# Assessment of Intraoperative Mesenteric Portovenography in Congenital Portosystemic Shunt Surgery

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# Abstract

The aim of this study was to assess the hepatic portal vasculature visible on an intraoperative mesenteric portovenogram. The portovenograms of 100 animals were independently assessed by two experienced observers. Two scoring systems were developed, a subjective visual analogue scale and a novel objective scoring system. These two systems were assessed for repeatability, reproducibility and interchangeability. The portovenograms studied consisted of an initial portovenogram, prior to manipulation of the portosystemic shunt, and a second portovenogram following temporary full occlusion of the shunting vessel.

The hepatic portal vasculature was compared between the pre-occlusion and post-occlusion portovenograms. These findings were used to investigate the relationship between portal atresia / hypoplasia and the pre-occlusion portovenograms.

The surgical records of the 100 animals were examined and the portovenograms of those animals which underwent only partial ligation of their portosystemic shunt were compared with those which tolerated full ligation.

There was no statistical difference between the two observers when scoring the same portovenogram for either the visual analogue scale ($P = 0.730$, reproducibility coefficient = 17.85 units) or the objective scoring system (scores identical, reproducibility coefficient = 0). There was no statistical difference, for either of the observers, when the same portovenogram was assessed on two separate occasions using the visual analogue scale (observer 1, $P = 0.35$, repeatability coefficient = 17.93 units; observer 2, $P = 0.42$, repeatability coefficient = 8.27 units) or the objective scoring system (scores given by both observers were identical, repeatability coefficient = 0 for both observers). The results of comparison between the visual analogue scale and objective scoring system

confirmed that the two scoring systems were not directly interchangeable. Although both scoring systems demonstrated good reproducibility and repeatability, the objective scoring system possessed a number of inherent deficiencies that suggested it was not the method of choice for the assessment of the subjective data obtained from intraoperative mesenteric portovenography.

The pre-occlusion scores were significantly different to the post-occlusion scores using both scoring systems ($P<0.01$ for both). Animals with apparent portal atresia / hypoplasia on pre-occlusion portovenography were found to have a wide range of scores on post-occlusion portovenograms, as high as 98 units using the visual analogue scale and 13 on the objective scoring system.

The full ligation group had significantly higher portovenogram scores than the partial ligation group both pre- and post-occlusion, using either scoring system ($P<0.01$ for all tests).

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# List of Abbreviations

| | |
|---|---|
| AC | ameroid constrictor |
| ALP | alkaline phosphatase |
| cm | centimetre |
| CVC | caudal vena cava |
| CVP | central venous pressure |
| HPV | hepatic portal vasculature |
| kg | kilogram |
| mg | milligram |
| ml | millilitre |
| mm | millimetre |
| OSS | objective scoring system |
| PDV | patent ductus venosus |
| PSS | portosystemic shunt |
| PVG | portovenogram |
| T13 | 13$^{th}$ thoracic vertebra |
| UK | United Kingdom |
| US | United States |
| VAS | visual analogue scale |
| VD | ventrodorsal |

# Introduction

This study aims to investigate the use of intraoperative mesenteric portovenography in the diagnosis and surgery of portosystemic shunts (PSS) in the dog and cat. In particular, the appearance of the hepatic portal vasculature (HPV) will be assessed and evaluated, before and after temporary occlusion of the shunting vessel, in order to investigate the incidence of portal vein atresia and hypoplasia. Two methods for defining the degree of HPV were devised. The novel objective scoring system (OSS) of the HPV was compared with a subjective visual analogue scale (VAS) for repeatability, reproducibility and agreement. Comparisons of the groups of animals undergoing full and partial ligation of their PSS were also undertaken in an attempt to consistently and safely identify animals which will tolerate full ligation of their PSS.

# Literature Review

Portosystemic shunts are abnormal vascular communications between the portal circulation, which drains the gastrointestinal tract, and the systemic circulation. They allow blood from the intestines, spleen and pancreas containing nutrients, hormones, toxins, bacteria and oral drugs to bypass the liver which would normally utilise, modify or remove them (Vulgamott, 1985). Portosystemic shunts were first identified in the dog in 1949, as an incidental finding during post mortem examination (Hickman and others, 1949). Subsequently Ewing and others (1974) reported the condition in dogs and these authors attempted to define the different types of PSS in the dog using various angiographic methods and post mortem examinations. No attempts were made to manage the condition in these cases.

## Anatomy of the Liver and Portal Venous System

*Gross Anatomy*

The liver constitutes three to four per cent of the bodyweight in adult animals, while in younger animals it is relatively heavier. It lies in the cranial abdomen apposed to the diaphragm cranially, with the stomach, pancreas, duodenum and right kidney immediately caudal to it. Classically, the liver is divided into four lobes (left, quadrate, right and caudate), four sublobes (left lateral, left medial, right medial and right lateral) and two processes (caudate and papillary processes of the caudate lobe) (Evans, 1993). Figure 1 shows the arrangement of the lobes of the liver in the dog.

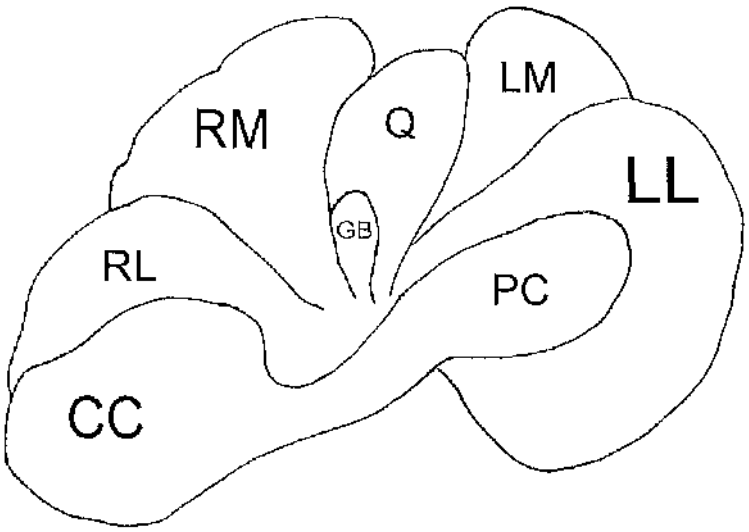


**Figure 1: The lobes of the liver in the dog, view from the visceral surface. *Key:* CC – caudate process of caudate lobe, RL – right lateral lobe, RM – right medial lobe, Q – quadrate lobe, GB – gall bladder, LM – left medial lobe, LL – left lateral lobe, PC – papillary process of caudate lobe.**

Sleight and Thomford (1970) divided the liver into three divisions defined by the hepatic arterial supply. By this classification the right division contains the caudate process of the caudate lobe and the right lateral lobe, the central division contains the right medial and quadrate lobes and the left division contains the left medial and left lateral lobes (Figure 2).

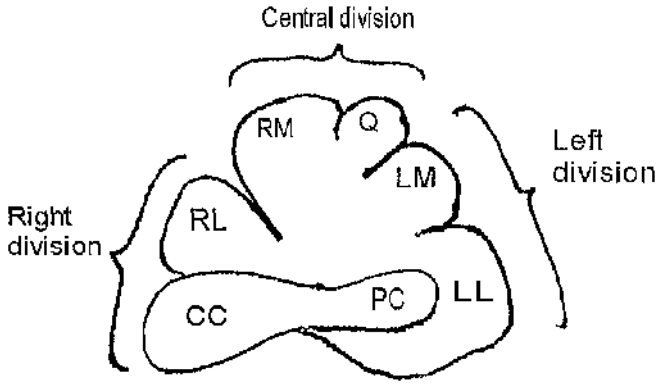


**Figure 2: The divisions of the liver in the dog, view from the visceral surface. *Key:* CC – caudate process of caudate lobe, RL – right lateral lobe, RM – right medial lobe, Q – quadrate lobe, LM – left medial lobe, LL – left lateral lobe, PC – papillary process of caudate lobe.**

The liver is attached to the diaphragm cranially by the coronary ligament, which surrounds the CVC as it passes through the caval hiatus, and also by the right and left triangular ligaments. The falciform ligament also has its origins at the ventral part of the coronary ligament and the diaphragm before passing to the umbilicus. Sleight and Thomford (1970) suggested the main attachment is the left triangular ligament because the right triangular ligament is very small and the falciform ligament between the diaphragm and liver is absent in fifty per cent of dogs.

*Vasculature*

The afferent blood supply to the liver consists of the portal venous system and the hepatic arteries. The portal blood comprises 75-80 per cent of total hepatic blood flow and provides 50 per cent of the oxygen supply, as well as nutrients and hormones which maintain the liver (Payne and others, 1990). The hepatic arterial flow, although only a minor proportion of total flow, is essential and death will ensue if it is occluded without the use of antibiotics (Evans, 1993). The portal vein is relatively constant in location and structure. In the dog, it originates at the conjunction of the cranial and caudal mesenteric veins, at the root of the mesentery, before receiving tributaries from the splenic vein and gastroduodenal vein (Kalt and Stump, 1993). At the porta of the liver, the portal vein provides a branch (right main branch) which further ramifies before entering the right lateral lobe and the caudate process of the caudate lobe. The main portal trunk continues to the left providing branches to the right medial lobe, papillary process of the caudate lobe, quadrate lobe, left medial lobe, ending in the left lateral lobe (Figure 3) (Sleight and Thomford, 1970; Kalt and Stump, 1993). In the cat, the portal vein receives tributaries from the cranial mesenteric and caudal mesenteric, splenic, gastroduodenal and cranial pancreaticoduodenal veins. At the porta of the liver it trifurcates, sending one branch to the caudate lobe, one to the right lateral and right

medial lobes and the final branch supplies the left division (Perry and Lowrie, 1993).

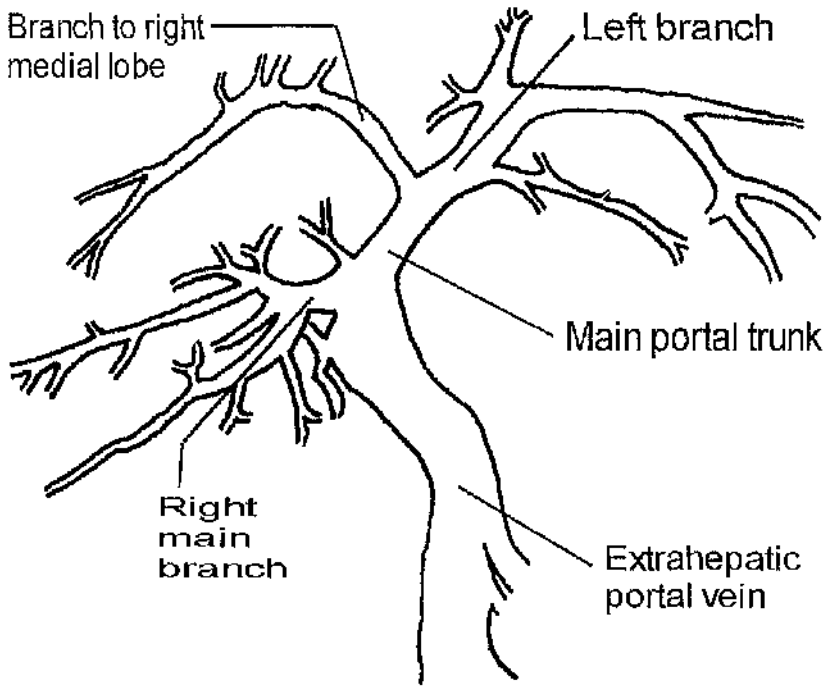


**Figure 3: Anatomy of the intrahepatic portal vein in the dog, ventrodorsal view.**

The hepatic arteries originate from the celiac artery and show considerable variation in their branching pattern. The hepatic artery descends towards the porta of the liver where it forms an arch usually passing ventral to the portal vein. The number of branches arising from the arch varies considerably, from one trunk, which then splits to provide branches to each lobe, up to five separate branches. Two or three branches are the most common configurations (Sleight and Thomford, 1970; Schmidt and others, 1980).

There are normally six to eight hepatic veins of significant size and numerous other tiny tributaries. The most consistent vessel is the left hepatic vein which drains the left divisional hepatic lobes and is also described as draining the quadrate and right medial lobes in some dogs (Evans, 1993). It enters the vena cava on the ventral surface at the left side and is the most cranial of the hepatic veins (Swalec Tobias and

Rawlings, 1996). The remaining hepatic veins, further caudal, are of an inconsistent position and drainage (Sleight and Thomford, 1970).

In the prenatal and neonatal animal at least fifty per cent of oxygenated placental blood is allowed to bypass the hepatic sinusoids by way of the ductus venosus. The umbilical vein terminates in the left branch of the portal vein. The ductus venosus is a straight vessel of uniform diameter which passes from the left branch of the portal vein (opposite the umbilical vein) between the left lateral lobe and papillary process of the caudate lobe into a venous dilatation (ampulla) at the confluence of the ductus venosus, left hepatic vein and left phrenic vein. This ampulla then drains into the vena cava. The ductus venosus is normally functionally closed by three days post partum and anatomically closed by six days (Burton and White, 1999).

**Embryology of the Abdominal Veins**

*Normal Development*

Three main embryological venous systems, the cardinal, vitelline and umbilical systems, form all the major abdominal veins. The formation of these veins by degeneration, anastomosis and persistence of embryological vessels is driven by the pattern of the flow of blood itself and so any minor changes in the development of the embryo may cause major changes in the eventual form of the vessels.

The portal vein is derived from the vitelline veins, which are originally paired (left and right) veins with three anastomoses connecting the two. It is formed from the caudal portion of the left vitelline vein, the middle anastomosis and the cranial portion of the right vitelline vein. The vitelline veins also form the hepatic veins and the hepatic and posthepatic caudal vena cava (CVC). These are normally separated from the portal vein by the developing hepatic sinusoids (Payne and others, 1990; Hunt and others, 1998a). The intrahepatic portal venous system is formed

predominantly from the vitelline veins. The left branch of the intrahepatic portal vein, between its termination in the left lobe and the ductus venosus, develops from the umbilical-portal sinus which is of both vitelline and umbilical origin (Payne and others, 1990).

There are initially two umbilical veins but the right umbilical vein degenerates. The cranial portion of the left umbilical vein forms the ductus venosus which connects the portal sinus and the left hepatic vein until after birth. Cranial to the liver both veins degenerate meaning all umbilical blood flow to the heart passes via the ductus venosus (Payne and others, 1990).

The cardinal system consists of three pairs of veins, the caudal cardinal, the supracardinal and the subcardinal veins. The supracardinal and subcardinal veins develop in association with the mesonephros and both connect to the caudal cardinal veins cranially. When the mesonephros degenerates and the metanephros develops the vascular segments degenerate and anastomose to form the adult veins. The prerenal (caudal to the kidneys) CVC is formed from the right supracardinal vein, the renal segment from the anastomosis of both right supracardinal and subcardinal veins and the prehepatic CVC (between the kidneys and the liver) develops from the right subcardinal vein. Where the prehepatic (cardinal) and hepatic (vitelline) segments of the CVC join is the only normal connection between the vitelline and cardinal venous systems caudal to the liver. The precursor of the azygos vein is the right caudal cardinal vein, this is connected to the supracardinal vein early in development but the intervening segment degenerates to ensure separation of the azygos vein and CVC.

**Classification of Portosystemic Shunts**

Portosystemic shunts have been classified in several different ways: acquired or congenital, multiple or single and intrahepatic or extrahepatic. Congenital PSS are present at birth, usually single or double, and can be

either intrahepatic or extrahepatic. Acquired PSS are usually not present at birth because they develop secondarily to conditions leading to raised portal pressure. They have been associated with hepatic parenchymal disease, hepatic arteriovenous fistulae and surgical manipulation of congenital PSS (Van den Ingh and others, 1995). They are narrow, tortuous vessels commonly found connecting the two venous systems close to the left kidney and are found in large numbers. Multiple congenital PSS have been described (Hunt and others, 1998b) but are uncommon. The majority of congenital PSS are single vessels although double congenital PSS do exist and comprised 11 per cent of PSS in one report (Johnson and others, 1987). Meyer and others (1999) described two dogs in which a second shunting vessel was demonstrated. These dogs had continued evidence of shunting despite ligation of a PSS previously.

Although the differenzbe tween intrahepatic and extrahepatic PSS appears to be simply whether the shunting vessel is within the liver parenchyma or not, intrahepatic PSS are more correctly defined as those which originate from the portal vein branches after their bifurcation (trifurcation in cats) at the porta of the liver (Hunt and others, 2000). This definition will then include those PSS which may not actually pass through the liver parenchyma itself. Intrahepatic PSS are usually classified by the division of the liver through which they pass (Swalec Tobias and Rawlings, 1996; White and others, 1998; Lamb and White, 1998). Left divisional PSS follow a pattern consistent with continued patency of the ductus venosus (White and Burton, 2000). Several authors have described all intrahepatic PSS as PDVs (Rothuizen and others, 1982; Martin and Payne, 1990; Payne and others, 1990) but as there appears to be no embryological basis for this structure in the right or central divisions, it is likely that PSS passing through these divisions are, in fact, anomalous vessels or bizarre sinusoidal malformations (Burton and White, 1999). Central divisional PSS in dogs, often take the form of a window-like connection between the dilated portal branch and hepatic

vein or vena cava. Central divisional PSS in cats and right divisional PSS in dogs and cats most often take the form of a tortuous vessel forming a loop entirely within one of the lobes (Lamb and White, 1998).

Extrahepatic PSS are always abnormal vessels, which can arise from anywhere in the portal circulation. They are often large tortuous vessels and commonly originate from the portal, left gastric, splenic, cranial mesenteric, caudal mesenteric, colonic, umbilical or gastroduodenal veins (Payne and others, 1990). They usually insert directly into a systemic vein, most commonly the CVC (portocaval) or the azygos vein (portoazygos) (Johnson and others, 1987). They can, however, drain into more obscure vessels such as the internal thoracic, renal or colonic veins (Berger and others, 1986). The portal vein cranial to the PSS may appear hypoplastic or may be completely absent as in cases of portal atresia (Hunt and others, 1998a). Portocaval shunts can often be located at the epiploic foramen whereas portoazygos shunts can be found by opening the omental bursa and observing any vessel crossing the diaphragm (Swalec Tobias and others, 1998).

Two further types of hepatic vascular anomalies have been described. Hepatic microvascular dysplasia is characterised by presentation with clinical signs consistent with portosystemic shunting but with no evidence of a macroscopic PSS, angiographically or at surgery. Diagnosis is by histopathology with the portal veins and hepatic veins connected at a microvascular level (Phillips and others, 1996; Schermerhorn and others, 1996; Christiansen and others, 2000). Hepatic arteriovenous (or arterioportal) fistulae are direct connections between the hepatic arteries and portal (or hepatic) vein, usually within a single lobe and can be congenital or acquired (Bailey and others, 1988). This produces dilated vessels within the parenchyma of this lobe, hepatofugal portal flow and portal hypertension usually leading to the development of multiple acquired PSS. Clinical signs are consistent with portal hypertension, such as ascites, and treatment is by removal of the affected lobe.

**Portal Atresia and Portal Hypoplasia**

Portal atresia is the total absence of a portal vein between the PSS and the liver (Hunt and others, 1998a). This is an uncommon finding with published incidences of 6.8 per cent (Hunt and others, 1998a) and 7.4 per cent (Center and Magne, 1990) of dogs with PSS. The very first reported finding of PSS also had portal atresia (Hickman and others, 1949). Hunt and others (1998a) described five dogs with portal atresia, in four of these the portal vein inserted directly into the CVC and in the other case it joined the left hepatic vein. If the PSS were closed in these animals then fatal portal hypertension would invariably result, this means that no surgical correction of the problem is possible. Hunt and others (1996) also identified a case with an intrahepatic PSS which had intrahepatic portal atresia, this was euthanased.

Portal hypoplasia describes portal vessels which are narrower or less well developed than normal. Primary portal hypoplasia has been reported in 42 dogs in which it was thought to be the cause of portal hypertension and multiple acquired PSS (Van den Ingh and others, 1995). It is more commonly found secondary to reduced portal blood flow, usually as a consequence of a PSS. It affects the portal vein proximal to the PSS including the HPV. Secondary portal hypoplasia was a finding in several case series of animals with PSS (Ewing and others, 1974; Gofton, 1978; Rothuizen and others, 1982). This was either observed at surgery or portovenography demonstrated little or no HPV. Breznock (1979) also found portal hypoplasia but described it as portal atresia despite some portal flow being present through the vessels. In this same study improved visualisation of the HPV was demonstrated angiographically several weeks after surgical attenuation or closure.

## Embryology of Portosystemic Shunts and Vascular Anomalies

Patent ductus venosus (PDV) is due to the failure of closure of the normal embryological structure the ductus venosus. Multiple acquired PSS are normal microscopic portosystemic communications which open in response to portal hypertension (increased blood pressure in the portal system). All other types of PSS are abnormal embryological connections. Other types of intrahepatic PSS (right and central divisional) may be due to failure of the hepatic sinusoids to separate the portal and caval portions of the vitelline vein, as normally occurs. They may be a remnant of the embryonic vitelline vein or a sinusoidal malformation (White and others, 1998). All extrahepatic PSS are abnormal connections between the vitelline and cardinal venous systems (Payne and others, 1990). This may be due to changes in embryological blood flow encouraging abnormal anastomoses between the vitelline and cardinal systems. Hawe and Mullen (1984) postulated that a delay in formation of the anastomosis between the left and right vitelline veins may have encouraged anastomosis with the cardinal veins to allow adequate venous flow, thus creating the PSS.

True portal atresia commonly involves the insertion of the portal trunk (vitelline system) into the prehepatic CVC, derived from the right subcardinal vein. The right subcardinal vein anastomoses with the hepatic CVC (vitelline origin) normally, so this vessel may have a special affinity for anastomosis with the vitelline system (Hunt and others, 1998a). For the animal to suffer from portal atresia, degeneration or aplasia of the vitelline system between the shunting vessel and the hepatic veins must also occur. Some reports of portal atresia describe insertion of the portal trunk into the left hepatic vein, this is a normal vitelline to vitelline anastomosis but the abnormality may be due to persistence of the left rather than right vitelline vein and failure of closure of the ductus venosus (Hunt and others, 1998a).

## Diagnosis

*Signalment*

Animals with congenital PSS usually present at a young age, although some do not show clinical signs until later in life (Center and Magne, 1990). Acquired PSS, although often presenting in the older animal, have also been diagnosed in very young animals (Rand and others, 1988). Most case series' reported no significant sex predilection for PSS (Johnson and others, 1987; Center and Magne, 1990). Some have suggested that large breed dogs are likely to have intrahepatic PSS and small breed dogs extrahepatic PSS (Bostwick and Twedt, 1995; Lamb, 1996). There are, however, exceptions to this rule (Center and Magne, 1990; White and others, 1998; Hunt and others, 2000). Some breeds are reported as having a higher incidence of intrahepatic PSS in the UK including golden and Labrador retrievers (central, right and left divisional PSS), Irish wolfhounds (PDV) and Old English sheepdogs (central divisional PSS) (White and others, 1998). In the US, Yorkshire terriers and miniature schnauzers have a high incidence of extrahepatic PSS (Center and Magne, 1990). In Australia, the Australian Cattle dog has a high incidence of right sided intrahepatic PSS and Maltese terriers are also over-represented with extrahepatic PSS (Tisdall and others, 1994). A survey of 160 dogs in the Netherlands revealed the commonest small breeds were Yorkshire, Cairn and Maltese terriers and large breeds were golden retrievers, Old English sheepdogs, Bernese mountain dogs and Irish wolfhounds (Wolschrijn and others, 2000). Irish wolfhounds in the Netherlands are the only breed to have had an inheritance of the disease demonstrated (Meyer and others, 1995). In cats, PSS are commonly found in domestic short hairs, Persians and Siamese. Extrahepatic PSS arising from the left gastric vein and intrahepatic PSS are common, but other types have been reported as well as variation between the breeds (Levesque and others, 1982; Berger and others, 1986; Scavelli and others, 1986; VanGundy and others, 1990).

*Clinical Signs*

Animals with PSS predominantly show signs associated with the nervous, gastrointestinal and urinary systems. They are usually episodic in nature and associated with the ingestion of protein-rich food, gastrointestinal bleeding, and periods of constipation or dehydration (Center and Magne, 1990; Watson, 1997). The list of reported clinical signs is large and varied. Neurological signs are predominantly due to hepatic encephalopathy and include depression, bizarre behaviour, hyperexcitability, apparent hallucinations, amaurotic blindness, ptyalism, ataxia, weakness, stupor, head pressing, staring, repetitive pacing and circling, aggression, grand mal seizures and coma (Vulgamott, 1985; Center and Magne, 1990; Watson, 1997). The pathophysiology of hepatic encephalopathy is not as yet fully understood. Current theories which have been suggested are ammonia acting as a neurotoxin with or without other synergistic toxins, alteration of monoamine neurotransmitters due to a change in aromatic amino acid metabolism, alteration in amino acid neurotransmitters, γ-aminobutyric acid and glutamate and increased cerebral levels of endogenous benzodiazepine-like substances (Maddison, 1992). Previous theories such as false neurotransmitters, decreased cerebral energy levels, lack of a brain protective factor and changes in the blood brain barrier are not currently favoured (Maddison, 1992). Gastrointestinal signs include intermittent anorexia, polyphagia, pica, vomiting, diarrhoea and constipation (Vulgamott, 1985; Maddison, 1992). Urinary tract signs include polyuria and polydipsia, which is postulated to be caused by either a low urea level resulting in a decreased medullary concentration gradient, psychogenic polydipsia, or derangement of hepatic metabolism of renin and adrenal steroids (Vulgamott, 1985; Watson, 1997). Haematuria and pollakiuria are caused by ammonium biurate uroliths present due to the impaired metabolism of ammonia and urea by the liver. Other commonly seen signs are failure to grow as well as littermates and intolerance to certain drugs, especially anaesthetics and tranquillisers. Rarer signs include weight loss, intermittent pyrexia, recurrent apparent upper respiratory tract problems

in the cat, intense pruritus in the dog, and jaundice and ascites in cases of acquired PSS (Center and Magne, 1990).

*Physical Examination*

Clinical examination of animals with PSS is often unremarkable. However, small stature, renomegaly, difficulty palpating a liver margin and bladder calculi may be detected. Affected cats commonly have a characteristic copper coloured iris (Center and Magne, 1990). Many animals are first presented with neurological deficits such as central blindness or are in the post-ictal phase of a seizure.

*Clinical Pathology*

The results of routine haematology, biochemistry and urinalysis do not definitively diagnose a PSS but may raise the index of suspicion for the clinician. Haematological abnormalities consist of mild anaemia and erythrocyte microcytosis. Postulated explanations for this include alteration in iron metabolism and altered erythrocyte production or survival. Poikilocytosis has been reported to occur commonly in cats and also in some dogs (Center and Magne, 1990). Biochemical changes are more varied. Alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase (ALP) may all be mildly elevated, although in younger animals the raised ALP may be extrahepatic in origin (bone). Blood urea and glucose levels are often low.

Two dynamic biochemical tests are available to diagnose PSS, both are highly specific. The first, the ammonia tolerance test involves measuring fasting blood ammonia level then giving ammonium chloride either by stomach tube (100 mg/kg at 20 mg/ml or less) or into the rectum (2 ml/kg of 5 per cent solution). Further samples are then taken after 30 minutes for the oral test, or 20 and 40 minutes for the rectal method (Leveille-Webster, 2000). Dynamic serum bile acid measurement requires a fasted blood sample and a further sample two hours after feeding a meal

(Leveille-Webster, 2000). In both tests the results are usually significantly raised for both samples in PSS. In the normal animal, ammonia is almost completely removed from the portal circulation by the liver. When blood bypasses the liver, ammonia remains at higher concentrations. Bile acids are released from the gall bladder when a meal is ingested and are subsequently reabsorbed from the GI tract and returned to the liver, via the portal vein, to be recycled. In cases of PSS, they remain in the systemic circulation for longer and in higher concentrations. Both ammonia and bile acid concentrations are sensitive indicators of portosystemic shunting but the latter is often preferred because bile acids are stable in blood for longer periods (Center, 1990). There are documented deficiencies of the ammonia tolerance test, it may precipitate an encephalopathic crisis itself and oral administration of ammonium chloride can cause vomiting, invalidating the test (Center, 1990).

Urinalysis reveals a variable specific gravity (hypersthenuria, isosthenuria or hyposthenuria). Ammonium biurate crystalluria or urolithiasis is also commonly found in animals with PSS (Center and Magne, 1990).

*Radiography*

Plain abdominal radiography may reveal microhepatica, renomegaly and loss of detail in the abdomen due to lack of intra-abdominal fat. Urate uroliths are radiolucent and so are not easily visible on plain films. Definitive diagnosis of a PSS relies on accurate imaging of the portal venous system to demonstrate the anomaly. Angiography is described as the definitive means for diagnosing, locating and determining the extent of a PSS (Moon, 1990). The functions of portovenography are to demonstrate the presence of a PSS, to document whether it is intrahepatic or extrahepatic, to determine its location (the affected vessels for extrahepatic PSS or the hepatic division for intrahepatic PSS), to determine its morphology (especially important for intrahepatic PSS) and to assess the status of the HPV. Birchard and others (1989) found, using portovenography, that if the caudal extent of a shunting vessel was

cranial to the vertebra of the 13th thoracic vertebra (T13) it was likely to be intrahepatic and if it was caudal to T13 it was likely to be extrahepatic. Angiography may also be used to monitor cases after surgery. It can be used to assess the patency of the shunting vessel, assess any improvement in the HPV and diagnose any acquired PSS which may have been formed (Martin and Payne, 1990). As this is an invasive procedure, however, it is usually only performed in animals showing clinical signs or with abnormal biochemistry or scintigraphy results, often in conjunction with repeat surgery.

In humans, many different angiographic procedures have been developed to investigate the different vascular systems of the liver. The hepatic circulation has a major effect on liver regeneration, metabolism and histology, thus angiography is of great value in assessing patients with hepatic fibrosis, cirrhosis, chronic active hepatitis, neoplasia, portal hypertension and PSS. Several indications for angiography in humans have now been superseded by the use of ultrasonography (Schmidt and Suter, 1980). The choice of techniques available for humans is very wide encompassing techniques which highlight arteries, hepatic veins and the portal vein either directly or indirectly (Table 1). Only a small number of these techniques have gained widespread use in veterinary medicine.

**Table 1: Human hepatic angiography methods (Veterinary options in bold) (Schmidt and Suter, 1980)**

| Arteriography | Indirect Portography | Direct Portography | Venography | Panangiography |
|---|---|---|---|---|
| **Selective celiac arteriography** | **Arterial portography** | **Operative mesenteric portography** | Free hepatic vein catheterisation | Percutaneous kinetic hepatography |
| **Superselective hepatic arteriography** | **Splenoportography** | Percutaneous transhepatic portography | Wedge hepatic vein catheterisation | |
| | Umbilical vein portography | Transjugular transhepatic portography | | |

The indications for hepatic angiography in animals include portal hypertension with ascites, hepatic encephalopathy and PSS (Schmidt and Suter, 1980).

Arteriography in cats and dogs is most useful in the diagnosis of arterioportal or arteriovenous fistulae. Superselective catheterisation of the hepatic artery gives a better quality image, reducing superimposition of other vessels. Arterial portography, most commonly cranial mesenteric arterial portography, may be used in the demonstration of PSS. Both arteriography techniques are minimally invasive, using the femoral artery to enter the aorta. The only minor complication is haematoma formation at the site of puncture and this can be minimised by cutting down onto the vessel. Special catheters and fluoroscopy or a cut film changer are required. It can be quite technically demanding to successfully place the catheter tip, especially if superselective catheterisation is attempted. Image quality is satisfactory but compared to direct portovenography there is superimposition of the arteries over the portal venous system and the density of the contrast in the portal system is diminished due to dilution as it passes through the capillaries (Suter, 1975; Schmidt and Suter, 1980).

Splenoportography involves placing a catheter into the splenic pulp before injecting contrast medium, this can be achieved either percutaneously or at laparotomy. The percutaneous method is unsuitable for cats and can be difficult in some dogs, as it can lead to puncture of other abdominal structures and haemorrhage from the puncture sites. These problems can be avoided by using ultrasound guidance or by placing the catheter at full or mini-laparotomy. The advantages of the technique are that it is minimally invasive when performed percutaneously, highlights most PSS with the exception of mesenteric to systemic PSS, and is very useful for assessing the direction of portal blood flow. Compared to other techniques, filling of the hepatic portal vein branches can be poor, due to the slow rate of drainage of contrast from

the splenic parenchyma. Contrast density is not as good as the direct methods and superimposition of the spleen can occur in lateral views (Schmidt and Suter, 1980).

Umbilical vein portovenography requires cannulation of the umbilical vein and as such is restricted to use in neonatal animals, usually as a research tool (Suter, 1975). Image quality is good but the flow of contrast is non-physiological (Burton and White, 1999).

Operative mesenteric portovenography is performed by catheterising a mesenteric vein at laparotomy. This method provides excellent detail of the portal vein in a physiological manner (Schmidt and Suter, 1980). The disadvantages of this technique are the requirements for laparotomy and sacrifice of a mesenteric vein. This technique is preferable to splenoportography if a laparotomy is to be performed and is often combined with surgery.

Techniques used in human patients which have not gained widespread use in veterinary medicine include percutaneous transhepatic portovenography, where the portal vein at the hilus of the liver is directly catheterised, transjugular transhepatic portography, in which the catheter is passed through the jugular vein, right atrium, hepatic vein and parenchyma into a portal vein, hepatic vein catheterisation, where a hepatic vein is catheterised through the jugular and percutaneous kinetic hepatography where a long needle is placed deep in the parenchyma and slowly withdrawn while injecting contrast. These have all met with sufficient drawbacks to minimise their use in animals (Schmidt and Suter, 1980).

The most commonly used veterinary techniques for PSS visualisation have been reviewed by Moon (1990). These include operative jejunal portovenography, splenoportography, cranial mesenteric arterial portography and superselective hepatic arterial catheterisation. The most

commonly performed techniques are splenoportography and operative mesenteric portovenography as these require the least equipment and expertise. In recent years, newer imaging technologies have been introduced to improve the detection of PSS. Subtraction portovenography improves visualisation of the shunting vessel which is undoubtedly useful for inexperienced observers but the extra time or expensive equipment mean that it is not an essential technique (Wrigley and others, 1987; Swalec Tobias and others, 1996). Seguin and others (1999) utilised magnetic resonance angiography (MRA) for the diagnosis of PSS. This technique uses the MRI scanner to create contrast in the portal vein without the need to introduce foreign agents into the animal. It has enormous potential as it is minimally invasive, obviating the need for contrast materials which may produce side effects. However, general anaesthesia is still necessary, further experimentation is required to produce the best results and in most cases it will be prohibitively expensive for some time to come.

*Ultrasonography*

The use of abdominal ultrasonography in the diagnosis of PSS is becoming more widespread. It has the advantage of being a non-invasive technique which in the hands of experienced operators provides a tremendous amount of information regarding the type, location and morphology of the shunting vessel (Lamb, 1998). Knowledge of the morphology of intrahepatic PSS before surgery is helpful in selecting the correct procedure and allows advanced preparations to be made. One study of 63 cases (Holt and others, 1995) correlated ultrasound findings with surgical, portovenographic and post mortem findings and found a sensitivity for detection of extrahepatic PSS of 81 per cent and a specificity of 66.7 per cent. For detection of intrahepatic PSS the sensitivity was 100 per cent. Colour flow Doppler and Doppler measurement techniques can greatly ease the identification of PSS but also require an experienced operator (Lamb, 1996).

*Nuclear Scintigraphy*

The degree of portosystemic shunting of blood can be quantified using portal scintigraphy. Technetium$^{99}$ labelled molecules are introduced into the portal system, either transcolonically or percutaneously into the splenic vein, and a gamma camera is used to monitor the change in radioactivity in the liver and heart. In animals with PSS the rise in activity in the heart precedes the liver, the opposite of normal animals. The degree of shunting can be estimated by comparing the rise in activity in the two organs. Portal scintigraphy, however, cannot determine the type of PSS, this requires a further diagnostic procedure (Moon, 1990; Van Vechten and others, 1994; Forster-van Hijfte and others, 1996). This technique has been used to monitor progressive changes in shunting after partial ligation of PSS (Van Vechten and others, 1994).

*Surgical Visualisation*

Experienced surgeons will often be able to locate PSS without the need for portovenography (Martin and Freeman, 1987; Bellenger and others, 1995). Many extrahepatic PSS occupy relatively consistent anatomical locations within the abdomen (Swalec Tobias and others, 1998). Intrahepatic PSS not immediately visible can be located by palpating a soft spot in the parenchyma of the affected lobe, observing indentations which move in time with breathing, examining the portal or hepatic vein for dilatation and locating fluid thrills over the PSS (Breznock and others, 1983; Swalec Tobias and Rawlings, 1996). Occluding the various portal vein branches while monitoring changes in haemodynamic measurements (portal pressure, central venous pressure (CVP)) may also indicate which portal vein branch supplies the PSS (Breznock and others, 1983).

**Treatment**

Treatment of PSS can be by medical management to alleviate the clinical signs or by surgical narrowing or closure of the shunting vessel.

*Medical Treatment*

Medical treatment is often implemented to stabilise an animal for surgery, in conditions where surgery is contraindicated (multiple acquired PSS) and where surgery is declined. The aim of the treatment is to control the clinical signs of hepatic encephalopathy (Taboada and Dimski, 1995). There are three main areas of medical therapy, dietary change, oral antibiotics and oral lactulose (Watson, 1997). A reduced protein diet is instituted to reduce blood levels of ammonia and aromatic amino acids and the post-prandial peaks. Oral antibiotics, such as ampicillin, alter the bacterial flora of the intestines to reduce bacterial ammonia production. Lactulose reduces ammonia levels by decreasing production and absorption, trapping ammonia in the colon and altering bacterial flora (Johnson, 2000).

*Surgical Treatment*

It is generally agreed that surgery is the best treatment option for PSS. Surgical ligation of a PSS can recreate the normal portal system and thus allow the liver to regenerate, obviating the need for continued medical therapy.

Attenuation of extrahepatic PSS is usually much more straightforward than intrahepatic PSS. The vessels are usually easily visible and accessible and require little dissection to allow ligature placement. Intrahepatic PSS may require considerable dissection through the liver parenchyma and can involve intravascular surgery, total hepatic vascular occlusion and thoracotomy.

The aim of surgical treatment is to narrow or, if possible, completely close the PSS. In the majority of cases the shunting vessel cannot be completely closed. This is because the HPV is unable to accommodate the increased blood flow and portal hypertension will result. If severe, this

will progress to hypovolaemic shock and will be fatal unless the ligature is removed. Milder portal hypertension will lead to formation of multiple acquired PSS and continuing signs of hepatic encephalopathy. To overcome these problems several methods of vessel attenuation have been proposed.

For the majority of the last twenty years silk has been the most commonly used ligation material. To reduce the incidence of complications, guidelines have been published on physical and haemodynamic parameters which should avoid excessive portal hypertension. Mathews and Gofton (1988) reported the visual signs of portal hypertension as splanchnic visceral pallor or cyanosis (particularly the pancreas), intestinal hypermotility, increased jejunal artery pulsation and congestion of splanchnic veins. Many authors have provided recommendations of maximum post ligation portal pressures, and increases in portal pressure, which should avoid portal hypertension (Breznock and others, 1983; Birchard, 1984; Martin and Freeman, 1987; Butler and others, 1990; Bostwick and Twedt, 1995). Accepted recommendations are that the portal pressure should not exceed 20 cm of water or the increase in portal pressure should not be greater than 10 cm of water. White and others (1998) discussed the unreliability of this method of judging attenuation and recommended visual assessment of viscera and assessment of the systemic arterial blood pressure and CVP (Swalec and Smeak, 1990).

Some partially ligated PSS will, with time, close completely without further manipulation (White and others, 1998; Meyer and others, 1999). Others may be fully ligated at a second surgery, while a final group will not tolerate any further narrowing (White and others, 1998). Silk is thought to provoke an acute inflammatory reaction within the vessel wall which lasts approximately seven days, followed by a fibroblastic reaction until day fifteen. This is proposed to produce further closure by scar formation and contracture (Van Vechten and others, 1994). However, in an experimental study (Youmans and Hunt, 1999), it was demonstrated that

a silk ligature did not cause progressive attenuation of a femoral vein. This may be due to different conditions between the leg and the abdomen, or different blood flow patterns.

In an attempt to produce slow, progressive attenuation after a single procedure, two further materials have been used, the ameroid constrictor (AC) and cellophane.

The AC is a ring of casein, a hygroscopic clay, which expands when exposed to tissue fluid. It is contained in a metal ring to direct the expansion to close the central hole, gradually occluding the vessel in the middle. The exact length of time to complete closure of the vessel has not been reliably documented, as such, controversy remains over whether attenuation can occur too rapidly. The AC has been shown to produce progressive attenuation of PSS. Youmans and Hunt (1999) found the AC completely occluded the femoral vein in two weeks, whereas Vogt and others (1996) reported that splenic veins became occluded by four to five weeks and half of extrahepatic PSS by 30 days. The central hole is not completely obliterated, so it is possible that this relies on thrombus formation to completely close the vessel. The reported mortality rate of 14 per cent and the rate of formation of acquired PSS, also 14 per cent (Vogt and others, 1996), using the AC compare less favourably with the 2.1 per cent reported for extrahepatic PSS ligated with silk (Hunt and Hughes, 1999). Possible reasons for this include too rapid closure of the vessel leading to fatal hypertension or acquired PSS, or kinking of the vessel by the ring causing immediate total occlusion and fatal portal hypertension.

Cellophane banding involves wrapping a strip of cellophane around the vessel and securing this with a titanium clip. This was first discussed by Breznock in 1979, and has been shown to produce progressive attenuation over four to six weeks (Youmans and Hunt, 1999), causing a chronic foreign body inflammatory reaction and subsequent contracture and thrombus formation.

A final method of attenuation reported is the implantation of thrombogenic coils into the PSS. This has been described for the closure of a PDV. It involves passing a long catheter through the jugular vein into the ductus venosus under fluoroscopic guidance. The coils are then positioned in the shunt through the catheter, usually two coils per procedure. In this report (Partington and others, 1993) four separate procedures were required to finally close the PSS, but the authors thought that with better coil size selection and greater experience it could be performed in just two procedures. The coil caused progressive closure due to thrombus formation. This procedure is less invasive than other surgical methods but because it is difficult to measure portal pressure and impossible to visualise the splanchnic viscera, there is a risk of portal hypertension.

*Surgical procedures*

The techniques for extrahepatic PSS attenuation and their common locations are outlined by Swalec Tobias and others (1998). The surgical procedures are relatively standard, with only the method of PSS attenuation varying between surgeons. Hunt and Hughes (1999) reported on the outcome of 49 dogs ligated with silk, finding a mortality rate of 2.1 per cent and that outcome after partial ligation was related to surgeon experience.

Intrahepatic PSS can vary in location, morphology and amount of parenchymal coverage. This has meant a variety of different procedures have been described, categorised as extravascular or intravascular techniques. Extravascular techniques involve either ligation of the portal vein branch leading into the PSS, the vessel itself, or the hepatic vein which drains the PSS. Breznock and others (1983) described the techniques of portal vein branch ligation which can be used for PSS of any division, and the technique of placing a ligature around the vessel as it entered the hepatic vein cranial to the liver. The portal branch ligation technique may require some parenchymal dissection, which carries a risk

of haemorrhage and makes this method far from ideal. The second technique is usually restricted to left divisional PSS, which drain into the left hepatic vein, the only easily accessible vein cranial to the liver. This technique is quick and relatively free of complications and is the technique of choice even today for left divisional PSS (White and others, 1998). Wrigley and others (1983) used intraoperative ultrasound to visualise the PSS before ligating it without parenchymal dissection. Martin and others (1986) reported the technique of ligating the left hepatic vein or ampulla instead of the shunting vessel. This may sometimes be more easily accessible but has deleterious, albeit temporary and not life threatening, effects on the lobes drained by this vein (Payne and others, 1991). One of the major risks of extravascular techniques is haemorrhage from the parenchyma or torn vessels. To overcome this Swalec Tobias and others (1996) outlined the use of an ultrasonic aspirator, which selectively destroys parenchyma while leaving blood vessels and bile ducts intact.

The ideal technique for PSS attenuation would seem to be to directly close the shunting vessel, rather than the portal or hepatic vein, and to reduce tissue dissection to an absolute minimum. It was with these goals in mind that the intravascular techniques were developed.

Rawlings and Wilson (1983) and Breznock and others (1983) described two similar intracaval techniques for closure of intrahepatic PSS. Both techniques utilised a longitudinal venotomy in the posthepatic vena cava to allow visualisation of the PSS opening before partially closing it with polypropylene sutures. In the second method, an additional suture with the ends outside the lumen of the vena cava allowed further vessel closure after the venotomy was repaired. These procedures are technically demanding and time consuming, but considerably reduce the risks of haemorrhage.

A similar technique of PSS attenuation via portal venotomy has been reported (Hunt and others, 1996). Total hepatic vascular occlusion was achieved and a portal venotomy made longitudinally in the dilated part of the portal vein branch supplying the PSS. A single mattress suture was passed, at right angles to the venotomy incision, through the lumen of the PSS. The suture was passed through Teflon-felt pledgets at each end and the ends left untied. The venotomy was closed with a continuous suture before the ligature was tightened. The final degree of ligation was decided with reference to portal pressure, CVP, arterial blood pressure and the appearance of the splanchnic viscera. This technique has also been used in two dogs with multiple congenital intrahepatic PSS which had a single opening into the portal vein (Hunt and others, 1998b).

White and others (1998) reported on a series of 45 dogs with intrahepatic PSS operated on using several of the above techniques and found a mortality rate of 18 per cent. These procedures were originally described for dogs but several have been used in cats (White and others, 1996b)

Due to the difficulty of partially ligating intrahepatic PSS and the possibility of rupturing the vessel (requiring total ligation), two methods of surgically creating an extrahepatic shunting vessel have been described, allowing easier control of attenuation. White and others (1996a) used an autologous jugular vein graft as an extrahepatic portocaval shunt in two dogs undergoing central divisional PSS ligation. This allowed total closure of the window-like PSS. Poy and others (1998) described a technique of creating a splenocaval shunt by end-to-side anastomosis of the splenic vein to the CVC. This technique can be used as an emergency procedure if a vessel ruptures which must be totally ligated to stop haemorrhage. Both of these procedures may require a second operation to close the surgically created PSS.

*Complications*

Several reports of large numbers of cases have discussed complications and their rate of occurrence (Johnson and others, 1987; Mathews and Gofton, 1988; Komtebedde and others, 1991; White and others, 1998; Hunt and Hughes, 1999; Wolschrijn and others, 2000). Intraoperative complications consist of anaesthesia related problems (2 of 33), arterial hypotension (8 of 13), hypothermia (5 of 33), acute hepatic congestion (6 of 13), haemorrhage (3 of 13 and 1 of 33) and vessel rupture during intrahepatic PSS dissection (3 of 45). After recovery from anaesthesia the most common problems are portal hypertension, neurological problems, hyperthermia and abdominal haemorrhage. Portal hypertension manifests itself in a number of ways depending on the severity. Mild signs are abdominal distension, self-limiting ascites which progress through abdominal pain to cardiovascular collapse and arrest or, in severe cases, sudden death. Most cases of portal hypertension are thought to be due to excessive attenuation of the shunt vessel although many animals appear to have had portal pressures within the recommended limits at surgery. Another possible cause is portal vein thrombosis and several reports of this condition exist. Roy and others (1992) reported two (of sixteen) cases, one intrahepatic and the other extrahepatic, both of which had been fully ligated and subsequently died acutely. Mathews and Gofton (1988) outlined a similar case which underwent full ligation and died acutely after 48 hours. All these cases underwent post mortem examination which revealed the thrombus. It is possible that other cases of portal hypertension which do not undergo further investigation may be caused by this condition. The incidence of portal hypertension may decrease with greater experience of assessing PSS attenuation intraoperatively, and this seems to be borne out in the decreased incidence in some of the larger case series (White and others, 1998; Hunt and Hughes, 1999; Wolschrijn and others, 2000). Neurological problems separate from any encountered preoperatively are quite a common complication and, if they progress to uncontrollable seizures, can be fatal. Hunt and Hughes (1999) found neurological signs as their most frequent

complication describing them as 'postligation neurological dysfunction'. Tisdall and others (2000) investigated this further and found no evidence of hypoglycaemia or hyperammonaemia to explain the syndrome. They found phenobarbitone administration appeared to reduce the progression of mild neurological dysfunction to status epilepticus.

*Anaesthesia*

Animals with PSS can be difficult to safely anaesthetise, due to their impaired liver function. With careful consideration of agents and diligent monitoring the risks can be greatly reduced. The liver is used to metabolise many anaesthetic drugs so preference should be given to those drugs which undergo minimal metabolism by the liver or those which can be easily reversed. The main agents which fall into these categories are isoflurane, opioid analgesics and propofol (Raffe, 1992). Monitoring of arterial blood pressure, CVP and end tidal carbon dioxide levels are very useful in detecting signs of cardiovascular instability before, during and after PSS attenuation. Blood glucose concentration should be monitored frequently because small dogs especially have difficulty regulating this and both hypoglycaemia and hyperglycaemia can be harmful to the patient (Butler and others, 1990). Core temperature should be monitored and maintained as close to normal as possible, as anaesthesia impairs normal thermoregulation.

**Methods of assessment of the HPV**

The HPV is extremely difficult, if not impossible, to objectively measure in a live animal. Possible options might include three dimensional computed tomography or magnetic resonance imaging to measure the volume of the vessels. It might be possible to quantify its volume post mortem but this would be unlikely to be reliable. It was for this reason that a subjective assessment was chosen. Several methods of measuring subjective criteria such as pain, pain relief and lameness have been described in the medical and veterinary literature. Human pain

assessment is the most common subject for which they have been utilised. The methods range from simple descriptive scales using four or five verbal descriptions (none, mild, moderate, severe, very severe), through numerical rating scales giving values from 0 to 10 or 0 to 20, to visual analogue scales or graphical rating scales (Downie and others, 1978). The VAS usually utilises a 100 mm line with perpendicular lines at each end, a verbal description of each end-point is placed at these extremes and the observer marks a point on the line corresponding to their assessment of the subject. The graphical rating scale is a VAS with further descriptive terms placed along the line. Visual analogue scales have been utilised with success in assessment of pain in humans and domestic animals (Reid and Nolan, 1991) and in lameness assessment of domestic animals (Welsh and others, 1993). It is considered to be an accurate and reproducible method for the assessment of a subjective criterion such as pain. The technique is considered as reliable as and more sensitive than a simple descriptive scale (Joyce and others, 1975).

## Hypotheses

This study aims to examine the following hypotheses:

The portal venous system demonstrated on a post-occlusion PVG is significantly different from the portal venous system shown on a pre-occlusion PVG.

Hypoplasia / atresia of the portal venous system can only be diagnosed using a post-occlusion PVG.

The novel OSS will give comparable results to the subjective VAS.

# Materials and Methods

## Case Details

The surgical records and videotaped angiographic studies of 100 consecutive dogs and cats treated surgically for the attenuation of a PSS at the Queen Mother Hospital for Animals, Royal Veterinary College and Davies White Veterinary Specialists between 1997 and 2000 were reviewed.

The surgical records were examined for details of the species, breed, age at surgery, sex, type of PSS and degree of ligation (partial or full).

The video records consisted of a ventrodorsal (VD) PVG used to identify the location of the PSS and a second PVG obtained after temporary full occlusion of the shunting vessel. The HPV was assessed by two different methods - a subjective VAS and a novel OSS.

## Scoring Systems

VAS scores were produced by making a mark on a 100 mm scale corresponding to the degree of HPV visualised. Factors taken into account when using the VAS are the degree of branching of the HPV, the rate at which the HPV fills with contrast, the width of the vessels, the rate at which contrast cleared and the degree and rate of parenchymal opacification. The end points of the scale were defined as 0 mm, showing no evidence of a portal vein entering the liver and 100 mm showing normal HPV. A well developed HPV, such as that shown in Figure 4, will show rapid filling of a well branched, relatively wide vascular tree with good opacification of the parenchyma, which is then rapidly cleared by the liver. The marks were then measured and recorded as the number of millimetres from the zero end-point. The VAS data were recorded to the nearest 1.0 mm.

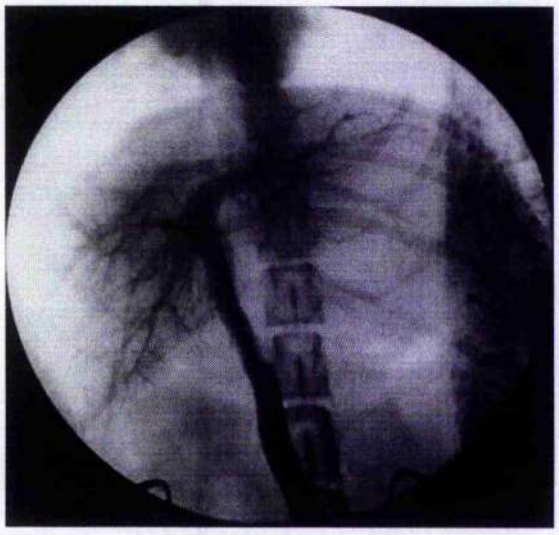

**Figure 4: Ventrodorsal intraoperative mesenteric portovenogram demonstrating a well developed hepatic portal vasculature**

The OSS consisted of thirteen questions with a 'yes' or 'no' answer devised to assess the development of the HPV (Table 2). A point was scored for each 'yes' answer giving a possible total of 13 points. When directly comparing the OSS with the VAS these results were converted to a figure out of 100.

**Table 2: Objective scoring system questions. *Key:* Yes=1 No=0**

| | |
|---|---|
| Is there a portal vein entering the liver? | Yes/No |
| Can we see the right principal branch? | Yes/No |
| Can we see the left principal branch? | Yes/No |
| Can we see branching to the right medial lobe? | Yes/No |
| Can we see branching to the left lobes? | Yes/No |
| In the right branches (right medial and lateral) – | |
| Can we see primary arborisation? | Yes/No |
| Can we see secondary arborisation? | Yes/No |
| Can we see tertiary arborisation? | Yes/No |
| In the left branches – | |
| Can we see primary arborisation? | Yes/No |
| Can we see secondary arborisation? | Yes/No |
| Can we see tertiary arborisation? | Yes/No |
| Is there opacification to the right side of the liver? | Yes/No |
| Is there opacification to the left side of the liver? | Yes/No |

## Exclusions

Animals were excluded from the study if both pre-occlusion and post-occlusion PVGs were not present and of diagnostic quality, or if the surgical records were incomplete.

## Observer Trials

To assess inter-observer variability, 40 PVGs were randomly selected and scored by both observers using both scoring systems. Both observers were experienced in the assessment of PVGs. Neither observer was aware of the score assigned by the other, and the VAS and OSS scores were recorded separately.

A further 20 PVGs were selected at random and scored as described above, on two separate occasions, one hour apart, to investigate the between-observer and within-observer variability of both scoring systems.

## Investigative Method

Each animal underwent a full clinical examination and blood samples were collected for routine haematological and biochemical evaluation. Diagnosis was initially based on signalment, history, physical examination, pre- and post-prandial serum bile acid concentrations, abdominal ultrasonography and, in some animals, portal scintigraphy. Intraoperative portovenography was performed immediately prior to, and immediately following, temporary PSS occlusion.

### *Portovenography*

The portovenography technique was consistent for each individual. Animals were premedicated with 0.02 mg/kg acepromazine maleate (ACP; C-Vet Veterinary Products) and 1 mg/kg pethidine hydrochloride (Pethidine injection; Martindale Pharmaceuticals) both given by intramuscular injection. Anaesthesia was induced by mask with isoflurane

(Isoflo vet; Schering-Plough Animal Health) vaporised in an oxygen and nitrous oxide carrier gas. Once unconscious the animal was intubated and maintained using the same agents. Perioperative antibiosis was provided with ampicillin (Penbritin Veterinary Injectable; SmithKline Beecham Animal Health) 20 mg/kg administered intravenously and an arterial catheter (Jelco; Critikon) was placed percutaneously into the dorsal metatarsal artery allowing arterial blood pressure to be monitored directly. A central venous catheter was placed into the external jugular vein allowing CVP to be monitored. Intravenous fluids (0.18 per cent saline with 4 per cent glucose) were administered at a sufficient rate to maintain central venous and arterial blood pressures within normal limits.

A cranial midline celiotomy was performed and a loop of jejunum was exteriorised allowing a jejunal vein to be catheterised using a 20 or 22 gauge over-the-needle cannula (Jelco; Critikon). The mesenteric venous pressure was estimated by saline manometry before mesenteric portovenography was performed using a mobile C-arm fluoroscope with image intensification (Phillips BV22; Phillips Medical Systems) to obtain VD and in some cases lateral images of the cranial abdomen. The fluoroscope was connected to a video recorder for image storage. Iohexol (Omnipaque 350; Nycomed) was injected as a 3-15 ml bolus into the catheterised mesenteric vein ensuring that the total dose of iodine did not exceed 600 mg/kg.

The PSS was identified and a ligature of 2-0 polypropylene (Prolene; Ethicon) was passed around the vessel allowing the vessel to be temporarily occluded with a Rummel tourniquet. The mesenteric venous pressure was again estimated and a second PVG was obtained. Central venous, arterial and portal pressure measurements, effects on splanchnic viscera and the results of portovenography were all assessed to determine the degree of attenuation the animal could tolerate. The PSS was then ligated with two or three 0 silk (Mersilk; Ethicon) ligatures. In some animals a polypropylene ligature would also be placed to allow

further attenuation of the PSS if a subsequent surgery was required. A liver biopsy was obtained and the celiotomy incision was repaired routinely.

**Data Collection**

The 200 PVGs obtained from the 100 animals were assessed by one observer. For each animal, two images were reviewed, the first prior to any manipulation of the shunting vessel and the second following its temporary, complete occlusion. Each PVG was assessed using both the OSS and VAS.

**Statistical Analyses**

The assessment of the PVGs provided a score before and after PSS occlusion for the OSS and the VAS. The results of these PVG scores were analysed using Minitab (Minitab Ltd) and Statistix (Analytical Software).

All data obtained using both the VAS and the OSS was assessed for Normal distribution.

The statistical significance of the differences between observers when assessing the same PVG using the VAS was determined by the use of the paired Student's *t*-test. Differences within the same observer when scoring the same PVG on two occasions were also analysed using the paired Student's *t*-test. When comparing these results for the OSS the Wilcoxon signed rank test was used.

The repeatability of the two scoring systems was quantified using the methods of Bland and Altman (1999). A repeatability coefficient was calculated for both the VAS and OSS using the formula: $1.96 * \sqrt{2} * SD$. The repeatability coefficient is the value within which two scores of the same PVG will lie for 95 per cent of those observations when scored

using the same scoring system by the same observer. These coefficients were used to assess whether any lack of agreement between the methods was explained by poor repeatability.

The data for both observers in the between- and within-observer trials were combined to allow assessment of reproducibility. Graphical techniques were used to plot the difference between the measurements obtained by each observer against their mean values. A reproducibility coefficient was calculated, using the formula: $1.96 * \sqrt{2} * SD$, for both the VAS and the OSS. The reproducibility coefficient is the value within which two scores of the same PVG will lie for 95 per cent of those observations when scored by the two observers using the same scoring system. These were used to quantify the reproducibility of the scoring systems.

The two scoring systems were compared using the method comparison techniques described by Bland and Altman (1999) to assess bias, 95 per cent limits of agreement and interchangeability. Graphical techniques were used to plot the difference between measurements by the two scoring methods for each PVG against their mean value. The 95 per cent limits of agreement were produced using the formula: mean $\pm$ 1.96*SD. Acceptable limits of agreement were defined in advance as 15.4 units in either direction. This is equivalent to two questions in the OSS.

The statistical significance of the differences between the pre- and post-occlusion scores for both scoring systems was assessed using the Wilcoxon signed rank test.

Animals which had a PVG score of 10 units or less using the VAS or 1 or less using the OSS were described as having portal hypoplasia or atresia. The post-occlusion PVGs of animals with apparently hypoplastic HPV on their pre-occlusion PVG were examined to assess the usefulness of the pre-occlusion PVG.

The Mann-Whitney U test was used to assess the statistical significance of the differences between animals undergoing full ligation of their PSS and those tolerating only partial ligation. This test was performed for both scoring systems, before and after temporary PSS occlusion.

The 95 per cent confidence intervals of the median score were calculated for the groups undergoing partial and full ligation in an attempt to produce ranges within which full ligation will be safe. These ranges were compared with the minimum scores of successful full ligations.

In all analyses statistical significance was set at $P<0.05$.

# Results

## Case Details

One hundred animals (80 dogs and 20 cats) met the inclusion criteria. The most common breeds of dog were the Irish wolfhound, the Yorkshire terrier and the West Highland white terrier. The most common cat breeds were the domestic short hair and the Persian. The breed distributions of the dogs and cats are shown in Tables 3 and 4.

**Table 3: Breed distribution of dogs studied (only breeds represented more than once included)**

| Breed | Number |
|---|---|
| Irish wolfhound | 8 |
| Yorkshire terrier | 8 |
| West Highland white terrier | 7 |
| Labrador retriever | 6 |
| Shih tzu | 6 |
| Golden retriever | 5 |
| Norfolk terrier | 5 |
| Border collie | 4 |
| Cairn terrier | 4 |
| Irish setter | 4 |
| Maltese terrier | 3 |
| Bichon frise | 2 |
| Jack Russell terrier | 2 |
| Miniature schnauzer | 2 |
| Other breeds | 14 |
| Total | 80 |

**Table 4: Breed distribution of cats studied**

| Breed | Number |
|---|---|
| Domestic short hair | 6 |
| Persian | 4 |
| British short hair | 3 |
| Siamese | 3 |
| Domestic long hair | 1 |
| Havana | 1 |
| Rag doll | 1 |
| Tonkinese | 1 |
| Total | 20 |

The mean age at the time of surgery was 15.9 months (range 2 to 108 months, SD 20.4) for dogs, 14.8 months (range 4 to 48 months, SD 13.0) for cats with a combined mean of 15.7 months (range 2 to 108 months, SD 19.1). Figures 5 and 6 show the distribution of the ages of dogs and cats. The majority of dogs studied were under 12 months of age and the majority of cats less than 24 months old.

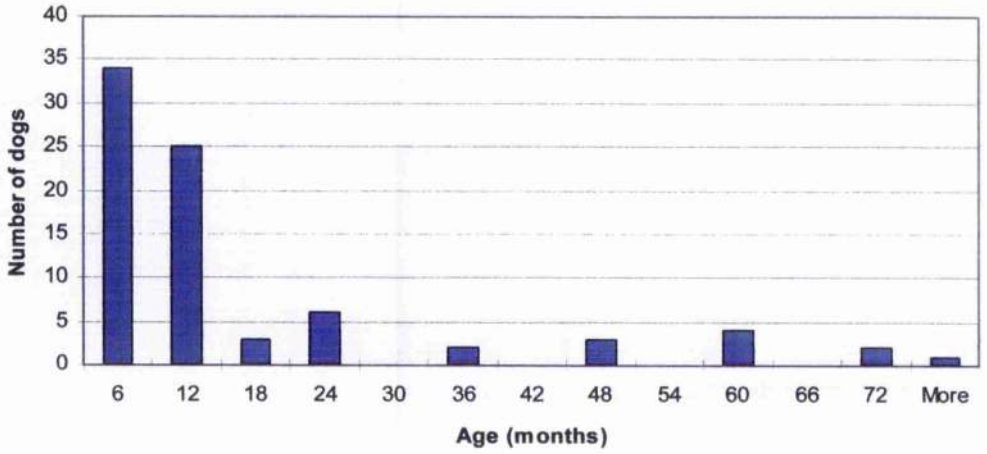


**Figure 5: Distribution of age at surgery for dogs studied**

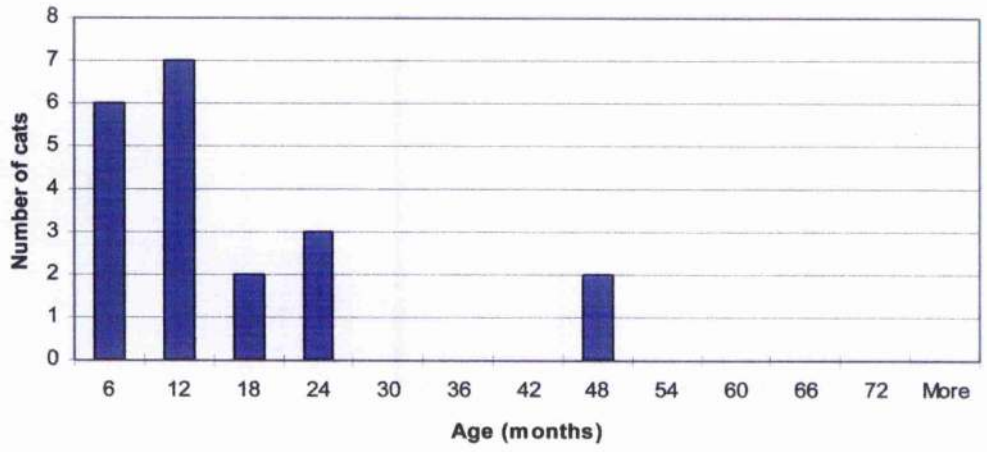


**Figure 6: Distribution of age at surgery for cats studied**

The overall ratio of sex distribution for dogs and cats was 1:1 (Table 5).

**Table 5: Sex distribution of dogs and cats studied**

| Sex | Number of dogs | Number of cats | Total |
|---|---|---|---|
| Male | 38 | 12 | 50 |
| Female | 42 | 8 | 50 |
| Total | 80 | 20 | 100 |

Of the PSS recognised in the dog, 64 per cent were extrahepatic, 34 per cent were intrahepatic and two PSS involved the umbilical vein. Of these, the commonest extrahepatic PSS was the portocaval (80 per cent) and the commonest intrahepatic PSS was the PDV (85 per cent). This data is presented in Table 6.

**Table 6: Type of portosystemic shunt identified in dogs**

| Shunt Type | Number |
|---|---|
| Extrahepatic | 51 |
| Portocaval | 41 |
| Portoazygos | 5 |
| Left gastric vein | 4 |
| Colonic vein | 1 |
| Intrahepatic | 27 |
| Patent ductus venosus | 23 |
| Right divisional | 2 |
| Central divisional | 1 |
| Left divisional | 1 |
| Umbilical vein (intra/extrahepatic) | 2 |
| Total | 80 |

In cats, an even greater proportion of PSS identified were extrahepatic (85 per cent). Of these, the left gastric to caudal vena cava was most frequently observed (65 per cent) (Table 7).

Table 7: Type of portosystemic shunt identified in cats

| Shunt Type | Number |
|---|---|
| Extrahepatic | 17 |
| Left gastric vein | 11 |
| Portocaval | 6 |
| Intrahepatic | 3 |
| Patent ductus venosus | 3 |
| Total | 20 |

With regards to the degree of attenuation achieved at surgery, in the majority of cases only partial PSS attenuation was possible (80 per cent of dogs and 70 per cent of cats) (Table 8).

Table 8: Degree of ligation of portosystemic shunt in dogs and cats

| Degree of ligation | Number of dogs | Number of cats | Total |
|---|---|---|---|
| Full ligation | 16 | 6 | 22 |
| Partial ligation | 64 | 14 | 78 |
| Total | 80 | 20 | 100 |

## Inter-observer differences (reproducibility)

Normal probability plots for the VAS scores obtained for the 80 portovenograms from the two trials indicated that the scores for both observers were not Normally distributed. A Normal probability plot for the differences between the VAS scores of the same portovenogram by the two observers confirmed this data to be Normally distributed.

Using the VAS, the mean difference between the two observers was 0.36 units (range -15 to 18 units, SD 6.44). Statistical analysis showed the difference between the two observers when scoring the same PVG using the VAS was not statistically significant (P=0.62).

The reproducibility coefficient for the VAS was 17.85 units.

The mean score of the VAS for both observers when scoring the 80 portovenograms was plotted against the difference in scores for each observer (Figure 7). This data demonstrated no error due to magnitude, that is, the difference between the scores did not increase with an increase in the mean score. Figure 7 also showed that there was greater agreement between the two observers at the extremities of the VAS.

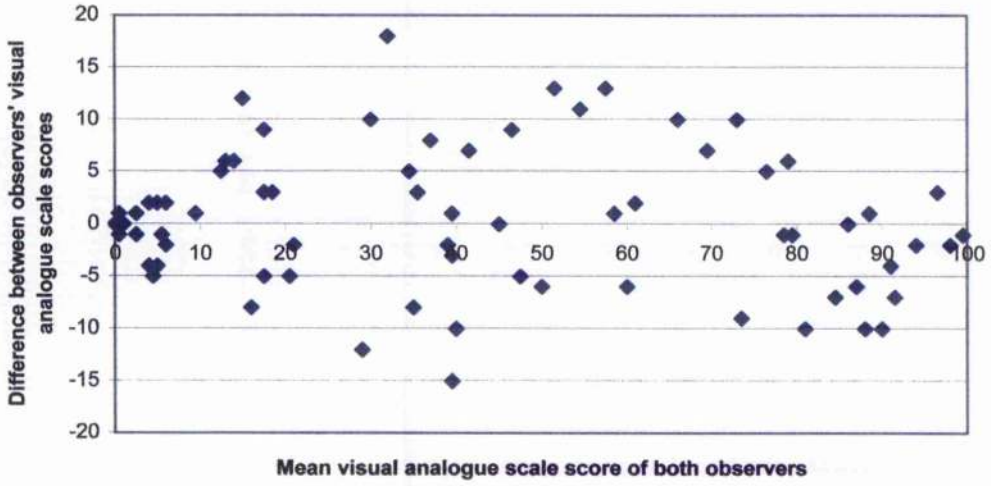


**Figure 7: Graph of the difference between observers' visual analogue scale scores plotted against their mean score**

Using the OSS, the scores of the two observers assessing the same PVG were identical. There was, therefore, no statistical difference between the two observers when scoring the same portovenogram using the OSS and the reproducibility coefficient for this OSS data was zero.

### Within-observer differences (repeatability)

Normal probability plots for the VAS scores obtained for the 20 portovenograms that were scored on two separate occasions indicated that the scores for both observers were not Normally distributed. A Normal probability plot for the differences between the VAS scores of the

same portovenogram by the same observer confirmed these data to be Normally distributed.

The mean difference between the scores recorded at two separate instances was -1.4 units (range -14 to 11 units, SD 6.47) for observer 1 and -0.55 units (range -5 to 4 units, SD 2.98) for observer 2. Statistical analysis showed no significant difference for either observer (observer 1, P=0.35 and observer 2, P=0.42) between the scores of the same portovenogram on two separate occasions.

Repeatability coefficients were 17.9 units (observer 1) and 8.3 units (observer 2).

When using the OSS, the scores for the same portovenograms on two separate occasions were identical, therefore no statistical difference was found and the repeatability coefficients were both zero.

**Comparison of scoring systems**

Following conversion of the OSS scores to a value out of 100, the mean difference between the two scoring systems was -11.0 units (range –55 to 22 units, SD 12.1).

The mean score of the two systems was plotted against the difference between the scores (Figure 8). This showed no error due to magnitude, that is, the difference did not increase as the mean score increased.

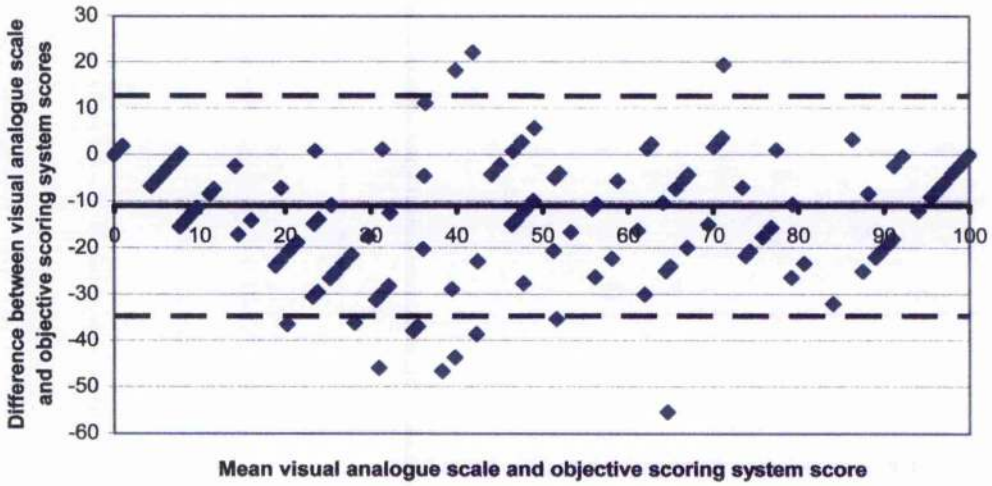


**Figure 8: Graph of difference between visual analogue scale and objective scoring system scores plotted against their mean score for 200 portovenograms. *Key:* Solid line indicates mean difference in scores. Dashed lines indicate 95 per cent limits of agreement of the two scoring systems**

The 95 per cent limits of agreement of the two scoring systems were calculated to be 12.7 and -34.8 units.

### Assessment of HPV on pre-occlusion and post-occlusion PVGs

Normal distribution of both pre-occlusion and post-occlusion data using the VAS was not demonstrated (Figure 9). The median score for the pre-occlusion VAS was 4.5 units (range 0 to 88 units) and for the post-occlusion VAS it was much higher at 53 units (range 2 to 100 units). Statistical analysis of these VAS data showed that pre-occlusion and post-occlusion scores were significantly different ($P<0.01$).

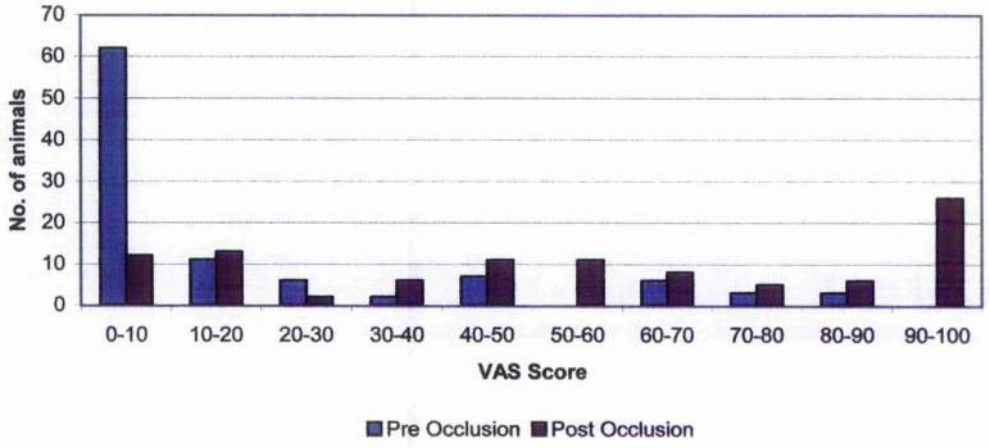


**Figure 9: Distribution of pre-occlusion and post-occlusion VAS scores**

Normal distribution of both pre-occlusion and post-occlusion data using the OSS was not demonstrated (Figure 10). The median score for the pre-occlusion OSS was 2 units (range 0 to 13 units) and for the post-occlusion OSS was also higher at 9 units (range 2 to 13 units). Statistical analysis of these OSS data showed that pre-occlusion and post-occlusion scores were significantly different ($P<0.01$).

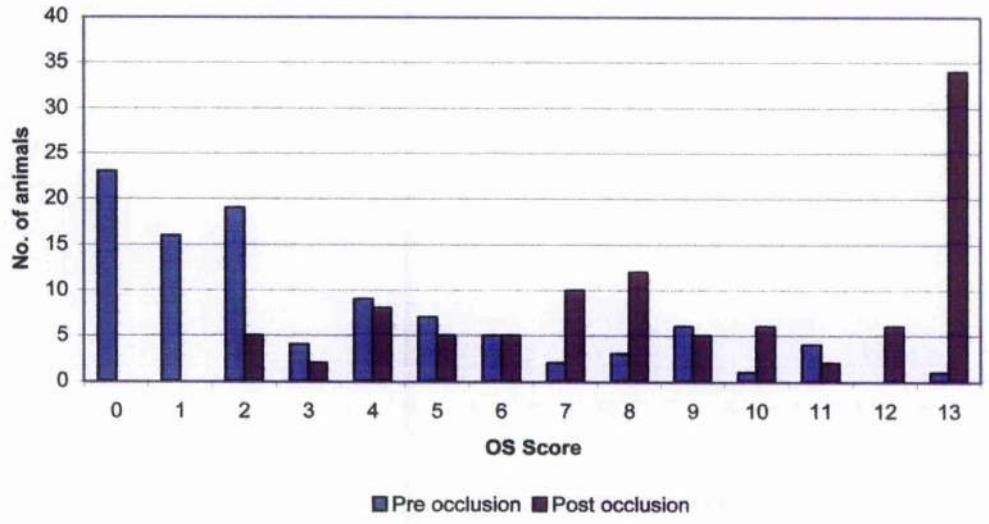


**Figure 10: Distribution of pre-occlusion and post-occlusion OS scores**

When using the VAS, 62 animals had HPV scores of 10 or less and were thus designated as showing evidence of portal atresia or hypoplasia.

Interestingly, for these individuals their median post-occlusion score was much higher at 41 units (range 2 to 98 units). This post-occlusion VAS data is presented in Figure 11.

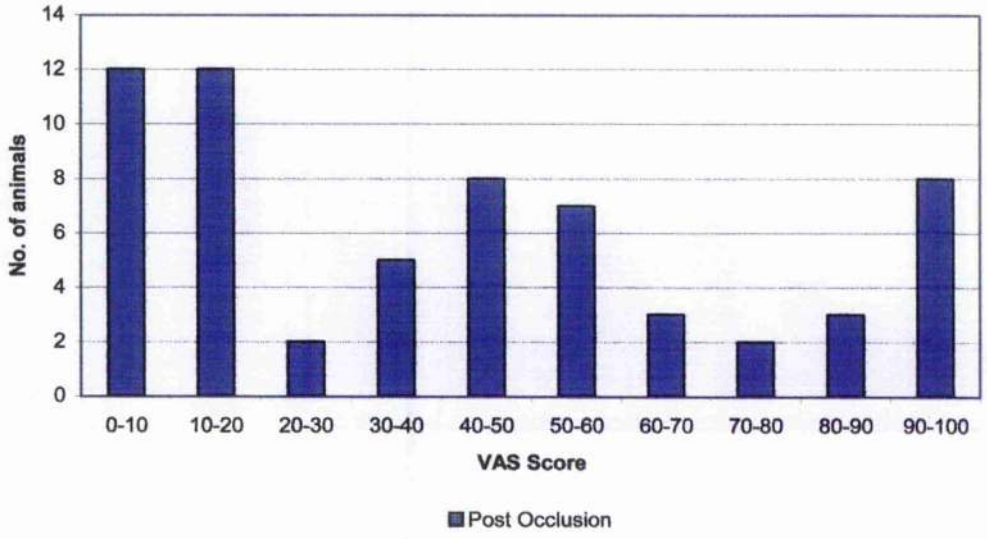


**Figure 11: Distribution of post-occlusion VAS scores of animals with apparently hypoplastic pre-occlusion HPV (n=62)**

When using the OSS, 39 animals had OSS scores of 0 or 1 and were also designated as having portal atresia or hypoplasia. Similarly, for these individuals their median post-occlusion score was much higher at 8 units (range 2 to 13 units). This post-occlusion OSS data is presented in Figure 12.

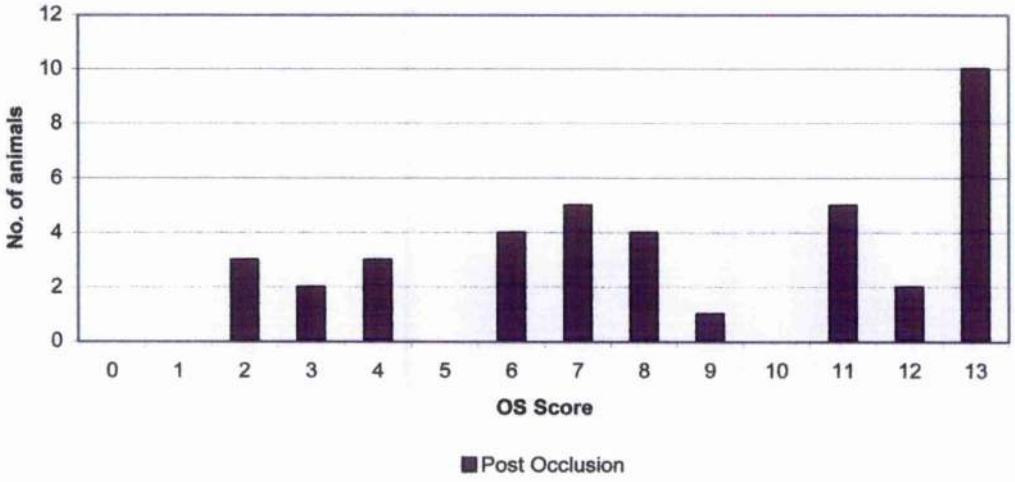


**Figure 12: Distribution of post-occlusion OS scores of animals with apparently hypoplastic pre-occlusion HPV (n=39)**

Figure 13 and 14 show pre- and post-occlusion PVGs of the same animal. This demonstrates an extrahepatic PSS with minimal apparent HPV development on the pre-occlusion study and a significantly improved, although still poor, HPV after PSS occlusion.

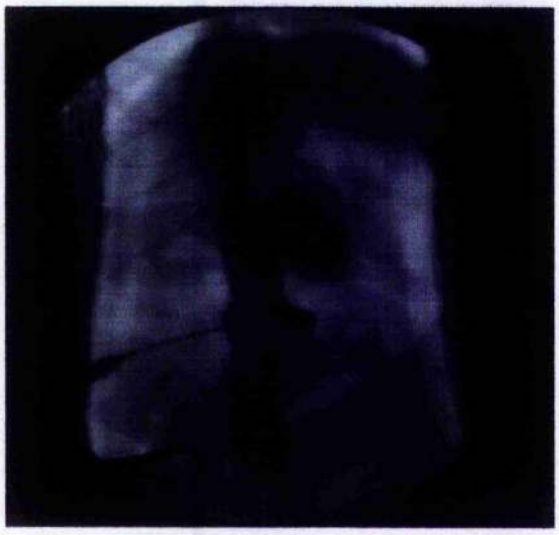

**Figure 13: Ventrodorsal intraoperative mesenteric portovenogram before temporary occlusion of the shunting vessel**

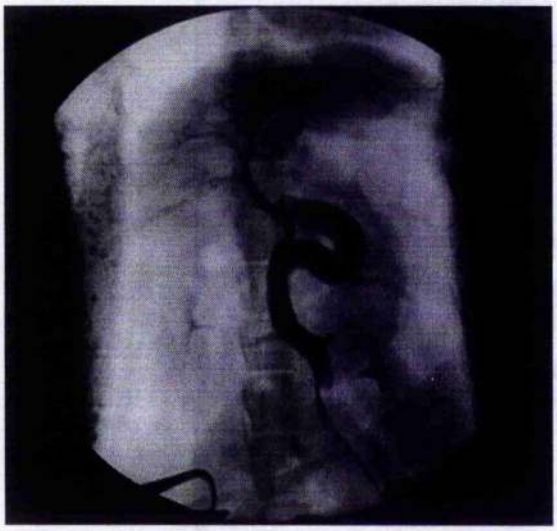

**Figure 14: Ventrodorsal intraoperative portovenogram of the same animal as Figure 13, after occlusion of the portosystemic shunt**

## Comparison of HPV between animals with full and partial ligation

Normal probability plots of the pre- and post-occlusion scores of animals with full or partial ligation of their PSS, using either scoring system did not demonstrate a Normal distribution for any data sets.

A greater number of animals with partial ligation of their PSS had VAS scores at the lower end of this scale, whereas for animals with full ligations the distribution was more evenly distributed. For clarity this data is presented as the number of animals in each group, as a percentage, against their respective pre-occlusion VAS score, in Figure 15.

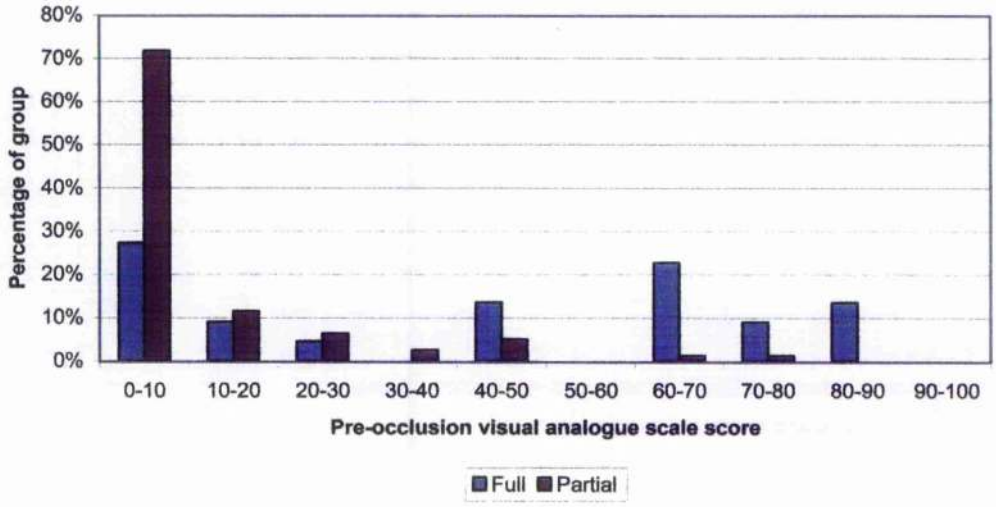


**Figure 15: Distribution of pre-occlusion visual analogue scale scores of animals undergoing partial and full ligation of their portosystemic shunt**

Conversely, similar data for post-occlusion VAS scores shows an even distribution of partial ligation scores with the full ligation data distributed at the higher end of the scale. This information is similarly presented in Figure 16.

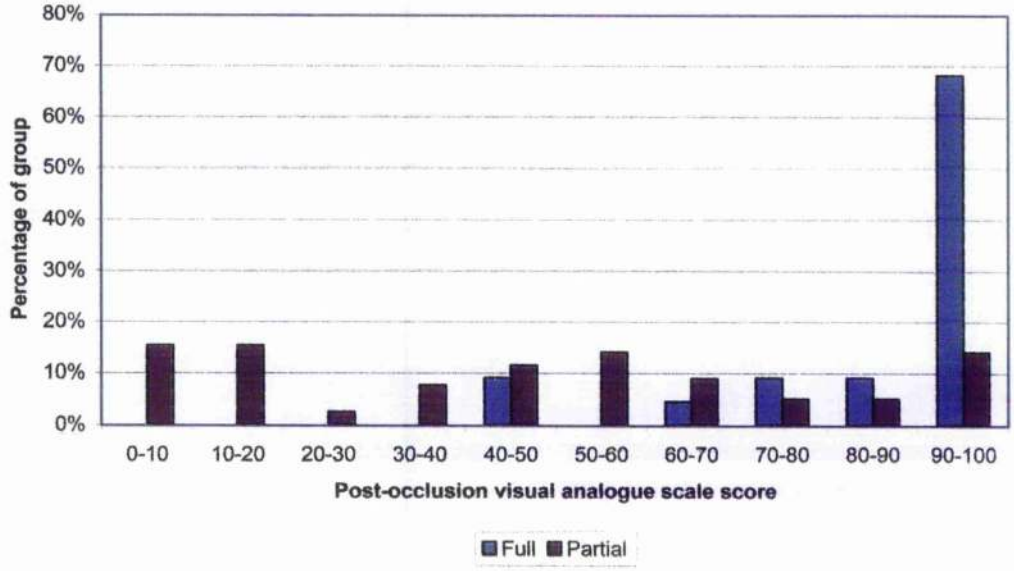


**Figure 16: Distribution of post-occlusion visual analogue scale scores of animals undergoing partial and full ligation of their portosystemic shunt**

When using the OSS, the data for pre-occlusion scores shows the partial ligation scores are distributed at the lower end of the scale while the full ligation scores are more evenly distributed but towards the higher end (Figure 17).

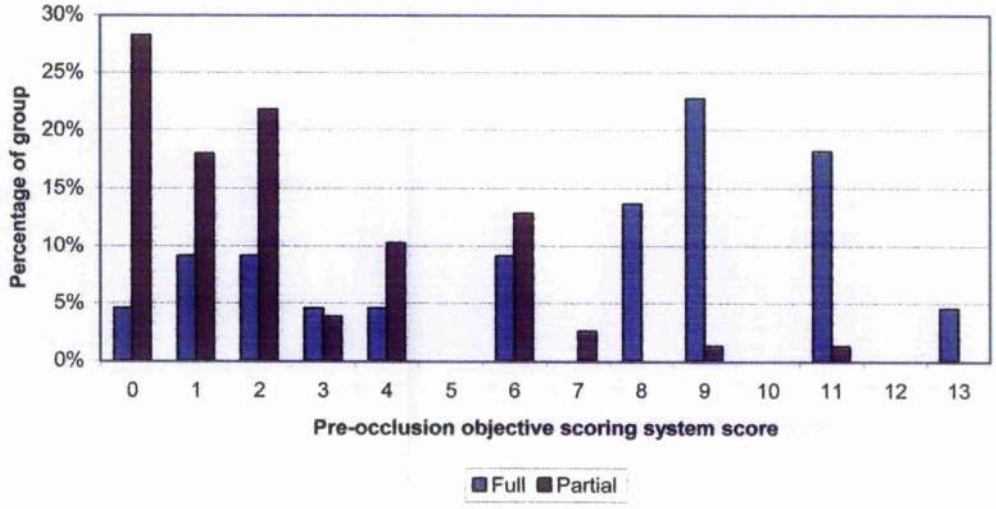


**Figure 17: Distribution of pre-occlusion objective scoring system scores of animals undergoing partial and full ligation of their portosystemic shunt**

Post-occlusion OSS data shows a more even distribution of partial ligation scores but the full ligation scores are largely confined to the upper scores (Figure 18).

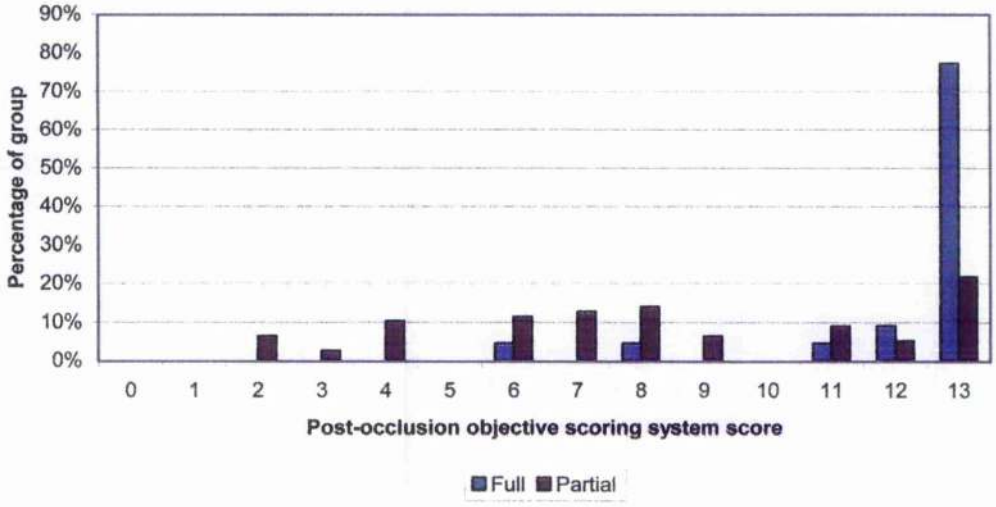


**Figure 18: Distribution of post-occlusion objective scoring system scores of animals undergoing partial and full ligation of their portosystemic shunt**

There was a significant difference between the results of animals with partial and full ligation of their PSS using either scoring system, both before and after temporary PSS occlusion ($P<0.01$ for all four tests).

Using the above data, the 95% confidence intervals of the median VAS scores were calculated for animals with partial and full ligation of their PSS for PVGs both before and after occlusion of their PSS. The 95% confidence intervals of the median OSS scores were calculated similarly. These results are presented in Table 9.

**Table 9: 95 per cent confidence intervals of pre- and post-occlusion scores for animals with partial and full ligation of their portosystemic shunt, using both scoring systems**

| | Partial ligation | Full ligation |
|---|---|---|
| Pre-occlusion VAS | 3.0 – 9.5 | 31.5 – 60.5 |
| Post-occlusion VAS | 39.5 – 54.0 | 80.0 – 96.0 |
| Pre-occlusion OS | 1.5 – 2.5 | 5.0 – 9.0 |
| Post-occlusion OS | 7.5 – 9.0 | 12.5-13.0 |

Examination of the post-occlusion scores show the lowest HPV score of an animal which tolerated full ligation of its PSS was 41 units using the

VAS and 6 units with the OSS. These figures are significantly lower than the low 95% confidence interval for each scoring system.

# Discussion

This is the first study which has attempted to quantitatively assess the HPV in a large group of dogs and cats with PSS. The development and assessment of the two scoring systems enabled reproducible and repeatable evaluation of intraoperative mesenteric PVGs. A number of previously reported studies have subjectively described the appearance of PVGs in animals with a PSS (Gofton, 1978; Wrigley and others, 1987; Martin and Payne, 1990; Swalec and Smeak, 1990; Swalec Tobias and others, 1996; White and others, 1996b; White and others, 1998). These methods are, however, unsatisfactory when comparing large numbers of PVGs both pre- and post-ligation. The variability of reported assessment methods makes comparison of data from these studies difficult. Previous assessment techniques remain crude, which is understandable given that data provided by a dynamic PVG lends itself to subjective rather than objective analysis. A form of precise and, ideally, objective measurement of portal vasculature development both prior to and following shunt attenuation is, therefore, highly desirable in the short and long term assessment of PSS in the dog and cat. It should, therefore, provide an ideal starting point on which to base further investigations of the effects of the various therapeutic interventions which are now available for the surgical management of PSS.

Several problems with the investigative method were identified during the study. True blind assessment was not possible because an experienced observer will recognise the shunting vessel on a pre-occlusion PVG, and an occluded shunting vessel on a post-occlusion PVG. This may introduce an element of bias, assigning higher VAS scores to post-occlusion PVGs. This bias should not apply to the OSS, as structures are either present or absent. It would be extremely difficult to negate this problem as this would require the PSS and the CVC to be edited out of the stored video image. This study was retrospective in nature and as such the decision whether to fully or partially ligate the PSS was made at

the time of surgery. In most cases the post-occlusion HPV development was one of several factors used to determine the degree of possible ligation, thus selecting for higher scores in those animals which tolerated full ligation. Only in a prospective study in which the surgeon had no opportunity to view the PVGs prior to making a decision on degree of shunt attenuation would this element of bias be eliminated. Those animals which underwent partial ligation of their PSS did so because the surgeon believed that full ligation of the vessel was unsafe. It is possible that in some of these cases, full ligation could have been safely tolerated and, therefore, the number of partial ligations performed may have been greater than was necessary. Again this may have produced an error in the interpretation of the results. Comparisons between intrahepatic and extrahepatic PSS will be skewed when using the OSS. Intrahepatic PSS all have a visible extrahepatic portal vein on PVG, whereas this does not apply to all animals with extrahepatic PSSs. This will increase the score of an intrahepatic PSS compared with an extrahepatic PSS with identical development of their HPV. This is particularly evident in individuals with PVGs scoring at the low end of the OSS, as in effect no animal with an intrahepatic PSS can score zero on the OSS. In this study the animals with intrahepatic PSS are not representative of the complete population of animals with this condition, as those individuals with a central divisional PSS were excluded from the study since the intravascular techniques required to close their shunts do not allow the performance of a post-occlusion PVG.

Although the two scoring methods used in this study are considerably more refined than those previously reported, some problems were encountered. The lack of statistical difference in the results of the inter-observer studies and the low reproducibility coefficients confirmed that both scoring systems had acceptable reproducibility. Good reproducibility is important when measuring a clinical parameter because it ensures validity in comparison of scores obtained by different observers. With poor reproducibility, an observer may produce a series of highly

repeatable scores that are still not comparable with results obtained from other centres or observers. Although the VAS had good reproducibility, it was interesting to note that the differences between the observers were greatest for values near the centre of the VAS and, conversely, the scores were found to be most similar at both the low and high ends of the scale. This is a recognised phenomenon in the use of a VAS, and it is suggested that scores falling in the region ± 20 mm of the centre point of the VAS line cause the most difficulty in both reproducibility and repeatability (Welsh and others, 1993).

The results of the within-observer studies confirmed that both scoring systems had excellent repeatability. The repeatability of a measurement is fundamental in ensuring that a measurement system is useful. If changes in a clinical parameter are to be measured over time, it is vital that any observed changes in the measured parameter are due to alteration in that variable and not due to variation in the accuracy of the scoring system. From the point of view of repeatability, both scoring systems appeared to be applicable to the assessment of dynamic intraoperative mesenteric portovenography. The fact that both scoring systems were highly repeatable was also of importance when consideration was given to a comparison of the two systems. If one or both of the scoring systems had poor repeatability, it would have proved difficult to compare the two systems. When evaluating a new scoring system with respect to a previously accepted system, if the older system has poor repeatability, even a perfectly accurate new system will have poor agreement. This lack of agreement might imply that the new scoring system is inaccurate when, in fact, the problem actually lies with the poor repeatability of the older, accepted system. Fortunately, in this study both scoring systems had a high level of repeatability and, therefore, a comparison between the two systems was considered both appropriate and meaningful. Assessment of the repeatability of portovenography itself was not performed, this would require repeated PVGs of the same animal

under similar conditions, which was not possible due to the retrospective nature of the study.

The two scoring systems were compared using a previously described method (Bland and Altman, 1999). This method measures the agreement between two systems which both measure a quantity of which the true value is not known. The 'new' system of measurement is compared to an 'established' system which itself may not provide a correct measurement. The method provides for assessment of how much the 'new' system differs from the 'established' system and this can then be used to determine whether the two systems are interchangeable. In this study the OSS was considered the 'established' method of measurement and the VAS the 'new' system of measurement although both systems were new. The results of the comparison between the VAS and OSS confirmed that the two scoring systems were not directly interchangeable. There are a number of reasons for this. As already eluded to, the degree of repeatability of each scoring system may have a profound effect on the findings of system comparison. The effect of repeatability was assessed by comparing the 95% limits of agreement of the two scoring systems with their repeatability coefficients. The repeatability coefficients were smaller than the limits of agreement suggesting there was some factor other than repeatability that was making the two systems not directly interchangeable. Bias was also demonstrated between the two systems, with the mean VAS score being 11.4 units higher than the OSS score. This, however, also does not completely explain the lack of agreement.

The reliability of both scoring systems may also contribute to their lack of interchangeability. Problems with reliability differed for each scoring system. For example, although the VAS had acceptable reproducibility, there was a central length (± 20 mm of the centre point) over which scores were considered less reliable. The conversion of OSS into values out of 100 so that the comparison with the VAS could be made produced its own set of inaccuracies. Firstly, the OSS produced discrete values

between zero and 13, so that even if there were perfect agreement between the two systems, there would be a range of VAS scores corresponding to a single OSS. This produces an inherent disagreement between the two scoring systems. As a factor in producing an overall disagreement between the two scoring systems, this was considered of little importance because the maximum disagreement for each score would only be half of an OSS point, that is, 3.8 units out of 100, well within the acceptable limits of agreement. Secondly, each of the questions in the OSS system did not represent an equal linear progression from the previous question and yet when the OSS scores were converted to a score out of 100 they were assumed to progress in an equal, linear manner of 7.7 units for each OSS question. For example, a portovenogram where there were only major portal branches with no arborisation would produce a low score on the visual analogue scale, although the same image might produce an OSS score of up to 5 out of 13. This may represent the most likely reason why the two systems cannot be directly interchanged.

Despite these inherent problems, the method of Bland and Altman (1999) is accepted to be the most appropriate way of quantifying agreement providing these limitations are understood. Measuring correlation between the two scoring systems would give very high correlation coefficients. This is because both systems are measuring the same criteria and would be expected to give increasing scores as the HPV improves, thus correlating well. Correlation does not give any information on whether the systems agree. Complete agreement will place all results along a line of equality when plotted against each other whereas results which correlate will be placed along any straight line, this can misleadingly give the impression of agreement.

The two scoring systems used in this study were devised for different reasons. The overall aim of a scoring system was to give a quantitative value for the degree of development of the HPV. The OSS was created to

provide an objective measurement of the HPV that could be used by any individual with knowledge of the normal anatomy of the portal vessels. Unfortunately, this scoring system proved to have a number of serious deficiencies. Several of the criteria used to interpret PVGs were found to be impossible to quantify. For example, it was not possible to quantify the degree of opacification of the liver parenchyma using the OSS. The system also proved problematic when interpreting intrahepatic shunts, such as a patent ductus venosus. Since all intrahepatic shunts emanate from either the left or right branch of the portal vein, a number of points will be scored on the OSS regardless of their further portal vasculature appearance. On the contrary, an extrahepatic shunt with similar portal development may have a lower score on the OSS since there may not be either a left or right portal vein branch. The OSS also assumes that both left and right portal vein branches are equal and symmetric in appearance. In the majority of normal individuals this is not true, with the left branch having greater development than the right (Burton and White, 1999). The VAS was devised because it would allow all gradations of portal development, from normal vasculature to complete portal atresia, to be assessed subjectively. This scoring system could take account of several factors that the OSS failed to assess. For example, the width of portal vessels rather than just their presence, the speed with which the vessels were observed to fill with contrast medium and the rate at which the contrast medium was seen to clear from these vessels. A disadvantage of the VAS was that it required the observer to be experienced in the assessment of PVGs.

The portovenograms for this study were produced using fluoroscopy and recorded on videotape. The procedure was performed in the operating theatre itself thus avoiding problems such as increased procedure time, hypothermia and loss of sterility associated with movement of the animal to a radiography room. The use of fluoroscopy and video storage and retrieval removes the possibility of missing the contrast medium outlining the vessels which may occur using static films due to sluggish portal

blood flow, or operator inexperience. A dynamic image of the portal vasculature gives a better indication of the flow rate of the contrast and allows the point of maximum contrast enhancement to be identified with confidence. By utilising video storage and retrieval, the study may be reviewed several times while in theatre negating the use of multiple contrast medium injections. Routinely only the ventrodorsal projection is used, however, if the vessel was unusual in appearance or difficult to locate a lateral projection would also be produced. The ventrodorsal view provides better information about the anatomical position of both intrahepatic and extrahepatic PSS. Several authors consider the use of intraoperative portovenography to be unnecessary for experienced surgeons due to the relatively common anatomical positions of many PSS (Komtebedde and others, 1991; Hunt and Hughes, 1999). Although it is often possible to locate the anomalous vessel during surgical exploration, a pre-occlusion PVG will provide more information about the morphology and location of the vessel than gross observation alone. A post-occlusion study will allow verification that the correct vessel has been identified, ensure the presence of as second PSS has been ruled out and allow assessment of the HPV to exclude portal atresia and assist in the decision regarding the degree of shunt attenuation.

In this study the decision whether to fully or partially ligate the PSS was made with reference to several factors, including post-occlusion HPV appearance, gross observation of splanchnic viscera, systemic arterial pressure, central venous pressure and portal pressure. These criteria were used in combination with the experience of the surgeon to determine the degree of shunt ligation. Although there are published figures of recommended post-occlusion portal pressures (Martin and Freeman, 1987; Swalec and Smeak, 1990; Bostwick and Twedt, 1995), these figures are considered by many authorities to be unreliable (Swalec and Smeak, 1990; White and others, 1998). Portal pressure measurement is notoriously unreliable due to many factors including position of the abdominal viscera, hypothermia, anaesthetic depth,

splanchnic venous compliance, arterial blood pressure and venospasm in the PSS following its manipulation (White and others, 1998). The post-occlusion PVG gives a visual representation of the perfusion of the liver, and is thus a useful additional tool when deciding whether to partially or fully ligate a PSS.

This study shows that the HPV visualised on a post-occlusion PVG is significantly different to the HPV outlined on a pre-occlusion PVG. This is to be expected as the flow of contrast medium (and thus portal blood) through the portal vasculature is vastly different when a shunting vessel is present compared to when it is occluded. With the PSS patent, it provides a route for the contrast to bypass the liver via the caudal vena cava or azygos vein. This route is chosen because of the lower pressure found in these veins compared to the hepatic portal vasculature with its higher resistance. Once the vessel is occluded, the contrast is forced to pass through the liver producing a more accurate view of the HPV. It will still flow through the path of least resistance producing the differential perfusion of the portal vein branches commonly observed. The initial degree of HPV visualisation is therefore related to the diameter of the PSS and the pressure gradients rather than any portal hypoplasia. With portal atresia the contrast will have no path to the liver and will therefore pool in the extrahepatic portal vein after PSS occlusion. This may also be seen in cases of severe portal vein hypoplasia. Whether the post-occlusion PVG gives an entirely accurate depiction of the maximum HPV depends on whether the hypoplasia is due to anatomical or functional factors. If the restriction is physical, with the vessels stretched as wide as possible without further growth, then the PVG will be a true representation of the HPV. However, if it is a functional problem, with sphincters or smooth muscle contraction restricting flow until the portal pressure gradually increases, then the PVG may not be accurate.

The correct diagnosis of portal atresia is essential. Attempting to ligate or attenuate the PSS in these animals will, at best, lead to the formation of multiple acquired PSS with continued clinical signs, and at worst be fatal. Similar problems can occur in animals with severe portal hypoplasia which are treated using materials which cause progressive attenuation (ameroid constrictors, cellophane bands). If the vessel becomes attenuated before the portal vasculature has developed enough to accept the increased blood flow then the formation of multiple acquired PSS is inevitable, this may explain the higher rate of long-term failure in these animals (Vogt and others, 1996) when compared with the use of silk (Hunt and Hughes, 1999). While it is important to make the correct positive diagnosis, it is also important that surgeons do not, by performing only pre-occlusion PVGs, over-diagnose portal atresia and deny many animals the benefit of corrective surgery. In this study 62 animals appeared to have hypoplastic or atretic portal veins on a pre-occlusion PVG using the VAS, of these, 50 showed improved HPV after vessel occlusion and all were at least partially ligated successfully.

An attempt was made to define a range of HPV scores for which full ligation of the PSS is safe, the 95% confidence intervals this produced are relatively narrow and the range of scores quite high. This is partly due to the small number of full ligations performed, with more data the ranges may become wider and lower values may be indicated. Lower values are certain to be safe in some animals as indicated by the lowest safe VAS score shown in this trial of 41, and 6 using the OSS, which are both substantially lower than their respective confidence intervals. Further work could be aimed at more accurately defining these safe limits although because several factors are taken into account when deciding the safe degree of attenuation for each animal it may not be ethical to use the HPV score alone in this decision in clinical cases.

Of the two scoring systems devised, the inherent deficiencies of the OSS suggest the VAS will be the most useful system for assessing HPV. With

a repeatable and reproducible scoring system, it will be possible to more accurately track the changes in the HPV due to surgical intervention. This would provide an ideal way of monitoring the effectiveness of the different methods of surgical attenuation available. If portovenography could be performed regularly after attenuation an explanation may be found for the different outcomes of attenuation in animals undergoing similar degrees and methods of attenuation, that is, why some animals progress to full closure of the vessel, some show continued flow through it and others form multiple acquired PSS (Burton and White, 2001). Portovenography, although more invasive than serum bile acid measurement and portal scintigraphy, is more sensitive at diagnosing portosystemic shunting. It provides significantly more information about the portal vasculature and can provide anatomical details of any continued portosystemic flow. Using the HPV scoring system described, it is possible to quantitatively assess the changes in the portal vasculature caused by the surgical intervention. With this information it may become possible to determine which animals are likely to progress to a normal HPV following surgery and which animals would not show further HPV development and thus would be at risk of developing multiple acquired portosystemic shunts.

In conclusion, both the OSS and VAS demonstrated acceptable reproducibility and repeatability when they were used to assess PVGs. The OSS had a number of inherent deficiencies that suggested it was not the method of choice in the assessment of PVGs. The VAS was considered most accurate in the assessment of PVGs characterised by either a very well-developed or a very poorly developed portal vasculature. Although optimal use of the VAS requires an experienced observer, it has been shown to provide a useful method for the assessment of the subjective data obtained from PVGs. The HPV observed on a post-occlusion PVG is significantly different to that seen before the vessel is occluded. This indicates that the HPV visualised on a pre-occlusion PVG is unreliable and more likely to be related to the flow through the PSS than the development of the portal vasculature. Many

animals in this study with apparent portal atresia or severe hypoplasia on pre-occlusion PVG were shown to have adequate to excellent HPV development after occlusion of the PSS.

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# Appendix

# Appendix I – Inter-observer trial

## Visual Analogue Scale

| Trial Number | Observer 1 | Observer 2 |
|---|---|---|
| 1 | 0 | 0 |
| 2 | 0 | 1 |
| 3 | 7 | 5 |
| 4 | 1 | 0 |
| 5 | 0 | 0 |
| 6 | 10 | 15 |
| 7 | 3 | 5 |
| 8 | 1 | 0 |
| 9 | 0 | 1 |
| 10 | 49 | 60 |
| 11 | 35 | 23 |
| 12 | 0 | 0 |
| 13 | 0 | 0 |
| 14 | 20 | 12 |
| 15 | 5 | 7 |
| 16 | 38 | 45 |
| 17 | 4 | 6 |
| 18 | 0 | 1 |
| 19 | 0 | 0 |
| 20 | 1 | 1 |
| 21 | 53 | 47 |
| 22 | 10 | 16 |
| 23 | 68 | 78 |
| 24 | 78 | 69 |
| 25 | 17 | 20 |
| 26 | 45 | 58 |
| 27 | 39 | 31 |
| 28 | 11 | 17 |
| 29 | 9 | 10 |
| 30 | 99 | 97 |
| 31 | 100 | 99 |
| 32 | 20 | 15 |
| 33 | 63 | 57 |
| 34 | 51 | 64 |
| 35 | 90 | 84 |
| 36 | 42 | 51 |
| 37 | 95 | 98 |
| 38 | 47 | 32 |
| 39 | 10 | 15 |
| 40 | 23 | 18 |

## Objective Scoring System

| Trial Number | Observer 1 | Observer 2 |
|---|---|---|
| 1 | 1 | 1 |
| 2 | 0 | 0 |
| 3 | 1 | 1 |
| 4 | 1 | 1 |
| 5 | 1 | 1 |
| 6 | 2 | 2 |
| 7 | 2 | 2 |
| 8 | 0 | 0 |
| 9 | 0 | 0 |
| 10 | 8 | 8 |
| 11 | 5 | 5 |
| 12 | 0 | 0 |
| 13 | 1 | 1 |
| 14 | 1 | 1 |
| 15 | 1 | 1 |
| 16 | 7 | 7 |
| 17 | 2 | 2 |
| 18 | 0 | 0 |
| 19 | 0 | 0 |
| 20 | 0 | 0 |
| 21 | 7 | 7 |
| 22 | 3 | 3 |
| 23 | 10 | 10 |
| 24 | 10 | 10 |
| 25 | 4 | 4 |
| 26 | 8 | 8 |
| 27 | 5 | 5 |
| 28 | 3 | 3 |
| 29 | 3 | 3 |
| 30 | 13 | 13 |
| 31 | 13 | 13 |
| 32 | 4 | 4 |
| 33 | 8 | 8 |
| 34 | 7 | 7 |
| 35 | 12 | 12 |
| 36 | 8 | 8 |
| 37 | 13 | 13 |
| 38 | 5 | 5 |
| 39 | 4 | 4 |
| 40 | 4 | 4 |

# Appendix II – Within-observer trial

## Visual Analogue Scale

| Trial Number | Observer 1 | Observer 1 +1 hour | Observer 2 | Observer 2 +1 hour |
|---|---|---|---|---|
| 1 | 50 | 45 | 45 | 45 |
| 2 | 76 | 79 | 82 | 78 |
| 3 | 58 | 60 | 59 | 62 |
| 4 | 74 | 88 | 79 | 81 |
| 5 | 61 | 66 | 71 | 73 |
| 6 | 93 | 95 | 89 | 93 |
| 7 | 22 | 16 | 20 | 19 |
| 8 | 80 | 86 | 79 | 76 |
| 9 | 25 | 32 | 35 | 37 |
| 10 | 2 | 7 | 3 | 2 |
| 11 | 33 | 41 | 41 | 38 |
| 12 | 7 | 3 | 3 | 2 |
| 13 | 45 | 39 | 35 | 40 |
| 14 | 40 | 33 | 38 | 41 |
| 15 | 86 | 93 | 86 | 83 |
| 16 | 6 | 6 | 2 | 5 |
| 17 | 34 | 23 | 37 | 41 |
| 18 | 88 | 95 | 89 | 85 |
| 19 | 13 | 9 | 22 | 21 |
| 20 | 90 | 95 | 84 | 88 |

## Objective Scoring System

| Trial Number | Observer 1 | Observer 1 +1 hour | Observer 2 | Observer 2 +1 hour |
|---|---|---|---|---|
| 1 | 7 | 7 | 7 | 7 |
| 2 | 10 | 10 | 10 | 10 |
| 3 | 8 | 8 | 8 | 8 |
| 4 | 10 | 10 | 10 | 10 |
| 5 | 8 | 8 | 8 | 8 |
| 6 | 12 | 12 | 12 | 12 |
| 7 | 3 | 3 | 3 | 3 |
| 8 | 10 | 10 | 10 | 10 |
| 9 | 3 | 3 | 3 | 3 |
| 10 | 0 | 0 | 0 | 0 |
| 11 | 4 | 4 | 4 | 4 |
| 12 | 1 | 1 | 1 | 1 |
| 13 | 6 | 6 | 6 | 6 |
| 14 | 5 | 5 | 5 | 5 |
| 15 | 11 | 11 | 11 | 11 |
| 16 | 1 | 1 | 1 | 1 |
| 17 | 34 | 23 | 37 | 41 |
| 18 | 88 | 95 | 89 | 85 |
| 19 | 13 | 9 | 22 | 21 |
| 20 | 90 | 95 | 84 | 88 |

## Appendix III - Data collected from Visual Analogue Scale and Objective Scoring System evaluation of portovenograms (including conversion to a score out of 100 of the Objective Scoring System)

| Study No. | Species | Breed | Sex | Age (months) | Type of PSS | FL or PL | VAS | | OSS | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Pre | Post | Pre | Post | Pre(100) | Post(100) |
| 1 | Cat | Siamese | F | 5 | PDV | PL | 24 | 62 | 3 | 9 | 23 | 69 |
| 2 | Dog | SBT | F | 36 | EHPC | FL - died | 1 | 47 | 0 | 6 | 0 | 46 |
| 3 | Cat | BSH | M | 24 | L gastric | PL | 5 | 53 | 1 | 9 | 8 | 69 |
| 4 | Dog | Pekinese | M | 13 | EHPC | PL | 49 | 96 | 4 | 13 | 31 | 100 |
| 5 | Dog | Irish wolfhound | M | 2.75 | PDV | PL | 1 | 4 | 2 | 2 | 15 | 15 |
| 6 | Dog | Norfolk terrier | M | 12 | EHPC | PL | 42 | 99 | 6 | 13 | 46 | 100 |
| 7 | Dog | Labrador | M | 3.5 | EHPC | PL | 2 | 39 | 0 | 7 | 0 | 54 |
| 8 | Dog | Norfolk terrier | M | 12 | EHPC | PL | 44 | 90 | 6 | 12 | 46 | 92 |
| 9 | Dog | Shih Tzu | M | 5 | EHPC | PL | 72 | 78 | 9 | 10 | 69 | 77 |
| 10 | Cat | Persian | F | 6 | L gastric | FL | 49 | 99 | 6 | 13 | 46 | 100 |
| 11 | Dog | Cairn terrier | M | 60 | EHPC | PL | 34 | 96 | 5 | 13 | 38 | 100 |
| 12 | Cat | Persian | M | 24 | EHPC | PL | 0 | 49 | 0 | 7 | 0 | 54 |
| 13 | Dog | Cairn terrier | F | 60 | EHPC | FL | 73 | 93 | 9 | 13 | 69 | 100 |
| 14 | Dog | Irish Setter | F | 5 | PDV | PL | 1 | 53 | 2 | 4 | 15 | 31 |
| 15 | Dog | YT | M | 18 | Colonic | PL | 16 | 99 | 4 | 13 | 31 | 100 |
| 16 | Dog | WHWT | M | 7 | EHPC | PL | 32 | 56 | 4 | 8 | 31 | 62 |
| 17 | Dog | G retriever | M | 4 | PDV | PL | 1 | 12 | 2 | 4 | 15 | 31 |
| 18 | Dog | YT | F | 4 | EHPC | PL | 0 | 57 | 0 | 10 | 0 | 77 |
| 19 | Dog | YT | F | 12 | EHPC | PL | 0 | 16 | 0 | 3 | 0 | 23 |
| 20 | Cat | BSH | M | 24 | L gastric | PL | 8 | 50 | 1 | 7 | 8 | 54 |
| 21 | Dog | Labrador | M | 3 | CIH | PL | 2 | 17 | 1 | 7 | 8 | 54 |
| 22 | Cat | Persian | F | 6 | L gastric | PL | 0 | 69 | 0 | 11 | 0 | 85 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 23 | Dog | Boxer | M | 3.75 | PDV | PL | 1 | 10 | 2 | 6 | 15 | 46 |
| 24 | Dog | Border collie | F | 5 | Umbilical v | PL | 1 | 17 | 0 | 4 | 0 | 31 |
| 25 | Dog | crossbred | M | 24 | EHPC | PL | 12 | 88 | 4 | 13 | 31 | 100 |
| 26 | Dog | Shih Tzu | F | 12 | L gastric | FL | 64 | 97 | 9 | 13 | 69 | 100 |
| 27 | Dog | Irish wolfhound | M | 8 | PDV | PL | 1 | 4 | 2 | 2 | 15 | 15 |
| 28 | Cat | Rag doll | M | 15 | L gastric | PL | 2 | 78 | 1 | 13 | 8 | 100 |
| 29 | Dog | Bichon Frise | F | 5 | EHPC | PL | 7 | 70 | 1 | 10 | 8 | 77 |
| 30 | Cat | DSH | F | 13 | L gastric | PL | 4 | 98 | 1 | 13 | 8 | 100 |
| 31 | Dog | English setter | F | 5 | EHPC | PL | 0 | 7 | 0 | 2 | 0 | 15 |
| 32 | Dog | Irish wolfhound | M | 2 | PDV | PL | 1 | 9 | 2 | 5 | 15 | 38 |
| 33 | Dog | Irish wolfhound | F | 2 | PDV | PL | 0 | 8 | 2 | 4 | 15 | 31 |
| 34 | Dog | Lhasa Apso | M | 2 | EHPC | PL | 0 | 13 | 0 | 2 | 0 | 15 |
| 35 | Dog | JRT | F | 5 | EHPC | PL | 1 | 11 | 0 | 4 | 0 | 31 |
| 36 | Dog | Irish wolfhound | M | 3 | PDV | PL | 3 | 31 | 2 | 7 | 15 | 54 |
| 37 | Dog | YT | F | 12 | EHPC | FL | 74 | 96 | 11 | 13 | 85 | 100 |
| 38 | Dog | Irish wolfhound | M | 2 | PDV | PL | 1 | 7 | 2 | 4 | 15 | 31 |
| 39 | Dog | Irish wolfhound | F | 2 | PDV | PL | 2 | 16 | 2 | 6 | 15 | 46 |
| 40 | Cat | DSH | M | 10 | L gastric | PL | 6 | 52 | 1 | 10 | 8 | 77 |
| 41 | Dog | BMD | F | 3 | PDV | PL | 6 | 8 | 3 | 7 | 23 | 54 |
| 42 | Cat | DSH | F | 48 | L gastric | FL | 43 | 96 | 9 | 13 | 69 | 100 |
| 43 | Dog | Irish wolfhound | F | 3 | PDV | PL | 44 | 51 | 7 | 8 | 54 | 62 |
| 44 | Dog | Labrador | F | 12 | PDV | PL | 0 | 25 | 2 | 7 | 15 | 54 |
| 45 | Cat | Siamese | M | 6 | EHPC | PL | 0 | 24 | 0 | 3 | 0 | 23 |
| 46 | Dog | Maltese terrier | F | 9 | EHPC | FL | 88 | 96 | 11 | 13 | 85 | 100 |
| 47 | Dog | Maltese terrier | M | 7 | EHPC | FL | 67 | 97 | 11 | 13 | 85 | 100 |
| 48 | Dog | Bearded collie | F | 7 | EHPC | PL | 0 | 51 | 0 | 8 | 0 | 62 |
| 49 | Dog | YT | M | 10 | EHPC | PL | 0 | 15 | 0 | 5 | 0 | 38 |
| 50 | Cat | Siamese | F | 12 | PDV | PL | 0 | 2 | 2 | 5 | 15 | 38 |
| 51 | Dog | Irish setter | F | 10 | EHPA | PL | 4 | 93 | 1 | 13 | 8 | 100 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 52 | Dog | YT | M | 24 | EHPC | PL | 5 | 94 | 1 | 13 | 8 | 100 |
| 53 | Dog | Border Collie | F | 24 | RIH | PL | 2 | 10 | 2 | 4 | 15 | 31 |
| 54 | Dog | Irish setter | F | 7 | EHPC | FL | 65 | 96 | 9 | 13 | 69 | 100 |
| 55 | Dog | G retriever | F | 11 | EHPC | PL | 16 | 66 | 7 | 12 | 54 | 92 |
| 56 | Cat | Persian | M | 4 | L gastric | FL | 63 | 98 | 8 | 13 | 62 | 100 |
| 57 | Cat | Tonkinese | F | 7 | EHPC | PL | 0 | 45 | 0 | 8 | 0 | 62 |
| 58 | Dog | Shih Tzu | M | 4 | EHPC | PL | 3 | 47 | 1 | 10 | 8 | 77 |
| 59 | Dog | G retriever | F | 10 | PDV | PL | 1 | 17 | 2 | 5 | 15 | 38 |
| 60 | Cat | DSH | M | 8 | L gastric | FL | 15 | 81 | 6 | 13 | 46 | 100 |
| 61 | Dog | WHWT | F | 16 | EHPC | FL | 6 | 96 | 1 | 13 | 8 | 100 |
| 62 | Dog | Std oodle | F | 2 | RIH | FL | 1 | 75 | 2 | 13 | 15 | 100 |
| 63 | Dog | YT | M | 6 | EHPC | PL | 20 | 99 | 4 | 13 | 31 | 100 |
| 64 | Dog | Clumber s | M | 7 | PDV | PL | 8 | 52 | 2 | 6 | 15 | 46 |
| 65 | Dog | G retriever | F | 3 | PDV | PL | 11 | 64 | 4 | 8 | 31 | 62 |
| 66 | Cat | BSH | M | 4 | EHPC | PL | 0 | 42 | 0 | 7 | 0 | 54 |
| 67 | Dog | WHWT | F | 5 | EHPC | PL | 11 | 79 | 4 | 13 | 31 | 100 |
| 68 | Cat | DSH | F | 12 | EHPC | PL | 3 | 51 | 1 | 8 | 8 | 62 |
| 69 | Dog | Shih Tzu | M | 6 | L gastric | PL | 5 | 68 | 1 | 13 | 8 | 100 |
| 70 | Dog | YT | F | 36 | EHPA | PL | 26 | 100 | 5 | 13 | 38 | 100 |
| 71 | Dog | GSD | M | 12 | LIH | PL | 26 | 59 | 6 | 9 | 46 | 69 |
| 72 | Dog | Labrador | F | 3 | PDV | PL | 0 | 50 | 2 | 8 | 15 | 62 |
| 73 | Cat | Havana | M | 48 | PDV | PL | 3 | 42 | 1 | 4 | 8 | 31 |
| 74 | Dog | Labrador | F | 4 | PDV | PL | 13 | 40 | 5 | 7 | 38 | 54 |
| 75 | Cat | DSH | M | 8 | EHPC | FL | 1 | 94 | 2 | 13 | 15 | 100 |
| 76 | Dog | Labrador | M | 48 | PDV | PL | 21 | 45 | 5 | 8 | 38 | 62 |
| 77 | Dog | Shih Tzu | M | 7 | EHPC | FL | 62 | 96 | 10 | 13 | 77 | 100 |
| 78 | Dog | Maltese terrier | F | 72 | EHPC | FL | 9 | 84 | 3 | 12 | 23 | 92 |
| 79 | Dog | WHWT | M | 7 | EHPC | PL | 0 | 82 | 0 | 13 | 0 | 100 |
| 80 | Dog | Cairn terrier | F | 12 | EHPC | PL | 3 | 93 | 1 | 13 | 8 | 100 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 81 | Dog | WHWT | M | 8 | EHPC | PL | 0 | 37 | 0 | 12 | 0 | 92 |
| 82 | Cat | DLH | M | 12 | L gastric | FL | 17 | 79 | 4 | 13 | 31 | 100 |
| 83 | Dog | Border collie | F | 6 | PDV | PL | 9 | 18 | 3 | 8 | 23 | 62 |
| 84 | Dog | Irish setter | F | 60 | EHPA | PL | 0 | 92 | 0 | 13 | 0 | 100 |
| 85 | Dog | JRT | M | 48 | EHPC | PL | 1 | 18 | 0 | 6 | 0 | 46 |
| 86 | Dog | Min Schnauzer | M | 24 | L gastric | PL | 64 | 69 | 11 | 12 | 85 | 92 |
| 87 | Dog | Bichon Frise | M | 108 | EHPC | PL | 9 | 34 | 5 | 9 | 38 | 69 |
| 88 | Dog | Norfolk terrier | F | 8 | EHPA | PL | 0 | 34 | 0 | 8 | 0 | 62 |
| 89 | Dog | Norfolk terrier | M | 60 | EHPA | FL | 82 | 91 | 13 | 13 | 100 | 100 |
| 90 | Dog | Chihuahua | M | 48 | EHPC | FL | 23 | 41 | 8 | 8 | 62 | 62 |
| 91 | Dog | WHWT | F | 5 | EHPC | PL | 1 | 8 | 0 | 5 | 0 | 38 |
| 92 | Dog | Shih Tzu | M | 4.25 | EHPC | FL | 47 | 63 | 9 | 11 | 69 | 85 |
| 93 | Dog | G retriever | F | 3 | PDV | PL | 2 | 15 | 2 | 8 | 15 | 62 |
| 94 | Dog | PMD | F | 3 | PDV | PL | 12 | 53 | 5 | 10 | 38 | 77 |
| 95 | Dog | Sheltie | F | 11 | Umbilical v | PL | 1 | 4 | 0 | 2 | 0 | 15 |
| 96 | Dog | WHWT | F | 24 | L gastric | FL | 1 | 92 | 1 | 12 | 8 | 92 |
| 97 | Dog | Min Schnauzer | M | 5 | EHPC | FL | 81 | 96 | 8 | 13 | 62 | 100 |
| 98 | Dog | Border collie | M | 12 | PDV | PL | 21 | 49 | 5 | 7 | 38 | 54 |
| 99 | Dog | Norfolk terrier | F | 72 | EHPC | PL | 0 | 88 | 0 | 13 | 0 | 100 |
| 100 | Dog | Cairn terrier | F | 24 | EHPC | PL | 20 | 71 | 4 | 9 | 31 | 69 |



**Key:** SBT Staffordshire Bull terrier, BSH British shorthair, YT Yorkshire terrier, WHWT West Highland White terrier, G retriever Golden retriever, DSH Domestic shorthair, JRT Jack Russell terrier, BMD Bernese Mountain dog, Std Standard, sp spaniel, GSD German Shepherd dog, DLH Domestic longhair, Min. miniature, PMD Pyrenean Mountain dog, Sheltie Shetland sheepdog, M male, F female, EHPC Extrahepatic portocaval, L gastric Left gastric vein, EHPA Extrahepatic portoazygos, PDV Patent ductus venosus, LIH Left divisional intrahepatic, CIH Central divisional intrahepatic, RIH Right divisional intrahepatic, PL Partial ligation, FL full ligation, VAS Visual analogue scale, OSS Objective scoring system