Interventional cardiovascular procedures: overview and veterinary applications

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Interventional cardiovascular procedures are nowadays readily available for the correction or palliation of congenital and acquired cardiovascular defects. This involves the passing of catheters via peripheral vessels, such as the femoral artery or the jugular vein, under general anesthesia. Access to the vessels involves a direct surgical dissection for arteries, although in some circumstances venous access using a Seldinger technique can also be used. Vascular access sheaths with a hemostatic valve are used to enter the vessels, allowing repeated and easy passage of various guide wires and catheters, with minimal blood loss.

Manipulation and directional control of the catheters and guide wires is observed under fluoroscopy with image intensification. The image on the monitor is typically magnified, thus accurate scaling is necessary. Scaling is achieved by measurement markings on a catheter placed within the esophagus, the heart or great vessels and the exact magnification measured at the commencement of the intervention. An angiographic pressure injector is required for rapid delivery of radiographic contrast and a blood pressure monitor for direct manometry.

The common conditions which nowadays are commonly managed by interventional techniques are (in the lecture I will use a case-based approach to discuss these indications) :

- Patent ductus arteriosus (PDA): Commonly closed using an Amplatz canine duct occluder (ACDO) in patients over 4 kg, or with a coil in cats and smaller dogs (catheterization of the femoral artery in a tiny puppy is not possible because the catheter needed to deliver the ACDO device typically exceeds the diameter of the lumen of the artery)
- Severe valvular pulmonic stenosis (PS): Using balloon dilation catheters the valvular stenosis can be reduced in severity in the majority of dogs with pure valvular stenosis
- Cor triatriatum dexter: This rare congenital heart diseases causes right-sided heart failure (ascites) due to a persistent membrane in the right atrium. Using balloon dilatation catheters (sometimes using a combination of cutting balloon and high-pressure) the obstruction can be relieved.
- Pacemaker implantation is now a well-recognized procedure for the management of various bradyarrhythmias causing symptoms, such as syncope, or for the risk of death such as third-degree AV block, advanced/symptomatic second-degree heart block (intermittent heart block), sinus arrest (often part of sick sinus syndrome), vasovagal syncope when the bradycardia portion is profound. Modern pacemakers are typically rate responsive, allowing an increase in the heart rate with exercise.
- Electrophysiological studies and ablation of tachyarrhythmias caused by congenital electrical bypass tracts is now available with increasing success rates
- Intra-hepatic portosystemic shunts (PSS): Numerous surgical techniques have been described for intrahepatic PSS attenuation, ranging from careful liver dissection around the shunting vessel to more technically demanding and complicated procedures involving temporary vascular hepatic inflow occlusion for intravascular repair, all with an unacceptable high mortality rate. Nowadays interventional techniques for intrahepatic PSS are available, using stenting of the caudal vena cava and placement of thrombogenic coils; these procedures are safe, successful, and improve the clinical situation of most affected dogs.

Pulmonic Stenosis (PS)

Pulmonic stenosis is characterized by congenital malformation of the pulmonic valves and sometimes hypoplasia of the the pulmonic annulus region, resulting in obstruction to right ventricular outflow, pressure overload and concentric hypertrophy of the right ventricle. Left uncorrected, syncope, right-sided congestive heart failure (ascites, pleural effusion) and arrhythmias with risk of sudden cardiac death can occur. Especially in Bulldogs pulmonic stenosis may be accompanied by coronary artery anomalies. Pulmonic stenosis produces a systolic left base murmur that must be differentiated from aortic/subaortic stenosis. Femoral pulses are often normal in PS whereas they are often weak with severe AS/SAS. Thoracic radiography may reveal right-sided cardiomegaly and a distinct main pulmonary artery bulge on the DV view. Echocardiography is necessary to make the diagnosis, rule out other concurrent cardiac defects, and assess severity and suitability for intervention.

Aortic/Subaortic Stenosis (AS/SAS)

The majority of dogs with AS have a ridge of fibrous tissue below the aortic valve in the LV outflow tract, therefore subaortic in nature (SAS). This causes obstruction to left ventricular outflow, pressure overload and concentric hypertrophy of the left ventricle. Exercise intolerance, syncope, left-sided congestive heart failure (pulmonary edema) and arrhythmias with risk of sudden cardiac death are potential consequences, with sudden death being the most common outcome, and may occur at a young age in severe cases. Importantly, the lesions may not be present at birth but develop during the first 4–8 weeks of life. Therefore, it is not uncommon to not be able to hear a murmur at the first puppy check. SAS produces a systolic left base murmur that must be differentiated from PS. Femoral pulses are often weak. Severity of SAS increases with growth and maturity therefore murmur intensity increases during puppyhood. Thoracic radiography may reveal a prominent ascending aorta/aortic arch bulge and left ventricular enlargement, though concentric hypertrophy often does not produce gross cardiomegaly radiographically to the same extent that cardiac dilation does. Echocardiography is necessary to make the diagnosis, assess severity and candidacy for any therapy, and rule out other concurrent cardiac defects. Even with echocardiography, diagnosis of mild SAS can be very challenging.

Atrial and Ventricular Septal Defects

Defects in the development of the embryonic ventricular septum, atrial septa, or endocardial cushions may result in atrial septal defects (ASD) and/or ventricular septal defects (VSD). When the degree of left-to-right shunting is substantive, ASD results in volume overload of the right atrium, the right ventricle, and pulmonary tree, whereas VSD results in volume overload of the left side of the heart and pulmonary tree (the right ventricle just acts as a passive conduit). Atrial septal defects can be challenging to detect on physical exam since they may produce no murmur due to low flow velocity, or a soft murmur similar to that of mild PS or an innocent murmur. A split second heart sound can sometimes be detected. Small VSDs produce very intense murmurs, typically loudest on the right, whereas larger VSDs may be associated with much softer murmurs.

Atrioventricular valvular dysplasia

Dysplasia of either of the atrioventricular valves (mitral valve, tricuspid valve) can occur in both the dog and cat. It occurs when malformation of either valve leads to stenosis, and/or regurgitation. Mitral valve dysplasia can also cause a dynamic obstruction of the left ventricular outflow tract, especially in kittens. This leads to dilatation of the atria, which in turn, can lead to heart failure and arrhythmias. Clinical signs with mitral valve (MV) dysplasia include a left sided heart murmur, syncope (often associated with tachyarrhythmias), and left sided congestive heart failure. Thoracic radiography may show evidence of left atrial and left ventricular enlargement, and if present, pulmonary congestion and edema. Echocardiography is useful to assess the severity of valvular malformation, dynamic obstruction, regurgitation, stenosis (if present) and chamber enlargement. Treatment is usually limited to managing heart failure and arrhythmias. Once these occur, prognosis becomes guarded. Clinical signs with tricuspid valve (TV) dysplasia are similar except that a right sided heart murmur and right sided heart failure can occur.