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Aortic stenosis in dogs: A brief review

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Abstract:

Congenital heart diseases are more prevalent in purebred dogs. The prevalence rate of congenital cardiac diseases have been reported to about 2.8 percent. The three most common congenital heart defects in canine patients are aortic stenosis (AS), pulmonary stenosis (PS) and patent ductus arteriosus (PDA). Congenital aortic stenosis can be of supravalvular, valvular or subvalvular type. Out of the three, subvalvular form is most common accounting for more than 95 percent of identified cases of aortic stenosis. In dogs this defect occurs mainly in Newfoundlands, Boxers, Golden Retrievers, Rottweilers, German Shepherds, Samoyeds and Dogue de Bordeaux breeds. The prevalence of congenital heart disease in boxers is very high i.e. 17.8% with subaortic stenosis being the most frequent finding with an incidence of 89.9% of all congenital defects. For diagnosis, evaluation of heart is performed via assessment of heart sounds and murmurs, arterial pulses, degree of jugular vein distention, strength and location of apex beat. Initial non-invasive testing usually includes thoracic radiographs, an electrocardiogram (ECG), cardiac biomarkers (NT-proBNP, BNP, ANP, troponin I) and echocardiographic studies. Medicinal therapy with a β -blocker is advocated in patients with moderate to severe subaortic stenosis. Several palliative surgical techniques have been used in dogs with severe subaortic stenosis. Cardiopulmonary bypass and open-heart surgery are necessary to reach the lesion directly. The prognosis in dogs with mild or moderate stenosis is fair while that of severe stenosis is guarded.

Keywords: congenital heart defects, aortic stenosis, subvalvular stenosis, dogs

Introduction

Congenital heart defects most often involve either a valve or an abnormal communication between the systemic and pulmonary circulations. Abnormally formed valves can have insufficiency, stenosis or both. Other malformations can exist and multiple anomalies occur in some patients.

Congenital heart diseases are more prevalent in purebred dogs. The prevalence rate of congenital cardiac diseases have been reported to about 2.8% (Brambilla *et al.*, 2003) [1]. The three most common congenital heart defects in canine patients include aortic stenosis (AS), pulmonic stenosis (PS) and patent ductus arteriosus (PDA). The lesser common defects include ventricular septal defects (VSD), mitral valve dysplasia (MD), tricuspid valve dysplasia (TD) and tetralogy of Fallot (TF). Most (60%) congenital heart defects are recognized in dogs older than one year of age as dogs less than one year of age remain asymptomatic (Garncarzet *et al.*, 2017) [2].

Aortic stenosis is defined as a congenital obstructive malformation of the heart with narrowing of the aortic valve, aorta, or left ventricular outflow tract. It has been reported in the dog, cat, cow, sheep, horse, pig and man. Congenital aortic stenosis can be of supravalvular, valvular or subvalvular type. Out of the three, subvalvular form is most common accounting for more than 95% of identified cases of aortic stenosis. Subvalvular aortic stenosis (SAS) is characterized by a subvalvular obstruction, typically a ring or ridge of fibrous or fibromuscular tissue immediately below the aortic valve, that narrows the left ventricular outflow tract. Consequences of SAS include left ventricular hypertrophy, myocardial ischemia and cardiac arrhythmias (Meurset *et al.*, 2005) [3]. In dogs this defect occurs mainly in Newfoundlands, Boxers, Golden Retrievers, Rottweilers, German Shepherds, Samoyeds and Dogue de Bordeaux breeds. The disorder is inherited as a dominant trait with variable penetrance in Newfoundland dogs (O'Grady *et al.*, 1989) [4]. The prevalence of congenital heart disease in boxers is very high i.e. 17.8% with subaortic stenosis being the most frequent finding with an incidence of 89.9% of all congenital defects (Bussadori *et al.*, 2001) [5].

For diagnosis, evaluation of heart is performed via assessment of heart sounds and murmurs, arterial pulses, degree of jugular vein distention, strength and location of apex beat. Initial noninvasive testing usually includes thoracic radiographs, an electrocardiogram (ECG), cardiac biomarkers (NT-proBNP, BNP, ANP, troponin I) and echocardiographic studies (Nowak, 2011) [6].

Echocardiography is excellent to confirm severity of valvular and stenotic lesions, evaluate chamber enlargement and quantify systolic and diastolic myocardial function. Cardiac catheterization with selective angiography can also be useful. The patient's prognosis and options for therapy depend on the definitive diagnosis as well as severity.

Most cases of aortic stenosis represent mild lesions, are detected as incidental findings and result in no loss of longevity or quality of life. Moderate or severe stenosis may result in exertional weakness, syncope or sudden death. Signs of congestive heart failure are rare unless mitral valve insufficiency is present. Cardiac catheterization and intracardiac pressure measurements, including the systolic gradient across the stenosis, provide an indication of the severity of the disease and thus the need for surgical correction. A gradient of less than 50 mm Hg suggests a minor lesion which does not require correction. Gradients of greater than 80 to 100 mm Hg indicate severe stenosis and the necessity for surgical correction. Without surgery, most dogs with gradients of greater than 100 mm Hg die within the first two years of life.

Several palliative surgical techniques have been used in dogs with severe subaortic stenosis. Cardiopulmonary bypass and open-heart surgery are necessary to reach the lesion directly. Resection of the stenotic area can significantly reduce the left ventricular systolic pressure gradient and improve exercise ability. Transvascular balloon dilation of the stenotic area reduces the measured gradient in some dogs, although narrowing may partially reoccur. Medical therapy with a β -blocker is advocated in patients with moderate to severe subaortic stenosis to reduce myocardial oxygen demand and minimize the frequency and severity of arrhythmias. Exercise restriction is advised for animals with moderate to severe subaortic stenosis. Prophylactic antibiotic therapy is recommended for animals with subaortic stenosis before the performance of any procedures with the potential to cause bacteremia.

The prognosis in dogs with mild or moderate stenosis is fair while that of severe stenosis is guarded. More than half of dogs with severe SAS die suddenly within their first three years. The overall prevalence of sudden death in dogs with SAS appears to be just over 20%. Infective endocarditis and chronic heart failure may be more likely to develop after three years of age. Atrial and ventricular arrhythmias and worsened mitral regurgitation are complicating factors. Dogs with mild stenosis are more likely to survive longer and without clinical signs (Nelson and Couto, 2009) [7].

Incidence

According to information disseminated by the American Veterinary Medical Association, one in ten dogs suffer from heart disease (Dove, 2001) [8]. The overall prevalence of cardiac diseases in the dog is around 4.4% (Manczur *et al.*, 2003). As per a study conducted by Kumar (2012) the highest incidence of cardiovascular abnormality in India were found in Pomeranian/ spitz (47.56%), followed by Labrador (24.39%), German shepherd (8.53%), Doberman (4.87%),

Great Dane (2.43%), Pug (3.65%), Non-descript (3.65%), Rampur hound (1.21%), Bhutia (1.21%) and Terrier (1.21%). The incidence of cardiovascular diseases in this study was found highest in 01-03 years of age group (23.17%), followed by 03-05 years (20.73%), 5-7 years 21.95% and >10 years (9.75). The lowest incidence was recorded in 00-01 year age group (8.53%) for cardiovascular abnormalities. The cardiovascular diseases were found more common in male (54.89%) than female (45.12%).

Diagnosis

Clinical signs

Dogs with SAS may be asymptomatic or may exhibit following clinical signs-

- Fatigue, exercise intolerance or exertional weakness due to reduced skeletal muscle perfusion and inadequate blood supply to the tissues.
- Collapse or syncope due to reduced cerebral perfusion
- Difficulty in breathing (dyspnea), rapid breathing (tachypnea) and nocturnal cough due to pulmonary edema or pleural effusion
- Pallor, cold extremities and prolonged capillary refill time due to inadequate blood supply.

Physical Examination Findings

- The predominant physical finding in animals with subaortic stenosis is a systolic ejection murmur, heard best at the left heart base during auscultation. This murmur often radiates equally or more loudly to the right heart base because of the orientation of the aortic arch. The intensity of the murmur correlates fairly well with the degree of stenosis and may increase as animal matures, reflecting progressive stenosis.

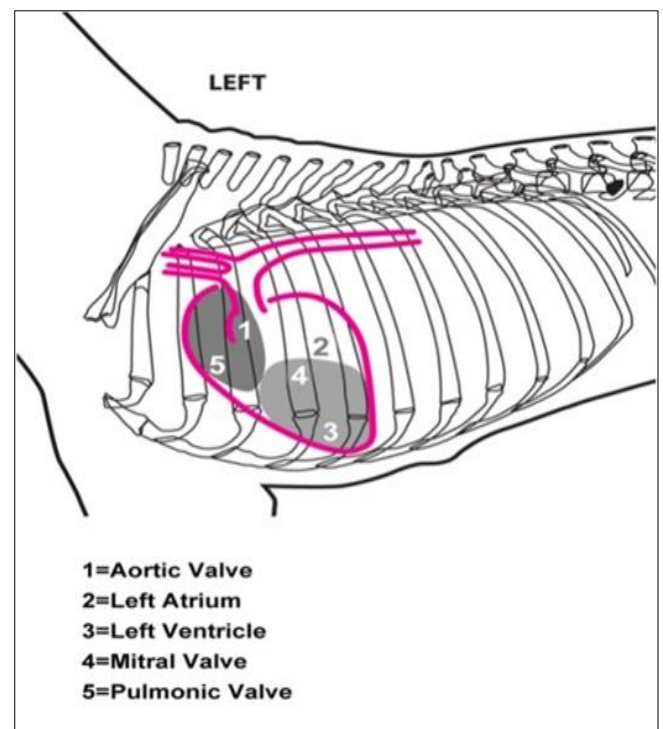


Fig 1: Areas of auscultation

- Muscular weakness
- In moderate to severe cases femoral pulse strength is diminished as the cardiac output falls. Femoral pulse is noticeably weak and late rising or hypokinetic.

- In severe cases most animals die suddenly due to severe ventricular arrhythmias and left sided heart failure. Clinical signs of left sided heart failure include:
 - ✓ Pulmonary crackles
 - ✓ Tachypnea
 - ✓ Mitral regurgitation
 - ✓ Cyanosis caused by ventilation-perfusion inequality

Electrocardiograms

Mild aortic stenosis is unlikely to cause echocardiographic abnormalities. They can often identify moderate to severe atrial or ventricular enlargement or conduction disturbances that may indicate the presence of moderate to severe aortic

stenosis. Severe lesions can result in increases in R-wave amplitude and QRS duration indicating left ventricular enlargement, depression of ST segment indicating myocardial hypoxia and arrhythmias.

Diagnostic Imaging Radiography (X-Ray)

It is used to determine if the heart is enlarged (particularly the left atrium and left ventricle), if the veins from the lungs to the heart are distended, or if fluid is beginning to develop in the lungs. Thoracic radiographs may reveal a normal cardiac silhouette or mild left ventricular and left atrial enlargement. Enlargement of the ascending aorta frequently is evident.

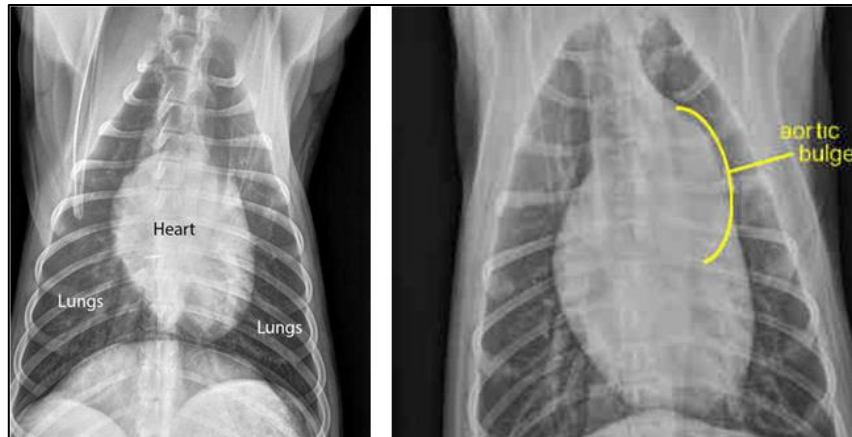


Fig 2: Normal heart versus aortic bulge radiograph

In severe cases where hypertrophy of left ventricle and left atrium occurs, cardiac enlargement can be seen.

Angiocardiography

Angiocardiography is performed by advancing cardiac catheters into specific areas of the heart or great vessels. Left sided specific cardiac catheterization is performed by placing dog in left lateral recumbancy and introducing 6-8 french pigtail catheter into the common carotid artery. This catheter is guided to heart by moving it across the artery and then it is introduced into the left ventricle. Contrast material i.e. 37% solution of diatrizoate meglumine is injected @ 0.5ml/kg body weight into the left ventricle and six thoracic films per second are taken during the injection (Linn and Orton, 1992)^[10]. This technique allows identification of anatomic abnormalities and the path of blood flow. Obstruction to blood flow in the left ventricular outflow tract can be visualized by varying degrees of poststenotic dilatation of the

aorta occur in dogs with congenital subaortic stenosis. These variations are probably the result of differences in the duration, degree, shape or location of obstruction, the extent of pathologic changes in the aortic wall and possibly other factors difficult to measure. Comparative angiocardiographic studies in normal dogs and dogs with subaortic stenosis have shown that relative aortic measurements provide reliable criteria for detecting poststenotic dilatation of the aorta in instances where this is not initially obvious. The most reliable test is the A/S ratio. In a series of normal dogs, the maximum diameter of the ascending aorta (A) distal to the sinus of Valsalva is always less than the diameter of the sinus of Valsalva (S). Therefore, the normal A/S ratio is less than one. In dogs with confirmed, isolated subaortic stenosis, the diameter of the ascending aorta is greater than the diameter of the sinus of Valsalva in one or more angio- cardiograms thus, A/S ratio is greater than one.

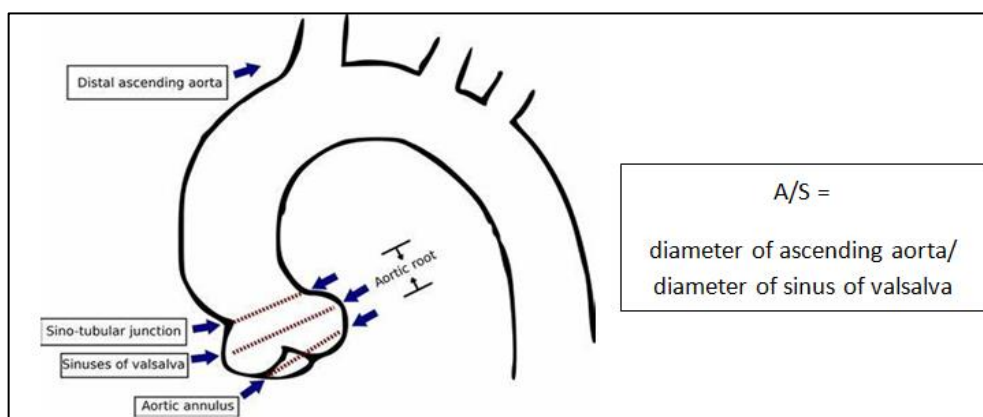


Fig 3: Calculating A/S Ratio

Hence, angiocardiography is useful for -

- delineating the site and geometry of obstruction
- identifying poststenotic dilatation of ascending aorta
- enlargement of left coronary artery and its extramural branches
- a small left ventricular cavity and
- hypertrophy of the papillary muscle and left ventricular wall (Buchanadn and Pattersodn, 1965) ^[11].

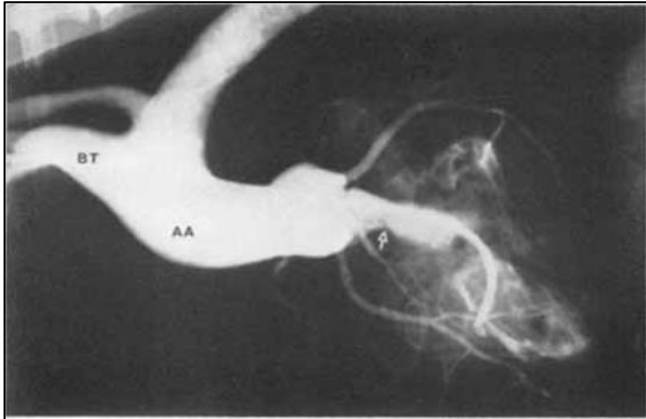


Fig 4: Left ventricular injection angiogram in an eight-month-old male German Shepherd Dog with subaortic stenosis. The area of subaortic stenosis (arrow) can be seen in the outflow tract of the left ventricle. Mild poststenotic dilatation involving the ascending aorta (AA) and the origin of the brachiocephalic trunk (BT) is apparent

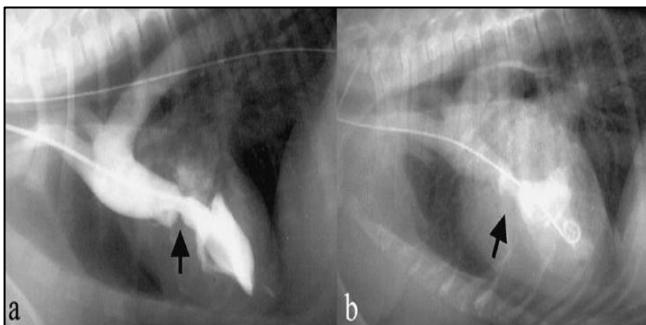


Fig 5: (a) Preoperative left ventricular angiography of a three-month-old golden retriever showing subvalvular stenosis (arrow), post stenotic dilatation of ascending aorta and mild mitral regurgitation
(b) Postoperative left ventricular angiography showing dialation of subvalvularstenotic area (arrow).

Echocardiography (M-mode)

The echocardiogram is the optimal method for identifying and quantifying moderate to severe AS in the dog. The findings that form the basis for diagnosis include:

- a narrowed LVOT
- left ventricular concentric hypertrophy and increased blood flow velocity or pressure gradient within the LVOT.

These features can be identified using two-dimensional, M-mode and Doppler echocardiography (Oyama and Thomas, 2002) ^[13].

Doppler ultrasound

Normal blood flow proceeds in a streamlined way, in the presence of aortic stenosis the flow becomes turbulent and is characterized by fluid whirlpools, stirring in all directions, within the vessel.

Doppler ultrasound allows

- Measurement of velocity,
- Direction of blood flow and
- Nature of blood flow (Oyama and Sisson, 2001) ^[13]

For the diagnosis of aortic stenosis, spectral Doppler technology is coupled to routine echocardiography and allows one to measure the velocity of blood flow across the stenotic orifice. As the stenosis progresses, the velocity of blood flow increases. The modified Bernoulli equation is used as the tool to examine hydrostatic pressure within individual cardiac chambers and is used to quantify disease severity. The peak systolic pressure gradient across the aortic valve can be calculated from the peak aortic velocity. Maximal velocity of flow is measured and can be converted to units of pressure (mmHg) via this equation:

$$\Delta P = 4 V^2$$

ΔP : left ventricular systolic pressure gradient

V: peak aortic velocity or left ventricular outflow tract velocity

Thus, measurement of peak aortic velocity or left ventricular systolic pressure gradient is commonly used as the principal diagnostic means to assess the severity of AS and establish a prognosis. In the normal dog, the mean Doppler-derived velocity across the aortic valve is reported as 1.18 m/s. In mild cases of aortic stenosis, subtle changes in the velocity spectra are detected. These include maximal velocities of between 1.7 and 2.2 m/sec. On the other hand, in cases of moderate to severe obstruction, changes in velocity spectra are dramatic (above 3.5m/s) and maximal Doppler gradients of greater than 50mmHg are considered moderate and above 80mmHg (4.5m/s) are severe (Machado, 2018) ^[14].

Peak systolic pressure gradients less than 5-6mm Hg are considered normal. Systolic gradients of 25 to 50 mmHg are mild, 50 to 75 mmHg are moderate and greater than 75 mmHg are severe when measured in unsedated or unanesthetized animals (Fossum *et al.*, 2013) ^[15].

Two-dimensional echocardiography enables to visualize the internal anatomy of the heart and provides direct visualization of the various morphologic components of the lesion. It can demonstrate variable left ventricular wall and septal thickening, depending on severity.

Dynamic obstruction is indicated by systolic anterior motion (SAM) of the mitral valve and may cause mitral insufficiency. Early closure of the aortic valve also suggests that dynamic obstruction may be present.

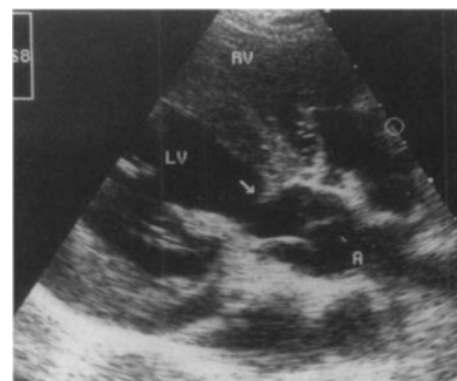


Fig 6: Echocardiogram from a 6-month-old German Shepherd Dog with severe subaortic stenosis. Note the discrete ridge of tissue (arrow) below the aortic valve, creating a fixed outflow tract obstruction. A, Aorta; LV, left ventricle; RV, right ventricle.

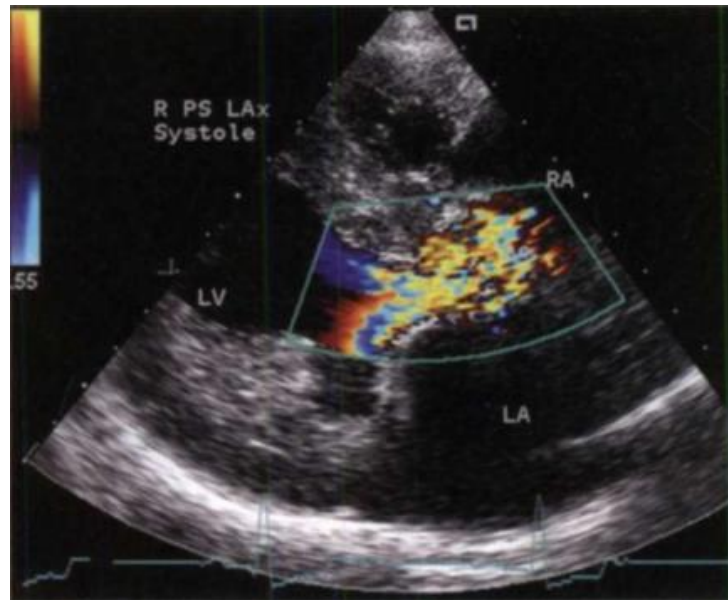


Fig 7: Color flow Doppler frame of the left ventricular outflow region in systole from a 2-year-old female Rottweiler with severe subaortic stenosis. Note the turbulent flow pattern originating below the aortic valve, as well as the thickened septum, papillary muscle and left ventricular free wall. Right parasternal long axis view; Ao, aorta; LA, left atrium; LV, left ventricle; RA, right atrium.

Laboratory Findings

Dogs with SAS may have abnormalities in platelet function and von Willebrand factor multimer distribution. A form of platelet dysfunction detected at high shear rates was associated with mitral regurgitation and SAS in dogs; both are diseases in which turbulent high-velocity blood flows are seen. The clinical significance of this is unknown.

Differential Diagnosis

Aortic stenosis must be differentiated from other conditions that cause systolic murmurs such as:-

- Pulmonary Stenosis
- Ventricular Septal Defect
- Tetralogy of Fallot
- Physiologic (flow/innocent) systolic murmurs commonly detected in large breed dogs.

The differential diagnosis can be done on the basis of radiographic, angiocardiographic and echocardiographic studies.

Medicinal Management

Medical management of dogs with subaortic stenosis is limited to symptomatic treatment of the arrhythmias or heart failure in complicated cases. In the case of congestive heart failure diuretics, low-salt diets and exercise restriction are recommended. β -Adrenergic blockade therapy with propranolol or atenolol may reduce the risk of sudden death by decreasing myocardial oxygen requirements, improving perfusion through a reduction in heart rate and suppressing ventricular arrhythmias during exercise. Symptomatic treatment e.g., furosemide, enalapril for CHF is indicated if it occurs (Meurset *et al.*, 2005)^[3].

Medical Management of Congestive Heart Failure β -Adrenergic blockade therapy

- Propranolol @ 0.33 mg/kg orally every 12hrs initially and gradually can be increased to 1.0 mg/kg every 12hrs.
- Atenolol @ 0.46 to 1.5 mg/kg, PO, every 12 hrs.

Acetyl cholinesterase inhibitors

They reduce pulmonary edema by increasing systemic venous capacitance, lowering pulmonary venous pressure and reducing systemic arterial resistance.

- Benazepril (Lotensin)@0.25-0.5 mg/kg PO every 12-24hr
- Enalapril (Vasotec)@0.25-0.5 mg/kg PO every 12-24hr

Diuretics

Rapid diuresis in cases of pulmonary oedema can be achieved with IV furosemide; effects begin within 5 minutes, peak by 30 minutes and last about 2 hours. This route also provides a mild venodilating effect. Once diuresis has begun and respiration improves, the dosage is reduced to prevent excessive volume contraction or electrolyte depletion

- Furosemide (Lasix)@ 2-4 mg/kg PO, IV, SC every 6-24hr as needed

Positive inotropes

Positive inotropic therapy is indicated when heart failure is caused by poor myocardial contractility. It strengthens the heart contractability so it can pump more blood with fewer heart beats.

- Pimobendan (Vetmedin)@ 0.25-0.3 mg/kg PO every 12hr IV, PO, SC.

Surgical Treatment

Surgical intervention should be considered for dogs with

- Substantial left ventricular hypertrophy and
- Systolic gradients above 75-80mmHg.

If surgery is undertaken, it should be done early to minimize degenerative myocardial changes.

Surgical options for dogs with SAS include

1. Balloon valvuloplasty
2. Transventricular aortic dilation valvuloplasty
3. Open resection- open membranectomy with septalmyectomy (Orton, 2003)

Balloon Vulvoplasty

- A skin incision is made over the right jugular area to expose right carotid artery.
- A hair-wire is inserted into the carotid artery through 18 gauge over-the-needle catheter.
- A 6 Fr introducer sheath of 7 cm length is then inserted to the right carotid artery with guidance of preplaced hair-wire.
- A 200cm long, 0.35 or 0.38mm diameter, J-tipped guide wire with 5 Fr angiographic catheter is then inserted through the introducer and proceeded to left ventricle under C-arm monitoring simultaneously with electrocardiogram monitoring for fatal arrhythmia.
- The obstructive lesion of left ventricular outflow tract is confirmed and balloon dilatation catheter that matches the aortic valvular annulus size is prepared.
- After withdrawing angiographic catheter, the balloon dilatation catheter is then inserted over the guide wire and located at the stenotic left ventricular out flow tract lesion through the introducer.
- Balloon length of 4 or 5 cm is used; balloon diameter is chosen by measuring the diameter of the aortic valve annulus via a 2-dimensional echocardiographic image of the aorta obtained from the right parasternal position. A balloon with a diameter approximately equal to the diameter of the aortic valve annulus is chosen; in most dogs, a 20 or 23mm diameter balloon is used.
- With inflation device, the balloon is inflated until the indentation of balloon disappears and then quickly deflated while monitoring patient's heart rate and blood pressure. This inflation procedures are repeated 5 times until the indentation from stenotic lesion disappears and then all devices are removed.
- The right carotid artery is ligated with 6-0 polypropylene suture in a simple continuous pattern and incision line is sterilized and bandaged.

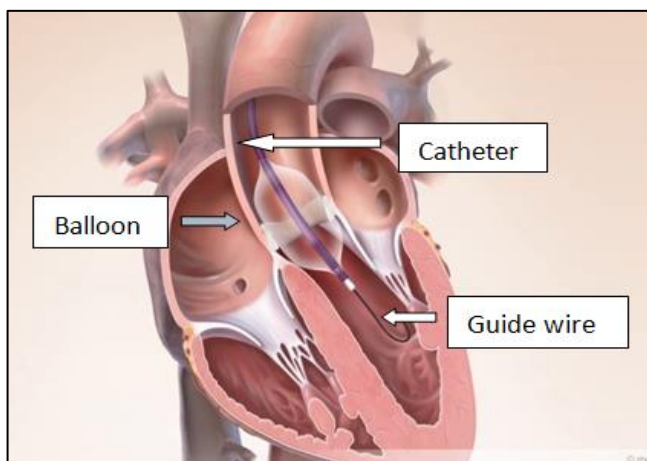


Fig 8: Balloon vulvoplasty of aortic valve

It is a palliative treatment option for moderate subvalvular aortic stenosis and can result in a significant decrease in the peak systolic pressure gradient in dogs with severe SAS, at least for the short term but the ability of this procedure to provide long term benefit is poor.

Transventricular Aortic Dialation Valvuloplasty

Before surgery, a triple lumen catheter is placed percutaneously into a jugular vein to provide central venous

access and to monitor central venous pressure. A dorsal pedal catheter is placed to monitor direct arterial pressure and for arterial blood gas analysis.

- The dogs are placed in dorsal recumbency and a median sternotomy is performed, leaving the first one or two sternbrae intact.
- Cardiopulmonary bypass is performed using a primary bypass circuit and heart-lung machine.
- A Finochietto retractor is used to expose thoracic structures.
- The pericardium is opened on its ventral midline and the heart is suspended by suturing the pericardium to the sternotomy incision.
- A deep horizontal mattress suture of a 3-0 polyester, buttressed with polytetrafluoroethylenepledgets is placed in the muscle of the left ventricular apex.
- The ends of the suture are passed through rubber tubing to form a Rommel tourniquet.
- A stab incision into the left ventricle is made through the center of this suture. The Rommel tourniquet is tightened as necessary to control bleeding during the procedure.
- A Cooley pediatric dilator is introduced into the left ventricle through the stab incision and its position in the left ventricular outflow tract is confirmed by palpation of the ascending aorta.
- The instrument is withdrawn slightly and opened until a "popping" sensation is felt. The dilating procedure is repeated four to six times until results of intraoperative measurement of the systolic pressure gradient confirmed a reduction in the degree of stenosis.
- The dilator and Rommel tourniquet are removed, the mattress suture is tied and the pericardium is sutured loosely. The sternotomy incision is closed.

Closed transventricular dilation provided acute relief of outflow obstruction in young dogs with discrete subvalvular aortic stenosis. However, the stenosis recurred in a relatively short time. Therefore, this procedure should not be considered an effective definitive treatment for discrete subvalvular aortic stenosis in dogs. The procedure may be useful in young dogs with critical aortic stenosis as a bridge to more definitive surgery (Linn and Orton,1992)^[10].

Open Membranectomy and Septal Myectomy

Cardiopulmonary bypass affords the best opportunity to resect the stenotic area. With additional experience, septal resection and patch grafting may prove beneficial in reducing transaortic pressures over the long term in affected dogs. Open resection during cardiopulmonary bypass should be considered in dogs with severe subaortic stenosis. Direct visualization of the defect through an aortotomy, excision of the discrete fibrous ring and septalmyectomy can be performed. Surgical correction of subaortic stenosis via right ventriculotomy and septal resection has been described in one dog with long-term benefits (Nelson *et al.*, 2004)^[17]

- The surgery is performed through a right fourth intercostals thoracotomy.
- Animal is placed on a primary bypass circuit and heart-lung machine. Arterial cannulation for cardiopulmonary bypass is done via the left femoral artery and venous cannulation is done bicaval involving cranial and caudal vena cava.
- The ascending aorta is cross clamped and cardioplegia

solution usually of temperature 4°C is administered antegrade to the aortic root. Cardioplegia solution contains a high concentration of potassium chloride (20mEq/L) which arrests the electrical and mechanical activities of myocardium.

- The defect is approached through an elliptical incision in the ascending aorta.
- The aortic valve leaflets are gently retracted and subvalvular fibrous membrane is sharply excised with number 11 scalpel. The membrane usually reflects onto the septal leaflet of the mitral valve, which tethers the leaflet to the outflow tract. This portion of membrane must be carefully excised to achieve good results, but great care must be taken to avoid injury to the mitral valve which can be a fatal complication.
- After resection of the subvalvular membrane, the outflow tract is evaluated for need to perform septalmyectomy.
- A large sponge is placed under the left ventricle causing the septum to bulge into the left ventricular outflow tract.
- Septalmyectomy is performed by making two partial thickness parallel incisions in the ventricular septum.
- The two incisions are connected by a transverse incision and a portion of muscular septum is excised.
- The conduction system is situated at the junction of the

noncoronary and the right coronary cusps so the myectomy is performed to the left of this area.

- The aortotomy is closed with a pledget-buttressed continuous horizontal mattress pattern oversewn with a simple continuous pattern.
- Air is removed from the left side of the heart just before closure of aortotomy.
- The aortic cross clamp is removed and dog is weaned from cardiopulmonary bypass.

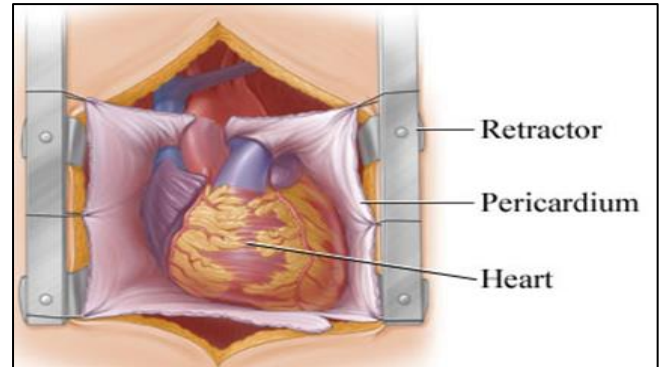


Fig 9: Opening of thoracic cavity

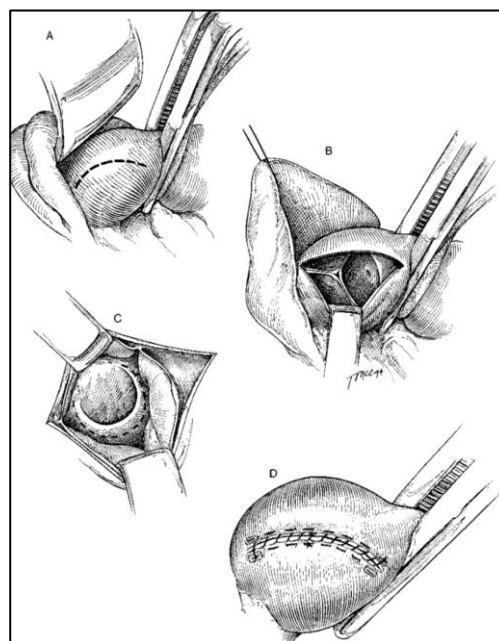


Fig 10: A- aortotomy; B- gentle retraction of aortic valve leaflets; C- resection of subvalvular fibrous membrane; D- aortotomy is closed with buttressed continuous horizontal mattress pattern of 4-0 polypropylene suture oversewn with simple continuous pattern

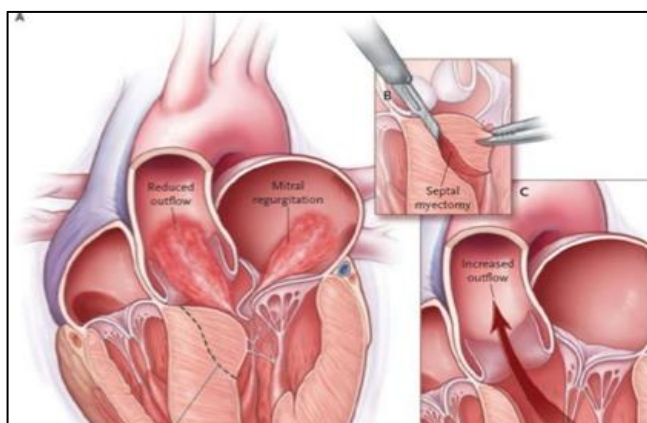


Fig 11: Septalmyectomy

Open surgical resection for subvalvular aortic stenosis produces a greater and more sustained relief of outflow obstruction compared with simple dilation valvuloplasty.

Suture materials and special instruments

Cardiopulmonary bypass having primary bypass circuit and heart lung machine is required for open repair of subaortic stenosis. A glutaraldehyde-fixed pericardial graft may be sutured into the defect caused by resecting the obstructing portion of the septum to prevent scarring and emboli formation.

Postoperative care and assessment

Ventilation should be monitored carefully in the early postoperative period. Poor ventilatory efforts may be

associated with residual pneumothorax, hemorrhage or pain. Heart rate and rhythm should be monitored postoperatively for 48 to 72 hours and hemodynamically significant arrhythmias should be treated. Blood pressure should be measured by direct or indirect means until the animal is fully recovered from anesthesia. Analgesics (local anesthetic techniques and systemic opioids) should be given to decrease postoperative discomfort. Urine output should be monitored closely, specially if hypotension occurred during or after surgery.

Prognosis

Dogs with systolic gradients above 75 mmHg are at substantial risk for sudden death in the first several years of life. Valve dilation can be performed at an early age with low operative mortality and without cardiopulmonary bypass. However, little evidence suggests that valve dilation results in sustained reduction in the systolic pressure gradient. The long-term benefit of this procedure is questionable. Modest gradient reduction (30% to 40%) has been achieved in approximately 33% of dogs that undergo valve dilation by balloon catheter. It is unclear whether valve dilation reduces the risk for sudden death.

Open resection of SAS under cardiopulmonary bypass may result in a substantial reduction of the systolic pressure gradient that is sustained for at least several years after surgery. However, in dogs undergoing open surgical correction of subvalvular aortic stenosis, a positive benefit on survival was not found despite reduction of the systolic pressure gradient (Orton *et al.*, 2000). These dogs appear to remain at high risk for sudden death associated with profound reflex vasodilation and bradycardia or fatal ventricular dysrhythmia. For balloon valvuloplasty, no clear benefit was seen in dogs with severe SAS that underwent this procedure, despite a significant decrease in the peak systolic pressure gradient (Meurset *et al.*, 2005)^[3].

Conclusion

Congenital heart defects are prevalent in canine population and they remain a major cause of asymptomatic death in dogs less than one year of age. Major contributor to congenital heart anomaly comprise of subaortic stenosis affecting the left ventricular outflow tract. With advancement in diagnostic techniques this defect can be diagnosed on clinical presentation with a parallel differential diagnosis from other congenital heart defects. This disease decrease longitivity and quality of life which can be managed by using conventional medicinal therapy or performing major surgical interventions such as balloon vulvoplasty, transventricular aortic dilation vulvoplasty and open membranectomy or septalmyectomy. The prognosis of surgical interventions is fair in mild cases whereas guarded in moderate to severe cases.

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