Ultrasonographic identification and characterization of congenital portosystemic shunts and portal hypertensive disorders in dogs and cats

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ABSTRACT

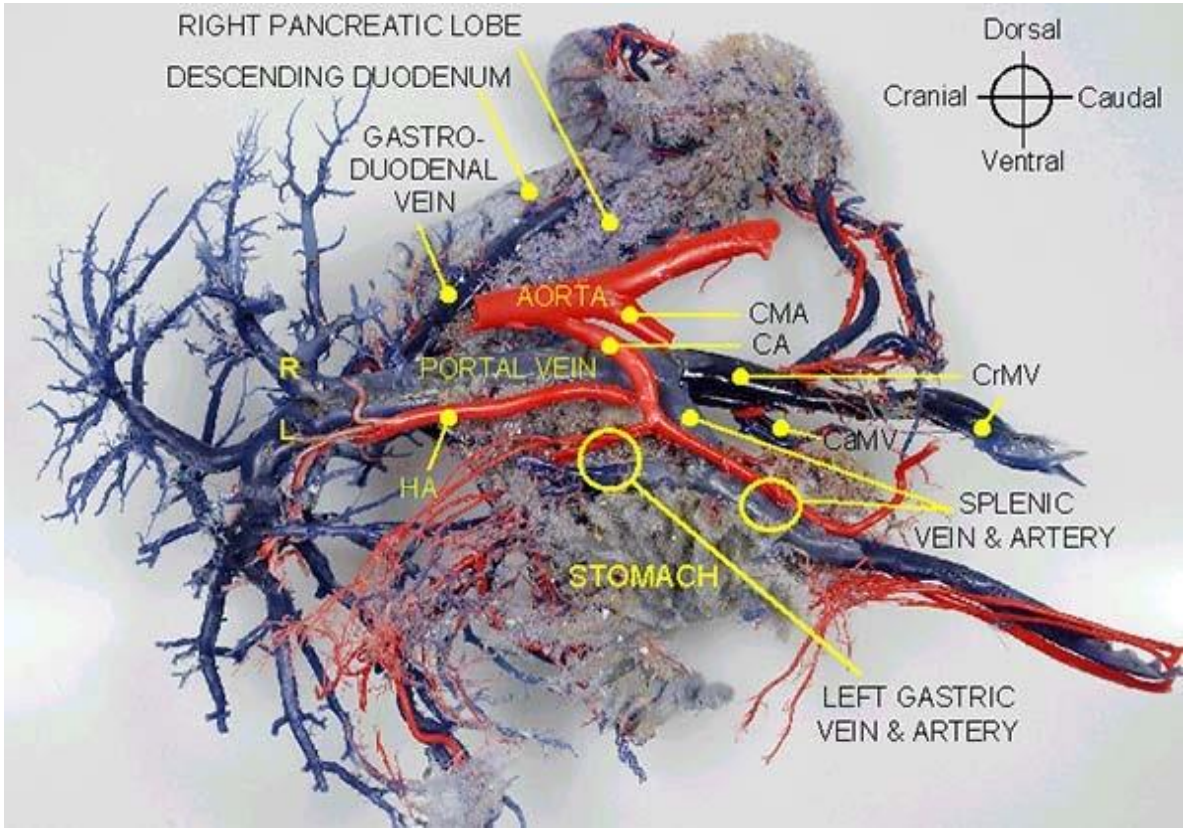
In the first part of this chapter the hemodynamic, anatomic and pathophysiologic features of canine portal vein disorders are described. Understanding these principles is necessary for correct interpretation of the ultrasonographic images. In the second part of the chapter an ultrasonographic scanning protocol is described, which is recommended to be used for thorough evaluation of the portal venous system. A short section at the very end will discuss the specific features of feline portal vein disorders.

INTRODUCTION

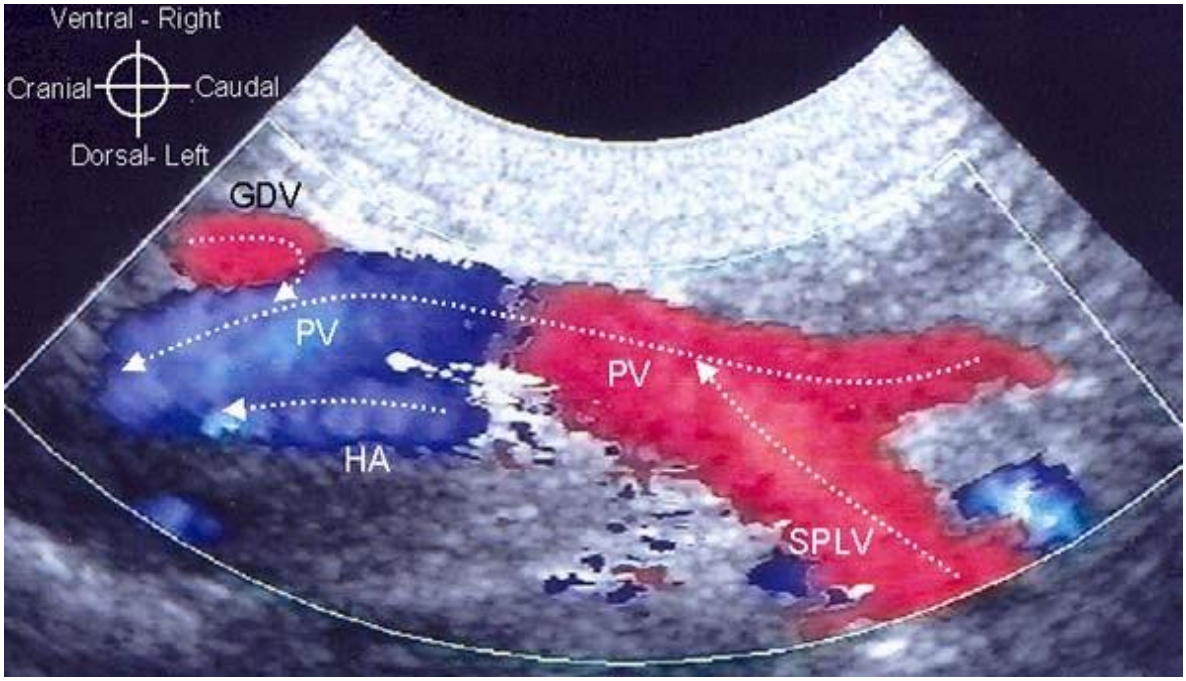
In the first part of this chapter the hemodynamic, anatomic and pathophysiologic features of canine portal vein disorders are described. Understanding these principles is necessary for correct interpretation of the ultrasonographic images. In the second part of the chapter an ultrasonographic scanning protocol is described, which is recommended to be used for thorough evaluation of the portal venous system. A short section at the very end will discuss the specific features of feline portal vein disorders.

Normal abdominal vascular anatomy in dogs

The aorta, the caudal vena cava (CVC) and the portal vein (PV) are the three great abdominal vessels, which all course parallel to the vertebral column. The aorta is the most dorsal one 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 CVC (Fig. 1). All the above described vessels can be visualized with ultrasound. The smallest intrahepatic branches of the hepatic artery terminate in the hepatic sinusoids.



A



B

Figure .1. Normal canine abdominal blood vessels.

A. *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. Before preparation the spleen, jejunum, ileum and colon were removed and the descending duodenum was retracted.*

B. *Color Doppler ultrasound image of a normal portal vein (PV) of a Yorkshire terrier. The diameter of the portal vein is uniform along its whole length. The image was made via the right flank with the dog in left lateral recumbency (plane-4). Dotted arrows indicate the direction of blood flow. Gastroduodenal vein (GDV), Splenic vein (SPLV), Hepatic artery (HA) (From Szatmári V et al. Standard planes for ultrasonographic examination of the portal system in dogs. J Am Vet Med Assoc 2004;224:713-716, with permission)*

The CVC is formed by the confluence of the right and left common iliac veins at the level of the aortic trifurcation. The CVC courses ventral to the aorta and after entering the thoracic cavity it terminates in the right atrium. The abdominal CVC collects the blood of the left and the right renal veins, and 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. 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)] The renal and hepatic veins can be visualized with ultrasound, but the gonadal veins because of their small diameter cannot.

The PV is formed by the confluence of the cranial and caudal mesenteric veins. [(2)] The PV collects the blood of the splenic vein at the level where the celiac artery originates from the aorta, as well as the blood of the gastroduodenal vein immediately caudal to the portal bifurcation. The left gastric vein is a tributary of the splenic vein. The right gastric vein is either a tributary of the gastroduodenal vein or of the PV. In the latter case it enters the PV 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 PV bifurcates into a larger left and a smaller right portal branch. The right branch courses dorsally, the left one ventrally. The right branch supplies the right lateral and the caudate hepatic lobes, whereas the left branch supplies the left lateral, left medial, quadrate and right medial lobes. The smallest portal branches terminate in the hepatic sinusoids where their blood mixes with the hepatic arterial blood. The splenic vein, the gastroduodenal vein and the left and right portal branches can be visualized with ultrasound.

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 latter terminates in the right atrium. [(2)]

The CVC 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 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. [(4-6)]

Diagnostic approach to dogs suspected of having portosystemic shunting

Because portosystemic shunting can cause a great variety of clinical signs, and ultrasonographic visualization of the anomalous veins has been a diagnostic challenge, measuring fasting venous blood ammonia level has become a routine procedure to justify or rule out the presence of portosystemic shunting. [(7, 8)]

Determining the blood ammonia level before performing an abdominal ultrasound examination can greatly increase the positive and negative predictive values of ultrasonography in finding the anomalous vein since hyperammonemia can only be caused by a few diseases such as congenital portosystemic shunts (CPSSs), acquired portosystemic collaterals (APSCs), or urea cycle enzyme deficiency. [9, 10] Differentiating these conditions non-invasively (e.g. by ultrasound) is crucial because CPSSs is the only disease that requires surgical treatment, the other two do not. Some other diseases may also cause hyperammonemia, but they can be differentiated from the above mentioned disorders by clinical examination and laboratory tests. [9]

Abdominal ultrasonography can readily diagnose CPSSs in non-sedated dogs. Moreover, the anatomy of the shunting vessel (i.e. intra- or extrahepatic) can be precisely determined. [9, 11-13] This knowledge is important not only for the surgeon, but also for predicting the prognosis. [14] The other great advantage of ultrasonography is that the other conditions causing elevated blood ammonia and bile acids levels, such as acquired portosystemic collaterals, may also be diagnosed. [9]

Pathophysiology of canine portal hypertension

Acquired portosystemic collaterals (APSCs) 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] The term prehepatic refers to disorders that affect the portal vein (i.e. extravascular compression or intravascular obstruction). [6] The term hepatic refers to the diseases of the liver itself, and the term posthepatic refers to conditions that affect the thoracic caudal vena cava (CVC) or the heart. [6] Posthepatic portal hypertension (e.g. right-sided congestive heart failure) never results in APSC-formation because not only the portal, but also the caval pressure increases. [6] Posthepatic portal hypertension results in an enlarged liver and dilated hepatic veins due to congestion, whereas prehepatic portal hypertension results in a small liver due to insufficient portal venous perfusion. In hepatic portal hypertension the small or normal sized liver have a slightly or severely abnormal (echo-)structure. A common, but not consistently occurring consequence of any kind of portal hypertension is accumulation of free abdominal fluid (pure or modified transudate). [6]

Acquired portosystemic collaterals in dogs

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. [5, 6] Collateral veins run simultaneously in several anatomic ways, however dogs tend to consistently develop the so called spleno-renal collaterals. [6] As a result of the spleno-renal collateral circulation the left gonadal vein (testicular vein in males and ovarian vein in females) becomes dilated [15] because a portion of the portal venous blood is forced to flow via the splenic vein through the preexisting embryonic connections to the left gonadal vein, and from here through the left renal vein eventually to the CVC (Fig. 2).

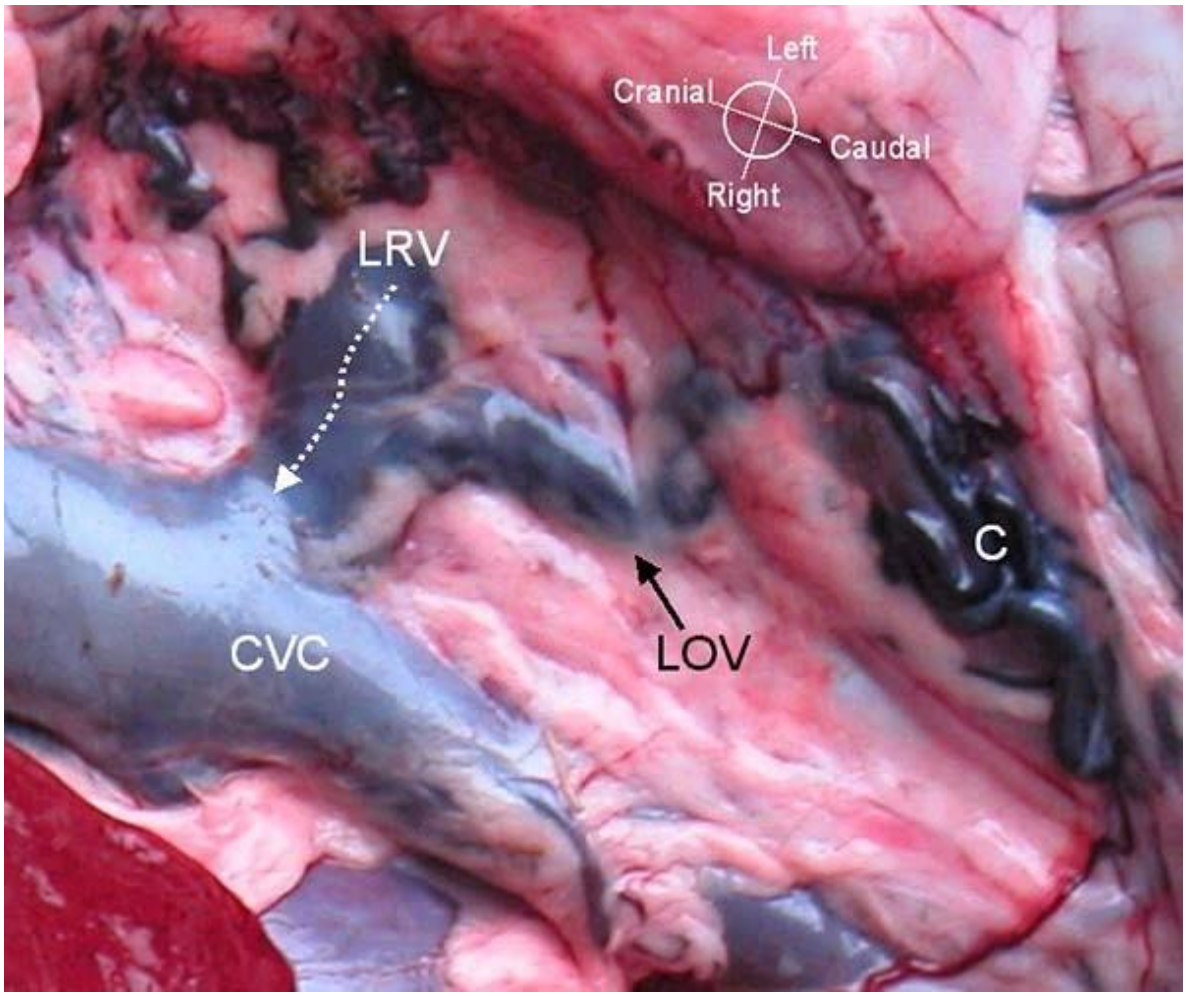
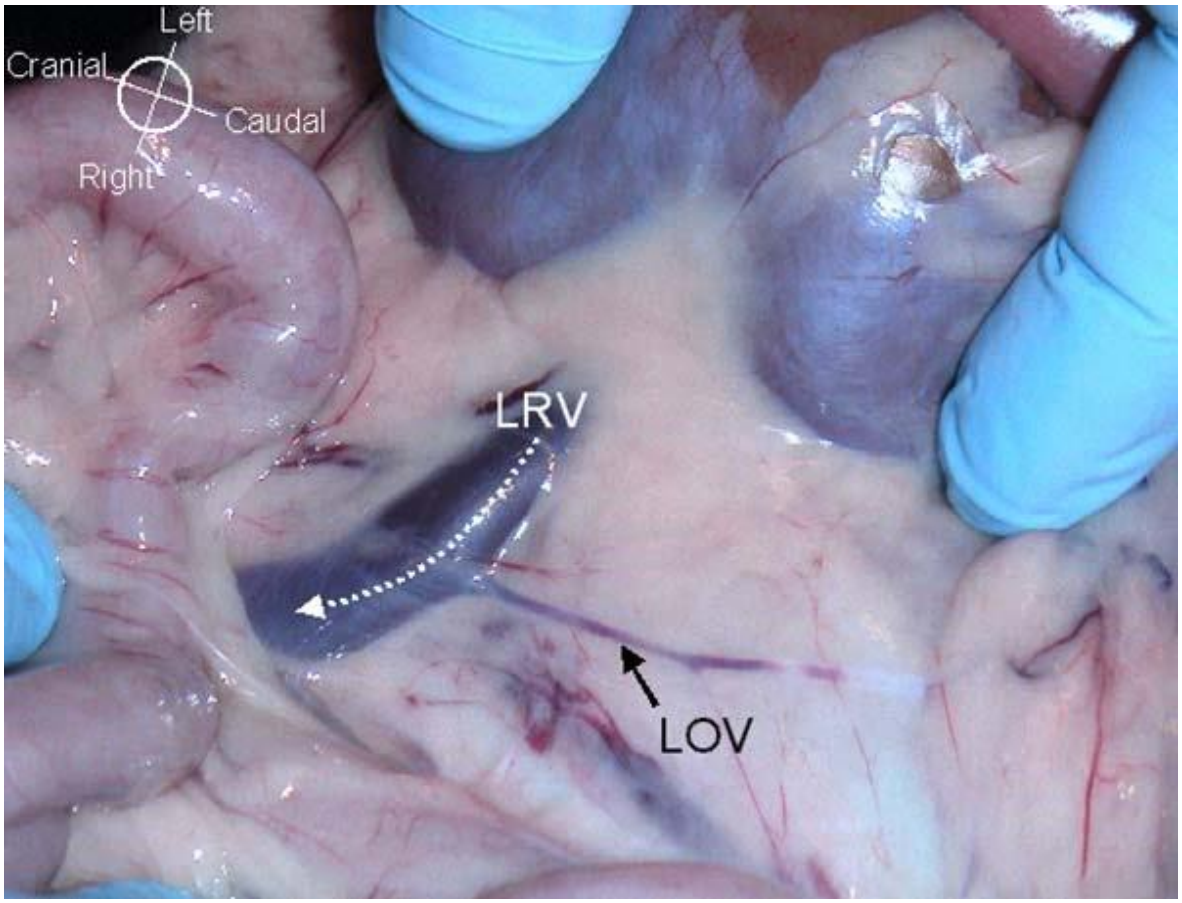


Figure 2. The left gonadal vein, which is a normal tributary of the left renal vein.

***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.*



***B**. Normal left ovarian vein in a 9-month-old female Labrador retriever. The left ovarian vein (LOV) enters the left renal vein from caudal It is much thinner than the left renal vein (LRV).*

In addition to the dilated left gonadal vein, the origin of an APSC can occasionally be found with ultrasound at the point where congenital extrahepatic spleno-caval shunts arise from the portal vein (PV) (see later). In these cases normal flow may be seen caudal to the APSC-origin and hepatofugal (i.e. away from the liver) flow cranial to it (Fig. 3A). In other dogs with portal hypertension the flow in the PV may be so slow that no color signals can be detected (Fig. 3B).

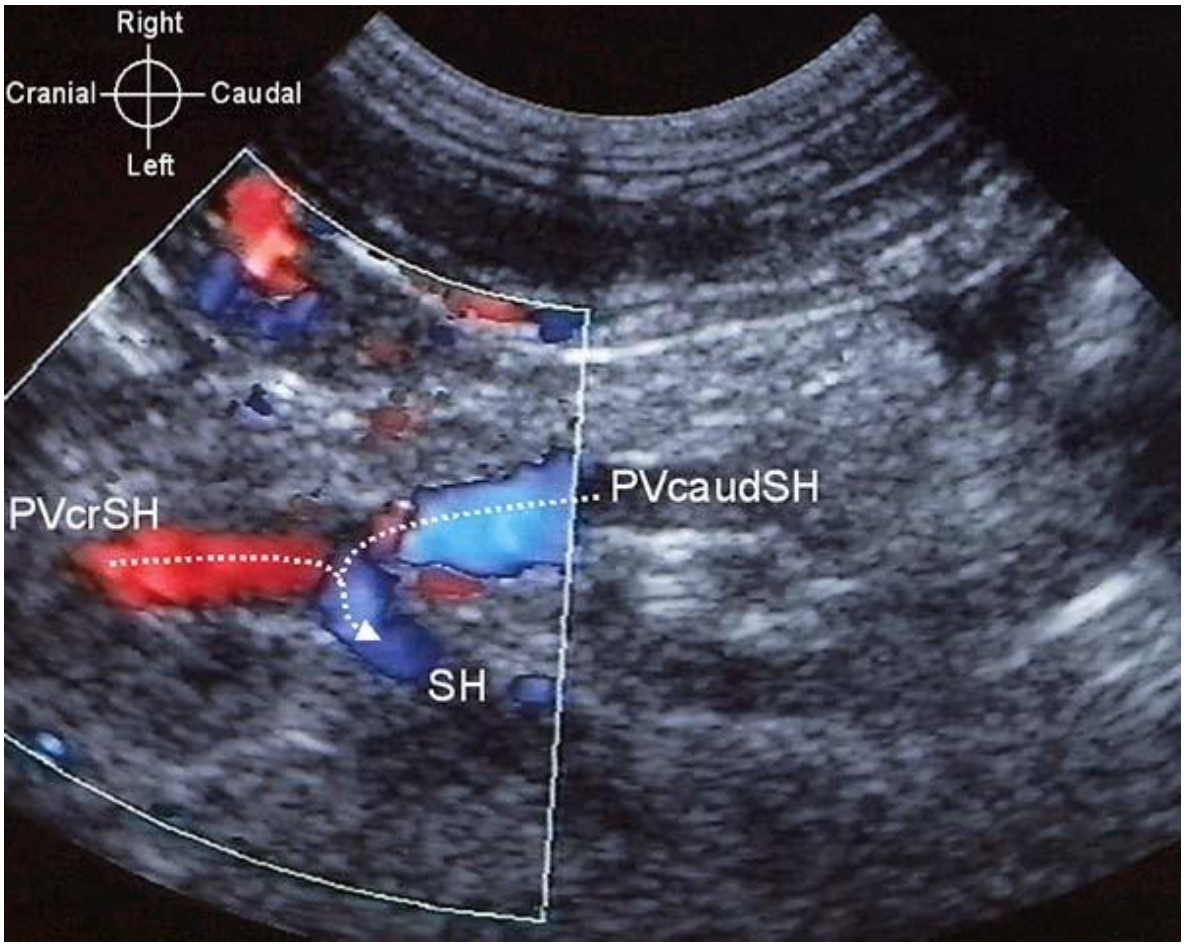


Figure 3. Portal venous flow in two dogs with hepatic portal hypertension.

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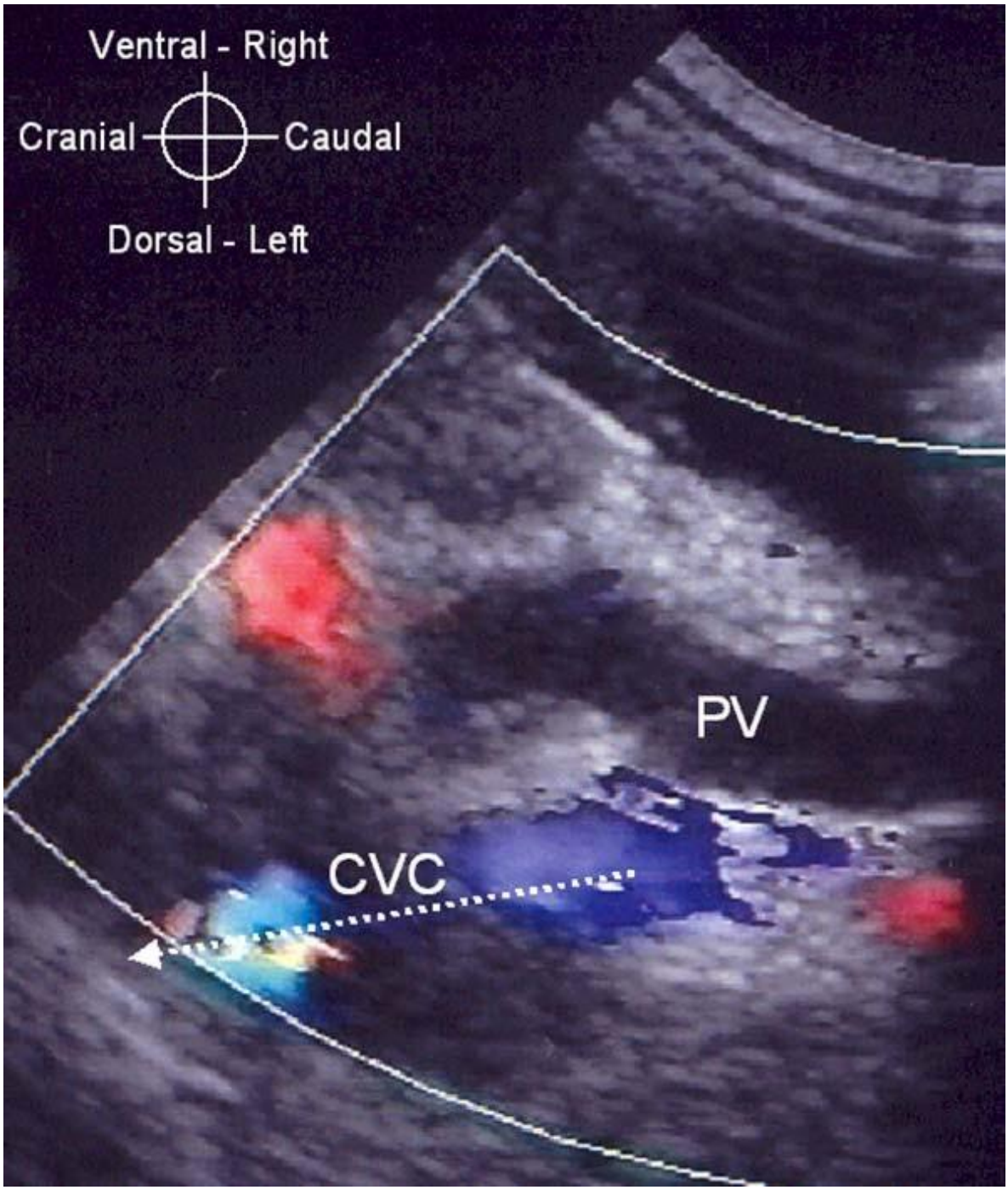
A. *Color Doppler ultrasound image of the portal vein and the origin of an acquired portosystemic collateral (SH) in a 5-year-old West highland white terrier with sustained portal hypertension of hepatic origin. Cranial to the collateral-origin (PVcrSH) hepatofugal portal flow can be seen. Note that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



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 artifact is apparent in the caudal vena cava (CVC). The dotted arrow indicates the direction of blood flow.*

Etiology of canine portal hypertension

Once the presence of portal hypertension has been established among others by visualizing a dilated left gonadal vein, the next diagnostic step is to identify the underlying cause. Ultrasonography is able to determine the cause of prehepatic portal hypertension as well as

diagnose arterioportal fistula. Hepatic portal hypertension may only be suspected with ultrasound.

Prehepatic portal hypertension can be caused by compression of the portal vein by a neoplasia or a cyst or by an obstruction of the portal vein by a thrombus or a tumor. Both conditions can readily be diagnosed with ultrasound.

Congenital arterioportal fistula is a developmental anomaly characterized by a direct connection between a portal venous and a hepatic arterial branch. [9, 16] The high arterial pressure is responsible for the classical ultrasonographic changes:

(a) extremely dilated and tortuous portal branch in a liver lobe
(b) hepatofugal flow in the PV (with a variable or an arterial Doppler spectrum)
(c) APSCs (Fig 4). [9]

Ascites (pure transudate) is usually present.

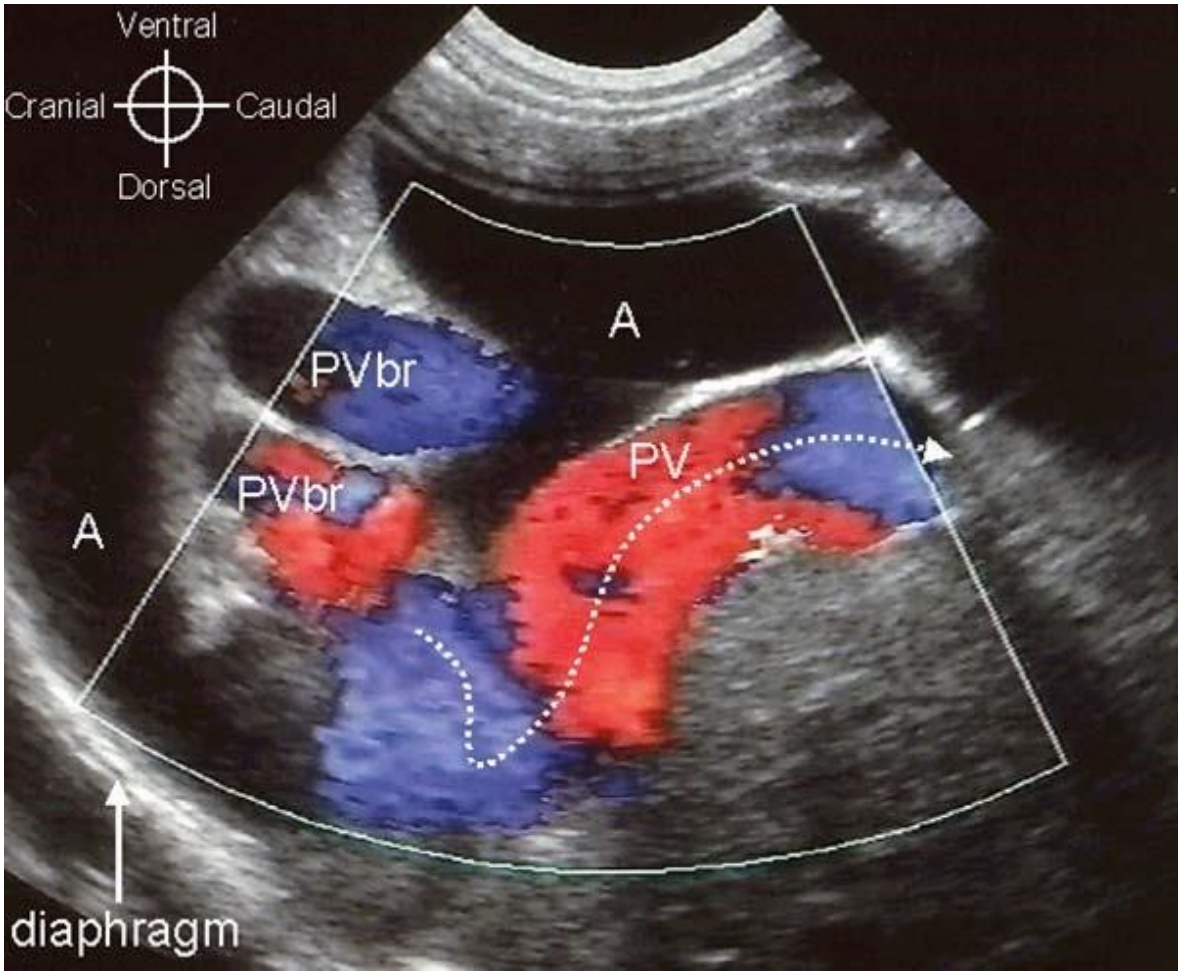


Figure 4. Congenital arterio-portal fistula.

Color Doppler ultrasound image of the liver of a 6-month-old male American Staffordshire terrier reveals dilated portal branches (PVbr) in the affected liver lobe. The flow-direction in the portal vein (PV) is hepatofugal indicated by the dotted arrow. A ascites

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Hepatic portal hypertension must be suspected in dogs with high blood ammonia level if ultrasonography discloses a dilated left gonadal vein and excludes arterioportal fistula and compression or obstruction of the PV. Hepatic portal hypertension can be caused by parenchymal liver diseases (chronic hepatitis of various etiologies) [17-19] or anomalies of the portal branches [e.g. primary hypoplasia of the portal vein (PHPV)]. [9, 20] Diagnosing and differentiating these conditions require histopathologic examination of liver biopsy specimens.

In mild cases of PHPV portal hypertension does not develop, hence blood ammonia level remains normal, however in severe cases APSCs develop as a consequence of sustained hepatic portal hypertension. [9, 20]

Primary hypoplasia of the portal vein is simultaneously present with arterioportal fistula and might accompany CPSSs. [9] When a CPSS and PHPV coincide in a dog, PHPV cannot be diagnosed preoperatively. In these dogs portal hypertension and APSCs will not develop because there is an already existing connection between the portal and the systemic veins (i.e. the CPSS). [21] Currently, the earliest time point when PHPV can be suspected in a dog with CPSS is during surgical attenuation of extrahepatic CPSSs, with intraoperative Doppler ultrasonography. [22]

Congenital portosystemic shunts in dogs

Portosystemic shunting is considered to be congenital when a single, usually large-bore vein is present without a concurrent portal hypertension. [4] Congenital portosystemic shunts are classified as intrahepatic and extrahepatic. Since a CPSS has equal or larger diameter compared to the PV-segment caudal to the shunt, it offers a lower resistance way for the blood to reach the systemic veins than the way through the hepatic sinusoids. As blood tends to flow towards the lowest possible resistance, the vast majority of the portal blood flows via the shunt because hepatic sinusoids represent much higher resistance to flow. Therefore, the liver receives no or only a trivial fraction of the portal blood, which is insufficient for a normal hepatic development and function. [4, 23]

Intrahepatic congenital porto-caval shunts

Intrahepatic porto-caval shunts occur predominantly in large breed dogs, [24] particularly often in Bernese mountain dogs and retrievers. Intrahepatic CPSSs originate either from the left or from the right portal branch and appear as the direct continuations of the PV as the diameters of the shunt and of the affected portal branch are the same as that of the PV. [9] Because the majority of blood flows through the portal branch that continues as the shunt, the contralateral portal branch remains very thin due to hypoperfusion. All intrahepatic CPSSs terminate in the CVC either directly or via a hepatic vein. [25] The intrahepatic CPSS is usually a single vein, but exceptionally they can have two loops. [9, 26]

Intrahepatic CPSSs that originate from the left portal branch courses cranioventrally and to the left (similarly to a normal left portal branch) to the diaphragm, then turns abruptly dorsally to enter the CVC via a dilated segment of the left hepatic vein. [25] In these dogs the right portal branch is very thin (Fig. 5). [9]

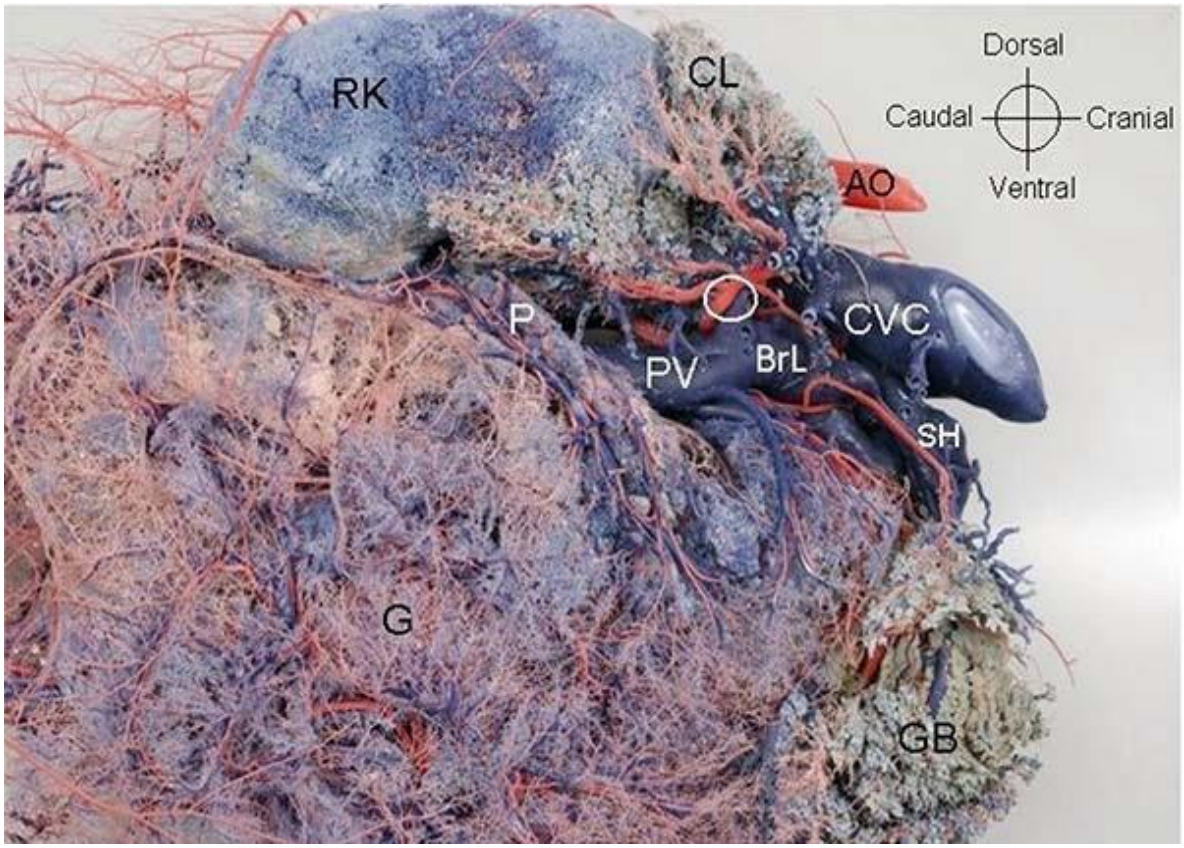


Figure 5. Patent ductus venosus.

Corrosion cast of a left divisional intrahepatic portocaval shunt of a 2-month-old Irish wolfhound. The portal vein (PV) has the same diameter as the left portal branch (BrL) and the shunt (SH). The right portal branch (O blue) is very narrow, however the corresponding hepatic arterial branch (O red) is relatively wide. CVC caudal vena cava, RK right kidney, AO aorta, CL caudate liver lobe, P pancreas, G guts, GB gallbladder.

Intrahepatic CPSSs that originate from the right portal branch appear as the direct continuation of the right portal branch (Fig. 3.6). The dilated right portal branch runs consistently dorsolaterally and to the right from the PV, like a normal right portal branch, but then, instead of tapering, it turns medially to enter the CVC. [9, 25] The dorsolaterally running segment is either short or long (i.e. central and right divisional shunt, respectively). Whatever morphology of a right-sided intrahepatic CPSS has, the left portal branch is severely underdeveloped.

A dog with simultaneous intrahepatic CPSS and arterioportal fistula has been reported. [27]

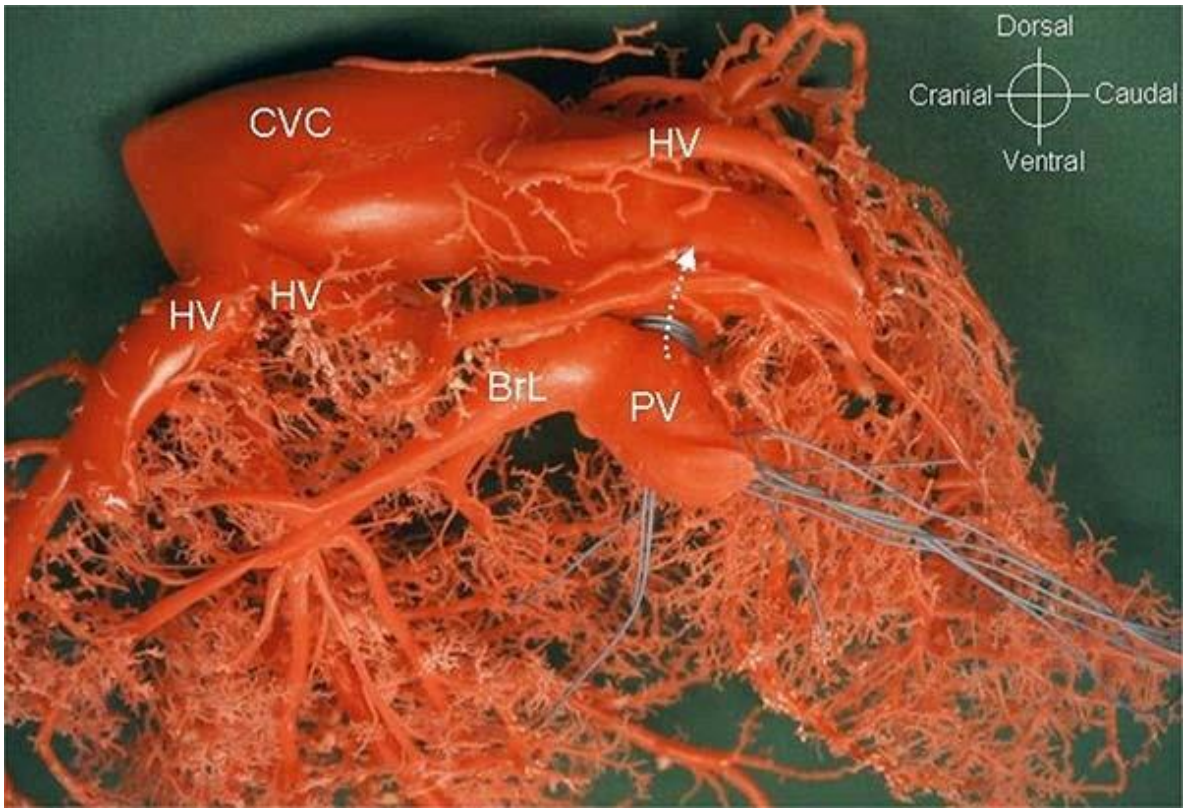


Figure 6.

Congenital intrahepatic portocaval shunt from the right portal branch.

Corrosion cast of a central divisional intrahepatic portocaval shunt of a 7-month-old mixed breed dog 3 months after partial shunt ligation. The portal vein (PV) continues via the right portal branch (arrow) to the caudal vena cava (CVC). Three months after shunt attenuation the left portal branch (BrL) is still rather narrow. HV hepatic vein. There is ligature around the shunt. The PV and CVC slightly caudal to the liver have been removed.

Extrahepatic congenital portosystemic shunts

Extrahepatic portosystemic shunts occur mostly in small breeds, particularly often in Maltese dogs, miniature schnauzers and small terriers (Yorkshire, Jack Russell, cairn), but are occasionally seen in large breeds. Extrahepatic CPSSs originate from the splenic vein, from the right gastric vein, or from both as communicating loops and enter either the abdominal CVC or the thorax: spleno-caval, right gastric-caval, spleno-azygos and right gastric-azygos shunt. [(9)] Shunts with two loops (one arising from the right gastric vein and the other one from the splenic vein) are categorized as right gastric shunts because the right gastric vein is the main loop and this has an identical morphology with the single shunts that arise from the right gastric vein. Though extrahepatic CPSSs are named after a portal tributary, they all divert the blood of the PV via a short and dilated segment of the involved tributary. Extrahepatic CPSSs with other anatomy than the above described four types are extremely rare in dogs. [(28)]

Extrahepatic CPSSs arising from the splenic vein

Spleno-caval shunts are the most common type of congenital extrahepatic CPSSs. They usually form a short loop between the PV and the CVC. Although the anomalous vein may have a long cranially extending loop, the points of origin and termination are always the same. Since the point of shunt origin is very close to the point where the splenic vein enters the PV and the short segment of the splenic vein that is between the PV and the shunt-origin is dilated

and the flow hepatofugal (i.e. away from the liver) in it, the CPSS seems to originate from the PV itself and the splenic vein seems to enter the shunting vessel (Fig. 7A). The origin of the spleno-caval shunts is slightly cranial to the level where the celiac artery originates from the aorta (i.e. approximately at the level of the cranial pole of the right kidney, Fig. 7B). The termination of the spleno-caval shunts in the CVC is always at the same point, i.e. slightly cranial to the level of the shunt-origin (Fig 8A). [9, 13]

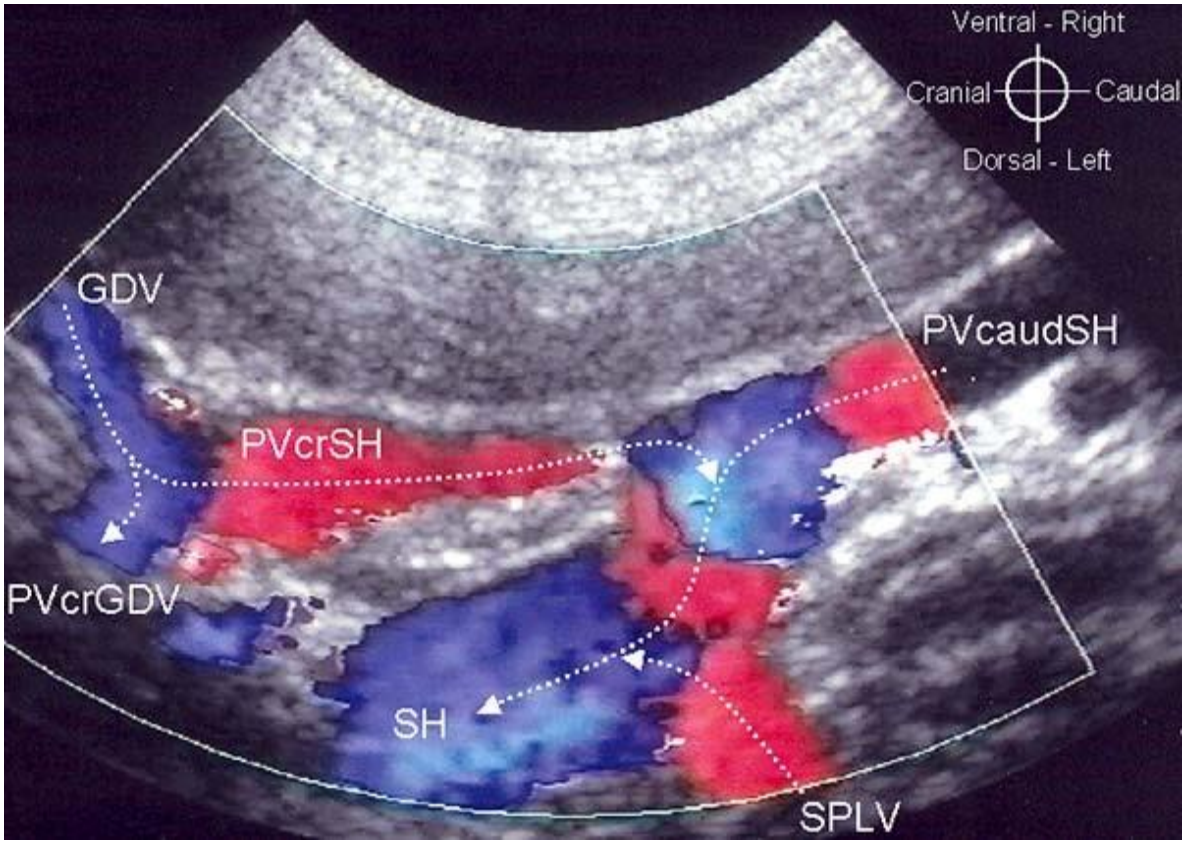
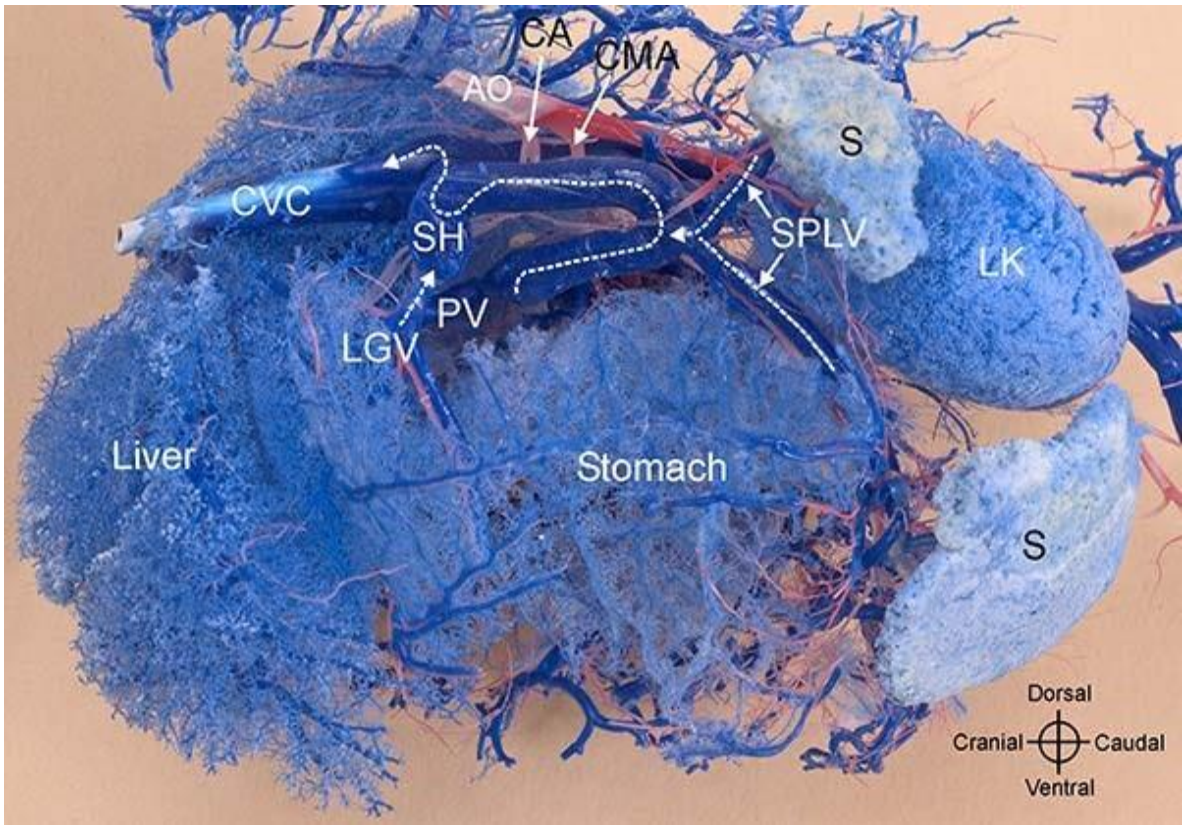


Figure 7. Congenital extrahepatic spleno-caval shunt.

A. Color Doppler ultrasound image of the portal vein in longitudinal section made via the right flank with the dog in left lateral recumbency (plane-4). The shunt (SH) originates from the splenic vein (SPLV), very close to the point where the SPLV normally enters the portal vein. Both the intestinal and the splenic blood are diverted. In the portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein (PVcrSH) the flow is hepatofugal. This PVcrSH is thinner than the portal vein caudal to the shunt-origin (PVcaudSH). Cranial to the entering point of the gastroduodenal vein (GDV) the portal flow is hepatopetal (PVcrGDV). Dotted arrows indicate the direction of blood flow. (From Szatmári V et al. Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in 14 dogs. Vet Rec 2004;155:448-456, with permission)



B. Corrosion cast of the abdominal blood vessels of a 2-month-old cairn terrier. The veins are blue and the arteries are red. The shunt (SH) terminates in the caudal vena cava (CVC) slightly cranial to the point where the celiac artery (CA) originates from the aorta (AO). The left gastric vein (LGV) and both the dorsal and ventral branches of the splenic vein (SPLV) enter the SH. The portal vein (PV) is thin cranial to the SH-origin. CMA Cranial mesenteric artery, LK left kidney, S spleen (the middle part of the spleen has been removed). Dotted arrows indicate the direction of blood flow in a living animal.

In cases of spleno-azygos shunts, the shunting vessel approaches the CVC at the point where the spleno-caval shunts terminate, but instead of entering it the shunting vessel courses dorsal to the CVC and eventually enters the thorax (Fig 8B). The point of origin of these spleno-azygos shunts is the same as that of the spleno-caval shunts (Fig 7A).

Since the diameter of the shunt is always wider than that of the PV caudal to the shunt-origin the PV cranial to the shunt-origin and the intrahepatic portal branches remain hypoperfused. Therefore, the PV-segment cranial to the shunt-origin is always thinner than the PV-segment caudal to the shunt-origin. In most cases of extrahepatic CPSSs the left and right portal branches are also very thin. When they have a relatively normal diameter, color Doppler shows an undetectably slow or very slow hepatopetal (i.e. towards the liver) flow in them. The flow-direction in the PV-segment cranial to the shunt-origin is hepatofugal in most dogs, however could be slow hepatopetal (i.e. flow to the liver) or alternating ("to-and-fro"). 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 (i.e. to caudal) than towards the hepatic sinusoids (Fig 3.7A).

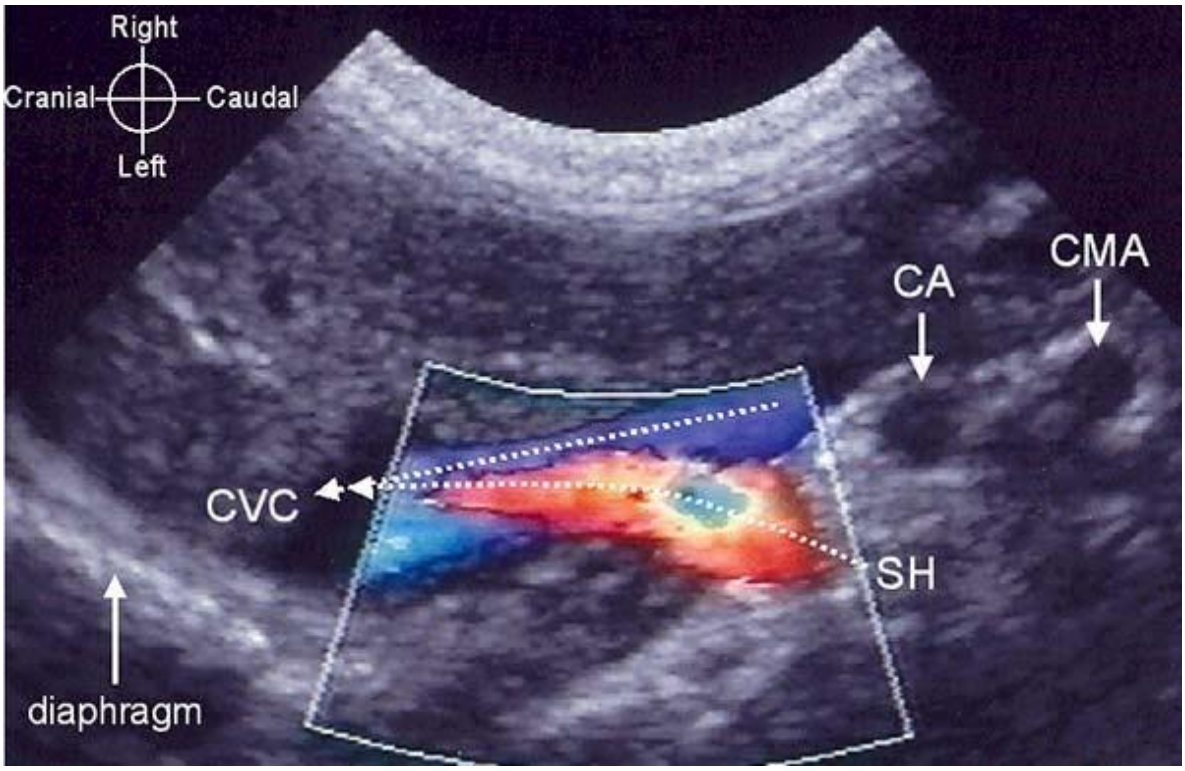
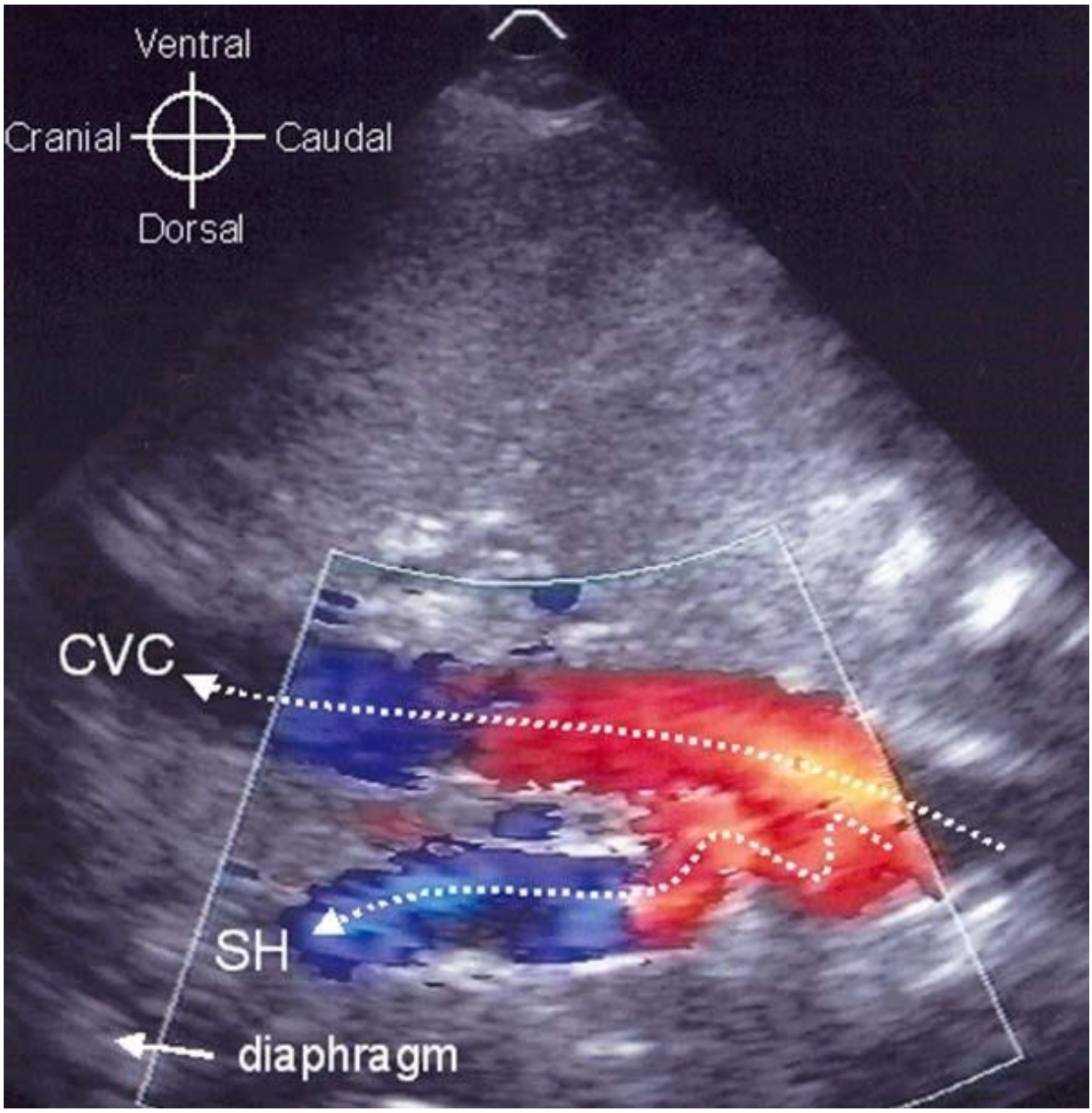


Figure 8. Congenital extrahepatic portosystemic shunts in two dogs.

Color Doppler ultrasound images show the termination of congenital extrahepatic portosystemic shunts. The transducer was positioned caudal to the last right rib (flank) with the dog in dorsal recumbency. Dotted arrows indicate the direction of blood flow. (From Szatmári V et al. Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). J Am Vet Med Assoc 2004;224:717-727, with permission)

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



B. Spleno-azygos shunt in a 3.5-month-old male Jack Russell terrier. The shunting vessel (SH) courses dorsal to the CVC, and enters the thorax.

The morphology of the APSCs that originate from the PV can be very similar to that of congenital extrahepatic spleno-caval shunts (Figs 3A,.7A). Moreover, in both cases hepatofugal portal flow cranial to the origin of the anomalous vein could be seen. The differences are: an APSC runs caudally from its origin and tends to disappear among the intestines, furthermore the diameters of the PV cranial and caudal to the APSC-origin are roughly equal (Fig 3A). In contrast, congenital extrahepatic spleno-caval or spleno-azygos shunts tend to run cranially from the origin and can always be followed to their terminations (CVC or diaphragm). Thirdly, the PV-segment that is cranial to the origin of a spleno-caval or a spleno-azygos shunt is always thinner than the PV-segment caudal to it (Fig.7). Moreover, an APSC is thinner than the PV caudal to the anomalous vein, unlike in cases of CPSSs. The simultaneous presence of the dilated left gonadal vein proves that an extrahepatic anomalous vein originating from the PV with a hepatofugal flow is the origin of an APSC and not that of a CPSS. [(9)]

Extrahepatic CPSSs arising from the right gastric vein

Right gastric-caval shunts have either one or two loops. In the former case only the cranial loop (right gastric-caval loop) is present (Fig 9 A, B). In the latter case both the cranial loop and the caudal loop (spleno-caval loop) are present and they anastomose before entering the CVC (Fig 10).

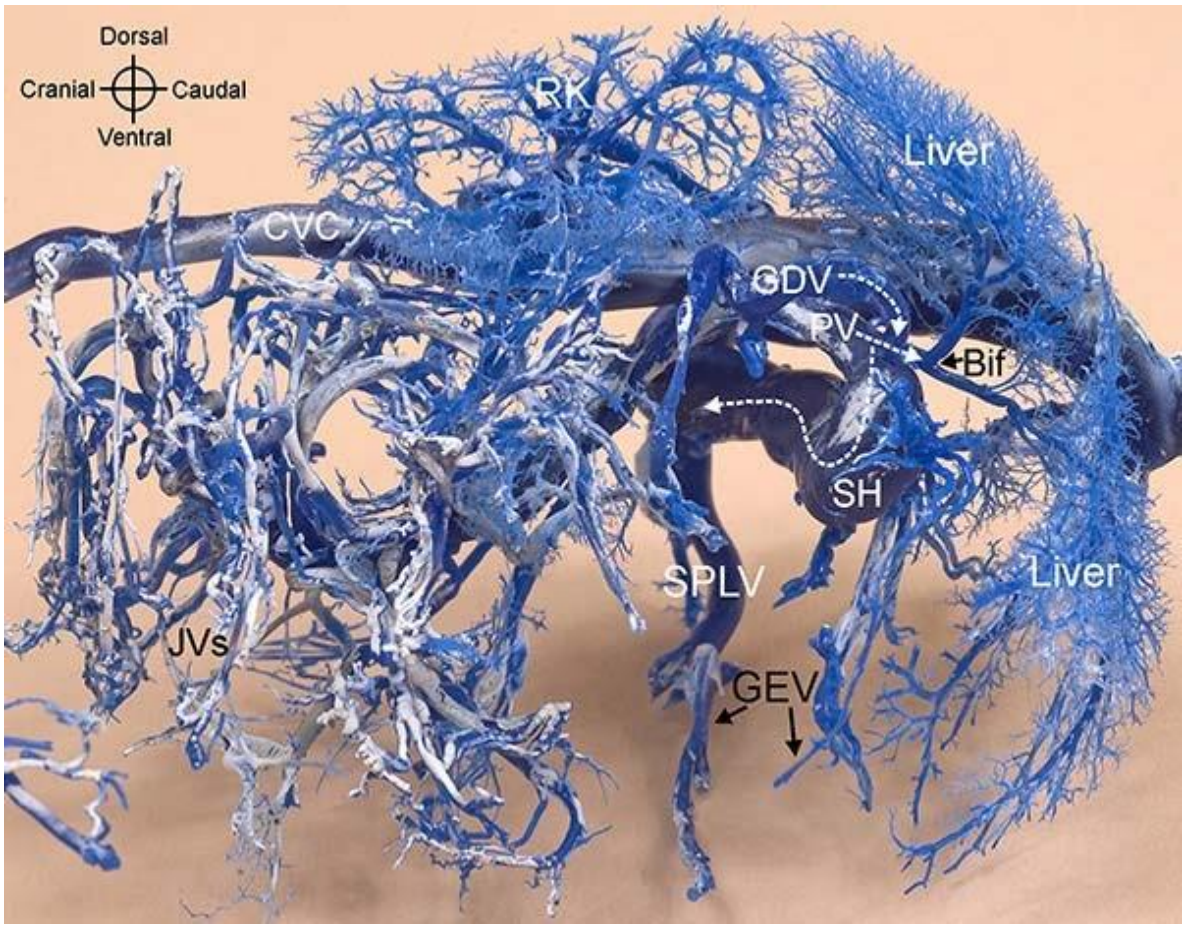
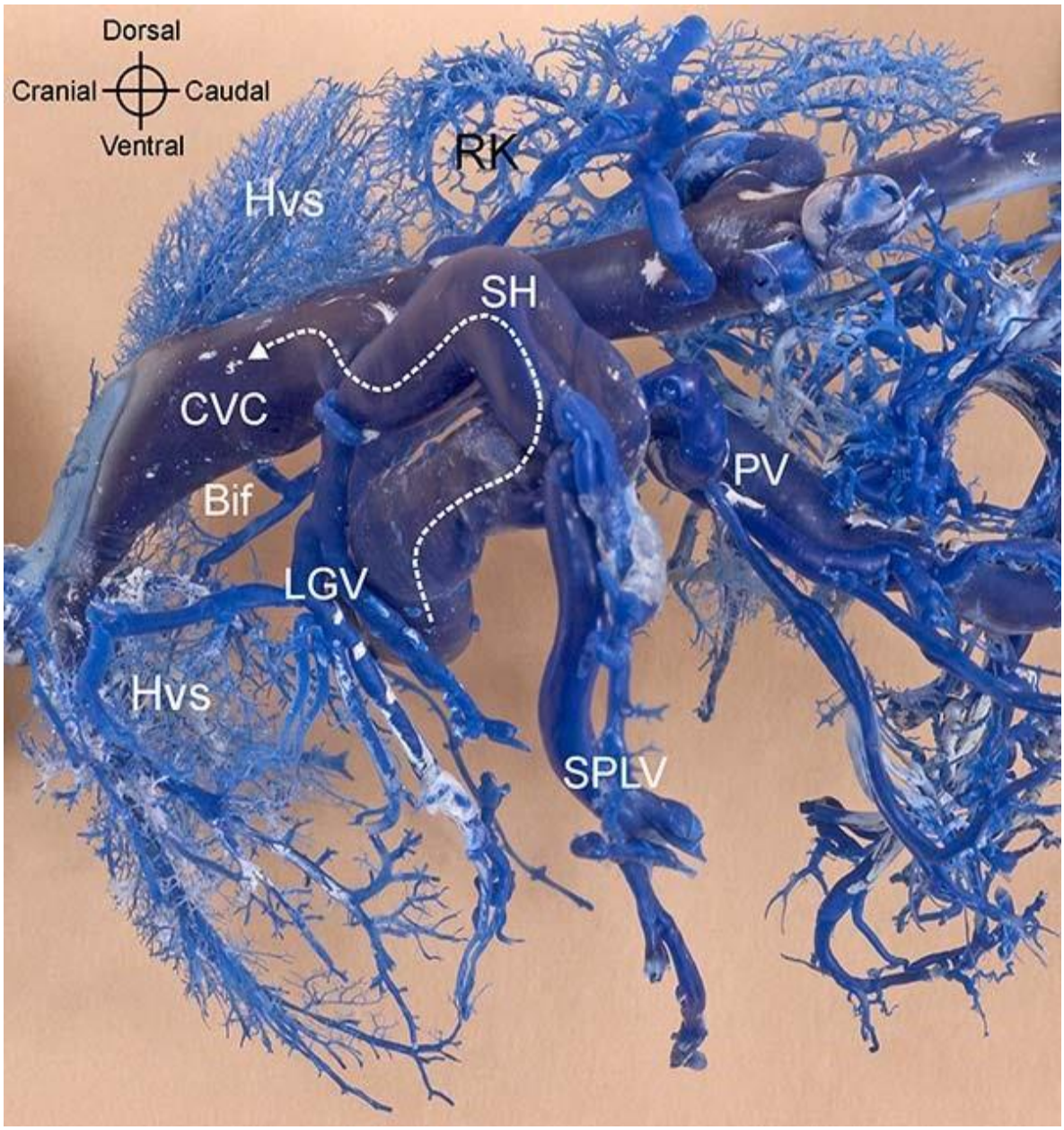


Figure 9. Right gastric-caval shunt with one shunt loop.

Corrosion cast of the abdominal veins of a Dachshund with congenital extrahepatic right gastric-caval shunt. In this patient only the cranial shunt-loop (SH) has developed. The left kidney has been removed. RK right kidney

A. The shunt (SH) originates at the point where the gastroduodenal vein (GDV) enters the portal vein (PV). The right gastric vein is a tributary of the GDV. The PV caudal to the SH-origin is thinner than the SH and becomes very thin cranial to the SH-origin. The right portal branch runs to dorsal and the left portal branch to ventral. Bif portal bifurcation, CVC caudal vena cava, GEV left and right gastroepiploic veins, JVs jejunal veins, SPLV splenic vein



B. The portal vein (PV) becomes very thin cranial to the origin of the SH. Compare the PV-diameter caudal to the SH with that just caudal to the PV-bifurcation (Bif). Both the splenic vein (SPLV) and the left gastric vein (LGV) enter the SH. The SH terminates in the caudal vena cava (CVC). HVs hepatic veins.

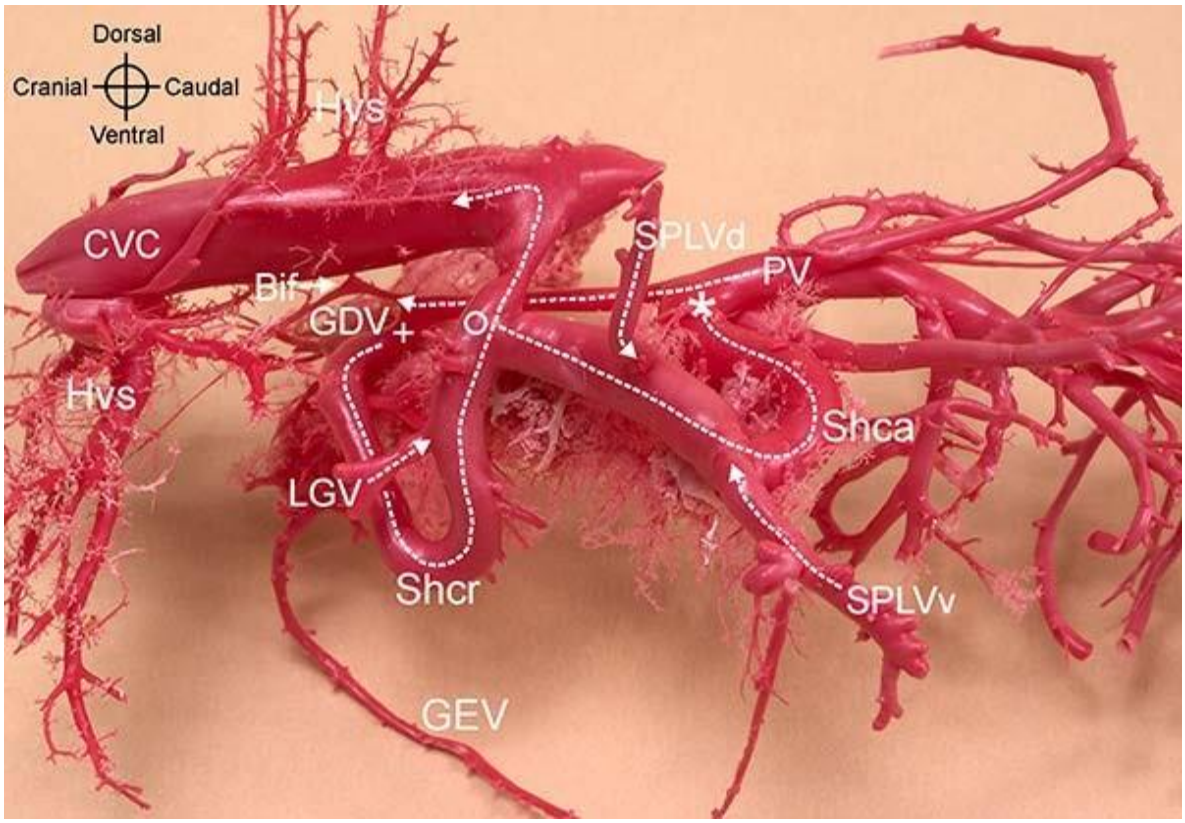


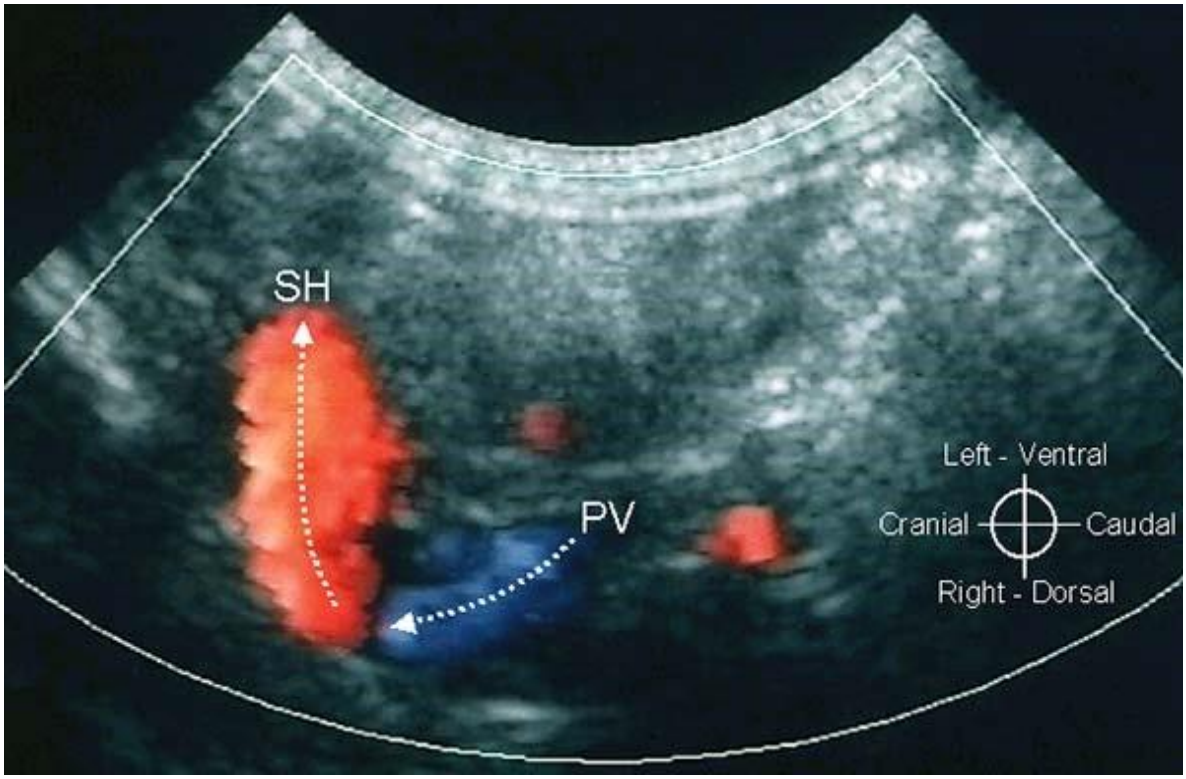
Figure 10. Right gastric-caval shunt with two shunt loops.

Corrosion cast of the abdominal veins of a Yorkshire terrier with a right gastric-caval shunt. Both the cranial shunt-loop (Shcr) and the caudal shunt-loop (SHca) are present and they anastomose with each other (o). The portal vein (PV) becomes narrower cranial to the point of the Shca-origin (*), and even more narrow cranial to the point of the Shcr-origin (+). The Shcr originates at the point where the gastroduodenal vein (GDV) enters the PV. By surgical closure of the common trunk of the SH adjacent to the caudal vena cava (CVC) both shunt-loops can be ceased. The arrows indicate the flow-directions in the vessels in a living animal. Bif Portal bifurcation with the very thin right and left portal branches, HVs Hepatic veins, GEV Right and left gastroepiploic veins, SPLVv Ventral branch of the splenic vein, SPLVd Dorsal branch of the splenic vein. (The CVC has been removed caudal to the SH-termination.)

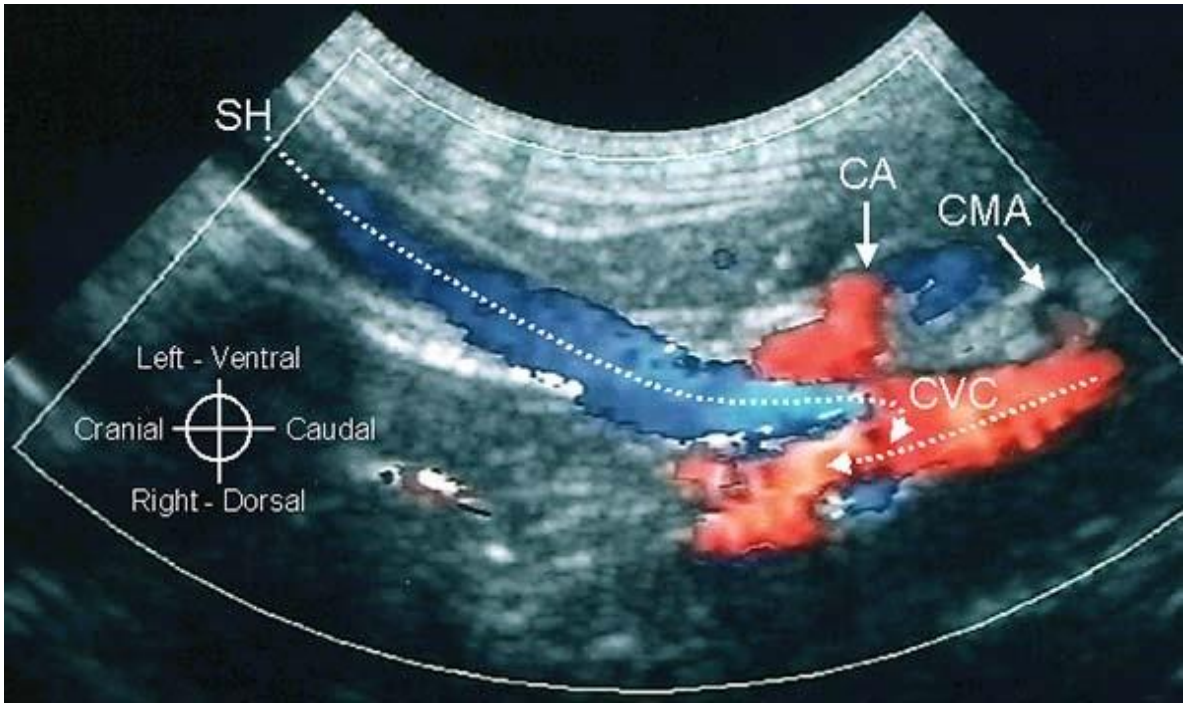
The cranial loop arises from the right gastric vein and the morphology of this loop varies slightly depending whether the right gastric vein is a tributary of the gastroduodenal vein or of the PV itself originating between the portal bifurcation and the gastroduodenal vein. [(3)] In both cases the shunt originates immediately caudal to the portal bifurcation via the dilated right gastric vein. When the right gastric vein is a direct tributary of the PV, the blood of the PV is drained via the dilated right gastric vein to the CVC. When the right gastric vein is a tributary of the gastroduodenal vein, 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 and the blood of the gastroduodenal vein flows through the shunt (i.e. the right gastric vein) without reaching first the PV. Regardless of the anatomic variation of the right gastric vein, the course of the shunt (i.e. the right gastric vein) is always the same, namely it makes a long loop from the liver hilus first laterally to the left body wall, then from here to caudomedially to eventually enter the CVC at the point where the spleno-caval shunts terminate (i.e. slightly cranial to the celiac

artery, Fig 11). The caudal shunt-loop of a right-gastric-caval shunt resembles a spleno-caval shunt.

Exceptionally, the shunting vessel does not enter the CVC, but courses dorsal to it and enters the thorax (right gastric-azygos shunts).



A



B

Figure 11. Right gastric-caval shunt.

Color Doppler ultrasound images of (the cranial loop of) a congenital extrahepatic right gastric-caval shunt in a 6.5-month-old female Yorkshire terrier. The dog is in right lateral recumbency and the transducer is placed caudal to the last left rib (plane-6). Dotted arrows indicate the direction of flow.

A. The shunt (SH) originates at the liver hilus and runs towards the left body wall making a roughly 90° angle with the portal vein (PV). The continuation of the shunt (traced caudally) is shown in Figure B. The PV cannot be seen cranial to the shunt origin because it is extremely thin due to hypoperfusion (see the corrosion cast).

B. The shunt (SH) terminates in the caudal vena cava (CVC) cranial to the celiac artery (CA), similarly to a congenital extrahepatic spleno-caval shunt. Note the large-caliber shunting vessel (SH) immediately under the left body wall. CMA cranial mesenteric artery. (From Szatmári V et al. Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). J Am Vet Med Assoc 2004;224:717-727, with permission)

All CPSSs that arise from the right gastric vein are very wide, with a diameter comparable to that of the CVC. At surgical exploration the cranial loop of the right gastric-caval shunt is found to follow the lesser curvature of the stomach, similarly to a normal right gastric vein. The caudal shunt loop, which is not consistently present, originates at the region where spleno-caval shunts are expected, but unlike the spleno-caval CPSSs, it courses from caudal to cranial and not from ventral to dorsal like the spleno-caval shunts do. The caudal loop drains the blood of the PV via the dilated segment of the splenic vein to the common trunk (Fig 10). The PV becomes slightly thinner cranial to the origin of the caudal shunt-loop (i.e. cranial to the splenic vein) with hepatopetal flow direction. The PV cranial to the origin of the cranial shunt-loop is so thin that cannot be visualized by ultrasound. [9] Right gastric-caval shunts are frequently found in Maltese dogs.

Hyperammonemia without portal vein disorder

Urea cycle enzyme deficiency is a rare congenital metabolic disease. [10] Since no morphologic changes are present, the abdominal ultrasound examination reveals normal sized and structured liver and kidneys and the absence of vascular abnormalities. [9]

Peritoneal absorption of ammonia containing urine may result in hyperammonemia, [29] however this condition can easily be differentiated from portosystemic shunting by measuring high plasma creatinine concentration.

Irish wolfhound pups have a physiologic period of hyperammonemia. [30]

Abdominal ultrasonography of portal vein disorders

Secondary changes

Before starting the examination of abdominal vessels, a detailed B-mode ultrasonographic study of the abdominal organs has to be performed. Determining the presence and amount of free abdominal fluid, the size and structure of the liver and kidneys is particularly important. The size of the left and right halves of the liver should be separately evaluated. The urinary bladder should also be examined for the presence of sediment or stone.

Typical findings in dogs with CPSS is a small liver, normal or enlarged kidneys (often with hyperechoic medulla) and no free abdominal fluid, [9, 11, 13] whereas dogs with APSCs have small, normal sized or asymmetric liver (usually the left side is small and the right side is enlarged), variable amount of ascites (from none to large amount) and slightly or markedly abnormal hepatic echo structure. [9] The kidneys are of normal size of acquired diseases, however may be enlarged with congenital portal hypertensive disorders such as PHPV. Normal sized liver and kidneys do not exclude CPSSs.

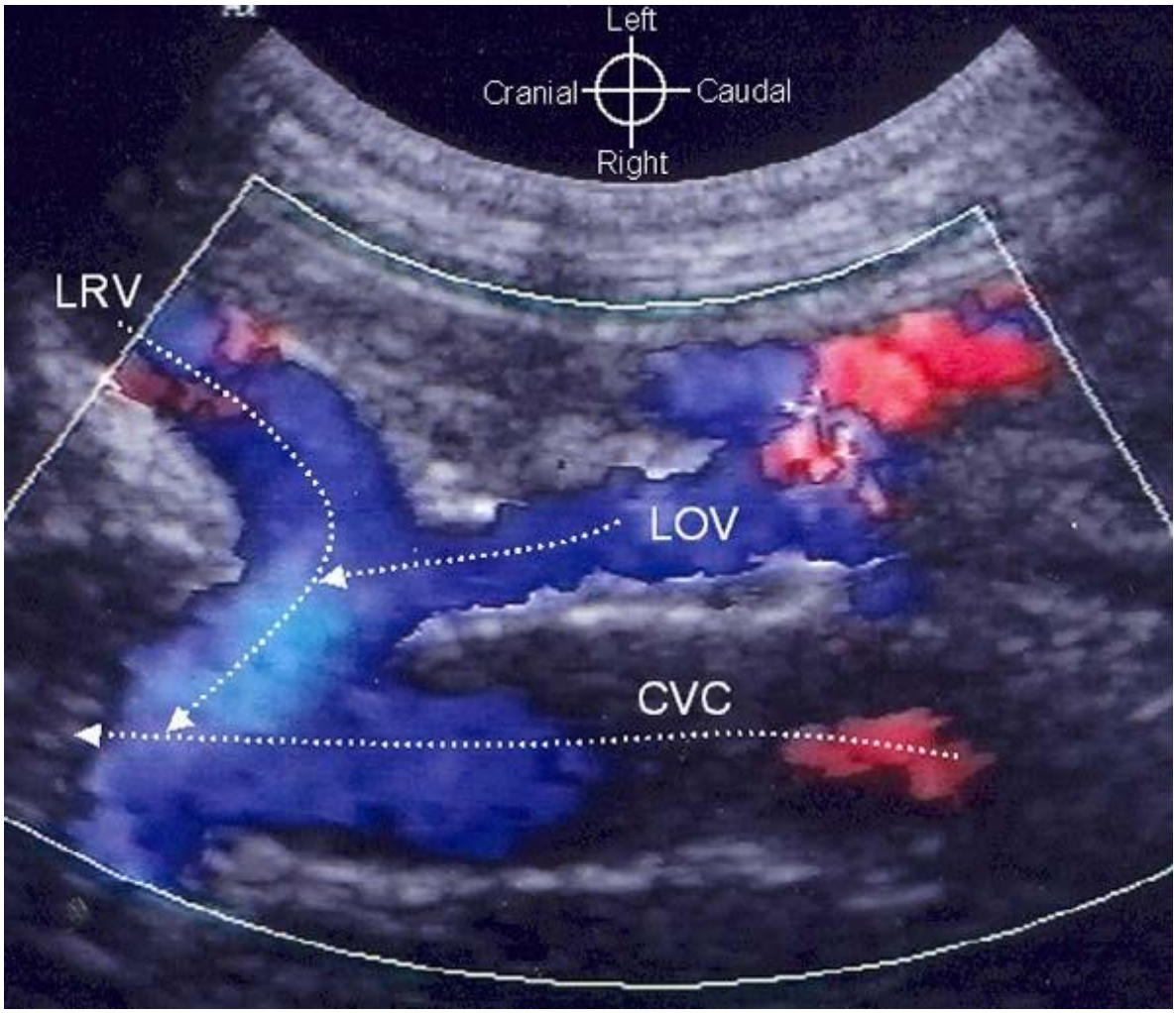
Large amount of free abdominal fluid may hinder ultrasonographic visualization of the abdominal vessels, but hyperammonemia in dogs with severe peritoneal effusion cannot possibly be the result of CPSSs or an urea cycle enzyme deficiency, since a dog with CPSS cannot have portal hypertension or so severe hypoalbuminemia that would result in formation of a large amount of transudate in the abdominal cavity. [6] Small amount of free abdominal fluid is normal in healthy pups, hence it may also be seen in puppies with CPSSs or with urea cycle enzyme deficiency. [9]

The spleen has usually a normal size in dogs with both CPSSs and APSCs. [9] The reason why splenomegaly does not develop in dogs with portal hypertension could be that acquired spleno-renal collaterals are the most consistently developing APSCs in dogs, and these collateral veins prevent the spleen from congestion. [9]

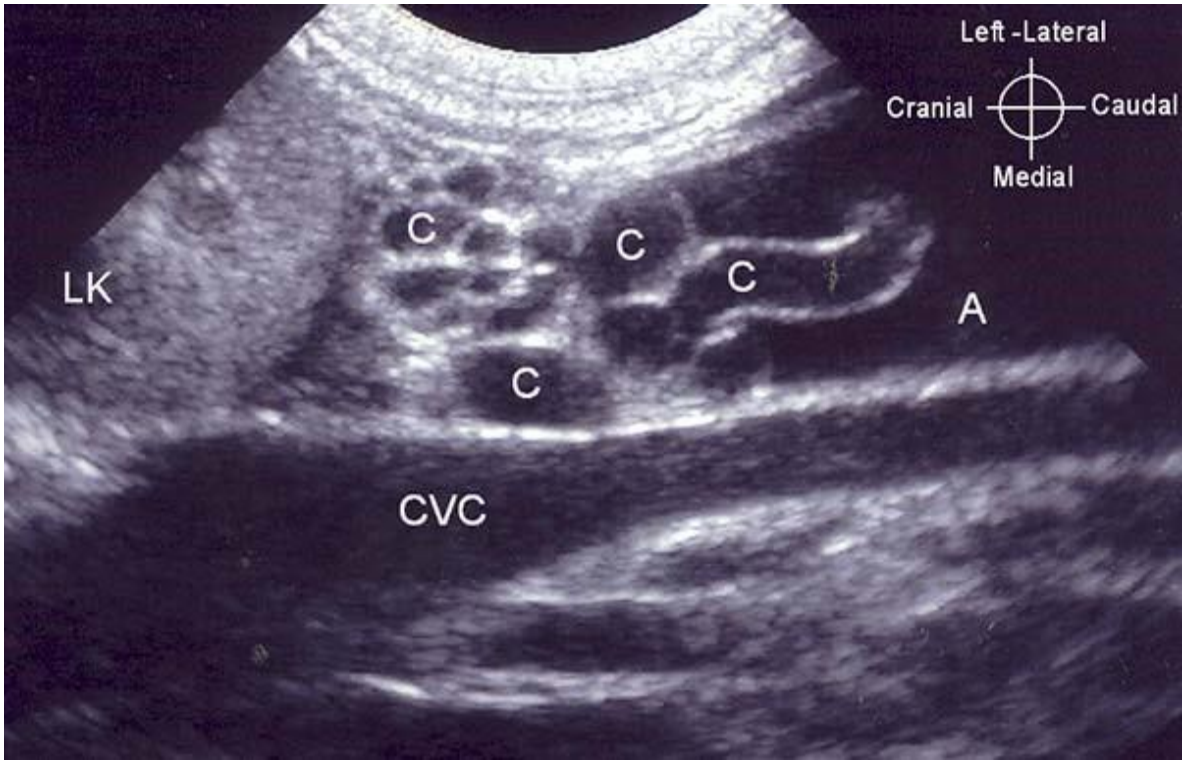
Diagnosis of CPSS is based on *visualizing* the anomalous vessel. Measuring flow velocities has no use. Ultrasonographic evaluation of the abdominal vasculature is recommended to be performed in 7 standard planes. [15]

Accurate recognition of CPSSs by ultrasound is only possible, if the anomalous vein is traced from its origin to its termination, or the other way around. Finding the point where a CPSS enters the CVC may be easier than finding the origin of a CPSS, [13] however finding a vein that enters the CVC does not mean that it is a CPSS because several other veins enter the CVC. In contrast, when a vein that originates from the PV 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 and do not originate from the PV.

Diagnosing APSCs ultrasonographically requires a different approach from that of CPSSs because recognizably large 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 ultrasonographically revealed. However, the dilated left gonadal vein, i.e. the termination of the spleno-renal collaterals, was found to be a highly specific and sensitive indicator of APSCs in dogs, and its ultrasonographic visualization is simple (Fig 12 A,B). [9]


Left
Cranial
Caudal
Right
LRV
LOV
CVC

A


Left -Lateral
Cranial
Caudal
Medial
C
C
C
C
LK
A
CVC

B

Figure 12. Acquired portosystemic (spleno-renal) collaterals.

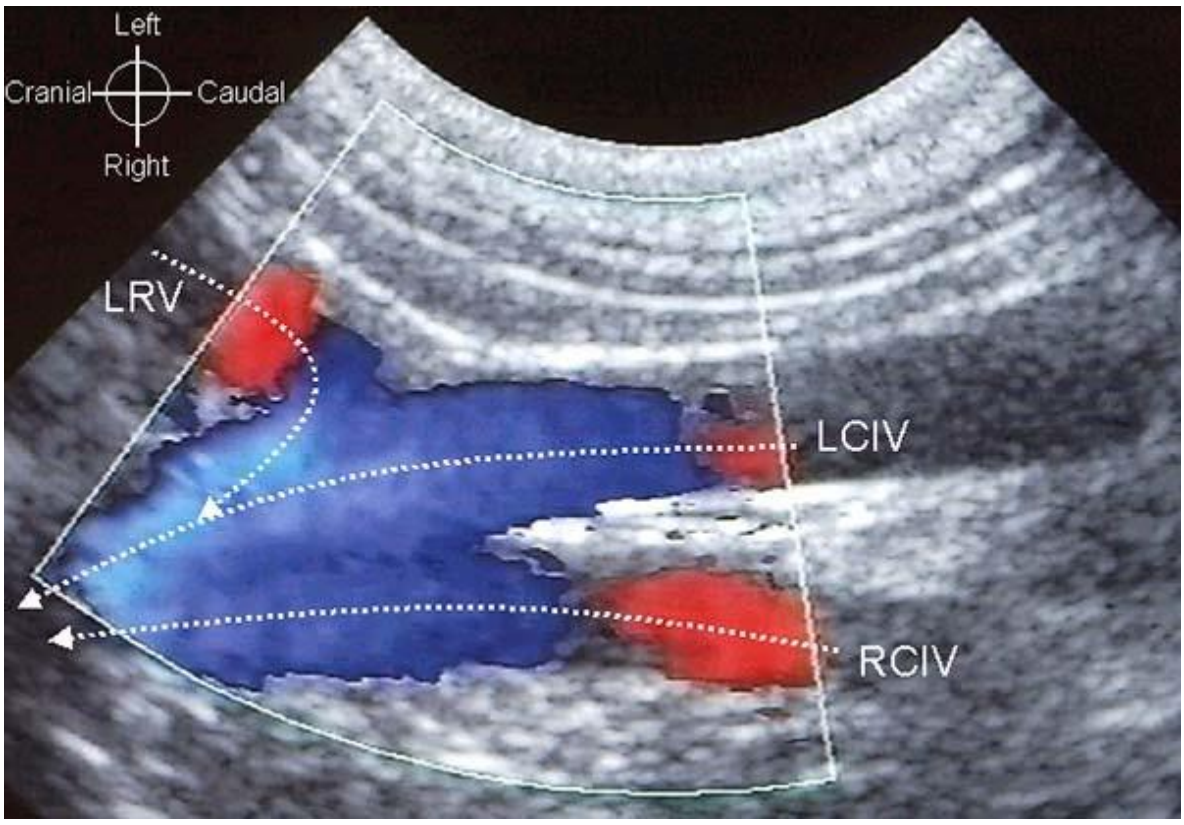
A. Dilation of the left ovarian vein (LOV) results from acquired spleno-renal collaterals in a miniature schnauzer after partial attenuation of a congenital extrahepatic spleno-caval shunt. This color Doppler ultrasound image was made via the left flank with the dog in right lateral recumbency (plane-7). Arrows indicate the directions of blood flow. LRV left renal vein, CVC caudal vena cava.

(From Szatmári V et al. Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in 14 dogs. Vet Rec 2004;155:448-456, with permission)

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 (From Szatmári V et al. Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). J Am Vet Med Assoc 2004;224:717-727, with permission).

"Double caudal vena cava" refers to an innocent congenital anomaly: the left and right common iliac veins fuse to form the CVC more cranial than normal, namely between the left and right renal veins. [31] Thus, 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 have the same diameters and run symmetrically on the respective side of the aorta (Fig 13 A,B). The only significance of this anomaly is that ultrasonographically the left common iliac vein may be mistaken with a dilated left gonadal vein. [9] However, careful examination can overcome this mistake. Of course, a "double caudal vena cava" does not cause high blood ammonia level, but it can be simultaneously present with a CPSS or with APSCs.



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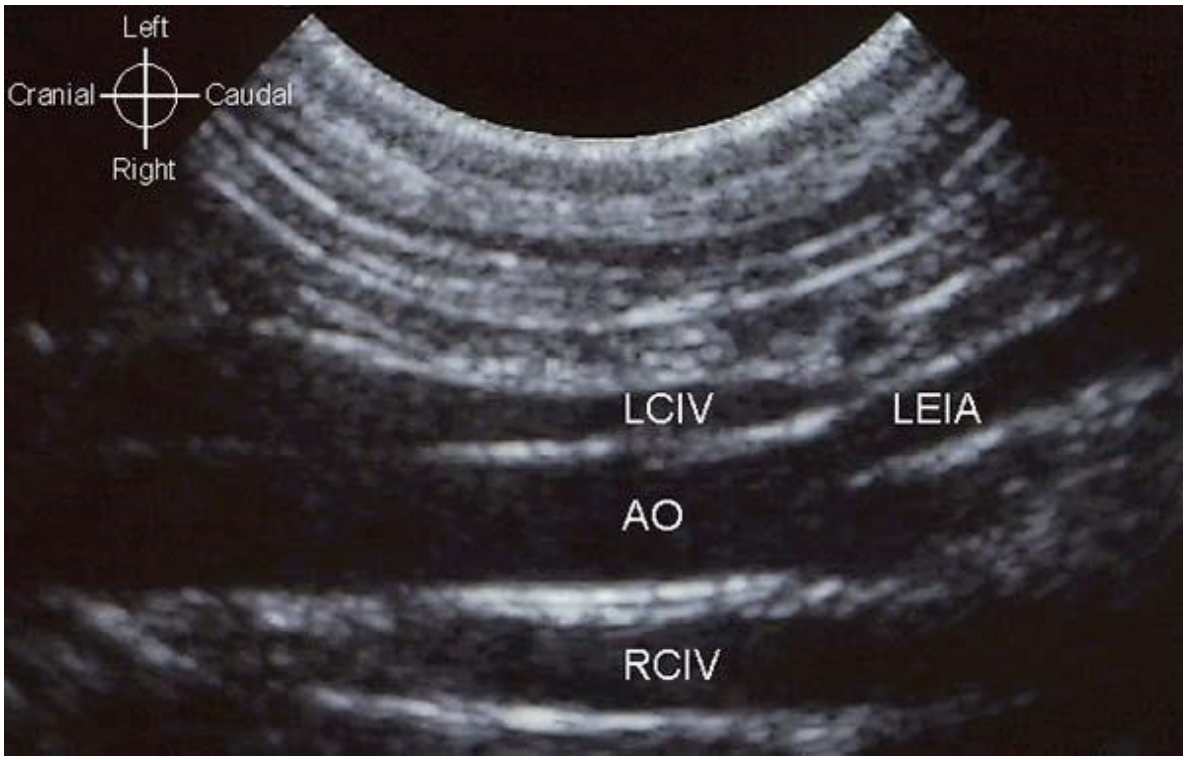


B

Figure 13. "Double caudal vena cava" (From Szatmári V et al. Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). J Am Vet Med Assoc 2004;224:717-727, with permission)

A. Color Doppler ultrasound 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 9A! Dotted arrows indicate the direction of blood flow. The LCIV and RCIV fuse to form the CVC cranial to the LRV.

B. Gray scale ultrasound image of the aortic trifurcation of the dog shown on Figure 10A. The left and right common iliac veins (LCIV, RCIV) run on the corresponding side of the aorta (AO). Plane-7, LEIA left external iliac artery.

Machine settings

Color Doppler parameters should be adjusted with care so that a vessel would be uniformly colored. Namely, the color gain must be set so that color signals would be seen in the entire lumen of a given vessel, but not outside the vessel. Traditionally, flow towards the transducer is coded with red, and flow away from the transducer with blue. Higher flow-velocities are coded with lighter hues of the appropriate color. The pulse repetition frequency (PRF) should also be appropriately adjusted, since a given PRF-setting is able to detect only a limited range of velocities. [32] If the PRF is set too high, slow flow may be missed. When the flow-velocity is higher than the upper limit of the velocity-range set that belongs to that particular PRF setting, then aliasing artifact occurs. Aliasing artifact means that if a flow velocity is higher than the upper limit of the velocity range belonging to a particular PRF, then no more lighter shades are available of the appropriate color, therefore these velocities will be coded with the opposite color, i.e. the color that indicates flow to the opposite direction. [32]

Standard scanning planes for evaluating portal vein disorders

When a portal vein disorder is suspected, a routine abdominal ultrasound examination should be done and the abdominal vessels in all the following seven scanning planes should be examined.

Plane-1: left lateral recumbency, transverse intercostal section — the starting point

The transducer is placed in one of the last right intercostal spaces. One should find the intercostal space through which only the liver is seen without the right kidney, and the cross-sections of the aorta, the CVC and the PV are visualized. When the right kidney appears, the transducer should be angled cranially or slided to a more cranial intercostal space, whereas, when air-containing lungs appear, the transducer should be angled caudally or slided to a more caudal intercostal space. When the PV cannot be imaged because of the duodenal gas, the transducer should be shifted dorsally within the same intercostal space and directed ventromedially.

Normal anatomy. From dorsal to ventral the cross-sections of the aorta, CVC and PV are seen; their cross-sectional areas are roughly equal (Fig 14).

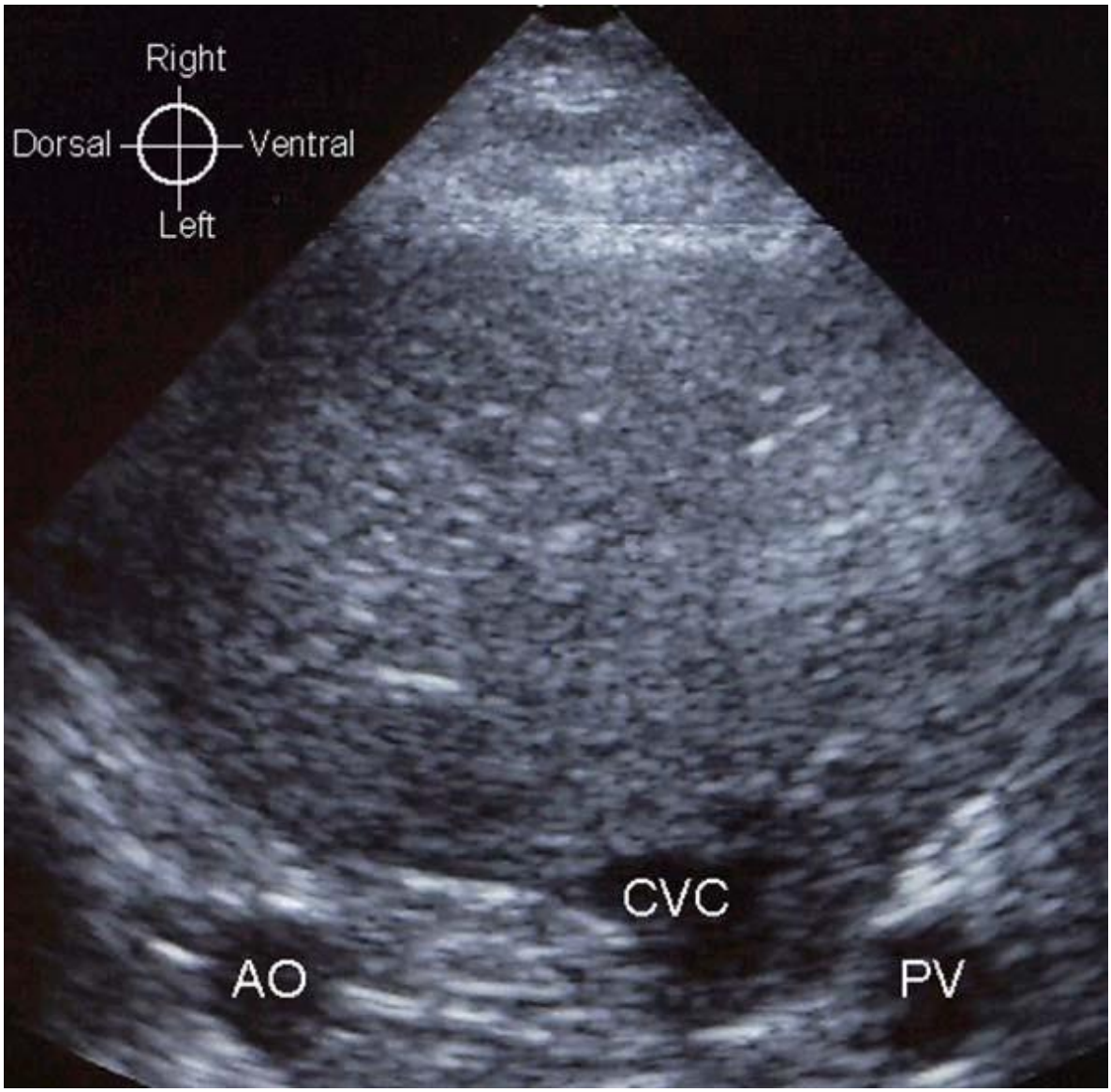


Figure 14. Normal great abdominal vessels.

Gray scale ultrasound image of the liver of a healthy male adult beagle in plane-1 (i.e. transverse section via one of the last right intercostal spaces with the dog in left lateral recumbency). This is the starting point of the systematic ultrasound examination of the portal system. 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. (From Szatmári V et al. Standard planes for ultrasonographic examination of the portal system in dogs. J Am Vet Med Assoc 2004;224:713-716, with permission).

Congenital intrahepatic porto-caval shunts. The images do not differ from normal, except for the presence of a prominent hepatic artery between the CVC and PV. The hepatic artery is 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 is thinner than the aorta, sometimes to such an extent that cannot even be recognized. The shunt might directly appear in this section.

Portal hypertension with APSCs. Visualization of the PV may often be hindered by ascitic fluid. If the PV is visible, the diameter is either smaller or larger compared to the aorta.

Plane-2: left lateral recumbency, transverse intercostal section to image the right portal branch

Starting from plane-1, the PV is traced by angling or sliding the transducer cranially to the point where the longitudinal image of the right portal branch appears.

Normal anatomy. The right portal branch can consistently be found as a well-defined vein originating from the PV and running dorsolaterally to the right while becoming gradually thinner due to ramification (Fig 15A).

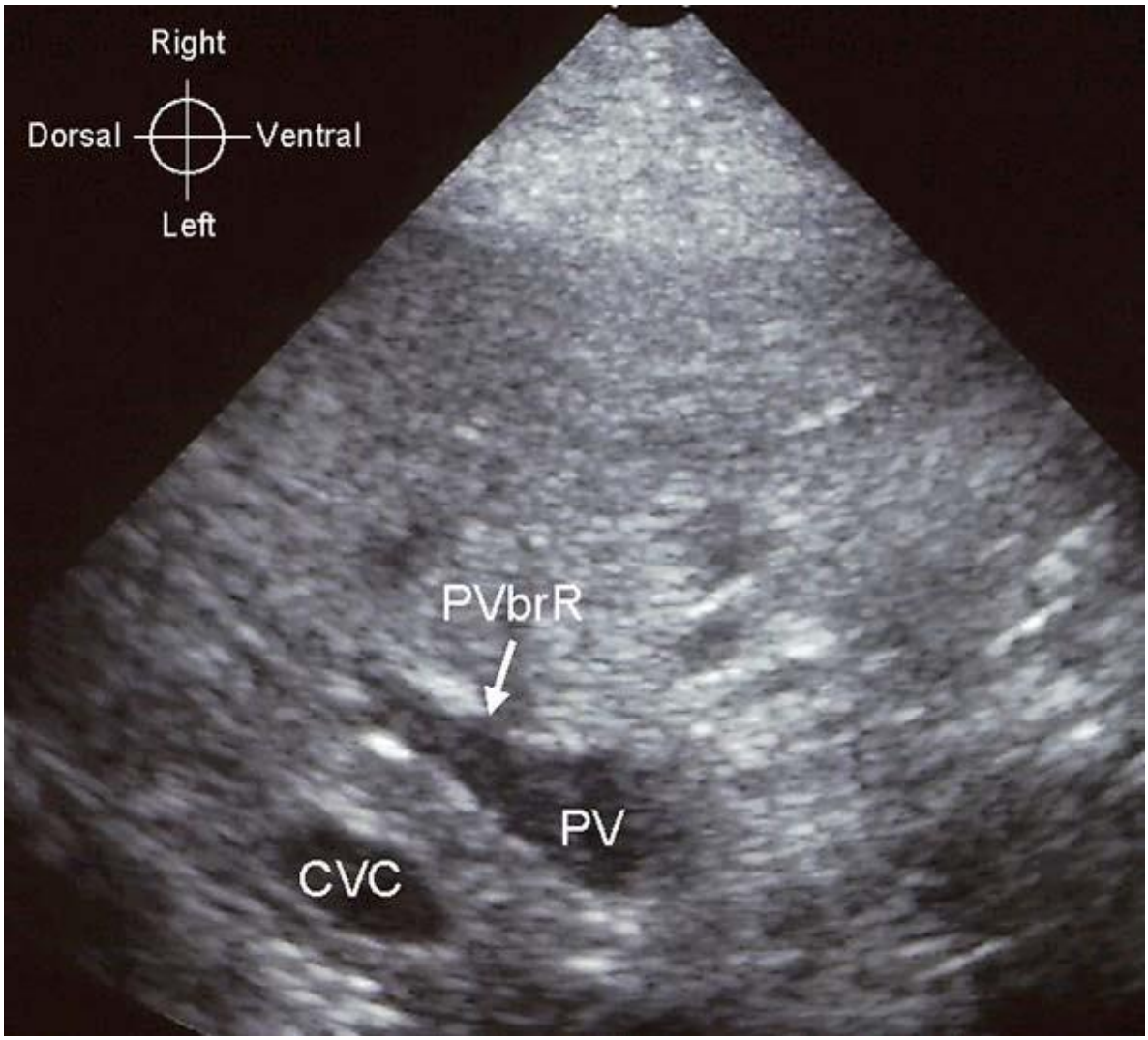
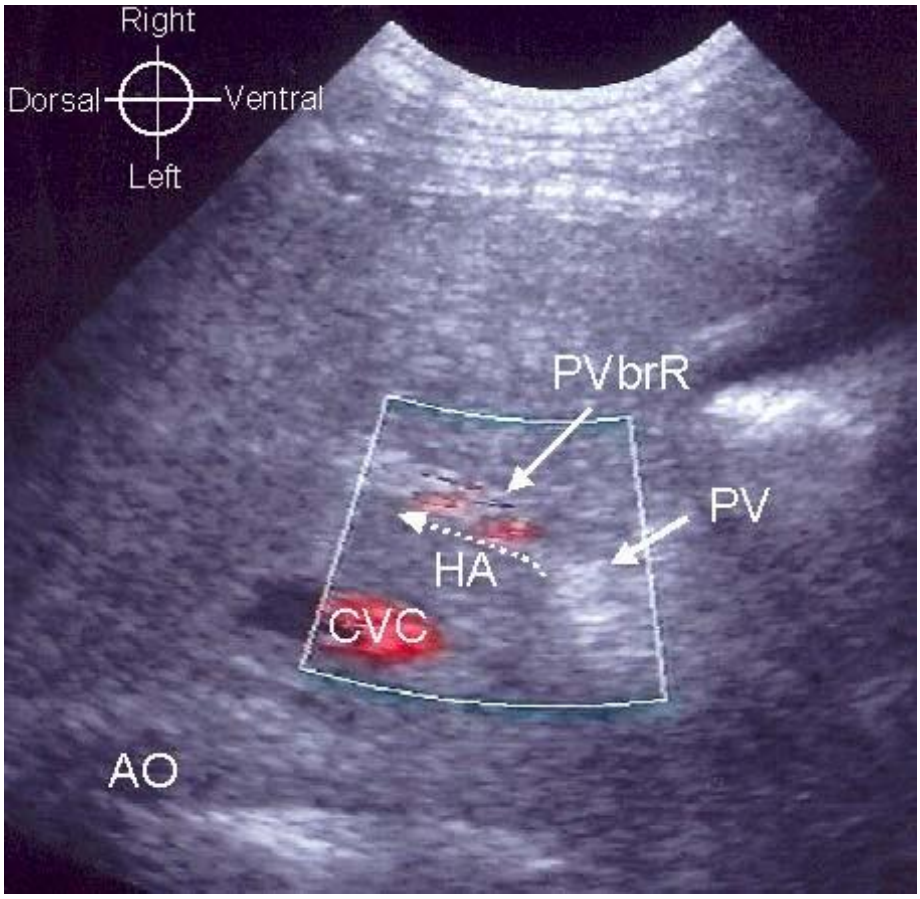


Figure 15. Right portal branch in 6 dogs.

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

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 to the periphery due to ramification. The right portal branch runs dorsolaterally and to the right. CVC caudal vena cava. (From Szatmári V et al. Standard planes for ultrasonographic examination of the portal system in dogs. J Am Vet Med Assoc 2004;224:713-716, with permission).



B. Color Doppler ultrasound image shows 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.

Congenital intrahepatic porto-caval shunts. Each right-sided intrahepatic CPSS originates from the right portal branch as its direct continuation. The right portal branch is wide and does not taper to the periphery. The first segment of the shunt consistently runs dorsolaterally to the right, like a normal right portal branch, but then instead of ramification, it turns medially to enter the CVC (Figs 15C, D). With little transducer-manipulation the entire course of the shunt can be traced to its caval termination.

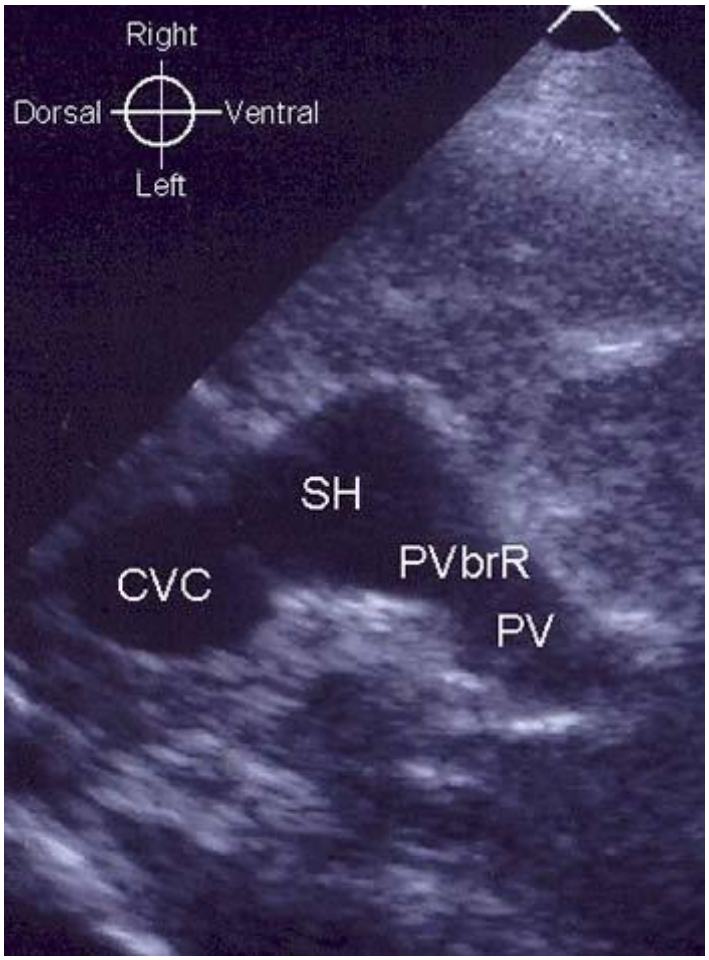


Figure 15.C. Central divisional congenital porto-caval shunt. Gray scale ultrasound image of a 4-month-old male large mixed breed dog with a short intrahepatic porto-caval shunt (SH) that originates from the right portal branch (PVbrR). Compare the length of the shunt with the one shown in Figure D! CVC caudal vena cava, PV portal vein.

Three months after the surgical attenuation of this shunt the dog was euthanized and a corrosion cast of the abdominal veins was made (see Fig 3.5).

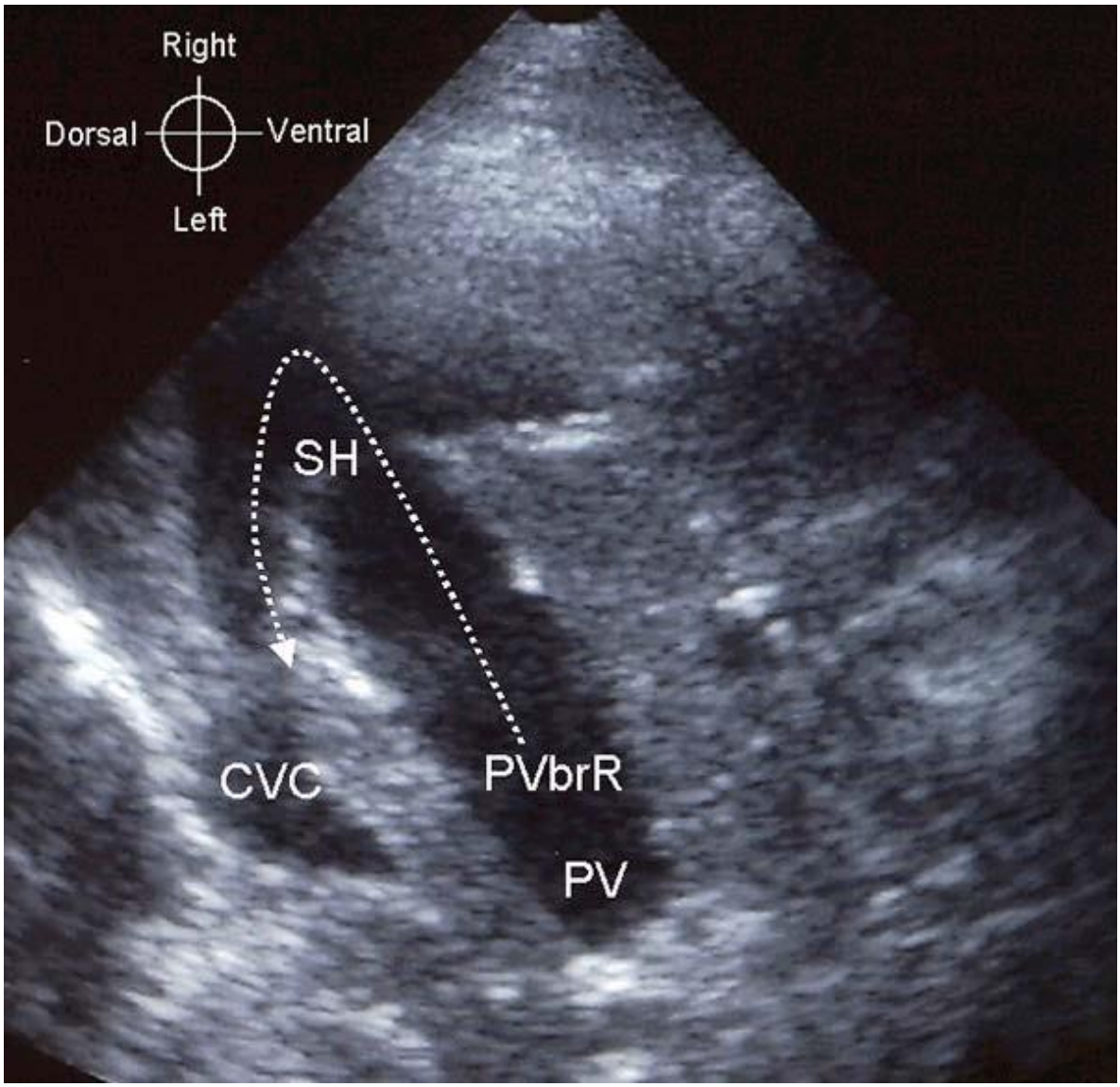


Figure 15.D. Right divisional congenital intrahepatic portocaval shunt. Gray scale ultrasound image of an intrahepatic porto-caval shunt that originates from the right portal branch (PVbrR) in a 5.5-month-old male Labrador retriever. In this single image the direct connection between the right portal 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. (From Szatmári V et al. Ultrasonographic findings in dogs with hyperammonemia: 90 cases (2000-2002). J Am Vet Med Assoc 2004;224:717-727, with permission).

Left-sided intrahepatic CPSS must be suspected, when the findings described in plane-1 are compatible with an intrahepatic CPSS, and the right portal branch is absent or very thin in plane-2 (Fig 15E). Often a hepatic artery branch is found at the place where the right portal branch is expected (Fig 15F). On B-mode images this artery looks like a very thin right portal branch, but color Doppler mode reveals fast flow and pulsed-wave Doppler mode shows arterial spectrum in it confirming that it is a hepatic artery branch that courses adjacent to the hypoperfused right portal branch.

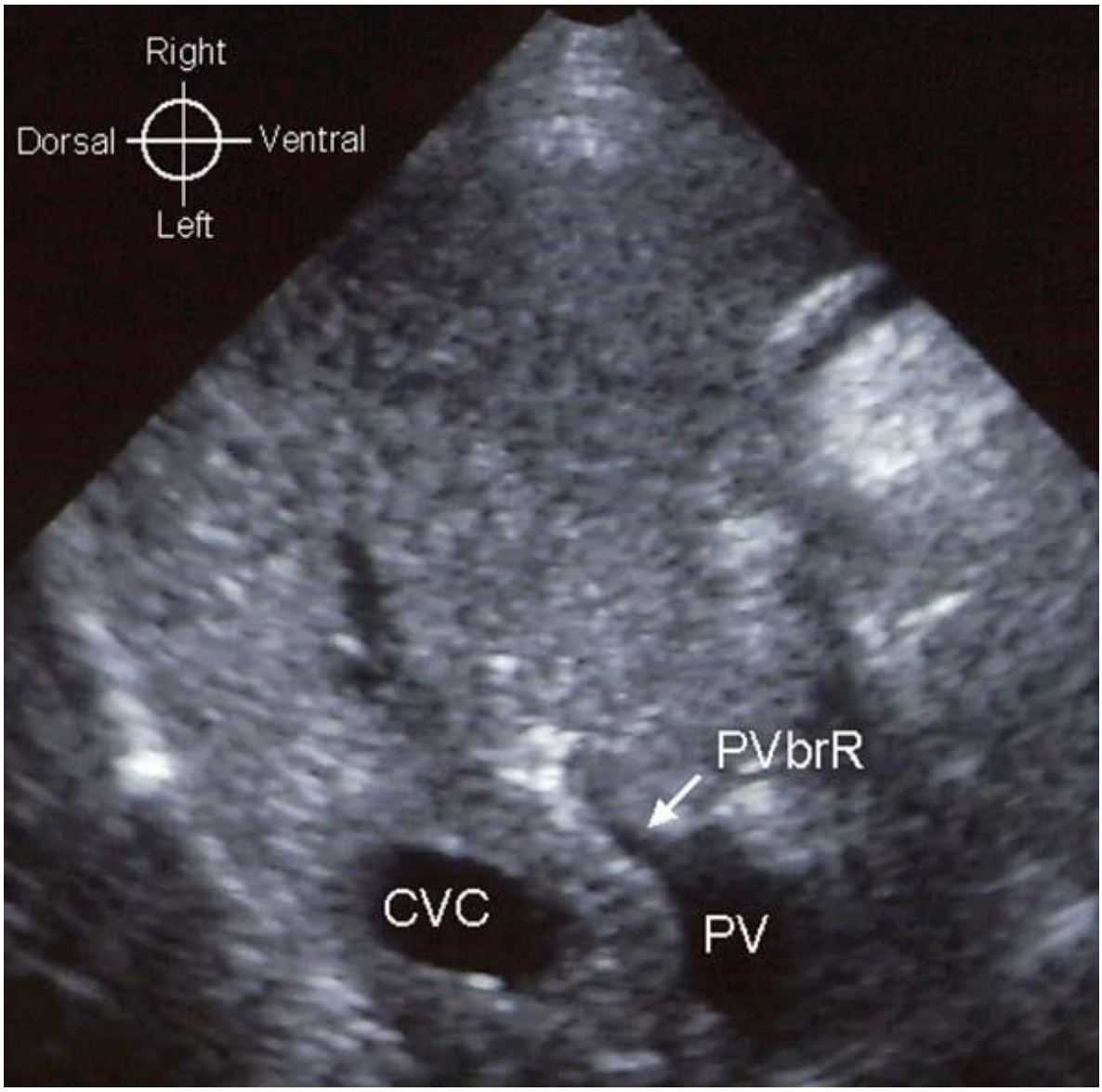


Figure 15.E. Gray scale ultrasound image of the right portal branch (PVbrR) in a 5.5-month-old male Bernese mountain dog with an intrahepatic porto-caval shunt that originates from the left portal branch. The portal vein (PV) has similar diameter to that of the caudal vena cava (CVC), however the right portal branch is very thin.

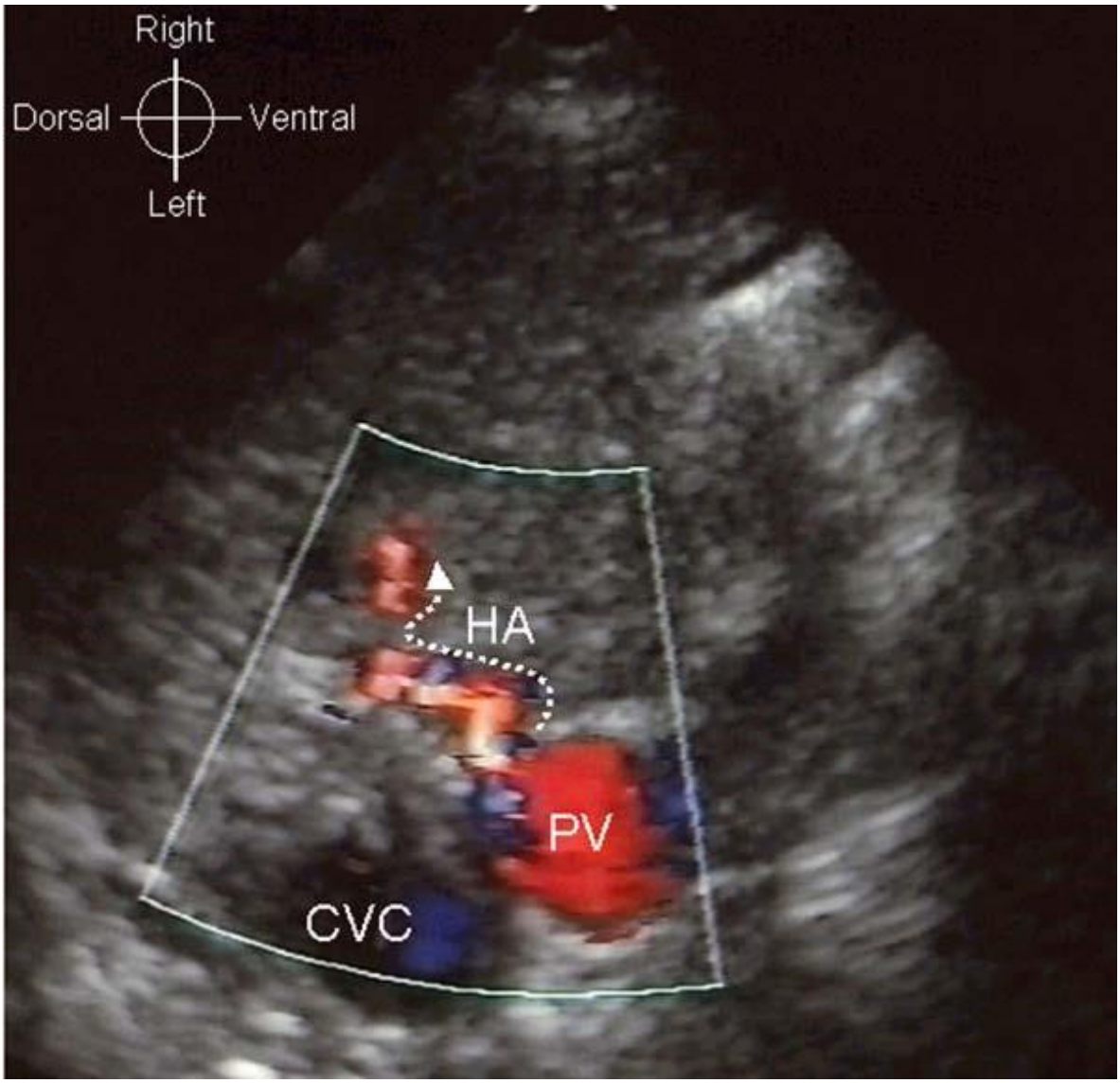


Figure 15.F. Undetectable right portal branch of a dog with a left divisional congenital intrahepatic portocaval shunt. An artery is seen at the place where the right portal branch is expected. The localization and course of this vessel is compatible with a right portal branch, but the flow velocity is much higher in it (color aliasing). The hepatic artery and portal branches run 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.

Congenital extrahepatic spleno-caval, spleno-azygos, right gastric-caval and right gastric-azygos shunts. The right portal branch as well as the PV itself are usually so thin that they cannot be visualized either on B-mode or on color Doppler images (Fig 15B).

Portal hypertension with APSCs. The right portal branch could only be exceptionally visualized because of the ascites and small liver size. It could be thinner or wider than normal or might have a normal diameter corresponding to the diameter of the PV, and shows normal arborization, but undetectably slow flow (i.e. no color signals with the lowest possible PRF).

Plane-3: left lateral recumbency, transverse intercostal sections to image congenital extrahepatic portosystemic shunts

Starting from plane-2, the transducer is gradually slided to caudal keeping the PV and CVC in the image, to the level where the celiac artery originates from the aorta. Scanning should be performed first with B-mode, then repeated with color Doppler mode. The aim is to look for a direct connection between the PV and CVC, or for a vessel that originates from the PV with a hepatofugal flow direction.

Normal anatomy and congenital intrahepatic porto-caval shunts. Immediately caudal to the portal bifurcation, the gastroduodenal vein may be imaged as it enters the ventral aspect of the PV from the right; however the gas-filled descending duodenum often hinders its visualization. Sliding the transducer further caudally, the splenic vein could be seen entering the left aspect of the PV from ventrolateral direction. Color Doppler mode reveals hepatopetal flow in the splenic vein. Slightly caudal to this point, the origins of the celiac and then the cranial mesenteric arteries from the aorta can be seen.

Congenital extrahepatic portosystemic shunts. The origin of the cranial loop of the right gastric-caval shunts can sometimes be seen, however the gastrointestinal gas often hinders their visualization. Therefore, plane-6 is recommended to be used when a CPSS with a right gastric vein origin is suspected based on the findings in planes-1, -2 and -3. The point where the shunt enters the CVC can regularly be detected, but the course of the shunt-loops can rarely be visualized from this side.

The entire length of spleno-caval shunts, and the origin of spleno-azygos shunts can always be visualized. Spleno-caval CPSSs make a short loop on the left side of the PV and CVC. The direct connection can usually be appreciated on B-mode images (Fig 16), however occasionally, when the shunt does not appear on B-mode images because of the insufficient gray scale resolution, color Doppler mode is helpful to visualize the shunt. Spleno-azygos shunts can be followed to the diaphragm.

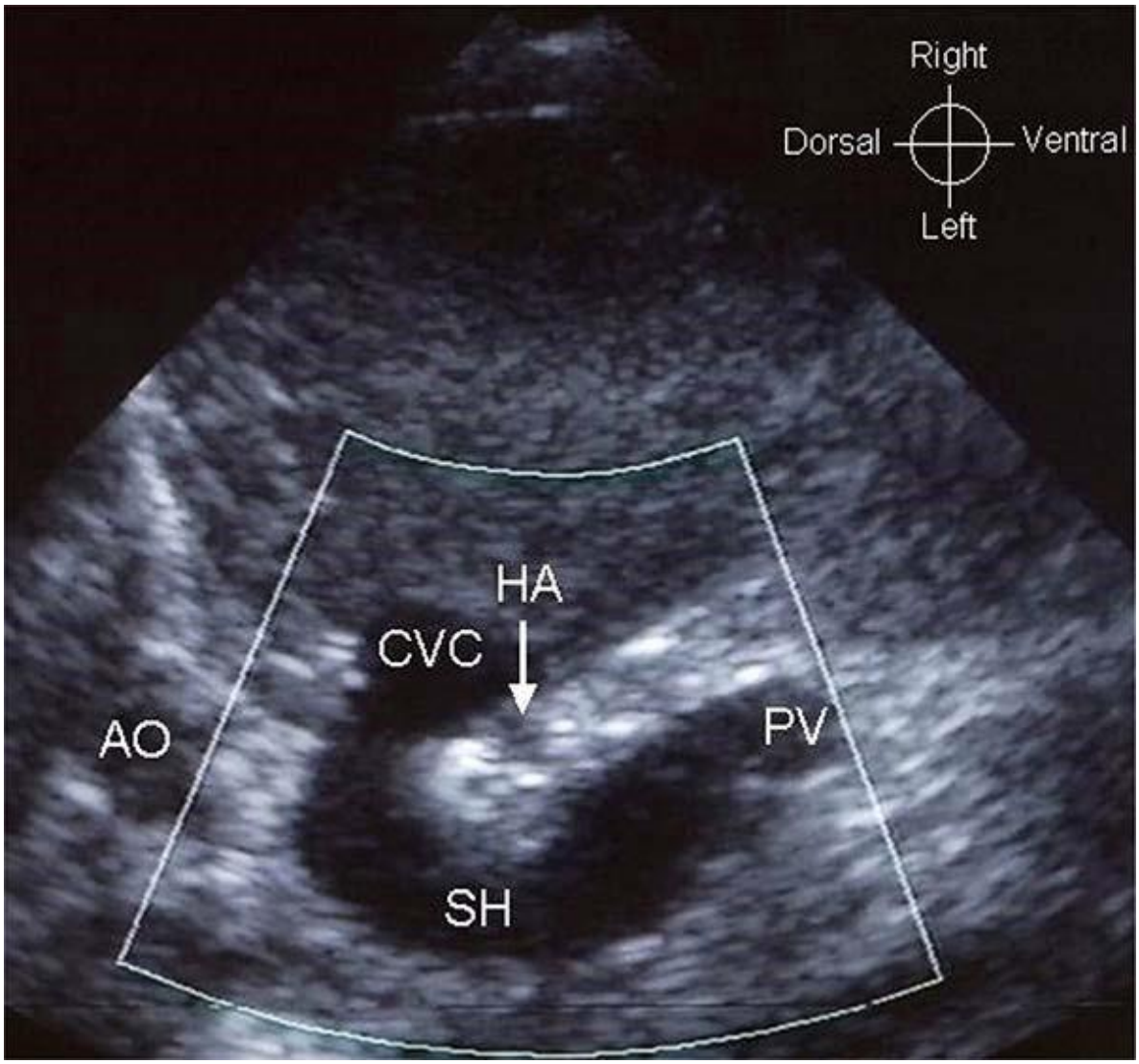


Figure 16. Congenital extahepatic spleno-caval shunt.

B-mode ultrasonogram shows the most common type of congenital portosystemic shunt in a 3.5-month-old female Jack Russell terrier made 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.

Portal hypertension with APSCs. The visualization of the PV is often difficult because of the presence of ascites. When the PV is imaged, the PV has a uniform diameter along its whole length. The origin of an APSC can occasionally be found as an anomalous vein with hepatofugal flow at the region where congenital extrahepatic spleno-caval shunts are expected to arise.

Plane-4: left lateral recumbency, longitudinal section via the right flank 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 are 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 should be obtained immediately ventral to the vertebrae. By ventral angulation of the transducer, the CVC becomes visible. Further ventral angulation results in the longitudinal image of the PV at the point where the splenic vein enters the PV. Firm transducer-pressure is often required to image the portal bifurcation.

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

Normal anatomy. The splenic vein can be seen to enter the PV from caudolateral direction from the left (Fig 1 B). Tracing the PV cranially, the portal bifurcation can be seen with the wider left and the thinner right portal branch. Both branches become gradually thinner towards the periphery.

Congenital intrahepatic porto-caval shunts. The PV at the level of the splenic vein looks similar to that of normal dogs. Tracing the PV cranially, an intrahepatic CPSS appears as the direct continuation of the PV that enters the CVC. Plane-4 does not allow the differentiation whether the intrahepatic CPSSs originates 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 is used to confirm the presence of left-divisional intrahepatic CPSSs by direct visualization of the porto-caval connection (Fig 17). Since the intrahepatic CPSSs that originates from the left portal branch courses adjacent to the diaphragm, plane-4 allows their better visualization than plane-2.

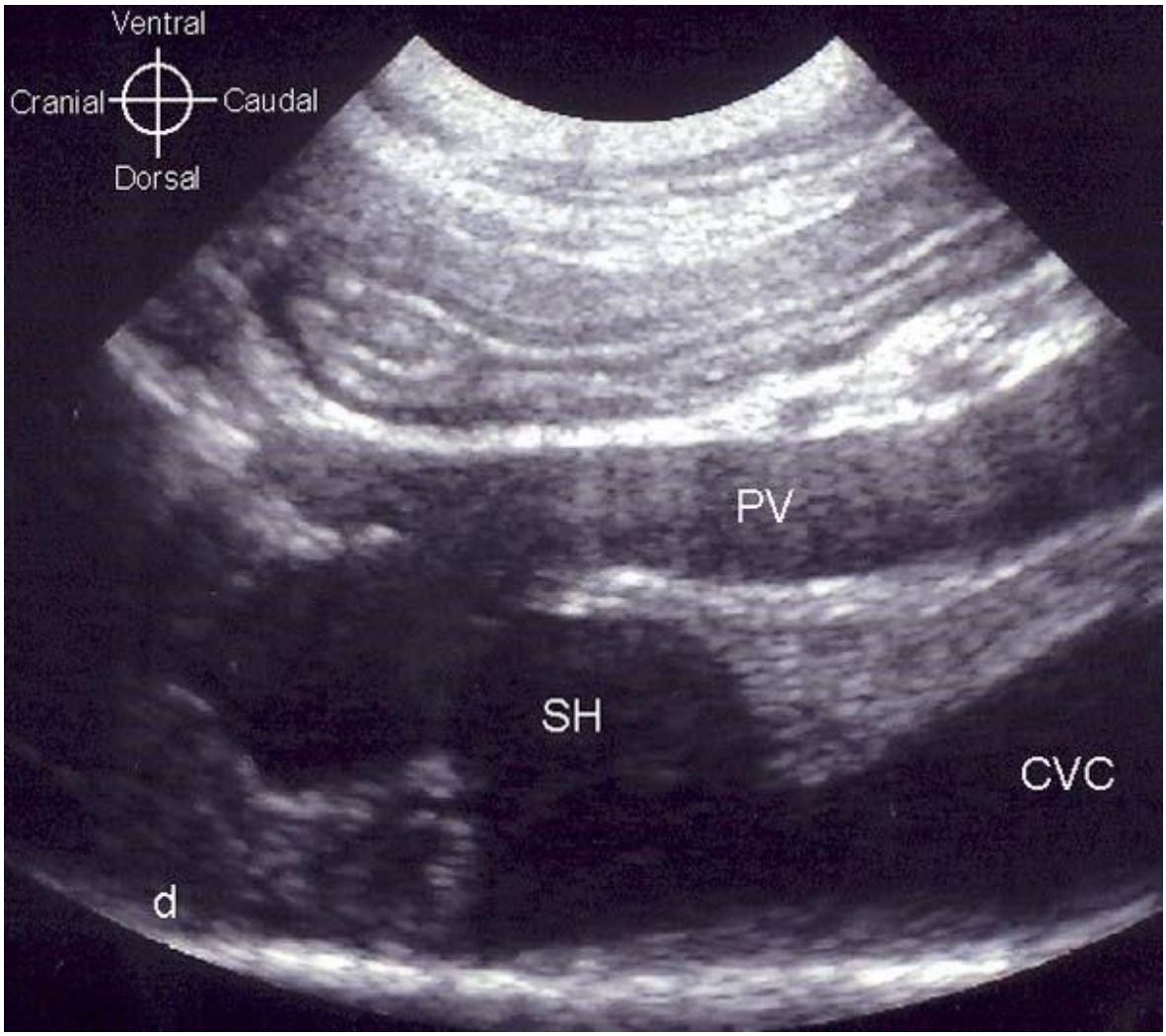


Figure 17. Patent ductus venosus.

This intrahepatic porto-caval shunt originates from the left portal branch (i.e. left divisional shunt) in a 9-month-old hovawart. The portal trunk (PV) continues via a wide and tortuous anomalous vessel (SH) and terminates in the caudal vena cava (CVC). The image was made in plane-5. An empty stomach can be seen between the portal vein and the abdominal wall. Gas in the stomach would hinder the visualization of the shunt. 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 can also be seen in addition to the shunt, and the direction of flow can be determined in them (Fig 7A). In cases of spleno-caval CPSSs, the termination of the CPSS can be found with little transducer-manipulation by tracing the shunting vessel. In cases of spleno-azygos shunts, the shunt can be traced to the point where it enters the thorax. Occasionally the PV-segment cranial to the point where the gastroduodenal vein enters the PV may also be imaged.

Congenital extrahepatic right gastric-caval and right gastric-azygos shunts. Occasionally, the origin of the dilated right gastric vein can be visualized using this view, but not the course of the cranial loop of the shunt. The caudal shunt-loop (when it is present) can regularly be visualized, which originates at the same point as a congenital extrahepatic spleno-caval shunt, however it courses cranially, unlike a spleno-caval shunt, which latter courses dorsally. The PV cranial to the origin of the caudal shunt-loop can always be seen, and the flow-direction is always hepatopetal in it. The anastomosis between the cranial and caudal loops of the shunt cannot be readily visualized with ultrasound, however the termination of the shunt can usually be found.

Portal hypertension with APSCs. This view makes the assessment of portal flow-velocity and of portal flow-direction possible. The origin of APSCs arising from the PV may also be seen at the region where congenital extrahepatic spleno-caval shunts arise. It is usually impossible to image the PV cranial to the entering point of the gastroduodenal vein.

Plane-5: dorsal recumbency, longitudinal sections via the ventral abdominal wall — an alternative for plane-4

To find the PV in dorsal recumbency, the dog should be slightly tilted towards the sonologist. The right kidney with the caudate liver lobe are imaged first. Then the transducer is angled a bit medially to image the CVC, then further medially to image the PV. To image the portal bifurcation, the PV is traced cranially. The transducer has often to be pushed firmly to move away the gas-filled intestinal loops.

The findings are the same as described under plane-4, but in some cases plane-5 allows better visualization of the shunt or of the PV (Fig 17), and provides better incidence angles for Doppler studies.

Plane-6: right lateral recumbency, longitudinal sections via the left flank to image the (cranial loop of) congenital extrahepatic right gastric-caval and right gastric-azygos shunts

The transducer is placed immediately caudal to the last left rib and the PV is imaged longitudinally at the hilus of the liver. Finding the PV using this approach is difficult and is only necessary when in plane-3 an extrahepatic CPSS was suspected, but its entire visualization was impossible.

Another way to find the right gastric-caval shunts is to follow the hepatic artery from its origin to the liver, as the hepatic artery crosses the cranial shunt-loop. To find the hepatic artery, the celiac artery has to be imaged as it originates from the aorta, cranial to the left kidney. [32] The hepatic artery is the widest branch of the celiac artery, which courses cranially to the liver between the PV and the CVC. Color Doppler mode helps to find the hepatic artery, when the gray scale resolution is insufficient to visualize this thin vessel. The color signals of the hepatic artery indicate higher flow velocity compared to that of the CVC and the PV.

Congenital extrahepatic right gastric-caval or right gastric-azygos shunts. Usually, when the PV is being searched, a large-caliber anomalous vein (i.e. the shunt) appears just under the body wall, even before the PV is actually found. The diameter of this CPSS is comparable to that of the CVC. Tracing the shunt cranially it seems to originate from the PV, at the hilus of the liver (Fig 11A). From its origin, the shunt should be traced to its termination with and also without color Doppler mode (Fig 11B). The PV cranial to the shunt-origin is too thin to be visualized.

Normal anatomy, congenital intrahepatic porto-caval shunts, congenital extrahepatic spleno-caval and spleno-azygos shunts, portal hypertension with APSCs. The large-caliber anomalous vein described above is absent. The normal right gastric vein is so thin that cannot be visualized ultrasonographically.

Plane-7: right lateral recumbency, longitudinal sections via the left flank to image the dilated left gonadal vein, i.e. the termination of acquired spleno-renal collaterals

The CVC is 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 is moved cranially to image the left renal vein as it enters the CVC. With B- and color Doppler modes a vein has to be searched that enters the left renal vein from caudal: this is the left gonadal vein.

Normal anatomy, congenital intra- and extrahepatic portosystemic shunts. Caudal to the left kidney two great vessels, namely the aorta and the CVC can be seen running parallel to each other; the aorta is located more to the left. The left gonadal vein can never be visualized because it is too thin. The left renal artery (occasionally double) runs adjacent to the left renal vein and can be differentiated from the vein even on gray scale images by its smaller diameter and pulsation.

Portal hypertension with APSCs. The dilated left gonadal vein can always be seen entering the left renal vein from caudal (Figs 2A, 12A); except for the cases of tense ascites, when not even the CVC can be visualized. When the left gonadal vein is very wide, it appears as a third great vessel on the left side of the aorta. This image has to be carefully differentiated from a “double CVC”. Several small tortuous veins (spleno-renal collaterals) may often, but not always be seen around the left renal vein (Fig 12B).

Summary

Ultrasonography is a highly sensitive and specific, non-invasive diagnostic method to diagnose and exclude CPSSs and APSCs in non-sedated 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 morphologic and hemodynamic features of the abdominal vessels are different in dogs with normal vascular anatomy from those with portal vein disorders. The diagnosis of a particular portal venous disorder is the result of a puzzle that is based on the results of clinical, laboratory and ultrasonographic findings.

Specific features of feline portal vein anomalies

Portal vein disorders occur much less frequently in cats than in dogs, and there are several differences in the etiology of APSCs and in the anatomy of the CPSSs and APSCs. This short chapter will focus only on these differences. The same scanning technique is recommended as in dogs (i.e. the seven standard planes), but the findings and their interpretation may be different.

Etiology of feline hyperammonemia

High blood ammonia level in cats can be caused by CPSS, APSCs and by arginine deficiency [33, 34]. Arginine deficiency develops in anorectic cats along with hepatic lipidosis, which can be diagnosed with abdominal ultrasonography and cytology of a hepatic fine needle aspiration biopsy. [35] Urea cycle enzyme deficiency has not been reported in the cat.

Routine abdominal ultrasound examination often reveals no abnormalities in cats with CPSS or with APSC due to hepatic portal hypertension. Ascites is absent not only in cats with CPSS, but usually also in cats with APSCs. The liver and the kidneys have most of the time normal size both with CPSS and with APSCs. Splenomegaly could be present in cats with portal hypertension.

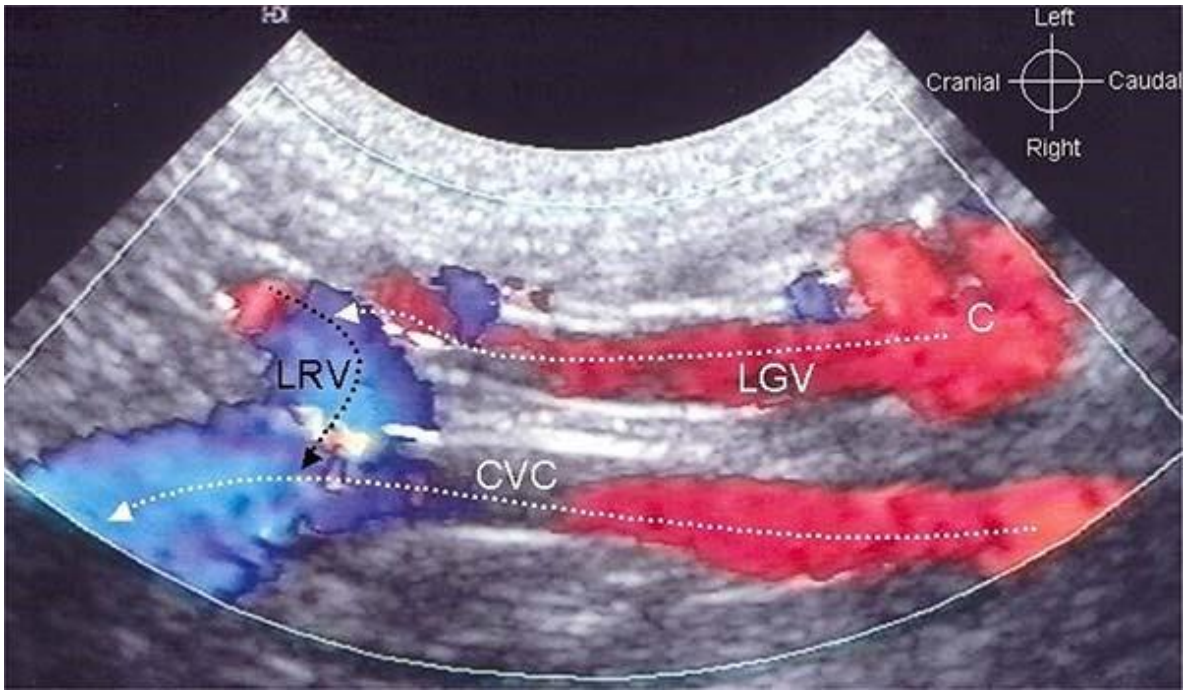
Portal hypertension in cats

Acquired portosystemic collaterals can develop as a result of hepatic or prehepatic portal hypertension. Prehepatic portal hypertension can be caused by an obstruction of the PV by a thrombus or by a compression by a tumor. [33] Congenital arterioportal fistula has also been found as the cause of APSCs. [33, 36] Hepatic portal hypertension is often the result of hepatic fibrosis caused often by congenital hepatic fibrosis due to polycystic kidney and liver disease (PKD). [33, 37] Free abdominal fluid is either absent or if it present its amount is very small.

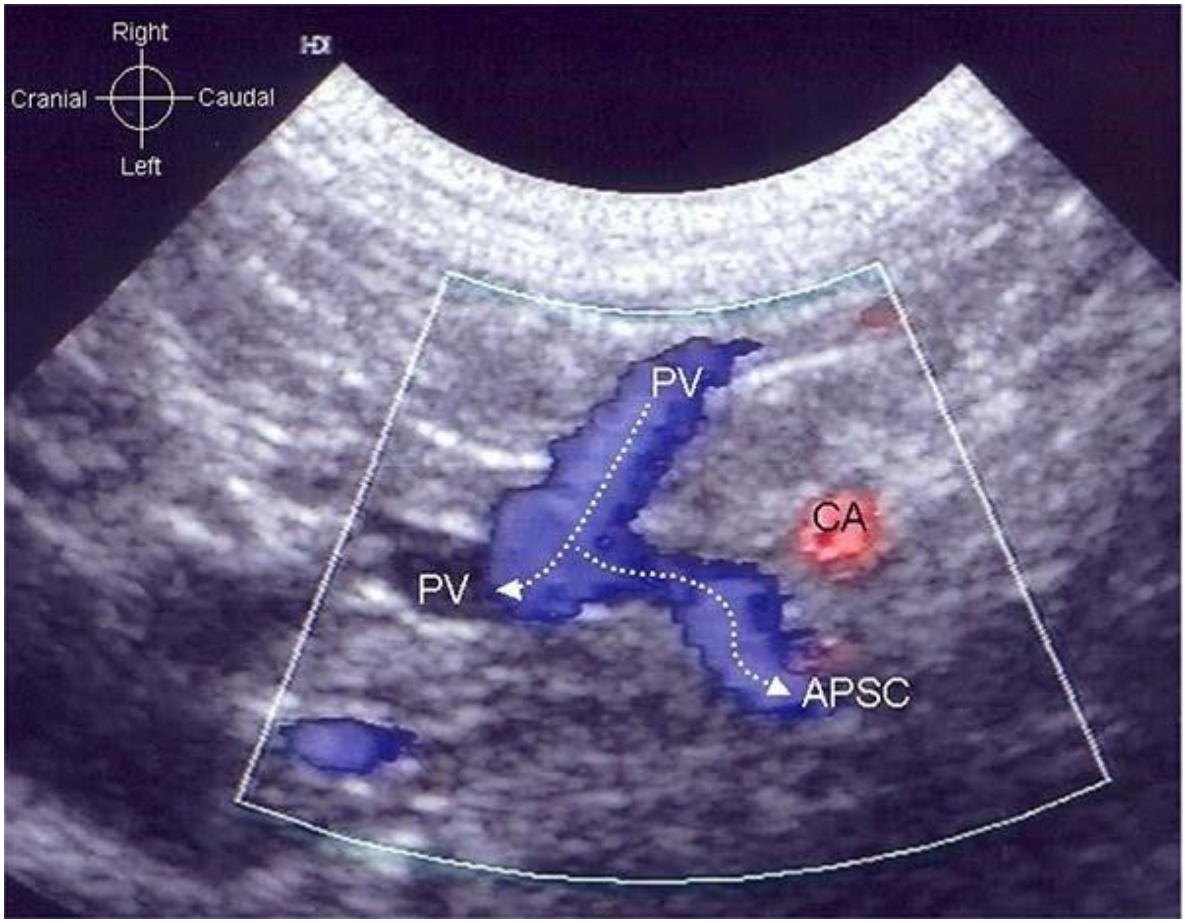
Acquired portosystemic collaterals in cats

The anatomy of feline APSCs is slightly different from that of the dogs. [38-40] Since spleno-renal collaterals do not develop consistently, the left gonadal vein may not be dilated. [33] When spleno-renal collaterals do develop, a dilated left gonadal vein will be present, similarly to dogs (Fig 18A). However, a sort of extrahepatic CPSS terminates in the left renal vein mimicking a dilated left gonadal vein. The difference is that in case of a dilated left gonadal vein (i.e. APSCs) color Doppler usually reveals lots of small tortuous veins around the gonadal vein, and the left gonadal vein cannot be followed to the PV because it ends up in a conglomeration of small collaterals. In contrast, a CPSS that enters the left renal vein is a very wide vein (wider than the CVC) and can be traced back to its PV-origin.

The origin of an APSC is often found arising directly from the PV at the point where congenital extrahepatic spleno-caval shunts originate in dogs, i.e. slightly cranial to the point where the celiac artery originates from the aorta (Fig 18B). Color Doppler mode may reveal hepatofugal, hepatopetal or zero flow in the PV cranial to the collateral origin. This APSC runs to caudal direction and tends to disappear among the intestines.



A



B

Figure 18. Feline portal hypertension.

Ultrasonographic signs of portal hypertension are shown in a three year-old female British short hair cat due to congenital hepatic fibrosis on the basis of polycystic kidney and liver

disease. No renal cysts were seen with ultrasonography in this cat. Dotted arrows indicate the direction of blood flow.

A. The left gonadal vein (LGV) becomes dilated when spleno-renal collateral circulation develops. CVC caudal vena cava, LRV left renal vein, C conglomeration of portosystemic collateral vessels; color Doppler image made in plane-seven.

B. Color Doppler ultrasound image of the portal vein (PV) and the origin of an acquired portosystemic collateral vein (APSC) made in plane-four. CA celiac artery.

When the presence of APSCs is suspected, ultrasound-guided biopsy of the liver should be taken for histopathologic examination. The suspicion should especially be high when renal cysts have been detected or when an abnormal abdominal vein in a Persian or British short hair breed or their crosses has been found.

Congenital portosystemic shunts in cats

In contrast to dogs, CPSSs in cats have a great anatomic variety. [41] The shunting vessel is usually a single extrahepatic wide vein.

Intrahepatic CPSSs are very rare in cats and unlike in dogs, they are not necessarily the continuations of the left or right portal branches, therefore they may be fairly thin.

Extrahepatic CPSSs. Feline spleno-caval and spleno-azygos shunts are similar to that of the dogs, however the shunt may originate directly from the PV adjacent to the point where the splenic vein enters the PV (Fig 19A). If the shunt originates more caudal from the PV then hepatofugal flow can often be seen in the PV segment between the shunt origin and the entering point of the splenic vein. However, the blood flow may be hepatopetal in the entire PV. The PV may get thinner cranial to the origin a CPSS, but may have a uniform diameter throughout its whole length.

Extrahepatic CPSSs arising from the right gastric vein are rather common in dogs, but do not occur in cats. However, there are two types of CPSSs that are quite specific for the cat. One originates slightly caudal to the portal bifurcation and courses cranially among liver lobes along the esophagus and eventually enters the CVC (often via a hepatic vein) between the diaphragmatic surface of the liver and the diaphragm. Ultrasonographically this shunt seems to be intrahepatic because the vessel is surrounded by liver (Fig 19B). The other type of CPSS originates from the cranial mesenteric vein or from the PV at the region where it is formed from the mesenteric veins. From here the wide shunting vessel courses caudally along the colon as caudal as the aortic trifurcation, where it makes a 180° turn and courses cranially on the left side of the CVC, and terminates in the left renal vein or in the CVC caudal to the left kidney (Fig 20). This type of CPSS can easily be found using plane-7 and should carefully be differentiated from a dilated left gonadal vein, which latter is the result of spleno-renal APSCs.

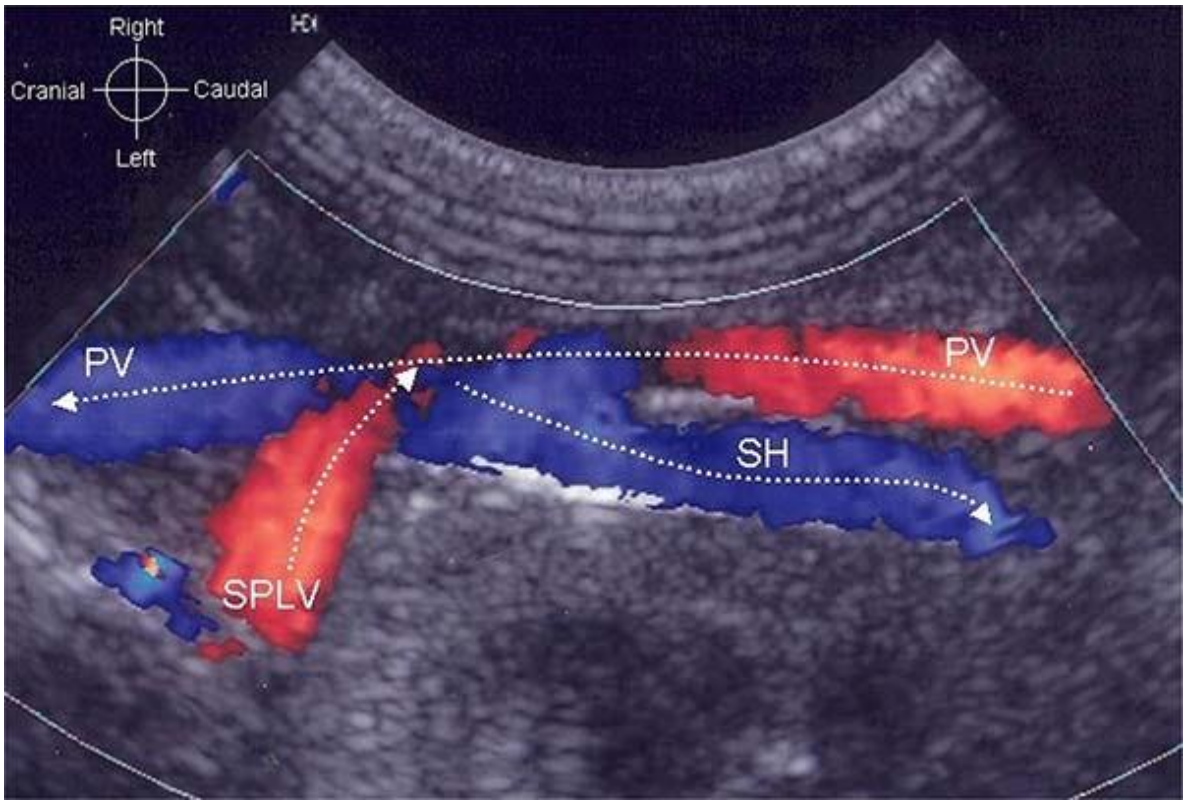
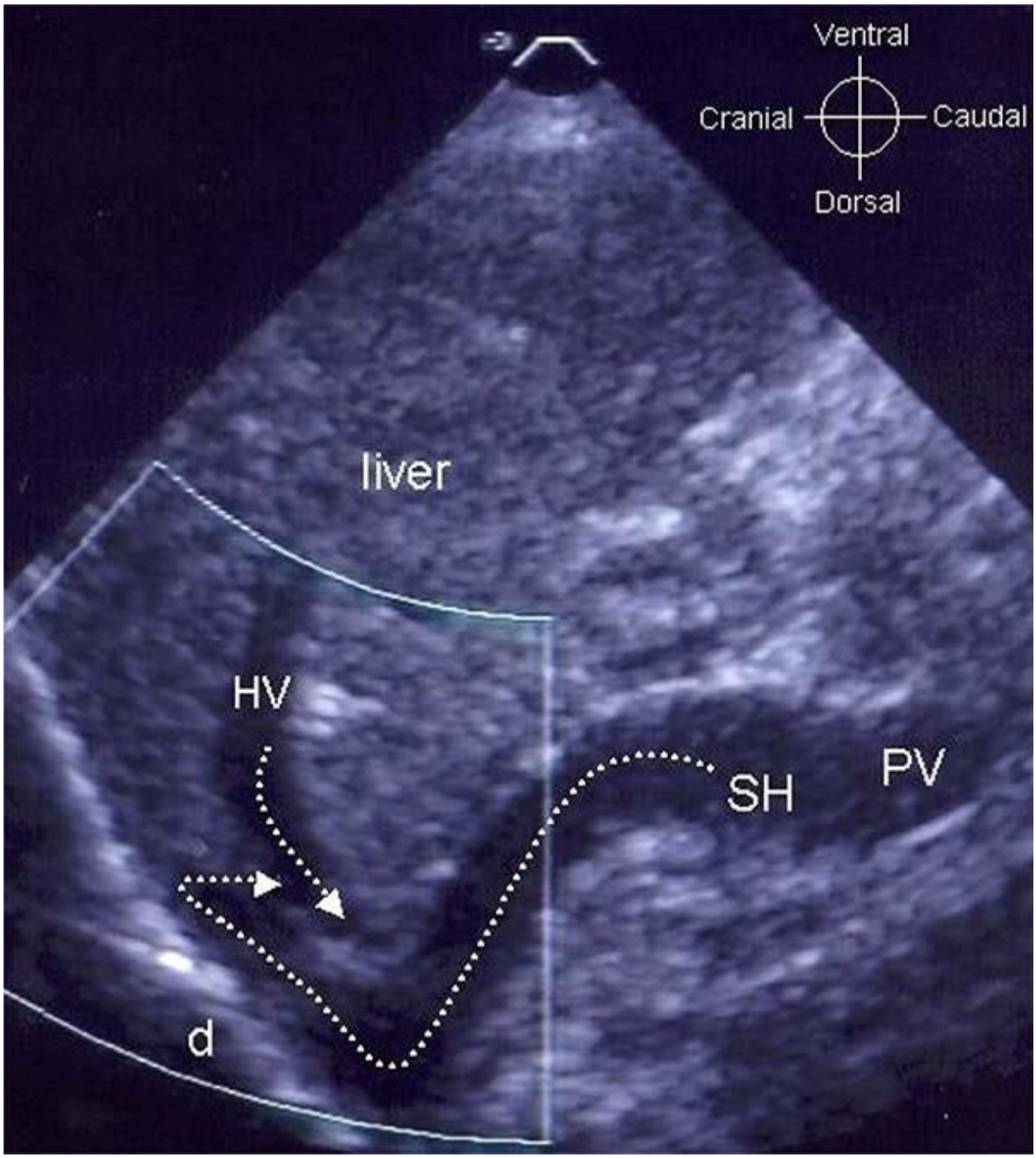


Figure 19: Congenital extrahepatic portosystemic shunts in two cats.

Dotted arrows indicate the direction of blood flow.

A. The shunt (SH) of this European short hair cat originates directly from the portal vein (PV) just caudal to the point where the splenic vein (SPLV) enters the PV. Interestingly, the PV does not become thinner cranial to the shunt origin and it displays hepatopetal flow. This Color Doppler image was made in plane-four.



B. The extrahepatic shunt (SH) of this Maine coon cat seems to be intrahepatic, but surgery revealed that it actually ran between the liver lobes. The shunt connects the portal vein (PV) and a hepatic vein (HV). This B-mode image was made in plane-five. d - diaphragm.

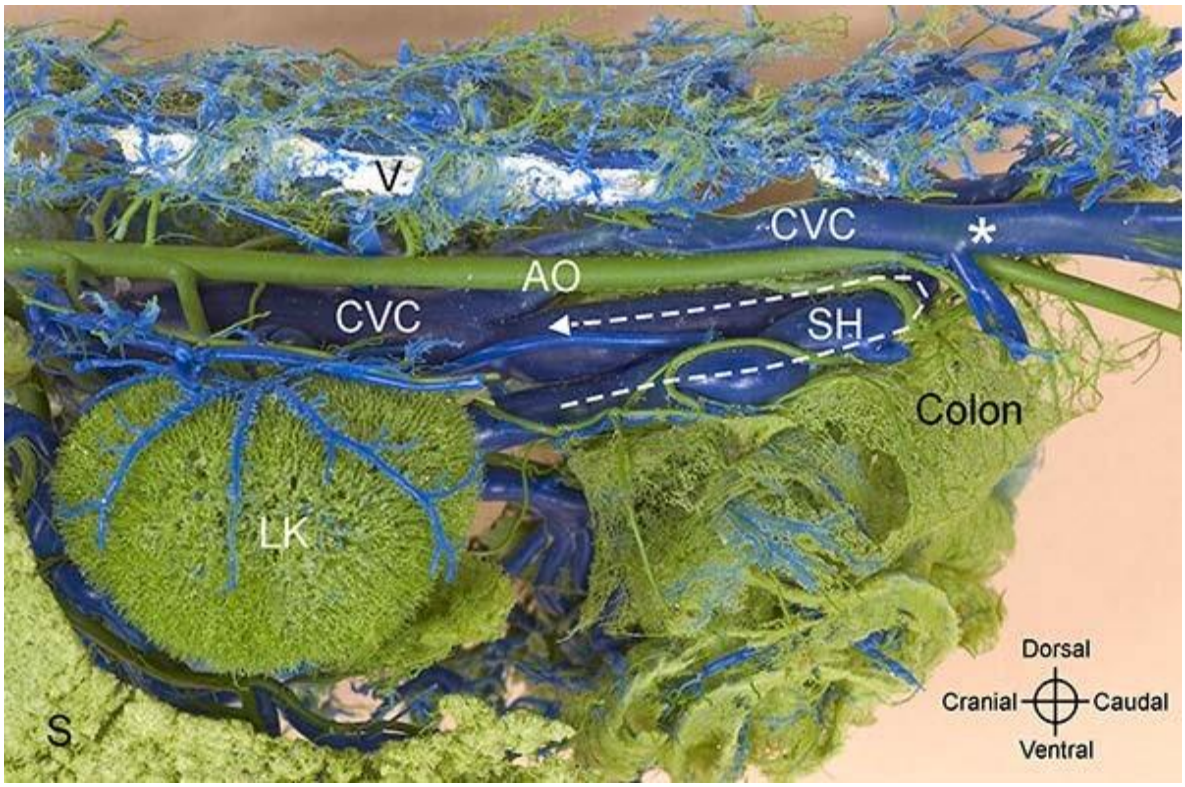


Fig 20: Congenital extrahepatic portosystemic shunt in a cat.

Corrosion cast of the abdominal blood vessels of a kitten with a congenital extrahepatic portocaval shunt. The veins are blue and the arteries are green. The shunt (SH) is a wide vessel that courses to caudal from its origin (cranial mesenteric vein) along the descending colon (colon). At the level of the aortic trifurcation (*) the SH turns back to cranial direction and courses on the left side of the caudal vena cava (CVC) to eventually terminate in the CVC slightly caudal to the left kidney (LK). AO - aorta, S - spleen, V - vertebrae.

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