Portosystemic shunts in dogs and cats: imaging portosystemic shunts in small animals – hepatic vascular anatomy, shunt morphology, radiography

Portosystemische shunts bij honden en katten: beeldvorming van portosystemische shunts bij kleine huisdieren – hepatische vasculaire anatomie, shuntmorfologie, radiografie

[1]H. Haers, [2]D. Paepe, [1]K. Vermote, [1]J.H. Saunders, [2]S. Daminet, [1,2]M. Risselada

[1]Department of Medical Imaging of Domestic Animals
[2]Department of Small Animal Medicine and Clinical Biology,
Faculty of Veterinary Medicine, Ghent University
Salisburylaan 133, B-9820 Merelbeke, Belgium

hendrikhaers@hotmail.com

ABSTRACT

Portosystemic shunts (PSSs) are anomalous vascular communications between the portal vein or its branches and the systemic venous system. Signalment, history, clinical signs, and laboratory findings can already provide a presumptive diagnosis of a PSS. However, imaging techniques such as portography, ultrasonography (US), nuclear scintigraphy, computed tomography (CT), or magnetic resonance imaging (MRI) are required to provide a definitive diagnosis. Nuclear scintigraphy is the gold standard for detecting PSSs, but it is not useful in distinguishing the different types of shunts. Due to its high sensitivity, portography was for a long time considered the gold standard for the differentiation of PSSs, even though it is an invasive technique. However, the recent development of a standard protocol for ultrasound (US) and the routine use of Doppler modalities resulted in the same sensitivity as portography. Therefore, with the additional benefit of being fast and noninvasive, US is now more commonly performed. It may be suggested as a "new" gold standard, depending upon the experience of the radiologist. Computed tomography and MRI provide detailed anatomic information. In this third part about portosystemic shunts in dogs and cats the different types of shunts and their diagnosis using portography will be explained.

SAMENVATTING

Portosystemische shunts (PSS's) zijn abnormale vasculaire verbindingen tussen de vena porta of zijn vertakkingen en de systemische veneuze circulatie. Via het signalement, de anamnese, de klinische symptomen en de laboratoriumbevindingen kan een waarschijnlijkheidsdiagnose gesteld worden. Beeldvormingstechnieken, zoals portografie, echografie, nucleaire scintigrafie, computertomografie (CT), of magnetische resonantie (MR), zijn echter noodzakelijk om een definitieve diagnose te bekomen. Nucleaire scintigrafie is de gouden standaard voor de detectie van PSS's, maar is niet nuttig om het onderscheid te maken tussen de verschillende shunttypen. Omwille van de hoge sensitiviteit werd portografie lange tijd beschouwd als de gouden standaard voor de differentiatie van PSS's, alhoewel het een invasieve techniek is. De recente ontwikkeling van een standaardprotocol voor echografie en het routinematig gebruik van dopplertechnieken resulteerden in dezelfde sensitiviteit als die bij portografie. Echografie heeft als voordeel dat het snel en niet-invasief is; tegenwoordig wordt ze meer en meer uitgevoerd. Echografie kan beschouwd worden als een "nieuwe" gouden standaard, afhankelijk van de ervaring van de radioloog. Computertomografie en MR geven gedetailleerde anatomische informatie. In dit derde deel over portosystemische shunts bij honden en katten worden de verschillende shunttypen en hun diagnosestelling met behulp van portografie beschreven.

INTRODUCTION

Portosystemic shunts (PSSs) are macroscopic vascular connections between the portal vein (PV) system and a systemic vein such as the caudal vena cava (CVC). These abnormal communications allow the portal blood from the intestine to bypass the liver and enter the systemic circulation. PSSs can be classified as congenital or acquired, single or multiple, and intra- or extrahepatic (Birchard *et al.,* 1989; Lamb, 1996; Lamb and Daniel, 2002; Tillson and Winkler, 2002; Santilli and Gerboni, 2003; Broome *et al.,* 2004; Cole *et al.,* 2005). Ferrell *et al.* (2003) described the rare simultaneous occurrence of congenital and acquired extrahepatic PSSs in dogs.

Congenital portosystemic shunting is considered if a single or double abnormal vessel is present without accompanying portal hypertension. Eighty percent of all PSSs in dogs are congenital PSSs (CPSSs), which are typically classified as intra- or extrahepatic, based upon their location (Broome *et al.,* 2004; Szatmári *et al.,* 2004b). Intrahepatic shunts make up approximately one-third of the CPSSs (Lamb, 1996; White *et al.,* 2003; Broome *et al.,* 2004; Szatmári *et al.,* 2004b).

Acquired PSSs (APSSs) are considered when multiple small extrahepatic collateral vessels occur secondary to portal hypertension (Boothe *et al.,* 1996; Johnson, 1999; Lamb and Daniel, 2002; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004a). Acquired PSSs constitute approximately 20% of all PSSs in dogs (Boothe *et al.,* 1996; d'Anjou *et al.,* 2004). They are less commonly detected in cats. Acquired PSSs originate from normal, preexisting, nonfunctional communications between the PV and the systemic circulation. These communications open secondary to and compensate for sustained portal hypertension (Bostwick and Twedt, 1995; Boothe *et al.,* 1996; Center, 1996b; Johnson, 1999; Lamb and Daniel, 2002; Tillson and Winkler, 2002; Ferrell *et al.,* 2003; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004a).

Acquired PSSs are commonly caused by increased intrahepatic portal venous resistance due to liver fibrosis or cirrhosis, but can also appear secondary to arterioportal fistulae, PV thrombosis, PV hypoplasia (also known as idiopathic noncirrhotic portal hypertension), and PV aplasia or atresia (Johnson, 1999; Szatmári *et al.,* 2000; Lamb and Daniel, 2002; Ferrell *et al.,* 2003; Santilli and Gerboni, 2003; White *et al.,* 2003; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b; Zandvliet *et al.,* 2005). Portal vein hypoplasia also occurs very commonly secondary to reduced hepatic portal flow, in association with a CPSS (White *et al.,* 2003).

Hepatic microvascular dysplasia (HMD), unlike PSSs, involves intrahepatic microscopic shunting of blood. It may occur alone or associated with a macroscopic shunting vessel (Broome *et al.,* 2004). The relationship between HMD and CPSSs is unclear (Johnson, 1999). Hepatic microvascular dysplasia is characterized by the presence of residual "juvenile-like" intralobular blood vessels, through which portal blood flow runs directly to the central veins, bypassing the sinusoids (Santilli and Gerboni, 2003). It is being reported in an increasing number of dog breeds and represents an important differential diagnosis of PSSs. It causes high serum bile acids concentrations without demonstrable macroscopic portosystemic shunting (Center, 1996a; Johnson, 1999; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b).

Signalment, history, clinical signs, and laboratory findings can already provide a presumptive diagnosis of a PSS (Levy *et al.,* 1995; Center, 1996b; Tillson and Winkler, 2002; Santilli and Gerboni, 2003; Broome *et al.,* 2004). However, imaging techniques such as radiography (Wrigley *et al.,* 1987a; Birchard *et al.,* 1989; Santilli and Gerboni, 2003; Broome *et al.,* 2004), ultrasonography (US) (Lamb, 1996; Lamb, 1998; Lamb and White, 1998; Szatmári *et al.,* 2004d), nuclear scintigraphy (Daniel *et al.,* 1990; Koblik *et al.,* 1990a and 1990b; Cole *et al.,* 2005; Morandi *et al.,* 2005), computed tomography (CT) (Frank *et al.,* 2003; Thompson *et al.,* 2003; Zwingenberger and Schwarz, 2004; Zwingenberger *et al.,* 2005), and magnetic resonance imaging (MRI) (Seguin *et al.,* 1999) are required to provide a definitive diagnosis and are very helpful in localizing the shunt and assessing its morphology before surgical attenuation. These imaging techniques enable efficient and rapid surgical manipulation of the shunt, which results in less anesthetic time (Birchard *et al.,* 1989; Johnson, 1999; Lamb and Daniel, 2002; Santilli and Gerboni, 2003).

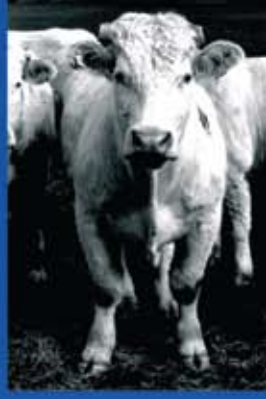
Portography was considered the gold standard in dogs suspected of having a PSS (Johnson, 1999; Lamb and Daniel, 2002; Miller *et al.,* 2002; Tillson and Winkler, 2002), because the interpretation of the portographic images is quite easy. It allows differentiation of CPSSs versus APSSs and it can be used to distinguish intra- from extrahepatic shunts (Szatmári *et al.,* 2004a). However, it is time-consuming, invasive and requires radiation (Lamb and Daniel, 2002; Broome *et al.,* 2004; Szatmári *et al.,* 2004a). These downsides, combined with the development of more advanced and less-invasive techniques, have led to decreased use of portography.

Ultrasonography has been used for diagnostic imaging of dogs with CPSSs since the 1980s (Wrigley *et al.,* 1987b). It has become popular because it is quick and noninvasive, and it does not require anesthesia or ionizing radiation. It was found to be nearly as sensitive a method for detecting extra- and intrahepatic PSSs as portography (Holt *et al.,* 1995; Scrivani *et al.,* 2001; Szatmári *et al.,* 2004a and 2004b). Furthermore, it allows simultaneous evaluation of the abdominal organs and, when using Doppler, it can be used to assess abnormal blood flow (Szatmári *et al.,* 2003, Szatmári *et al.,* 2004a).

Although scintigraphy is considered the gold standard for detecting PSSs, it is not useful for distinguishing CPSSs from APSSs, or intrahepatic from extrahepatic shunts (Broome et al. 2004; Szatmári *et al.,* 2004a; Cole *et al.,* 2005; Morandi *et al.,* 2005).

Computed Tomography and MRI can provide anatomic information. However, unlike US, these modalities cannot give information on the direction of blood flow in the vessels examined. Moreover, both procedures are time-consuming, use expensive equipment, and require general anesthesia (Seguin *et al.,* 1999; Frank *et al.,* 2003; Szatmári *et al.,* 2004a;

Vétoquinol
a Sign of Passion



Zin om je grenzen te verleggen?

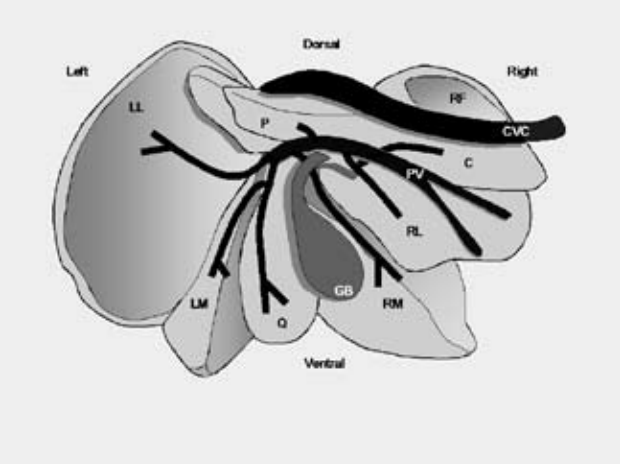


Figure 1. Normal hepatic divisional anatomy and intrahepatic portal branching in a dog. Left divisions: LL = left lateral lobe; LM = left medial lobe; P = papillary process of caudate lobe. Central divisions: RM = right medial lobe; Q = quadrate lobe. Right divisions: C = caudate process of the caudate lobe; RL = right lateral lobe. (PV = portal vein; CVC = caudal vena cava; RF = renal fossa; GB = gallbladder)

Zwingenberger and Schwarz, 2004).

The hepatic vascular anatomy, its shunt morphology, and the diagnosis using portography are discussed in more detail.

HEPATIC VASCULAR ANATOMY AND SHUNT MORPHOLOGY

The portal system receives blood from the cranial and caudal mesenteric veins, the splenic vein and the gastroduodenal vein, which drain the gastrointestinal tract, pancreas, spleen, caudal thoracic esophagus and the greater part of the rectum (Center, 1996b; Tillson and Winkler, 2002; Broome *et al.,* 2004).

In dogs, the intrahepatic PV consists of a right and a left branch.The left branch supplies the left (the papillary process of the caudate lobe and the left late-ral and medial lobes) and central divisions of the liver (right medial and quadrate lobes). The right branch supplies the right hepatic divisions (the caudate process of the caudate lobe and the right lateral lobe) (Figure 1; Center, 1996b; Tillson and Winkler, 2002; Broome *et al.,* 2004). The right branch reaches the liver before the left branch, which may be noted during positive-contrast portography (Center, 1996b). The PV in the cat divides into right-, central- and left divisions (Tillson and Winkler, 2002; Broome *et al.,* 2004).

The hepatic artery originates from the celiac artery. The celiac artery lies dorsal to the PV and common bile duct. It completes an arch before terminating as the right gastric and gastroduodenal arteries. Usually, each hepatic division has its own arterial supply. Two or three arterial branches originate from the arch and supply the liver (Center, 1996a).

The hepatic veins are variable in number and location, and they enter the CVC before it crosses the diaphragm. Usually there are 6 to 8 hepatic veins. The left hepatic vein is the largest and the most consistently and most cranially located segment (Center, 1996b; Broome *et al.,* 2004).

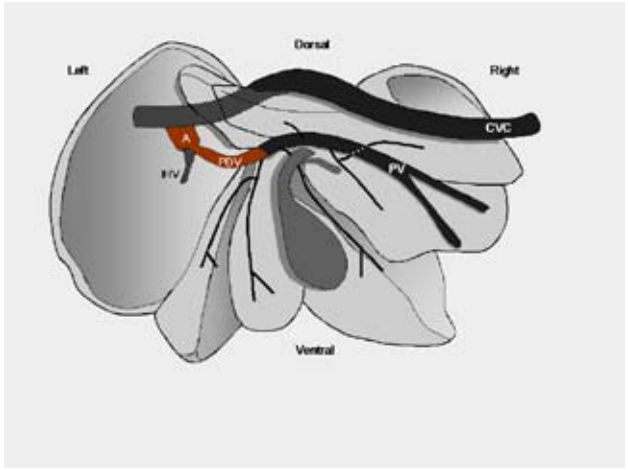


Figure 2A. Left-divisional shunts arise from a left branch of the PV, then turn abruptly dorsally via the patent ductus venosus (PDV) to enter the CVC or left hepatic vein (lHV). The confluence of PDV and lHV is usually dilated and is known as the ampulla (A).

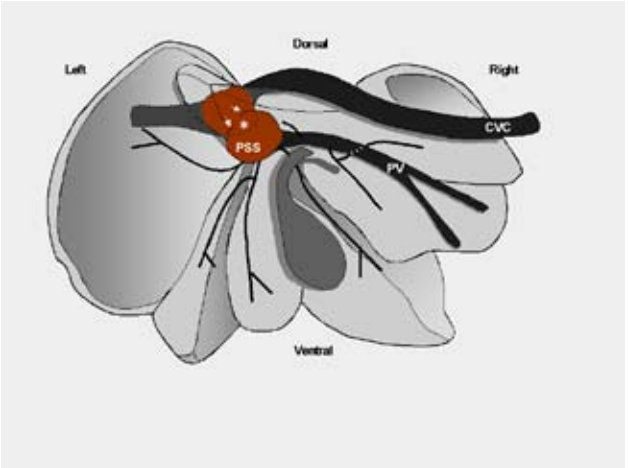


Figure 2B. Central-divisional shunts appear as a kind of foramen (*) between marked, aneurysmal dilations of the intrahepatic PV and CVC. In some cases, a thin membrane-like structure (white arrows) can be visible separating these two vessels.

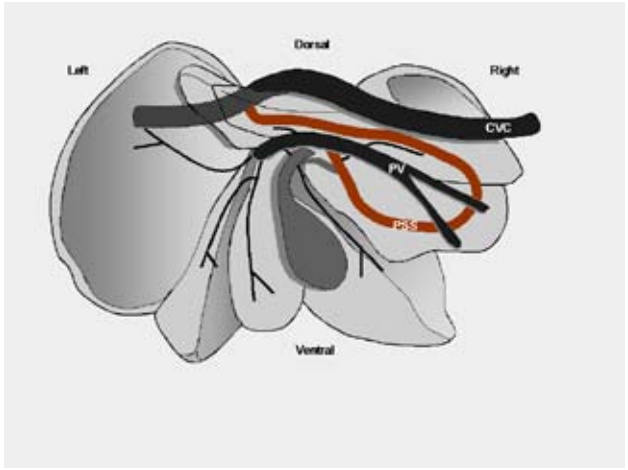


Figure 2C. Right-divisional shunts are long, tortuous shunts originating from the right portal branch. They pass through the right lateral or caudate lobe, sometimes forming a wide loop, before entering the CVC.

Vétoquinol
a Sign of Passion

Marbocyl®S is echt innovatief:

Zijn concentratie afhankelijke bactericiede werking is zo snel en intens dat één enkele injectie volstaat voor een optimale antibacteriële werking.

Een doorbraak bij de behandeling van respiratoire problemen

Expovet
Stand Vétoquinol

Marbocyl®

Pushing back the boundaries

www.marbocyl.com

Benaming Marbocyl®S 10%, oplossing voor injectie – **Doeldieren:** Runderen – **Indicaties:** Therapeutische behandeling van luchtweginfecties veroorzaakt door gevoelige stammen van *Pasteurella multocida, Mannheimia haemolytica* en *Histophilus somni* – **Contra-indicaties:** Niet gebruiken bij dieren met een gekende overgevoeligheid voor fluoroquinolones. Niet gebruiken als de betrokken ziekteverwekker resistent is voor andere fluoroquinolonen (kruisresistentie) – **Bijwerkingen:** Het is bekend dat fluoroquinolones arthropatieën kunnen induceren, doch dit effect werd nooit vastgesteld voor marbofloxacine in runderen. Toediening via de intramusculaire weg kan voorbijgaande plaatselijke reacties veroorzaken zoals pijn en zwelling op de injectieplaats en ontstekingslaesies die minstens tot 12 dagen na injectie aanwezig kunnen blijven. Er werden geen andere bijwerkingen vastgesteld bij runderen – **Dosering en toedieningwijze:** De aanbevolen dosering is 8 mg/kg lichaamsgewicht, dit komt overeen met 2ml/25 kg lichaamsgewicht als een eenmalige intramusculaire injectie. Als het in te spuiten volume meer is dan 20 ml, moet dit verdeeld worden over twee of meer injectieplaatsen – **Wachttijd:** vlees en vleesafval: 3 dagen; melk: 72 uren – **Registratiehouder:** Vétoquinol NV, Kontichsesteenweg 42, B – 2630 Aartselaar – *Bijkomende informatie is verkrijgbaar op aanvraag.*

Intrahepatic shunts have been classified as left-, central- or right-divisional (Figure 2(A-C); Bostwick and Twedt, 1995; Lamb *et al.,* 1996; White *et al.,* 1996; Lamb and White, 1998; Lamb and Daniel, 2002; Szatmári *et al.,* 2004b). Left-divisional shunts (patent ductus venosus) are shunts originating from the left portal branch. Right-divisional shunts are long shunts originating from the right portal branch, whereas central-divisional shunts are short shunts originating from the right portal branch (Szatmári *et al.,* 2004b). Left-divisional shunts occur most commonly (Center, 1996b; Broome *et al.,* 2004; Tobias *et al.,* 2004). They are caused by failure of the fetal ductus venosus to close properly after birth. The ductus connects the embryologic left umbilical vein and the cranial portion of the right vitelline vein (Bostwick and Twedt, 1995; Center, 1996b; Santilli and Gerboni, 2003; Broome *et al.,* 2004). In puppies, the ductus venosus normally closes two to six days after birth (Lamb and White, 1998; Broome *et al.,* 2004; Lamb and Burton, 2004; Tobias *et al.,* 2004). The origin of central- and right-divisional intrahepatic shunts is unknown (Lamb and White, 1998; Broome *et al.,* 2004).

Intrahepatic PSSs are diagnosed as large vessels connecting the intrahepatic portal and hepatic venous vascular beds. They usually keep a uniform diameter throughout, are larger than normal hepatic vessels, and have abnormal, tortuous courses (Holt *et al.,* 1995). Left-divisional CPSSs have a relatively consistently bent tubular shape. They run cranioventrally, extending from the PV and to the left (as the normal left portal branch) until the level of the diaphragm, then turn abruptly dorsally to enter the CVC via a dilated segment of the left hepatic vein (Figure 2A; Bostwick and Twedt, 1995; Lamb *et al.,* 1996; White *et al.,* 1996; Lamb and White, 1998; Szatmári *et al.,* 2004b). The confluence of the patent ductus and left hepatic vein is usually dilated and is known as the ampulla (Lamb and Daniel, 2002). Central-divisional shunts appear as a kind of foramen between marked, aneurysmal dilations of the intrahepatic PV and CVC. In some cases, a thin membrane-like structure can be visible separating these two vessels (Figure 2B; Lamb and White, 1998; Lamb and Daniel, 2002). Right-divisional shunts are large, tortuous vessels. These shunts consistently run dorsolaterally and to the right from the portal vein (as normal right portal branch), after which, instead of ramifying, they turn medially to enter the CVC (Lamb *et al.,* 1996; Lamb and White, 1998; Lamb and Daniel, 2002; Szatmári *et al.,* 2004b). They pass through the right lateral or caudate lobe, sometimes forming a wide loop, before entering the CVC (Figure 2C; Bostwick and Twedt, 1995; White *et al.,* 1996; Lamb and White, 1998; Lamb and Daniel, 2002).

In cats, left-divisional shunts are not very different from those in dogs. A right-divisional shunt was found to be similar to those in dogs (Lamb and White, 1998).

Embryologically, extrahepatic PSSs result from abnormal connections between the fetal cardinal and vitelline venous sytems (Bostwick and Twedt, 1995; Center, 1996b; Tillson and Winkler, 2002; Ferrell *et al.,* 2003; Santilli and Gerboni, 2003; Broome *et al.,* 2004). They originate from the main trunk of the PV or one of its tributaries, for example the splenic vein, the left gastric vein, or the right gastric vein. The most common type of CPSS is a splenic-caval shunt (Bostwick and Twedt, 1995; Center, 1996b; Lamb and Daniel, 2002; Broome *et al.,* 2004; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b; Cole *et al.,* 2005). Extrahepatic PSSs are diagnosed when an abnormal branch of the PV or one of its tributaries is identified caudal to the porta hepatis. PSSs that do not terminate in the CVC, but can be traced cranially and dorsally to the diaphragm, are presumed to be portoazygos shunts (Holt *et al.,* 1995; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b).

A splenic-caval shunt usually shows as a short dilated loop between the PV and the CVC. The point of origin of the shunt is very close to the point where the splenic vein enters the PV. The CPSS seems to originate from the PV itself and the splenic vein seems to enter the shunting vessel. The origin of a splenic-caval shunt is slightly cranial to the level where the celiac artery originates from the aorta (Szatmári *et al.,* 2004b).

Fewer shunts have been described that involve the gastroduodenal and mesenteric vessels (Center, 1996b; Broome *et al.,* 2004). Most dogs with right gastric-caval shunts have double shunts. There is a cranial shunt loop and a caudal shunt loop, which anastomose and form a common trunk before entering the systemic venous system. The cranial loop originates from the right gastric vein. The right gastric vein can be a tributary of the gastroduodenal vein or of the PV itself. In both cases, however, the shunt originates immediately caudal to the portal bifurcation. The course of the shunt was found to be always the same: a long loop starting from the porta hepatis, along the left body wall, turning caudomedially and terminating into the CVC at the point where splenic-caval shunts terminate. The (occasionally absent) caudal shunt loop originates in the region where splenic-caval shunts are expected. This loop, however, has a caudal-to-cranial direction instead of a ventral-to-dorsal direction as in splenic-caval shunts (Szatmári *et al.,* 2004b).

Congenital PSS's commonly terminate into the left aspect of the CVC just cranial to the phrenicoabdominal veins, or into the azygos vein (Bostwick and Twedt, 1995; Center, 1996b; Lamb and Daniel, 2002; Broome *et al.,* 2004; Szatmári *et al.,* 2004b; Cole *et al.,* 2005). Besides terminating into the abdominal portion of the CVC, CPSSs can also empty into its thoracic portion (Szatmári *et al.,* 2004b). The shunting vessel of splenic-azygos shunts runs towards the CVC and does not terminate, but continues further dorsal to the CVC. It eventually enters the thorax (Holt *et al.,* 1995; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b). Right gastric-azygos shunts do not enter the CVC, but pass it and enter the thorax (Szatmári *et al.,* 2004b). Occasionally, CPSSs empty into the hepatic veins, renal vein, phrenicoabdominal vein or the internal thoracic vein (Tillson and Winkler, 2002; Santilli *et al.,* 2003).

Acquired PSSs are small tortuous veins in the omentum or retroperitoneum near the left renal vein (Boothe *et al.,* 1996; Johnson, 1999; Lamb and Daniel,

2002; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b). They have typical patterns. In small animals, splenorenal and mesenteric collaterals are most commonly encountered, but gastrophrenic, pancreaticoduodenal, or rarely hemorrhoidal collaterals can also be found (Cole *et al.,* 2005; Morandi *et al.,* 2005). These last two cannot be identified on splenoportography and trans-splenic portal scintigraphy (Cole *et al.,* 2005). The collateral vessels of APSSs only occasionally arise directly from the PV (Szatmári *et al.,* 2004a). The morphology of APSSs that originate from the PV shows several differences compared with congenital extrahepatic splenic-caval shunts. The most important difference is that APSSs run caudally from the point of origin and it is very difficult to follow their path (Boothe *et al.,* 1996; Ferrell *et al.,* 2003; d'Anjou *et al.,* 2004; Szatmári *et al.,* 2004b), whereas CPSSs go cranially from their origin and can always be followed from their origin to their termination. Furthermore, APSSs are never wider than the PV caudal to the shunting vessel, whereas a CPSS is usually wider (Szatmári *et al.,* 2004b).

PLAIN ABDOMINAL RADIOGRAPHY

Abdominal survey radiographic findings are usually unremarkable but can include a small liver (microhepatica), enlarged kidneys, urinary calculi, and loss of serosal detail (Boothe *et al.,* 1989; Gonzalo-Orden *et al.,* 2000; Steyn, 2000; Lamb and Daniel, 2002; Santilli and Gerboni, 2003). Microhepatica, a common finding in dogs with a CPSS, is a less consistent finding in cats (Levy *et al.,* 1995; Johnson, 1999). Mild renomegaly (normal kidney size: 2.5-3.5 times the length of L2 in dogs; 2.4-3.0 times the length of L2 in cats) (Feeney and Johnston, 2002) is occasionally noted in dogs and cats with a CPSS. Ammonium urate calculi, when present, are usually invisible, unless they contain considerable amounts of magnesium and phosphate (Johnson, 1999; Steyn, 2000). Animals with PSSs frequently have reduced intraabdominal fat or ascites, resulting in a loss of serosal detail (Birchard *et al.,* 1989; Boothe *et al.,* 1996; Johnson, 1999; Steyn, 2000; Tillson and Winkler, 2002; Ferrell *et al.,* 2003).

POSITIVE-CONTRAST PORTOGRAPHY

Positive-contrast portography was considered the gold standard for imaging PSSs (Johnson, 1999; Lamb and Daniel, 2002; Miller *et al.,* 2002; Tillson and Winkler, 2002; Frank *et al.,* 2003; Broome *et al.,* 2004). Water-soluble iodinated contrast agents are used. Several methods can be used: the contrast can be introduced into arteries supplying the bowel (cranial mesenteric arteriography), into a mesenteric vein (intraoperative mesenteric portography), into the pulp of the spleen (percutaneous splenoportography), or retrograde into subsequently the azygos and the CVC through the jugular vein (transvenous retrograde portography) (Schmidt and Suter, 1980; Birchard *et al.,* 1989; Johnson, 1999; Steyn, 2000; Lamb and Daniel, 2002; Miller *et al.,* 2002; Broome *et al.,* 2004).

Portography confirms the presence of a PSS and it provides information on the anatomical location of the vessel, the direction of portal blood flow, and the patency of the PV and its intrahepatic branches (Levy *et al.,* 1995; Lamb and Daniel, 2002; White *et al.,* 2003; Broome *et al.,* 2004).

During portography, some confusion may exist about the intra- or extrahepatic location of the PSS. For example, poor visualization of the liver due to loss of serosal detail can hamper classification of the shunt (Birchard *et al.,* 1989). To avoid this problem, Birchard *et al*. (1989) evaluated the shunt location relative to the thoracolumbar spine. They found that if the caudal aspect of the PSS (where the shunt diverges from the PV) is cranial to T13, the shunt is probably intrahepatic. If this caudal aspect is caudal to T13, there is a high probability of the shunt being extrahepatic.Furthermore, if the caudal extent of the shunt does not extend caudally to the T12/T13 intervertebral disc space during full inspiration, it is likely to be intrahepatic. If any portion of the shunt is at T13 or caudally during full expiration, it is likely to be extrahepatic (Birchard *et al.,* 1989).

Recently, Scrivani *et al*. (2001) found that left lateral positioning during portography resulted in the highest sensitivity for detection of PSSs. The sensitivity of mesenteric portography for detecting an abnormal portosystemic blood vessel was 85, 91 and 100% in dorsal, right lateral, and left lateral recumbency, respectively.

The most important disadvantage of operative mesenteric portography is its invasive nature (Lamb and Daniel, 2002; Frank *et al.,* 2003; Broome *et al.,* 2004), and the need for mobile radiographic equipment or a radiography room clean enough for a surgical procedure (Lamb and Daniel, 2002; Szatmári *et al.,* 2003; Broome *et al.,* 2004). Moreover, using contrast portography, the ability to outline a vessel is dependent on the flow rate, concentration and volume of the contrast agent, and the position of the patient (Scrivani *et al.,* 2001; Cole *et al.,* 2005).

Radiographic subtraction techniques make portography more sensitive. These techniques remove the images of the overlying intra-abdominal structures, which improves the radiographic contrast of the portal system on portal venograms. The improved quality of the subtracted portograms aids in the detection of PSSs and intrahepatic PV branches. Subtraction studies are able to reveal PSSs not detected on the initial portal venogram (Wrigley *et al.,* 1987a; Steyn, 2000).

Operative mesenteric portography

This is the most commonly used and preferred technique because it allows a high-quality study of the portal system, it does not require special equipment, and it results in few complications (e.g. infection, bleeding, extravasation of contrast medium). It is performed with the patients under general anesthesia. A loop of jejunum is isolated through a ventral midline incision. A catheter is placed surgically in a jejunal vein and secured with ligatures, after which the iodinated contrast medium is injected as a bolus (1-2 ml/kg) (Holt *et al.,* 1995; Levy *et al.,* 1995; Center, 1996b; Johnson, 1999; Steyn, 2000; Lamb and Daniel, 2002; Tillson and Winkler, 2002; San-

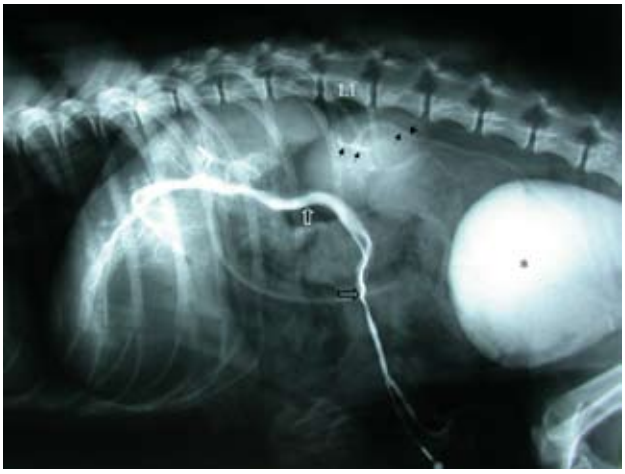


Figure 3A. Mesenteric portogram in a dog with a normal portal system. The contrast medium is injected in a jejunal vein (open black arrow) which terminates into the portal vein (PV) (open white arrow) at the level of the first lumbar vertebra (L1). A normal portogram reveals the PV trunk and its multiple intrahepatic branches. Note also the presence of contrast into the renal pelvices, ureters (black arrows) and bladder (*).

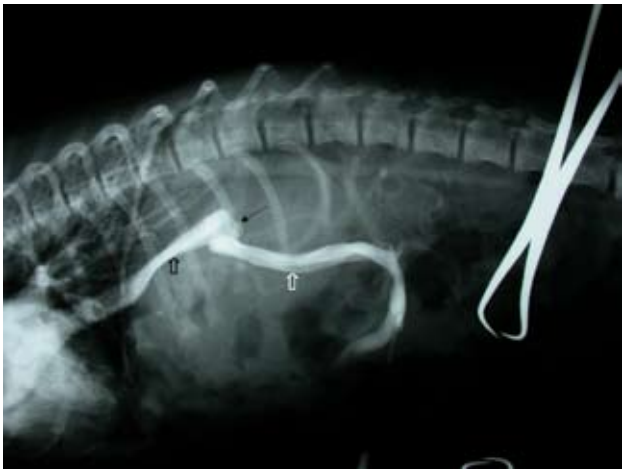


Figure 3B. Mesenteric portogram in a dog with a left-divisional intrahepatic PSS. The contrast medium is directly flowing from the portal vein (open black arrow) into the caudal vena cava (open white arrow) through the shunt (black arrow). There is no opacification of the intrahepatic portal system.

tilli and Gerboni, 2003) with a maximum dose of 2-4 ml/kg (Broome *et al.,* 2004). In the early days, the injection was recorded using a rapid film changer (Steyn, 2000; Lamb and Daniel, 2002; Tillson and Winkler, 2002; Santilli and Gerboni, 2003; Broome *et al.,* 2004). Nowadays, however, videotape, CDs, and DVDs are used.

A normal portogram reveals the PV trunk and its multiple intrahepatic branches (Figure 3A). The portogram of a patient with a PSS initially shows contrast in the PV, which then moves into the CVC or azygos vein. A tortuous vessel starting from the portal system usually represents the shunt (Figure 3B; Johnson, 1999; Tillson and Winkler, 2002).

Intrahepatic PV branches may or may not be opacified. Animals with portal atresia do not show any evidence of either hepatic parenchymal opacification, an intrahepatic portal vasculature, or a PV entering the liver (Johnson, 1999; White *et al.,* 2003). In cases of portal hypoplasia, portography shows dilation of the extrahepatic PV, APSSs, an insufficient number of middle-sized intrahepatic PV branches, and sudden ending of the peripheral venous branches (Johnson, 1999; Bunch *et al.,* 2001). Therefore, failure to outline the intrahepatic portal system may suggest higher intrahepatic vascular resistance. These patients are more likely to develop postoperative complications (Johnson, 1999).

Cranial mesenteric arteriography

With this alternative technique, contrast medium is selectively delivered to the cranial mesenteric artery via a femoral arteriotomy using fluoroscopic guidance (Schulz *et al.,* 1993; Center, 1996b; Steyn, 2000; Lamb and Daniel, 2002). The catheter tip is placed in the origin of the cranial mesenteric artery (Center, 1996b; Steyn, 2000). Following injection of contrast medium, the contrast bolus passes through the intestinal capillary bed and opacifies the PV (Schulz *et al.,* 1993; Steyn, 2000; Lamb and Daniel, 2002). An important disadvantage of this technique is the dilution of the contrast by the time it reaches the PV (Wrigley *et al.,* 1987a; Schulz *et al.,* 1993; Center, 1996b; Steyn, 2000). It is used less often than operative mesenteric portography because it requires more experience (Lamb and Daniel, 2002).

Splenoportography

Splenoportography is performed by percutaneous (or operative) injection of contrast into the splenic parenchyma. The contrast is then taken up rapidly by the sinusoids into the portal system (Schulz *et al.,* 1993; Center, 1996b; Steyn, 2000). It is a simple and relatively easy technique to perform. In right lateral recumbency, the needle is advanced through the splenic parenchyma and the tip of the needle is positioned as close to the hilus as possible. This results in optimal venous drainage of the contrast medium (Schmidt and Suter, 1980). In right lateral recumbency, the position of the spleen is more consistent, and its inclination to be displaced when introducing the needle is less than in ventrodorsal or dorsoventral positions (Schmidt and Suter, 1980). Schulz *et al*. (1993) were able to position the catheter into a major splenic vein at the hilus (transsplenic portal catheterization).

Complications are splenic laceration and bleeding from the puncture site, and dislodgement of the catheter (Schmidt and Suter, 1980; Schulz *et al.,* 1993; Center, 1996b). The risk of complications decreases with experience, and when a correct technique is followed (Schmidt and Suter, 1980). The number of failures can be reduced by using fluoroscopy (Schmidt and Suter, 1980) or US (Moon, 1990).

The most important disadvantage of this technique is that shunts caudal to the splenic vein will not be recognized, thus resulting in a possible false-negative diagnosis (Schmidt and Suter, 1980; Center, 1996b; Steyn, 2000; Cole *et al.,* 2005). Moreover, the amount of contrast medium needed to opacify the PV within a short time is limited by the draining capacity of the

splenic pulp and the blood flow in the splenic vein (Schmidt and Suter, 1980).

Transvenous retrograde portography (TRP)

As for cranial mesenteric arteriography and percutaneous splenoportography, TRP provides an alternative method of identifying and characterizing PSS's without requiring abdominal surgery (Miller *et al.,* 2002).

The balloon-tipped catheter is inserted through the jugular vein and is directed caudally in the cranial vena cava and then in a dorsal direction into the azygos vein. The catheter is advanced as far as possible until resistance is observed. The balloon is inflated, which occludes the azygos vein. Radiographic contrast medium (1-2 mL/kg) is injected during continuous fluoroscopic evaluation. This results in retrograde filling of the intercostal and intervertebral veins. Via the intervertebral veins, the contrast flows in a retrograde fashion into the prehepatic portion of the CVC (Miller *et al.,* 2002).

Following this injection, the catheter is withdrawn into the cranial vena cava and then advanced caudally through the right atrium into the CVC. The catheter is positioned immediately cranial to the diaphragm, after which the balloon is inflated, occluding the CVC. The second injection, performed immediately after occlusion, results in retrograde filling of the abdominal portion of the CVC and any PSS (Miller *et al.,* 2002). A PSS could be identified and characterized with TRP in 18 of 20 dogs (Miller *et al.,* 2002).

Once the shunt is identified, selective catheterization and injection of contrast medium allows more specific opacification of the shunt, providing more detailed anatomic information. Furthermore, the catheter can be left in place in the lumen of the shunt, thus assisting in identification of the shunt during surgery (Miller *et al.,* 2002).

CONCLUSION

Because of the non-specificity of the clinical signs and laboratory findings, imaging techniques such as portography, ultrasonography, nuclear scintigraphy, computed tomography, and magnetic resonance imaging are required to provide a definitive diagnosis of a PSS.

Due to its high sensitivity in detecting PSSs (85, 91 and 100% in dorsal, right lateral, and left lateral recumbency, respectively) (Scrivani *et al.,* 2001) and its simple interpretation, portography was considered the gold standard in dogs suspected of having a PSS. However, it is time-consuming, invasive and requires radiation.

Nowadays, US can be considered the "new" gold standard for the differentiation of PSSs because it is quick and noninvasive, because it does not require anesthesia or ionizing radiation, and because it has the same sensitivity as portography. This will be further explained in the next part of this review of the literature.

LITERATURE

An extended literature list can be obtained from the authors.

Uit het verleden

DUIVENSCHADE AAN GEBOUWEN

Dendermonde 1366

Schade en hinder in gebouwen door nestelende, rondfladderende en vooral ... schijtende duiven zijn helemaal geen verschijnsel eigen aan onze tijd. Dat bewijst een rekeningenpost uit 1366 voor de (in oorsprong 10de-eeuwse, nu sinds lang verdwenen) feodale burcht van Dendermonde. In dat jaar werden de rijvormig aangebrachte vooruitspringende boogjes (arkaden) onder de dakrand gedicht: *omme de arkette van d' ouder zalen te slutene jeghen de duven, costen te taetsen ende te lukene.*

Uit: de Vlamynck, A. (1897). L'ancien château féodal de Termonde. In: *Annales de la Fédération archéologique et historique de Belgique. Congrès de Gand 1896*, Siffer, Gent, p. 317.