ULTRASONOGRAPHY OF PORTOSYSTEMIC SHUNTING IN DOGS

DOPPLER STUDIES BEFORE, DURING AND AFTER SURGERY

Echografie van portosystemische shunting bij de hond
Dopplerondersoeken voor, tijdens en na chirurgie

(*met een samenvatting in het Nederlands*)

Kutyák portoszisztémás söntjének ultrahangvizsgálata
Doppler vizsgálatok műtét előtt, alatt és után

(*magyar nyelvű összefoglalóval*)

Proefschrift ter verkrijging van de graad van doctor aan de Universiteit Utrecht
op gezag van de Rector Magnificus, Prof. Dr. W. H. Gispen,
ingevolge het besluit van het College voor Promoties in het openbaar te verdedigen
op donderdag 22 april 2004 des middags te 16.15 uur

door

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Geboren op 3 mei 1972, te Debrecen

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Szatmári Viktor: Ultrasonography of portosystemic shunting in dogs; Doppler studies before, during and after surgery

PhD thesis, Utrecht University, 2004

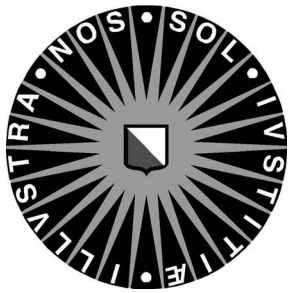
ISBN 90-393-3625-3

Cover design: Chiharu Nakamura, Aart van der Woude & Viktor Szatmári

Illustrations: Aart van der Woude & Viktor Szatmári

Printer: OPTIMA [Dick Ladage], Rotterdam, the Netherlands

The studies described in this book were conducted at and supported by



The author wishes to thank the Office for International Cooperation (BIC) of the Faculty of Veterinary Medicine of Utrecht University for establishing the International PhD Programme.

The author was financially supported during 2 years of his research by

Hungarian State Eötvös Scholarship

Additional financial assistance was provided by

Contents

CHAPTER 1

General introduction

Abdominal vascular anatomy in normal dogs

The aorta, the caudal vena cava and the portal vein are the three great abdominal vessels. They all course parallel to the vertebral column. The aorta is the most dorsal and its major branches from cranial to caudal are: the celiac, the cranial mesenteric, the right and left renal arteries, and before the final trifurcation: the right and left external iliac arteries.[1] The celiac artery branches further into three arteries, of which the largest is the common hepatic artery, which runs cranially between the portal vein and the caudal vena cava (**Fig 1**). The smallest intrahepatic branches of the hepatic artery terminate in the capillary system of the liver, in the hepatic sinusoids.

The caudal vena cava is formed by the confluence of the right and left common iliac veins at the level of the aortic trifurcation. The caudal vena cava courses ventral to the aorta and after entering the thoracic cavity it terminates in the right atrium of the heart. The abdominal caudal vena cava collects the blood of the left and the right renal veins, then as it passes through the liver collects the blood of the hepatic veins. The hepatic veins drain the blood of the hepatic sinusoids and are straight vessels running in the liver lobes to a craniomedial direction, entering the caudal vena cava. The left gonadal vein (ovarian vein in females and testicular vein in males) enters the left renal vein, whereas the right gonadal vein is a direct tributary of the caudal vena cava.[2]

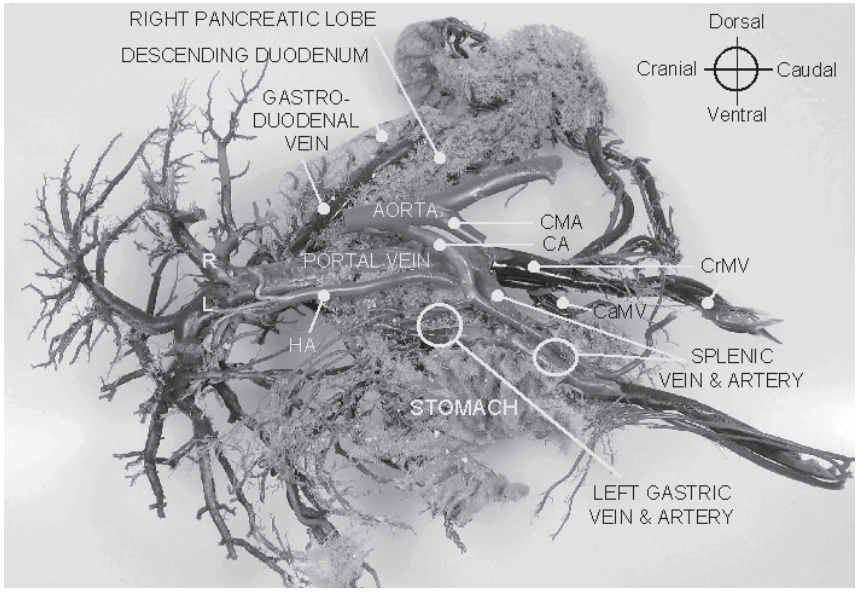


Figure 1.

Corrosion cast of the portal vein (blue) and the cranial part of the abdominal aorta (red) in an adult normal beagle. R right portal branch, L left portal branch, CA celiac artery, CMA cranial mesenteric artery, HA hepatic artery; CrMV cranial mesenteric vein, CaMV caudal mesenteric vein. (Full **color** illustration on page 177.)

The positions of the vessels do not show the real *in situ* situation because the spleen, jejunum, ileum and colon were removed and the descending duodenum was retracted.

The portal vein is formed by the confluence of the cranial and caudal mesenteric veins.[2] The trunk of the portal vein collects the blood of the splenic vein at the level where the celiac artery originates from the aorta, and the blood of the gastroduodenal vein immediately caudal to the portal bifurcation. The left gastric vein is a tributary of the splenic vein and may anastomose with the right gastric vein. The right gastric vein is either a tributary of the gastroduodenal vein or enters the portal vein trunk directly cranial to the gastroduodenal vein after a course along the lesser curvature of the stomach.[3] At the hilus of the liver the trunk of the portal vein bifurcates into a larger left and a smaller right portal branch. The right branch runs dorsally, the left one ventrally. The right branch supplies the right lateral and the caudate lobes, whereas the left branch supplies the left lateral, left medial, quadrate and right medial lobes. The smallest portal branches are continued in the hepatic sinusoids where their blood mixes with the hepatic arterial blood.

The azygos vein is a thin vessel that courses dorsal to the aorta and after passing through the diaphragm it enters the cranial vena cava, which terminates in the right atrium of the heart.[2]

The caudal vena cava and the azygos vein together with their tributaries belong to the systemic venous system, and the portal vein together with its tributaries form the portal venous system. No macroscopic communication exists between the systemic and the portal venous systems.

Portosystemic shunting

Portosystemic shunting occurs when anomalous veins allow the portal blood to enter the systemic veins directly without first flowing through the hepatic sinusoids.[4] Portosystemic shunting can occur via acquired portosystemic collaterals or via congenital portosystemic shunts.

Acquired portosystemic collaterals are formed as the result of sustained hepatic or prehepatic portal hypertension by enlargement of extrahepatic rudimentary vessels, through which no blood normally passes.[5,6] Collateral-formation is a compensatory mechanism to maintain normal portal pressure by allowing the portal blood to be drained into the lower pressure systemic veins.

Portosystemic shunting is considered to be congenital if a single or double anomalous vein is present without a concurrent portal hypertension.[4,6] Congenital portosystemic shunts are classified as intrahepatic and extrahepatic.

Consequences of portosystemic shunting

Under normal circumstances the blood from the gastrointestinal tract flows via the portal vein to the sinusoids of the liver. Subsequently, the detoxified blood is drained via the hepatic veins to the systemic circulation. Shunting of the portal blood has two important consequences: (1) the blood from the guts, containing toxins and nutrients, enters the systemic circulation directly resulting in hyperammonemia among other things, and (2) the amount of portal venous blood that perfuses the liver is insufficient resulting in impaired hepatic development and function.

Ammonia is one of the toxins that plays a role in the development of hepatic encephalopathy, which is the most characteristic clinical sign of dogs with portosystemic shunting. Therefore, an increased blood ammonia level confirms that the central nervous symptoms of a dog are indeed in association with hepatic encephalopathy, or can reveal that the underlying cause of aspecific clinical symptoms is related to hyperammonemia. Dogs with portosystemic shunting can show clinical signs from the age of 2 months, but even a congenital portosystemic shunt may remain clinically silent until old age.[4]

Clinical signs of dogs with portosystemic shunting vary substantially.[4,7] In dogs with acquired portosystemic collaterals, signs of portal hypertension such as ascites may dominate, whereas in dogs with congenital portosystemic shunts, signs of hepatic encephalopathy such as circling, head pressing, blindness, seizures, dementia, or coma may be the leading symptoms. Often, however, only aspecific signs such as anorexia, vomiting, diarrhea, or polyuria/polydipsia are found. The presenting signs may also be hematuria or stranguria in association with ammonium-urate urolithiasis. Stunted growth can occur in young dogs both with congenital or acquired portal vein anomalies. In an otherwise healthy dog a prolonged or complicated recovery from a sedation or anesthesia can draw the veterinarian's attention to an underlying portosystemic shunting.

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CHAPTER 2

The aim and scope of the thesis

Ultrasonography for diagnostic imaging of abdominal blood vessels

Real time gray scale ultrasonography provides detailed anatomic information of the abdominal organs and blood vessels non-invasively without ionizing radiation in unsedated dogs.[1] With color and spectral Doppler techniques the presence, direction and velocity of flow can be determined in any given vessels. In Chapter 3 the principles of canine abdominal vascular Doppler ultrasonography are described and the normal spectral Doppler waveforms of the major abdominal vessels are illustrated.

Diagnosis of portosystemic shunting with ultrasound Can it be accurate?

Differentiating **congenital portosystemic shunts** (**CPSSs**) non-invasively from other conditions that cause hyperammonemia such as **acquired portosystemic collaterals** (**APSCs**) and urea cycle enzyme deficiency is crucial because CPSSs require surgical treatment and the other two conditions do not. Portosystemic shunting is suspected based on the history, clinical and laboratory findings (particularly elevated blood ammonia and bile acid concentrations), however direct visualization of the aberrant vein is essential for a definitive diagnosis.[2] Though ultrasonography is a quick and non-invasive technique for imaging the abdominal vessels and organs in unsedated dogs, veterinarians tend to rely on the results of angiographic studies.[3,4] Descriptions of the ultrasound-anatomy of the different types of CPSSs and APSCs in dogs were not found in the literature. The few reports that have been published about ultrasonography of portosystemic shunting focus only on some aspects of CPSSs without detailed anatomic descriptions and the ultrasonographic features of APSCs are discussed only superficially, or not at all.[2,3,5-8]

In Chapter 4 the abdominal ultrasonographic findings of the conditions that result in hyperammonemia in dogs and the ultrasonographic anatomy of the different kinds of CPSSs are described. Ultrasonography proved to be a reliable method to diagnose CPSSs and APSCs, and to identify the subtypes of CPSSs as well as to rule out portosystemic shunting.

For diagnostic imaging of portal vein disorders, angiography was the first modality that has been used and is still the most frequently used technique.[9,10] The interpretation of the portographic images is simple and the differentiation of APSCs from CPSSs, and intra- from extrahepatic CPSSs is possible.[9,11] However, portography is a time-consuming and invasive procedure that involves radiation.[12] Although scintigraphy is the gold standard for the detection or exclusion of portosystemic shunting, it does not allow differentiation of CPSSs from APSCs.[13,14] Computed tomography and magnetic resonance imaging give detailed anatomic information of the vessels,[15,16] however cannot determine the flow-direction in them, moreover both procedures are time-consuming and require general anesthesia. Ultrasonography has been used for diagnostic imaging of CPSSs since the 1980`s,[5] and has become popular because the examination is quick, non-invasive, does not require anesthesia or radiation, and the abdominal organs can be simultaneously evaluated. Currently no other modalities can compete with the advantages of color Doppler ultrasonography in abdominal vascular imaging, but veterinarians still tend to rely on the results of other or additional diagnostic procedures (e.g. explorative laparotomy and portography) because of the reported low sensitivity and specificity of ultrasound.[8] The

reason for the insufficient accuracy of the published ultrasound-studies could be that no standardized protocol has been used; and no descriptions have been available how the different types of CPSSs can be recognized and differentiated from one another.

In Chapter 5 an examination protocol for systematic ultrasonographic evaluation of the canine portal system is described. This protocol has allowed us not only to recognize and characterize the different types of CPSSs and APSCs accurately, but also to exclude portosystemic shunting in a large number of patients.

Surgical treatment of congenital portosystemic shunts Are the complications predictable and preventable?

The definitive therapy for CPSSs would ideally be complete occlusion of the shunt at a location closest to the systemic venous circulation.[17,18] However, in most dogs, only partial shunt ligation can be performed because attenuating the shunt vessel forces blood to flow through the portal branches, which are frequently hypoplastic, resulting in post-ligation portal hypertension.[13,17,19-22] The degree of post-ligation portal hypertension depends on the degree of attenuation and the capacity of the previously hypoperfused portal branches. A greater degree of attenuation is associated with development of more severe portal hypertension. Portal vein hypoplasia may be primary (i.e. a congenital anomaly) or may arise secondary as a result of portal hypoperfusion.[23] 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 blood flow.[21] When severe post-ligation portal hypertension develops, the patient may die during or shortly after surgery as a result of circulatory collapse or thrombosis of the portal vein.[17,24,25] Even when these acute complications do not occur, APSCs may develop as a result of sustained portal hypertension.[17]

During surgical ligation of a CPSS, the narrowest possible shunt diameter is determined in steps by assessing portal hypertension.[18] To avoid acute fatal portal hypertension, two 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.[9,12,22,24,26-30] Although this method allows quantitative assessment of portal hypertension, several factors can make the interpretation of pressure changes unreliable.[26,27,29,30] In addition, fatal hemorrhage may occur as a complication of the catheterization.[24,27] 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 acceptable degree of post-ligation portal hypertension.[18,20,22,27,31,32] Both methods allow acute portal hypertension to be successfully avoided; however, development of chronic portal hypertension remains a frequent complication.[17,20,22,24,27]

Some years ago, ameroid constrictors were introduced in portosystemic shunt surgery. These devices are placed around the shunt vessels and cause their gradual attenuation, resulting in complete shunt occlusion in one to several weeks' time.[30,33,34] In theory, gradual shunt attenuation would allow 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 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, subacute or chronic portal hypertension may develop.[33,35] Application of ameroid constrictors on intrahepatic shunts involves further 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.[34,35] 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 or resection of the affected liver lobe.[35,36]

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.[34,37]

Regardless of the technique used for shunt attenuation and for assessing postligation portal hypertension, the clinical outcome remains unpredictable,[22,24,27-31,33,35,38] 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.[13,18,23,24] Use of histopathologic changes in the liver,[21,23] portal pressure,[17,20,25] partial versus complete shunt ligation,[17,20,26,28,32,39] age of the dogs,[18,39] and portographic images[17,20] have not yielded satisfactory results.

In Chapter 6 intraoperative ultrasonography as a noninvasive technique for quantitative assessment of postligation portal flow is described in dogs with intrahepatic portosystemic shunts as an alternative of portal pressure measurement. Moreover, intraoperative ultrasonography appears to be an excellent alternative to mesenteric portography.

In Chapter 7 the portal hemodynamic changes associated with surgical ligation of extrahepatic CPSSs and recommend ultrasonographic criteria for determining the optimal degree of shunt narrowing are described.

Postoperative follow-up of attenuated congenital portosystemic shunts Is a second surgery necessary after a partial closure?

An improved clinical outcome has been related to complete occlusion of a CPSS.[10,20,26,28,32,40] However, other groups found no difference in outcomes between dogs with partial and complete shunt-ligation.[17,27,31,39,41] Although the underlying causes of poor outcome in most of the studies remained undetermined, it has been suggested that, if complete shunt-occlusion is not feasible during a surgical attenuation of a CPSS because it would cause the development of a fatal portal hypertension, a second surgery should be performed to attempt a complete shunt-occlusion.[10,28] The underlying idea is that an initial partial ligation would allow the portal system to adapt to an increased flow and the portal branches would have become gradually wider by the time of the second surgery.

A second surgery should only be considered when portosystemic shunting persists exclusively through the CPSS. When shunting occurs through APSCs, further attenuation of the CPSS is contraindicated. To determine whether post-ligation portosystemic shunting occurs via the CPSS, APSCs or both, mesenteric portography has been used.[33] In addition to the fact that this angiographic technique involves the use of ionizing radiation, it requires general anesthesia and a laparotomy, both of which are high risk procedures in animals with

hepatic insufficiency.[42] Though scintigraphy is suitable to detect portosystemic shunting,[17] and with labeled microspheres to quantify it,[13] with this technique it is impossible to distinguish between congenital and acquired shunting. Doppler ultrasonography offers a non-invasive way to examine the abdominal blood vessels in unsedated dogs, and the direction and velocity of flow can easily be determined. However, no reports were found in the literature about the ultrasonographic findings of dogs that underwent partial surgical ligation of a CPSS.

In Chapter 8 the post-ligation hemodynamic changes of the portal system are described. We give ultrasonographic criteria for deciding whether a dog with a partially attenuated CPSS should or should not undergo a second laparotomy for complete shunt-ligation are suggested. Furthermore, an explanation at the level of portal hemodynamics as to why certain dogs with a partially attenuated extrahepatic CPSS have excellent and others poor clinical outcomes is proposed.

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CHAPTER 3

Principles of abdominal vascular Doppler ultrasonography in dogs

based on the article by

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Normal duplex Doppler waveformsof major abdominal blood vessels in dogs: a review

Veterinary Radiology & Ultrasound
2001;42:93-107.

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Summary

The normal flow velocity profile and duplex Doppler waveform of the major abdominal blood vessels (aorta, caudal vena cava and the portal vein as well as their major branches) were examined by Doppler ultrasound. The flow velocity profile of an artery is largely determined by its diameter. The pulsatility of the waveform is related to the vascular impedance downstream to the point of measurement. Early systolic peak is present in the Doppler pattern of some vessels in some dogs. The waveform of the veins is mainly affected by the pressure conditions of the right atrium and the intrathoracic and intraabdominal pressure changes due to the respiration. Simultaneous electrocardiogram was used to reveal the effect of the heart beats on the Doppler patterns of the veins.

Introduction

Doppler ultrasonography is relatively new within small animal sonography. Duplex Doppler ultrasonography provides both real time anatomic and dynamic information. The presence, direction and type of blood flow can be determined during Doppler examinations.

The knowledge of the normal Doppler signs of each blood vessel is important in their identification because a spectral Doppler signal is fairly specific to a particular vessel, or even vessel site.[1,2] Recognizing the pathologic changes in a Doppler spectrum is only possible if the sonographer knows the variations of normal.

The aim of this paper is to describe the normal Doppler patterns of the major abdominal blood vessels in the dog by reviewing the literature and using results from our own examinations. According to our knowledge, there is only one review article dealing with the Doppler waveforms of canine abdominal blood vessels.[3] However, there are reports where the results of Doppler examinations of renal[4-6], hepatic[7,8], prostatic[9], fetal and maternal arteries[10], as well as portal[8,11-14], hepatic[15] and splenic veins[16], in healthy dogs or in dogs with various diseases are described. In our study, simultaneous electrocardiography was used during the examination of veins.

The sonographic examinations were performed by the first author using a commercially available ultrasound machine (Panther 2002, Brüel & Kjaer, equipped with a transducers of 3.5 and 5.0 MHz; the images of Figs 1, 2, 5, 18, 20, 21B were made with an ATL HDI 3000, Philips Medical Systems, Advanced Technical Laboratories Ultrasound). At least 40 dogs were examined, but not all vessels were evaluated in each animal. Doppler tracings were recorded from conscious animals except in dogs with high respiratory and heart rate. In these dogs it was difficult to separate the effect of heart beat and respiration on the Doppler signal. Therefore, the combination of xylazine and diazepam was administered intramuscularly. Approximately 10 examinations were conducted with sedation in 5 healthy animals.

Two-dimensional B-mode real-time image of blood vessels

Finding and recognising the major abdominal vessels during abdominal ultrasound examinations is essential because (1) vessels may serve as landmarks to localise certain organs (e.g. adrenal glands), (2) vascular disorders (e.g. thrombosis) can be readily diagnosed, and (3) finding an aberrant vessel can give a definitive clinical diagnosis (e.g. portosystemic shunts). To know the anatomic relations of blood vessels to one another and to the abdominal organs is essential. Corrosions casts are great study-materials because the topographic anatomy of the arteries and veins as well as of the adjacent organs can simultaneously be appreciated.

Abdominal blood vessels in longitudinal plane have a tubular structure with well-defined walls. The walls are parallel, hyperechoic, thin smooth lines. Non-compressed vessels imaged transversely appear circular or oval. The lumen is anechoic because of the echofree blood inside.[2] When blood flow is slow and the diameter of a vessel is large enough, aggregated red blood cells can be seen as moving echoes.[17]

Two-dimensional real-time sonography is suitable to detect abnormally located vessels (e.g. shunts), to measure the diameter of the vessels (e.g. dilation), to see the smoothness and thickness of the vessel wall or to identify abnormal intraluminal or perivascular structures (e.g. thrombus or tumor). However, a fresh thrombus can be echofree like blood itself.[3] Pulsation of arteries may also be seen, but it must be realized that the pulsation of an artery can be referred to an adjacent vein.[2]

Physical principles of Doppler ultrasonography, the Doppler effect

The frequency of the reflected sound changes, if the reflector moves with respect to the sound source.[18] During the examination of blood vessels, the reflectors are the moving blood cells (actually the red blood cells, because of their great majority among the blood cells). The difference between the frequency of the transmitted and the reflected sound is called Doppler-frequency-shift or *Doppler-shift* (Δf).[19] If the direction of blood flow is towards the transducer, then the Doppler-shift will be positive; i.e. the returning echo will have a higher frequency.[3] If the direction of flow is away from the probe, then the Doppler-shift is negative (i.e. the frequency of the reflected ultrasound is lower, than that of the transmitted one).

The Doppler-shift is displayed in various ways depending on the Doppler technique. Using spectral Doppler mode, a Doppler tracing can be seen and simultaneous sound can be heard. Using color or power Doppler mode, the Doppler-shift is displayed as shades of two or one color within a color box, respectively.

The Doppler equation ($\Delta f=2^{x}f^{x}v^{x}\cos\Theta/c$) contains parameters that influence the magnitude of the Doppler frequency shift (Δf). The parameter f = ultrasonic beam frequency (frequency of the transducer), v = red blood cell velocity, Θ = Doppler angle (the intersection angle of the axis of the vessel and the axis of the ultrasound beam), and c = mean velocity of the ultrasound in living tissues (1540 m/s).[1,3,18,19]

The magnitude of the frequency change (Doppler-shift) is the greatest when the ultrasound beam and the axis of the blood vessel are parallel to each other ($\cos0°=1$). There is no useful Doppler-shift if they are perpendicular to each other because $\cos90°=0$.

Spectral Doppler ultrasonography

Spectral Doppler ultrasound techniques display a Doppler spectrum, and has two types: continuous and pulsed wave Doppler modes. Continuous wave Doppler is used in cardiology to measure high velocities, whereas pulsed wave Doppler mode is used in abdominal or peripheral vascular studies.

A *pulsed wave* Doppler transducer contains one piezoelectric crystal. The crystal transmits short bursts of ultrasound (pulses) at regular intervals and the same crystal receives the reflected signal in the rest of the time and compares it with the transmitted reference.[18,20] Pulsing the ultrasound wave allows Doppler measurements to be taken from a specific region within the imaged field, thus allowing velocity measurements from selected vessels.[11] How often the pulses are emitted is called *pulse repetition frequency* (PRF). The time delay between the pulse and the echoes depends on the site of the reflector, therefore it is possible to locate the source of the returning echoes.[18] This technique allows

precise localization of the volume of tissue from which the Doppler blood flow signal is sampled in contrast with the continuous wave Doppler technique.[1] The region from which Doppler signals are measured is called the *sample volume* (or *gate*).[19]

Duplex imaging combines pulsed wave Doppler with two-dimensional real-time image. The location of the target volume is displayed on the B-scan tomogram.[1] The sampling gate can be moved into the lumen of the vessel as seen on the real-time image.[21] The velocity changes that occur in a vessel throughout each cardiac cycle is displayed graphically. The running time is on the horizontal axis (*baseline*), the Doppler shift frequency can be read on the vertical axis in kHz. If the manually rotated cursor is aligned parallel to the blood vessel (angle-correction), the velocity of the moving cells (i.e. blood flow) can be seen in cm/s on the vertical axis.[18] By convention, flow towards the transducer is displayed above the baseline, and flow away from the transducer is displayed below the baseline. Baseline represents zero flow. True velocity-values can only be obtained, if the Doppler angle does not exceed 60º, because a small error in estimation of the angle makes a great error above this value (this is because of the nature of the cosine-curve).[18,19] The angle correction can also be done on the frozen image (postprocessing), but always should be done before measuring velocities.

The brightness of the spectrum indicates the quantity of blood.[1] The brighter a site of the spectral tracing is, the more cells flowed at that particular velocity at a specific time through the sample volume.

During duplex Doppler examination, the Doppler-shift is not only displayed graphically, but the Doppler shift frequencies are also audible to human beings at the same time (in the frequency-range of diagnostic ultrasound transducers). The arteries have swish-like sounds, whereas most of the veins have continuous wind-blow-like sounds. The intensity of the audible sound is in direct proportion to the quantity of moving blood cells. The height of the sound depends on the Doppler-shift. The higher the velocity of the flowing blood is (i.e. the larger the Doppler-shift is), the higher the audible sound is.[22]

Pulsed wave Doppler sonography allows the assessment of the presence, direction and velocity of blood flow within the sample volume. The technique is also suitable to differentiate between arteries and veins. Furthermore, discrimination between the various flow velocity profiles of the arteries as well as between arteries of low and high resistance of flow is also possible. Every blood vessel has its own characteristic Doppler-pattern, like its “signature”, which can be used to aid its identification. Changes in the waveform may also have pathologic meaning.

The most frequently used methods to measure the blood flow velocity in a vessel are the *uniform insonation method* (i.e. the entire lumen of the vessel is incorporated into the gate) and the *maximum velocity method* [i.e. the small sample volume (approximately half of the diameter of the vessel) is placed in the center of the vessel].[3,19]

Color Doppler imaging

The basics of this technique are the same as that of the pulsed wave Doppler mode. In contrast with the pulsed wave Doppler technique, there are multiple sample volumes within a circumscribed region called a *color box* instead of one visible one. None of these sample volumes is displayed on the monitor, only the border of the color box is imaged on the B-mode tomogram. The size and position of the color box on the B-mode image is determined by the operator. Color Doppler sonography displays the two-dimensional flow information in color superimposed on the B-mode image of the vessel and surrounding tissue. Within the color box, every moving point is a shade of red or blue instead of a shade of the gray. The direction of flow relative to the transducer is illustrated on a color bar beside the image.[23] By convention, flow towards the transducer is red, flow away from the transducer is blue. Since these colors could be inverted with a single button control, checking the color bar is always advisable. The higher flow velocities are indicated by lighter hues of the same color.

Color Doppler ultrasound technique gives movement-information about a large part of the image, like a map (in contrast to spectral Doppler). Color Doppler flow imaging allows assessment of the presence, direction, and quality of blood flow more rapidly than does any other noninvasive technique, even in areas that do not appear as vessels on B-mode examination.[21] Differentiation between slow and fast flow is also possible.

A major advantage of colour Doppler ultrasonography is that not only the flow in a selected vessel can be detected (like with angiography), but also the vessels' anatomic relations to the abdominal organs and to other vessels can be appreciated.

To obtain good quality color Doppler images, the PRF (or scale) should be adjusted for each vessel individually by setting it high enough to prevent aliasing artefact (see below), and low enough so that a slow flow will not be misinterpreted as an absence of flow. However, PRF should not be so low that causes color pixels to appear outside the vessels. With a given PRF, only a range of velocities can be detected. When a flow velocity in a vessel is lower than the lowest value of this range, then the lumen of this vessel will remain anechoic without color signals. However, if a velocity is higher than the highest value of this range, the lumen will be colored with an inappropriate color, i.e. aliasing artifact occurs.

During an examination, color Doppler and gray scale images should alternatively be obtained by regularly switching on and off the color overlay.

Power Doppler imaging

The image is similar to that of the color Doppler mode, however only one color (orange) and its shades can be seen within the color box. *Brighter* shade indicates larger volume of flow, ie. *more blood cells* flowed at that particular region. This technique does not give any information about the direction or velocity of the flow in contrast with the color Doppler imaging technique.

The power Doppler technique has been useful to detect very tiny vessels or very slow flow. However, the newer ultrasound machines have much more sensitive color Doppler mode compared to older machines, which makes the use of the power mode unnecessary.

Doppler artifacts

Aliasing (color and pulsed-wave Doppler)

Aliasing occurs when the velocity of blood exceeds an upper measurement limit (so called Nyquist-limit). Aliasing is present, when the PRF is less than twice the frequency of the reflected Doppler shift signal.[3]

On color Doppler images, a velocity beyond the upper measurement limit (beyond the lightest shade of the proper color) is displayed with an incorrect color, i.e. with the color, which is to code the opposite direction of flow (e.g. red instead of blue). This often results in a concentric color pattern. Circumscribed color aliasing can help to identify stenotic regions, however aliasing that is present in the entire vessel should be avoided.

Aliasing in pulsed wave Doppler mode is shown in **Fig 1A.** The part of the spectrum that is beyond the upper measurement limit is cut down, and displayed wrongly on the opposite side of the baseline. The height of the transferred peak is twice as it would be on the proper side (i.e. without aliasing).

In sum, an increased, or aliased Doppler shift frequency is seen as a "folding over" of the spectral display, which shows an ambiguous relationship between velocity of the motion and the displayed Doppler shift.[20]

The possible solutions to overcome aliasing are to increase the PRF or/and to shift the baseline (**Figs 1B, 1C**), or to select another transducer with a lower frequency.

Absence of Doppler signal (color and spectral Doppler)

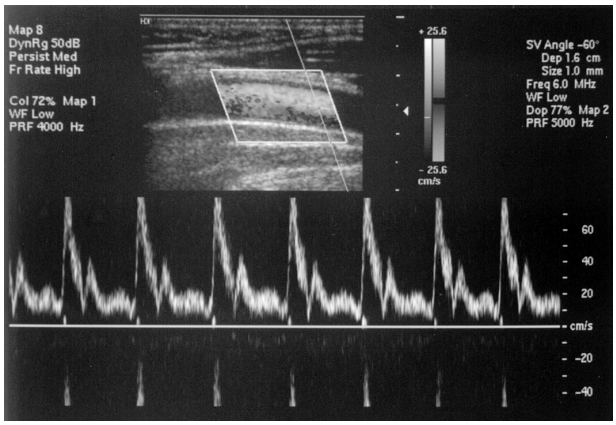
When the axis of the intersection angle of the blood vessel and the ultrasound beam is perpendicular to each other, then there is no detectable Doppler signal. The vessel should then be imaged in another position to get a more acute angle. If this is not possible, *beam steering* may be used with linear transducers. Beam steering makes both the color and the pulsed wave Doppler ultrasound beam emit in an acute angle from the transducer meanwhile the two-dimensional image remains unchanged.

When in spite of the adequate Doppler angle, there are not appropriate Doppler signs, then the PRF should be decreased or/and the Doppler gain should be increased.

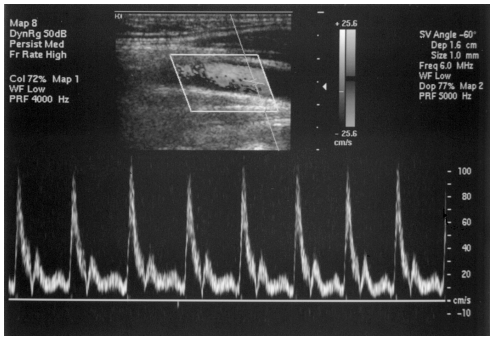
Mirroring (spectral Doppler)

Besides the normal spectrum, the same spectrum may also be seen simultaneously on the other side of the baseline with less intensity (the baseline behaves like a mirror). This artifact disappears by reducing the Doppler gain. If the background of the spectrum is not clear black but gray-spotted or gray, the Doppler gain should be reduced (**Fig 2**).

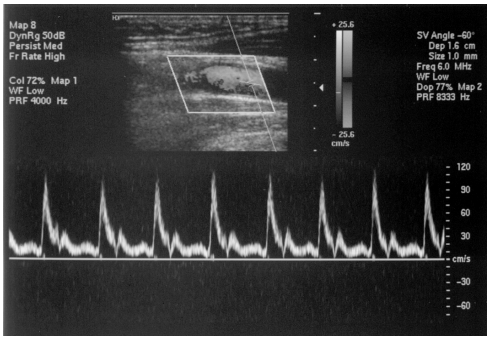
Figure 1.



A. Aliasing artifact during duplex Doppler ultrasound examination of an artery. The tops of the systolic peaks are cut and appear on the other side of the baseline.
In the left upper corner the color Doppler parameters are displayed: the colour gain (72%), the wall filter (WF low) and the pulse repetition frequency (PRF). In the right upper corner the pulsed-wave Doppler parameters are displayed: SV angle 60° (the incidence angle after angular correction), Dep 1.6 cm (the position of the sample volume, i.e. how deep it is located), Size 1.0 mm (the size of the sample volume). The pulsed-wave Doppler wall filter is also set "low". Below and above the colour bar the + and - velocity values indicate the maximum mean velocities that can be displayed into both directions using this specific color PRF-setting (4000 Hz) without an aliasing artefact. If a velocity exceeds these values, aliasing will occur. A flow that is coded with red on the color image is traditionally displayed above the baseline on the spectral Doppler image. With this pulsed-wave Doppler PRF-setting (5000 Hz), the maximum velocity that can be displayed without aliasing is 40 cm/s when the flow is away from the transducer and 70 cm/s when the flow is towards the transducer. These values can be changed by shifting the baseline and changing the PRF.
To keep the incidence angle as small as possible (i.e. the angle between the vessel's axis and the ultrasound beam) the color and pulsed-wave Doppler beams are steered.



B. Shifting the baseline downwards eliminates aliasing artifact. Compare the scales on the right side of the tracings of Figs 1 A and 1B! The pulse repetition frequency has not been changed (right upper corner: PRF 5000 Hz).



C. Increasing the PRF is another way to eliminate aliasing artifact. The PRF is 8333 Hz (right upper corner). The baseline has not been shifted in comparison with Fig 1A. Compare the scales on the right side of the tracings of Figs 1A and 1C!

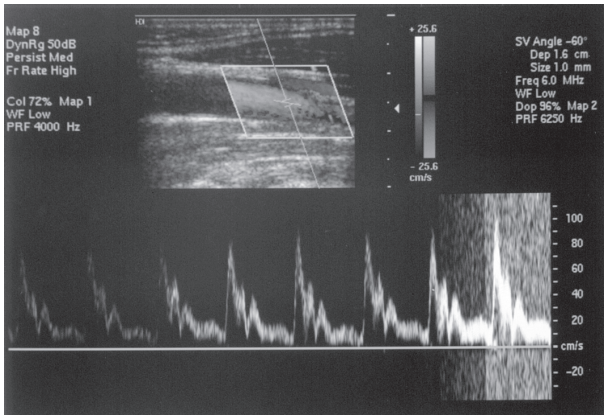


Figure 2.
In the beginning of the tracing the Doppler gain is too low (the first two and a half waves are not sharp), at the end of the tracing the Doppler gain is too high (gray-spotted background around the last one and half complexes). In the middle the Doppler gain is optimal. Note that the Doppler gain artificially influences the peak velocities.

Sonographic examination of blood vessels

Both color and pulsed-wave Doppler examinations should only be performed on a vessel that is imaged in a longitudinal section, and the incidence angle is as small as possible. During the examination of a blood vessel, the first step is to image the vessel using a real-time two-dimensional technique in the *longitudinal* plane. Ideally, the axis of the ultrasound beam and the axis of the vessel are parallel to each other, but their intersection angle should not exceed 60º. Then colour Doppler should be switched on and the PRF adjusted properly. Since smaller vessels can often be visualised only with color Doppler because of the insufficient gray scale resolution, looking for a certain vessel can be facilitated by having the color overlay on.

With the use of color mode it is possible to determine the presence or absence of flow in the vessel. The color parameters should be set so that the lumen of the vessel will be filled with only one color (i.e. without aliasing) and color information should not be seen outside the vessel lumen (because of e.g. too much color gain), but the entire vessel lumen (peripherally to the walls) should be filled with color.

After switching on the pulsed-wave Doppler mode, the sample volume is placed in a straight segment of the vessel that is uniformly colored on the color image (with either blue or red). Within this segment the sample volume is positioned so that the incidence angle will be as small as possible. The size of the sample volume has to be adjusted so that only the vessel of interest will be included in the sample volume. When the sample volume is placed in a vessel segment that is red, the Doppler spectrum will appear above the baseline (flow towards the transducer). Once the spectrum tracing appears in the screen, aliasing should be eliminated by adjusting the PRF and shifting the baseline. With other words the spectrum should vertically fit in the available space, otherwise the top of the spectrum would be cut down and placed on the other side of the baseline (aliasing). Once the Doppler tracing is free from artifacts (aliasing, moving), the image should be frozen and after angle correction, the waveform analysis can be performed.

Flow velocity profiles of arteries

The distribution of flow velocities across the vessel lumen is displayed as a frequency or velocity range in the Doppler spectrum. Undisturbed (laminar) flow velocity profiles are described as plug (narrow range of frequencies/velocities), parabolic (wide range of frequencies/velocities), or blunted parabolic (intermediate range). Turbulence can also be detected.[1] The audible Doppler signal produced by undisturbed (laminar) flow has a "whistling" quality.[23] Blood flow in most blood vessels is undisturbed (laminar) with blood moving in thin concentric layers or laminae. Central layers flow the fastest whereas frictional forces cause energy loss and slowing of layers near the vessel wall.[3]

The terms laminar and turbulent are not recommended to be used to describe blood flow, since they require steady-state flow of a newtonian fluid through a rigid tube.[24]

Types of undisturbed (laminar) flow

Plug flow velocity profile. In larger arteries (e.g. aorta), the velocity of blood is nearly the same in the center of the vessel as near the vessel wall. The great majority of blood cells are moving at a uniform speed, thus the velocity distribution is very narrow across the vessel lumen. As a result, the spectrum is characterized by a thin line in systole that outlines a clear space called the *spectral window* (or *systolic window*) (**Figs 3, 4, 6**).[23]

Parabolic flow velocity profile. In smaller arteries (e.g. renal artery) the centrally moving blood has higher velocity compared to the blood adjacent to the vessel wall, so the velocity distribution is broad across the vessel lumen. The spectral window is absent during systole on the spectrum display, because blood cells with a range of velocities (from zero to the peak velocity) flow through the sampling gate (**Figs 4, 11**).

Blunted parabolic (intermediate) flow velocity profile. In middle-sized arteries (e.g. celiac trunk), the flow is similar to the plug flow velocity profile in the center of the vessel (i.e. cells moving at a uniform speed), however the flow is more similar to the parabolic flow velocity profile in the peripheral parts of the vessel lumen. Cells with a small range of velocities are represented in peak systole (zero and slow velocities are absent). The velocity distribution across the vessel lumen is broader, than in a vessel with plug flow velocity profile, but not so broad as is in a vessel with parabolic flow velocity profile. Thus, a spectral window is present during systole but it is smaller than in the spectrum of an artery with plug flow velocity profile (**Fig 4**).

The classes mentioned above (plug, parabolic and blunted parabolic flow velocity profiles) are not strict categories as a flow velocity profile can be transitional.

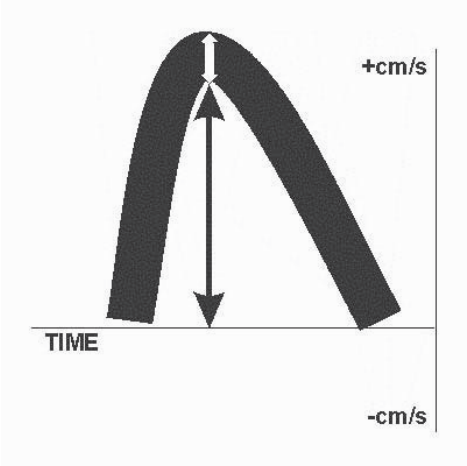


Figure 3.
The spectral window and the bandwidth in the spectral tracing. The horizontal axis is the baseline (zero flow) and the running time is indicated along it. The magnitude of the Doppler shift is represented along the vertical axis. If the Doppler correction is done on the two-dimensional image, the velocity can be read in cm/s on the vertical axis. The flow towards the transducer is displayed above the baseline, the flow away from the transducer is displayed below the baseline. On the spectrum display of an artery with a plug flow velocity profile, a curve can be seen in systole that outlines a clear space called the *spectral window* (black arrow) The thickness of the bandwidth (i.e. the velocity distribution across the vessel lumen) is indicated by the white arrow.

Disturbed (turbulent flow)

At bifurcations, curves or branches, a broad velocity distribution (i.e. spectral broadening) or even flow reversal may be present, where cells with a wide range of velocities from zero to both the negative and positive maximum velocities are represented.

Size of the sample volume

The size and position of the sample volume strongly influences the Doppler pattern. Using the uniform insonation method results in a Doppler spectrum that is different from the spectrum obtained using the maximum velocity method. This is because of the difference in the size of the sampling gate. Not only the size, but also the site of the sample volume (gate) across the vessel lumen influences the Doppler spectrum. When a small sample volume is placed in the center of an artery with a parabolic flow velocity profile according to the maximum velocity method, the spectrum will be different from that obtained with the same sized sample volume placed into the periphery of the same artery, or when a larger sample volume was used according to the uniform insonation method (**Fig 5**).

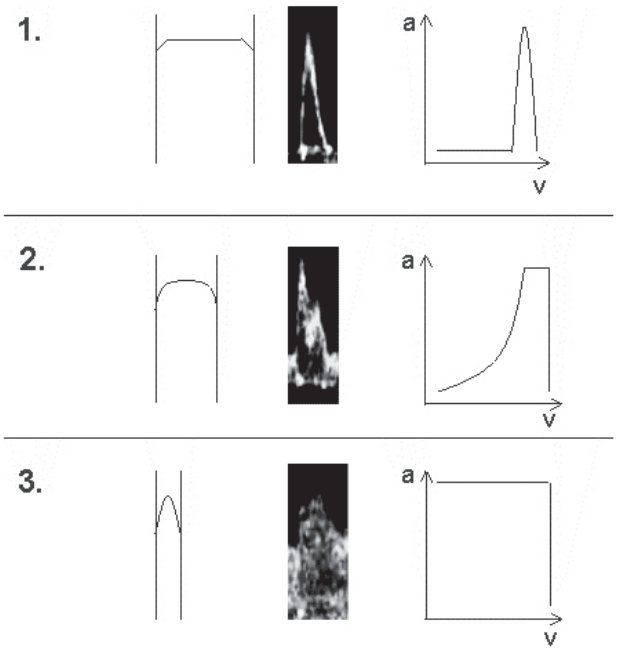


Figure 4.

The relationship between the flow velocity profiles, the Doppler spectra (systolic peaks) and the Doppler shift power spectra in arteries (v = velocity of blood flow, a = quantity of blood cells moving at a specific speed).[20] The width of the systolic peak depends *not* on the flow velocity profile.

1. Plug flow velocity profile. The great majority of blood cells are moving at a uniform speed, thus the velocity distribution is very narrow across the vessel lumen. As a result, the spectrum is characterized by a thin line in systole that outlines a clear space called the *spectral window*.

2. Blunted parabolic flow profile. The flow is similar to the plug flow velocity profile in the center of the vessel (i.e. cells moving at a uniform speed), however the flow is more similar to the parabolic flow in the peripheral parts of the vessel lumen.

3. Parabolic flow velocity profile. The centrally moving blood has higher velocity comparing to the blood adjacent to the vessel wall, so the velocity distribution is broad across the vessel lumen. Spectral window cannot be seen during systole on the spectrum display, because blood cells with a wide range of velocities (from zero to the peak velocity) flow through the sampling gate.

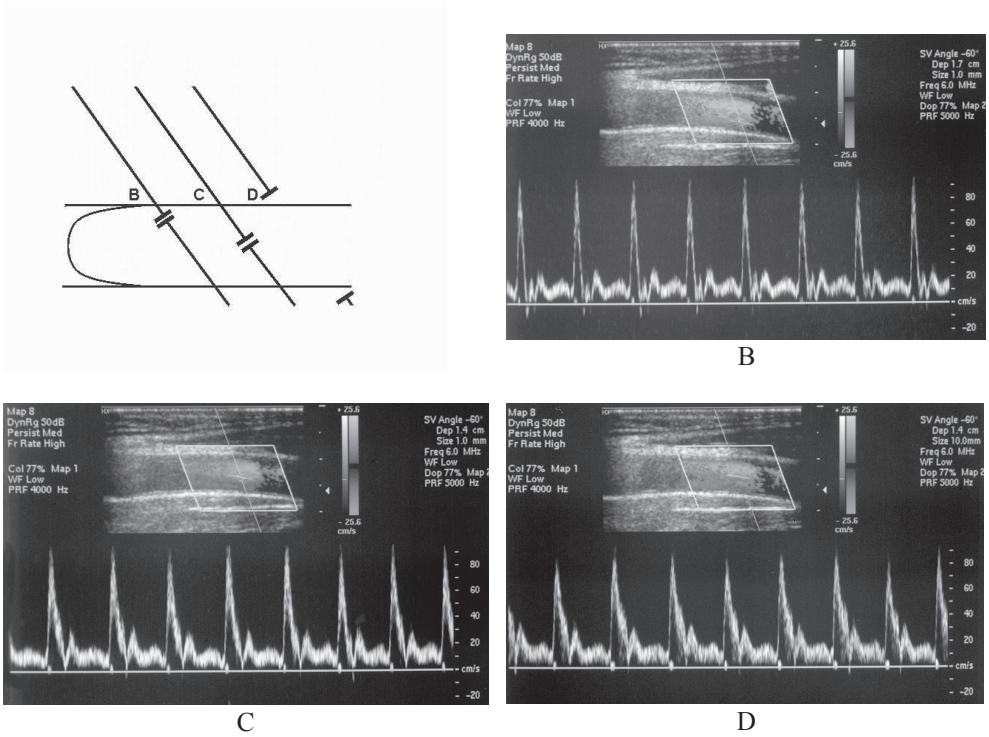


Figure 5.
The effect of sample volume size on the Doppler spectrum of an artery. A small sample volume placed in the periphery of an artery (Fig 5B) results in a different Doppler pattern from that in which the same sized sample volume was placed in the center of the vessel (Fig 5C) or a large sample volume incorporates the whole diameter of the vessel (Fig 5D).

Doppler waveforms of the blood vessels

Arterial flow

The outline of the maximum Doppler shift frequency corresponds to the time variation of the maximum flow velocity within the vessel. The pulsatility of the waveform is related to the vascular impedance downstream to the point of measurement.[1] The perfusion requirements of each organ determine the state of its vascular bed, which in turn affects the flow characteristics of its associated vessels.[1]

Each cardiac contraction causes forward blood flow, and results in distension of arteries. The diastolic reverse flow is due to blood actually rebounding up the aorta as the velocity wave is reflected from the high impedance of the peripheral vascular bed of the hind limbs.[1] As vessel diameters return to normal, the rebound energy provides necessary energy to promote continuous flow in diastole.[3] Blood flow entering arteries feeding parenchymal organs (e.g. kidneys, liver, spleen) that constantly require perfusion normally have continuous and gradually decreasing forward flow in diastole without diastolic flow reversal.

High resistance flow pattern. High pulsatility and high resistance to flow are indicated by sharp systolic peaks and flow reversal in early diastole (e.g. aorta).

We observed in cases of marked sinus arrhythmia when the next heart beat is delayed (i.e. the diastole is long), there are not only two diastolic waves (i.e. the systolic peak is followed by a reverse flow wave, which is followed by a forward flow wave), but continuous oscillations can be seen: the first forward diastolic flow wave is followed by another reverse flow wave, then a forward wave comes, etc., until the next systole. The amplitude of the forthcoming diastolic waves are gradually smaller, i.e. the first diastolic reverse flow wave is the highest among the diastolic reverse flow waves of a particular systole-diastole complex and the first diastolic forward flow wave is the highest among the diastolic forward flow waves (**Fig 6**).

Low resistance flow pattern. Low pulsatility and low resistance to flow are indicated by broad systolic peaks and continuous, high velocity flow in diastole with gradually decreasing velocity. The low resistance arteries (e.g. renal artery) supply organs that have continuous blood demand. This demand, together with the Doppler flow pattern of the vessel, may change according to the functional state of the organ (e.g. pre- and postprandial state of the intestines).[3]

Intermediate resistance flow pattern. Intermediate pulsatility and intermediate resistance to flow are indicated by sharp systolic peaks (broader, than in arteries of high resistance flow pattern), and forward flow in diastole without reverse flow (e.g. celiac trunk). The diastolic peak velocity compared to the peak systolic velocity is lower than in the low resistance flow pattern.

Venous flow

Usually, the flow in the veins is undisturbed (laminar).[24] The intrathoracic and intraabdominal pressure conditions, during inspiration and expiration, influence blood flow velocities in the veins making phasic changes. Most veins have mildly periodic flow except for the hepatic veins and the cranial part of the abdominal caudal vena cava, whose Doppler patterns are markedly periodic because of the strong effect of the right atrial pressure changes during the cardiac cycle.

The following terms are used to describe the spectral changes of flow. *Pulsatility* refers to arterial flow, *phasicity* refers to changes in flow secondary to respiration, and *periodicity* refers to variation in the hepatic veins secondary to cardiac activity.[25]

When the direction of flow in the portal vein is towards the liver it is called hepatopetal, and the reversed flow is called hepatofugal flow.[25]

Early systolic peak (ESP)

Generally, in an arterial Doppler tracing one systolic peak can be seen during each systole. In some dogs, however, there are double systolic peaks in given arteries. Based on in vitro studies it has been suggested that the ESP represents a transmitted pulse and that the second broader systolic peak that is seen in "low resistance arteries" is a compliance peak.[26] The presence as well as the absence of an ESP is normal and is likely related to the degree of vascular compliance.[26]

We found that when a double systolic peak is present in the Doppler spectrum of the celiac trunk, cranial and caudal mesenteric arteries or deep circumflex iliac artery, the sharp peak becomes notched, and the first peak dominates over the second, smaller compliance peak. In the splenic and renal arteries the second peak (i.e. the compliance peak) is rounded and more dominant.

Doppler spectra and waveforms of the major abdominal blood vessels

Abdominal aorta

In our experience, the best image of the caudal abdominal aorta can be obtained with the animal in right lateral recumbency. The transducer is placed on the caudodorsal part of the abdomen, where the pulsating aorta can be seen longitudinally just ventral to the vertebrae, to the left and parallel to the adjacent caudal vena cava. The cranial part of the abdominal aorta (cranial to the left phrenicoabdominal artery) is not easy to image, especially in deep chested dogs because of the artifacts caused by the gastrointestinal gas and ribs. Sometimes, when there is not much gastrointestinal gas it may be easier to get a better sagittal image from the cranial part of the abdominal aorta in dorsal recumbency, medial to the right kidney. The aorta has a typical plug flow velocity profile and its waveform is a typical high resistance flow pattern. It has a sharp systolic peak with large and clear spectral window. The velocity distribution is narrow. The systolic peak is followed by a retrograde flow wave, then a forward flow wave can be seen (**Fig 6**). If there is a longer pause between two ventricular contractions, additional waves with lower velocities occur (see also *High resistance flow pattern)*.

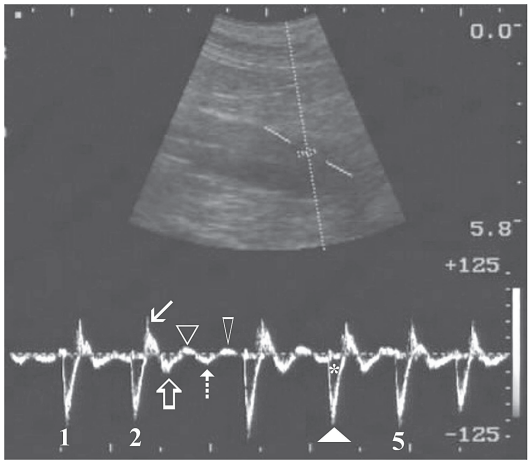


Figure 6.
Spectral Doppler pattern of the abdominal *aorta* (PRF=9014, Θ=51°). Waves under the baseline represent flow away from the transducer. The systolic peaks (arrowhead) are sharp. The narrow velocity distribution with large and clear spectral window (*) represents plug flow velocity profile. The systolic peak is followed by a retrograde flow wave (arrow) in early diastole (high resistance flow pattern). The retrograde flow wave is followed by a forward flow wave (empty arrow), then the flow becomes retrograde again (empty rectangular arrowhead), then the flow gets forward again (dotted arrow), then retrograde again (narrow empty arrowhead). The velocity of the second diastolic forward flow wave and the second and third retrograde flow waves is always gradually smaller, than that of the first ones. The systolic peak of the second complex (2) is followed by a longer pause (diastole), meanwhile three retrograde and two forward diastolic flow waves occurred. The fifth complex (5) has only one retrograde and one forward diastolic flow wave, because the next systolic peak occurs earlier, than in the case of the second complex.

Internal iliac arteries

The right and left internal iliac arteries are the last branches of the aorta. They originate symmetrically from the lateral surface of the aorta, forming a narrow angle with it. Lateral recumbency can be suggested to image them. Caudal from this point, the aorta becomes the sacral median artery. Blunted parabolic flow profile and a high resistance flow pattern can be detected. Sharp systolic peaks with a very small spectral window can be seen. There is a small amount of retrograde early diastolic flow, then the flow is forward in the rest of the diastole (**Fig 7**).

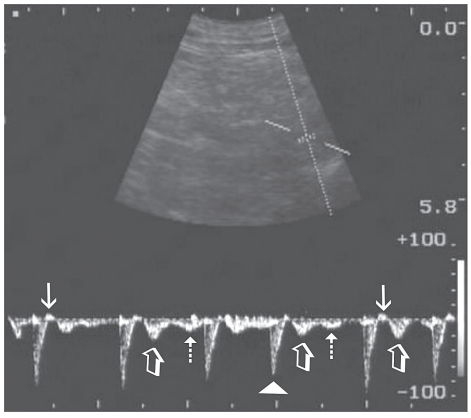


Figure 7.
Spectral Doppler pattern of the right *internal iliac artery* (PRF=7022, Θ=51°). Flow is away from the transducer (below baseline). The systolic peaks (arrowhead) are sharp with a small spectral window (blunted parabolic flow velocity profile). The systolic peak is followed by a small retrograde wave (arrow) in early diastole (high resistance flow pattern). The retrograde flow wave is followed by a forward flow wave (empty arrow), then the flow velocity becomes lower, then it gets higher again (dotted arrow). The second diastolic forward flow wave is always smaller, than the first one.

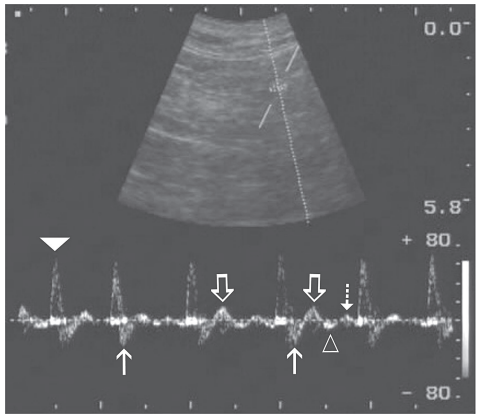


Figure 8.
Spectral Doppler pattern of the left *external iliac artery* (PRF= 7022, Θ=39°). Flow is towards the transducer (above baseline). The systolic peaks (arrowhead) are sharp. Its spectral window is large and clear, the velocity distribution is narrow (plug flow velocity profile). The systolic peak is followed by a small retrograde wave (arrow) in early diastole (high resistance flow pattern). The retrograde flow wave is followed by a forward flow wave (empty arrow), then the flow velocity becomes retrograde again (empty arrowhead), then the velocity gets forward again (dotted arrow). The second diastolic forward flow waves and the second retrograde flow waves are always smaller, than the first ones.

External iliac arteries

The right and left external iliac arteries originate slightly cranial to the origin of the internal iliac arteries, symmetrically from the lateral surface of the aorta, forming about an angle of 45º with it. Its waveform and flow profile are similar to that of the aorta, i.e. plug flow velocity profile (clear spectral window and narrow velocity distribution) and high resistance flow pattern (**Fig 8**). We found that it was often hard to maintain the sample volume in the lumen of the artery because each pulse wave produced extremely large abaxial movement of the vessel.

Deep circumflex iliac arteries and veins

The thin, symmetric, paired arteries originate laterally, almost perpendicularly from the aorta, slightly cranial to the origin of the external iliac arteries. The corresponding veins flow into the caudal vena cava. The ipsilateral artery and vein course parallel close to each other. Lateral recumbency is preferred to image these vessels. These arteries have a blunted parabolic flow velocity profile (small, sometimes hardly detectable spectral window) and a high resistance flow pattern (sharp systolic peak and small amount of reverse flow in early diastole). Double systolic peaks can be present in some dogs (**Fig 9**).

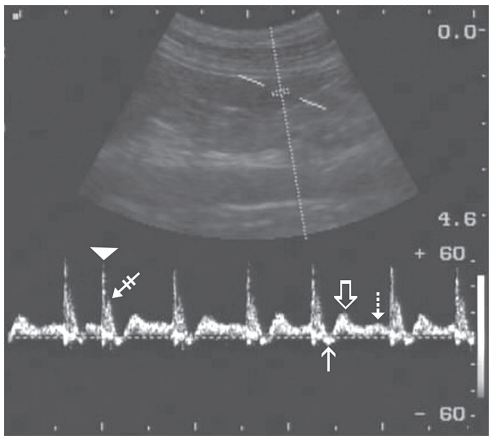


Figure 9.
Spectral Doppler pattern of the left *deep circumflex iliac artery* (PRF=3511, Θ=60°). Flow is towards the transducer (above baseline). The systolic peaks are sharp (arrowhead) with very small spectral window (blunted parabolic flow velocity profile). Note the double systolic peaks (arrowhead: first systolic peak, crossed arrow: the second systolic peak). The double systolic peak is followed by a very small retrograde wave (arrow) in early diastole (high resistance flow pattern). The retrograde flow wave is followed by a forward flow wave (empty arrow), then the flow velocity becomes lower, then it gets higher again (dotted arrow). The second diastolic forward flow wave is always smaller, than the first one.

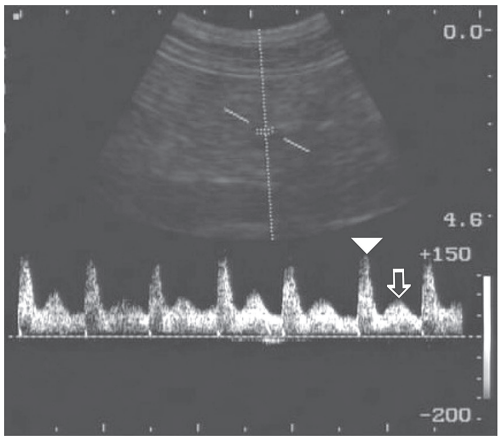


Figure 10.
Spectral Doppler pattern of the *caudal mesenteric artery* (PRF=11029, Θ=60°). Flow is towards the transducer (above baseline). Continuous forward flow can be seen during the whole cardiac cycle. The systolic peaks are broader (arrowhead) with a very small, not clear spectral window (blunted parabolic flow velocity profile). The systolic peak is not followed by retrograde wave (intermediate resistance flow pattern). In mid-diastole a forward flow wave with higher velocity (empty arrow) can be seen. Then the flow velocity becomes lower again.

Caudal mesenteric artery

It is a thin, unpaired vessel, originating from the ventral surface of the aorta, slightly cranial to the origin of the deep circumflex iliac arteries. The vessel is difficult to visualize without color Doppler mode, and artifacts made by the intestinal gas and ingesta may further hinder visualization. Blunted parabolic flow profile with broader systolic peaks and very small spectral window as well as intermediate resistance flow pattern can be seen. During diastole, the flow is continuous without retrograde flow. After the systolic peak, the velocity drops but remains forward, then it becomes faster in mid-diastole, then drops again in late diastole. ESP may be present in some dogs (**Fig 10**).

Lumbar arteries and veins

Multiple pairs of lumbar vessels can be seen between the renal and the iliac vessels, coursing dorsolaterally.[2] The thin arteries originate from the aorta; the adjacent veins enter the caudal vena cava. Blunted parabolic flow profile with sharp systolic peaks and small spectral window as well as high resistance flow pattern can be recorded. A small amount of early diastolic reverse flow is generally seen. Double systolic peaks may be present in some dogs.

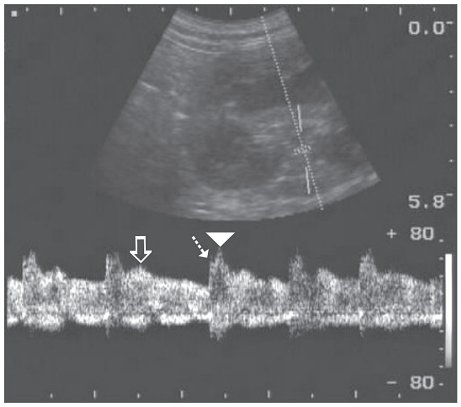


Figure 11.
Spectral Doppler pattern of the left main *renal artery* (PRF=9014, Θ=12°). Flow is towards the probe (i.e. above the baseline). The systolic peaks (arrowhead) are broad with ESP (dotted arrow). The velocity distribution is broad (typical parabolic flow velocity profile), that is why the spectral window is absent. High velocity continuous diastolic flow without reverse flow can be detected (typical low resistance flow pattern). The velocity suddenly decreases after the systolic peak, then after a wave of high velocity (empty arrow) gradually decreases again.

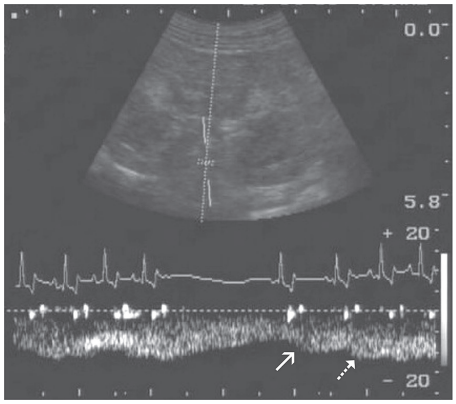


Figure 12.
Spectral Doppler pattern of the left main *renal vein* with simultaneous electrocardiogram (PRF=2003, Θ=9°). Flow is away from the transducer (under the baseline). Each ventricular contraction is followed by an increased flow wave (arrow). The forthcoming wave superimposes on the previous one increasing the velocity further (dotted arrow), if the two ventricular contractions are close enough to each other. If there is no heart beat (in the middle of the tracing), the velocity in the renal vein gradually decreases.

Renal arteries and veins

The renal artery and vein can be imaged from the hilus of the kidneys until their origin from the aorta and to the caudal vena cava, respectively. Interlobar branches can be seen around the central echocomplex, as they radiate from the pelvis toward the corticomedullary junction. Interlobar arteries branch into arcuate arteries, coursing in the corticomedullary junction. The interlobular arteries originating from the arcuate arteries. They become visible in the cortex using color Doppler ultrasound technique. The veins course parallel to the arteries and they are generally wider, than the adjacent arteries. The renal arteries have typical parabolic flow velocity profile (i.e. systolic peaks with broad velocity distribution and without spectral window). The systolic peak is always broad and ESP is sometimes visible. Low resistance flow can be detected with high, continuous diastolic flow, which gradually decreases during diastole. After the systolic peak, the

velocity drops a little, then gets higher again (diastolic peak velocity), and in the rest of diastole gradually decreases. (**Fig 11**). The flow in the renal veins changes slightly due to the right atrial and intraabdominal pressure changes. We realized that each heartbeat is followed by an increased forward flow wave. If the ventricular contractions are close to each other enough, the forthcoming wave on the Doppler tracing is superimposed on the previous wave, producing faster flow. If there is a longer pause between two ventricular contractions, the velocity gradually decreases in the renal veins (**Fig 12**).

Phrenicoabdominal arteries and veins

They are thin, paired vessels, originating from the aorta and flowing into the caudal vena cava, respectively. They can be visualized only with color Doppler ultrasound as the ipsilateral artery and vein course dorsal and ventral to the corresponding adrenal gland. These vessels divide the adrenals into a cranial and a caudal part. The flow pattern is similar to the Doppler pattern of the lumbar artery. It is very hard to hold the sample volume in the lumen of the tiny vessel; moreover, to get a proper Doppler angle is rarely possible.

Cranial mesenteric artery

This unpaired vessel is the second branch of the abdominal aorta. Its origin is slightly cranial to the origin of the right renal artery and just a little caudal to the origin of the celiac trunk. If the animal is in dorsal recumbency, the celiac trunk and the cranial mesenteric artery form a V-shape in sagittal scan plane (**Fig 13**). When the dog is in right lateral recumbency, the celiac trunk and the cranial mesenteric artery are parallel vessels (**Fig 14**). Blunted parabolic flow velocity profile and intermediate resistance flow pattern can be seen. The systolic peak is broader with small spectral window. There is no reverse diastolic flow. After the systolic peak, the velocity drops, then the flow becomes faster, then slower again (**Fig 16**). The blood has higher resistance to flow during fasting, and lower resistance postprandially.[3]

Celiac trunk

This is the first unpaired branch of the abdominal aorta. After a short trunk, it branches into three arteries (so called tripus Halleri): the common hepatic, splenic and left gastric arteries. In our experience, the nicest image can be achieved in dorsal or right lateral recumbencies, medial to the right kidney, although gas often precludes imaging (**Fig 15**). Its imaging is more difficult in deep chested breeds. Blunted parabolic flow velocity profile and intermediate resistance flow pattern can be detected. Broader systolic peaks with quite clear, middle-sized spectral window can be seen. Reverse diastolic flow cannot be detected. The velocity during diastole is alternating similarly to the pattern of other arteries with intermediate resistance flow pattern. In some dogs double systolic peak can be present (**Fig 17**).

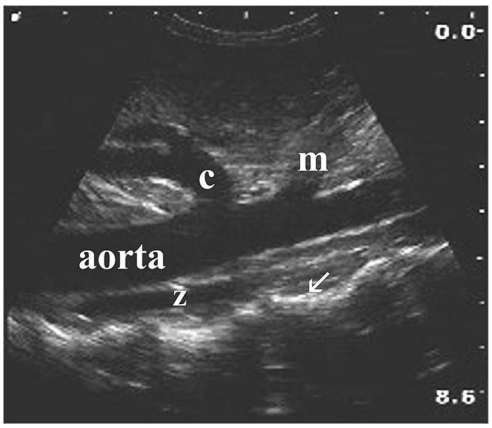


Figure 13.
B-mode sagittal image of the aorta, celiac trunk (c), cranial mesenteric artery (m) and the right azygos vein (z). The dog is in dorsal recumbency. The azygos vein courses between the aorta and the vertebrae (arrow).

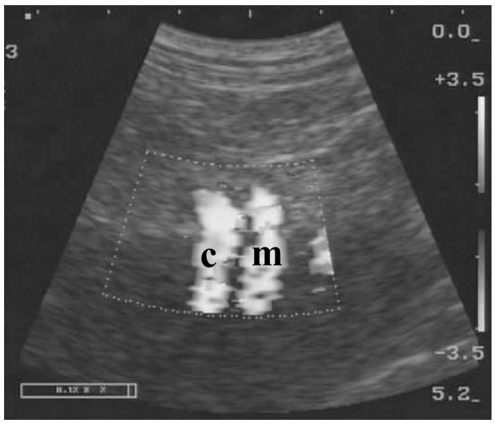


Figure 14.
Color Doppler image of the celiac trunk (c) and the cranial mesenteric artery (m). The dog is in right lateral recumbency, sagittal scan plane. Flow is towards the probe in both vessels. The pulse repetition frequency is too low because both arteries are orange instead of red. Aliasing artifact can be appreciated as light blue areas. (Full **color** illustration on page 177.)

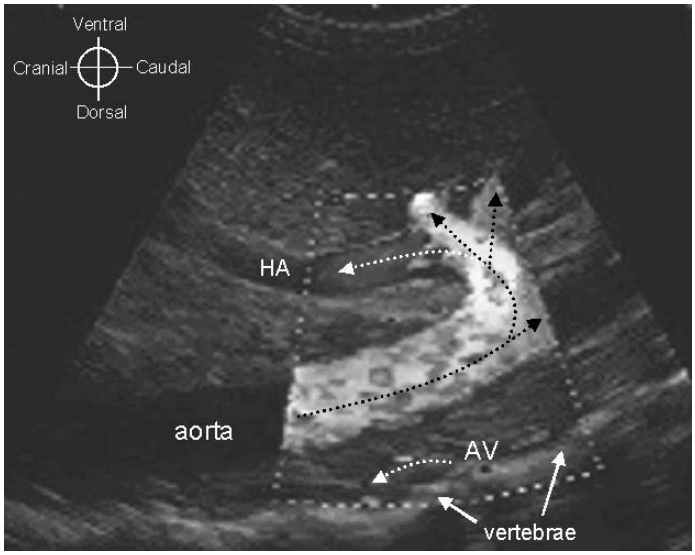
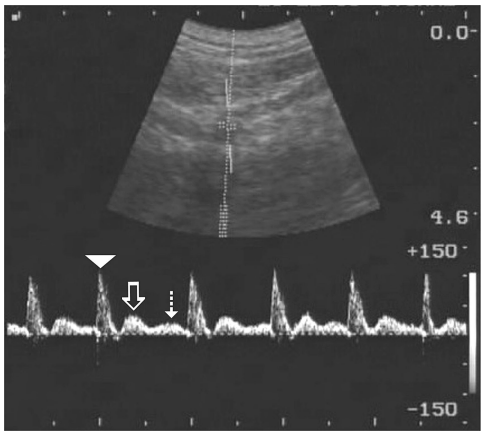


Figure 15.
Color Doppler image of the *celiac trunk* and its trifurcation (tripus Halleri). The dog is in dorsal recumbency, sagittal scan plain. The azygos vein (AV) can be seen between the aorta and the vertebrae. The hepatic artery (HA) courses cranially, whereas the other two branches of the celiac artery (the splenic and the left gastric arteries) ventrally. Arrows indicate the direction of blood flow. (**Color** version on page 177.)



Figures 16.
Spectral Doppler pattern of the *cranial mesenteric artery* (PRF=15000, Θ=5°). Flow is towards the transducer (above baseline). Forward flow can be seen during the whole cardiac cycle. The systolic peaks are broader (arrowhead) with a very small, not clear spectral window (blunted parabolic flow velocity profile). The systolic peak is not followed by retrograde wave (intermediate resistance flow pattern). In mid-diastole a forward flow wave with higher velocity (empty arrow) can be seen. Then the flow velocity becomes lower (between the arrows), then higher (dotted arrow) again.

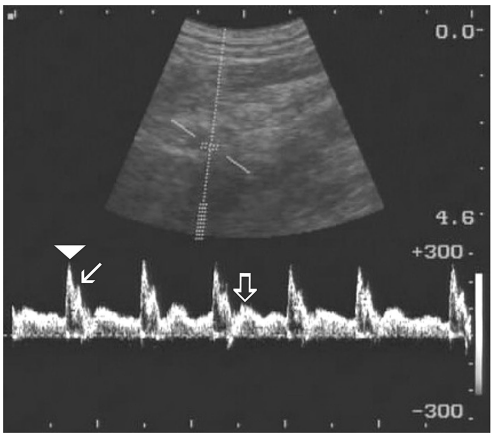


Figure 17.
Spectral Doppler pattern of the *celiac trunk* (PRF=15000, Θ=63°). Forward flow can be seen during the whole cardiac cycle. Flow is towards the transducer (above baseline). The systolic peaks are broader (arrowhead) with a quite clear spectral window (blunted parabolic flow velocity profile). Note the double systolic peaks. The first peak (arrowhead) is higher than the second one (arrow). The systolic peak is not followed by retrograde flow wave (intermediate resistance flow pattern). In mid-diastole a forward flow wave with higher velocity (empty arrow) can be seen, then the flow velocity becomes lower again.

Hepatic artery

Sometimes, the common hepatic artery can be imaged only with color Doppler ultrasound. It originates from the celiac trunk, and courses cranially parallel to and between the caudal vena cava and the main portal vein. The intrahepatic branches of the hepatic artery generally cannot be found in healthy dogs, because of their small size, unless the color mode of the ultrasound machine is very sensitive. Its Doppler flow pattern is similar to the spectrum of the renal artery (i.e. parabolic flow velocity profile and low resistance flow pattern). Broad systolic peaks without spectral window (i.e. broad velocity distribution) and continuous, gradually decreasing high diastolic flow can be detected. In some dogs the spectrum is more similar to that of the celiac trunk (**Fig 18**).

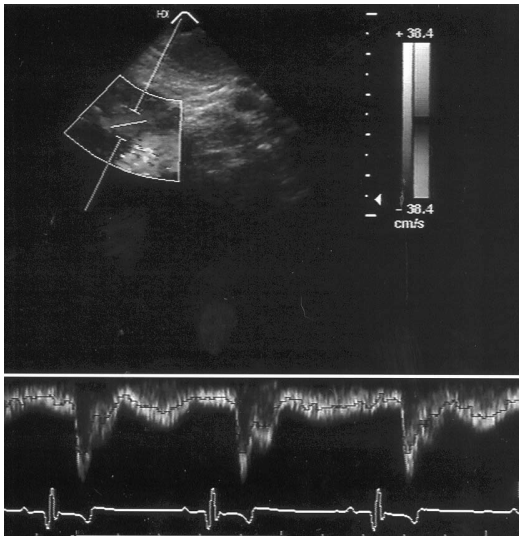


Figure 18.
The common *hepatic artery* courses between the portal vein and the caudal vena cava. The Doppler spectrum is similar to that of the celiac trunk.

Splenic artery and vein

Parenchymal splenic veins can be seen by B-mode echography as they break through the capsule of the spleen, but the thinner arteries can only be found with color Doppler technique, adjacent to the veins. The main splenic artery originates from the celiac trunk and terminates by branching in the splenic hilum, but because of its tortuous course and the gastrointestinal gas, its imaging from the celiac trunk until the spleen is rarely possible. The main splenic vein and artery can be seen in the hilus of the spleen caudal to the stomach. The splenic vein flows into the portal vein. A typical low resistance waveform (i.e. broad systolic peaks and continuous high diastolic flow) as well as parabolic flow velocity profile (i.e. systolic peaks without spectral window) can be found, similarly to the Doppler pattern of the renal or hepatic arteries. ESP is present in some animals (**Figs 19, 20**). In the splenic veins, the velocity changes are due to the respiratory pressure changes, similarly to the Doppler pattern of the portal vein (**Fig 20**).

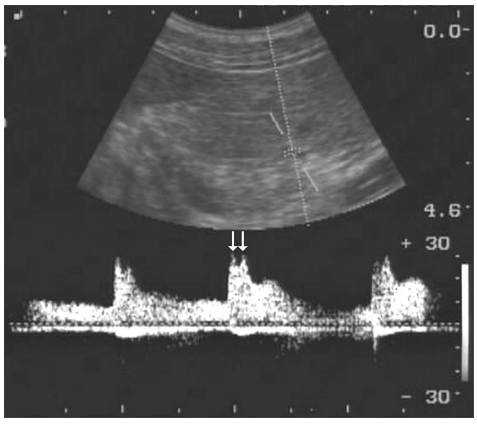
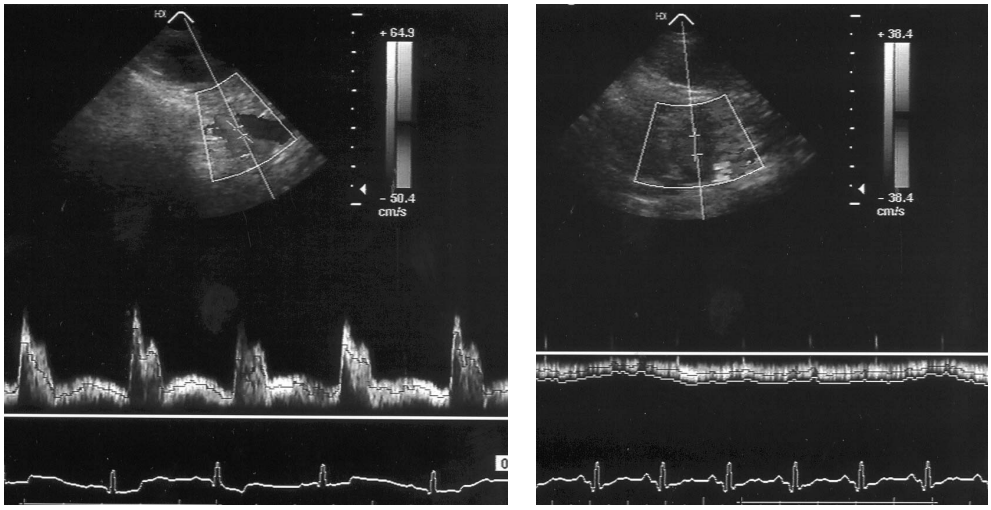


Figure 19.
Spectral Doppler pattern of a *parenchymal splenic artery* (PRF=3511, Θ=20°). Flow is towards the transducer (above baseline). The systolic peaks (arrow) are broad without spectral window (parabolic flow velocity profile). Early systolic peaks are present (arrow). The systolic peak is not followed by retrograde flow wave. Continuous forward flow can be seen during the whole cardiac cycle (low resistance flow pattern). In mid-diastole a forward flow wave with higher velocity can be seen. Then the flow velocity becomes gradually lower again.



A B

Figure 20.
The main splenic vein and artery visualized via the left abdominal wall.

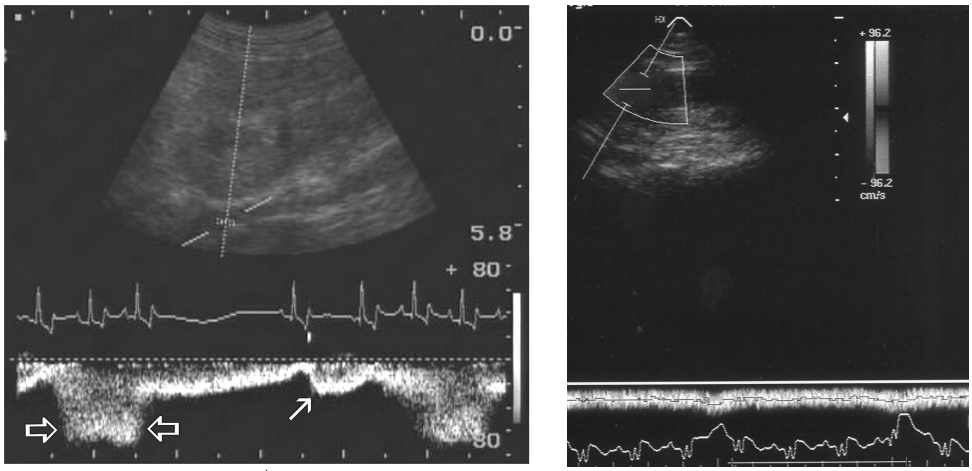
A. The main *splenic artery* can be found between the stomach and the cranial extremity of the spleen. The Doppler spectrum is similar to that of the celiac artery.

B. The main *splenic vein* can be found adjacent to the main splenic artery. The Doppler spectrum is identical to that of the portal vein.

Abdominal caudal vena cava

The caudal part of the abdominal caudal vena cava is adjacent to the abdominal aorta. They have about the same diameter, however the aorta is usually thinner. Cranial to the entering of the renal veins, the caudal vena cava deviates in a cranioventral direction and enters the thoracic cavity through the diaphragm. The cranial part of the caudal vena cava is easier to be imaged in dorsal or left lateral recumbency, medial to the right kidney. The caudal vena cava is always a straight vessel in contrast with the main portal vein, whose latter course is curving. The caudal vena cava and the main portal vein are roughly parallel to each other. The main portal vein is ventral and slightly to the left to the caudal vena cava. Within the cranial part of the caudal vena cava marked perodic and phasic changes can be seen in the spectrum in contrast to its caudal part, corresponding to the pressure conditions of the right atrium as well as of the intrathoracic and intraabdominal pressure changes due to the respiratory movements.

We observed that each ventricular contraction was followed by a forward flow wave with slightly increased velocity. In addition, every inspiration caused a forward flow wave with high velocity (**Fig 21**).



A B

Figure 21.

Spectral Doppler pattern of the *caudal vena cava* with simultaneous electrocardiogram. Flow is away from the probe (under the baseline).

A. Cranial to the left kidney (PRF= 4507, Θ=56°). Each heart beat causes an increase in the velocity of the blood flow in the caudal vena cava (arrow). In addition each inspiration results in a forward flow wave with high velocity (inspiration is between the empty arrows). If there is no ventricular contraction (between the right empty arrow and the arrow), the velocity gradually decreases.

B. Caudal to the left kidney the effect of the respiratory and right atrial pressure changes is damped.

Common iliac veins

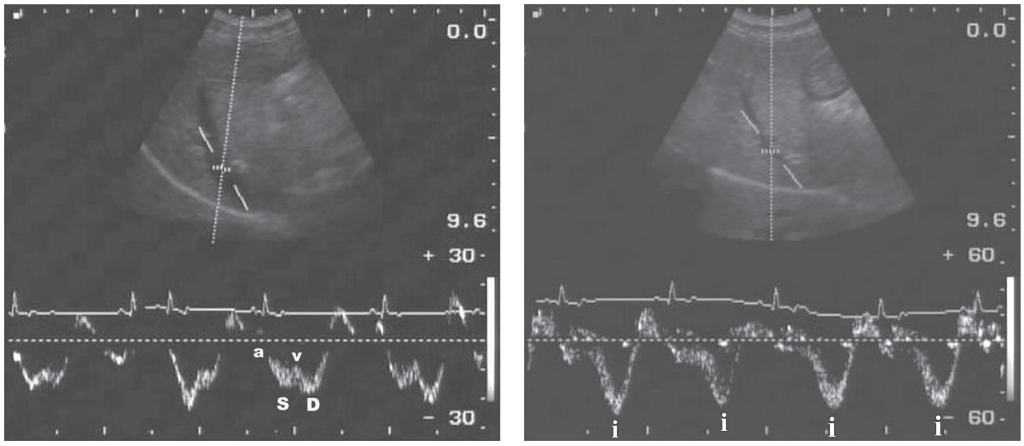
At the level of the final bifurcation of the aorta, the confluence of the left and right common iliac veins forms the caudal vena cava. These veins are almost parallel to the internal iliac arteries. The Doppler waveform is continuous forward flow without significant alterations.

Hepatic veins

Hepatic veins and their convergence into the caudal vena cava can be imaged in the liver. The best image for Doppler studies can be reached in left lateral recumbency with a transducer placed intercostally, in a sagittal plane. The number and anatomy of the hepatic veins is written elsewhere.[25,27,28] The Doppler pattern of the hepatic veins is strongly periodic, and the waves correspond to right atrial pressure.[29] The following nomenclature of the waves is based on a human study.[25,29] The first, large antegrade wave (S-wave) is caused by the movement of the tricuspid annulus toward the cardiac apex. The second component of the Doppler pattern of the hepatic veins is a small retrograde wave (v-wave) caused by right atrial overfilling. The third component is the second antegrade wave (D-wave) caused by the opening of the tricuspid valves and flow of blood from the right atrium to the right ventricle. This second antegrade wave is generally smaller, than the first one. The fourth component is the second retrograde wave (a-wave) caused by the right atrial contraction. Sometimes a small retrograde fifth wave (c-wave) can be seen as the

result of the closure of the tricuspid valves. Inspiration caused a slight increase in D-wave, and a slight decrease in S-wave in this human study.

The same waves can also be identified in dogs (**Fig 22A**). In our experience, the intrathoracic and intraabdominal pressure changes due to the respiratory movements influence the Doppler waveform of the hepatic veins more prominently in the dog than that in human beings. The effect of the respiratory movements can be so strong that it conceals the effect of the right atrial pressure changes. Generally, during panting or deep inspiration it is impossible to hold the sample volume within the lumen of the vessel. We found, that inspiration caused increased flow velocity in forward direction (**Fig 22B**).



A B

Figure 22.
Spectral Doppler pattern of a *hepatic vein* with simultaneous electrocardiogram.

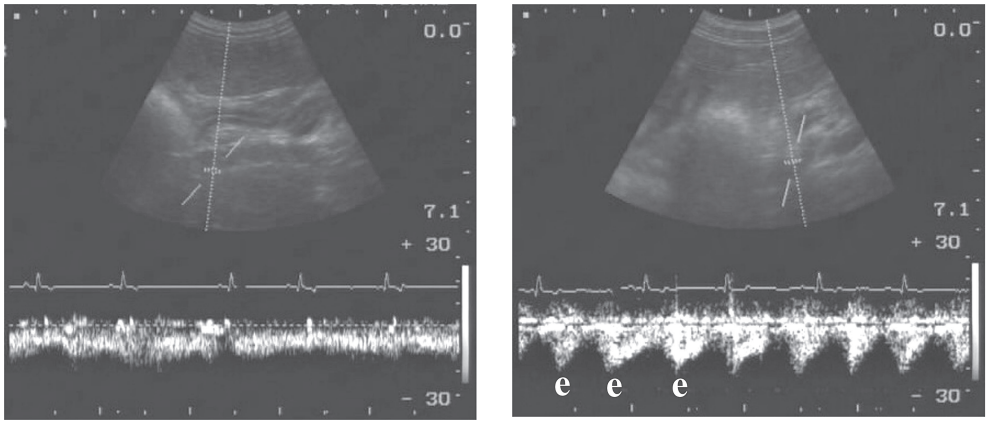
A. When the dog is breathing quietly. (PRF=2500, Θ=38°). S-wave and D-wave are forward flow waves (away from the transducer, i.e. under the baseline). The v-wave is also a forward flow wave (under the baseline), but its velocity is lower. The a-wave is a retrograde flow wave (above the baseline) following the P-wave of the ECG. See the text for details.

B. During deep inspirations (PRF=5514, Θ=38°). Each inspiration results in a marked forward (away from the transducer, i.e. below the baseline) flow wave (i). The effect of the respiratory movements on the flow velocity in the hepatic vein is stronger, than the effect of the right atrial pressure changes.

Portal vein

In our experience, the main portal vein can be the best imaged in dorsal recumbency in an oblique sagittal plane, when the cranial part of the transducer is on the linea alba and turned slightly to the right or via the right flank with the dog in left lateral recumbency. This vein is never straight like the caudal vena cava. The main portal vein turns dorsal in front of its bifurcation at the porta hepatis. The intrahepatic portal vein branches have echogenic wall, which makes their identification easier from the hepatic veins, which latter ones do not have echogenic walls. The wall of the hepatic veins is hyperechoic only in instances when the incident ultrasound beam and the vessel are perpendicular. According to a study in humans,[30] the explanation for this phenomenon is that the wall of the hepatic veins is composed of parallel tightly packed collagen fibers. In

contrast, portal vein walls are composed of loosely arrayed, non-parallel connective tissue fibers, which are separated by multiple intervening spaces and only a minority of which are collagenous. Perivascular fat was not identified adjacent to intrahepatic vessels beyond the liver hilus. Portal vein wall fibers are oriented in multiple directions, so that the ultrasonographic beam can be returned to the transducer at a wide range of incident angles. In addition, fibers in portal vein walls are loosely arrayed, so that multiple interfaces are present to reflect the ultrasound beam in contrast with the hepatic vein wall. Normally, the flow is hepatopetal (i.e. towards the liver). The flow is slightly phasic, with variable velocities during expiration and inspiration (**Fig 23**). According to a study, this alteration can be an artifact, because the sample volume passes from the center of the lumen to the edge and back again with movement of the vessel during respiration.[31]



A B

Figure 23.

Spectral Doppler pattern of the *portal vein* with simultaneous electrocardiogram. The flow is away from the transducer (below the baseline).

A. The dog was in rest (PRF=3004, Θ=37°). Continuous hepatopetal flow (towards the liver) can be seen.

B. The dog was panting (PRF=3511, Θ=25°). Each expiration (e) caused an increased forward velocity wave.

Cranial and caudal mesenteric veins

The cranial mesenteric vein can be imaged in lateral recumbency in the middle of the abdomen in sagittal plane parallel to the vertebral column, adjacent to the cranial mesenteric artery, when there is not much gastrointestinal gas.[3] In dorsal recumbency, following the main portal vein into caudal direction is also a good option to image them. The convergence of the cranial and caudal mesenteric veins forms the main portal vein. The Doppler waveform is similar to the Doppler pattern of the portal vein, with slight changes according to the respiratory movements (**Fig 24**).

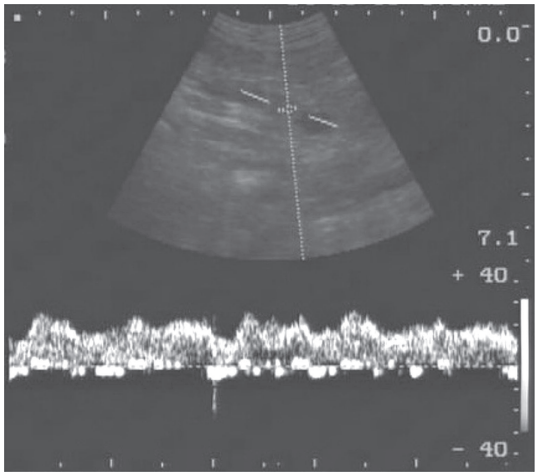


Figure 24.
Spectral Doppler pattern of the *cranial mesenteric vein* (PRF=2003, Θ=64°). Continuous flow is towards the probe (above baseline), similar to the spectrum of the portal vein. The slight alterations in the velocity are due to the respiratory movements.

Other abdominal vessels

The ovarian, uterine, or testicular and prostatic arteries and veins, the vessels of the stomach, pancreas, urinary bladder and adrenal glands can not be regularly found, mainly because of their small size as well as the artifacts caused by the gastrointestinal gas and ingesta. The detection of the tiny vessels can be improved by the use of high quality ultrasound machines with high frequency transducers and with color Doppler mode. The right azygos vein cannot be regularly seen either. It may be imaged in dorsal recumbency medial to the right kidney, between the vertebral column and the aorta coursing parallel with them. The blood flows towards the heart in it (**Figs 13, 15**).

Acknowledgement

The first author is grateful to Csilla Becskei, DVM for her continuous help and patience during the ultrasound examinations. Zsolt Abonyi-Tóth and Aart van der Woude helped in preparing the illustrations.

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CHAPTER 4

Ultrasonographic differentiation of hyperammonemic conditions in dogs

based on the article by

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Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002)

Journal of the American Veterinary Medical Association
2004;224:717-727.

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Summary

Abdominal ultrasound examinations were performed in 90 dogs with hyperammonemia. Hyperammonemia was the result of urea cycle enzyme deficiency, or portosystemic shunting either via congenital shunts or acquired collaterals. Six basic types of congenital portosystemic shunts were found. Intrahepatic shunts originated from the left or from the right portal branch and entered the caudal vena cava, whereas extrahepatic shunts originated either from the splenic vein or from the right gastric or both (double shunts) and terminated in the abdominal caudal vena cava or entered the thorax. Acquired portosystemic collaterals were the result of arterioportal fistulae, primary hypoplasia of the portal vein, or chronic hepatitis. Arterioportal fistulae were diagnosed ultrasonographically, and primary hypoplasia of the portal vein and chronic hepatitis by histopathologic examination of liver biopsy specimen. Nine hyperammonemic pups with normal abdominal vascular anatomy were suspected to have urea cycle enzyme deficiency.

Structured abstract

Objectives—To describe the ultrasonographic findings of the various conditions that lead to hyperammonemia in dogs, and the criteria how these conditions can be ultrasonographically identified.
Design—Prospective study.
Animals—90 client-owned dogs with high blood ammonia levels.
Procedure—Ultrasound examinations of the abdominal vessels and organs were performed in a systematic way. When ultrasonography revealed a congenital portosystemic shunt, surgical shunt-ligation was performed. When ultrasonography revealed acquired portosystemic collaterals or normal abdominal vasculature, liver biopsies were taken for histopathologic examination.
Results—Ultrasonography excluded portosystemic shunting in 11 dogs. Acquired portosystemic collaterals were found in 17 dogs, of which 3 had arterioportal fistulae and 14 other hepatic pathologies. Congenital portosystemic shunts were found in 61 dogs, of which 19 had intrahepatic and 42 extrahepatic shunts. Intrahepatic shunts originated from the left portal branch in 14 dogs and from the right portal branch in 5 dogs. Shunts from the right portal branch were either short or long vessels. Extrahepatic shunts originated either from the splenic vein, or from the right gastric vein (single shunts) or simultaneously from both of them (double shunts) and entered the abdominal caudal vena cava or the thorax. Ultrasonography revealed spleno-caval shunts in 24 dogs, right gastric-caval shunts in 9 dogs, spleno-azygos shunts in 8 dogs and right gastric-azygos shunt in 1 dog.
Conclusions and Clinical Relevance—Ultrasonography is a reliable diagnostic tool to identify and characterize congenital portosystemic shunts and to recognize acquired portosystemic collaterals as well as to exclude portosystemic shunting. A dilated left gonadal vein as a result of spleno-renal collateral-circulation was a reliable indicator of acquired portosystemic collateral-circulation.

Introduction

High blood ammonia levels in dogs are usually the results of one of 3 well-defined conditions: **acquired portosystemic collaterals (APSCs)**, **congenital portosystemic shunts (CPSSs)** or urea cycle enzyme deficiency.[1,2]

Portosystemic shunting occurs when anomalous veins allow the portal blood to enter the systemic veins directly without first flowing through the hepatic sinusoids. Acquired portosystemic collaterals are formed as the result of sustained hepatic or prehepatic portal hypertension by enlargement of extrahepatic rudimentary vessels, through which no blood normally passes.[2,3] Collateral-formation is a compensatory mechanism to maintain normal portal pressure by allowing the portal blood to be drained into the lower pressure systemic veins. Since the causes that lead to APSC-formation are either congenital (e.g. arterioportal fistulae) or acquired disorders (e.g. liver cirrhosis), APSCs can develop at any age and in any breed.[2,4-7] Posthepatic portal hypertension (e.g. right-sided heart failure) never results in APSC-formation because not only the portal pressure, but also the pressure in the **caudal vena cava (CVC)** increases.[2,6] A common consequence of any kind of portal hypertension is accumulation of free abdominal fluid, however portal hypertension cannot be excluded in the absence of ascites.[2,8]

Portosystemic shunting is considered to be congenital when a single or double anomalous vein is present without a concurrent portal hypertension.[5] Congenital portosystemic shunts are classified as intrahepatic and extrahepatic.[5,9] Intrahepatic CPSSs tend to occur in large breeds and result in hepatic encephalopathy usually under the age of one year,[9,10] whereas extrahepatic CPSSs occur predominantly in small breeds and can cause symptoms in young animals but may also remain clinically silent until old age.[5] The etiology of CPSSs is obscure except for the left-sided intrahepatic CPSS, which is believed to be a persistent ductus venosus.[11,12]

Urea cycle enzyme deficiency is a rarely detected congenital disease characterized by decreased activity of one or more enzymes that transform ammonia to urea.[1]

In addition to these 3 pathological conditions, healthy Irish wolfhound pups have a physiological period of transient hyperammonemia.[13]

Exceptionally, hyperammonemia may also be caused by conditions when ammonia-containing urine, as a result of the activity of urea-splitting microorganisms, has the possibility to be absorbed into the systemic circulation. This could happen in dogs with ruptured urinary bladder or urethra[14] or after a surgical ureterocolonic anastomosis.[15]

Ammonia is one of the toxins that plays a role in the development of hepatic encephalopathy, therefore an increased blood ammonia level can confirm that the central nervous symptoms of a dog are indeed in association with hepatic encephalopathy, or can reveal that the underlying cause of aspecific clinical symptoms is related to hyperammonemia. Elevated bile acids levels are also sensitive, however in contrast to hyperammonemia, are less specific indicators of portosystemic shunting,[a] since cholestasis and diffuse hepatocellular dysfunction, which may accompany several hepatic disorders, can also result in increased serum bile acids levels.[16]

Differentiating CPSSs from APSCs and from urea cycle enzyme deficiency non-invasively is crucial because CPSSs require surgical treatment and the other two conditions do not. Though ultrasonography is a quick and non-invasive technique to image the abdominal vessels and organs in unsedated dogs, veterinarians tend to rely on the results of angiographic studies.[6,17,18] According to our knowledge, the ultrasound-anatomy of the

different types of CPSSs and APSCs have not been described in dogs yet. The few reports that have been published about ultrasonography of portosystemic shunting focus only on some aspects of CPSSs without detailed anatomic descriptions.[12, 18-22]

Our aims are to describe the ultrasonographic findings of the conditions that result in hyperammonemia in dogs and the ultrasonographic anatomy of the different kinds of CPSSs. We demonstrate that ultrasonography is a reliable method to diagnose CPSSs and APSCs, and to identify the subtypes of CPSSs as well as to rule out portosystemic shunting.

Materials and methods

Of the dogs that underwent an abdominal ultrasound examination because of hyperammonemia at the Companion Animal Clinic of Utrecht University between July 2000 and December 2002 those were selected that were scanned by the first author and in which a definitive diagnosis was available based on laparotomy or necropsy or histopathologic examination of the liver or had characteristic ultrasonographic signs of arterioportal fistulae (ie, an extremely dilated and tortuous portal branch). Hyperammonemia was diagnosed when the 12-hour-fasting blood ammonia level exceeded the upper normal value (reference range, 24 to 45 μmol/L). Ninety client-owned dogs fulfilled the inclusion criteria. The animals were not sedated or anesthetized for the ultrasound examination except for a litter of 4 pups because anesthesia was necessary for blood sampling, and one dog that was anesthetized because the suspected anomalous vein could not be visualized in a conscious state, whereas the ultrasonographic findings were compatible with a left-sided intrahepatic CPSS. The examination of this dog was repeated under general anesthesia. No other cases required anesthesia or repeated examination.

When CPSS was the ultrasonographic diagnosis, the dog was subjected to surgical shunt-ligation, unless the owner did not agree with the surgical intervention or the dog was older than 5 years. Only the dogs that underwent a laparotomy or a necropsy were included in the study. Each surgery was performed by one surgeon (FJvS) according to a described protocol.[23]

When portal hypertension with APSCs was the ultrasonographic diagnosis, ultrasound-guided core biopsies of the liver were taken for histopathologic examination, unless the blood coagulation parameters were unsatisfactory. Only the dogs whose livers were histologically examined were included in the study.

When ultrasonography revealed a normal abdominal vascular anatomy without any evidence of portal hypertension or anomalous vessels, then a rectal ammonia tolerance test was performed according to a described method.[24] When the test indicated abnormal ammonia metabolism, a scintigraphy was performed by injecting ^{99m}Tc-macroaggregates into a parenchymal splenic vein under ultrasound guidance according to a described protocol,[25] and ultrasound-guided core biopsies of the liver were taken for histopathologic examination.

When a dog was examined more than once because of complains related to hyperammonemia in a later time point, only the first examination was included in this study, ie, re-checks are not.

First a B-mode abdominal ultrasound examination was performed [ATL HDI 3000 (Advanced Technology Laboratories) high definition ultrasound system equipped with a 7-4 MHz broad band phased array and a 8-5 MHz broad band curved linear array transducer,

Philips Medical Systems] paying special attention to the presence and amount of free abdominal fluid. The quantity of peritoneal effusion was subjectively estimated and the following categories were established: large amount when the ascites was detectable by physical examination, moderate amount when the ascites was undetectable by physical examination but seemed to be reasonably large amount ultrasonographically, and small amount if thorough ultrasonographic search was needed to detect free abdominal fluid. Ultrasonographic evaluation of the abdominal vasculature was performed in 7 standard planes according to a described protocol (**Chapter 5**). Shortly, with the dog in left lateral recumbency transverse sections of the portal vein and the portal branches were obtained via the right intercostal spaces and longitudinal sections via the right flank. With the dog in dorsal recumbency longitudinal sections of the portal vein were obtained via the ventral body wall. With the dog in right lateral recumbency longitudinal sections were made via the left flank to image the right gastric-caval shunts and the left gonadal vein.

Results

Ultrasonography of the 90 dogs revealed: normal abdominal anatomy in 11 dogs, CPSS in 61 dogs, APSCs due to arterioportal fistulae in 3 dogs and APSCs due to portal hypertension of hepatic origin in 14 dogs. In one dog both arterioportal fistulae and a CPSS were simultaneously present.

Normal ultrasound-anatomy

No abnormalities were found ultrasonographically in 11 dogs. In 2 of these dogs repeated measurements of blood ammonia levels and the results of the rectal ammonia tolerance tests were normal.

In the remaining 9 dogs (puppies of 3 litters) the ammonia tolerance tests were abnormal, the serum bile acids levels were normal, scintigraphy excluded portosystemic shunting, ultrasonography revealed a small amount of free abdominal fluid and histopathologic examination of the liver biopsy specimens revealed normal findings. This was found in a litter of 4 Belgian Malinois pups, a litter of 2 cairn terrier pups and in 3 Irish wolfhounds of a litter of 4 pups. (The fourth Irish wolfhound pup of the litter had a left-sided intrahepatic CPSS.) The age of the litters were 48, 59 and 63 days. Metabolic examination of blood and liver samples of the 3 Irish wolfhound pups revealed arginino-succinate synthetase enzyme deficiency.

Congenital portosystemic shunts

Congenital portosystemic shunt was the ultrasonographic diagnosis in 61 dogs. The liver was always small, sometimes to such an extent that parts of the stomach were in direct contact with the diaphragm. Free abdominal fluid was seen only in young puppies and its amount was always small.

Congenital portosystemic shunts were categorized into intrahepatic and extrahepatic CPSSs based on the origin of the shunt.

Intrahepatic portosystemic shunts were diagnosed in 19 dogs. The mean (range) age of the dogs at surgery was 5.9 (2.5-17.6) months. Only large breeds were represented except for a border collie. Intrahepatic CPSSs originated either from the left (14/19 dogs) or from the right portal branch (5/19 dogs) and appeared as the direct continuations of the

portal vein trunk (PV) as the diameters of the shunt and of the affected portal branch were the same as that of the PV. All intrahepatic CPSSs terminated in the CVC either directly or via a hepatic vein. The intrahepatic CPSS was a single vein in 18 dogs and was double in one dog (**Fig 1**).

Intrahepatic CPSSs that originated from the left portal branch ran always cranioventrally and to the left (similarly to a normal left portal branch) until the diaphragm, then turned abruptly dorsally to enter the CVC via a dilated segment of the left hepatic vein. In these dogs the right portal branch could not be visualized. A thin vessel with fast hepatopetal flow that was found at the place where the right portal branch was expected, appeared to be a hepatic arterial branch examined by pulsed-wave Doppler mode.

Intrahepatic CPSSs that originated from the right portal branch had a segment that consistently ran dorsolaterally and to the right from the PV, like a normal right portal branch, but then, instead of ramifying, turned medially to enter the CVC. This dorsolaterally running segment was either short (2/5 dogs) or long (3/5 dogs). One of the long shunts (in the border collie) was double (**Fig 1**). Whatever morphology of a right-sided intrahepatic CPSS had, the left portal branch could never be found.

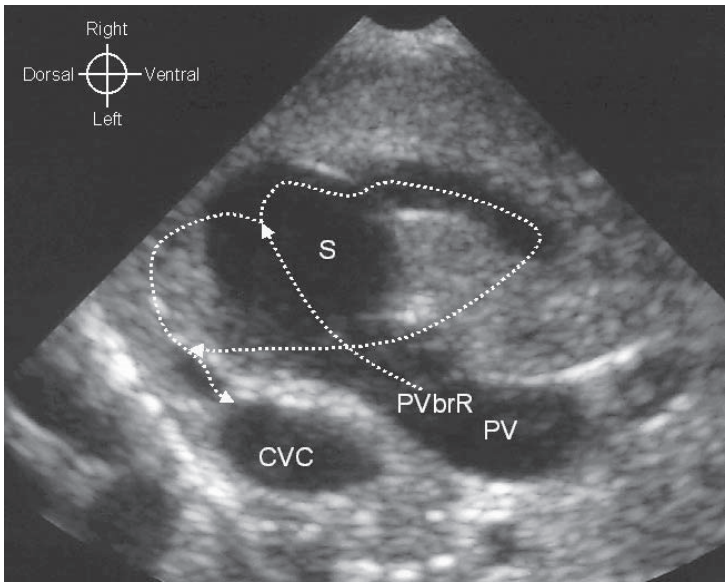


Figure 1.

Right divisional congenital *intrahepatic porto-caval* shunt. Gray scale ultrasound image of the right portal branch of a 3-month-old female border collie with a double intrahepatic porto-caval shunt originating from the right portal branch (PVbrR). At the end of the wide right portal branch a sinus (S) is seen, from which two shunt-loops arise: one courses ventrally and the other one dorsally (the dorsal loop cannot be seen in this image, but is indicated with an arrow). The ventral loop turns back to dorsal after a medial loop, then after the confluence with the dorsal loop a short common trunk enters the caudal vena cava (CVC).

Extrahepatic portosystemic shunts were diagnosed in 42 dogs. The mean (range) age of the dogs at surgery was 16.0 (2.9-66.4) months. Only small breeds were represented except for a Labrador retriever. Extrahepatic CPSSs originated from the splenic vein, from the right gastric vein, or from both and entered either the abdominal CVC or the thorax. Extrahepatic CPSSs were divided into 4 groups based on their origins and terminations: 24 dogs had a spleno-caval, 9 a right gastric-caval, 8 a spleno-azygos and 1 a right gastric-azygos shunt. Shunts with two loops (one arising from the right gastric vein and the other one from the splenic vein) were categorized as right gastric shunts because the right gastric vein was the main loop and this had an identical morphology with the single shunts that arise from the right gastric vein. These categories were established based on the ultrasonographic results and were in agreement with the surgical findings.

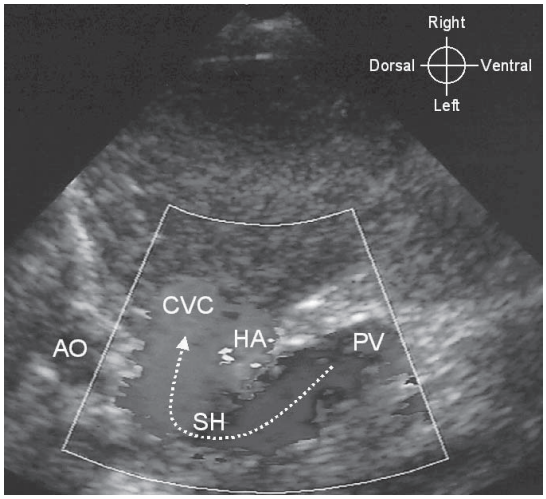


Figure 2.
Congenital *extrahepatic spleno-caval* shunt in a 3.5-month-old female Jack Russell terrier. Cross sections of the aorta (AO), caudal vena cava (CVC), hepatic artery (HA) and portal vein (PV) are seen. A short anomalous vein (SH) makes direct connection between the PV and the CVC on their left side. Color Doppler mode reveals that the direction of flow in the shunt is from the PV towards the CVC. The arrow indicates the direction of blood flow. (**Color** version on page 177.)

The most common type of congenital CPSS was the spleno-caval shunt. This usually formed a short single loop between the PV and the CVC (**Fig 2**). Although in 2 dogs the anomalous vein had a long cranial loop, the points of origin and termination were the same as in cases of short loops. Spleno-caval CPSSs originated from the splenic vein. Since the point of origin was very close to the point where the splenic vein entered the PV and the short segment of the splenic vein that was between the PV and the shunt-origin was dilated and the flow hepatofugal in it, the CPSS seemed to originate from the PV itself and the splenic vein seemed to enter the shunting vessel (**Fig 3A**). The origin of the spleno-caval shunts was slightly cranial to the level where the celiac artery originated from the aorta. The termination of the spleno-caval shunts in the CVC was always at the same point, slightly cranial to the level of shunt-origin (**Fig 4A**).

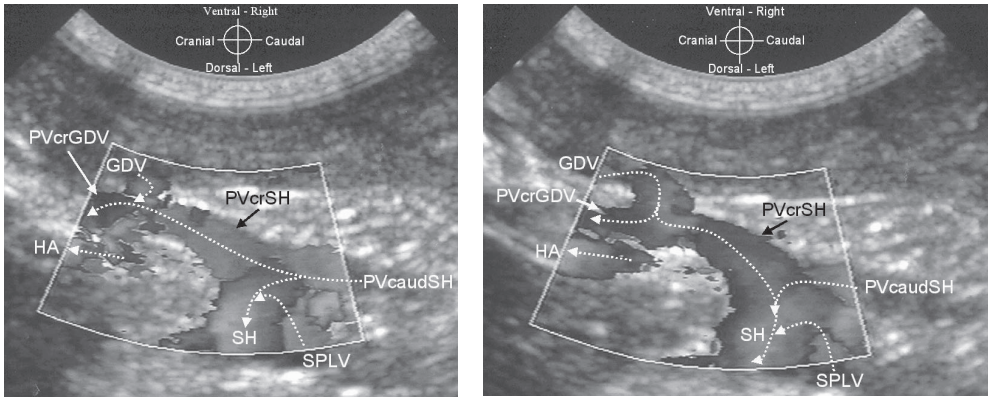


Figure 3.

Color Doppler images of a congenital extrahepatic spleno-caval shunt in a 4-month-old male Yorkshire terrier with the dog in dorsal recumbency. A and B images were made at the same point with a difference of 2 seconds. Compare A and B images and note the "to-and-fro" flow-direction (alternating hepatopetal and hepatofugal flow) in the portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein (PVcrSH). Continuous hepatopetal flow is seen in the portal vein cranial to the entering point of the gastroduodenal vein (PVcrGDV) and in the portal vein caudal to the shunt-origin (PVcaudSH). Dotted arrows indicate the direction of blood flow, shunt (SH), HA hepatic artery, SPLV splenic vein. GDV gastroduodenal vein.

A. Hepatopetal flow in the entire portal vein. (Full **color** illustration on page 178.)

B. Hepatofugal flow in the portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein. (Full **color** illustration on page 178.)

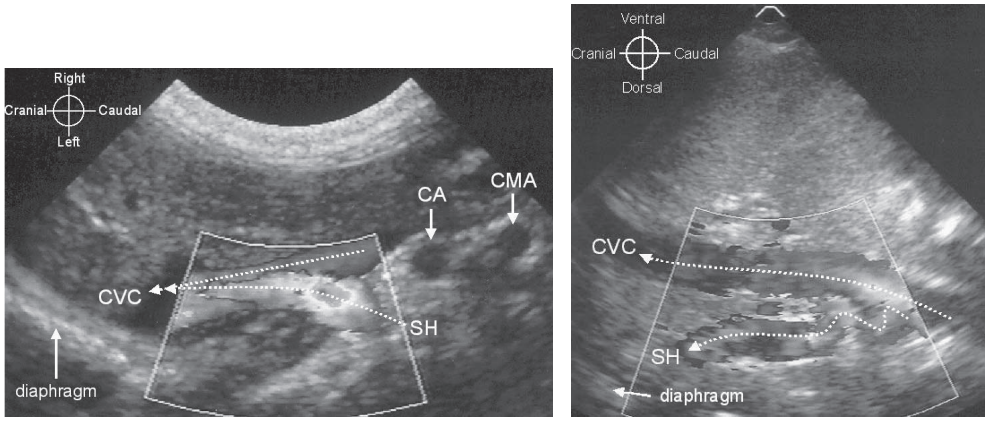


Figure 4.

Color Doppler images showing the termination of congenital extrahepatic portosystemic shunts imaged with a transducer positioned caudal to the last right rib with the dog in dorsal recumbency. Dotted arrows indicate the direction of blood flow.

A. Spleno-caval shunt in a 3-month-old female cairn terrier at the point where the shunt (SH) enters the caudal vena cava (CVC). This point is located always slightly cranial to the point where the celiac artery (CA) originates from the aorta. CMA cranial mesenteric artery. (Full **color** illustration on page 178.)

B. Spleno-azygos shunt in a 3.5-month-old male Jack Russell terrier. The shunting vessel (SH) runs dorsal to the CVC, and enters the thorax. Dotted arrows indicate the direction of blood flow. (Full **color** illustration on page 178.)

In cases of spleno-azygos shunts, the shunting vessel ran towards the CVC, but instead of entering it at the point where the spleno-caval shunts terminated, it ran further, dorsal to the CVC and eventually entered the thorax (**Fig 4B**). The point of origin of these spleno-azygos shunts was the same as that of the spleno-caval shunts.

The diameter of the shunt with a splenic vein origin was always wider than that of the PV caudal to the shunt-origin (**Fig 3**). The PV-segment cranial to the shunt-origin was thinner than the PV-segment caudal to it in all 32 dogs whose shunt originated from the splenic vein. Sometimes the PV cranial to the shunt-origin was so thin that it could not be visualized ultrasonographically. The flow-direction in the PV-segment cranial to the shunt-origin was hepatofugal or "to-and-fro" in 17/32 dogs determined by color Doppler mode (**Fig 3**). In the remaining dogs this segment could not be visualized or the flow was normal, ie, hepatopetal. The PV-segment cranial to the origin of the gastroduodenal vein could only occasionally be imaged. Whenever this PV-segment was visualized, its flow was always hepatopetal, even if cranial to the shunt-origin hepatofugal portal flow was seen (**Fig 3B**).

Right gastric-caval shunts were in most of the dogs double shunts, since they had a cranial and a caudal shunt-loop, however the two loops anastomosed before entering the systemic venous system. The cranial loop arose from the right gastric vein and the morphology of its origin was slightly variable depending whether the right gastric vein was a tributary of the gastroduodenal vein or of the PV itself. In both cases the shunt (ie, the dilated right gastric vein) originated immediately caudal to the portal bifurcation. When the right gastric vein was a tributary of the gastroduodenal vein, the origin of the shunt (ie, the right gastric vein) was very close to the point where the gastroduodenal vein entered the PV and the short segment of the gastroduodenal vein that was between the PV and the origin of the right gastric vein was dilated and the flow hepatofugal in it. Regardless of the anatomic variation of the right gastric vein, the course of the shunt was always the same, namely it made a long loop from the liver hilus: running laterally to the left body wall, then turned caudomedially and entered the CVC at the point where the spleno-caval shunts terminate, ie, slightly cranial to the level where the celiac artery originates from the aorta (**Fig 5**). In one dog, the shunt did not enter the CVC after making the above described course, but ran dorsal to the CVC to cranial and entered the thorax. In this dog the ultrasonographic diagnosis was right gastric-azygos shunt. All CPSSs that arise from the right gastric vein were very wide, with a diameter comparable to that of the CVC. At surgical exploration the cranial loop of the right gastric-caval shunts were found to follow the lesser curvature of the stomach, similarly to a normal right gastric vein. The caudal shunt loop, which was occasionally absent, originated at the region where spleno-caval shunts are expected, but unlike the spleno-caval CPSSs, the caudal loop of a right gastric-caval shunt ran from caudal to cranial and not from ventral to dorsal like the spleno-caval shunts, moreover it formed a longer shunt-loop than the spleno-caval shunts. The caudal loop drained the blood of the PV via the dilated segment of the splenic vein, through the left gastric vein to the right gastric vein. As the cranial shunt-loop (right gastric vein—CVC) and the caudal shunt-loop (splenic vein—left gastric vein—right gastric vein) anastomosed, a common trunk entered the CVC. The PV was slightly thinner cranial to the origin of the caudal shunt-loop (ie, cranial to the splenic vein) and the flow direction was always hepatopetal in it. The PV cranial to the origin of the cranial shunt-loop was so thin that could not be visualized by ultrasound.

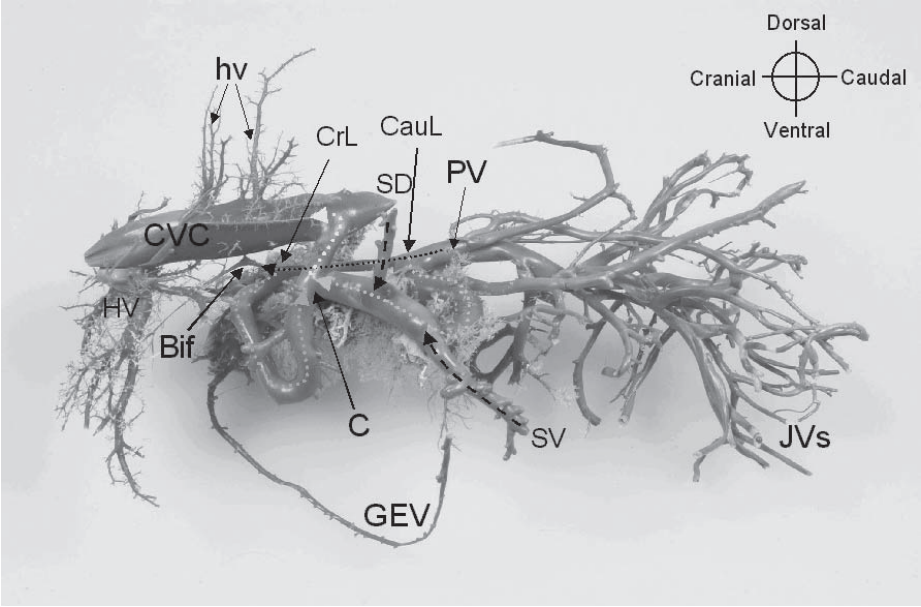
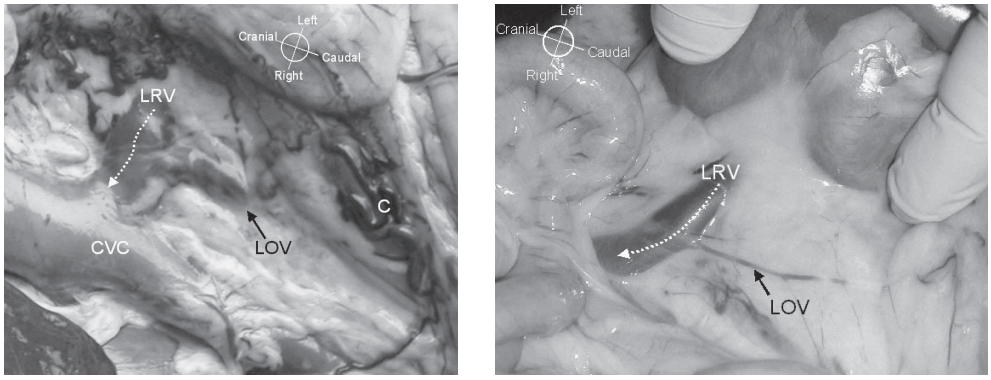


Figure 5.
Corrosion cast of the abdominal veins of a Yorkshire terrier with a right gastric-caval shunt. The cranial shunt-loop (interrupted green arrow) and the caudal shunt-loop (interrupted cyan arrow) anastomose with each other. From the point of confluence (C) the shunting vessel (interrupted yellow arrow) drains the portal blood to the caudal vena cava (CVC). Note that the portal vein becomes narrower cranial to the point of the origin of the caudal shunt-loop (CauL), and even more narrower cranial to the point of the origin of the cranial shunt loop (CrL). The cranial shunt-loop originates at the point where the gastroduodenal vein enters the portal vein. (The gastroduodenal vein is on the other side of the cast, so is not visible on this photo.) The ligature is usually placed around the common trunk (interrupted yellow arrow). The arrows indicate the flow-directions in the vessels.
Bif Portal bifurcation with the very thin right and left portal branches. HV Left hepatic vein, hv Hepatic veins, JVs Jejunal veins, GEV Right and left gastroepiploic veins, SV Ventral branch of the splenic vein, SD Dorsal branch of the splenic vein. (Full **color** illustration on page 178.)

Acquired portosystemic collaterals

Portal hypertension with APSCs was the ultrasonographic diagnosis in 17 dogs. In all of these dogs a dilated left gonadal vein (testicular vein in males and ovarian vein in females) was seen. This vein ran parallel to the CVC and entered the mid-portion of the left renal vein from caudal (**Figs 6A, 6B**). The diameter of the gonadal vein was usually similar to that of the renal vein, but in some cases it was much wider, and either had a straight or a tortuous course. Lots of tiny tortuous veins were often observed around the left renal vein (**Fig 7**). In one dog the dilated left gonadal vein could be followed caudally to a conglomeration of smaller vessels, whereas in another dog it could be followed all the way to its origin, to the splenic vein at the splenic hilus. In the remaining dogs the dilated left gonadal vein could not be followed.

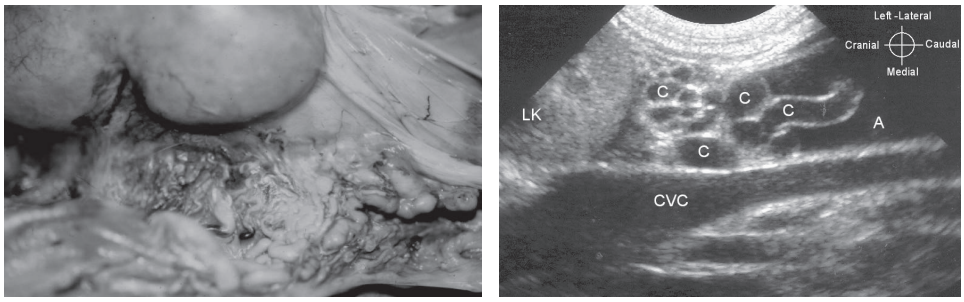


A B

Figure 6.
Left gonadal vein.

A. Dilated left ovarian vein (LOV) in a 5-month-old female great Dane with spleno-renal collaterals as a result of sustained portal hypertension of hepatic origin. C conglomeration of collateral vessels, CVC caudal vena cava, LRV left renal vein. (Full **color** illustration on page 179.)

B. Normal left ovarian vein in a 9-month-old female Labrador retriever. The left ovarian vein (LOV) is much thinner than the left renal vein (LRV). (Full **color** illustration on page 179.)



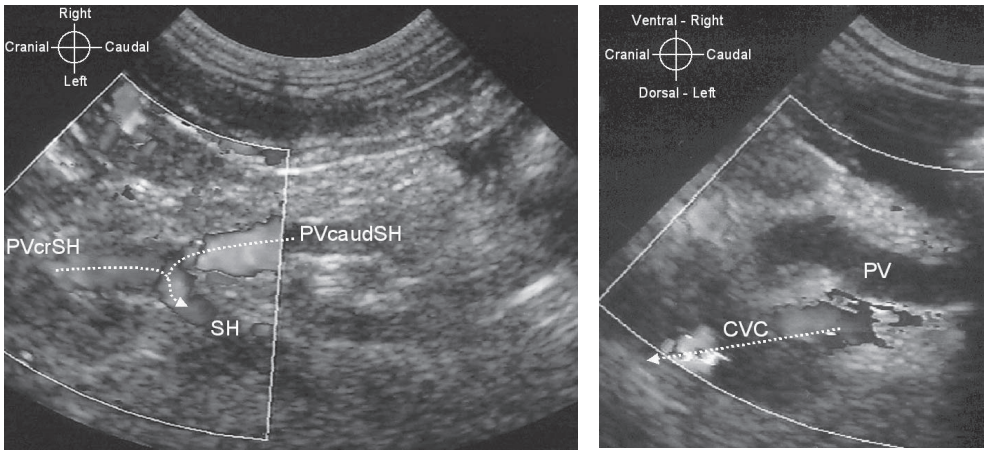
A B

Figure 7.
Acquired portosystemic collaterals

A. Spleno-renal collaterals at the region of the left kidney in a 2.5-year-old German shepherd dog with a sustained prehepatic portal hypertension. Purple silicon rubber was injected into the caudal vena cava and yellow into the splenic vein to outline the portosystemic communication in this formalin-perfused specimen. (Full **color** illustration on page 177.)

B. Gray scale ultrasound image of spleno-renal acquired collaterals (C) caudal to the left kidney (LK) in a 1-year-old female Dutch schapendoes with sustained portal hypertension of hepatic origin. A ascites, CVC caudal vena cava

In addition to the dilated left gonadal vein, the origin of an APSC from the PV was seen in 4 dogs at the point where congenital extrahepatic spleno-caval shunts arise (**Fig 8A**). These APSCs originated directly from the PV, and their relations to the splenic vein could not be revealed. In 2/4 dogs the flow in the entire PV was so slow that no color signals were detected with the lowest possible pulse repetition frequency setting (**Fig 8B**), whereas in the 2 other dogs the flow velocity caudal to the APSC-origin was not slow estimated with color Doppler mode, however cranial to the APSC-origin the flow-direction in the PV was hepatofugal (**Fig 8A**).



A B

Figure 8.

Hepatic portal hypertension. (Full **color** illustrations on page 179.)

A. Color Doppler ultrasound image of the portal vein and the origin of an acquired portosystemic collateral (SH) in a 5-year-old West highland white terrier with sustained portal hypertension of hepatic origin. Cranial to the collateral-origin (PVcrSH) hepatofugal portal flow can be seen. Note that the anomalous vein (SH) runs caudally. Dotted arrows indicate the direction of blood flow, PVcaudSH portal vein caudal to the collateral-origin. Compare with Figure 3B!

B. Color Doppler ultrasound image of the portal vein in a 6.5-year-old female Jack Russell terrier with sustained portal hypertension due to primary hypoplasia of the portal vein shows undetectably slow flow in the portal vein. Note that no color signals are seen in the portal vein (PV), whereas aliasing artefact is seen in the caudal vena cava (CVC). The dotted arrow indicates the direction of blood flow.

The morphology of APSCs that originated from the PV was very similar to that of congenital extrahepatic spleno-caval shunts. Moreover, in both cases hepatofugal portal flow cranial to the origin of the anomalous vein could be seen. The differences were: an APSC ran caudally from the point of origin and tended to disappear among the intestines, furthermore the diameters of the PV cranial and caudal to the APSC-origin were approximately equal. In contrast, congenital extrahepatic spleno-caval or spleno-azygos shunts ran cranially from the origin and could always be followed to their terminations (CVC or thorax). Thirdly, the PV-segment that was cranial to the origin of a spleno-caval or a spleno-azygos shunt was always thinner than the PV-segment caudal to it. Moreover, an APSC was never wider than the PV caudal to the anomalous vein, unlike in cases of CPSSs. In addition to these features, the simultaneous presence of the dilated left gonadal vein always proved that an extrahepatic anomalous vein that originated from the PV was the origin of an APSC.

Three kinds of underlying diseases were found that led to the formation of APSCs: arterioportal fistulae, **primary hypoplasia of the portal vein (PHPV)**, and chronic hepatitis. Arterioportal fistulae were diagnosed ultrasonographically, whereas chronic hepatitis and PHPV were differentiated by histopathologic examination of liver biopsy specimens. The size of the spleen was normal in all portal hypertensive patients.

Arterioportal fistulae were diagnosed in 4 dogs, however the classical ultrasonographic appearance (**Fig 9**) was seen only in 2 of them (a 6-month-old American Staffordshire terrier and a 2.5-month-old bullmastiff); namely (1) ascites, (2) extremely dilated and tortuous portal branch in a liver lobe, (3) hepatofugal flow in the PV (with a variable or a clear arterial Doppler spectrum) and (4) APSCs. A 3-year-old basset hound was presented without any free abdominal fluid, but the other 3 features were found. A 9-month-old Labrador retriever had only an extremely dilated and tortuous portal branch without ascites or APSCs, and the flow-direction in the PV was normal, ie, hepatopetal. These findings suggested that in addition to the arterio-portal communication, an intrahepatic porto-caval communication must had existed, which was confirmed during necropsy. Histopathologic examination of the liver biopsy specimens of the Labrador retriever and of the basset hound revealed hypoplastic portal branches.

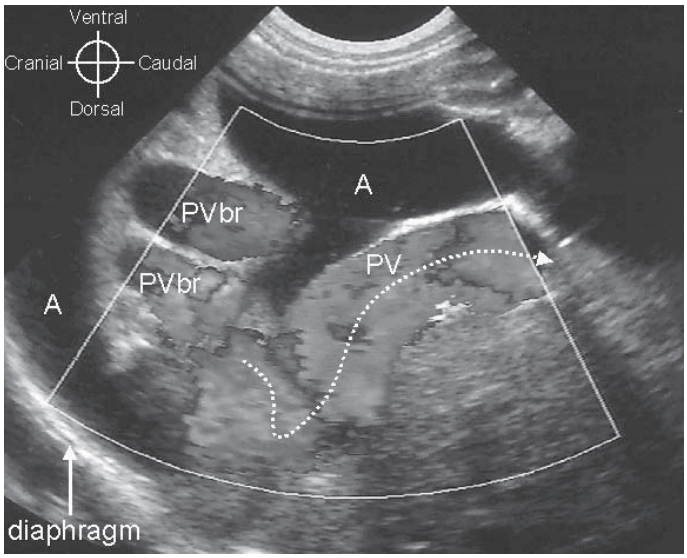


Figure 9.
Congenital arterio-portal fistula. Color Doppler ultrasound image of the liver of a 6-month-old male American Staffordshire terrier. Note the dilated portal branches (PVbr) in the affected liver lobe. The flow-direction in the portal vein (PV) is hepatofugal. The dotted arrow indicates the direction of blood flow, A ascites. (Full **color** illustration on page 179.)

Primary hypoplasia of the portal vein as the cause of APSC-formation was diagnosed in 7 dogs (6 female and 1 male) by means of histopathologic examination of the liver. The mean (range) age of the dogs was 3.4 (0.5-6.5) years. The liver was small in all dogs with either normal (2/7 dogs) or slightly irregular echo-structure (5/7 dogs). Ascites was found at physical examination only in 1/7 dog; in 3 other dogs moderate amount of free abdominal fluid was detected ultrasonographically, and in the remaining 3 dogs no free abdominal fluid was seen.

Chronic hepatitis with or without cirrhosis was the histopathologic diagnosis of the liver biopsy specimen in 7 dogs (3 females and 4 males). The mean (range) age of the

dogs was 5.5 (2.9-9.5) years. Six of the 7 dogs had severe ascites at physical examination and irregular liver structure detected by ultrasound. One dog had only moderate amount of free abdominal fluid and ultrasonographically normal liver.

Double caudal vena cava

Double CVC was found in 3 dogs in addition to an extrahepatic CPSS. In cases of double CVC the left and right common iliac veins fuse to form the CVC more cranial than normal, namely between the left and right renal veins, hence the left renal vein enters the left common iliac vein and the right renal vein enters the CVC. The left and right common iliac veins had the same diameters and ran symmetrically on the respective side of the aorta (**Fig 10**).

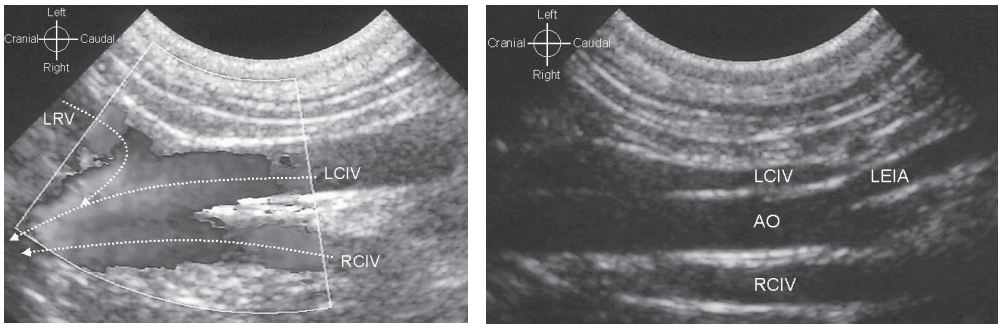


Figure 10.

Double caudal vena cava. (Full **color** illustration on page 179.)

A. Color Doppler image of the left renal vein (LRV) as it enters the left common iliac vein (LCIV) in a 3-month-old female cairn terrier. Compare to Figure 6! Dotted arrows indicate the direction of blood flow, RCIV right common iliac vein.

B. Gray scale image of the aortic trifurcation. The left and right common iliac veins (LCIV, RCIV) run on the corresponding side of the aorta (AO) beyond the level of the aortic trifurcation. LEIA left external iliac artery

Discussion

As a cause of hyperammonemia, the following diseases were found in our series of dogs: (1) CPSS, (2) APSCs due to PHPV, (3) APSCs due to arterioportal fistulae, (4) APSCs due to chronic hepatitis and (5) urea cycle enzyme deficiency.

The incidence of APSCs and CPSSs in the present study does not represent the incidence of these disorders in the clinic's population because the dogs with an ultrasonographic diagnosis of APSCs or with congenital extrahepatic spleno-azygos shunts were more often excluded from the study population than dogs with for example intrahepatic CPSSs. In cases of APSCs an exclusion criterion was an insufficient coagulation time, which did not allow liver biopsies to be taken, hence the ultrasonographic diagnosis remained unconfirmed. In cases of CPSSs, if the dog was older than 5 years, dietary and medical management was recommended instead of surgical shunt-ligation. Therefore, several dogs with an ultrasonographic diagnosis of congenital extrahepatic

spleno-azygos shunt were not included in the study population because the ultrasonographic diagnosis was not confirmed surgically.

Two basic types of intrahepatic CPSSs and four basic types of extrahepatic CPSSs were found in this study. The basic categories were established based on the origin and termination of the anomalous veins.[10]

Based on the origin and the course of the anomalous veins, the intrahepatic CPSSs were categorized into left-divisional shunts (originating from the left portal branch), right-divisional shunts (long shunts originating from the right portal branch) and central-divisional shunts (short shunts originating from the right portal branch). These categories are based and are in agreement with reported angiographic studies.[12]

Previous descriptions of extrahepatic CPSSs used the "porto"-caval term.[18,19] Indeed, on portographic and ultrasonographic images all extrahepatic CPSSs seem to arise from the PV itself and they divert the blood of the PV, however necropsy studies showed that they actually originate either from the splenic or from the right gastric veins.[10,20] Ultrasonographically we confirmed these findings and found that all extrahepatic CPSSs originated very close to the point where the splenic vein (in cases of spleno-caval or spleno-azygos shunts) or the right gastric vein (in cases of right gastric-caval shunts) entered the PV, and the short segment between the PV and the shunt-origin was always dilated, with hepatofugal flow. What actually happens in cases of extrahepatic CPSSs with splenic vein origin is that the shunt drains the blood of the PV and of the splenic vein via a short and dilated segment of the splenic vein to the CVC or to the azygos vein. In dogs, whose portal flow direction is hepatofugal in the PV-segment that is cranial to the shunt-origin, the blood of the gastroduodenal vein flows via the PV to the shunt as well. In cases of extrahepatic CPSSs with right gastric vein origin, there are two anatomic variations regarding the cranial loop of the shunt: (1) the right gastric vein is a tributary of the PV and is located between the portal bifurcation and the gastroduodenal vein, or (2) the right gastric vein is a tributary of the gastroduodenal vein.[26] When the right gastric vein is a direct tributary of the PV, the blood of the PV is drained via the right gastric vein to the CVC or to the azygos vein. When the right gastric vein is a tributary of the gastroduodenal vein, then the blood of the PV is drained via a short and dilated segment of the gastroduodenal vein through the right gastric vein into the CVC or into the azygos vein and the blood of the gastroduodenal vein flows through the shunt (ie, the right gastric vein) without reaching first the PV. The caudal shunt-loop of a right-gastric-caval shunt actually functions similarly to a spleno-caval shunt, except for the fact that the flow direction is always hepatopetal in the PV segment cranial to the origin of the caudal loop (ie, between the origins of the two shunt-loops). Since in cases of extrahepatic "porto"-azygos CPSSs the shunting vessel enters the azygos vein in the thorax,[10] we assumed that the CPSSs that entered the thorax terminated in the azygos vein,[20] however we could never visualize this segment by ultrasound because of the air-filled lungs.

Hepatofugal portal flow has been reported in association with extrahepatic CPSS in one dog, but no explanation was added.[18] Hepatofugal portal flow cranial to the shunt-origin was observed in our case series both with APSCs and congenital extrahepatic spleno-caval and spleno-azygos shunts (**Figs 3B, 8A**). The cause of hepatofugal portal flow however is probably different. In cases of CPSSs, the blood of the gastroduodenal vein was found to be responsible for the hepatofugal flow because the gastroduodenal blood finds lower resistance to flow towards the shunt than towards the hepatic sinusoids. This was confirmed by the presence of hepatopetal portal flow cranial to the entering point of the

gastroduodenal vein (**Fig 3**). Preoperative detection of the flow-direction in the PV-segment cranial to the origin of a CPSS may be important in determining the level of portal vein hypoplasia, hence to facilitate predicting the surgical outcome (**Chapter 7**). In cases of hepatopetal or "to-and-fro" flow excellent prognosis could be expected because the flow-direction is continuously or at least intermittently physiological, whereas in cases of continuous hepatofugal flow the surgical prognosis can range from poor to excellent. In human cirrhotic patients microscopic intrahepatic arterioportal communications were found to be responsible for the hepatofugal portal flow, and were thought to have been formed due to the disorganized hepatic architecture.[27,28] This might also be the case in dogs with chronic hepatitis,[5,29] however visualizing the portal branches in dogs with portal hypertension to detect whether the flow direction was indeed hepatofugal in them was impossible in any dogs of our study. Intraoperative Doppler ultrasonography of the portal branches[30] or angiography by superselective catheterization of a hepatic branch of the hepatic artery would be necessary to reveal whether intrahepatic arterioportal communications are indeed responsible for the hepatofugal portal flow in dogs with sustained portal hypertension.

Primary hypoplasia of the portal vein is a distinct congenital disorder characterized by hypoplasia of the portal branches and sometimes also of the PV.[4] In mild cases, portal hypertension and APSCs do not develop, hence blood ammonia level remains normal, however in severe cases APSCs develop as a consequence of sustained hepatic portal hypertension. The severe form of PHPV has also been described as non-cirrhotic portal hypertension.[6,31-33] "Hepatic microvascular dysplasia" was described as a condition that results in elevated serum bile acids levels without demonstrable macroscopic portosystemic shunting and was speculated to cause hepatic encephalopathy due to suspected microscopic intrahepatic portosystemic communications;[34-37] however blood ammonia levels of these dogs have never been determined. Since the histopathologic changes in the liver as well as the clinical and laboratory findings of these reported dogs were identical with the findings of dogs with the mild form of PHPV, the "WSAVA liver diseases and pathology standardization research group" agreed that the described condition is actually also PHPV.[b,33]

The etiology of secondary portal vein hypoplasia is different from that of the PHPV. Secondary hypoplasia of the portal branches can be found when the blood supply of the liver via the portal vein is reduced due to e.g. portal vein thrombosis or CPSS. Since a CPSS has equal or larger diameter compared to the PV-segment caudal to the shunt, it offers a lower resistance route towards the systemic veins than the route towards the hepatic sinusoids; therefore the PV cranial to the shunt-origin and the intrahepatic portal branches remain hypoperfused. Ultrasonographically this phenomenon is usually recognizable, namely in cases of intrahepatic CPSSs the non-affected portal branch is frequently unrecognizably thin, whereas in cases of extrahepatic CPSSs the portal vein segment that is cranial to the shunt-origin is much thinner than the segment that is caudal to it. In most cases of extrahepatic CPSSs the left and right portal branches are also very thin. The smallest portal branches, at microscopic level, are also thinner than normal, however this secondary portal vein hypoplasia is histologically indistinguishable from PHPV.[4,5] The situation can be more complicated because PHPV is either an independent disease, but can also be simultaneously present with arterioportal fistulae[38] and with CPSSs. When a CPSS and PHPV are simultaneously present in the same dog, PHPV cannot be diagnosed preoperatively. In these dogs portal hypertension and APSCs will not develop, because

there is already an existing connection between the portal and the systemic veins in the form of a CPSS. Currently, the earliest time point when PHPV can be suspected in a dog that has a simultaneous CPSS is during surgical attenuation of the CPSS, with the use of intraoperative Doppler ultrasonography (**Chapter 7**).

Congenital arterioportal fistula is a single or multiple connection of a portal branch and a hepatic arterial branch that results in development of portal hypertension and formation of APSCs. Ultrasonographically, the presence of an extremely dilated and tortuous portal branch in a liver lobe is a pathognomic finding.[7] The high arterial pressure causes dilation of the affected portal branch and hepatofugal flow in the affected portal branch and in the PV. Similarly to a reported case,[38] both dogs that underwent a liver biopsy in our case-series had coinciding PHPV. Surgical resection of the affected liver lobe(s) should only be considered when PHPV is excluded by histopathologic examination of liver biopsy specimens, because after having ceased the arterioportal communication surgically, the portal blood would continue to flow towards the lower resistance ie, via the APSCs, and not towards the hypoplastic portal branches.

The development of portal hypertension and APSCs in dogs with cirrhosis is well documented and is associated with the disorganized hepatic architecture due to the formation of connective tissue and regenerative nodules.[2,8] Portal hypertension due to chronic hepatitis develops as a result of intrahepatic inflammatory and fibrotic changes.[2,39] As a result of these pathological processes, the little portal branches and hepatic veins in the liver lobes are compressed and portal hypertension develops.

Since no previous studies aimed diagnosing APSCs ultrasonographically, the ultrasonographic features of APSCs have not been described in details yet.[19] Measuring portal flow velocity was suggested to differentiate APSCs from CPSSs[18,40] because B-mode ultrasonographic signs of APSCs were thought to be variable.[40] However, we showed that slow portal flow velocity is not necessarily present in all cases of portal hypertension (**Fig 8A**). Moreover, a dilated left gonadal vein was found to be a reliable indicator of APSCs. The dilated left gonadal vein is the result of the spleno-renal collateral circulation, which is the most consistently observed route of APSCs in dogs.[3,4,29] The left gonadal vein enters the left renal vein in normal dogs, but it is so thin that cannot be ultrasonographically visualized (**Fig 6**, **Chapter 5**). As a result of sustained hepatic or prehepatic portal hypertension, the virtual communications between the velar-omental radicles of the splenic vein and the mesocolic radicles of the left gonadal vein become functional and these rudimentary vessels dilate allowing the portal blood to shunt into the systemic veins, therefore the left gonadal vein gets dilated.[3] Non-invasive diagnosis of APSCs is essential because patients with portal hypertension and hyperammonemia are of a high anesthetic risk,[41] furthermore there is nothing in the abdominal cavity that could surgically be corrected in these dogs during a diagnostic laparotomy; except for some rare cases such as a circumscribed stenosis of the PV.[42] Banding the CVC was described as a surgical technique to reduce portosystemic shunting via APSCs by attenuating the CVC to increase its pressure above the portal pressure, however this method did not bring satisfactory results,[43] hence is not recommended.

In cases of spleno-renal collaterals, a relatively wide and straight collateral vein can regularly be seen. This vein should not be mistaken with a CPSS. We believe that the conditions that were described as spleno-mesenteric-renal shunt in 2 dogs[44] as well as the case report of a "suspected microscopic hepatic arteriovenous fistulae"[16] were both cases of PHPV with acquired spleno-renal collaterals. The fact that the histopathologic findings of

liver biopsy specimens are identical in cases of CPSS, PHPV and arterioportal fistula, these conditions should be differentiated by means of diagnostic imaging.

A double CVC is an innocent congenital vascular anomaly.[45] According to our knowledge ultrasonographic diagnosis of this condition has not been reported yet. The only significance of a double CVC is that the left common iliac vein may be mistaken with a dilated left gonadal vein. If a vein is found on the left side of the aorta caudal to the left kidney running parallel to the CVC, thorough evaluation of this vein and the abdomen is necessary to decide whether the dog has a double CVC or APSCs. The left and right common iliac veins in cases of a double CVC have the same diameter and they run symmetrically on the respective side of the aorta, whereas a dilated left gonadal vein may be tortuous and its diameter is usually different from that of the CVC. In addition, the common iliac veins can always be followed caudally until the point where they leave the abdomen, unlike a left gonadal vein.

Large amount of free abdominal fluid may hinder ultrasonographic visualization of the abdominal vessels, but hyperammonemia in dogs with severe peritoneal effusion cannot possibly be the result of CPSSs or an urea cycle enzyme deficiency,[2,19] since a dog with CPSS cannot have portal hypertension or so severe hypoalbuminemia that would result in a formation of a large amount of transudate in the abdominal cavity.[5,19] Naturally occurring simultaneous presence of APSCs and CPSSs is also very unlikely, if not impossible[c]. Small amount of free abdominal fluid is normal in healthy pups, hence may also be seen in puppies with CPSSs or with urea cycle enzyme deficiency.

Free abdominal fluid was absent in some dogs with sustained portal hypertension in our case-series. This is not surprising, since ascites-formation depends not only on the degree of portal pressure but also on the colloid osmotic pressure and albumin concentration of the blood as well as the effectiveness of the APSCs.[2] In the dog that had a combination of arterioportal fistulae and an intrahepatic porto-caval shunt, ascites and APSCs did not develop because the simultaneously present congenital porto-caval shunt did not allow portal hypertension to develop.

No splenomegaly was found in any of the dogs with portal hypertension, however it is a common finding in humans and was suggested to be present in dogs as well.[46] The reason why splenomegaly does not develop in dogs with APSCs could be that acquired spleno-renal collaterals are the most consistently developing APSCs in dogs in contrast to humans, and these collateral veins prevent the spleen from congestion.

Since in dogs with a urea cycle enzyme deficiency a functional disorder is responsible for the hyperammonemia, ultrasonographically the abdominal organs and vessels do not differ from normal.

The scanning time that was necessary to image the anomalous vein or to exclude portosystemic shunting was approximately 15-30 minutes. This was actually not or not much longer than a routine abdominal ultrasound examination. This was possible not only because of the experience and skill of the ultrasonographer and the use of a high quality ultrasound system, but also because of the use of our systematic scanning protocol (**Chapter 5**) and the knowledge of the ultrasound anatomy of the various types of vascular anomalies. Moreover, an important factor that made the examinations shorter was that the clinician's request was straightforward and based on the high blood ammonia level only three distinct conditions had to be distinguished with ultrasound: normal anatomy, CPSS and portal hypertension.

In sum, ultrasonography is a reliable diagnostic method to characterize the underlying disease of hyperammonemic dogs non-invasively. The cause of APSCs can be established ultrasonographically in cases of arterioportal fistulae, however in other cases taking an ultrasound-guided liver biopsy for histopathologic examination is necessary.

Acknowledgements

Dr Szatmári was supported by the Hungarian State Eötvös Scholarship. Mr Aart van der Woude helped in preparing the illustrations. This paper was presented in part at the WSAVA-FECAVA-AVEPA veterinary congress in Granada, Spain, October, 2002.

Footnote

a. Gerritzen-Bruning MJ, Rothuizen J. Sensitivity and specificity of ammonia and bile acids in diagnosing portosystemic shunting. Abstract. p. 264. "Voorjaarsdagen" Intrernational Veterinary Congress, April 25-27, 2003, Amsterdam, The Netherlands

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c. Szatmári V. Simultaneous congenital and acquired extrahepatic portosystemic shunts in two dogs — Letter to the Editor. *Vet Radiol Ultrasound* 2003;44:486-487.

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CHAPTER 5

Standard planes for ultrasonographic identification and characterization of congenital portosystemic shunts and acquired portosystemic collaterals in dogs

based on the article by

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Standard planes for ultrasonographic examination of the portal system in dogs

Journal of the American Veterinary Medical Association
2004;224:713-716.

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Summary

Seven planes were used to visualize the portal system in dogs that were suspected of having portosystemic shunting. To diagnose or to rule out portosystemic shunting, the right portal branch, the left gonadal vein, and the portal vein immediately caudal to the portal bifurcation as well as at the level of the splenic vein need to be imaged. In left lateral recumbency, transverse planes via the right intercostal spaces were used to find the right portal branch. Longitudinal planes, caudal to the last right rib, were used to reveal left-sided intrahepatic porto-caval shunts, and the extrahepatic spleno-caval and spleno-azygos shunts. In right lateral recumbency, longitudinal planes via the left abdominal wall were used to reveal the extrahepatic congenital shunts that arise from the right gastric vein, and to visualize the dilated left gondal vein, which is the result of spleno-renal collateral-circulation, as it enters the left renal vein from caudal.

Introduction

Portosystemic shunting occurs when anomalous veins allow the portal blood to directly enter the systemic veins without flowing first through the hepatic sinusoids. Portosystemic shunting is thought to be congenital if a single (or double) anomalous vein is present without a concurrent portal hypertension, whereas acquired portosystemic collaterals are formed as a result of a compensatory mechanism due to sustained hepatic or prehepatic portal hypertension.[1] Portosystemic shunting is suspected based on the history, clinical and laboratory findings, however direct visualization of the aberrant vein is essential for a definitive diagnosis[2] because elevated blood ammonia levels can be caused not only by **congenital portosystemic shunts (CPSSs)**, but also by **acquired portosystemic collaterals (APSCs)** and urea cycle enzyme deficiency.[3] Differentiating these three conditions non-invasively is crucial because only CPSSs require surgical treatment, the other two do not.

For diagnostic imaging, angiography was the first modality that has been used and is still the most frequently used technique. The interpretation of the portographic images is simple and the differentiation of APSCs from CPSSs, and intra- from extrahepatic CPSSs is possible.[4,5] However, portography is a time-consuming and invasive procedure that involves radiation.[6] Although scintigraphy is the gold standard diagnostic imaging technique to detect as well as to exclude portosystemic shunting, it does not allow differentiation of CPSSs from APSCs.[7,8] Computed tomography and MRI give detailed anatomic information of the vessels,[9,10] however cannot determine the flow-direction in them, moreover both procedures are time-consuming and require general anesthesia. Ultrasonography has been used for diagnostic imaging of CPSSs since the 1980`s,[11] and has become popular because the examination is quick, non-invasive, does not require anesthesia or radiation, and the abdominal organs can be simultaneously evaluated. Currently no any other modalities can compete with the advantages of color Doppler ultrasonography in abdominal vascular imaging, however veterinarians tend to rely on the results of other or additional diagnostic procedures (e.g. explorative laparotomy and portography) because of the reported low sensitivity and specificity of ultrasound.[12] The reason for the insufficient accuracy of the published ultrasound-studies could be that (1) no standardized examination protocol has been used; and (2) no descriptions have been available how the different types of CPSSs can be recognized and differentiated from one another. Moreover, the few reported studies focus only on some aspects of CPSSs, and the ultrasonographic features of APSCs are only superficially, or not discussed at all.[2,11-14] Our aim is to describe an examination protocol for systematic ultrasonographic evaluation of the canine portal system. This protocol has allowed us not only to recognize and characterize the different types of CPSSs and APSCs accurately, but also to exclude portosystemic shunting in a large number of patients.

Description of the technique

Abdominal ultrasound examinations were performed in conscious dogs. Hair was clipped over the entire abdomen and the region of the last few right ribs, and contact gel was applied. First, a routine B-mode real time abdominal ultrasound examination was performed [ATL (Advanced Technology Laboratories) HDI 3000 high definition

ultrasound system equipped with a 7-4 MHz broad band phased array and a 8-5 MHz broad band curved linear array transducers, Philips Medical Systems], then the abdominal vasculature was evaluated in 7 standard planes to identify or exclude CPSSs and APSCs with the simultaneous use of B- and color Doppler modes.

Color Doppler parameters were adjusted with care so that a vessel would be uniformly colored.[15] Namely, the color gain was set so that color signals would be seen in the entire lumen of a given vessel, but not outside the vessel. The **pulse repetition frequency (PRF)** was also appropriately adjusted, since a given PRF-setting is able to detect only a range of velocities. If the PRF is set too high, slow flow may be missed when the flow-velocity in a vessel is lower than the lower limit of the velocity-range that belongs to that PRF-value. When the flow-velocity is higher than the upper limit of the velocity-range, then aliasing artifact occurs. Traditionally, flow towards the transducer is coded with red, and flow away from the transducer with blue. Higher flow-velocities are coded with lighter shades of the appropriate color. If a velocity is higher than the upper limit of the velocity range, then no more lighter shade is available of the appropriate color, therefore these velocities will be coded with the wrong color, i.e. the color that indicates flow towards the opposite direction (blue instead of red).[15]

Plane-1: transverse intercostal section in left lateral recumbency as a starting point—The transducer was placed in one of the last right intercostal spaces. The aim was to find the intercostal space through which only the liver was seen without the right kidney, and the cross-sections of the aorta, the **caudal vena cava (CVC)** and the **portal vein trunk (PV)** could be visualized. When the right kidney appeared in the initial image, then the transducer was angled cranially or slided to a more cranial intercostal space, whereas, when air-containing lungs appeared, the transducer was angled caudally or slided to a more caudal intercostal space. When the PV could not be imaged because of the gastrointestinal gas, the transducer was shifted dorsally within the same intercostal space and directed ventromedially.

Plane-2: transverse intercostal section in left lateral recumbency to image the right portal branch—Starting from plane-1, the PV was followed by angling or sliding the transducer cranially to the point where the longitudinal image of the right portal branch appeared.

Plane-3: transverse intercostal sections in left lateral recumbency to image congenital extrahepatic portosystemic shunts—Starting from plane-2, the transducer was gradually slided caudally keeping the PV and CVC in the image, until the level where the cranial mesenteric artery originated from the aorta. Scanning was performed first with B-mode, then was repeated with color Doppler mode. The aim was to look for a direct connection between the PV and CVC, or for a vessel that originated from the PV with a hepatofugal flow direction (away from the PV).

Plane-4: longitudinal section in left lateral recumbency to image the portal vein and the left divisional intrahepatic congenital portocaval shunts as well as the congenital extrahepatic portosystemic shunts—Longitudinal images of the PV and of the main portal branches were obtained with a transducer placed immediately caudal to the last rib and directed craniomedially. To find the PV, first the longitudinal image of the aorta was

obtained immediately ventral to the vertebrae. By ventral angulation of the transducer, the CVC became visible. Further ventral angulation resulted in the longitudinal image of the PV at the point where the splenic vein entered the PV. Firm transducer-pressure was often necessary to image the portal bifurcation.

In deep-chested and in large dogs the PV could not be visualized via the right flank, hence an alternative approach was used, namely starting from plane-1 the transducer was rotated by 90° to obtain a longitudinal image of the PV intercostally.

Plane-5: longitudinal sections via the ventral abdominal wall in dorsal recumbency as an alternative for plane-4—To find the PV in dorsal recumbency, the dog was slightly tilted towards the sonologist, and the right kidney with the caudate liver lobe was imaged first. Then the transducer was angled a bit ventromedially to image the CVC, then further ventromedially to image the PV. To image the portal bifurcation, the PV was followed cranially. The transducer had often to be pushed firmly to move away the gas-filled intestinal loops.

Plane-6: longitudinal sections via the left lateral abdominal wall in right lateral recumbency to image the congenital extrahepatic right gastric-caval and right gastric-azygos shunts—The transducer was placed immediately caudal to the last left rib longitudinally and the PV was imaged at the hilus of the liver. Imaging the PV using this approach was rather difficult and was only necessary when using plane-3 an extrahepatic CPSS was suspected, but its entire visualization was impossible.

Another way to find the right gastric-caval shunts was to follow the hepatic artery from its origin to the liver, since the hepatic artery crossed the wide shunt-loop. To find the hepatic artery, the celiac artery was imaged as it originated from the aorta, cranial to the left kidney.[15] The hepatic artery is the widest branch of the celiac artery, which courses cranially to the liver between the PV and the CVC.[15] Color Doppler mode helped to find the hepatic artery, when the gray scale resolution was insufficient to visualize this thin vessel. The color signals of the hepatic artery indicated higher flow velocity compared to that of the CVC and the PV.

Plane-7: longitudinal sections via the left lateral abdominal wall in right lateral recumbency to image the dilated left gonadal vein, i.e. acquired spleno-renal collaterals—The CVC was imaged in longitudinal section by placing the transducer immediately ventral to the lumbar vertebrae and caudal to the left kidney. Keeping the longitudinal image of the CVC, the transducer was slided cranially to image the left renal vein entering the CVC. With B- and color Doppler modes a vein was searched that entered the left renal vein from caudal.

Results

Dogs

Twenty clinically healthy adult beagles of both sexes with normal serum bile acids and bilirubin levels as well as normal plasma alanine transaminase and alkaline phosphatase activities were examined to establish the normal abdominal vascular ultrasound anatomy. In addition to these normal dogs, 90 client-owned dogs with high

blood ammonia levels were examined. These patients were selected from the dogs that were examined because of hyperammonemia at the Companion Animal Clinic of Utrecht University between July 2000 and December 2002. Selection criteria were (1) laparotomy or necropsy or liver biopsy or scintigraphy was performed depending on the ultrasonographic diagnosis to confirm the ultrasonographic findings, and (2) the ultrasound examination was performed by the first author.

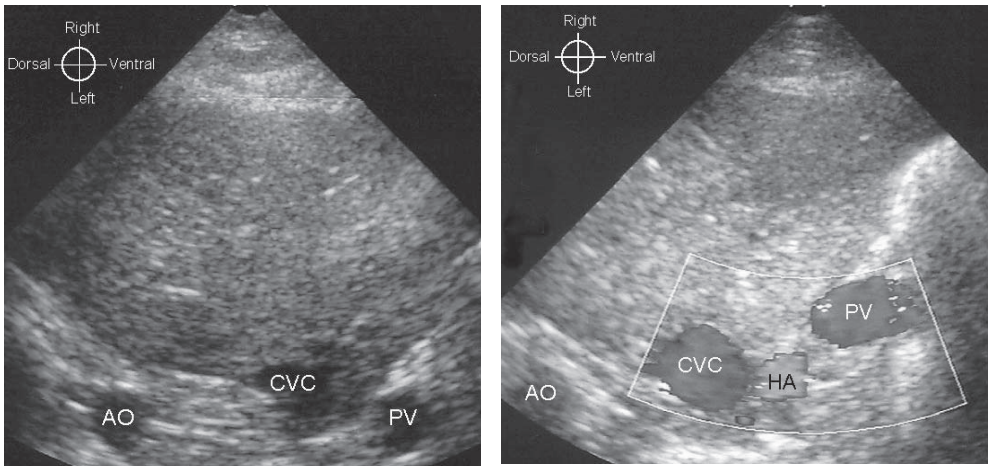
Findings in plane-1

Normal anatomy. From dorsal to ventral the cross-sections of the aorta, CVC and PV were seen; their cross-sectional areas were roughly equal (**Fig 1A**). The CVC was often latero-laterally collapsed.

Congenital intrahepatic porto-caval shunts. The images did not differ from normal, except for the presence of a dilated, hence clearly recognizable hepatic artery between the CVC and PV (**Fig 1B**). The hepatic artery was a pulsating vessel with a smaller diameter compared to that of the PV.

Congenital extrahepatic spleno-caval, spleno-azygos, right gastric-caval and right gastric-azygos shunts. The PV was thinner than the aorta, sometimes to such an extent that could not even be visualized.

Acquired portosystemic collaterals. Visualization of the PV was often hindered by ascitic fluid and the cranially displaced intestines due to the small liver. If the PV was visible, its diameter was either smaller or larger compared to the aorta.



A B

Figure 1.

Plane-1, transverse section via one of the last right intercostal spaces with the dog in left lateral recumbency as the starting point of the systematic ultrasound examination of the portal system.

A. Gray scale image of the liver of a healthy male adult beagle. From dorsal to ventral the cross-sections of the aorta (AO), caudal vena cava (CVC) and portal vein (PV) are seen. The cross-sectional areas of the three vessels are approximately equal.

B. Color Doppler image of the liver of a 5-month-old female Labrador retriever with a persistent ductus venosus. In the color box from dorsal to ventral the cross sections of the caudal vena cava (CVC), hepatic artery (HA) and portal vein (PV) are seen. The hepatic artery is wider than in a healthy dog. The flow velocity in the HA is faster the than that of the CVC and PV.

Findings in plane-2

Normal anatomy. The right portal branch was consistently found as a well-defined vein originating from the PV and running dorsolaterally to the right while becoming gradually thinner due to ramification (**Fig 2A**).

Congenital intrahepatic porto-caval shunts. Each right-sided intrahepatic CPSS originated from the right portal branch as its direct continuation. The right portal branch was wider than normal and did not get thinner towards the periphery. The first segment of the shunt consistently ran dorsolaterally to the right, like a normal right portal branch, but then instead of ramification, it turned medially to enter the CVC (**Figs 2B, 2C**). With little transducer-manipulation the entire course of the shunt could be traced to its caval termination.

Left-sided intrahepatic CPSS was suspected, when the findings described in plane-1 were compatible with an intrahepatic CPSS, and the right portal branch was absent or very thin (**Fig 2D**). Often a hepatic artery branch was found at the place where the right portal branch was expected (**Fig 2E**). On B-mode images this artery looked like a very thin right portal branch, but color Doppler revealed much faster flow in it than in a portal branch could be expected, moreover pulsed-wave Doppler mode showed low-resistance arterial spectrum in it confirming that this thin vessel was actually a hepatic artery branch, which coursed next to the hypoperfused and hypoplastic right portal branch.

Congenital extrahepatic spleno-caval, spleno-azygos, right gastric-caval and right gastric-azygos shunts. The right portal branch as well as the PV itself were usually so thin that they could not be visualized either on B-mode or on color Doppler images (**Fig 2F**). When they were wide enough to be seen (exceptionally), the blood flow velocity was either undetectably slow or very slow hepatopetal in them.

Acquired portosystemic collaterals. The right portal branch could only be exceptionally visualized because of the ascites and the small size of the liver. When it was visualized, it was either thinner or wider than normal, but showed normal arborization and undetectably slow flow (i.e. no flow with the lowest possible PRF-setting). When it was thin, then the PV was also thin; when it was wide, then the PV was also wide.

Findings in plane-3

Normal anatomy and congenital intrahepatic porto-caval shunts. Immediately caudal to the portal bifurcation, the gastroduodenal vein could occasionally be imaged as it entered the ventral aspect of the PV from the right; however the gas-filled descending duodenum often hindered its visualization. Sliding the transducer further caudally, the splenic vein could be seen entering the left aspect of the PV from ventrolateral direction.

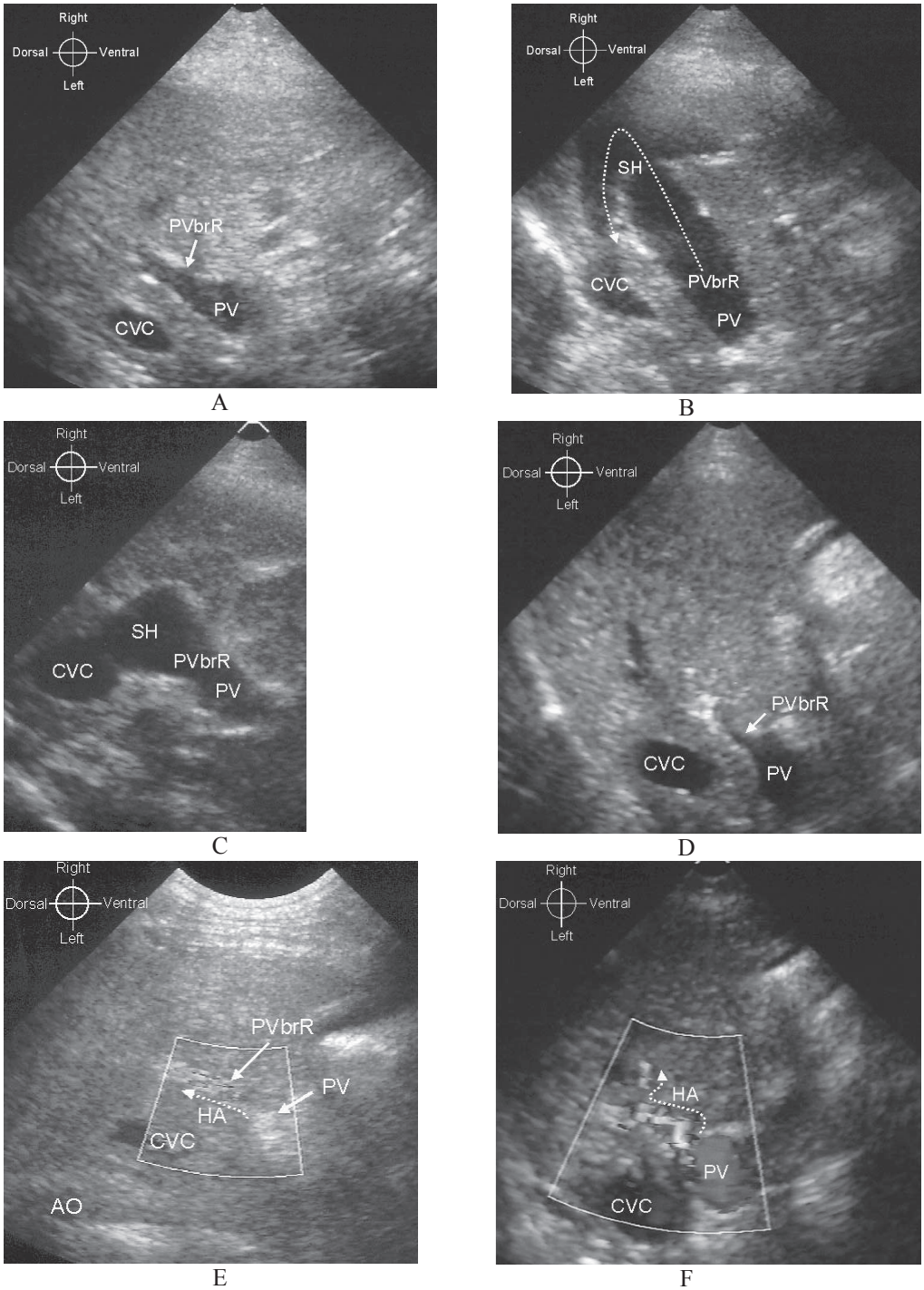


Figure 2.
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Figure 2.

Plane-2, transverse section via a right intercostal space with the dog in left lateral recumbency to image the right portal branch, cranial to plane-1.

A. Normal right portal branch (PVbrR) at the point of its origin from the portal vein (PV) in a healthy adult male beagle. The right portal branch is thinner than the PV and becomes gradually thinner towards the periphery due to ramification. The right portal branch runs dorsolaterally and to the right. CVC caudal vena cava.

B. Right divisional congenital intrahepatic portocaval shunt. Gray scale ultrasound image of an intrahepatic porto-caval shunt that originates from the right portal branch (PVbrR) in a 5.5-month-old male Labrador retriever. In this single image the direct connection between the right portal branch and the caudal vena cava (CVC) can be appreciated. The right portal branch is as wide as the portal vein (PV) and remains wide towards the periphery.

C. Central divisional congenital porto-caval shunt. Gray scale ultrasound image of a 4-month-old male Irish wolfhound mixed breed dog with a short intrahepatic porto-caval shunt (SH) that originates from the right portal branch (PVbrR). Compare the length of the shunt with the one shown in Figure 2B! CVC caudal vena cava, PV portal vein.

D. Gray scale ultrasound image of the right portal branch (PVbrR) in a 5.5-month-old male Bernese mountain dog with an intrahepatic porto-caval shunt that originates from the left portal branch (persistent ductus venosus). The portal vein (PV) has similar diameter to that of the caudal vena cava (CVC), however the right portal branch is very thin.

E. Color Doppler image of the right portal branch in a 1.5-year-old cairn terrier with a congenital extrahepatic spleno-caval shunt. The caudal vena cava (CVC) is well recognizable, however at the place of the portal vein (PV) and right portal branch (PVbrR), only the walls of these collapsed vessels can be seen as hyperechoic structures. The color signs originate from the adjacent hepatic artery branch (HA hepatic artery branch of the right lateral liver lobe). AO aorta. (Full **color** illustration on page 180.)

F. Undetectable right portal branch in plane-2 of a dog with a left divisional congenital intrahepatic portocaval shunt. An artery is seen at the place where the right portal branch is expected. The localization and course of this vessel is compatible with a right portal branch, but the flow velocity is very high in it (color aliasing). The hepatic artery and portal branches run normally adjacent to each other, but in this case the portal branch is undetectably thin. Dotted arrows indicate the direction of blood flow, CVC caudal vena cava, HA hepatic artery branch of the right lateral liver lobe, PV portal vein. (Full **color** illustration on page 180.)

Color Doppler mode revealed hepatopetal flow in the splenic vein. Slightly caudal to this point, the origins of the celiac and cranial mesenteric arteries from the aorta could be seen. Both arteries ran ventrally.

Congenital extrahepatic portosystemic shunts. The origin of the cranial loop of the right gastric-caval shunts were sometimes seen (**Fig 3A**), however the gastrointestinal gas often hindered their visualization. Therefore, plane-6 was used when a CPSS with a right gastric vein origin was suspected based on the findings in planes-1, -2 and -3. The point where the shunt entered the CVC could often be detected, but the course of the shunt-loops could not be visualized.

The whole length of spleno-caval shunts, and the origin of spleno-azygos shunts were always visualized. Spleno-caval CPSSs made a short loop on the left side of the PV and CVC. The direct connection was usually appreciated on B-mode images (**Fig 3B**), however occasionally, when the shunt did not appear on B-mode images because of the insufficient gray scale resolution, color Doppler mode was necessary to visualize the shunt.

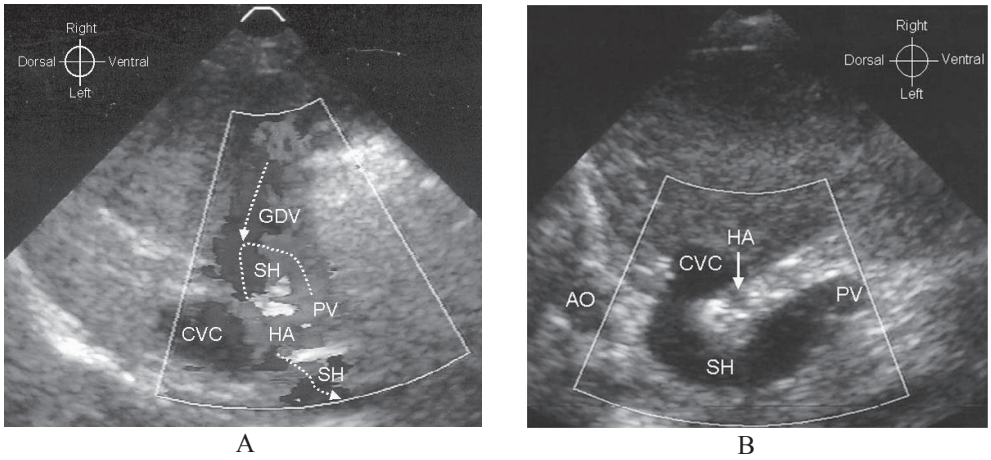


Figure 3.

Ultrasonograms of the two possible origins of canine congenital extrahepatic portosystemic shunts scanned from plane-3 with the dogs in left lateral recumbency.

A. Color Doppler image of the origin of a right gastric-caval shunt in a 10-month-old female Maltese dog. The gastroduodenal vein (GDV) seems to enter the shunt (SH) itself; in fact the red part of the shunt belongs to the gastroduodenal vein and the shunt originates at the point of the blue-red transition. The shunt drains not only the blood of the gastroduodenal vein, but also the blood of the portal vein (PV). The entire shunt cannot be visualized from this approach. Dotted arrows indicate the direction of blood flow, CVC caudal vena cava, HA hepatic artery. (**Color** version on page 180.)

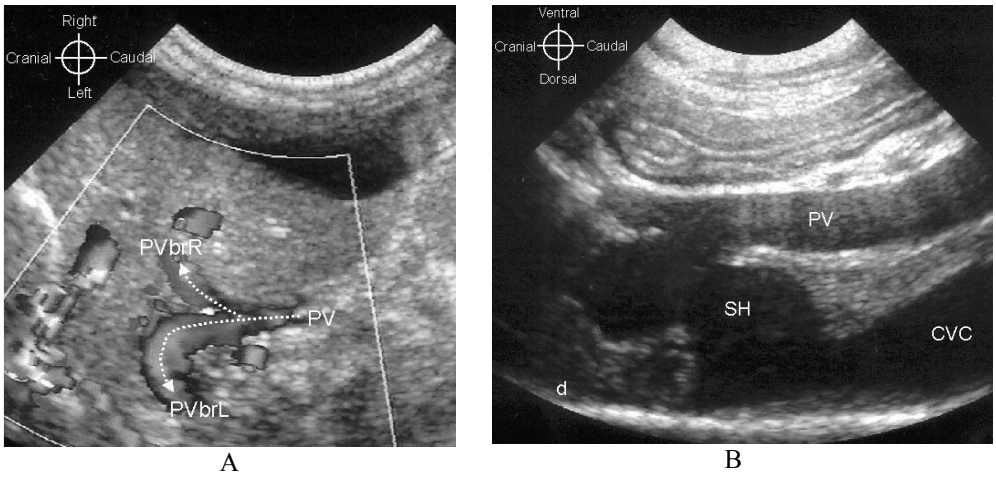
B. B-mode ultrasound image of a congenital extrahepatic spleno-caval shunt in a 3.5-month-old female Jack Russell terrier in plane-3. Cross sections of the aorta (AO), caudal vena cava (CVC) and portal vein (PV) are shown. Between the CVC and the PV the hepatic artery (HA) is seen. A short anomalous vein (SH) makes direct connection between the PV and the CVC on their left side. The diameters of the shunt and the PV are about equal. Note that not only the origin, but the entire shunt can be visualized in a single image from this approach.

Acquired portosystemic collaterals. The visualization of the PV was often difficult because of the presence of ascites and the cranially displaced intestines due to a small liver. When the PV was imaged, the PV had uniform diameter along its whole length. The origin of an APSC was occasionally found at the region where congenital extrahepatic spleno-caval shunts were expected to arise, i.e. an anomalous vein with hepatofugal flow.

Findings in plane-4

Normal anatomy. The splenic vein was seen to enter the PV from caudolateral direction from the left. Following the PV cranially, the portal bifurcation was seen with the wider left and the thinner right portal branch. Both branches became gradually thinner towards the periphery (**Fig 4A**).

Congenital intrahepatic porto-caval shunts. The PV looked similar to that of normal dogs. Following the PV cranially, an intrahepatic CPSS appeared as the direct continuation of the PV, and entered the CVC. Plane-4 did not allow the differentiation whether the intrahepatic CPSSs originated from the right or left portal branch, however in plane-2 the right- and central-divisional intrahepatic CPSSs could already be diagnosed and the left-divisional ones suspected. Plane-4 was only used to confirm the presence of left-divisional intrahepatic CPSSs by direct visualization of the porto-caval connection (**Fig 4B**). Since the intrahepatic CPSSs that originated from the left portal branch coursed adjacent to the diaphragm, plane-4 allowed their better visualization than plane-2 because the gas-containing intestinal loops could be pushed away with the transducer.



A B

Figure 4.
The portal bifurcation. (Full **color** illustration on page 180.)

A. Normal portal bifurcation in a 8-week-old mixed breed dog with hypoalbuminaemia. Color Doppler image of the portal vein (PV) at the hilus of the liver with the portal bifurcation. Note that the left portal branch (PVbrL) is wider than the right one (PVbrR) and they become gradually thinner towards the periphery. The image was made via the right flank with the dog in left lateral recumbency.

B. Intrahepatic porto-caval shunt that originates from the left portal branch in a 9-month-old hovawart. The portal trunk (PV) continuous via a wide and tortuous anomalous vessel (SH) and terminates in the caudal vena cava (CVC). The image was made in plane-5. d diaphragm

Congenital extrahepatic spleno-caval and spleno-azygos shunts. Using plane-4, the PV-segment cranial as well as caudal to the CPSS-origin could also be seen in addition to the shunt, and the direction of flow could be determined in them (**Fig 5**). In cases of spleno-caval CPSSs, the termination of the CPSS could be found with little transducer-manipulation by following the shunting vessel. In cases of spleno-azygos shunts, the shunt could be traced to the point where it entered the thorax through the diaphragm. Occasionally the PV-segment cranial to the point where the gastroduodenal vein entered the PV could also be imaged.

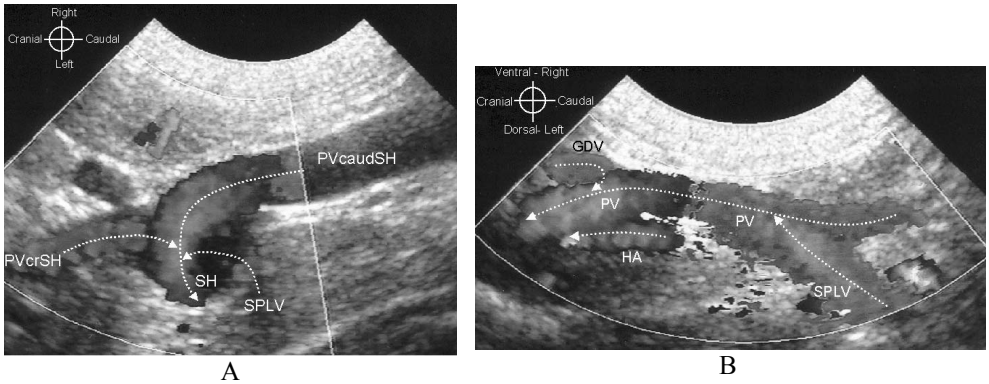


Figure 5.

Color Doppler ultrasound image of the portal vein in longitudinal section from plane-4. The transducer is placed longitudinally, caudal to the last right rib, with the dog in left lateral recumbency. Dotted arrows indicate the direction of blood flow.

A. Congenital extrahepatic spleno-caval shunt (SH). Cranial to the shunt-origin the direction of portal blood flow is hepatofugal. The portal vein cranial to the shunt-origin (PVcrSH) is thinner than the portal vein caudal to the shunt-origin (PVcaudSH). The shunt originates from the portal vein, very close to the point where the splenic vein (SPLV) enters the portal vein. (Full **color** illustration on page 180.)

B. Normal portal vein (PV) in a Yorkshire terrier. The diameter of the portal vein is uniform along its whole length.

GDV gastroduodenal vein, SPL splenic vein, HA hepatic artery. (Full **color** illustration on page 180.)

Congenital extrahepatic right gastric-caval and right gastric-azygos shunts. Occasionally the origin of the dilated right gastric vein was visualized using this view, but not the course of the cranial loop of the shunt. The caudal shunt-loop (when it was present) was regularly visualized, which originated at the same point as a congenital extrahepatic spleno-caval shunt, however it coursed cranially, unlike a spleno-caval shunt, which coursed dorsally. The PV cranial to the origin of the caudal shunt-loop was always seen, and the flow-direction was always hepatopetal in it. The anastomosis between the cranial and caudal loops of the shunt could never be visualized with ultrasound.

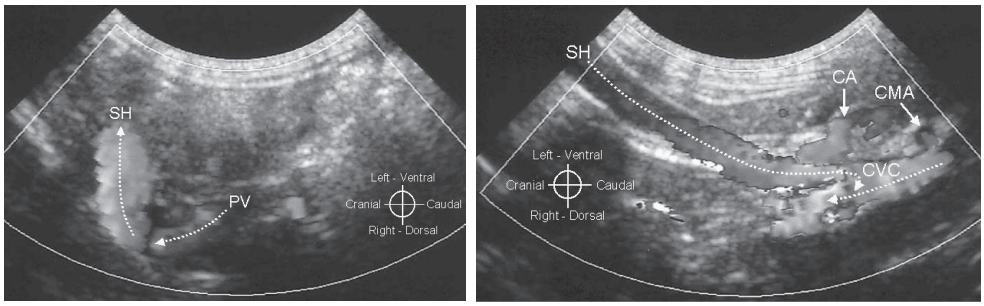
Acquired portosystemic collaterals. This view made the assessment of portal flow-velocity and of portal flow-direction possible. The origin of APSCs arising from the PV could also be seen at the region where congenital extrahepatic spleno-caval shunts arise. It was impossible to image the PV-segment that was cranial to the entering point of the gastroduodenal vein in any dog with APSCs.

Findings in plane-5

The findings were the same as described under plane-4, but in some cases plane-5 allowed better visualization of the shunt or of the PV (**Fig 4B**), and provided better incidence angles for Doppler studies.

Findings in plane-6

Congenital extrahepatic right gastric-caval or right gastric-azygos shunts. Usually when the PV was being searched, a large-caliber anomalous vein (i.e. the shunt) appeared just under the body wall, even before the PV was actually found. The diameter of this CPSS was comparable to that of the CVC. Following the shunt cranially it seemed to originate from the PV, at the hilus of the liver, making a 90° angle (**Fig 6A**). From its origin, the shunt was followed to its termination with or without the help of color Doppler mode (**Fig 6B**). The PV cranial to the shunt-origin could never be visualized.



A B

Figure 6.

Congenital extrahepatic right gastric-caval shunt, the cranial loop. Dotted arrows indicate the direction of flow. **A**. Color Doppler image of the origin of the cranial loop of a right gastric-caval shunt in a 6.5-month-old female Yorkshire terrier in right lateral recumbency, from plane-6. The shunt (SH) runs towards the left body wall making a 90° angle with the portal vein (PV). (Full **color** illustration on page 181.) **B**. The continuation of the shunt that is shown in Figure 6A (followed caudally). The right gastric-caval shunt (SH) terminates in the caudal vena cava (CVC). Note the large-caliber shunting vessel (SH) immediately under the left body wall. CA celiac artery, CMA cranial mesenteric artery. (Full **color** illustration on page 181.)

Normal anatomy, congenital intrahepatic porto-caval shunts, congenital extrahepatic spleno-caval and spleno-azygos shunts, acquired portosystemic collaterals. The large-caliber anomalous vein described above was absent. The right gastric vein is so thin that cannot be visualized ultrasonographically.

Findings in plane-7

Normal anatomy, congenital intra- and extrahepatic portosystemic shunts. Caudal to the left kidney 2 great vessels, namely the aorta and the CVC were seen; the aorta was located more to the left. The left gonadal vein could never be visualized. The left renal artery (occasionally double) ran adjacent to the left renal vein and could be recognized even on gray scale images by its smaller diameter and pulsation.

Acquired portosystemic collaterals. The dilated left gonadal vein could always be seen entering the left renal vein from caudal (**Fig 7**); except for the cases of tense ascites,

when not even the CVC could be visualized. When the left gonadal vein was very wide (**Fig 7A**), it appeared as a third great vessel on the left side of the aorta. Many small tortuous veins (portosystemic collaterals) could often, but not always be seen around the left renal vein (**Fig 7B**).

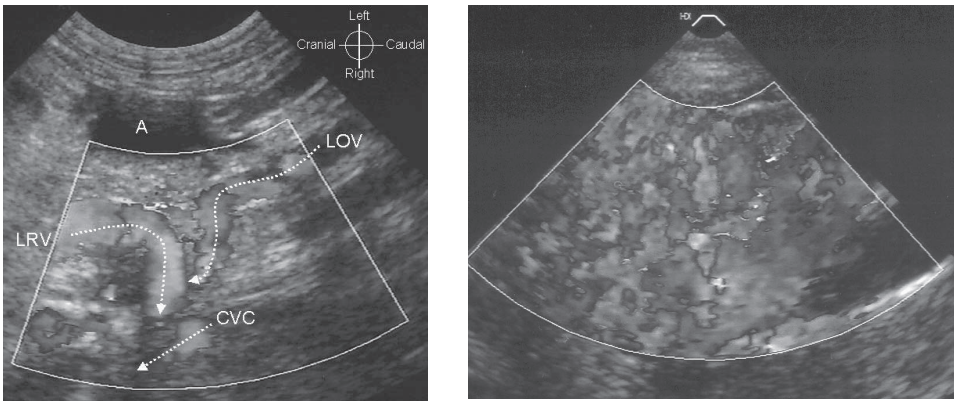


Figure 7.
Acquired portosystemic collaterals.
(Full **color** illustrations on page 181.)

A. Color Doppler image of a dilated left ovarian vein in a 6.5-year-old female Jack Russell terrier with sustained portal hypertension due to primary hypoplasia of the portal vein, from plane-7. Dotted arrows indicate the direction of blood flow. A ascites, CVC caudal vena cava, LOV left ovarian vein, LRV left renal vein.

B. Color Doppler image of several tortuous collateral veins caudal to the left kidney in a 5-month-old boxer with sustained portal hypertension due to primary hypoplasia of the portal vein, from plane-7.

Accuracy

The protocol described above allowed us to rule out portosystemic shunting non-invasively when hyperammonemia was the result of a laboratory error (2/90) or of a urea cycle enzyme deficiency (9/90). The abdominal ultrasonographic findings of the 90 hyperammonemic dogs are shown in **Table 1**. Acquired portosystemic collaterals were identified in all cases. None of the dogs with APSCs or with normal vascular anatomy underwent a laparotomy, whereas all the dogs with CPSS underwent a surgical shunt-ligation.

Table 1.
Ultrasonographic findings in 90 dogs with hyperammonemia

No portosystemic shunting:	**11**
Congenital portosystemic shunt:	**61**
Intrahepatic porto-caval:	*19*
Left divisional:	14
Central divisional:	2
Right divisional:	2
Right divisional double:	1
Extrahepatic portosystemic:	*42*
Spleno-caval:	24
Right gastric-caval:	9
Spleno-azygos:	8
Right gastric-azygos:	1
Acquired portosystemic collaterals:	**17**
Hepatic portal hypertension:	14
Arterioportal fistulae:	3
Other:	**1**
Arterio-portal fistula and intrahepatic porto-caval shunt:	1

In cases of CPSS, the shunting vessel was followed from its origin to its termination. One exception was a 5-month-old Weimaraner, in which the shunting vessel could not be visualized ultrasonographically, not even when the dog was sedated, though the ultrasonographic findings were compatible with a left-sided intrahepatic CPSS (i.e. wide PV and left portal branch, undetectable right portal branch, no extrahepatic CPSS or APSCs). Laparotomy revealed a left-sided intrahepatic CPSS. In cases of portal hypertension with APSCs, the direct connection between the portal and the systemic veins could only exceptionally be appreciated, however a dilated left gonadal vein was always seen.

In 4 dogs the ultrasonographic diagnosis was congenital extrahepatic spleno-caval shunt, whereas during surgery the termination of the shunt could not be visualized in the abdomen, hence they were reported to be congenital extrahepatic spleno-azygos shunts. In every other CPSS-cases the ultrasonographic diagnosis was in agreement with the surgical findings.

Discussion

Previous studies that aimed ultrasonographic differentiation of CPSSs from other diseases reported 74-95 % sensitivity and 57-100 % specificity.[2,13,14] The reason why not each extrahepatic CPSS was recognized could be that in two studies only a right intercostal approach was used. As we showed, the congenital extrahepatic right gastric-caval shunts cannot be readily diagnosed from this view, because the visualization of the cranial shunt-loop requires a right lateral recumbency, whereas the caudal shunt-loop can only be seen

via the right flank in a longitudinal section. The possible explanation why extra- and intrahepatic CPSSs were mistaken in these studies could be that the evaluation of the abdominal vasculature was not systematic and the right portal branch was not regularly imaged. Indeed, congenital extrahepatic spleno-caval shunts seem to be surrounded by liver when they are scanned via a right intercostal approach. Our systematic approach allowed us to diagnose both CPSSs and APSCs by ultrasound with 100 % sensitivity, 100 % specificity and 100 % accuracy. We did not consider the case of the Weimaraner false negative because though the direct connection of the left portal branch and CVC could not be visualized ultrasonographically, the morphology of the abdominal vessels was compatible with a left sided intrahepatic CPSS. In addition to the use of our scanning protocol, a high accuracy could only be achieved with the use of a high quality ultrasound machine with an excellent gray scale resolution. Using color Doppler is not essential, however it greatly facilitates the diagnosis of abdominal vascular diseases, especially if the ultrasound system is equipped with a sensitive color Doppler mode and the sonographer is familiar with the Doppler techniques.

Accurate recognition of CPSSs by ultrasound is only possible, if the anomalous vein is followed from its origin to its termination. Finding the point where a CPSS enters the CVC may be easier than finding the origin of a CPSS,[14] however finding a vein that enters the CVC does not mean that it is a CPSS because several other veins enter the CVC. If the origin and the course of a suspected CPSS is not visualized, a normal right renal or phrenicoabdominal vein could be mistaken with a CPSS. When a vein that is suspected to be a CPSS is found to enter the CVC, it has to be traced from its termination to its origin, i.e. to the PV. In contrast, when a vein that originates from the PV or from a portal tributary displays hepatofugal flow, it is surely an extrahepatic CPSS or an APSC, even without knowing the point of its termination because in normal animals veins only enter the PV and do not originate from it.[13]

In our opinion, technically the most difficult type of CPSS to find and visualize is the left divisional intrahepatic CPSS because it runs very close to the diaphragm. Therefore its visualization via the intercostal spaces is usually impossible, whereas its scanning via the right flank requires hard transducer-pressure because of the cranially displaced intestines due to the small liver, which causes discomfort to the dog, hence they do not usually stay still making the examination even more difficult.

Diagnosing APSCs ultrasonographically requires a different approach compared to that of the CPSSs because the collateral veins only occasionally arise directly from the PV, moreover they are thin and tortuous and most of the times are hidden among the intestines. Therefore, their origins and courses can only exceptionally be revealed. The dilated left gonadal vein, i.e. the termination of the spleno-renal collaterals, however was found to be a highly specific and sensitive indicator of APSCs,[16] and its ultrasonographic visualization is simple (**Chapter 4**).

In sum, ultrasonography is a highly sensitive and specific, non-invasive diagnostic method to diagnose or exclude CPSSs and APSCs in dogs. Excluding portosystemic shunting is not only based on the fact that no anomalous veins could be found, but also on the fact that the abdominal vessels of a dog with normal vascular anatomy look different from those of a dog with portosystemic shunting. The 4 sites that always need to be imaged when a portal vein anomaly is suspected are (1) the right portal branch, (2) the left gonadal vein, (3) the portal vein at the level of the celiac artery (splenic vein) and (4) the portal vein immediately caudal to the portal bifurcation (right gastric vein and gastroduodenal vein).

Acknowledgements

Dr Szatmári was supported by the Hungarian State Eötvös Scholarship. Mr Aart van der Woude helped in preparing the illustrations. This paper was presented in part at the WSAVA-FECAVA-AVEPA veterinary congress in Granada, Spain, October, 2002.

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

Journal of the American Veterinary Medical Association
2003;222:1086-1092.

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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.

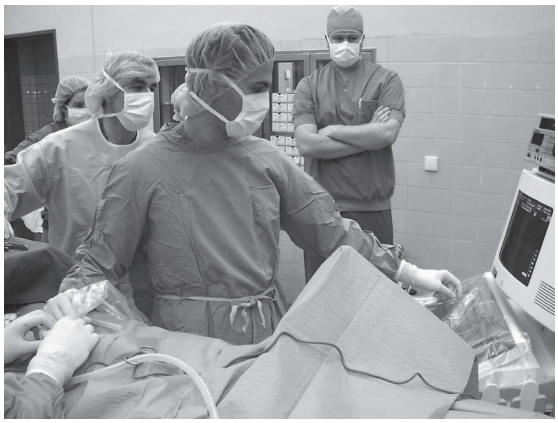


Figure 1.
Intraoperative ultrasonography during a surgical attenuation of a congenital portosystemic shunt.

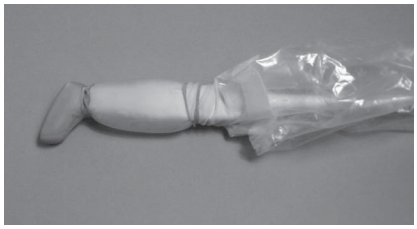


Figure 2.
An intraoperative linear array transducer (5-10 MHz) with sterile cover.

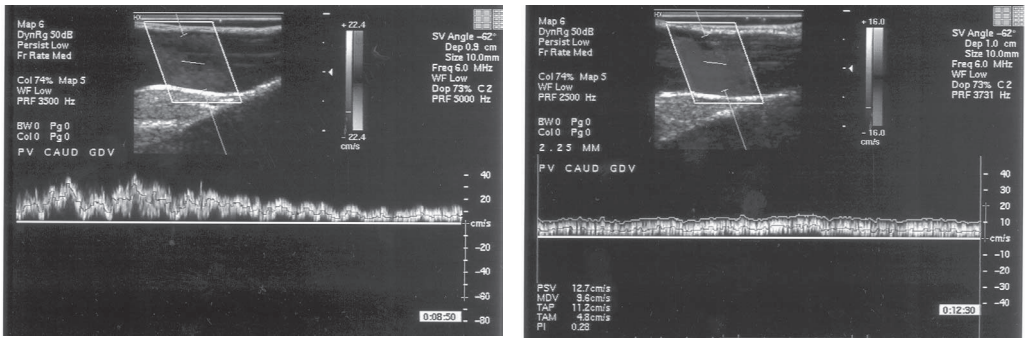
A midline celiotomy was performed, and all organs were left in the abdominal cavity. The descending duodenum was temporarily retracted toward the midline to expose the portal vein and was then released. The ultrasound transducer was placed directly on the trunk of the portal vein between the splenic 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.



A B

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

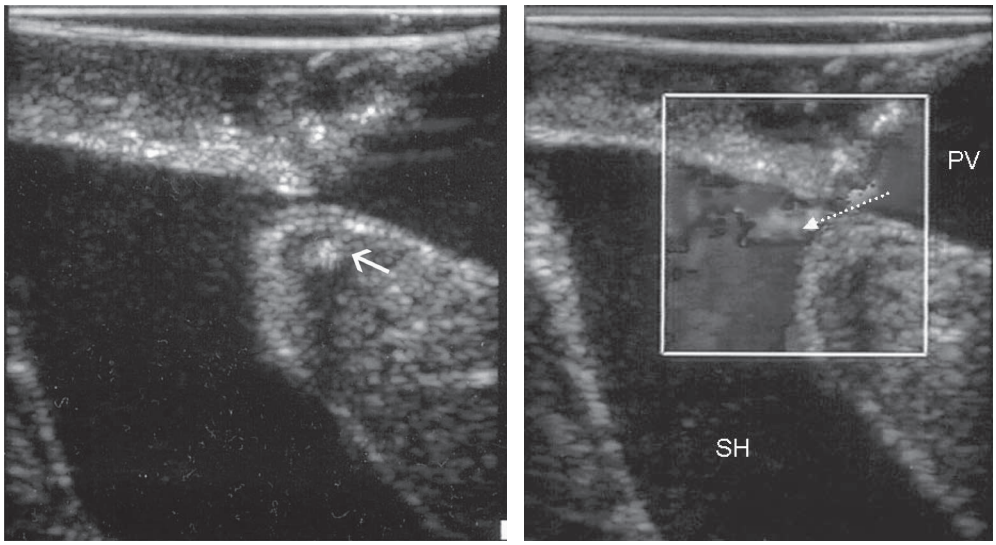
Nine client-owned dogs underwent surgical attenuation of an intrahepatic portosystemic shunt at the Utrecht University Clinic for Companion Animals during 2001 with this technique. Results for the first 2 are not included in the present report, because the

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.



A B

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.

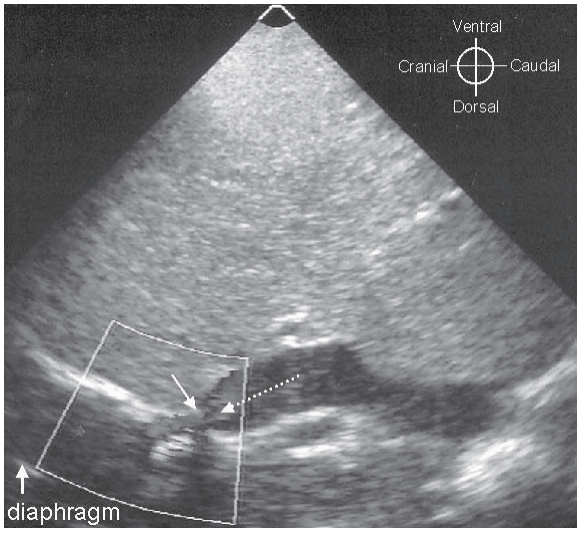
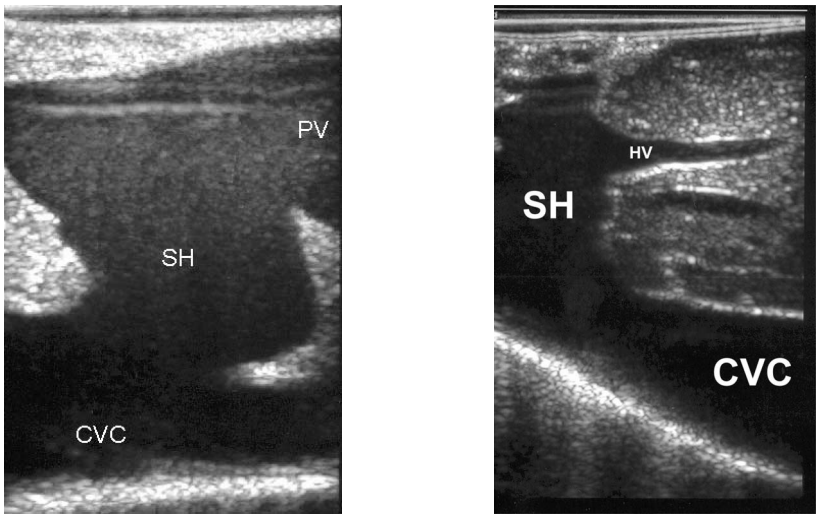


Figure 5.
Transabdominal color Doppler ultrasonogram of a partially ligated left-divisional intrahepatic portocaval shunt 1 month after surgery. The narrowing of the vessel caused by the ligature is clearly seen (arrow). The stenosis casued by the ligature results in a high flow velocity (poststenotic 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.



A B

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.

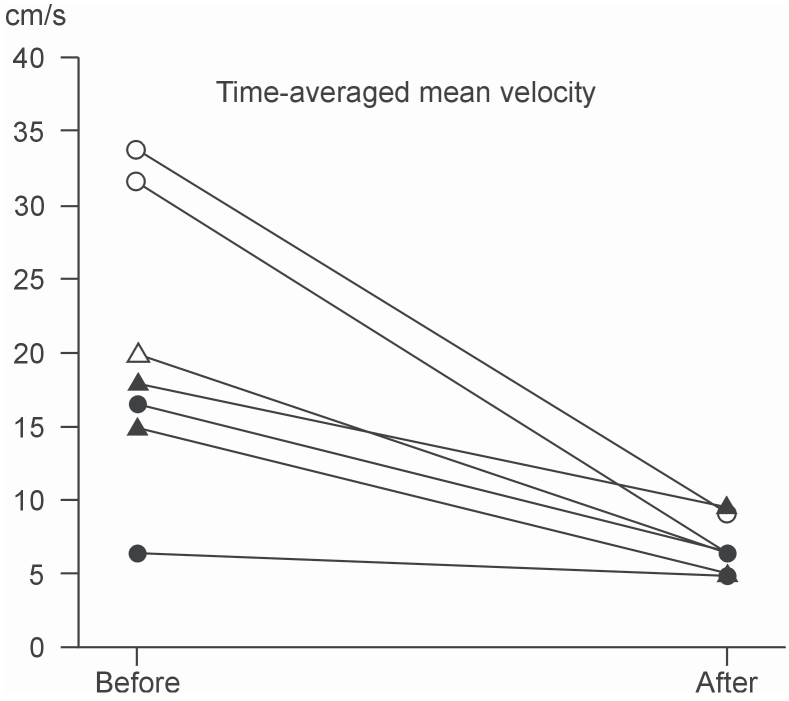


Figure 7.

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.

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CHAPTER 7

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

based on the article by

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Journal of the American Veterinary Medical Association
2004;224:395-402.

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Summary

Intraoperative Doppler ultrasonographic examination of the portal vein was performed in 17 dogs with single congenital extrahepatic portosystemic shunts before and after shunt ligation. Hepatofugal portal flow was often detected before shunt ligation in the portal segment between the shunt origin and the entering point of the gastroduodenal vein. Shunt ligation resulted in hepatopetal flow in this portal segment and the shunt, with favorable outcome. If severe hypoplasia of the portal branches did not allow hepatofugal flow to become hepatopetal flow, poor outcome resulted.

We recommend that the largest possible shunt diameter that ensures hepatopetal flow in the shunt and the entire portal vein should be the goal of ligation. Regardless of these flow directions, hepatopetal portal flow caudal to the shunt should always be maintained with a minimum time-averaged mean velocity of 3 cm/s. Increase of the portal congestion index > 3.5 times should be avoided.

Structured abstract

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 post-ligation 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 postoperatively 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 towards 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.

Introduction

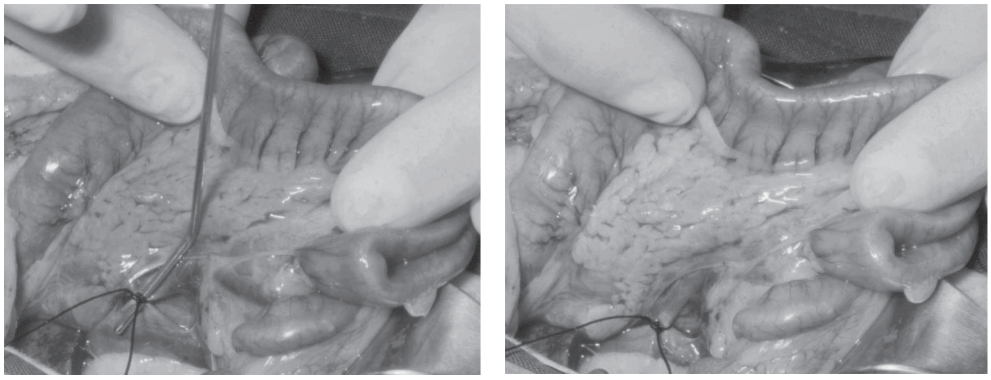
Portosystemic shunts are abnormal macroscopic vascular connections that allow the portal blood to directly enter the systemic venous circulation, bypassing the hepatic sinusoids, and can be classified according to their anatomy as intra- or extrahepatic.[1-9] An extrahepatic portosystemic shunt is thought 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] post-ligation 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 post-ligation 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 post-ligation portal hypertension that is acceptable (**Fig 1**).[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]

Several years ago ameroid constrictors were 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 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]



A B

Figure 1.
Surgical ligation of a congenital extahepatic spleno-caval shunt.

A. Completely closure of the shunt causes cyanosis of the descending duodenum and the pancreas (sign of portal hypertension). The metal rod has a known diameter. (Full **color** illustration on page 182.)

B. After removing the rod, the colors of the descending duodenum and the pancreas are normal again. The smallest diameter of the shunt equals to the diameter of the rod. (Full **color** illustration on page 182.)

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] have 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 (Ethibond Excel 2/0, Ethicon, Johnson & Johnson Intl, Hamburg, Germany) by a single surgeon (FJvS) by use of a reported technique.[8] After premedication {0.3 ml/kg [0.135 ml/kg] from the combination of droperidol and fentanyl, IM; 1 ml contains 2.50 mg droperidol and 0.05 mg fentanyl (Thalamonal, Janssen-Cilag B.V., Tilburg, The Netherlands)}, 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 (ATL HDI 3000, Philips Medical Systems, Advanced Technical Laboratories Ultrasound) was used, equipped with a 26-mm-long, 5- to 10-MHz intraoperative linear-array transducer. Both the keyboard and the transducer were covered with sterile material (Surgi-Tip Intraoperative Transducer Cover & 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.

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 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, the portal vein segment cranial and the portal vein segment caudal to the shunt origin (**Fig 2**). 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 (HighQ ATL HDI 3000), and the mean of 3 measurements was calculated.

After automatic analysis of at least 3 good-quality Doppler spectra, the transducer was rotated 90^{o} 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 (High Q of the ATL HDI 3000). The congestion index of the portal vein was calculated from the cross-sectional area divided by the time-averaged mean velocity.[28]

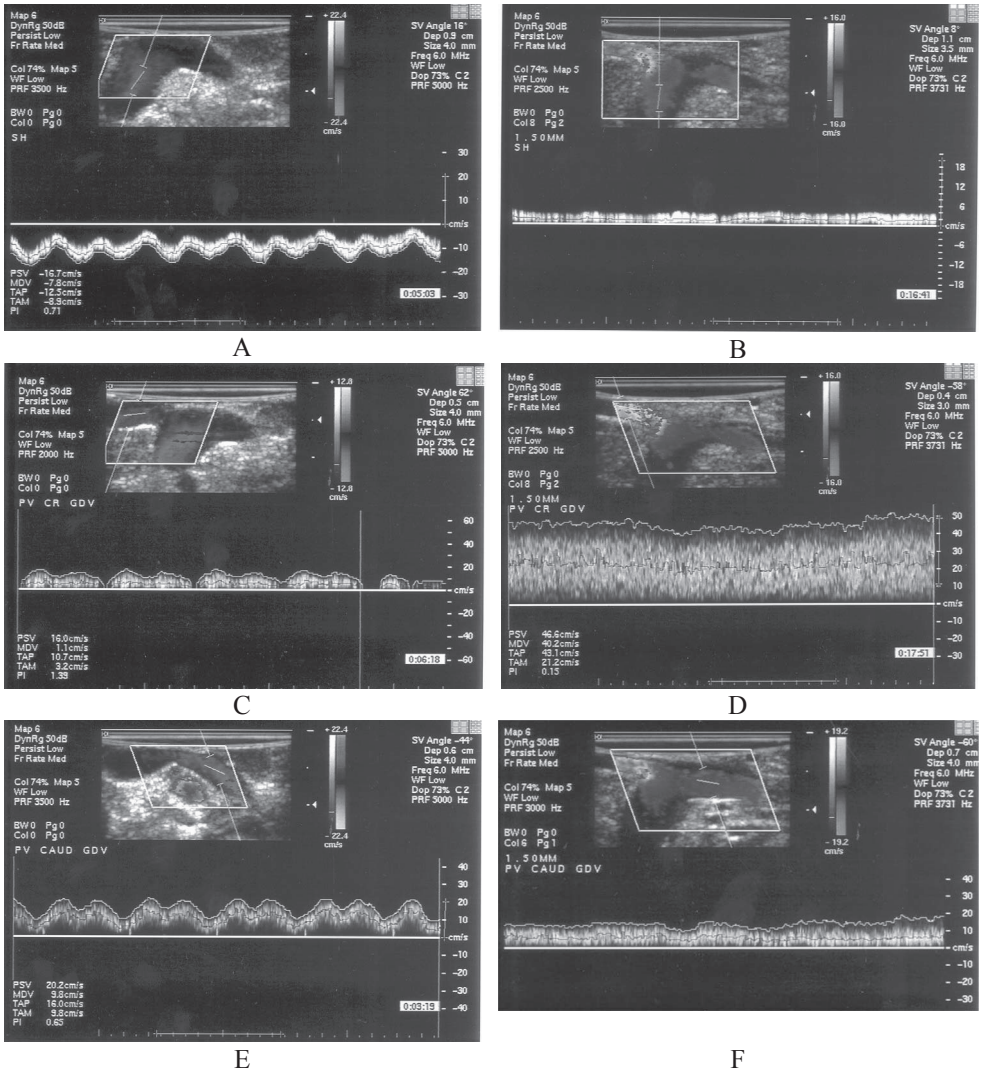


Figure 2.

Intraoperative pulsed-wave Doppler ultrasonographic tracings *before* (A,C,E) and *after* (B,D,F) partial ligation of a congenital extrahepatic spleno-caval shunt in a dog with excellent clinical outcome.

A, B In the shunt the flow direction becomes hepatopetal and the variability of the spectrum becomes damped.

C, D In the portal vein cranial to the shunt-origin the flow becomes hepatopetal and very fast.

E, F In the portal vein caudal to the shunt-origin the velocity decreases and the variability becomes damped subsequent to shunt attenuation.

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 in the shunt were revealed with color Doppler ultrasonography. **Acquired portosystemic collaterals (APSCs)** were diagnosed ultrasonographically, when a wide left gonadal vein was found (**Chapter 5**), as a result of splenorenal collaterals entering the left renal vein from 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 the 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 cause of poor, fair, and fatal outcomes is either 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 for the outcome, a graph was made (SPSS 11.0 for Windows statistical program, SPSS Inc., Chicago, Illinois) 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 whose CPSSs had been attenuated. Dogs with fair and fatal outcomes 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 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 and the thrombus was attributed to an exaggerated shunt closure. 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 pre-attenuation time-averaged mean portal velocity measured cranial to the shunt origin (v_{PVcrSH}) from the pre-attenuation time-averaged mean

portal velocity measured caudal to the shunt origin ($v_{PVcaudSH}$). 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

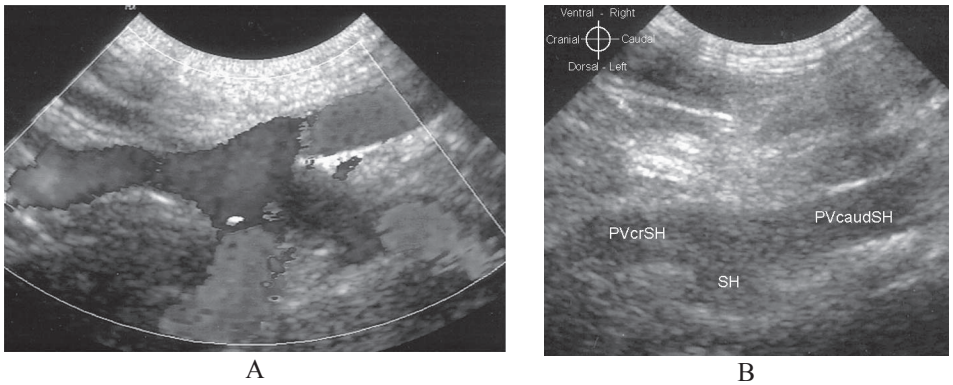
Mean duration of ultrasonography during each surgery was 22 minutes (range, 14 to 35 minutes). Mean duration between the pre- and post-ligation ultrasonographic measurements was 37 minutes (range, 16 to 60 minutes).

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.



A B

Figure 3.

A. Preoperative color Doppler ultrasound image of the congenital extrahepatic spleno-caval shunt that is shown on Fig 3B.

B. Gray scale ultrasonogram of a portal vein thrombosis 2 days after a complete surgical ligation of a congenital extrahepatic portosystemic shunt. The shunt (SH) and the portal vein cranial (PVcrSH) as well as caudal (PVcaudSH) to the shunt-origin are filled with a thrombus.

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, **Fig 3**), 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.

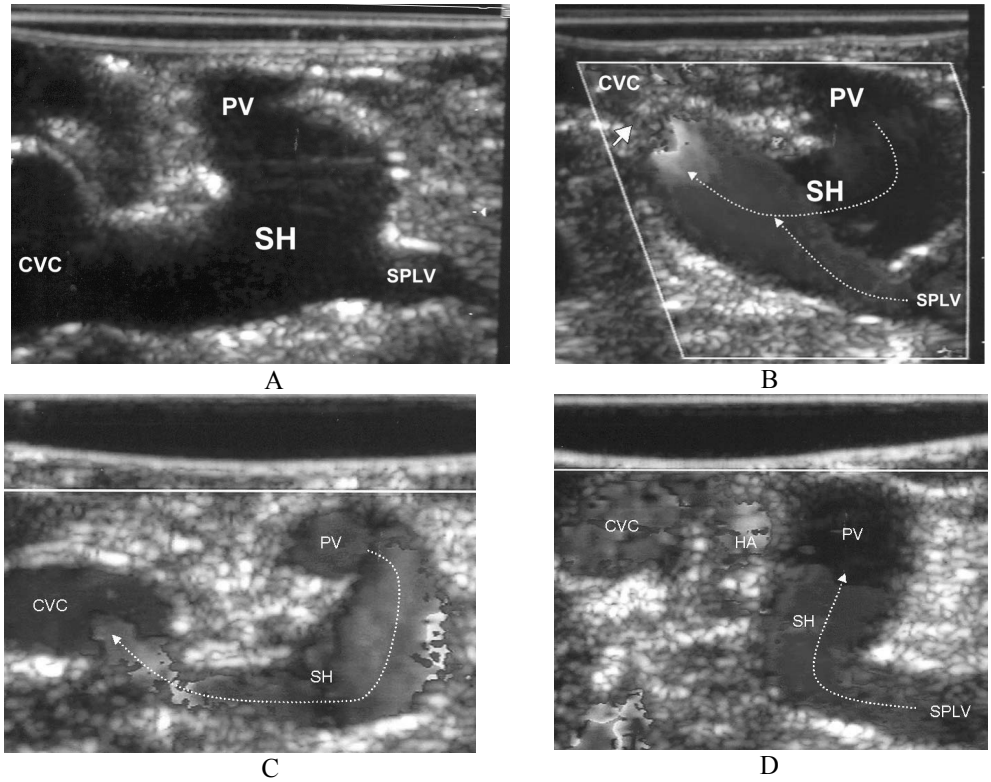


Figure 4.

Intraoperative ultrasound images of congenital extrahepatic spleno-caval portocaval shunts. Transverse sections *before* (A, C) and *after* partial ligation (B, D) in two dogs: one (A, B) with fair outcome and the other one (C, D) with excellent outcome. The dotted arrows indicate the direction of blood flow. SH shunt, PV portal vein, CVC caudal vena cava, SPLV splenic vein, HA hepatic artery. (Full **color** illustrations on page 182.)

A. Gray scale ultrasonogram shows the anatomy of the shunt. Figure 4B shows the postoperative situation of this dog.

B. Color Doppler image shows a functional shunt after a partial shunt ligation. The arrow indicates the ligature. The shunt remained functional because of a coinciding portal vein hypoplasia.

C. Color Doppler ultrasonogram before ligation the blood from the portal vein (PV) flows via the shunt (SH) to the caudal vena cava (CVC). Figure 4D shows the postoperative situation of this dog.

D. Partial ligation of the shunt (SH) adjacent to the caudal vena cava (CVC) results in a hepatopetal shunt flow because the splenic blood (SPLV) finds lower resistance to flow towards the PV than towards the attenuated shunt segment.

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

Portal hemodynamics cranial to the shunt origin before shunt ligation. In 4 of the 5 dogs that had 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 (**Figs 5, 7**). 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 5**).

Portal hemodynamics cranial to the shunt origin after shunt ligation. After attenuation, the pre-attenuation hepatopetal flow direction remained unchanged, but increased in velocity. 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 (**Figs 5, 7**). In the 2 dogs in which portal flow-directions remained hepatofugal, the shunt-flow also remained hepatofugal. These 2 dogs developed APSCs postoperatively and hyperammonemia persisted; ultrasound-guided liver biopsies 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.

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 (**Figs 5, 8**). 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.

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.

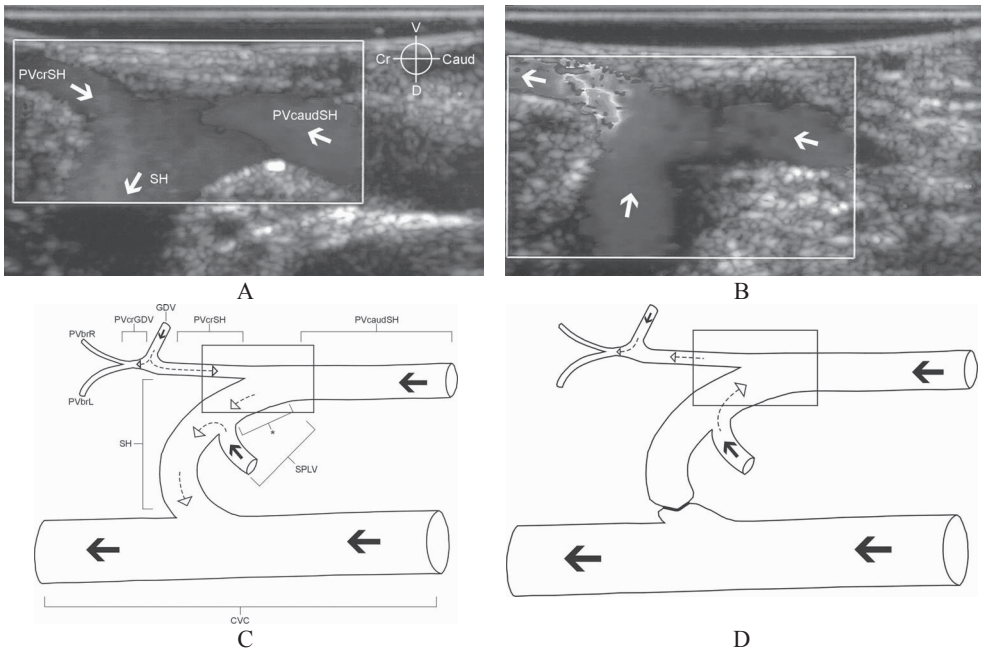


Figure 5.

Intraoperative color Doppler ultrasound images (A,B) and the corresponding schematic drawings (C,D) of a congenital extrahepatic spleno-caval shunt in a dog *before* (A,C) and *after* (B,D) partial shunt-ligation. The framed regions of the drawings correspond with the ultrasound images.

Red color represents flow towards the transducer and blue away from the transducer. Arrows indicate flow-directions. Cr cranial, Caud caudal, V ventral, D dorsal, SH shunt, PVcrSH portal vein cranial to the shunt-origin, PVcaudSH portal vein caudal to the shunt-origin, PVbrL & PVbrR left and right portal branches, GDV gastroduodenal vein, PVcrGDV portal vein cranial to the point where the gastroduodenal vein enters the portal vein. Black arrows indicate the flow that do not change directions subsequent to shunt-attenuation, open interrupted arrows indicate the flow that may change. (Full **color** illustrations on page 182.)

A. Before shunt attenuation the portal flow is hepatofugal cranial to the shunt. The portal vein cranial to the shunt-origin is narrower than caudal to it.

B. After partial shunt attenuation the flow-direction became hepatopetal both in the shunt and in the portal segment cranial to the shunt-origin. The portal flow velocity caudal to the shunt-origin is decreased.

C. The blood from the gastroduodenal vein (GDV) is responsible for the hepatofugal portal flow between the shunt-origin and GDV. The splenic vein (SPLV) seems to enter the shunt, however in reality the shunt originates from the splenic vein, and the shunting blood from the portal vein results in dilation of this segment of the splenic vein and generates hepatofugal flow in it (*).

D. Shunt-attenuation made the resistance towards the shunt higher than towards the portal branches. This results in hepatopetal flow both in the shunt and in the portal vein cranial to the shunt-origin. A portion of the splenic venous blood remains to flow towards the CVC via the narrowed segment.

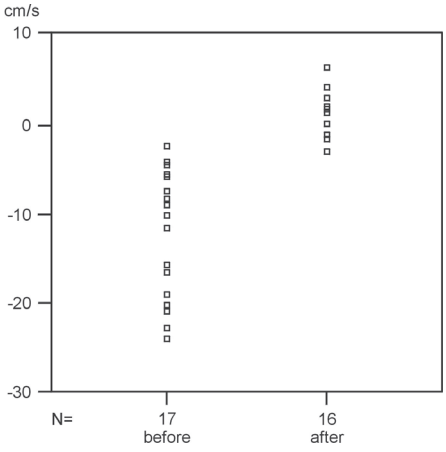


Figure 6. Comparison of the time-averaged mean velocities in the *shunt* measured adjacent to the portal vein before (n = 17) and after shunt attenuation (n=16). Hepatopetal flow-direction is coded with positive signs, hepatofugal flow-directions with negative signs. Every dog had hepatofugal flow before shunt attenuation; this turned to hepatopetal in all but four dogs after shunt attenuation. When it remained hepatofugal, the velocity decreased.

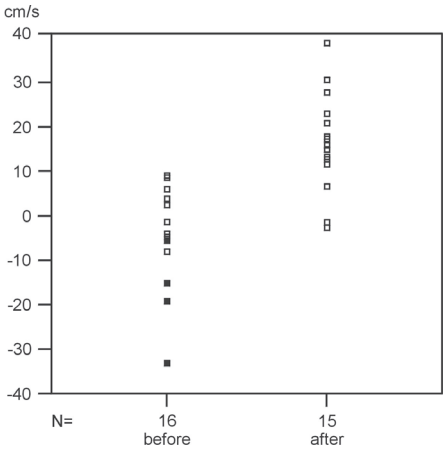


Figure 7. Comparison of the time-averaged mean velocities in the *portal vein cranial to the shunt* origin (PVcrSH) before (n = 17) and after shunt attenuation (n=16). Hepatopetal flow-direction is coded with positive signs, hepatofugal flow-directions with negative signs. Several dogs had hepatofugal flow before shunt attenuation; this turned to hepatopetal after shunt attenuation in all but two dogs. The pre-ligation values of the two dogs that developed acquired portosystemic collaterals one month after surgery, the dog with aplasia of the cranial portal vein, and the dog that died suddenly are in the high range of this group (black squares).

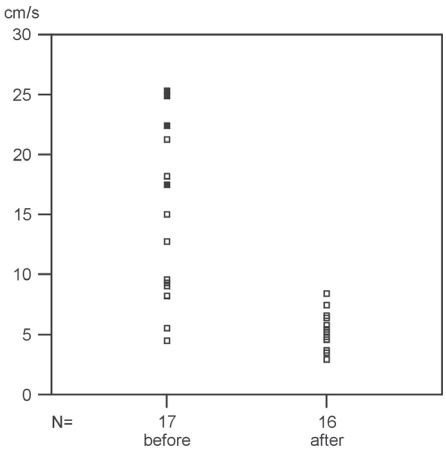


Figure 8. Comparison of the time-averaged mean velocities in the *portal vein caudal to the shunt* (PVcaudSH) before (n = 17) and after shunt attenuation (n=16). Hepatopetal flow-direction is coded with positive signs, hepatofugal flow-directions with negative signs. Every dog had hepatopetal flow-direction before shunt attenuation; this decreased after shunt attenuation. Note that the pre-attenuation wide range of velocities converged to a narrow range subsequent to attenuation. The pre-ligation values of the two dogs that developed acquired portosystemic collaterals one month after surgery and of the dog with aplasia of the cranial portal vein as well as the one that died suddenly are in the high range of the group (black squares).

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 9**).

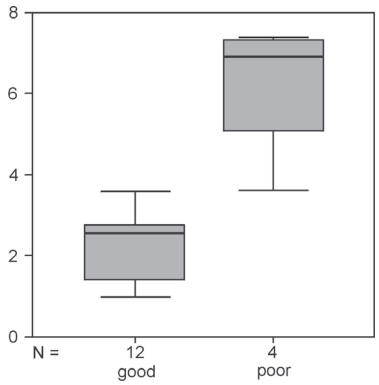


Figure 9. Comparison of the magnitude of increase of the portal congestion indices (CI) caudal to the shunt (PVcaudSH) in dogs with "good" outcomes (n = 12) and in the dogs with "poor" outcomes, i.e. the ones that died, was euthanized because of portal thrombosis or developed chronic portal hypertension (n = 4). The magnitude of increase was calculated by dividing the post-attenuation CI by the pre-attenuation CI. Complications occurred when the magnitude of increase was more than 3.6-fold. The two graphs show the median, the 25 and 75 percentiles and the range of each group.

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 after shunt-attenuation. 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 post-ligation 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, liver biopsy specimens obtained 1 month after surgery revealed portal vein hypoplasia. In these dogs and in 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 (**Figs 7, 8**); but there were overlaps among the values of the group with good outcomes and those of the group with poor outcomes. However, if differential portal velocity (DPV) was > +28, complications developed (formation of

APSCs, inability to attenuate the shunt because of aplasia of the cranial portal vein, sudden death), and if DPV was < +28, the outcome was good (**Fig 10**).

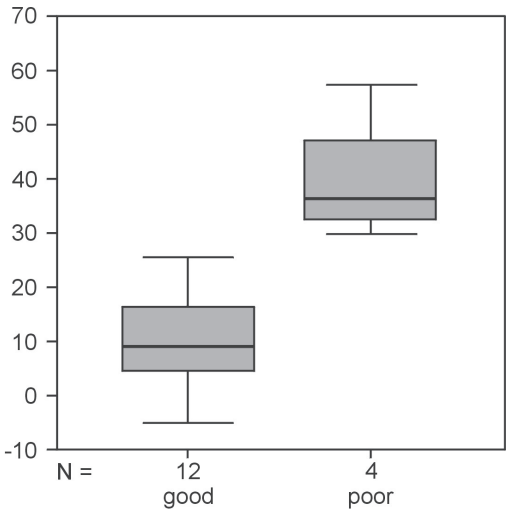


Figure 10. Comparison of the "differential portal velocities" (DPVs) in the dogs with "good" outcomes (n = 12) to the DPVs of the dogs with "poor outcomes", i.e. the ones that had severe portal vein hypoplasia (developed acquired portosystemic collaterals) or aplasia, or died after surgery (n = 4). The dog with portal vein thrombosis is included in the group of good outcome because thrombosis is believed to be a iatrogenic complication due to exaggerated shunt-closure without any underlying disease, therefore this complication is unpredictable from the pre-attenuation values. Hepatopetal flow is indicated by a positive sign, hepatofugal flow by a negative sign. Complications may be predicted before shunt attenuation, when the DPV is above +28 cm/s. The graphs show the median, the 25 and 75 percentiles and the range of the two groups.

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 post-attenuation flow in the shunting vessel has not been previously described to our knowledge, presumably because scintigraphy or portography have been predominantly used for follow-up studies[6,9] and these techniques are 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 because 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 spleno-caval 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 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 pre-attenuation 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 pre-attenuation 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 post-attenuation 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 post-attenuation 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 non-invasive and quantitative. The depth of anesthesia was unlikely to affect our measurements, since the cardiovascular state of the patients was monitored continuously, and the mean arterial blood pressure was maintained above 60 mmHg.

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 post-ligation portal vein thrombosis and of the dogs that have been described with postligation portal vein thrombosis in the literature (i.e. 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.

Pre-attenuation 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 pre-ligation portal flow velocities that were associated with good outcomes could be that if the pre-ligation velocity is already low, the magnitude of decrease in post-ligation velocity (as well as increase in pressure) is small, requiring only mild adaptation from the portal system and reducing the risk of complications. The differential portal velocity (DPV) would be an especially useful variable if it was 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]

We concluded that 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 in 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 more than 3.5-times

increase in the portal congestion index caudal to the shunt should be prevented. Because these recommended values and the threshold differential portal velocity (DPV) value were derived retrospectively from our case series, their overall validity should be tested prospectively.

In summary, intraoperative ultrasonography can reveal the hemodynamic features of the portal and shunt-flow quickly, non-invasively and without radiation exposure, next to the operating table. The portal flow-directions have showed better relationship with the clinical outcomes than any other variables that have been previously studied.[1,6-12,18,24,34-36] Surgical ligation of extrahepatic CPSSs guided by intraoperative Doppler ultrasound offers the best possibilities ever to perform a safe and effective shunt-closure among all the previously described shunt-attenuating techniques.

Acknowledgements

Dr. Szatmári was supported by the Hungarian State Eötvös Scholarship. The authors thank Aart van der Woude for the illustrations and Dr. Ted S. G. A. M. van den Ingh for histopathologic examinations.

This paper was presented in part at the "Voorjaarsdagen" Veterinary Congress in Amsterdam, The Netherlands (April 26-28, 2002) and at the 9th Annual Conference of the European Association of Veterinary Diagnostic Imaging in Archena (Murcia), Spain (July 24-27, 2002).

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CHAPTER 8

Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in dogs

based on the article by

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The Veterinary Record
in press

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Summary

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

Introduction

Portosystemic shunting occurs when anomalous veins allow the portal blood to enter the systemic veins directly without first flowing through the hepatic sinusoids.[1] Portosystemic shunting can be acquired or congenital. **Acquired portosystemic collaterals (APSCs)** develop as the result of sustained hepatic or prehepatic portal hypertension by enlargement of extrahepatic rudimentary vessels, through which no blood normally passes.[2,3] Collateral-formation is a compensatory mechanism to maintain normal portal pressure by allowing the portal blood to be drained into the lower pressure systemic veins. Portosystemic shunting is considered to be congenital if a single or double anomalous vein is present without a concurrent portal hypertension.[1] Definitive treatment of **congenital portosystemic shunts (CPSSs)** involves occlusion of the shunting vessel at a location closest to the systemic venous circulation.[4,5] Attenuating the shunt forces blood to flow through the portal branches, which are often hypoplastic, resulting in subsequent development of portal hypertension.[1,6] Portal vein hypoplasia may be primary or arise secondary to portal hypoperfusion.[1] The degree of portal hypertension that develops during attenuation of a CPSS depends on the severity of portal vein hypoplasia (i.e. the capacity of the portal branches to absorb the increased blood flow), and on the degree of shunt-attenuation. If severe post-ligation portal hypertension develops, the patient may die during or shortly after the surgery as a result of circulatory collapse or thrombosis of the portal vein.[4,7,8] If these acute complications do not occur, APSCs may develop as a result of sustained portal hypertension.[4]

Complete occlusion of a CPSS has been suggested to result in a better outcome compared to partial ligation.[9-14] However, other groups found no difference in clinical outcome between dogs with partial and complete shunt-ligation.[4,15-18] The underlying reasons of poor outcomes in most of the studies remained undetermined. It has been suggested that, if complete shunt-occlusion is not feasible during a surgical attenuation of a CPSS because it would cause the development of a fatal portal hypertension, a second surgery should be performed to attempt a complete shunt-occlusion.[11,14] The underlying idea is that an initial partial ligation would allow the portal system to adapt to an increased flow and the portal branches would become gradually wider by the time of the second surgery.

A second surgery should only be considered, when portosystemic shunting persists exclusively through the CPSS. When shunting occurs through APSCs, further attenuation of the CPSS is contraindicated. To determine whether post-ligation portosystemic shunting occurs via the CPSS, APSCs or both, mesenteric portography can been used.[19] In addition to involving the use of ionising radiation, this angiographic technique requires general anaesthesia and a laparotomy, both of which are high risk procedures in animals with hepatic insufficiency.[20] Though scintigraphy is suitable to detect and to quantify portosystemic shunting,[4,21] with this technique it is impossible to distinguish between congenital and acquired shunting. Doppler ultrasonography offers a non-invasive way to examine the abdominal blood vessels in unsedated dogs, and the direction and velocity of flow can easily be determined.[22] However, no reports were found in the literature about the ultrasonographic findings of dogs that underwent partial surgical ligation of a CPSS.

Our aims are to describe the post-ligation hemodynamic changes of the portal system and to suggest ultrasonographic criteria for decision making as to whether a dog should or should not undergo a second laparotomy for complete CPSS-ligation. Furthermore, we wanted to find an explanation at the level of portal hemodynamics as to

why certain dogs with a partially attenuated extrahepatic CPSS have excellent and others poor clinical outcomes.

Materials and methods

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

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

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

Gauged surgical attenuation of the CPSSs was performed by one surgeon (FJvS) according to a reported method[5] combined with intraoperative Doppler ultrasonography (**Chapter 7**).[23] The CPSS was attenuated to the narrowest diameter that did not result in cyanosis of the intestines. If signs of portal hypertension such as intestinal cyanosis were seen during the surgery, then the ligature was removed and another one with a larger diameter was placed around the shunting vessel. Portal pressure was not measured. Before and after placing the ligature around the shunt the presence and direction of blood flow was determined in the shunt and in the portal vein cranial as well as caudal to the shunt-origin with intraoperative color Doppler ultrasonography. The results of the postligation intraoperative ultrasonography did not influence the surgeon's decision making about the degree of shunt-attenuation. All pre-, intra- and postoperative ultrasound examinations were done by one ultrasonographer (VSz).

At the 4-week re-check, the 12-hour-fasting blood ammonia level was measured and an abdominal ultrasound examination was performed. Special attention was paid to the presence of free abdominal fluid and the visibility of the left gonadal vein (criteria to identify the presence of portal hypertension), and to the size and symmetry of the liver (indicator of portal venous perfusion of the liver). Acquired portosystemic collaterals were diagnosed ultrasonographically when the left gonadal vein was dilated (**Fig 1, Chapter 5**). Special efforts were made to image the right and left portal branches.

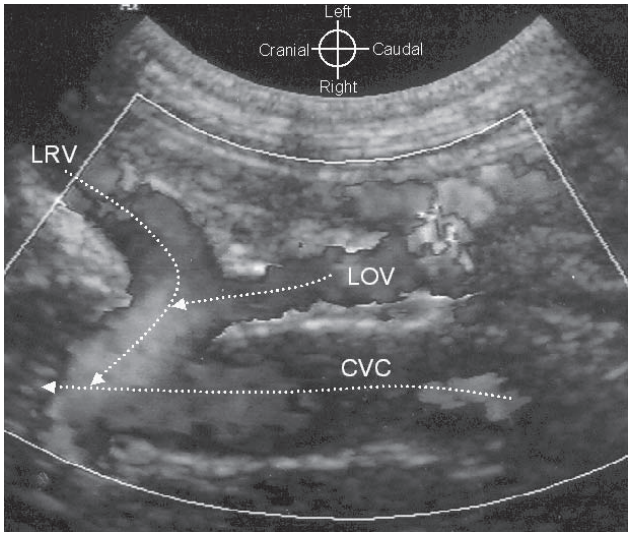


Figure 1.
Dilated left ovarian vein (LOV) as a result of acquired spleno-renal collaterals in a miniature schnauzer four weeks after partial attenuation of a congenital extrahepatic spleno-caval shunt. This color Doppler ultrasound image was made via the left flank with the dog in right lateral recumbency. Arrows indicate the directions of blood flow. LRV Left renal vein, CVC Caudal vena cava. (Full **color** illustration on page 183.)

Colour Doppler ultrasonography was used to determine the direction of flow in the shunt, the portal branches and the portal vein both cranial and caudal to the origin of the shunt. The direction of flow in the shunt was determined adjacent to the portal vein. Pulsed wave Doppler ultrasonography with uniform insonation method was used to measure the flow velocity in the portal vein caudal to the shunt-origin.[22] The time-averaged mean portal velocity was calculated by a built-in automatic spectrum analyser (HighQ ATL HDI 3000).

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

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

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

Results

Right gastric caval shunts

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

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

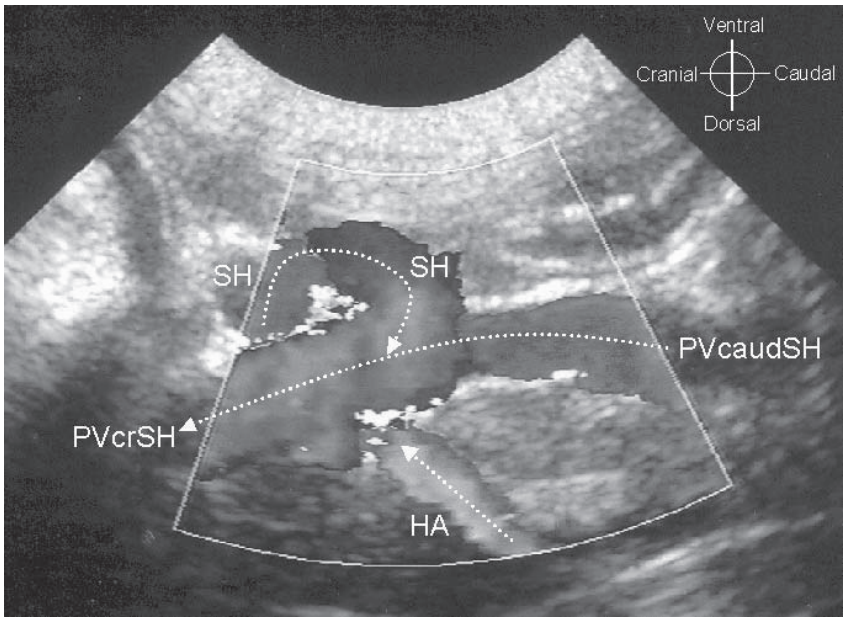


Figure 2.
Color Doppler ultrasound image of a congenital extrahepatic right gastric-caval shunt in a Maltese dog, four weeks after partial surgical attenuation made via the left flank with the dog in right lateral recumbency. The image shows the origin of the cranial shunt-loop. The flow-directions in the shunt (SH) and in the entire portal vein are hepatopetal. The portal vein cranial to the shunt-origin (PVcrSH) has the same diameter as the portal vein caudal to the shunt-origin (PVcaudSH). Arrows indicate the direction of blood flow. HA hepatic artery. (Full **color** illustration on page 183.)

Table 1.
Comparison of the blood flow directions before, immediately after and 4 weeks after partial surgical attenuation of a congenital extrahepatic portosystemic shunt in fourteen dogs.

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

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

OUTCOME	BEFORE NARROWING	IMMEDIATELY AFTER NARROWING	FOUR WEEKS AFTER NARROWING
EXCELLENT (n=2)	-↓← small liver	←↑←	←↑← (Fig 2) normal liver
GOOD (n=1)		←↓←	←↓← normal liver

B. Spleno-caval shunts (n=11)

OUTCOME	BEFORE NARROWING	IMMEDIATELY AFTER NARROWING	FOUR WEEKS AFTER NARROWING
EXCELLENT (n=5)	→↓← (Fig 3) small liver	←↑← (Fig 4A)	←↑← (Figs 4B,4C) normal liver
EXCELLENT (n=1)		←↓← (Fig 5A)	
GOOD (n=1)		←↓← (Fig 5A)	←↓← (Figs 5B,5C) normal liver
FAIR (n=2)		→↓← (Fig 6A)	←↓← ω (Figs 1,5B) asymmetric liver
FAIR (n=1)		←↑- (Fig 8)	←↓← ω (Figs 1,5B) normal liver
POOR (n=1)		→↓← (Fig 6A)	→↓← small liver

Spleno-caval shunts

The blood flow directions pre-, immediately post- and 4 weeks postoperatively in these eleven dogs are demonstrated in **Table 1B**. The preoperative ultrasonographic findings were identical in all the eleven dogs, namely the liver was small, the left and right portal branches could not be identified, and the flow-directions in the shunt and in the portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein were hepatofugal (**Fig 3**).

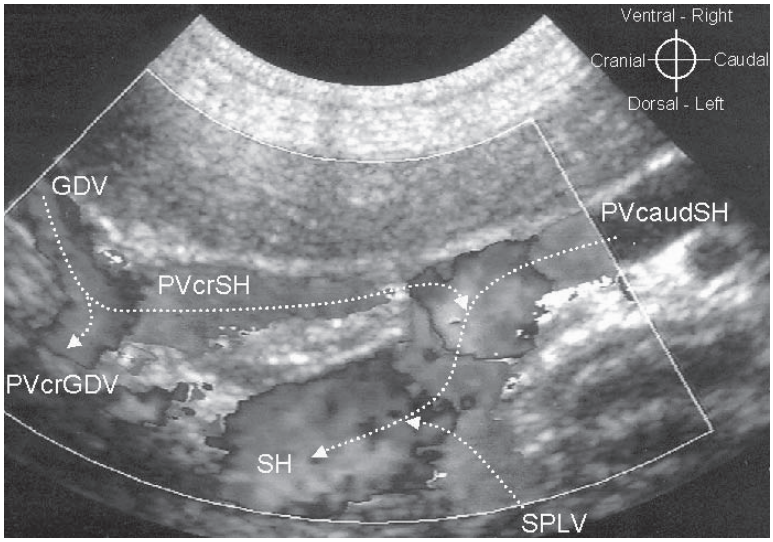
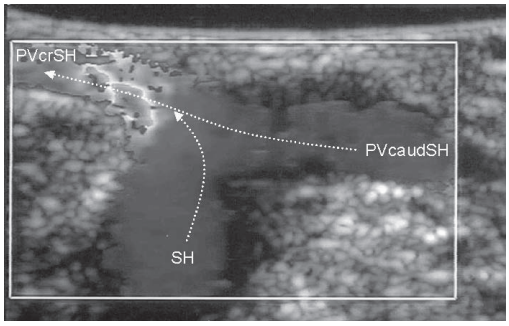


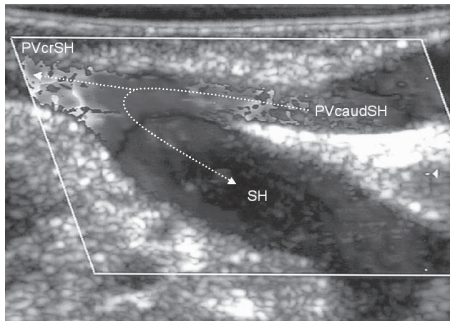
Figure 3.
Transabdominal color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt before shunt-attenuation. The flow-direction is hepatofugal in the portal vein segment between the origin of the shunt (SH) and the entering point of the gastroduodenal vein (GDV) because the majority of blood from the GDV flows caudally to the shunt. The blood that comes from the intestines and from the spleen is shunting. The direction of the blood flow is indicated by arrows. PVcaudSH Portal vein caudal to the shunt-origin, PVcrSH Portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein, PVcrGDV Portal vein cranial to the entering point of the gastroduodenal vein, SPL Splenic vein. (**Color** version on page 183.)

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

Four weeks postoperatively the owners of all six dogs reported complete resolution of the clinical signs. The blood ammonia levels were normal. The liver was symmetric and its size was estimated to be larger than before surgery. Ultrasonographic signs of portal hypertension were absent. The left and right portal branches were well recognisable. Color Doppler ultrasonography revealed that the direction of blood flow in the entire portal vein, in the portal branches and in the shunt adjacent to the portal vein was hepatopetal (**Fig 4B**). In the shunt-segment between the splenic vein and the portal vein the flow-direction was hepatopetal, and in the shunt segment between the splenic vein and the caudal vena cava it was hepatofugal, since a part of the blood from the splenic vein flowed via the shunting vessel to the portal vein and the other part flowed via the attenuated shunt-segment to the caudal vena cava (**Fig 4C**).



4A



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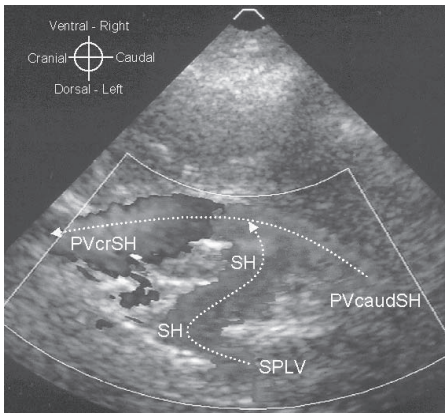

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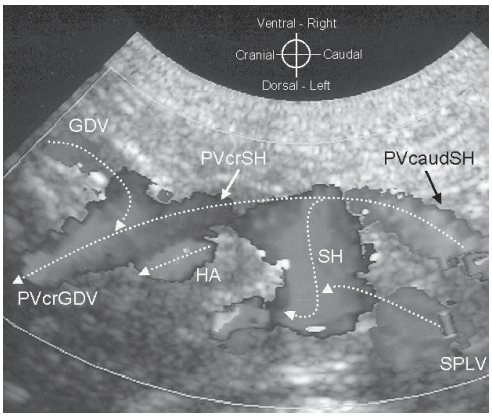

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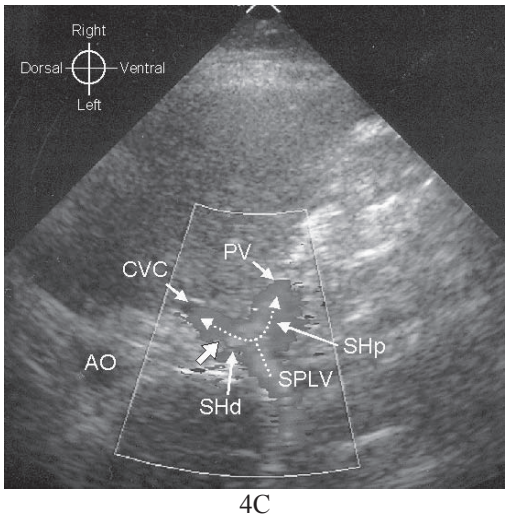

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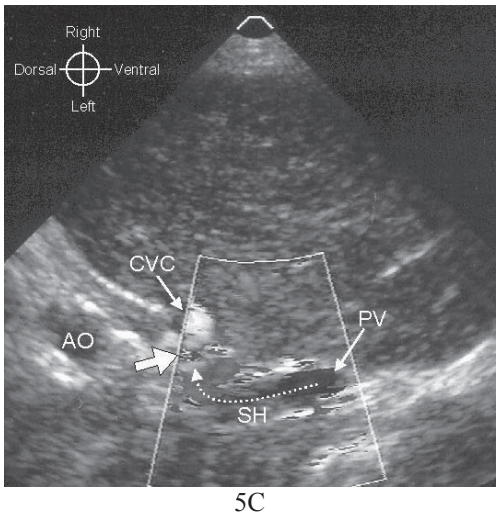


5C

Figure 4.

Figure 5.

See legends on opposite page.

(Full **color** illustrations on page 184.)

Figure 4.

Partially ligated spleno-caval shunt. Scenario of an excellent outcome. The direction of the blood flow is indicated by interrupted arrows.

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

B. Longitudinal transabdominal color Doppler ultrasound image via the right flank, four weeks after partial shunt-attenuation. The blood from the splenic vein (SPLV) flows via the SH to the portal vein (PV) preventing the portal blood from shunting. The flow-direction in the entire portal vein is hepatopetal. Compare to Figure 3!

C. Transverse transabdominal color Doppler ultrasound image via a right intercostal space, four weeks after partial shunt-attenuation. The SPLV enters the middle portion of the shunting vessel. One part of the splenic venous blood flows to the PV via the proximal shunt segment (SHp) and the other part flows to the caudal vena cava (CVC) via the distal shunt segment (SHd). Only splenic blood is shunting because the other portion of the splenic blood prevents the portal blood from shunting. The arrow points at the ligature. AO Aorta

Figure 5.

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

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

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

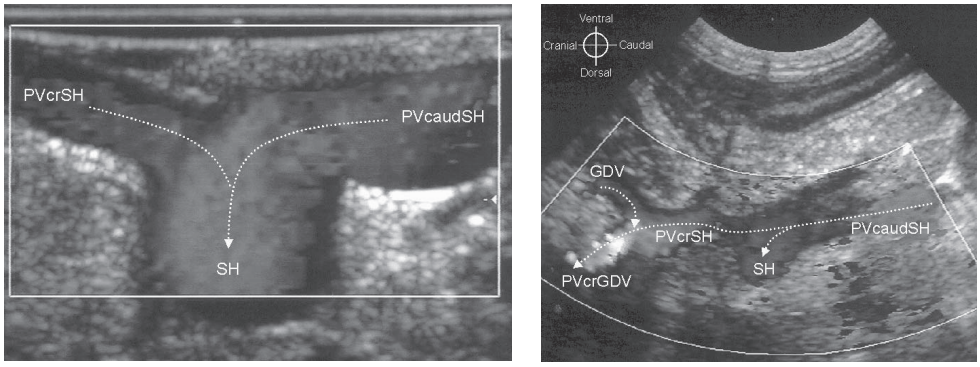
C. Transverse transabdominal color Doppler ultrasound image made via a right intercostal space, four weeks after partial shunt-attenuation. The direction of the blood flow is hepatofugal in the entire SH. This means that the toxin rich blood from the portal vein (PV) flows to the caudal vena cava (CVC).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

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

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

Dogs with fair outcome. In two of the three dogs (a 4-month-old cairn terrier and a 8-month-old miniature schnauzer) with fair outcome the postligation intraoperative ultrasonography revealed hepatofugal flow in the shunt and in the portal vein cranial to the shunt-origin (**Fig 6A**), whereas four weeks after surgery the flow-direction became hepatopetal in the entire portal vein (**Fig 5B, Table 1B**). Actually, the change of the flow-

direction in the portal vein cranial to the shunt-origin was detected already one day after surgery during a non-scheduled ultrasound examination in one dog (**Fig 6B**). At the 4-week re-check abdominal ultrasonography revealed a markedly asymmetric liver. In both dogs the left half of the liver was very small, however the right half was enlarged. The diameters of the portal branches corresponded with the liver size, namely the left portal branch was narrower in both cases than the right one, however normally the left branch is wider. Histopathologic examination of the liver biopsy specimens of the small left liver half revealed the presence of portal vein hypoplasia (**Figs 7A, B**), whereas the hepatic architecture was normal of the specimens that were taken form the enlarged right liver half (**Figs 7C, D**).



A B

Figure 6.

Spleno-caval shunt, scenario of a fair outcome due to concurrent primary hypoplasia of the portal vein. Arrows indicate the direction of blood flow. (Full **color** illustrations on page 183.)

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

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

The third dog with fair outcome (a 9-month-old Yorkshire terrier) displayed hepatopetal flow both in the shunt and in the portal vein cranial to the shunt-origin and zero flow in the portal vein caudal to the shunt-origin at the intraoperative ultrasonography immediately after shunt-attenuation (**Fig 8**). However, four weeks after surgery the flow-direction in the shunt was hepatofugal (**Fig 5C**) and the flow in the entire portal vein hepatopetal (**Fig 5B**). The liver was symmetric and its size was estimated to be larger than before surgery (**Table 1B**).

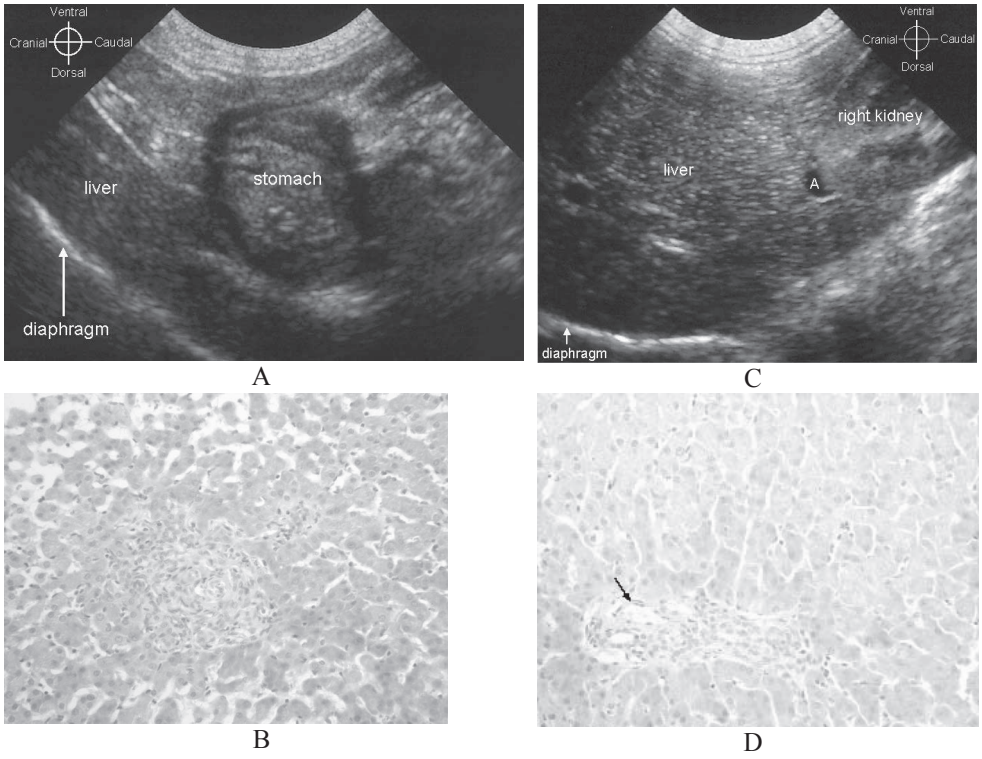


Figure 7.

Hypoplasia of the left portal branch. Ultrasound images (A, C) and photomicrographs of biopsy specimens (B, D) of the left (A, B) and right (C, D) liver halves of a cairn terrier four weeks after a partial attenuation of a congenital extrahepatic spleno-caval shunt. Staining (B, D): haematoxylin-eosin, magnification: 20x.

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

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

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

Dog with poor outcome. In this dog (a 4-month-old Rottweiler) the results of the pre-, intra- and postoperative ultrasonographic examinations did not differ from one another (**Table 1B, Fig 6A**). Four weeks postoperatively the owner observed no clinical improvement, and the blood ammonia level was elevated. The whole liver was small and the left and right portal branches could not be identified. Ultrasonographic signs of portal hypertension were absent. Color Doppler ultrasonography revealed hepatofugal flow both in the shunt and in the portal vein cranial to the shunt-origin.

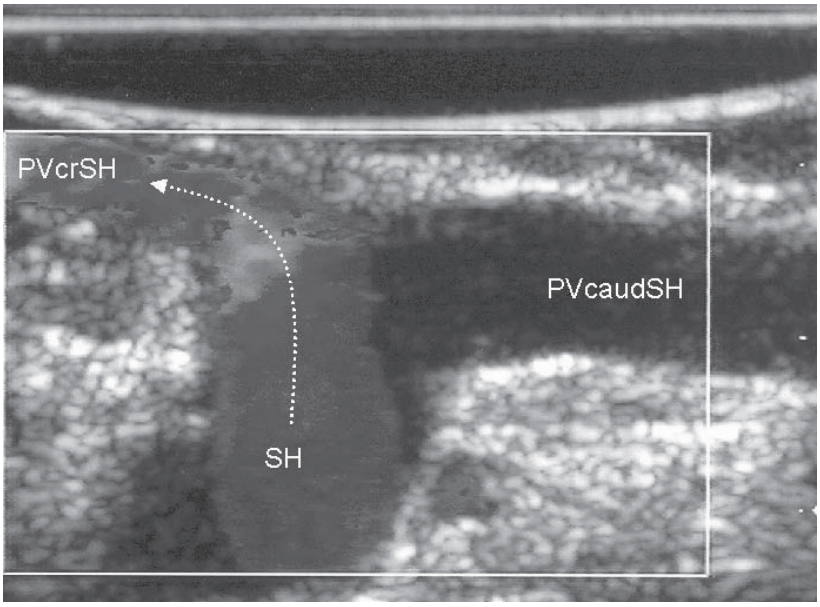


Figure 8.
Spleno-caval shunt, intraoperative color Doppler ultrasound image of a Yorkshire terrier with a fair outcome due to exaggerated shunt-attenuation. The flow-directions in the shunt (SH) and in the portal vein cranial to the shunt-origin (PVcrSH) are hepatopetal. No flow can be detected in the portal vein caudal to the shunt-origin (PVcaudSH), which indicates the presence of portal hypertension. Compare to Figures 4A and 6A!. (Full **color** illustration on page 183.)

In the eleven dogs with spleno-caval CPSS the time-averaged mean velocity in the portal vein caudal to the shunt-origin ranged from 10.7 to 25.3 cm/s at the 4-week re-check.

Discussion

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

CPSS, it does not mean that the toxin-rich blood of the portal vein is shunting, since the shunting blood may originate from a portal tributary.

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

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

Progressive remission of portosystemic shunting has been documented in partially attenuated intra- and extrahepatic CPSSs, and spontaneous mechanical closure of the CPSS due to thrombosis and scar-formation was suspected to be responsible for this phenomenon.[4,21,23] However, the present study has demonstrated that a patent extrahepatic CPSS is not necessarily functional when it diverts only the splenic venous blood, which does not contain more ammonia and other toxins than a systemic vein.

Scintigraphy and portography have been used for follow-up diagnostic imaging of portosystemic shunting.[4,21] Since scintigraphy is unable to differentiate between portosystemic shunting that occurs via CPSSs and that via APSCs, this technique cannot be recommended for follow-up evaluation of attenuated CPSSs because it does not facilitate decision making about a second surgery. Angiography can differentiate whether shunting occurs via the attenuated CPSS or via APSCs, however the use of ionising radiation and anaesthesia is required. The most commonly used angiographic techniques for evaluating portosystemic shunting are intraoperative mesenteric portography and splenoportography. The advantage of intraoperative mesenteric portography is that a jejunal or the mesenteric vein is catheterised, therefore the route of the toxin-rich blood can be followed, however the procedure involves a laparotomy. In cases when mesenteric portography detects APSCs, a high anaesthetic risk was taken merely for diagnostic purposes.

Though splenoportography can be performed without a laparotomy by ultrasound-guided injection of contrast medium into a parenchymal splenic vein, the results of splenoportograms can be misleading. As color Doppler ultrasonography demonstrated in the present study, the splenic vein plays a central role in the portal hemodynamics in cases of partially attenuated extrahepatic CPSSs. This pressure balancing role of the splenic venous blood is the result of the anatomic location of the splenic vein, namely that it enters the shunt in half way between the portal vein and the caudal vena cava. In cases of a non-functional shunt, one part of the splenic blood keeps flowing through the attenuated shunt to the caudal vena cava, however the other part flows to the portal vein. Though the splenic vein belongs to the portal venous system, it does not contain more toxins than any systemic vein, therefore shunting of the splenic blood has no clinical significance. Moreover, the splenic blood that flows from the shunt to the portal vein prevents the toxin-rich mesenteric blood from shunting. Splenoportography however is unable to differentiate between shunting blood arising from the splenic or mesenteric vein. Therefore, splenoportography indicates portosystemic shunting also in cases when only the blood of the splenic vein flows through the shunt. This may result in false positive findings. This phenomenon explains the observations of Van Vechten and others,[4] namely that despite of the evidence of shunt-patency, shunt-values determined by scintigraphy were within the reference range.

The classification of the outcome in the present study was based on the ultrasonographically detected hemodynamics. If telephone interviews or questionnaires had been used to evaluate the surgical outcome as in other studies,[9,13] only the Rottweiler would

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

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

In four dogs the ultrasonographic findings were different immediately after shunt-attenuation and four weeks after surgery indicating that clinically relevant hemodynamic changes can still occur postoperatively both to positive and to negative directions. However, the postoperative portal hemodynamic changes may be predicted based on the intraoperative ultrasound findings in the majority of the cases, especially in dogs with fair outcome.

In one dog with excellent outcome the flow-direction in the shunt changed from hepatofugal to hepatopetal. Local inflammation and scar formation around the ligature may have further reduced the shunt-diameter, and the resistance towards the liver may have been reduced by dilation of the small portal branches. As a result, the attenuated shunt-segment had insufficient capacity to allow the whole amount of splenic venous blood to flow through it, thus a part of it was forced to flow to the portal vein.

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

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

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

Mean portal flow velocities measured four weeks postoperatively in all the fourteen dogs were in the reference range (18.1±7.6 cm/s) that was established in normal dogs by Nyland and Fisher.[24] Apparently, the first priority of the portal circulation after the onset of portal hypertension due to shunt-ligation is to restore and maintain the physiological blood flow in the portal vein. In the present study portal flow velocity in the portal vein caudal to the shunt-origin did not give any additional information regarding the presence of portal hypertension or the level of portal perfusion. However, hepatopetal portal flow direction cranial to the shunt-origin at the 4-week re-check was correlated with a favourable clinical outcome. Though dogs with APSCs or with functional CPSS were clinically healthy four weeks after surgery, they might have an increased chance for recurrence of the clinical signs in the future.

Primary and secondary portal vein hypoplasia cannot be differentiated by histopathologic examination of the liver.[1] Therefore, a possible unfavourable outcome in dogs with a coexisting primary portal vein hypoplasia cannot be predicted before surgical ligation of a CPSS because secondary portal vein hypoplasia, which is the result of hypoperfusion of the liver, is always present in dogs with CPSS. Once the portal circulation has become normal postoperatively (i.e. hepatopetal flow in the entire portal vein), the histopathologic signs of secondary portal vein hypoplasia disappear. In contrast to this,

primary portal vein hypoplasia cannot be solved by restoring normal portal flow. In two dogs with fair outcome the evidence of a coexisting primary portal vein hypoplasia was found both ultrasonographically and histologically at the 4-week re-check. Interestingly, in both dogs the abnormality affected only the left portal branch. The left portal branch supplies the larger part of the liver (four liver lobes) and the right portal branch supplies only two liver lobes, namely the right lateral and the caudate ones. A hypoplastic right portal branch may not have clinical significance. The reason why only the left portal branch was hypoplastic remains obscure. Though preoperative diagnosis of coinciding primary portal vein hypoplasia in dogs with CPSS is currently impossible, its recognition would not change decision making about whether or not a dog with an extrahepatic CPSS should undergo surgery. Observations from the present study suggest that every dog with a CPSS under the age of six years should undergo surgery because even if a coexisting primary portal vein hypoplasia is present and shunt-attenuation would cause the development of APSCs, normal portal circulation can be restored by changing the hepatofugal or zero flow to hepatopetal in the portal vein cranial to the shunt-origin, which subsequently ensures normal hepatic development and function. The clinical signs of hepatic encephalopathy usually also disappear once the flow becomes hepatopetal in the entire portal vein because the amount of mesenteric blood that used to shunt has markedly decreased, and the APSCs probably divert predominantly the splenic venous blood, since the spleno-renal collaterals are the most common types of APSCs in dogs.[2] The patients of the present study did not include dogs older than six years since surgical treatment had not been recommended to the owners if the dog at the time of the diagnosis of a CPSS was over six years of age, because an earlier study revealed significantly worse clinical outcome in older animals.[5] Since intraoperative and postoperative Doppler ultrasonography was not part of the protocol when the study of Wolschrijn and others[5] was performed, the reasons for the less favourable outcomes in older dogs remained unknown. A prospective study with the inclusion of older dogs and of the use of intra- and postoperative ultrasonography should be performed to find the portal hemodynamic reasons for the less favourable outcomes in older animals.

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

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

Acknowledgements

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

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CHAPTER 9

General discussion

Portosystemic shunting occurs when anomalous veins allow the portal blood, which carries toxin-rich blood from the guts, to enter the systemic veins directly without first flowing through the hepatic sinusoids.[1] Portosystemic shunting can occur via acquired portosystemic collaterals or via congenital portosystemic shunts and may result in clinical signs of hepatic encephalopathy due to high blood ammonia levels.

Preoperative ultrasonography

It is necessary to differentiate congenital portosystemic shunts non-invasively from other conditions that cause hyperammonemia, such as acquired portosystemic collaterals and urea cycle enzyme deficiency, since congenital portosystemic shunts require surgical treatment while the other conditions do not. Portography,[2-4] ultrasonography,[5,6] scintigraphy,[7,8], computed tomography,[9] and magnetic resonance imaging[10] have been used to diagnose portal vein disorders. Although Doppler ultrasonography is the only technique that does not require the use of anesthesia, and can provide detailed anatomic as well as hemodynamic information about the abdominal vasculature, its accuracy in detecting congenital portosystemic shunts was reported to be insufficient.[4-6]

In the present studies, real time two-dimensional gray scale ultrasonography combined with color Doppler mode appeared to be an accurate method to: (1) diagnose a congenital portosystemic shunt, (2) specify its type, (3) distinguish it from acquired portosystemic collaterals and (4) rule it out. Four elements are needed to be able to reach a high diagnostic accuracy. The first two, experience and a high quality ultrasound system, are a question of time and money, respectively. However, the other two points, the knowledge of the examination protocol and of the ultrasound anatomy of the abdominal vessels and portal vein anomalies, can be learned from this booklet. Once sonographers knows *what* they are looking for and *how* they can find it, they *will* find it. As a result of our studies, dogs with hyperammonemia do not have to undergo a diagnostic laparotomy. Furthermore, ultrasonographic examinations are quick and can be performed on unsedated animals without the use of ionizing radiation.

Intraoperative ultrasonography

The definitive therapy for congenital portosystemic shunts would ideally be complete occlusion of the shunt at a location closest to the systemic venous circulation.[11,12] However, in most dogs, only partial shunt ligation can be performed because attenuating the shunt vessel forces blood to flow through the portal branches that are frequently hypoplastic, which results in post-ligation portal hypertension.[1,7,11,13-15] Acute portal hypertension can usually be successfully avoided, though development of chronic portal hypertension remains a frequent complication.[11,14-17]

Regardless of the technique used for shunt attenuation and for assessing post-ligation portal hypertension, the clinical outcome remains unpredictable.[15-24] This is largely because there is no method currently available to determine the optimal degree of shunt narrowing.

Most surgeons aim to narrow a congenital portosystemic shunt as narrow as possible. However, as a consequence of an exaggerated closure, dogs may develop postoperative complications related to acute or chronic portal hypertension.

To help avoid this problem, intraoperative ultrasonography greatly increases the likelihood of determining the optimal shunt diameter. Congenital extrahepatic portosystemic shunts do not have to, and actually should not, be completely occluded. Once the flow becomes hepatopetal in the entire portal vein and in the shunt adjacent to the portal vein, further narrowing of the shunt is unnecessary, and would increase the chance for development of portal hypertension.

Surgical ligation of an intrahepatic portocaval shunt is challenging because the anomalous vessel is hidden in the liver lobes. We have shown that intraoperative ultrasonography helps the surgeon to select the optimal point for dissection of the liver and ligation of the shunt.

Postoperative ultrasonography

Complete occlusion of a congenital extrahepatic portosystemic shunt has been suggested to result in a better clinical outcome compared to partial ligation.[4,14,19,25-27] However, other surgeons have not been able to confirm this finding.[11,17,18,28,29] It has been suggested that, if complete shunt-occlusion is not feasible, a second surgery should be performed to attempt a complete shunt-occlusion.[4,19] Partial attenuation of a congenital portosystemic shunt is believed to result in a less favorable outcome than a complete occlusion. Therefore, several surgeons propose that a second surgery should be performed in every dog whose shunt has partially been attenuated in order to occlude their shunt completely.

We have shown that dogs that underwent a partial ligation of a congenital portosystemic shunt do not have to be routinely re-operated on. Color Doppler ultrasonography performed at least one month after surgical ligation of a shunt can reveal whether a persisting hyperammonemia is the result is of a functional shunt, acquired portosystemic collaterals, or both. Dogs that have developed acquired portosystemic collaterals should not be re-operated because they already suffer from portal hypertension. Dogs, whose extrahepatic portosystemic shunt was partially ligated and the flow direction in the entire portal vein and in the shunt adjacent to the portal vein became hepatopetal, should not be re-operated on. This is because their shunt is not functional (though it is patent) and their blood ammonia levels are within the normal reference range.

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CHAPTER 10

Summary
(in English, Dutch and Hungarian)

Samenvatting

Összefoglaló

Introduction

In Chapter 1 we begin by explaining the normal abdominal vascular anatomy of the dog and the consequences of portosystemic shunting.

In Chapter 2 we explore the problems that may occur during the diagnostic process of portal vein anomalies as well as during and after surgical ligation of congenital portosystemic shunts.

Chapter 3 reviews the principles of the Doppler ultrasonographic modalities. In addition, we describe how the various abdominal blood vessels can be localized and identified with Doppler ultrasonography in dogs.

Preoperative ultrasonography

In Chapter 4 we present a detailed description of the ultrasonographic features of the different conditions that lead to high blood ammonia level in dogs, including the various types of congenital portosystemic shunts and portal hypertensive disorders. Based on the ultrasonographic findings guidelines are given of how to decide whether a dog with hyperammonemia should or should not undergo a laparotomy.

We conclude that

- Intrahepatic portosystemic shunts arise either from the left or right portal branch and terminate in the caudal vena cava.
- Extrahepatic portosystemic shunts drain the blood of the portal vein via a short segment of the splenic or the gastroduodenal vein and terminate in the caudal vena cava slightly cranial to the celiac artery or enter the thorax.
- The abdominal ultrasonographic findings of dogs with urea cycle enzyme deficiency do not differ from normal.
- Arterioportal fistula has characteristic ultrasonographic features.

In Chapter 5 an examination technique for systematic ultrasonographic evaluation of the canine portal system is described in a “cookbook style”. This protocol allowed not only the accurate recognition and characterization of the different types of congenital portosystemic shunts and acquired portosystemic collaterals, but also the exclusion of portosystemic shunting in a large number of dogs.

We conclude that

- Ultrasonography allows accurate diagnosis of congenital portosystemic shunts and identification of their subtypes.
- A dilated left gonadal vein is indicative for the presence of spleno-renal acquired portosystemic collaterals as a result of hepatic or prehepatic portal hypertension.

Intraoperative ultrasonography

In Chapter 6 we show that intraoperative ultrasonography can replace both portal pressure measurement and mesenteric portography during surgical ligation of congenital intrahepatic portocaval shunts in dogs. In addition, intraoperative ultrasonography provides valuable anatomic details for the surgeon about the intrahepatic course of the shunting vessel.

We focus on the portal hemodynamic changes associated with surgical ligation of congenital extrahepatic portosystemic shunts in Chapter 7. Ultrasonographic criteria for determining the optimal degree of shunt narrowing are recommended. This method offers the safest and most efficient way to facilitate surgical decision making among all techniques that have ever been described in congenital portosystemic shunt surgery. Moreover, the clinical outcome may also be predictable.

We conclude that

- Hepatofugal flow (i.e. flow away from the liver) occurs often in the portal vein cranial to the shunt origin in dogs with congenital extrahepatic shunts of splenic vein origin.
- Hepatofugal portal flow in these cases is the result of the fact that the blood from the gastroduodenal vein finds lower resistance to flow caudally to the shunt than cranially to the hepatic sinusoids.
- During surgical ligation of a congenital extrahepatic portosystemic shunt the largest shunt diameter should be found that ensures hepatopetal flow (i.e. flow towards the liver) in the entire portal vein and in the shunt adjacent to the portal vein; a smaller shunt diameter would only increase the degree of portal hypertension, but would not reduce shunting, nor would it improve hepatic perfusion.
- Post-ligation hepatopetal flow in the shunt is the result of the anatomical fact that the splenic vein enters the shunting vessel and its blood flows via the shunt to the portal vein.
- When the flow in the portal vein cranial to the shunt remains hepatofugal during a temporary occlusion of a congenital extrahepatic portosystemic shunt with a splenic vein origin, a suboptimal surgical outcome may be predicted because of a possible coinciding primary hypoplasia of the left portal branch.

Postoperative ultrasonography

In Chapter 8 we describe how Doppler ultrasonography can be used for postoperative evaluation of attenuated congenital extrahepatic portosystemic shunts. Guidelines are given to decide whether a second surgery is recommended for further shunt attenuation. Furthermore, causes of postoperative formation of acquired portosystemic collaterals are described.

We conclude that

- Dogs with hepatopetal portal flow cranial to the shunt origin are clinically healthy regardless of the presence of portosystemic shunting.
- Hepatopetal flow in the shunt adjacent to the portal vein prevents the toxin-rich portal blood from shunting, whereas shunting of the splenic venous blood via the attenuated segment of the shunt has no clinical significance; these partially attenuated extrahepatic congenital shunts are non-functional, therefore do not require further narrowing.
- A second surgery that aims further narrowing of a partially attenuated congenital extrahepatic portosystemic shunt is recommended in dogs, only when one month after surgery the flow is hepatofugal in the portal vein cranial to the origin of the shunt.
- Dogs with a dilated left gonadal vein should not undergo a second surgery because they already developed a portal hypertension.

Echografie van portosystemische shunting bij de hond
Dopplerondersoeken voor, tijdens en na chirurgie

Introductie

In hoofdstuk 1 wordt de anatomie van de abdominale bloedvaten bij de hond beschreven en worden de gevolgen van een portosystemische communicatie uitgelegd.

In hoofdstuk 2 worden de problemen die tijdens de diagnostische beeldvorming van afwijkingen van de vena portae, en de problemen die tijdens de chrirurgische correctie van aangeboren portosystemische shunts kunnen voorkomen, behandeld.

In hoofdstuk 3 worden de principes van dopplerechografie en de indicaties voor de verschillende dopplertechnieken besproken. De auteurs leggen uit hoe de abdominale bloedvaten met behulp van echografie bij de hond kunnen worden zichtbaar gemaakt en geïdentificeerd.

Preoperatieve echografie

In hoofdstuk 4 worden de echografische bevindingen beschreven van de verschillende aandoeningen die verhoogde bloedammoniakconcentraties veroorzaken, zoals aangeboren portosystemische shunts en aandoeningen die leiden tot hepatische of prehepatische portale hypertensie. Er worden echografische criteria vermeld aan de hand waarvan kan worden vastgesteld welke honden wel en welke honden niet geopereerd moeten worden.

De volgende conclusies werden getrokken:

- De oorsprong van intrahepatische portocavale shunts is of de rechter, of de linker portatak, en deze shunts monden uit in de vena cava caudalis.
- Extrahepatische portosystemische shunts draineren het bloed van de vena portae via de vena lienalis of de vena gastroduodenalis naar de vena cava caudalis op een niveau net craniaal van de plaats waar de arteria coeliaca uit de aorta ontspringt of naar de vena azygos, in de borstholte.
- Echografische bevindingen bij onderzoek van de buikholte bij honden met ureumcyclusenzymdeficiëntie zijn normaal.
- Echografische bevindingen bij honden met arterioportale fistels zijn pathognostisch.

In hoofdstuk 5 wordt een onderzoeksprotocol beschreven, waarmee aangeboren portosystemische shunts en verkregen portosystemische collateralen kunnen worden gevonden en aangeboren portosystemische shunts kunnen worden getypeerd.

De volgende conclusies werden getrokken:

- Echografie is een betrouwbare methode om aangeboren portosystemische shunts te diagnosticeren en te typeren.
- Een gedilateerde vena ovarica of vena testicularis sinistra wijst op de aanwezigheid van verkregen splenorenale collateralen als gevolg van een hepatische of prehepatische hypertensie.

Intraoperatieve echografie

In hoofdstuk 6 wordt aangetoond dat intraoperatieve portografie en directe portale drukmeting tijdens de chirurgische sluiting van intrahepatische portocavale shunts bij honden kunnen worden vervangen door intraoperatieve echografie. Bovendien levert intraoperatieve echografie belangrijke anatomische informatie over het intrahepatische verloop van de shunt.

In hoofdstuk 7 worden de hemodynamische veranderingen beschreven die als gevolg van de chirurgische vernauwing van een aangeboren extrahepatische portosystemische shunt optreden. De veranderingen in de stroomrichting van het bloed kunnen worden gebruikt voor de bepaling van de ideale mate van shuntvernauwing. Deze methode is veiliger en betrouwbaarder dan andere, tot nu toe gebruikte technieken.

De volgende conclusies werden getrokken:

- Hepatofugale stroom (stroomrichting van de lever af) in de vena portae craniaal van de oorsprong van de shunt komt vaak voor bij honden met een aangeboren portosystemische shunt die uitgaat van de vena lienalis.
- De hepatofugale stroom wordt veroorzaakt door het bloed uit de vena gastroduodenalis dat naar caudaal, in de richting van de shunt, een lagere weerstand ondervindt dan naar craniaal, in de richting van de leversinusoïden.
- De chirurg moet de grootste shuntdiameter vinden die hepatopetale stroom (stroomrichting naar de lever toe) in de gehele vena portae en in de shunt vlakbij de vena portae veroorzaakt; een smallere shuntdiameter zou alleen de mate van portale hypertensie doen toenemen maar zou de circulatie van de lever niet verbeteren en de mate van shunting niet verminderen.
- Hepatopetale stroom in de shunt na vernauwing wordt veroorzaakt doordat het bloed van de vena lienalis, die uitmondt in de shunt, via de shunt naar de vena portae stroomt.
- Wanneer bij een tijdelijke afsluiting van een aangeboren portosystemische shunt, die uitgaat van de vena lienalis, de stroomrichting in de vena portae craniaal van de oorsprong van de shunt hepatofugaal blijft, is er vermoedelijk sprake van hypoplasie van de linker portatak en is de prognose minder gunstig.

Postoperatieve echografie

In hoofdstuk 8 wordt de waarde van dopplerechografie beschreven bij onderzoek van honden die een shuntoperatie hebben ondergaan. Er worden criteria gegeven op grond waarvan kan worden vastgesteld of een tweede operatie nodig is om de shunt verder te vernauwen. Bovendien worden de mogelijke oorzaken van de ontwikkeling van portosystemische collateralen beschreven.

De volgende conclusies werden getrokken:

- Honden met een hepatopetale bloedstroom in de vena portae craniaal van de oorsprong van de shunt zijn klinisch gezond ook al hebben ze portosystemisch shunting.
- Hepatopetale bloedstroom in het deel van de shunt vlakbij de vena portae zorgt ervoor dat het toxinerijke bloed van de vena portae niet meer shunt, terwijl de

shunting van het bloed van de vena lienalis klinisch niet relevant is; in deze gevallen is geen tweede operatie nodig om de shunt verder te vernauwen.
- Een tweede operatie moet alleen worden uitgevoerd wanneer de bloedstroom in de vena portae craniaal van de oorsprong van de shunt 1 maand na chirurgie hepatofugaal is.
- Honden met een gedilateerde vena ovarica of vena testicularis sinistra hebben al portale hypertensie en dienen dus geen tweede operatie te ondergaan.

Kutyák portoszisztémás söntjének ultrahangvizsgálata
Doppler vizsgálatok műtét előtt, alatt és után

Bevezetés

Az 1. fejezetben a hasi erek normál anatómiáját, a portoszisztémás sönt definícióját és klinikai következményeit ismerteti a szerző.

A 2. fejezetben a verőceér rendellenességeinek diagnosztikai nehézségei, majd a veleszületett portoszisztémás söntök műtéti úton történő gyógykezelése során fellépő problémák kerülnek megtárgyalásra.

A 3. fejezetben a Doppler-ultrahang alapjait, valamint a különféle Doppler technikák leírását találja meg az olvasó. Ezen kívül a szerzők vázolják, hogy miként lehet a kutyák fontosabb hasi ereit Doppler-ultrahang segítségével felkeresni és azonosítani.

Az ultrahangvizsgálat szerepe a műtéti indikáció felállításában magas vérammóniaszint esetén

A 4. fejezetben a kutyák magas vérammóniaszinttel járó betegségeinek hasi ultrahang lelete kerül ismertetésre, különös tekintettel a veleszületett portoszisztémás söntök variánsaira és a portális hypertóniával járó kórképekre. Az olvasó iránymutatást kap arra vonatkozóan, hogy a hasi ultrahang lelet alapján mely esetekben szükséges műtéti és mely esetekben konzervatív gyógykezelést alkalmazni.

Fontosabb megállapítások:

- Az intrahepatikus portoszisztémás söntök a verőceér jobb vagy bal főágának folytatásai, amelyek a verőceér vérét a hátulsó üresvénába vezetik.
- Az extrahepatikus portoszisztémás söntök a verőceér vérét a v. lienalis vagy a v. gastroduodenalis egy rövid szakaszán át a hátulsó üres vénába vagy a v. azygos mellkasi szakaszába juttatják.
- A hasi ultrahang vizsgálat nem mutat elváltozást azokban a kutyákban, amelyekben a magas vérammóniaszint oka a karbamidciklus egy vagy több enzimjének az aktivitáscsökkenése.
- Az intrahepatikus arterioportális fisztulák ultrahanglelete patognosztikus.

Az 5. fejezetben egy olyan részletes ultrahang vizsgálati séma kerül leírásra, amely segítségével nemcsak a különféle veleszületett portoszisztémás söntök felismerése válik lehetővé, hanem ezek kizárása is, valamint a szerzett portoszisztémás kollaterálisoktól való elkülönítése.

Fontosabb megállapítások:

- A hasi ultrahangvizsgálat megbízható módszer a veleszületett portoszisztémás söntök diagnosztizálására, tipizálására és kizárására.
- A kitágult bal v. ovarica vagy testicularis — máj- vagy verőceéreredetű — portális hypertónia fennállására utal.

Az ultrahangvizsgálat szerepe a veleszületett portoszisztémás söntök műtéte során

A 6. fejezetben az intraoperatív ultrahangvizsgálat szerepe kerül bemutatásra az intrahepatikus portocavalis söntök műtéti szűkítése során.

Fontosabb megállapítások:

- Az intraoperatív Doppler-ultrahangvizsgálat kiválthatja az invazív direkt portális vérnyomásmérést és portográfiát.
- Intraoperatív ultrahangvizsgálat értékes információt nyújt a sebész számára a sönt májlebenyen belüli lefutásáról.

A 7. fejezetben a veleszületett extrahepatikus portoszisztémás söntök szűkítése során fellépő hemodinamikai változások ismertetésére kerül sor. Ezen változások irányadó paraméterekként szolgálnak a söntszűkítés mértékének a meghatározására. Az intraoperatív ultrahangvizsgálat az extrahepatikus portoszisztémás söntök szűkítése során a korábban leírt módszereknél megbízhatóbb, ezen kívül a segítségével a műtét kimenetele is prognosztizálható.

Fontosabb megállapítások:

- A lépvénából eredő veleszületett extrahepatikus portoszisztémás söntök esetében gyakori a hepatofugális (vagyis a májtól távolodó) véráramlás a verőceérnek a sönt eredésétől craniálisan eső szakaszán, mivel a v. gastroduodenalis vére caudalisan, a kisebb ellenállást képviselő sönt irányába, és nem craniálisan, a nagyobb ellenállást képviselő májszinuszoidok felé áramlik
- A veleszületett extrahepatikus portoszisztémás söntök sebészi szűkítése során az ideális söntátmérő az a lehető legnagyobb átmérő, amelynél a véráramlás iránya mind a verőceér egész hosszában, mind pedig a sönt verőceér felőli szakaszán hepatopetalissá (a máj felé irányulóvá) válik; ennél szűkebb söntátmérő nem javítja a máj perfúzióját, és nem csökkenti a „söntölődő" vér mennyiségét sem, viszont fokozza a portális hypertónia súlyosságát.
- A sönt szűkítése utáni hepatopetális áramlás a sönt verőceér felé eső szakaszán annak a következménye, hogy a lépvéna a söntbe szájadzik, és a lépvéna vérének egy része a söntön keresztül a verőceérbe folyik.
- Kétes a prognózis — a verőceér bal főtörzsének hypoplasiája miatt —, ha a sönt ideiglenes lekötésekor a véráramlás iránya a verőceérnek a sönttől craniálisan eső szakaszán hepatofugális marad.

Az ultrahangvizsgálat szerepe a veleszületett portoszisztémás söntök műtéti szűkítése után

A 8. fejezetben iránymutatások találhatóak arra vonatkozóan, hogy mely esetekben szükséges egy újabb műtéti beavatkozás elvégzése a sönt további szűkítése céljából. Ezen kívül a műtét eredményeként szerzett portoszisztémás kollaterálisok lehetséges okai kerülnek megtárgyalásra.

Fontosabb megállapítások:

- Tünetmentesek azok a kutyák, amelyek verőceerének a sönt eredésétől craniálisan eső szakaszán a véráramlás iránya hepatopetalis, függetlenül egy funkcionális portoszisztémás sönt vagy portoszisztémás kollateralisok jelenlététől.
- A hepatopetális véráramlás a söntnek a verőceér felőli szakaszán megakadályozza, hogy a verőceér káros anyagcseretermékekben gazdag vére „söntölődjön", miközben a lépvéna „söntölődő" vérének nincs klinikai jelentősége; ebben az esetben bár a részlegesen lekötött sönt fizikailag átjárható, funkcinálisan mégis zártnak tekinthető, így további szűkítése nem indokolt.
- A részlegesen lekötött veleszületett extrahepatikus portoszisztémás söntök további szűkítése egy második műtét keretében csak akkor szükséges, ha egy hónappal az első műtét után a verőceér sönt eredésétől craniálisan eső szakaszán a véráramlás iránya hepatofugális.
- Azokat a kutyákat, amelyeknek a bal v. ovaricája vagy testicularisa kitágult, nem javasolt újra műteni, mivel a sönt további szűkítése csak a már amúgy is fennálló portális hypertóniát súlyosbítaná.

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Journal articles

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Viktor Szatmári

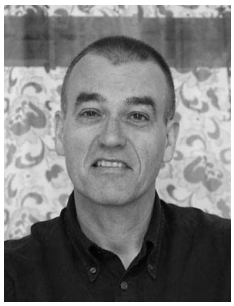
Curriculum vitae

The author of this thesis was born in Debrecen, Hungary on 3 May 1972. After finishing the secondary school (Tóth Árpád Gimnázium, Debrecen) in 1990, he studied veterinary medicine at the University of Veterinary Science in Budapest (September 1990 – December 1995). He received his diploma on 1 December 1995 with *summa cum laude*. After graduation the author completed an internship at the Small Animal Clinic of the Department of Internal Medicine of the University of Veterinary Science in Budapest (December 1995 – December 1996). He started to work as a PhD-student at the same department from January 1997 on the topic of Doppler ultrasonography of abdominal blood vessels in dogs. Besides his research activities he actively participated in the routine ultrasonography, cardiology and internal medicine service of the clinic. From October 1999 he continued his research at the Division of Diagnostic Imaging of the Faculty of Veterinary Medicine of the Utrecht University in the Netherlands focusing on Doppler ultrasonography of the portal vein in dogs. In February 2003 he became a junior teacher and researcher of the same division and performed routine ultrasonographic examinations on small animals. In September 2003 he started a residency program in companion animal internal medicine and cardiology at the Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University.

Acknowledgements — Köszönetnyilvánítás — Dankword


Dr. George Voorhout


Prof. Dr. Jan Rothuizen


Prof. Dr. Freek van Sluijs

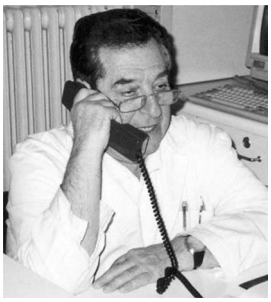

Dr. Ted van den Ingh

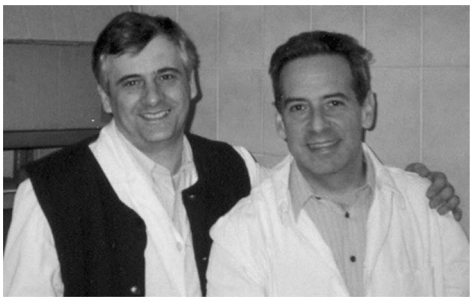
Beste George, thank you for taking the responsibility to guide this project and to be my major advisor. I really enjoyed working with you and appreciated your optimistic words even when things did not go the way they should have. I am grateful that your door was always open to discuss any problems, regardless whether they were professional or private. Your positive thoughts and continuous support gave me a lot of energy to go on with my research. I am grateful that you moved all expected and unexpected obstacles out of my way. I remember that intraoperative sonography was your idea. Thank you very much!

Beste Jan, thank you for being a really professional hepatologist and for finding so many dogs with portal vein anomalies. The fact that you always knew in advance what I would find with ultrasound made me learn a lot indeed. I am grateful for your positive way of thinking, continuous support, relaxed personality and good sense of humor. It was a pleasure to work with you.

Beste Freek, thank you for giving me the opportunity to perform intraoperative ultrasound studies during the shunt operations and for your patience during these surgeries.

Beste Ted, thank you for the explanations about the pathological details of the portal vein anomalies and the reliable histopathological diagnosis.


Dr. László Papp


Prof. Dr. Károly Vörös & Dr. Zoltán Harkányi


Dr. Ferenc Manczur

Kedves Laci bácsi, thank you for showing me your 'tricks' at the small animal clinic in Budapest. I will never forget your great sence of humor. You inspired me a lot!

Kedves Tanár Úr, thank you for giving me the opportunity to become a member of the clinic's ultrasound team and for guiding me to the best teachers to learn as much about this subject as possible. I am grateful for the freedom and support I got at your clinic in Budapest.

Kedves Zoltán, thank you for the ultrasound courses (**B**udapest **U**ltrasound **C**entre) and for allowing me, as a veterinarian, to enter the world of the human radiologists. I am especially grateful for your idea to start working with vascular Doppler ultrasonography.

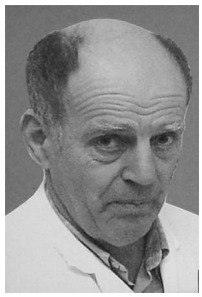
Kedves Feri, thank you for letting me leave Budapest for Utrecht and for keeping the ultrasound unit running at the University Clinic in Budapest.


Dr. Linda McPhee


Wim Kersten


Richard Lenters


Aart van der Woude


Ton van Os

Dear Linda, thank you for telling me the secrets of how to compose a scientific paper so that the readers will understand the story and get the message. I learned a lot from you!

Beste Wim en Richard, thank you for your invaluable help in making the corrosion casts.

Beste Aart, thank you for sharing some of your design tricks with me and for making me realize that less is often more, that pictures convey more than words and that tiny details can make huge differences.

Beste Ton, thank you for always managing to find someone to hold the dogs for me.

The cooperation and help that I received from colleagues in the Departments of Anesthesiology (particularly *Gert*, *Jo*, *Ron* and *Jan*) and Pathology in Utrecht are highly appreciated.


Division of Diagnostic Imaging in Utrecht (2002)

Beste Julia, Leonie, … thank you for your endless patience and company during the long hours of ultrasound examinations when you were holding the dogs for me in the "dark room".

Beste AJ, Elise, Flip, Hans, Lineke, Susanne, … thank you for being entertaining during the routine working days and for making each of them special. These days were not so gray after all.

Dear Tony, the efforts of all International Neighbour Group (ING) volunteers (especially Prof. Dr. Tony Hearn) in organizing social events for the foreign employees at the Utrecht University was greatly appreciated.

Dr. István Szatmári & Katalin Papp: my parents

Lujza Kubinyi

Dr. Csilla Becskei

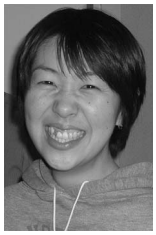
Kedves Anyu és Apu, I want to thank you for always inspiring me to study hard and for supporting me whichever way I chose to go on. Thank you for making a real home and for making my studies possible. You did *everything* to make my life easier by creating ideal conditions for me.

Kedves Lujza, thank you for giving me a place in your home to study and for placing your interest in the background.

Kedves Csillus, thank you for your support and for helping me after graduation in making a decision about staying on the academic path.

Dr. Walter van den Brom

Zsófia Halmi



Chiharu Nakamura

Dr. Allison Mills

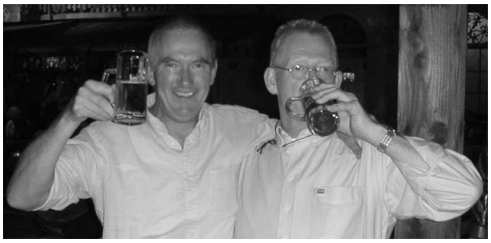
Dr. Paddy Royall

Beste Walter, thank you for your advice regarding statistical issues and for creating the term "differential portal velocity".

Kedves Zsózsó, Mária néni, Mackó és Nándi bácsi, thank you for your hospitality and for giving me a place to stay in Budapest.

Shi-n-ai-naru Chiharu-chan, thanks for designing the front cover of this booklet.

Dear Allison, Paddy and Eric, thanks a lot for correcting my English mistakes.



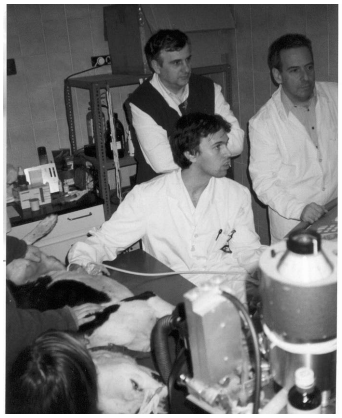
Dr. Anton Jan van den Belt & Hans van den Brinck

Beste Hans en AJ, whom I chose to support me during the PhD-defense, thank you for taking care of me from the very beginning and for making me feel comfortable at the department in Utrecht, in a different country and among different people ...

My apologies if I have forgotten to mention someone; I am just a human being who does make mistakes ...

Addendum — Ráadás

"People do not change after the age of 18." (Ferenc Manczur)



Hungary,1999

The Netherlands, 2003

„Az ember mindenét feláldozza egy reménységért; amit pedig szerzett és szerez, azt ismét egy újabb reménységért áldozza fel. Úgy látszik, ez a Természetnek valamiféle kaján rendje, amelyet azért teremtett, hogy mindig kivegye kezünkből azt, amit megkaptunk. Nem gondolunk boldogságunkkal a jelen pillanatban, hanem mindig az eljövendő időkre halasztjuk, mintha a jövő más volna, mint a jelen."
(Fontenelle, Nagy Orsolya nyomán)

* * *

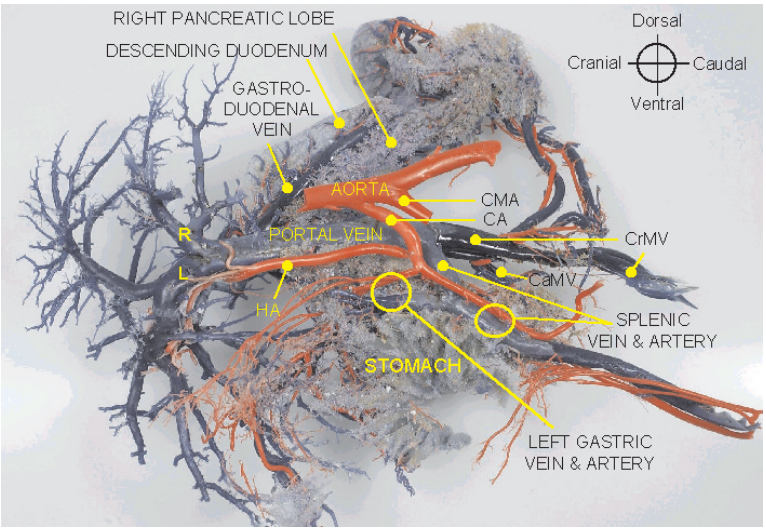
"You cannot stand on one leg." (Jan Rothuizen)

„Ha valami nem sikerül, az azért nem sikerül, mert valami sokkal jobb fog helyette történni."
(Szabó Árpád)

Looking at things from a different angle ...

COLOR FIGURES

CHAPTER 1
General introduction



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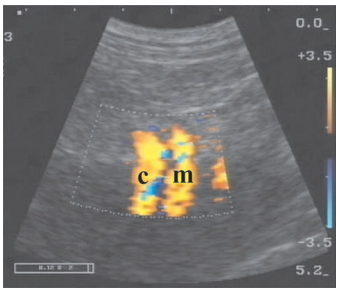
CHAPTER 3
Principles of abdominal vascular Doppler ultrasonography in dogs



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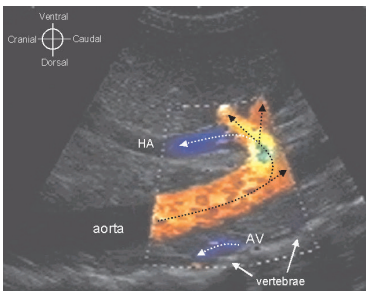

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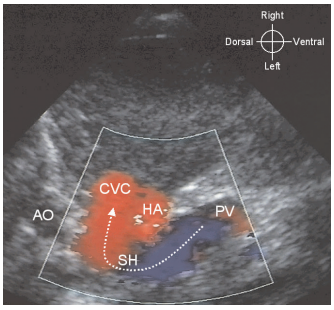
CHAPTER 4
Ultrasonographic differentiation of hyperammonemic conditions in dogs



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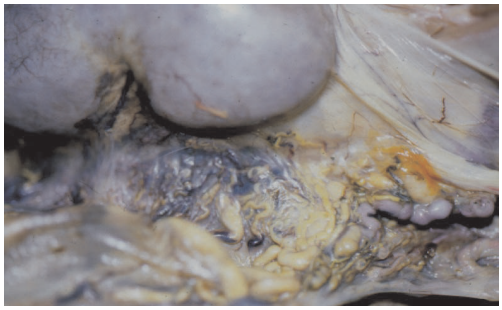

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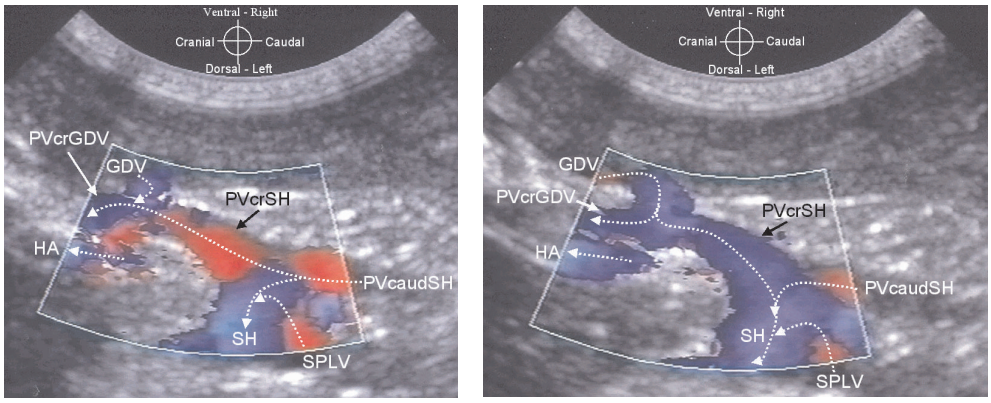

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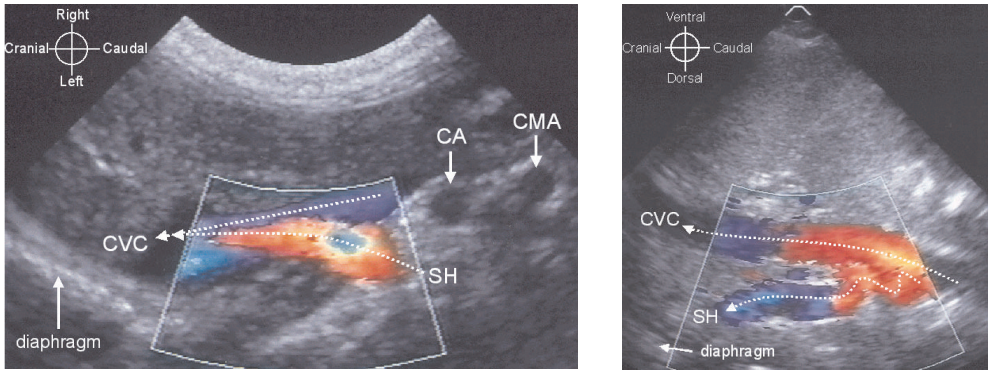

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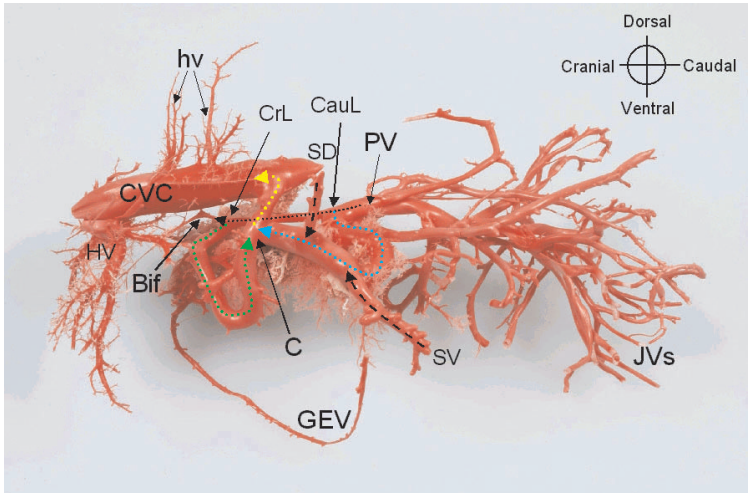

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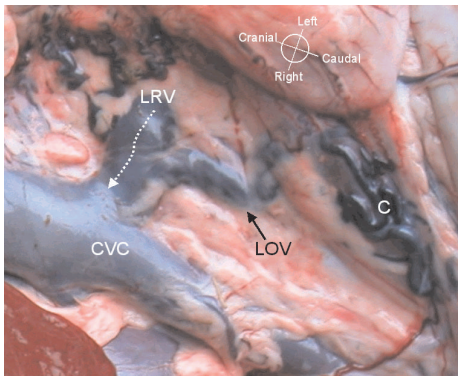

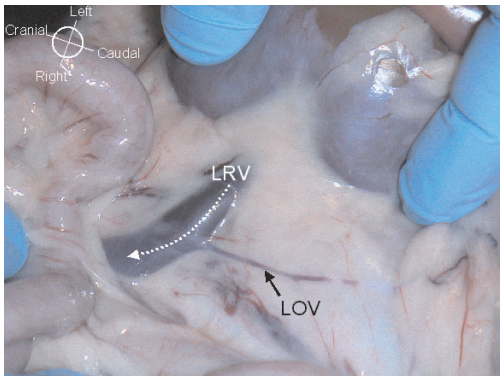

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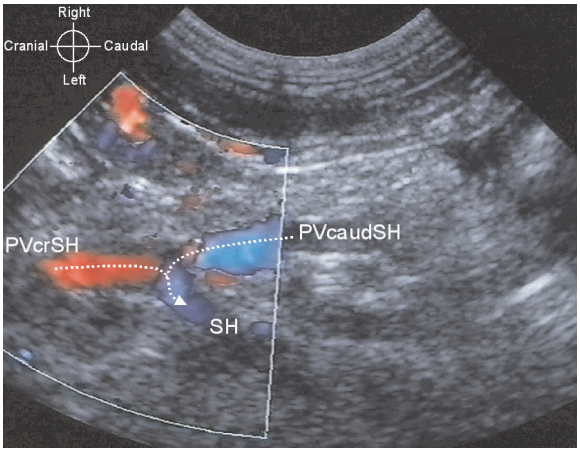

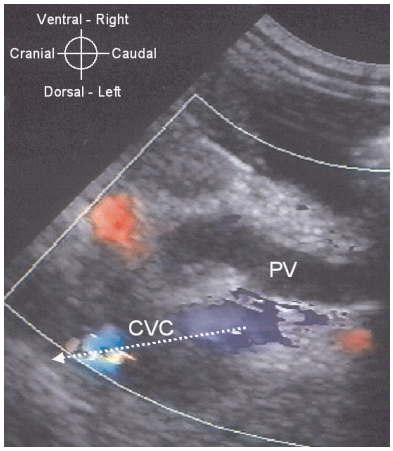

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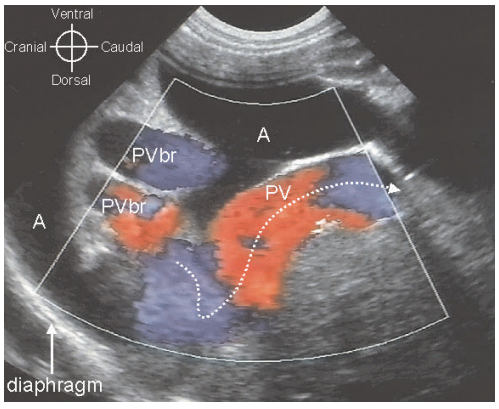


Figure 9 from Page 63.

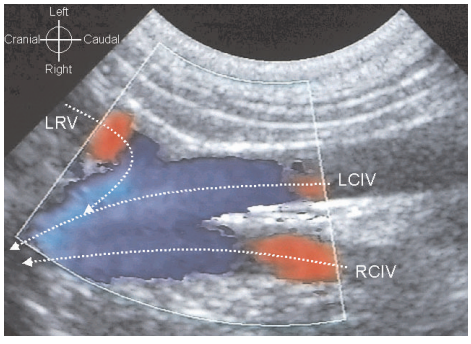
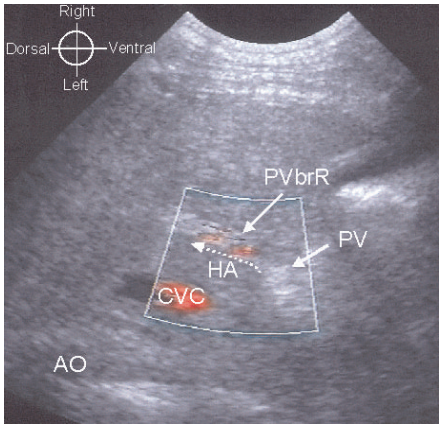
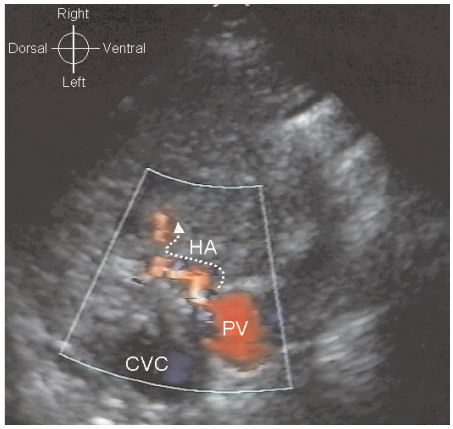


Figure 10A from Page 64.

CHAPTER 5

Standard planes for ultrasonographic identification and characterization of congenital portosystemic shunts and acquired portosystemic collaterals in dogs





Figures 2E & F from Page 80.

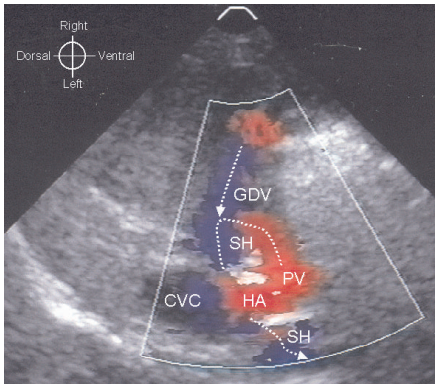


Figure 3A from Page 82.

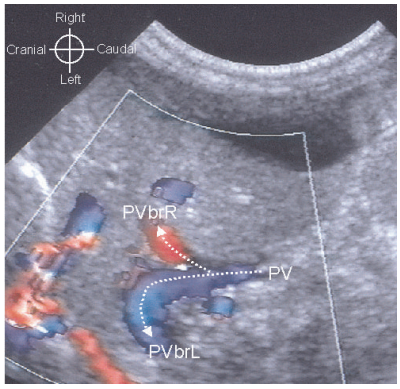
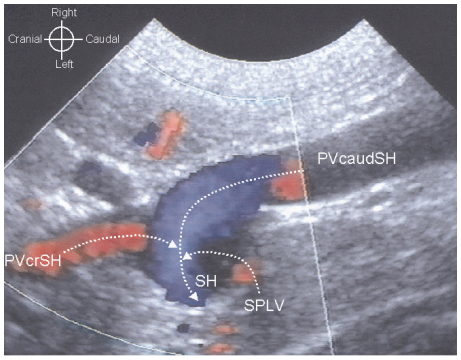
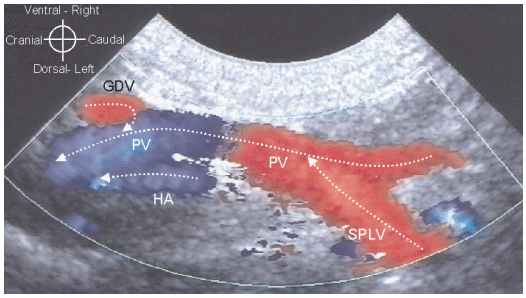


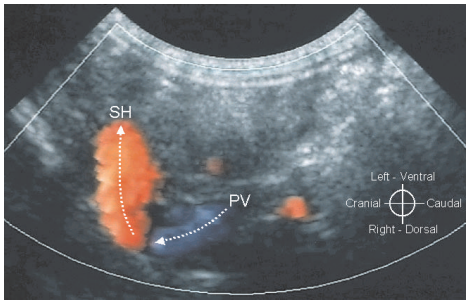
Figure 4A from Page 83.

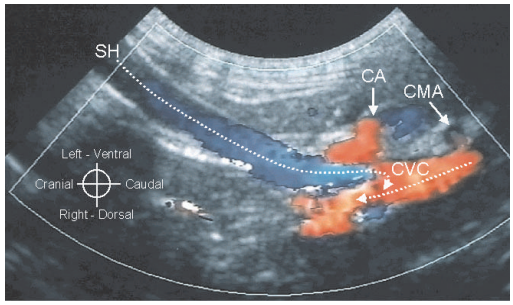




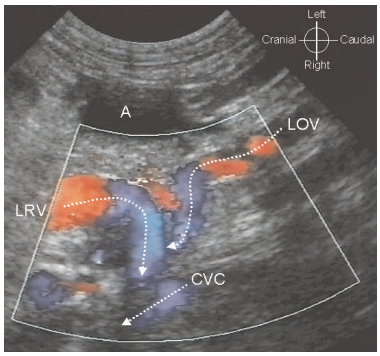
Figures 5A & B from Page 84.

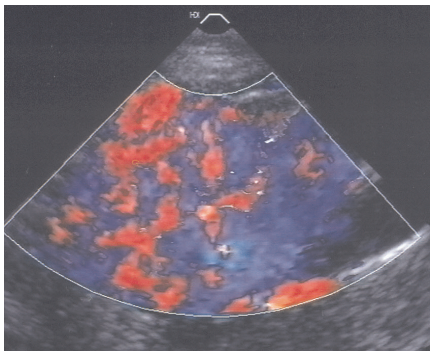
CHAPTER 5
Standard planes for ultrasonographic identification and characterization of congenital portosystemic shunts and acquired portosystemic collaterals in dogs





Figures 6A & B from Page 85.





Figures 7A & B from Page 86.

CHAPTER 6
Intraoperative ultrasonography of the portal vein during gauged attenuation of intrahepatic portocaval shunts in dogs

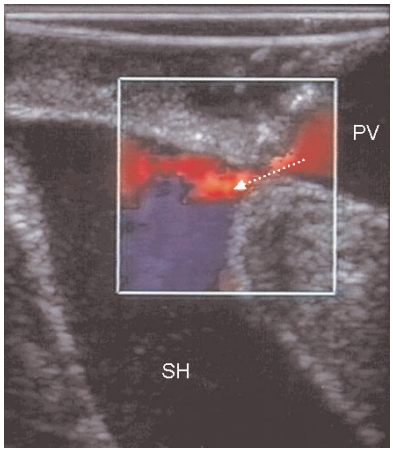


Figure 4B from Page 97. Inversed color bar!

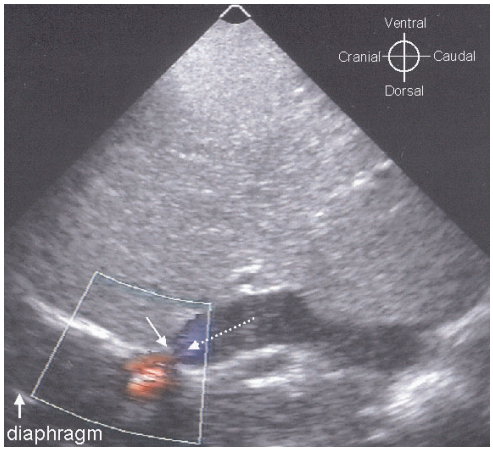
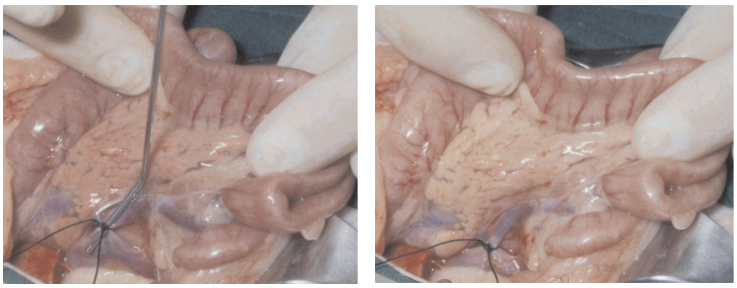


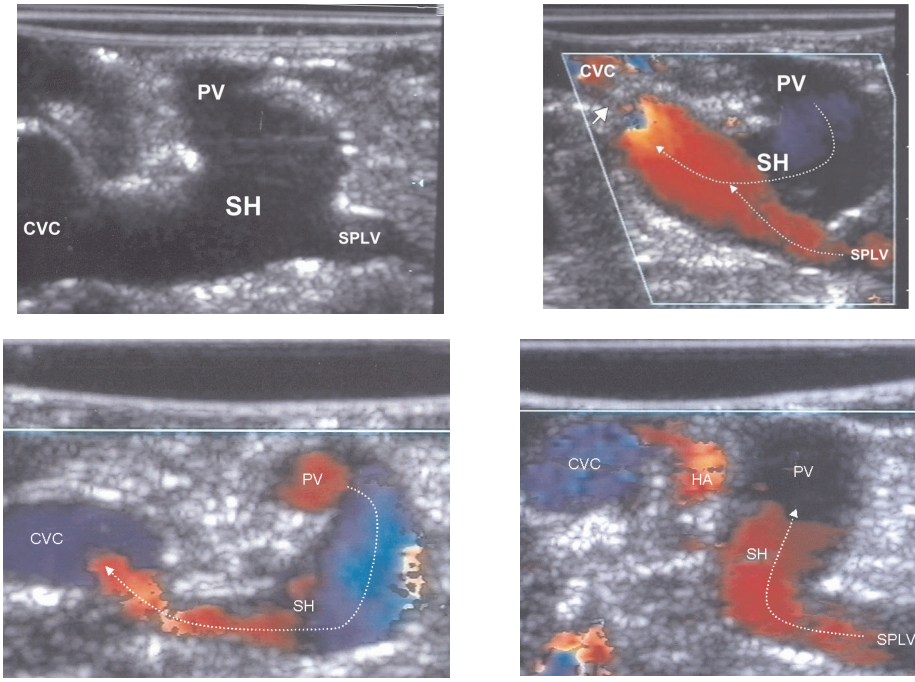
Figure 5 from Page 98.

CHAPTER 7

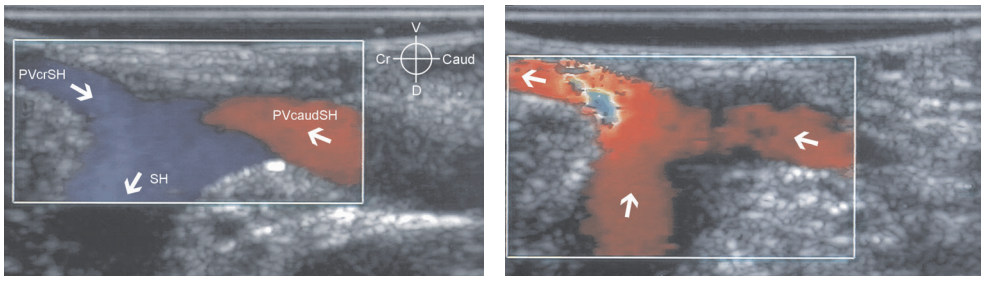
Ultrasonographic assessment of hemodynamic changes in the portal vein during surgical attenuation of congenital extrahepatic portosystemic shunts in dogs



Figures 1A & B from Page 112.



Figures 4 A, B, C & D from Page 117.



Figures 5A & B from Page 119.

CHAPTER 8
Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in dogs

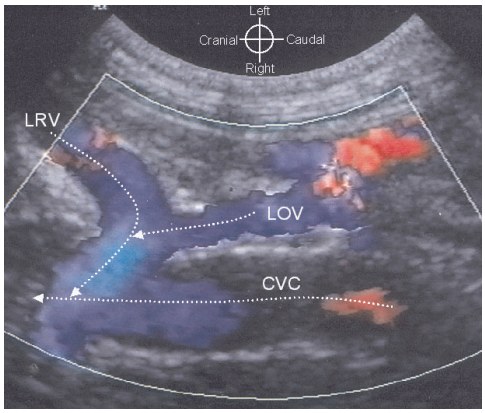


Figure 1 Page from 134.

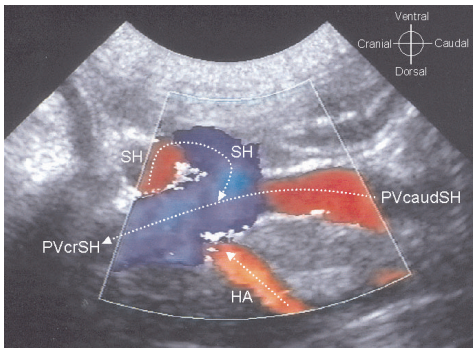


Figure 2 from Page 135.

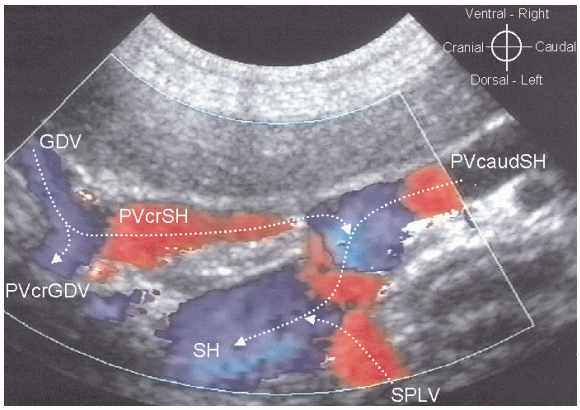


Figure 3 from Page 137.

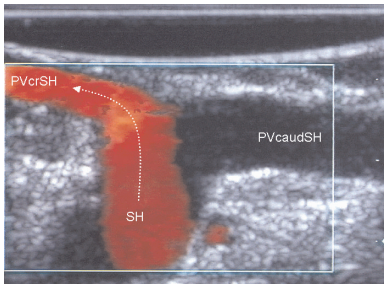
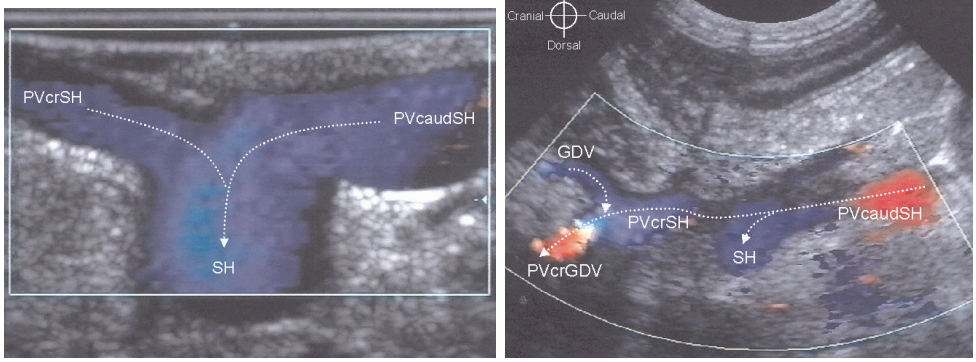
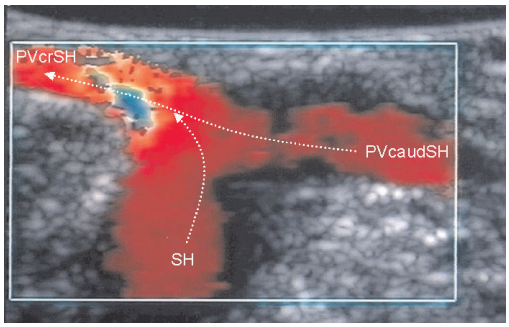


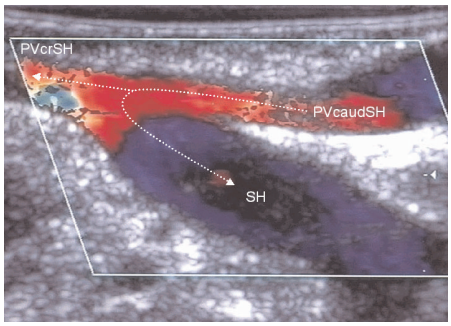
Figure 8 from Page 142.

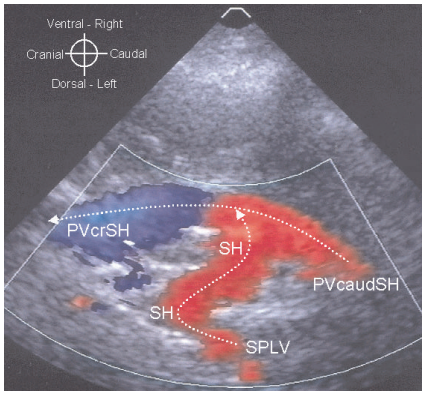


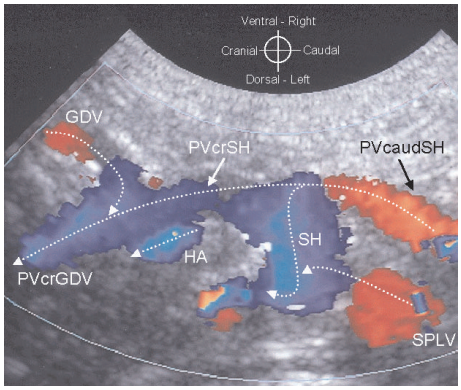
Figures 6A & B from Page 140.

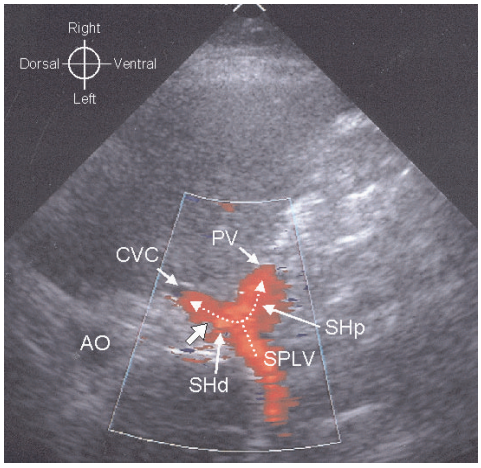
CHAPTER 8
Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in dogs

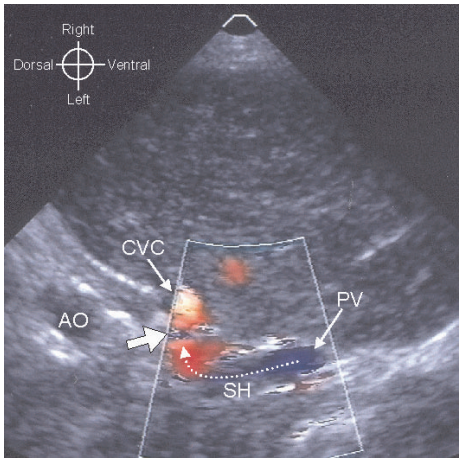












Figures 4A, B & C from Page 138.

Figures 5A, B & C from Page 138.