

STATE OF THE ART LECTURE



CARDIAC DISEASE IN HUMANS AND ANIMALS: MERGING RESEARCH AND CLINICS TO BENEFIT BOTH

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Cardiac surgery includes procedures performed on the pericardium, cardiac ventricles, atria, venae cavae, aorta, and main pulmonary artery. Closed cardiac procedures (i.e., those that do not require opening major cardiac structures) are most commonly performed; however, some conditions require open cardiac surgery (i.e., a major cardiac structure must be opened to accomplish the repair). Open cardiac surgery necessitates that circulation be arrested during the procedure by inflow occlusion or cardiopulmonary bypass

Inflow Occlusion

Inflow occlusion is a technique used for open heart surgery where all venous flow to the heart is temporarily interrupted. Because inflow occlusion results in complete circulatory arrest, it allows limited time to perform cardiac procedures. Ideally, circulatory arrest in a normothermic patient should be less than 2 minutes, but can be extended to 4 minutes if necessary. Circulatory arrest time can be extended up to 6 minutes with mild, whole-body hypothermia (32° to 34° C). Temperatures below 32° C may predispose to fibrillation and should be avoided. The advantage of inflow occlusion is that it does not require specialized equipment; however, the limited time available to perform the surgery requires that the procedure be well planned and executed with speed and expertise. We have used this technique primarily for right atrial tumors and cor-triatrarium dexter.

Cardiopulmonary Bypass

Cardiopulmonary bypass is a procedure whereby an extracorporeal system provides flow of oxygenated blood to the patient while blood is diverted away from the heart and lungs. This greatly extends the time available for open cardiac

surgery. Several advances (i.e., development of membrane oxygenators, improved methods of myocardial protection, increased availability of monitoring technologies, and improved veterinary critical care) have made cardiopulmonary bypass increasingly feasible in dogs. Cardiopulmonary bypass can be used to treat dogs with congenital or acquired cardiac defects. Readers are referred to a cardiovascular surgery text for details of performing cardiopulmonary bypass.

SUB-AORTIC STENOSIS

Surgical treatment of sub-aortic stenosis (SAS) in dogs has been successful in the short term in reducing the systolic pressure gradient across the aortic valve, but has not been shown to decrease the incidence of sudden death in this population. Reports of closed transventricular dilation showed marked post-operative decreases in pressure gradients, but restenosis is common, usually within three months. This restenosis is consistent with reports in the human literature following transventricular dilation. The most promising results thus far are found in techniques investigating the use of cardiopulmonary bypass and open surgical correction.

To date, 3 dogs with subaortic stenosis has undergone cardiopulmonary bypass and open-heart correction of this defect at Texas A&M University. These patients had severe SAS with a Doppler-derived gradient in excess of 200 mmHg and moderate to severe left ventricular hypertrophy without significant ventricular ectopy or mitral regurgitation. Through a median sternotomy, a right ventriculotomy was performed in 2. An initial incision into the hypertrophied septum allowed exploration of the left ventricular outflow tract (LVOT). An aortotomy was also performed to improve visualization of the LVOT and aortic

valve. A large portion (1.5 x 2 cm) of the dorsal septum was removed and the subvalvular fibrous tissue resected without damage to the mitral valve. The septal defect was repaired with autologous pericardium harvested at surgery and treated with glutaraldehyde to improve its handling characteristics. Full thickness resection was performed in an attempt to alleviate the late restenosis noted with alternate partial thickness resection techniques. One dog survived long-term

PULMONIC STENOSIS

Although supra and subvalvular lesions have been seen, the most common cause of pulmonic stenosis in dogs is valvular dysplasia. Dogs with moderate to severe stenosis may experience syncope or changes leading to congestive heart failure and are at risk for sudden death. Surgery or balloon valvuloplasty should be considered if the pressure gradient is above 80 mmHg. Valvuloplasty may be beneficial for primarily valvular lesions, but its efficacy may be reduced in those cases with significant subvalvular muscular hypertrophy. Restenosis, presumably due to scarring, has been reported.

Alternatively a patch graft technique, using PTFE or Gortex material, may be more likely to provide a greater and longer standing reduction in the pressure gradient, although survival data have not been previously evaluated. Patch grafting techniques may be performed under inflow occlusion and mild hypothermia; however, the use of cardiopulmonary bypass affords the surgeon more time for precise placement of the graft and thus may allow for improved post-operative outcomes.

Dogs with an aberrant coronary artery contributing to their pulmonic stenosis are not considered candidates for balloon valvuloplasty or patch grafting techniques due to the risk of disturbance of that coronary vessel. Surgery in these animals would generally require cardiopulmonary bypass and placement of a conduit from the right ventricle to the pulmonary artery to circumvent the stenosis.

MITRAL VALVE DISEASE

Despite mitral valve disease (MVD) being the most common cause of heart failure in dogs, no medical therapy has yet been identified that will delay or alter the progression of this disease. Valve repair or replacement has become the standard of care in human patients with chronic degenerative valve disease. If possible, valve repair is considered preferable to replacement as it eliminates the need for anti-coagulative therapy post-operatively and is less expensive. Depending

on the stage of disease, a variety of repair techniques are available to improve the dynamics of the valve. Ruptured chordae may be repaired with synthetic (Gortex) sutures to re-establish normal motion of the valve leaflets; an Alferari procedure ("bow-tie" or procedure in which a suture is placed between the anterior and posterior valve leaflets) can decrease the regurgitant area and provide support for leaflets and chordae. An annuloplasty is generally required and involves placement of a synthetic ring or sutures to reduce the size of the dilated mitral annulus. Once systolic function has deteriorated to the point that continued inotrope support (other than digoxin) is essential, mitral valve repair bypass surgery becomes substantially more risky.

Other congenital defects that may be amenable to definitive surgical repair

Ventricular septal defect (VSD) is the second most common congenital heart defect in cats and accounts for 5% to 10% of congenital heart defects seen in dogs. Most ventricular septal defects in small animals occur in the membranous septum. Perimembranous defects are located in the membranous septum, medial to the septal tricuspid leaflet, and inferior to the crista supraventricularis. Infundibular or supracristal defects are located in the right outflow tract superior to the crista supraventricularis. The pathophysiology of VSD depends on the size of the defect and on pulmonary vascular resistance. VSD typically causes a left-to-right shunt. A typical VSD overloads the left heart and, depending on its size and location, may overload the right heart as well. A large VSD can progress to left-sided congestive heart failure. Chronic overcirculation of the lungs can cause progressive pulmonary vascular remodeling leading to severe pulmonary hypertension and right-to-left shunting of blood (Eisenmenger's physiology). Aortic insufficiency is a fairly common secondary abnormality associated with VSD, particularly infundibular VSD. Aortic insufficiency results from prolapse of an aortic leaflet into the defect. This prolapse is due to the Venturi effect associated with VSD flow and loss of support of the aortic annulus. Aortic insufficiency adds to the left ventricular volume overload and is usually progressive.

Definitive patch closure of VSD can be accomplished with the aid of cardiopulmonary bypass in dogs over 4 kg in body weight. A perimembranous VSD is corrected from the right side via a right atriotomy approach. An infundibular VSD is corrected via a right ventriculotomy from a left thoracotomy or median sternotomy approach.

Tetralogy of Fallot (T of F) is the most common

congenital heart defect that causes cyanosis in small animals. It occurs in cats and a variety of canine breeds. Tetralogy of Fallot can be simplified into two physiologically significant defects: pulmonic stenosis and ventricular septal defect (VSD). The pathophysiologic consequences of tetralogy depend on the relative magnitude of these two defects. If a large VSD and hemodynamically insignificant pulmonic stenosis are present, the functional result is a left-to-right shunt and volume overload of the left heart similar to an isolated, large VSD. If severe pulmonic stenosis, suprasystemic right heart pressures, and right-to-left shunt are present, the result is moderate to severe cyanosis, exercise intolerance, and progressive polycythemia. A shortened life span is expected in these animals due to complications of hyperviscosity-induced thromboembolism or sudden death. Animals that have pulmonic stenosis and VSD that are somewhat balanced are functionally similar to those that have a VSD and pulmonary artery banding performed. Animals with predominantly left-to-right shunt are termed acyanotic tetralogy and may function reasonably

well as long as the shunt flow is insufficient to cause left heart failure. Progression of pulmonic stenosis due to infundibular hypertrophy is possible and may cause acyanotic animals to become cyanotic as they age. Surgery should be considered for severely cyanotic animals to lessen clinical signs and prolong life. Animals with a resting arterial oxygen saturation less than 70% should be considered candidates for surgery. Palliative surgeries for tetralogy include isolated correction of the pulmonic stenosis or creation of a systemic-to-pulmonary shunt (e.g., Blalock-Taussig shunt). Correction of the pulmonic stenosis risks overcorrection of the stenosis and an overwhelming left-to-right shunt. For this reason, valve dilation, either surgically or by balloon dilation, is preferred over a more definitive procedure such as a patch-graft. Definitive repair of tetralogy can be undertaken in medium- to large-breed dogs with cardiopulmonary bypass. Patch closure of the VSD and patch-grafting of the pulmonary outflow tract are undertaken through a right ventriculotomy approach.