Complications after surgical attenuation of congenital extrahepatic portosystemic shunts in dogs can be prevented by intraoperative Doppler ultrasonographic assessment of portal hemodynamics

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What is a congenital portosystemic shunt?

Under normal circumstances the blood from the gastrointestinal tract flows via the portal vein to the liver. The detoxified blood leaves the liver via the hepatic veins to the caudal vena cava, which latter enters the heart . (**Figure 1**).

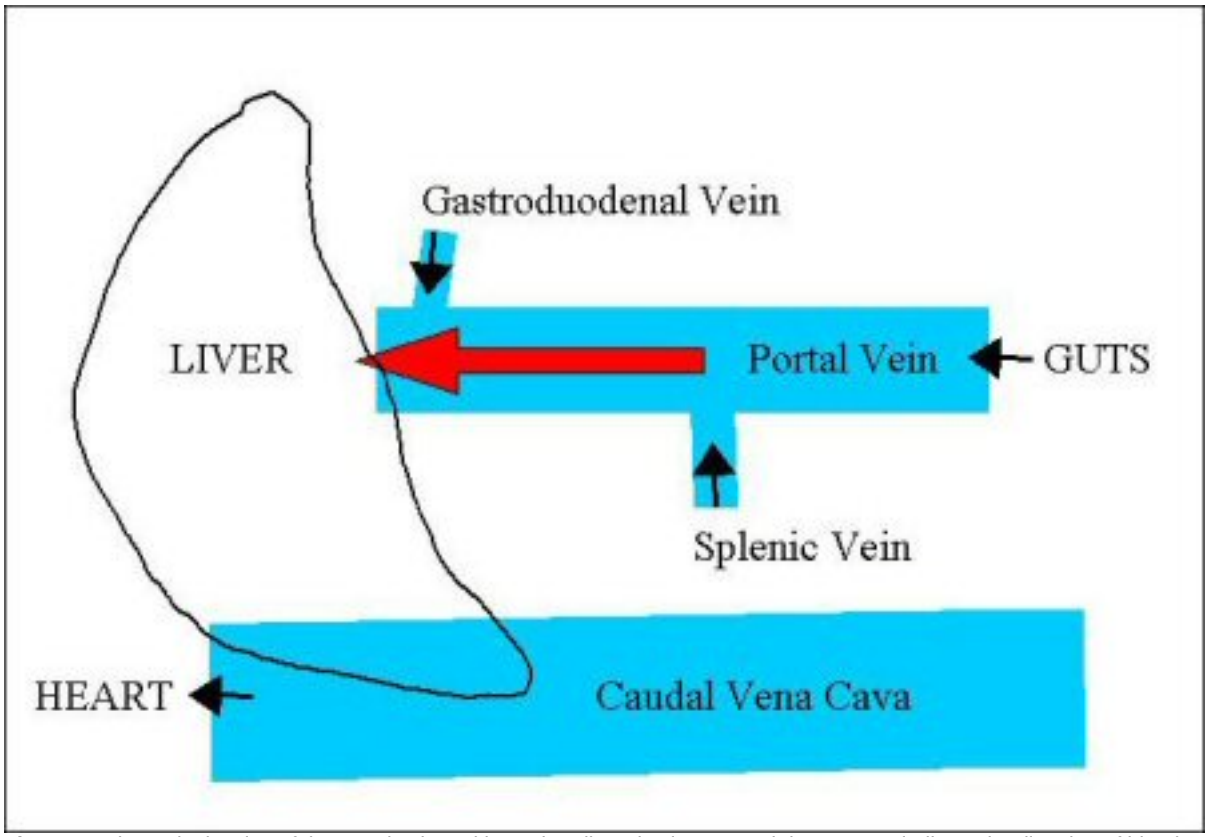

Figure 1 Schematic drawing of the portal vein and its major tributaries in a normal dog. Arrows indicate the direction of blood flow.

Portosystemic shunting occurs when an anomalous vein allows the portal blood to bypass the liver. This can happen via acquired portosystemic collaterals as a consequence of sustained portal hypertension or via a congenital portosystemic shunt (CPSS) as a result of a developmental anomaly. Congenital portosystemic shunts have been described in several mammals including humans, however the only species in which the anomaly is fairly common is the dog. Although there are several anatomic variations of the anomalous vessels a CPSS is always a large-bore vein that drains almost 100% of the portal blood to a systemic vein. The most common type of CPSS in dogs is the so-called spleno-caval shunt, which is an extrahepatic vessel that drains the portal blood via a splenic vein segment to the caudal vena cava.[1] (Figure 2, Video 1)

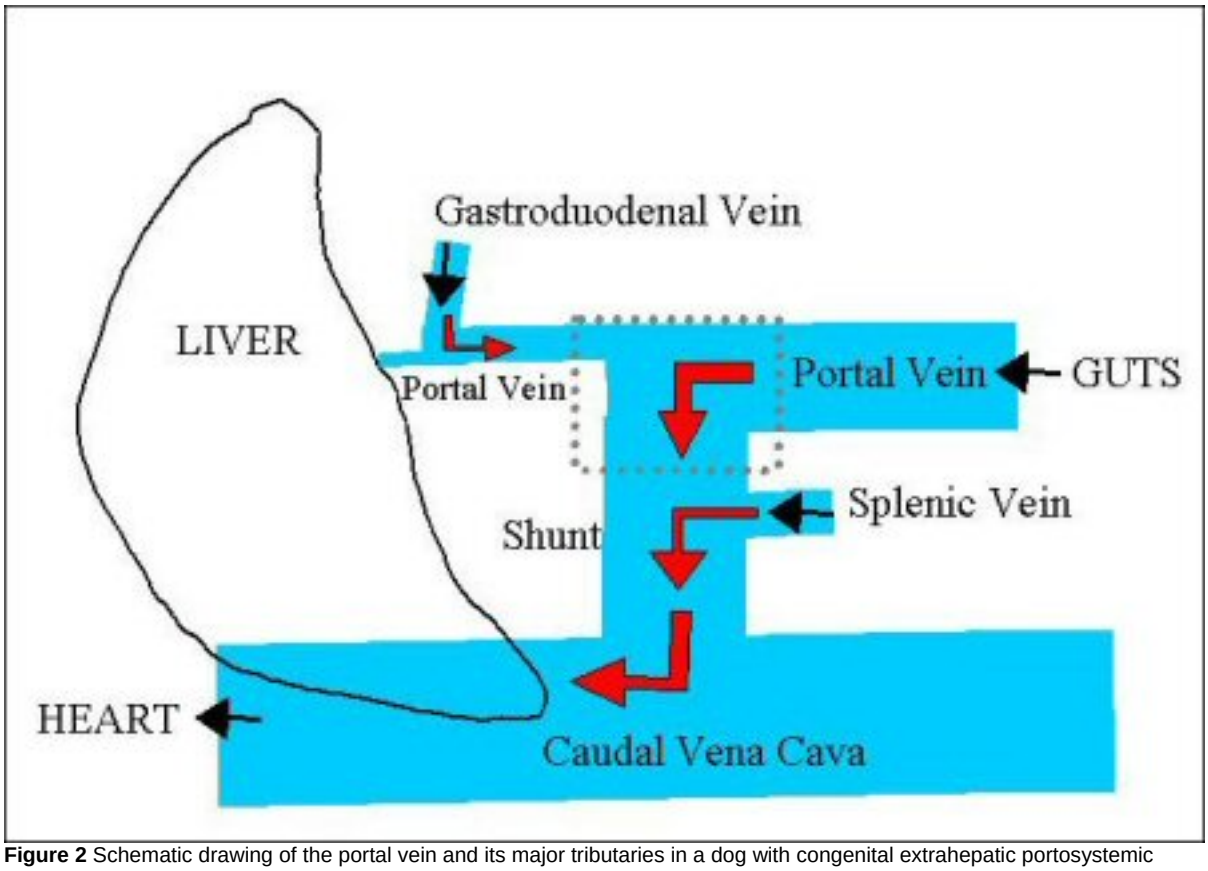

Figure 2 Schematic drawing of the portal vein and its major tributaries in a dog with congenital extrahepatic portosystemic (spleno-caval) shunt. Note that the portal vein becomes narrow cranial to the shunt-origin. The blood from the gastroduodenal vein (GDV) is responsible for the hepatofugal portal flow between the shunt-origin and GDV. The rectangular region can be seen in the videoclips 2-4, 6. Arrows indicate the direction of blood flow.

Video 1

Video 1 Intraoperative gray scale and color Doppler ultrasound images of a congenital extrahepatic spleno-caval shunt in a dog. In this image the shunt in its whole length can be seen (runs vertically) from the portal vein (top) to the caudal vena cava (left bottom). Note that the splenic vein (red colored) enters the middle portion of the shunt (from right). Red color represents flow towards the transducer and blue away from the transducer.

Affected dogs may show clinical signs at any age from as early as 3 months of age, however, for unknown reasons, the CPSS may remain clinically silent. Most symptoms are related to the high blood ammonia concentration and range from signs of hepatic encephalopathy, which is manifested in periods of abnormal behaviour, through stunted growth, to signs related to urinary tract problems due to urolithiasis. Congenital portosystemic shunt is suspected on the medical history and is justified by visualizing the abnormal vein by means of abdominal ultrasonography after measurement of an elevated blood ammonia level.[1] [2]

What can ultrasonography offer?

By means of gray scale (i.e. B-mode) ultrasonography detailed real-time anatomic information of any soft tissue (including blood vessels) can be obtained in a non-invasive way and without the use of ionizing radiation. Vessels on B-mode images appear black (i.e. echofree) with thin white (i.e. echorich) walls. In addition to the anatomic information color Doppler ultrasonography can detect the presence and direction of blood flow. Traditionally, a flow directed towards the transducer is coded with red, and a flow directed away from the transducer is coded with blue. Higher flow velocities are indicated by lighter hues.

How to treat congenital portosystemic shunts?

The definitive therapy for CPSSs would ideally be complete occlusion of the anomalous vessel. Most surgeons aim to narrow a CPSS as narrow as possible. However, in most dogs, only partial shunt ligation can be performed because occluding the shunt would often result in severe acute portal hypertension and subsequent shock or development of acquired portosystemic collaterals because of sustained post-ligation portal hypertension. The reason why portal hypertension develops is that shunt-ligation forces portal blood to flow through the underdeveloped (i.e. hypoplastic) portal

branches. Hypoplasia (i.e. small diameter, thus insufficient vascular capacity) occurs in the portal vein segment that is cranial to the origin of the CPSS because this segment has been hypoperfused since birth (Figure 2). This underdeveloped portal vein segment is unable to accommodate the normal amount of portal blood that will be forced to flow through it after the shunt has been ligated. The degree of post-ligation portal hypertension thus depends on (1) the degree of attenuation as well as (2) the severity of portal vein hypoplasia.

Surgical complications

Several methods have been advocated to overcome the above mentioned post-ligation complications, however, all of them failed to guarantee a favorable surgical outcome. Measuring portal pressure by direct catheterization of a portal tributary has been generally used. Although this method allows direct quantitative assessment of portal hypertension, several factors make the interpretation of pressure changes unreliable. [3] Another method is based on monitoring qualitative signs (changes of the intestine's color) 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. Both methods allow - in most cases - life-threatening acute portal hypertension to be successfully avoided. However, development of chronic portal hypertension remains a frequent complication.

Several years ago, ameroid constrictors were introduced in portosystemic shunt surgery. These devices are placed around the shunt vessels and cause their gradual occlusion. In theory, gradual shunt attenuation would allow the underdeveloped portal branches to become adapted to the increased blood flow. The major disadvantage of this method is that shunt-attenuation becomes an uncontrollable process and 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 does develop regularly. [3] Regardless of the technique used for shunt attenuation and for assessing post-ligation portal hypertension, the clinical outcome remains unpredictable.

Are the postoperative complications predictable and preventable with the use of ultrasonography?

We decided to evaluate the portal and shunt flow with color Doppler ultrasound during surgical attenuation of CPSSs with the hypothesis that if we could understand the portal hemodynamics better we might possibly prevent post-ligation complications. This technique had never been used before during surgery. To obtain color Doppler images of the portal vein both cranial and caudal to the shunt origin and of the shunt itself a sterile ultrasound probe (i.e. transducer) was placed directly on the portal vein at the point where the shunt originated from the portal vein. The evaluation was repeated after partial shunt attenuation.

Video 2

Video 2 Intraoperative color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt in a dog. 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. Red color represents flow towards the transducer and blue away from the transducer. See also Figure 2.

Already in the very first dog a very interesting phenomenon was noticed: the flow in the portal vein segment cranial to the shunt origin was hepatofugal (i.e. directed away from the liver; Video 2). This phenomenon turned out to be present in almost all dogs with extrahepatic CPSS. Actually, the hepatofugal flow was restricted to the portal vein segment between the shunt origin and the entering point of the gastroduodenal vein. The explanation of this hepatofugal flow must be that the blood from the gastroduodenal vein finds lower resistance to flow caudally towards the shunt than cranially to the hepatic sinusoids [3] (Figure 2). Ultrasonographic evaluation of the portal vein after partial shunt ligation made us even more excited because in the majority of the dogs the hepatofugal portal flow observed before shunt attenuation turned to hepatopetal (i.e. flow directed towards the liver) after shunt-attenuation.

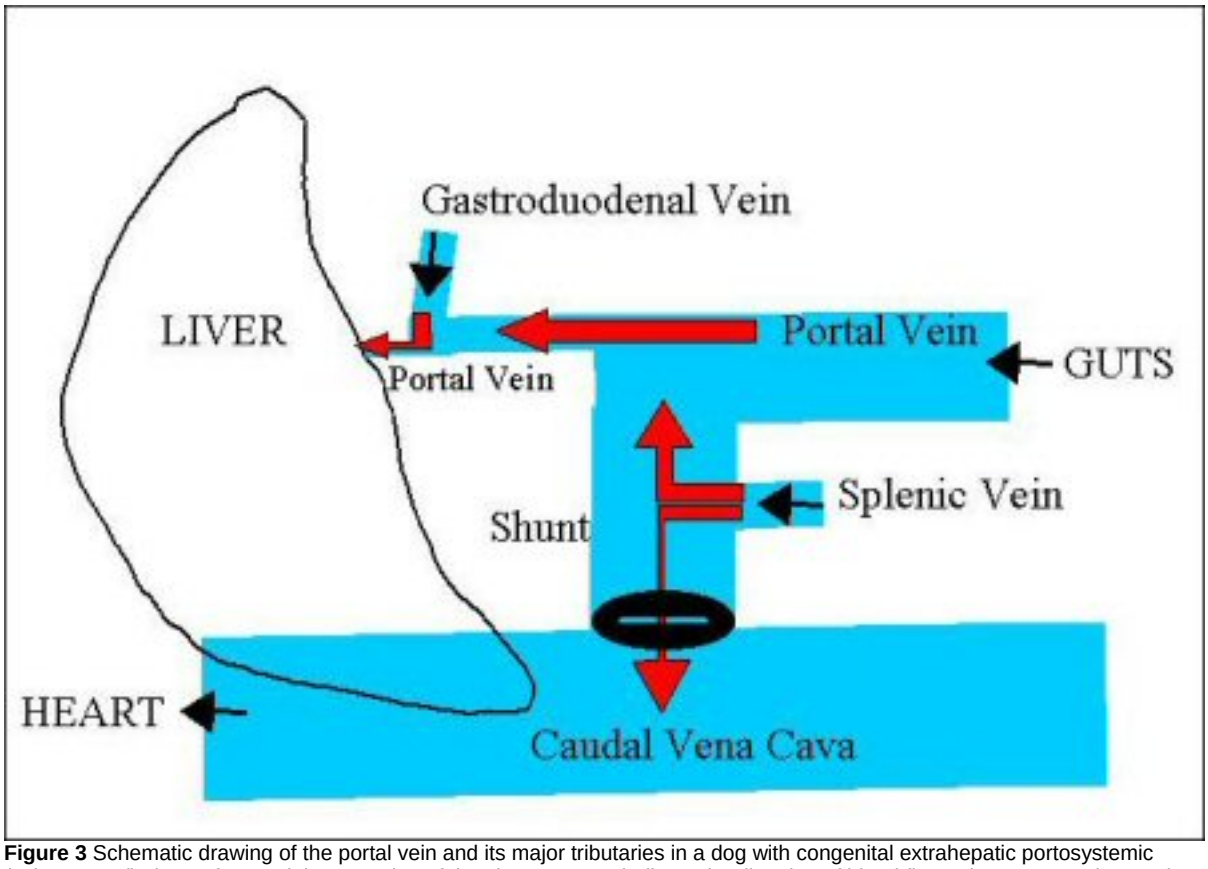

Figure 3 Schematic drawing of the portal vein and its major tributaries in a dog with congenital extrahepatic portosystemic (spleno-caval) shunt after partial attenuation of the shunt. Arrows indicate the direction of blood flow. Shunt-attenuation made the resistance towards the shunt higher than towards 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 caudal vena cava via the narrowed segment.

Video 3

Video 3 The same dog as on video 2. 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. Red color represents flow towards the transducer. See also Figure 3.

Post-operative follow-up studies revealed that the dogs, in which the portal flow remained hepatofugal after shunt attenuation failed to show clinical improvement after surgery, however, the dogs in which the portal flow became hepatopetal after surgery became clinically healthy. [3] When the flow in the portal vein cranial to the shunt origin remains hepatofugal during a temporary applied complete occlusion of a CPSS, a suboptimal surgical outcome may be predicted because of the presence of a severe portal vein hypoplasia [3] [4] (Video 4).

Video 4

Video 4 Intraoperative color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt in a dog. After partial shunt attenuation the flow-direction remained hepatofugal both in the shunt and in the portal segment cranial to the shunt-origin because of a severe portal vein hypoplasia. Blue color represents flow away from the transducer.

In these cases even complete shunt occlusion did not result in exceeding the resistance towards the systemic veins above the resistance of the severely underdeveloped portal branches. To our great surprise we found that shunt ligation made not only the portal flow hepatopetal, but also resulted in a hepatopetal flow in the shunt resulting in dogs with favorable outcome (Video 3). Hepatopetal flow in the shunt results from the anatomical fact that the splenic vein enters the shunting vessel and its blood finds lower resistance to flow via the shunt to the portal vein than towards the ligature after shunt ligation (Figure 3, Video 5).

Video 5

Video 5 Intraoperative color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt in a dog. These transverse images were made after occlusion of the shunt. The left vessel (blue) is the cross section of the caudal vena cava, the right side of the image shows how the splenic venous blood flows via the shunt vessel to the portal vein. Red color represents flow towards the transducer. See also Figure 3.

A great hemodynamic advantage of the hepatopetal flow in the shunt is that this flow prevents the toxin rich mesenteric venous blood from shunting (Figure 3). Though a fraction of the splenic venous

blood still flows through the partially attenuated shunt, this has no clinical significance, since the splenic vein contains equal amount of toxins than any systemic veins.

Is a second surgery necessary after a partial shunt closure?

Some surgeons believed in an improved clinical outcome after complete occlusion of CPSSs. However, others found no difference in outcomes between dogs with partial and complete shunt-ligation. It has been suggested that, if complete shunt-occlusion is not feasible during a surgical attenuation of a CPSS because it would cause fatal portal hypertension, a second surgery should be performed to attempt a complete shunt-occlusion. 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.

Actually, a second surgery should only be considered when portosystemic shunting persists exclusively through the CPSS. [4] If shunting occurs through acquired portosystemic collaterals, further attenuation of the CPSS is contraindicated because portal hypertension already exists. To determine whether post-ligation portosystemic shunting occurs via the attenuated CPSS, or via acquired portosystemic collaterals or both is therefore critical. For this purpose mesenteric portography has been used. In addition to the fact that this angiographic technique involves the use of ionizing radiation, it requires general anesthesia and an abdominal surgery. We described how acquired portosystemic collaterals can be recognized non-invasively with ultrasound in unsedated dogs. [1] [2] [4] We have shown that dogs that underwent a partial ligation of a CPSS do not have to be routinely re-operated. Dogs, in which the extrahepatic CPSS 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 because hepatopetal flow in the shunt adjacent to the portal vein prevents the toxin-rich portal blood from shunting. Persistent shunting of the splenic venous blood via the attenuated segment of the shunt has no clinical significance, therefore these shunts do not require further narrowing. These shunts are patent, but not functional and do not cause abnormal blood ammonia levels. A second surgery that aims at further narrowing of a partially attenuated extrahepatic CPSS is only recommended, if one month after surgery the flow in the portal vein cranial to the origin of the shunt is hepatofugal. We found that dogs with hepatopetal portal flow cranial to the shunt origin are clinically healthy regardless of the presence of portosystemic shunting. Thus, the clinical signs seem to be associated with the reduced portal venous perfusion of the liver rather than with the presence of shunting blood.

We discovered and explained a new pathophysiological phenomenon

In the portal vein of dogs with extrahepatic CPSS a new pathophysiological phenomenon was discovered and subsequently utilized in the development of novel criteria for determining the optimal diameter of a CPSS during its surgical attenuation. Based on the portal hemodynamic changes associated with surgical ligation of extrahepatic CPSSs we recommend the following Doppler ultrasonographic criteria for determining the optimal degree of shunt narrowing. The largest shunt diameter should be found that ensures hepatopetal flow in the entire portal vein and in the shunt adjacent to the portal vein. A smaller shunt diameter due to exaggerated shunt occlusion would only increase the degree of portal hypertension, but would not reduce shunting, nor would it improve hepatic perfusion (Video 6). Regardless of these flow directions, hepatopetal portal flow caudal to the shunt origin should always be maintained. [3] Hereby we demonstrated that extrahepatic CPSSs do not have to, and in fact should not be, completely occluded.

Video 6

Video 6 Intraoperative color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt in a dog. After partial shunt attenuation the flow-direction became hepatopetal both in the shunt and in the portal segment cranial to the shunt-origin, however, the flow in the portal vein caudal to the shunt origin became very slow indicating severe portal hypertension. This is presumably because of an exaggerated shunt closure. Red color represents flow towards the transducer.

Doppler ultrasonography is recommended to be used during every portosystemic shunt surgery

Source: Szatmári V. Ultrasonography of portosystemic shunting in dogs; Doppler studies before, during and after surgery. PhD-thesis, Utrecht University, Utrecht, The Netherlands, 2004.

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