Cardiac Surgery in Veterinary Medicine: Where are we and what does the future hold?

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INTRODUCTION

Cardiopulmonary bypass and inflow occlusion
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. Venous inflow occlusion provides brief circulatory arrest, allowing short procedures (less than 5 min) to be performed. Longer open cardiac procedures require establishing an extracorporeal circulation by cardiopulmonary bypass to maintain organ perfusion during surgery.

Cardiac surgery is not fundamentally different from other types of general surgery and similar principles of good surgical technique (i.e., atraumatic tissue handling, good hemostasis, and secure knot tying) apply. Consequences of poor surgical technique are often devastating. Cardiac surgery differs from other surgeries in that motion from ventilation and cardiac contractions adds to the technical difficulty of performing these procedures. Approaches that provide limited access to dorsal structures require that surgeons incise, suture, and/or ligate structures located deep within the thorax. Ligature placement using hand ties are useful in such situations and the ability to place hand-tied knots (vs. instrument tying) should be considered a fundamental skill for cardiac surgeons. Secure knot tying is critically important to successful cardiac surgery. Hand tying knots is fast and produces tighter and more secure knots than instrument tying. The one-handed knot tie technique is best suited to the fine sutures used in cardiac surgery. Tight knots are facilitated by throwing the first two or three throws in the same direction before finishing with square knots for security.

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-triatriatum dexter.

Depending on the cardiac procedure being done, a left or right thoracotomy or median sternotomy is performed. With a right thoracotomy or median sternotomy, the cranial and caudal vena cava and azygous vein are occluded with vascular clamps or Rumel tourniquets which can be made by passing umbilical tape around the vessel, then threading umbilical tape through a piece of rubber tubing that is 1 to 3 inches long. When the umbilical tape has been adequately tightened to occlude the vessel, a clamp is placed above the rubber tubing to hold it securely in place. Care must be taken to avoid injuring the right phrenic nerve during placement of the clamps or tourniquets. For left thoracotomies, separate tourniquets are passed around the cranial and caudal vena cavae. Then, dissecting dorsal to the esophagus and aorta, the azygous vein is occluded by placing a tourniquet around it.
**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.

**Preoperative concerns**

Animals requiring cardiac surgery often have prior cardiovascular compromise that should be stabilized medically when possible, prior to anesthetic induction. Congestive heart failure, particularly pulmonary edema, should be managed with diuretics (e.g., furosemide) and ACE inhibitors (e.g., enalapril, lisinopril) before surgery. Cardiac arrhythmias should be recognized and treated (see also postoperative care below). Ventricular tachycardia should be suppressed before surgery with class I antiarrhythmic drugs (i.e., lidocaine, procainamide). Lidocaine is effective for management of ventricular tachyarrhythmias during and immediately after surgery. Supraventricular tachycardia may require management with digoxin, beta-adrenergic blockers (e.g., esmolol, atenolol), or calcium channel blocking drugs (e.g., diltiazem) prior to surgery. Atrial fibrillation should be controlled prior to surgery with digoxin to lower the ventricular response rate below 140 bpm. This may require the addition of beta-adrenergic blockade or calcium channel blocking drugs if digoxin alone does not decrease the ventricular rate sufficiently. Animals with bradycardia should undergo an atropine response test before surgery. If bradycardia is not responsive to atropine, temporary transvenous pacing or may be required.

Most animals should undergo evaluation by echocardiography prior to cardiac surgery as an incomplete or inaccurate diagnosis can have devastating consequences. With the advent of Doppler echocardiography, cardiac catheterization is no longer routinely necessary prior to cardiac surgery.

**SUB-AORTIC STENOSIS**

To date, 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. The traditional approach to resection of the subvalvular stenosis is through an aortotomy made above the coronary ostia. Tearing of the aortic incision during resection is a potential complication of this approach. The typical defect is a discrete fibrous membrane located 1-5 mm below the base of the cusps of the aortic valve that reflects onto the septal cusp of the mitral valve from the septum. This portion of the ring must be excised to adequately reduce the pressure gradient but care must be taken not to damage the mitral valve during the resection. Varying degrees of muscular hypertrophy of the interventricular septum accompany this lesion. Removal of partial thickness sections of hypertrophied septum (septal myectomy) has been reported in the veterinary literature but a significant difference in survival using this technique was not demonstrated. Due to the continued problem of late recurrence of stenosis, alternate techniques are used in people to resect full thickness portions of septum and reconstruct the septal defect with a patch graft. The proximity of the conduction system is the primary concern when performing a septal myectomy.

To date, one dog with subaortic stenosis has undergone cardiopulmonary bypass and open-heart correction of this defect at Texas A&M University. The patient 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. 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. Although not substantiated in dogs, it is hoped that this will, at a minimum, delay the progression of disease and decrease the chance of sudden death.

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 anticoagulative 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.

Dogs that are considered good candidates for mitral valve surgery at our hospital should meet the following criteria:

- There should be no significant concurrent disease such as end stage renal disease, heartworm disease, hepatic failure, metastatic neoplasia, septicemia, etc.
- The patient should have a confirmed echocardiographic diagnosis of chronic degenerative valve disease (CVD) without presence of concurrent severe congenital disease such as sub-aortic stenosis, pulmonic stenosis and ventricular septal defects. There must be a history of cardiogenic pulmonary edema (congestive heart failure) with a radiographic response to diuretic therapy and the dog must require maintenance diuretic therapy.
- Indirect systolic blood pressure measurement by Doppler or Dynamap should be greater than 100 mmHg when the animal is awake and has been stabilized.
- Left ventricular systolic function should be preserved. Systolic function is considered to be within normal limits in dogs with CVD if the internal dimension of the left ventricle during systole (LVIDs) is within normal limits for that patient. This measurement is part of a routine 2-D guided m-mode echocardiogram. The following formula can be used as a guide 0.69 BW (kg) 0.41 = mean normal LVIDs (cm)
- The dog must not require continued inotropic support (except digoxin).
Presurgical screening evaluations that we require be performed within the previous 30 days:

- Complete blood count including platelet count
- Biochemical profile
- Canine blood typing
- Thoracic radiographs
- Indirect blood pressure
- Abdominal ultrasound
- Echocardiogram

Digital or VHS video of the echocardiogram should be provided for evaluation and should include standard 2-D imaging planes from the right and left parasternal positions as well as m-mode measurements of the left ventricle and left atrium/aorta.

**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 (see below under signalment). 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.