CHAPTER 2

The aim and scope of the thesis
Ultrasonography for diagnostic imaging of abdominal blood vessels

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

Diagnosis of portosystemic shunting with ultrasound
Can it be accurate?

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

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

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

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

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

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

During surgical ligation of a CPSS, the narrowest possible shunt diameter is determined in steps by assessing portal hypertension. To avoid acute fatal portal hypertension, two methods have been recommended for use during surgical shunt ligation, either separately or in combination. Measuring portal pressure by direct catheterization of a portal tributary has been generally used for direct quantitative assessment of portal hypertension. Although this method allows quantitative assessment of portal hypertension, several factors can make the interpretation of pressure changes unreliable. In addition, fatal hemorrhage may occur as a complication of the catheterization. The other method is based on monitoring qualitative signs (color changes of the intestines) and indirect quantitative variables (magnitude of change in mean systemic arterial blood pressure and heart rate) to determine the acceptable degree of post-ligation portal hypertension. Both methods allow acute portal hypertension to be successfully avoided; however, development of chronic portal hypertension remains a frequent complication.

Some years ago, ameroid constrictors were introduced in portosystemic shunt surgery. These devices are placed around the shunt vessels and cause their gradual attenuation, resulting in complete shunt occlusion in one to several weeks. In theory, gradual shunt attenuation would allow the underdeveloped portal branches to become adapted to the increased blood flow. Although using an ameroid constrictor does not require intraoperative assessment of portal hypertension, shunt attenuation becomes an
uncontrollable process with this method. A hypoplastic portal system may not be able to adapt to the increased blood flow at the same rate as the contraction rate of the device would force it to; therefore, subacute or chronic portal hypertension may develop.\textsuperscript{33,35} Application of ameroid constrictors on intrahepatic shunts involves further risks, such as perforation of the shunt because of the increased dissection needed and development of acute portal hypertension because of kinking of the shunt.\textsuperscript{34,35} To reduce these risks, a technically challenging procedure has been recommended, namely placement of an ameroid constrictor on an extrahepatic portocaval venograft after complete ligation of the intrahepatic shunt or resection of the affected liver lobe.\textsuperscript{35,36}

Cellophane banding of extrahepatic CPSSs is another method that has been used to create gradual shunt occlusion. Its application requires initial narrowing of the shunt, so intraoperative assessment of portal hypertension is necessary.\textsuperscript{34,37}

Regardless of the technique used for shunt attenuation and for assessing postligation portal hypertension, the clinical outcome remains unpredictable,\textsuperscript{13,18,23,24} largely because there is no method currently available to reliably evaluate the capacity of the hypoperfused portal branches to accept the increased blood flow that results from shunt attenuation.\textsuperscript{13,18,23,24} Use of histopathologic changes in the liver,\textsuperscript{21,23} portal pressure,\textsuperscript{17,20,25} partial versus complete shunt ligation,\textsuperscript{17,20,26,28,32,39} age of the dogs,\textsuperscript{18,39} and portographic images\textsuperscript{17,20} have not yielded satisfactory results.

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

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

**Postoperative follow-up of attenuated congenital portosystemic shunts**

**Is a second surgery necessary after a partial closure?**

An improved clinical outcome has been related to complete occlusion of a CPSS.\textsuperscript{10,20,26,28,32,40} However, other groups found no difference in outcomes between dogs with partial and complete shunt-ligation.\textsuperscript{17,27,31,39,41} Although the underlying causes of poor outcome in most of the studies remained undetermined, it has been suggested that, if complete shunt-occlusion is not feasible during a surgical attenuation of a CPSS because it would cause the development of a fatal portal hypertension, a second surgery should be performed to attempt a complete shunt-occlusion.\textsuperscript{16,28} The underlying idea is that an initial partial ligation would allow the portal system to adapt to an increased flow and the portal branches would have become gradually wider by the time of the second surgery.

A second surgery should only be considered when portosystemic shunting persists exclusively through the CPSS. When shunting occurs through APSCs, further attenuation of the CPSS is contraindicated. To determine whether post-ligation portosystemic shunting occurs via the CPSS, APSCs or both, mesenteric portography has been used.\textsuperscript{33} In addition to the fact that this angiographic technique involves the use of ionizing radiation, it requires general anesthesia and a laparotomy, both of which are high risk procedures in animals with
hepatic insufficiency.42 Though scintigraphy is suitable to detect portosystemic shunting,17 and with labeled microspheres to quantify it,13 with this technique it is impossible to distinguish between congenital and acquired shunting. Doppler ultrasonography offers a non-invasive way to examine the abdominal blood vessels in unsedated dogs, and the direction and velocity of flow can easily be determined. However, no reports were found in the literature about the ultrasonographic findings of dogs that underwent partial surgical ligation of a CPSS.

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

References


