INTRODUCTION
Interventional Radiology uses fluoroscopy to guide minimally-invasive therapies. There is currently expanding investigation into the use of these techniques in various areas of veterinary medicine, including but not limited to the arterial and venous systems. The morbidity associated with certain open surgical procedures in these areas, particularly in compromised patients, makes these minimally-invasive approaches increasingly appealing. Moreover, the lack of treatment alternatives available for more complex, terminal, or end-stage diseases when traditional therapies have failed has inspired research into potential uses for these techniques, many of which have become the standard-of-care in human medicine. This lecture will review some of the interventional procedures currently performed in the hepatic arterial and venous systems of veterinary patients and some of the lessons learned – or more importantly the new questions raised – using these techniques.

HEPATIC VASCULAR ANOMALIES
The categorization of liver vascular anomalies is often confusing and but the most recent classification suggests three separate categories of liver vascular disease: (1) Congenital portosystemic shunts (IHPSS and EHPSS), (2) Disorders associated with abnormal hepatic bloodflow or portal hypertension, currently termed Primary Hypoplasia of the Portal Vein (PVH), and (3) Disturbances in outflow. The second category (PVH) remains the most confusing, and includes processes that may or may not result in portal hypertension. These are termed PVH with portal hypertension and PVH without portal hypertension. Examples of PVH with portal hypertension include non-cirrhotic portal hypertension (NCPH), and hepato-portal fibrosis/veno-occlusive disease. PVH without portal hypertension was previously termed microvascular dysplasia (MVD).

IHPSS (and EHPSS): Single, extrahepatic PSSs are amenable to relatively uncomplicated surgical attenuation, however surgical repair of intrahepatic PSSs are consistently more challenging. Numerous techniques have been described for intrahepatic PSS attenuation, however morbidity and mortality rates can be very high, even for the most experienced surgeons. The goal of IR techniques for IHPSSs is to reduce the unacceptably high, peri-operative mortality rates associated with traditional open surgical techniques and hopefully improve the outcome for these cases. The author has performed over 100 percutaneous transvenous coil embolizations (PTCE) with a vena caval stent and thrombogenic coils placed within the shunt. Peri-operative complications were mostly minor and peri-operative mortalities were comparatively low versus that reported for traditional surgery.

PROCEDURE:
Percutaneous Transvenous Coil Embolization- All dogs are treated medically initially following diagnosis of the IHPSS for a period of weeks to months. When possible, CT or MR angiography is performed to delineate the shunt anatomy and obtain caval and shunt measurements under a separate anesthetic episode. All PTCE procedures are performed under general anesthesia using standard liver dysfunction protocols and often neuromuscular blockade to minimize respiratory artifact during digital subtraction angiography. Shunts are typically accessed by a percutaneous right jugular approach. Contrast venography is performed to delineate the portal vein, portosystemic shunt, and caudal vena cava anatomy. Intravascular pressure measurements are obtained in the caudal vena cava and portal vein. Shunt attenuation involves placement of a stent within the caudal vena cava, positioned so as to traverse the shunt entrance into the vena cava. Shunt PTCE is subsequently performed by passage of a catheter
through the stent interstices and deposition of the coils within the shunt lumen. Coils are subsequently added with intermittent shunt pressure measurements taken to avoid creating portal hypertension. Coils are typically added until the shunt mouth is covered with coils or the shunt pressures have increased between 6 and 10 cm H$_2$O or maximal pressures approached 20 cm H$_2$O. Ultimate shunt and caval pressures are recorded, the jugular sheath is exchanged for a 7 Fr multi-lumen catheter, and the animal is recovered from anesthesia. Following the initial procedure, medications are gradually weaned over the following 4-8 weeks. Additional PTCE is recommended if clinical signs returned.

LESSONS LEARNED:
Lessons learned or questions raised concerning DIAGNOSTIC IMAGING include:
- CT and MR angiography are well tolerated and facilitate identifications of uncommon shunt anatomy, however certain abnormalities are underestimated using these techniques. For instance, multiple small intrahepatic shunts are often not identified on standard cross-sectional imaging. These abnormalities are better identified with traditional contrast portography. It is not clear if dual phase CT provides us with additional important information at this time (as compared to single-phase).
- MRA (using gadolinium) may permit single procedure IHPSS imaging and treatment in order to avoid excessive iodinated contrast use associated with CT angiography following by angiography.
- Even “typical” IHPSS have variant hepatic vein anatomy. Does location of HV entrance into the PSS affect results of attenuation? It is conceivable that intra-hepatic vein shunting (acquired intrahepatic PSS) may occur more readily in these patients. Examples will be discussed.

Lessons learned or questions raised concerning TREATMENT include:
- A certain small population of IHPSS patients have “significant” portal:systemic venous pressure gradients (or resting portal pressures) before treatment. This is counter-intuitive in animals with PSS in that reduced portal pressure gradients would be anticipated, and this has prevented treatment in some cases. Are there small vascular windows or narrowings present that are not identified on cross sectional imaging with relatively wide slices? This suspicion has been raised as pull-out pressure tracings confirm short, focal areas where pressure gradients exist. The presence of a developed portal system may suggest a narrowing of the shunt in some location making access more difficult. This may be the same for EHPSS suggesting intermittent shunt compression in phrenic shunts for instance!!
- Which is more important in preventing the development of complications associated with portal hypertension following IHPSS treatment; Total portal pressure or pressure gradients? During surgery we rarely measured CVP and using IR techniques, we always measure CVP.
- During portography, when multiple small intrahepatic shunts are identified, this is almost exclusively associated with and elevated portal pressure and/or pressure gradient. Are these congenital shunts or acquired IHPSS resulting from a congenitally narrowed IHPSS?
- Acquired Intrahepatic Portosystemic shunts: Originally believed to only acquire EHPSS, there is more evidence that IHPSS can be acquired as well. Do the same criteria for shunt attenuation (no greater than ~10cmH2O rise in portal pressure and/or no greater than ~20cmH2O total portal pressure) hold for attenuation of IHPSS? Although there is no documented difference between HV attenuation and PV attenuation, the vascular bed receiving the congestion is intrahepatic with the former and extrahepatic with the latter. Does this matter?
- DO NOT PERFORM IHPSS SHUNT ATTENUATION IN THE FACE OF GI ULCERATION/HEMORRHAGE. The authors currently perform endoscopy and biopsy of all IHPSS cases before treatment and the overwhelming majority of these dogs have some degree of inflammatory bowel disease, sometimes including GI ulceration. Approximately 17% of patients have evidence of GI bleeding before treatment. Elevation of portal pressures with the presence of GI ulceration can lead to severe GI hemorrhage and death. All animals are maintained on omeprazole therapy for life. Initially a long-term mortality rate of 30% in IHPSS PTCE animals was caused by GI bleeding in ~50% of deaths. Lifelong antacid therapy has reduced the mortality rate to 12.5% with fewer than 4% secondary to GI bleeds. Is lifelong omeprazole therapy safe?

Lessons learned or questions raised concerning FOLLOW-UP include:
- Is return to normal bile acids concentrations necessary? Should this be the goal of therapy? The majority of patients receiving IHPSS PTCE do not have return to normal liver function and some have even been identified to have no development of portal branching, although pressure gradients continue to
HEPATIC AVMS:
The WSAVA liver study group has recently reclassified circulatory disorders of the liver. One of the less common congenital vascular anomalies present within the liver previously termed, “arteriovenous fistula (AVF),” is more appropriately termed “hepatic arteriovenous malformation (HAVM)” due to more recent evidence and a better understanding of the anatomy of such lesions. Little veterinary information exists on the nature of AVMs so most of the information presented here is based upon human experiences. Although both forms of “high-flow” vascular anomalies, an AVF is a single communication between an artery and vein and can typically be easily identified using cross-sectional imaging or angiography. A more common example of such a lesion could be considered a patent ductus arteriosus. On the other hand, an AVM is composed of multiple small communications involving a “nidus”, or nest, of vessels and can be more difficult to identify and treat. Treatment is often much more complicated, aimed at embolizing the nidus rather than the feeders, and inappropriate treatment can exacerbate the lesion by stimulating growth and making future treatments more difficult. In addition, treatment of AVMs is often considered more palliative than cure as repeat treatments can be anticipated in the human experience. AVMs are believed to be congenital lesions in veterinary patients even though the diagnosis may not be made until later in life depending upon location. AVFs can be congenital or acquired following trauma (bite wound or biopsy), ligation of an artery to a vein, etc.

HAVMs are most often diagnosed in young dogs (or less often cats) and typically involve too numerous to count communications between the hepatic artery and portal vein in the right or central divisions of the liver. Consequently, arterialization of the portal vein (rather than the substantially lower venous pressures of 7-9mmHg) results in development of multiple acquired extrahepatic portosystemic shunts, a similar clinical syndrome to that seen with IHPSS/EHPSS, and often ascites. The author’s experience covering only approximately 20 cases has not identified a hepatic artery to hepatic vein communication. Clinical signs are typically associated with ascites (75% of dogs) and/or hepatomegaly (less common than with PSS). As such, these patients are often misidentified as IHPSS dogs as the bloodwork changes and clinical syndrome is similar and a large intrahepatic vessel is identified on ultrasonography. The presence of ascites should raise suspicion (rare in IHPSS cases as these patients have portal hypotension while HAVM dogs have portal hypertension). While 25% of dogs will not have ascites presumptively due to the acquired EHPSS decompressing the portal system, a reliable diagnostic tool seems to be identification of hepatofugal (retrograde) portal bloodflow that is always present in these dogs examined to date by the author. Other clinical signs include GI signs (diarrhea, vomiting), stunted growth, lethargy, and heart murmurs (present in ~20% of dogs). The author has rarely appreciated the reportedly identified audible “bruit” when the AVM is auscultated.

Potential treatment options for HAVM have included nutrient artery ligation, surgical resection (liver lobectomy or lobectomies) and more recently trans-arterial embolization (TAE) with glue (cyanoacrylate). It is the author’s opinion that nutrient artery ligation should not be performed for AVMs as there are numerous contributing vessels and this limits access to the AVM in the future if necessary. Ligation is a reasonable option for AVFs although this form of vascular anomaly is exceedingly rare in the animal liver and embolization would likely be much easier. The latter two treatments are both reasonable options for discussion with the owner. As the majority of HAVMS are located in the right and central divisions of the liver (often surrounding or involving the gall bladder or caudal vena cava), and approximately 25% involve more than one lobe, surgery can be challenging. In addition, keep in mind the entire portal circulation is arterialized so there are no “minor bleeds” from the omentum or mesentery. When surgery is performed, temporary vascular occlusion has been recommended but the author has found that the cranial mesenteric artery needs to be temporarily attenuated along with both the celiac...
artery and portal vein to limit otherwise substantial intra-operative hemorrhage that has lead to a reported 39% intra-operative hemorrhage occurrence. Other reported surgical complications besides hemorrhage include portal hypertension, systemic hypotension, bradycardia, and portal or mesenteric vein thrombus formation. In dogs undergoing surgical therapy alone, peri-operative survival rates were 77% and long-term outcome was fair or good for 38% to 57%. Overall 75% of dogs continue to require dietary or medical management of clinical signs due to patent acquired EHPSS that will always persist. Transcatheter therapies typically involve cyanoacrylate glue (or more recently “Onyx”, (ethylene-co-vinyl alcohol [(EVOH] in dimethyl sulfoxide [DMSO]) a slower polymerizing agent. While sclerosants such as alcohol and particles such as PVA or microspheres have been described, these are not less typically used for high-flow lesions such AVMs.

The observations below have been made following surgery or glue embolization of approximately 20 HAVMs.

Lessons learned or questions raised concerning DIAGNOSTICS include:

- These animals often present for clinical signs associated with PSS and are often mistaken for IHPSS due to the ultrasonographic identification of a large intrahepatic vascular structure along with clinical signs consistent with PSS.
- While all of these animals have SEVERE PORTAL HYPERTENSION, about 25% of these cases will not present with ascites. This is presumably due to the multiple extrahepatic PSS that are acquired (along with other physiologic changes taking place) and have decompressed the portal system sufficiently to relieve the excessive hydrostatic pressure.
- Clinical signs are often less severe with HAVM than standard PSS presumably because the portal system is more developed in the former patients than in the latter ones.
- Other diagnostic signs that are less commonly discussed include hepatofugal portal blood flow (ALWAYS PRESENT), a reduction in aortic diameter caudal to the level of the celiac artery, and differing systemic blood pressures obtained from the forelimbs (higher) and the hindlimbs (lower) that is occasionally present. Identifiable abdominal bruit is rarely present in the author’s experience.

Lessons learned or questions raised concerning TREATMENT include:

- It has been suggested in the human literature that while AVFs can be ligated or coil embolized, multiple AVFs or AVMs should receive glue embolization (or alcohol ablation) in order to destroy the nidus that will otherwise recruit additional vessels over time. This has not been confirmed in the veterinary population but angiograms shown may support this notion.
- Initial angiography (and cross sectional imaging) often underestimates the extent of the disease. Following initial embolization, additional previously unidentified contributing vessels open up demonstrating the true infiltrative nature of these vascular anomalies.
- Complete HAVM embolization or resection appears to be required to prevent recurrence. Incomplete embolization or resection will lead to revascularization if the HAVM nidus remains.
- These patients can tolerate complete hepatic artery embolization and the cyanoacrylate glue appears to be permanent in the cases that have been followed to date, although the radio-opacity of the glue (Tantalum and Lipiodol) may diminish over time.
- Although return to hepatopetal portal bloodflow (towards the liver) would be considered the goal, this has not been seen in any of the cases treated to date. Acquired EHPSS are present in ALL patients and can be expected to remain in place providing the least resistance to portal bloodflow. Complete HAVM embolization could conceivable result in stagnant portal bloodflow as the direction changes from hepatofugal to hepatopetal and near complete stasis has been identified but to date has not required intervention.
- Vascular contributions to the HAVM are not only from the hepatic artery but have also been identified to arise from the gastroduodenal artery, left gastric artery, and phrenic aa. Performing an aortogram following embolization is recommended.
- More recently, the author has been investigating venous outflow occlusion/embolization for HAVMs. In humans, this approach has been considered the greatest advance in management of large AVMs in the past 5-10 years. It is too soon to know if this will be effective in HAVMS in veterinary patients but initial results are promising. Cases will be presented using this approach.

Lessons learned or questions raised concerning FOLLOW-UP include:
- As acquired EHPSS will never close, continued life-long medical therapy is often necessary (and indicated) in patients following treatment. Some animals may not need medical therapy however. It is unclear how venous embolization will change patient followup.

- It is unclear which animals may benefit from treatment. In certain cases with no overt clinical signs (for instance the cases without failure to thrive and massive ascites), these patients may not benefit from the treatment of the anomalies. Do all animals with HAVM require treatment? It has been suggested that chronic portal hypertension can lead to portal vein blunting and reduce portal perfusion. If so, is this an argument that all these animals should be treated as soon as possible?

References available upon request