrow). The stenosis casued by the ligature results in a high flow velocity (poststenotic jet), which can be appreciated as red color with mosaic pattern (aliasing artifact). The interrupted arrow indicates the direction of blood flow in the portal vein. (Full **color** illustration on page 181.)

Three of the 7 patients were also examined 3 months after surgery (1 with normal and 2 with markedly high blood ammonia concentrations 1 month after surgery). Blood ammonia concentrations and results of transabdominal ultrasonography were similar to those of 2 months earlier. A fourth dog, which had mild hyperammonemia and a patent shunt 1 month after surgery, was re-examined 1 year after surgery. Abdominal ultrasonography revealed that the shunt had subsequently become completely closed, and the blood ammonia concentration was within reference limits.

Intraoperative B-mode ultrasonography proved to be useful in all 7 dogs in identifying the shunt and the non-affected portal branches, as well as their course, and in guiding the surgeon to the optimal point for ligature placement by differentiating vessels in connection with the shunt into hepatic veins and portal branches (**Fig 6**). Hepatic veins were wider with hepatofugal or bi-directional blood flow, whereas portal branches were thin with rather slow hepatopetal flow. Sinus-like dilatation of the shunt was also precisely localized in each case and was successfully avoided during surgical dissection. Postligation intraoperative ultrasonographic evaluation was valuable to confirm that the ligature was indeed placed on the shunting vessel and at the best point possible. As a result of shunt attenuation, the previously hypoperfused portal branches became wider and the hepatopetal flow became faster. Upstream to the ligature, swirling movements of slowly flowing aggregated red blood cells could be seen in the dilated shunt in B-mode images.

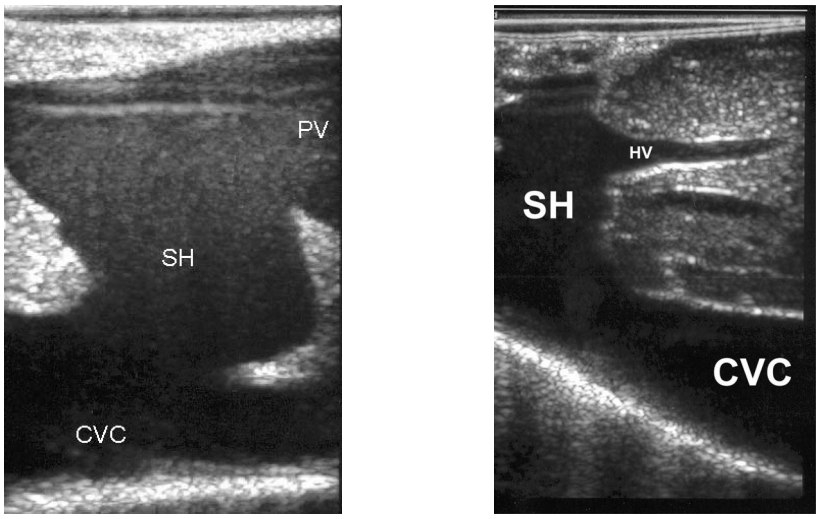

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Figure 6.
Intraoperative gray scale ultrasonograms of left divisional intrahepatic portocaval shunts in two dogs. SH shunt, CVC caudal vena cava, PV portal vein

A. The intrahepatic course of the shunt (SH) can be appreciated from its origin to its termination.

B. A hepatic vein (HV) is in direct connection with the shunt. This is an important piece of information when selecting the point of ligation.

Mean time spent on ultrasonography during each surgery was 19 minutes (range, 13 to 25 minutes). This included the time spent on obtaining anatomic information before and after shunt attenuation (ie, localizing the shunt, planning and confirming the placement of the ligature, obtaining cross-sectional areas of the portal vein and caudal vena cava) and the time spent on measuring portal flow velocity with duplex Doppler ultrasonography. Mean time between pre- and postligation ultrasonographic measurements was 83 minutes (range, 57 to 124 minutes); this represented the time necessary for the surgeon to dissect the shunt and to find, in a stepwise manner, the narrowest possible diameter that did not cause signs of severe portal hypertension.

Intraoperative duplex Doppler ultrasonography revealed that before shunt attenuation, the time-averaged mean velocity of blood flow in the portal vein ranged from 6.4 to 33.7 cm/s in the 7 dogs. After shunt attenuation, the direction of portal flow remained hepatopetal in every dog, and the time-averaged mean velocity ranged from 5.0 to 9.5 cm/s (**Fig 7**). In addition, the variability in the velocity spectrum attributable to the effect of right atrial pressure changes became damped after shunt attenuation.

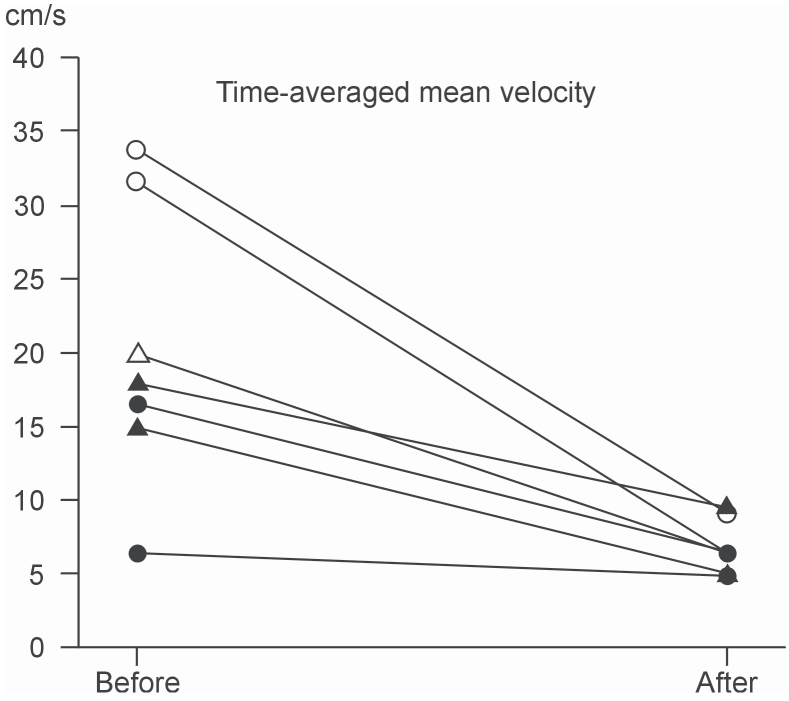

Figure 7.

Time-averaged mean portal blood flow velocity values for 7 dogs before and after partial ligation of an intrahepatic portosystemic shunt. Velocities were measured intraoperatively in the portal vein by means of duplex Doppler ultrasonography. Triangles indicate dogs in which the shunt originated from the right portal branch; circles indicate dogs in which the shunt originated from the left portal branch. Open symbols indicate dogs in which the blood ammonia concentration was within reference limits 1 month after surgery; closed symbols indicate dogs in which blood ammonia concentrations were still high 1 (3 dogs) and 3 (2 dogs) months after surgery.

Other variables were examined in an attempt to identify one that had similar values in all 7 dogs after shunt attenuation, but all other variables examined were more variable from patient to patient than the time-averaged mean portal flow velocities.

As with time-averaged mean velocities, time-averaged peak velocities decreased after shunt attenuation (time-averaged peak velocities ranged from 16.7 to 69.7 cm/s before shunt attenuation and from 9.4 to 25.9 cm/s after shunt attenuation). However, the range of peak velocities after shunt attenuation was wider than the range of mean velocities.

The cross-sectional area of the portal vein after shunt attenuation (0.59 to 1.30 cm^2) and the magnitude of its increase (20 to 148%), compared with values before shunt attenuation (0.44 to 1.27 cm^2) were calculated in each dog, but both sets of data were rather variable among dogs and would be unsuitable for guiding the degree of attenuation. A wide range of variability was also found when the relative cross-sectional area of the portal vein after shunt attenuation, corrected for body weight, (0.034 to 0.126 cm^2/kg) and the magnitude of its increase (106 to 248%) compared with preligation values, were examined.

Portal flow before and after shunt attenuation and the magnitude of the decrease was also calculated. The trend was similar to the velocity values, but flow values after shunt attenuation were more variable among patients (3.0 to 11.4 mL/s) than the time-averaged mean velocity values. The postligation mean portal flow values (11.2 to 71.7 mL/min/kg) were even more variable among patients.

The congestion index of the portal vein after shunt attenuation ranged from 0.092 to 0.218, and the magnitude of the increase in congestion index, compared with preligation values, was 3.1 to 5.9-fold. Both varied greatly from patient to patient.

The cross-sectional area of the portal vein was smaller than that of the caudal vena cava in each patient before shunt attenuation. If the cross-sectional area of the caudal vena cava in each dog was indexed as 100%, the cross-sectional area of the portal vein prior to ligation ranged from 17.7 to 53.1%. Neither this nor any of the other measured or calculated variables could be related to the outcome of the surgery (ie, normalization of the blood ammonia concentration).

The relative size of the portal vein seemed to depend on the age of the dog, with younger dogs having relatively wider portal veins. The cross-sectional area of the portal vein, expressed as a percentage of the cross-sectional area of the caudal vena cava, varied from 24 to 53% in the 5 dogs that ranged from 4 to 6 months old at the time of surgery, but was 18% in a 9-month-old dog and 20% in a 15-month-old one. Similar differences were seen when cross-sectional areas corrected for body weight were compared; this ranged from 0.027 to 0.051 cm^2/kg in dogs that were 4 to 6 months old, but was 0.022 cm^2/kg in the 9-month-old dog and 0.021 cm^2/kg in the 15-month-old one.

The cross-sectional area of the caudal vena cava changed markedly during each respiratory cycle. Images used for measurements were those in which the caliber seemed to be the largest.

Discussion

Although preoperative transabdominal ultrasonography allows identification of an intrahepatic portosystemic shunt and determination of whether the intrahepatic shunt originates from the left or right portal branch, intraoperative ultrasonography can be used to directly image the portal branch a surgeon suspects to be the shunt and to immediately

verify that suspicion. Localizing the origin and insertion of an intrahepatic portocaval shunt by visual inspection requires a great deal of experience, and the course of the shunting vessel still remains hidden within the liver.[30,31] In addition to assisting in identifying the correct vessel, ultrasonography is valuable in helping to select the optimal point for placing the ligature. The goal of shunt attenuation is to improve the perfusion of all liver lobes, and this requires localizing the origin of the most cranial portal branch and placing the ligature downstream to it. If the ligature is placed upstream to a portal branch, the corresponding liver lobe would atrophy. A third advantage of intraoperative ultrasonography during portosystemic shunt surgery is that any sinus-like dilatation of the shunt, which is common in dogs with intrahepatic shunts, can be precisely localized.[30,32] A surgeon lacking this information could place the ligature downstream to the sinus, and the resulting portal hypertension could cause the sinus to grow and, possibly, rupture. Fatal hemorrhage can also occur if the sinus is damaged during blind dissection of the shunt.[2]

Although intraoperative B-mode ultrasonography was used as early as 1983 to locate, as well as to guide the ligation of, an intrahepatic portocaval shunt in a dog,[30] portography has traditionally been used for intraoperative imaging of the shunt and the portal vasculature, and intraoperative abdominal ultrasonography has remained uncommon in veterinary medicine.[33] However, intraoperative ultrasonography offers several advantages over portography. When image intensifiers are used in the operating suite for mesenteric portography, the staff must wear lead aprons during the whole time of the surgery to protect themselves against radiation. This is certainly rather exhausting during a 1- to 3-hour surgery.[2] If portography is performed outside the operating suite, the abdomen must be closed after surgical insertion of a catheter into a portal tributary, and the patient must be transported to the radiology unit then back to the operating suite, which is time consuming and may cause the patient's temperature to drop dramatically.[2] Loss of sterility is another major concern with this procedure.[2] Some authors have described performing diagnostic intraoperative portography and the actual therapeutic procedure (ie, the shunt ligation) during 2 separate anesthetic sessions to reduce the risks of extended anesthesia; however, this approach requires 2 laparotomies.[9,10] In contrast to these difficulties associated with mesenteric portography, intraoperative ultrasonography can be performed by placing the ultrasound system next to the operating table, so the patient does not have to be transported to another unit, and the staff does not have to wear protective clothing. Moreover, within 10 minutes more detailed anatomic, as well as hemodynamic, information can be obtained than with portography. A major advantage of intraoperative ultrasonography is that any vessel can readily be identified by simply placing the transducer directly on it; whereas, after portography has been performed, the angiographic images must still be correlated to the macroscopic structures, which might be difficult. Ultrasonography also allows the improved circulation of the previously hypoperfused portal branches to be instantly appreciated after shunt attenuation. In addition, ultrasonography is particularly valuable in identifying the shunt vessel and avoiding damage to other structures when direct examination of the abdominal vessels is impossible because of extensive peritoneal adhesions; this is common in dogs that previously underwent a celiotomy.[8]

Measuring blood flow velocity by duplex Doppler ultrasonography is an accurate and reliable method[25,34-39] and has been used to identify portal flow and portal hypertension in dogs.[21,22,24] Care should be taken to avoid compressing the vein with the transducer, which will artificially increase velocities and artificially decrease cross-sectional areas.[23] In our dogs, velocity and area measurements were repeated at least 3 times and results were

averaged to reduce intra-observer variability.[8,26,35,36,40,41] Inter-observer variability was eliminated by having all examinations performed by a single ultrasonographer. To avoid erroneous velocity determinations, special attention was paid to adjusting the Doppler angle correctly, since the incidence angle was often still rather high (between 60 and 70°), despite beam steering.[24] The color Doppler mode was used to avoid areas of turbulent flow (eg, where a smaller tributary entered the portal vein), since this would result in an inadequate Doppler spectrum for analysis.

The portal flow velocity decreased in every dog after shunt attenuation as a result of peracute portal hypertension. Although reduced portal flow velocity in dogs with portal hypertension has been described, the magnitude of the decrease in velocity is not directly comparable with our data, because earlier studies obtained their data from dogs with subacute (3 days after portal branch ligation)[40] or chronic portal hypertension.[21,40]

Clinical observation of the abdominal viscera is an inexpensive and valuable method of avoiding acute fatal portal hypertension; however, to be able to correlate intraoperative findings with surgical outcome, quantifiable information such as portal flow velocity is required. In all 7 patients in the present report, the qualitative and indirect quantitative criteria used to determine the narrowest shunt diameter at which the degree of portal hypertension remained acceptable corresponded to a time-averaged mean velocity of 5.0 to 9.5 cm/s, regardless of the velocity before shunt attenuation. Portal blood flow velocity is an easily obtainable variable that can be measured directly, quickly, and noninvasively without any calculations. Moreover, it seems to be independent of the size of the dog.

The cross-sectional area of the portal vein was increased in every dog after shunt attenuation, and the percentage of this increase was calculated, since the cross-sectional area of the vein depends on the size of the dog. However, neither portal vein cross-sectional area itself nor the magnitude of the change in cross-sectional area after shunt attenuation showed a consistent relationship with macroscopic postligation surgical findings, even when corrected for body weight (ie, cross-sectional area divided by body weight). Thus, area values could not be used as a guide for shunt attenuation. A possible explanation for the variation in magnitude of the increase in portal vein cross-sectional area is that although measurements were performed in each dog at the same point on the portal vein, the ligatures were placed various distances from this measurement site. Placing the ligature further upstream may result in a larger increase in the portal vein diameter. Other individual variables, such as capacity of the previously hypoperfused portal branches and compliance and caliber of the portal system, must also play a role.[18]

Although an increase in cross-sectional area of the portal vein and a decrease in portal blood flow velocity are logical consequences of acute congestion secondary to postligation portal hypertension,[41] to our knowledge, this has not been documented in dogs previously. In dogs with experimentally induced chronic portal hypertension, the cross-sectional area of the portal vein did not differ from that of healthy dogs, probably because of the presence of acquired portosystemic collaterals.[21]

Calculated variables such as portal flow, average portal flow, and congestion index of the portal vein have been recommended as ways to assess portal hypertension in dogs and humans.[21,26,42] However, these variables may not be useful in guiding the degree to which intrahepatic shunts can be attenuated, because they vary too greatly from patient to patient after shunt attenuation. Calculation of each of these variables involves the cross-sectional area, and even when the substantial errors that are known to occur in determining

the area of the portal vein[25,35,42] are corrected through direct measurement (eg, tracing the lumen on a transverse image rather than calculating area from 1 or 2 diameters), cross-sectional area remains extremely variable.

The cross-sectional area of the portal vein was compared with that of the caudal vena cava before shunt attenuation to determine whether relative size of the portal vein had any relation with outcome of the surgery. Although a necropsy study[19] reported that dogs with primary portal vein hypoplasia may have normal or narrow extrahepatic portal veins, exact measurements were not performed. During intraoperative ultrasonography, the cross-sectional areas of the caudal vena cava and the portal vein can be precisely measured. However, because of the extreme changes in caliber of the caudal vena cava as a result of respiratory and cardiac pressure changes, the caudal vena cava cannot be used as a reference vessel for area comparisons. Instead, measuring the cross-sectional area of the aorta may be more reliable, because there will be fewer changes in caliber. In any event, the relative size of the extrahepatic portal vein has no apparent predictive value on the outcome of the surgery.

Comparing the relative sizes of the portal veins of dogs of different ages, however, seemed to confirm observations made by one of the authors (FJvS) that large-breed dogs are born with vessels with calibers relatively larger than those in small-breed dogs. As a consequence, the postnatal growth rate of the blood vessels in large-breed dogs is less than that of the body as a whole.

Result of shunt attenuation were evaluated 1 month after surgery in the 7 dogs in this report because approximately 1 month is necessary for acquired collaterals to develop following the onset of severe portal hypertension.[21,27] The second re-examination was performed 3 months after surgery on the basis of our clinical experience that changes in the portal system may still be expected during this period, but that after 3 months, further changes are typically not seen. One month after surgery, follow-up abdominal ultrasonography revealed that the liver had become larger, compared with its preoperative state, in all dogs, presumably as a result of more blood of portal origin flowing through the portal branches. Portal blood is rich in the nutrients and gastrointestinal hormones necessary for the normal function and development of the liver.[1,29] A substantial reduction of the cross-sectional area of the shunt was achieved in each dog, which in turn reduced the amount of blood that was shunted and increased the amount of portal blood that reached the liver. We also found that partial ligation of the shunt was sufficient to resolve signs of hepatic encephalopathy. Moreover, we could confirm the observation of Meyer et al[18] in 1 dog that partial shunt attenuation may progress to spontaneous shunt occlusion.

Although our population was small, all had a favorable clinical outcome and the intraoperative time-averaged mean portal flow velocity after shunt attenuation was < 9.5 cm/s in each patient. This value could be considered the minimum velocity that would not result in signs of severe portal hypertension; however, more patients must be examined to establish a safe cut-off value. If the time-averaged mean velocity before shunt attenuation is < 15 cm/s, we recommend that it be no less than 5 cm/s after attenuation, as this was the lowest registered velocity among our patients. However, as none of the 7 dogs developed fatal acute or sustained portal hypertension after surgery, we cannot report whether the postligation time-averaged mean portal velocity in such dogs would be different (ie, significantly lower), and further speculation would be inappropriate.

As a gold standard for determining the acceptable degree of shunt attenuation in these dogs, we used color of the viscera together with changes in arterial blood pressure and

heart rate and results of follow-up examinations. Previous studies showed that using viscera color assessment for predicting postoperative portal hypertension resulted in a similar percentage of false-negative results (6%)[4] as did portal pressure measurement (8 and 6%).[8,11] Direct portal pressure measurement, thus, would not have provided any additional information. Furthermore, we elected to not directly measure portal pressure because a variety of factors make the interpretation of pressure changes unreliable[2] and we did not want to perform more-invasive procedures on client-owned dogs (ie, catheterization of a jejunal vein) than they would otherwise experience, as portal pressure measurement is not part of the surgical protocol at our clinic.

A major problem with all of the techniques that have been recommended for the treatment of congenital portosystemic shunts in dogs is that the long-term surgical outcome has always been unpredictable. Many attempts have been made to identify pre- or intraoperative measurements that could be associated with an uncomplicated outcome,[4,5,10-18,43-47] as well as to identify newer techniques for shunt-attenuation that would not result in postoperative complications. However, all attempts have failed. Several investigators have suggested that fewer postoperative complications and fewer recurrences of hepatic encephalopathy can be expected after complete shunt ligation, compared with partial shunt attenuation.[5,11,12,45] Others, however, cannot confirm this theory,[13,18,44,46-48] and portal vein thrombosis, which is a rare but fatal complication, is thought to be associated with complete shunt ligation.[48] Another argument against complete shunt ligation is that partial shunt attenuation in many cases is followed by spontaneous complete shunt occlusion.[18,43] Our aim was to investigate another aspect of the portal circulation that has not been examined to date in relation to shunt attenuation, namely portal flow and portal flow velocity. We had hoped that these variables might be more reliable in predicting outcome. However, at the current stage of our research on intrahepatic portocaval shunts, we are unable to show that portal flow velocity values obtained intraoperatively would give any extra information to the surgeon.

In summary, with intraoperative ultrasonography, direct information can be obtained noninvasively, quickly, and without radiation exposure about portal flow and the anatomy of intrahepatic portosystemic shunts and the portal branches. Thus, intraoperative ultrasonography may be an alternative to both direct portal pressure measurements and mesenteric portography. However, it is necessary to examine more patients, especially patients with poor outcome, to determine whether portal flow velocity is more reliably related to surgical outcome than currently used variables.

References

1. Van den Ingh TSGAM, Rothuizen J, Meyer HP. Circulatory disorders of the liver in dogs and cats. *Vet Q* 1995;17:70–76.
2. White RN, Burton CA, McEvoy FJ. Surgical treatment of intrahepatic portosystemic shunts in 45 dogs. *Vet Rec* 1998;142:358–365.
3. Watson P. Decision making in the management of porto-systemic shunts. *In Pract* 1997;19:106–120.
4. Wolschrijn CF, Mahapokai W, Rothuizen J, et al. Gauged attenuation of congenital portosystemic shunts: results in 160 dogs and 15 cats. *Vet Q* 2000;22:94–98.

5. Hottinger HA, Walshaw R, Hauptman JG. Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. *Vet Surg* 1995;24:331–336.
6. Ewing GO, Suter PF, Bailey CS. Hepatic insufficiency associated with congenital anomalies of the portal vein in dogs. *J Am Anim Hosp Assoc* 1974;10:463–476.
7. Breznock EM. Surgical manipulation of portosystemic shunts in dogs. *J Am Vet Med Assoc* 1979;174:819–826.
8. Komtebedde J, Forsyth SF, Breznock EM, et al. Intrahepatic portosystemic venous anomaly in the dog. Perioperative management and complications. *Vet Surg* 1991;20;37–42.
9. Swalec Tobias KM, Rawlings CA. Surgical techniques for extravascular occlusion of intrahepatic shunts. *Compend Contin Educ Pract Vet* 1996;18:745–754.
10. Murphy ST, Ellison GW, Long M, et al. A comparison of the ameroid constrictor versus ligation in the surgical management of single extrahepatic shunts. *J Am Anim Hosp Assoc* 2001;37:390–396.
11. Johnson CA, Armstrong PJ, Hauptman JG. Congenital portosystemic shunts in dogs: 46 cases (1979–1986). *J Am Vet Med Assoc* 1987;191:1478–1483.
12. Scavelli TD. Complications associated with the diagnostic, medical, and surgical management of portosystemic shunts. *Probl Vet Med* 1989;1:147–158.
13. Mathews K, Gofton N. Congenital extrahepatic portosystemic shunt occlusion in the dog: gross observations during surgical correction. *J Am Anim Hosp Assoc* 1988;24:387–394.
14. Vogt JC, Krahwinkel DJ, Bright RM, et al. Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using the ameroid constrictor. *Vet Surg* 1996;25:495–502.
15. Kyles AE, Gregory CR, Jackson J, et al. Evaluation of a portocaval venograft and ameroid ring for the occlusion of intrahepatic portocaval shunts in dogs. *Vet Surg* 2001;30:161–169.
16. Youmans KR, Hunt GB. Cellophane banding for the gradual attenuation of single extrahepatic portosystemic shunts in eleven dogs. *Aust Vet J* 1998;76:531–537.
17. Youmans KR, Hunt GB. Experimental evaluation of four methods of progressive venous attenuation in dogs. *Vet Surg* 1999;28:38–47.
18. Meyer HP, Rothuizen J, van Sluijs FJ, et al. Progressive remission of portosystemic shunting in 23 dogs after partial closure of congenital portosystemic shunts. *Vet Rec* 1999;144:333–337.
19. Van den Ingh TSGAM, Rothuizen J, Meyer HP. Portal hypertension associated with primary hypoplasia of the hepatic portal vein in dogs. *Vet Rec* 1995;137:424–427.
20. Phillips L, Tappe J, Lyman R, et al. Hepatic microvascular dysplasia in dogs. *Prog Vet Neurol* 1996;7:88–96.
21. Nyland TG, Fischer PE. Evaluation of experimentally induced canine hepatic cirrhosis using duplex Doppler ultrasound. *Vet Radiol* 1990;31:189–194.
22. Lamb CR, Mahoney PN. Comparison of three methods for calculating portal blood flow velocity in dogs using duplex-Doppler ultrasonography. *Vet Radiol Ultrasound* 1994;35:190–194.
23. Szatmári V, Sótonyi P, Vörös K. Normal duplex Doppler waveforms of the major abdominal blood vessels in dogs: a review. *Vet Radiol Ultrasound* 2001;42:93–107.
24. Kantrowitz BM, Nyland TG, Fischer P. Estimation of portal blood flow using duplex real-time and pulsed Doppler ultrasound imaging in the dog. *Vet Radiol* 1989;30:222–226.
25. Bolondi L, Gaiani S, Barbara L. Accuracy and reproducibility of portal flow measurement by Doppler US. *J Hepatol* 1991;13:269–273.
26. Moriyasu F, Nishida O, Ban N, et al. "Congestion index" of the portal vein. *Am J Roentgenol* 1986;146:735–739.
27. Vitums A. Portosystemic communications in the dog. *Acta Anat (Basel)* 1959;39: 271–299.
28. Valentine RW, Carpenter JL. Spleno-mesenteric-renal venous shunt in two dogs. *Vet Pathol* 1990;27:58–60.

29. Johnson SE. Portal hypertension. Part I. Pathophysiology and clinical consequences. *Compend Contin Educ Pract Vet* 1987;9:741–748.
30. Wrigley RH, Macy DW, Wykes PM. Ligation of ductus venosus in a dog, using ultrasonographic guidance. *J Am Vet Med Assoc* 1983;183:1461–1464.
31. Breznock EM, Berger B, Pendray D, et al. Surgical manipulation of intrahepatic portocaval shunts in dogs. *J Am Vet Med Assoc* 1983;182:798–805.
32. Lamb CR, White RN. Morphology of congenital intrahepatic portocaval shunts in dogs and cats. *Vet Rec* 1998;142:55–60.
33. Penninck DG, Finn-Bodner ST. Updates in interventional ultrasonography. *Vet Clin North Am Small Anim Pract* 1998;28:1017–1040.
34. de Vries PJ, van Hattum J, Hoekstra JB, et al. Duplex Doppler measurements of portal venous flow in normal subjects—inter- and intra-observer variability. *J Hepatol* 1991;13:358–363.
35. Gill RW. Measurement of blood flow by ultrasound; accuracy and sources of error. *Ultrasound Med Biol* 1985;11:625–641.
36. Zironi G, Gaiani S, Fenyves D, et al. Value of measurement of mean portal flow velocity by Doppler flowmetry in the diagnosis of portal hypertension. *J Hepatol* 1992;16:298–303.
37. Haag K, Rössle M, Ochs A, et al. Correlation of duplex Doppler sonography findings and portal pressure in 375 patients with portal hypertension. *Am J Roentgenol* 1999;172:631–635.
38. Iwao T, Toyanaga A, Oho K, et al. Value of Doppler ultrasound parameters of portal vein and hepatic artery in the diagnosis of cirrhosis and portal hypertension. *Am J Gastroenterol* 1997;92:1012–1017.
39. Iwao T, Toyanaga A, Shigemori H, et al. Echo-Doppler measurements of portal vein and superior mesenteric artery blood flow in humans: inter- and intra-observer short-term reproducibility. *J Gastroenterol Hepatol* 1996;11:40–46.
40. Lee Y-W. Pulsed Doppler ultrasonographic evaluation of portal blood flow in dogs with experimental portal vein branch ligation. *J Vet Med Sci* 1999;61:59–61.
41. Finn-Bodner ST, Hudson JA. Abdominal vascular sonography. *Vet Clin North Am Small Anim Pract* 1998;28:887–942.
42. Sabbà C, Ferraioli G, Buonamico P, et al. Echo-Doppler evaluation of acute flow changes in portal hypertensive patients: flow velocity as a reliable parameter. *J Hepatol* 1992;15:356–360.
43. Van Vechten BJ, Komtebedde J, Koblik PD. Use of transcolonic portal scintigraphy to monitor blood flow and progressive postoperative attenuation of partially ligated single extrahepatic portosystemic shunts in dogs. *J Am Vet Med Assoc* 1994;204:1770–1774.
44. Lawrence D, Bellah JR, Diaz R. Results of surgical management of portosystemic shunts in dogs: 20 cases (1985–1990). *J Am Vet Med Assoc* 1992;201:1750–1753.
45. Hunt GB, Hughes J. Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. *Aust Vet J* 1999;77:303–307.
46. Komtebedde J, Koblik PD, Breznock EM et al. Long-term clinical outcome after partial ligation of single extrahepatic vascular anomalies in 20 dogs. *Vet Surg* 1995;24:379–383.
47. Bostwick DR, Twedt DC. Intrahepatic and extrahepatic portal venous anomalies in dogs: 52 cases (1982–1992). *J Am Vet Med Assoc* 1995;206:1181–1185.
48. Roy RG, Post GS, Waters DJ, et al. Portal vein thrombosis as a complication of portosystemic shunt ligation in two dogs. *J Am Anim Hosp Assoc* 1992;28:53–58.