CONGENITAL HEART DISEASES



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Congenital Heart Diseases: Uncommon in dogs and rare in cats.

Most Common Types in dogs:

- Subaortic Stenosis (SAS)
- Pulmonic Stenosis (PS)
- Patent Ductus Arteriosus (PDA)

Most Common Types in cats:

- Atrial Septal Defects (ASDs)
- Ventricular Septal Defects (VSDs)
- Valvular Dysplasias

- Many CHDs have breed predispositions; some are heritable traits, though not all are inherited.
- Often diagnosed via auscultation of a significant or unusual murmur in young patients. Early detection can lead to earlier treatment, improving prognosis and quality of life.

TABLE 8.12 Breed predisposition for congenital heart diseases

Congenital defect	Breed predisposition	Known heritable mutation
Subaortic stenosis	Newfoundland, Rottweiler, Golden Retriever, Boxer, Great Dane, German Shorthair Pointer, German Shepherd Dog, Bouvier des Flandres, English Bulldog, Samoyed, Bull Terrier (valvular stenosis)	Newfoundland
Pulmonic stenosis	English Bulldog, French Bulldog, West Highland White Terrier, Beagle, Mastiff, Samoyed, Miniature Schnauzer, American Cocker Spaniel, Keeshond, Boxer, Chihuahua, Airedale Terrier	Beagle
Tricuspid valve dysplasia	Labrador Retriever, Golden Retriever, German Shepherd Dog	Labrador Retriever
Ventricular septal defect	Beagle, English Springer Spaniel, Keeshond, English Bulldog	Beagle, English Springer Spaniel
Atrial septal defect	Poodle, Dobermann, Boxer, Samoyed	Poodle
Mitral valve dysplasia	Bull Terrier, Rottweiler, Golden Retriever, Newfoundland, Mastiff, German Shepherd Dog	
Patent ductus arteriosus	Poodle, German Shepherd Dog, Pomeranian, Shetland Sheepdog, Collie, Maltese, Yorkshire Terrier, Chihuahua, Bichon Frise, Keeshond, American Cocker Spaniel, Rottweiler, English Springer Spaniel, Labrador Retriever, Newfoundland	Poodle
Tetralogy of Fallot	Keeshond, English Bulldog, Wire-haired Fox Terrier, Miniature Schnauzer, West Highland White Terrier	Keeshond
Persistent right aortic arch	German Shepherd Dog, Irish Setter, German Short-haired Pointer, Greyhound	German Short-haired Pointer

• **Congenital heart defects often produce an audible murmur**, though some serious malformations may not.

• Murmur Intensity: Ranges from very loud to very soft, depending on defect type, severity, and hemodynamic factors.

• Characteristics of Innocent Murmurs:

Common in Young Animals: Relatively common in puppies and kittens.

Physiologic Causes: May involve growth rate mismatches between the heart and great vessels, relative anemia, or high sympathetic tone.

Presentation: systolic ejection-type murmurs, best heard at the left heart base.

• Typically get softer and disappear by about 4 months of age.

- **Persistence of Congenital Murmurs:** Usually persist beyond 6 months of age and may get louder, though not always.
- **Importance of Auscultation:** Essential in breeding animals, working dogs, and pets.
- Diagnostic Considerations for Murmurs:

Murmurs that are loud (audible on both sides of the chest)

persist beyond 4-6 months.

• Types of Congenital Heart Defects

Common Involvements:

• Valves or valve regions: Abnormal communications between systemic and pulmonary circulations

Valvular Abnormalities:

• Insufficient, stenotic, or both

• the murmur should be identified as to its timing in the cardiac cycle (e.g., systolic, diastolic, continuous). Also, the duration of the murmur (e.g., early systolic, holosystolic, pansystolic) should be noted.

• the site at which the murmur is loudest (PMI) (e.g., valve area) and where it radiates because of blood flow through the defect (e.g., other valve areas where it can be heard) should be noted.

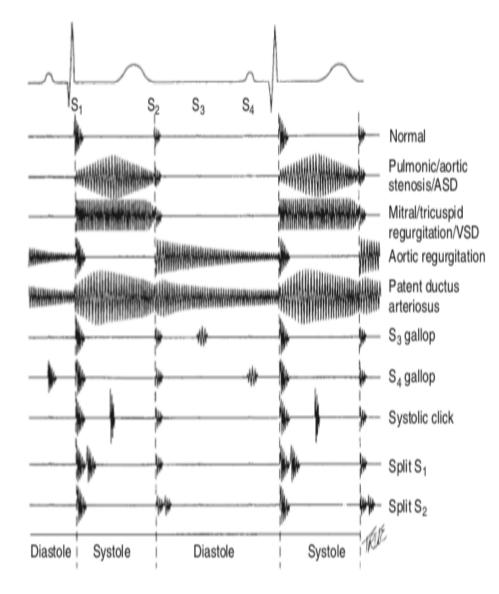


Figure 2-16 Cardiac cycle with electrocardiogram and phonocardiogram schematized. Both normal and abnormal sounds are included. *ASD*, Atrial septal defect; *VSD*, ventricular septal defect. (From Atkins CE: Abnormal heart sounds. In Allen DG, editor: Small animal medicine, Philadelphia, 1991, Lippincott Williams & Wilkins.)

- Systolic murmurs can occur in early (protosystolic), middle (mesosystolic), or late (telesystolic) systole or throughout systole (holosystolic).
- Diastolic murmurs generally occur in early diastole (protodiastolic) or throughout diastole (holodiastolic). Murmurs at the end of diastole are termed *presystolic*.
- Continuous murmurs begin in systole and extend through S2 into all or part of diastole. A holosystolic (plateau-shaped) murmur begins at the time of S1 and is of fairly uniform intensity throughout systole.
- Loud holosystolic murmurs may mask the S1 and S2 sounds. AV valve insufficiency and interventricular septal defects commonly cause this type of murmur because turbulent blood flour occurs throughout ventricular systole.

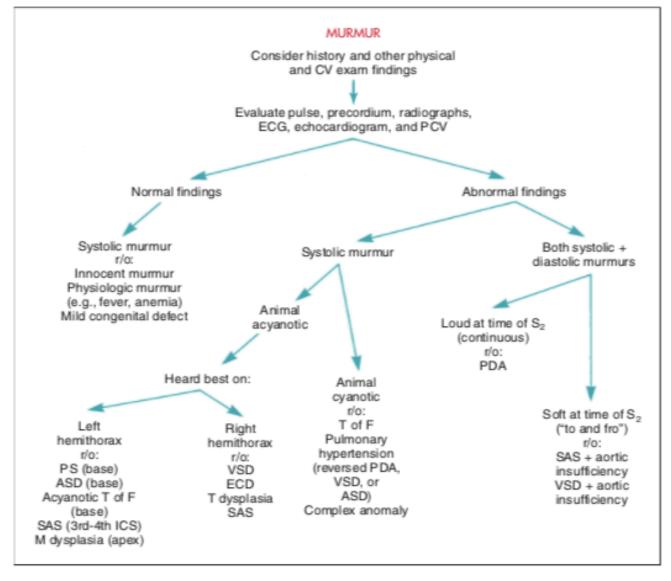


FIG 5.1

Flow chart for differentiating murmurs in puppies and kittens. Thoracic radiographs, ±ECG, also can help screen for structural abnormalities; however, referral to a veterinary cardiologist is recommended for more definitive evaluation. ASD, Atrial septal defect; ECD, endocardial cushion defect; ECG, electrocardiogram; ICS, intercostal space; M, mitral valve; PCV, packed cell volume; PDA, patent ductus arteriosus; r/o, rule out; SAS, subaortic stenosis; T, tricuspid valve; T of F, tetralogy of Fallot; VSD, ventricular septal defect.

راهنمای استفاده از سوفلهای قلبی جهت دستیابی به تشخیص بیماریهای مادرزادی قلب در سنگ و گربه سوفل ۱- ارزیابی نبض شریانی ۲- ضربان نزدیک قلبی ۳- رادیوکرافی ۴- الکتروکاردیوگرافی ۵- اکوکاردیوگرافی ۶- حجم فشرده گلبولی (PCV) dig yok and the Andre in the سوفل طبيعي سوفل غیرطبیعی (پاتولوژیک) سوفل سيستوليك سوفل سیستولیک ـ دیاستولیک سوفل فيزيولوژيک and the second s يصورت علائم در زمان بلندبودن سوفل در بدون علائم سيانور ۱ - بعلت أنمى ۲ - تب صداى دومقلب بصورت زمان صدای دوم قلب training and the 🖕 she had ۳- نقصهای علائم مادرزادی (To and fri) بصورتبلند و مداوم، شنيدن صداهاي قلبي ۱- تنگىزىردرىچە (بازبودنمجرای شریانی) آئورت -طرفراستناحيهقفسهسينه طرف چپ ناحیه قفسه سینه ۲- ئارسائى دريچە یا علائم سیانوز ۱-نقص ديوار مبين دوبطن ا - تنگی دریچهٔ ریوی أئورت همراه با ۱- تترالوژی فالوت ۲- نقص اندوكار ديت بالشتكي در قاعدهٔ قلب دوبطن ۲- افزایش فشار خون ۳- دیسپلازی تریکوسپید ۲- نقص ديواره بين دو ۳- نارسائی دریچهٔ شریان ریوی ۴- تنگىزىردرىچەأئورت دهليز در قاعدهٔ قلب أئورت ۳- بازبودن مجرای شریانی Contraction of the ۳- تترالوژی فالوت غیر ۴-نقص دیوار ذبین دوبطن سيانوزه در قاعدهٔ قلب ۵- نقص ديوار ذبين دو دهليز ۴- تنگى زيردريچة أنورت **ج- انومالیهای کمپلکس** در سومین و چهارمین مادرزادی فضاى بين دندهاى and the second sec

• Femoral pulses:

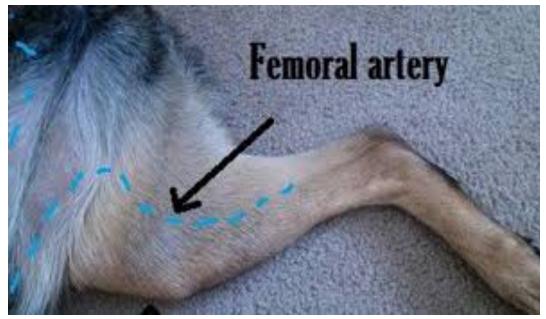
- □ Both femoral pulses should be felt.
- □ It is difficult to palpate the femoral pulses in cat.
- □ Normal rate in dog:70-180 beats/min
- □ Normal rate in cat: 160-240 beats/min
- □ Puppies>220 beats/min
- □ There is a pulse for every heart beat.
- □ Arterial pulse pressure is the difference
- between the arterial systolic and diastolic pressure.
- It is strong and have a rapid rate of rise and fall.
- Pulse quality depends on age, hydration, cardiac function

And level of excitement and activity

able 1.3	Types of Pulses and Their	
	Associated Causes	

Pulse	Cause
Absent pulses	Thromboembolism
Abrupt pulses	Mitral regurgitation
	Ventricular septal defects
Erratic pulses	Atrial fibrillation
Hypokinetic pulses	Heart failure
	Hypotension
	Hypovolemia
	Subaortic stenosis
Hyperkinetic pulses	Aortic regurgitation
	Fear
	Fever
	Patent ductus arteriosus
	Severe anemia
	Severe bradycardia
	Thyrotoxicosis
Pulse deficits	Arrhythmias
Pulsus alternans	Severe dilated cardio myopathy
Pulsus bigeminus	Arrhythmias
Pulsus paradoxus	Cardiac tamponade





Partial or complete occlusion: thromboembolism

Bacterial endocarditis of mitral or aortic valve in dog and cat, hyperadrenocorticism, PLN, cardiomyopathy in cat.

□ Hypokinetic(weak): decreased cardiac output(Congestive heart failure, hypovolemia)

Decreased vascular resistance

Hyperkinetic(strong): pulses rise and fall quickly: aortic regurgitation, fear, fever.

D Pulses alternance(pulse is weak and then strong): normal, severe DCM

Normal Mucous Membrane Colors

- Most healthy mucous membranes are pink in color .
- The color is a direct reflection of vascular health.
- Mucosa has a healthy blood supply and its tissues are thin. This makes changes in oxygenation and perfusion visible.
- When mucosa is well-oxygenated and there is adequate blood flow through the vasculature, no pigmented mucosa is pink.
- Another measure of the health of the patient's circulatory system is the capillary refill time (CRT).

Mucous membranes:

Mucous membranes are inspected for pallor, cyanosis

Pallor can indicate anemia , but can also be associated with severe peripheral vasoconstriction

Cyanosis indicates an increased concentration of desaturated hemoglobin and imparts a mucous membrane colour ranging from slightly 'dusky' in mild cases to nearly navy blue in patients with severe hypoxemia.

Cyanosis is most often seen with severe hypoxaemia:

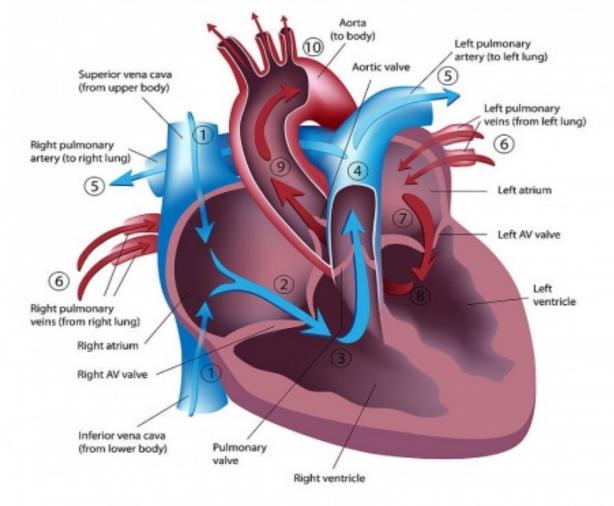
- respiratory disease (including pulmonary odema or pleural effusion with CHF)
- a right-to-Left congenital shunt. With right-to-Ieft shunts (sometimes termed 'cyanotic heart disease')
- polycythemia, which itself gives a dark red colour to the mucous membranes

• Abnormal Causes of Palpably Cool or Cyanotic Extremities:

Despite the fact that dogs have footpad vasculature that preserves the core body temperature at the expense of the peripheral body temperature, there are conditions in which palpably cool extremities are abnormal. These include:

- Arterial or aortic thromboembolism (ATE)
- Reverse patent ductus arteriosus (PDA)
- Shock

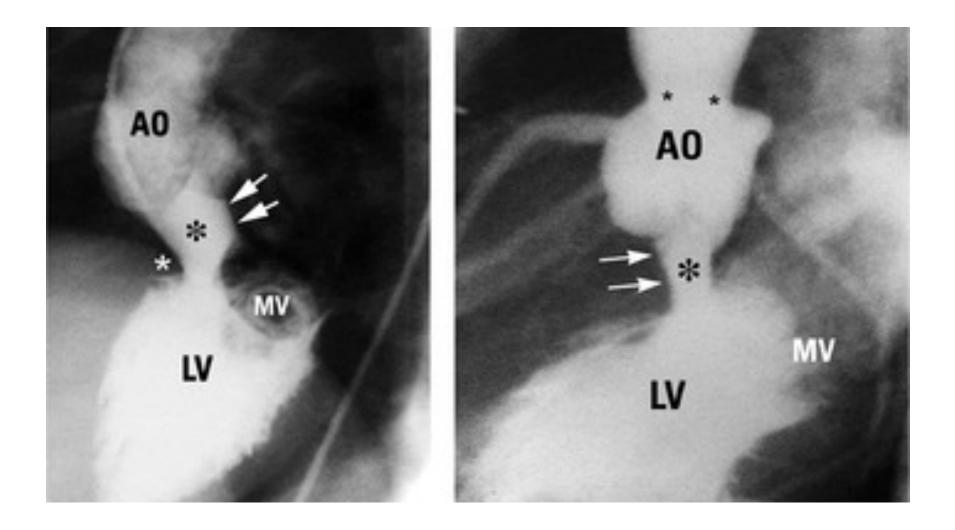




Key Factors that Influence Decision Making Process in CHD Patients

- Anatomy of the congenital defect
- Severity of the disease
- Patient size (including vessel size for access)
- Presence of concurrent congenital/acquired heart disease
- Presence of concurrent systemic disease
- Need for advanced imaging to characterize the anatomy
- Procedural availability
- Operator experience
- Equipment inventory and availability
- Cost

SUBAORTIC STENOSIS:



Aortic Stenosis:

- A narrowing of the left ventricular outflow tract near the aortic valve.
- Can occur at subvalvular, valvular, or supravalvular levels.
- Subaortic Stenosis (SAS) in Dogs: Most common manifestation in dogs.
- Genetic Basis: A genetic mutation linked to SAS has been identified in **Newfoundland.**



Pathophysiology of SAS

- Anomalous fibrous band or ring of tissue at the subvalvular level.
- **Pressure overload leads** to left ventricular concentric hypertrophy and sometimes left atrial enlargement.
- Turbulence and increased flow velocity create a left systolic basilar murmur.
- **Predisposition to ventricular arrhythmias due to myocardial ischemia** from increased myocardial volume, reduced coronary blood flow, and subendocardial fibrosis.
- Less commonly, increased left atrial pressure can lead to left-sided congestive heart failure (CHF).

Clinical Presentation of Subaortic Stenosis (SAS):

- Failure to thrive
- Exercise intolerance
- Syncope
- Signs in Older Dogs: Tachypnea or dyspnea associated with the development of congestive heart failure (CHF).

Severe SAS and Associated Risks

• Ventricular Tachyarrhythmias: Rapid and sustained arrhythmias can lead to sudden death.

Physical Examination Findings

- Heart Murmur:
- Left basilar systolic murmur, with grade correlating to the severity of SAS.
- Pulses: Weak femoral pulses in cases of severe stenosis.
- Arrhythmias: Occasionally detected during examination.

Differential Diagnosis for Subaortic Stenosis:

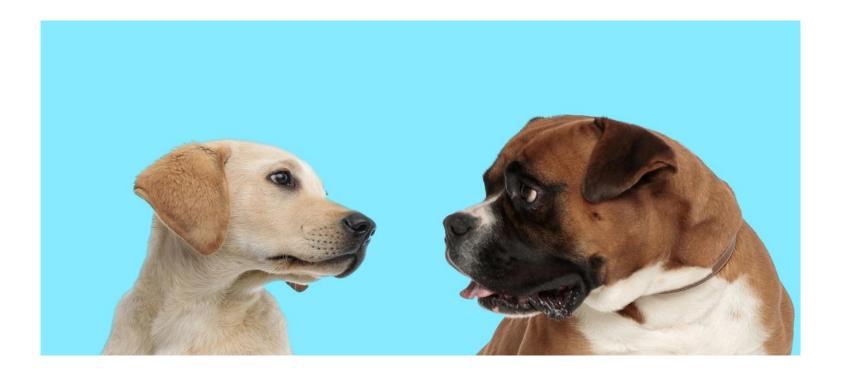
- Pulmonic Stenosis (PS)
- Ventricular Septal Defect (VSD)
- Physiologic Murmur

Diagnostic Imaging for SAS:

• **Thoracic Radiographs**: May show poststenotic dilation of the aortic root.

- Echocardiography in SAS Diagnosis: Echocardiography visualizes the stenosis location and fibrous anomaly.
- Assesses severity of secondary changes: left ventricular concentric hypertrophy and atrial dilation.
- Color-flow echocardiography reveals turbulent flow through the left ventricular outflow tract.
- Doppler echocardiography quantifies stenosis severity via peak systolic aortic blood flow velocity.

- Pressure Gradient Measurement: A pressure gradient above 80 mmHg indicates severe SAS.
- Boxers and Golden Retrievers: Predisposed to SAS, including mild forms with soft murmurs.



• Medical Management of Subaortic Stenosis (SAS)

Beta-Blockers:

• Atenolol (0.5–1 mg/kg orally every 12–24 hours) to slow heart rate, reduce myocardial oxygen demand, and provide antiarrhythmic effects.

Goals of Medical Therapy:

- Decrease syncopal episodes frequency
- Improve exercise tolerance
- Reduce risk of sudden death

Interventional Procedures for SAS

Balloon Valvuloplasty: Considered for severe SAS with poor prognosis. Utilizes cutting and/or high-pressure balloons to relieve pressure gradient. Effectiveness in improving quality of life and survival remains unproven. • Management of Congestive Heart Failure (CHF): Managed with diuretics and ancillary medications as appropriate.

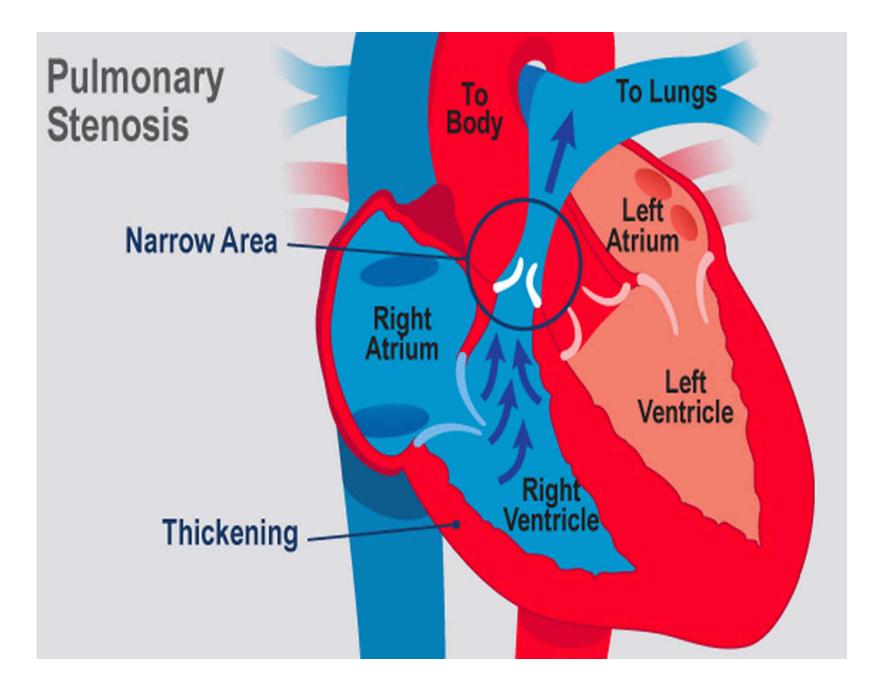
• Infective Endocarditis Risk:

Mildly increased in SAS patients.

Antibiotics recommended during surgeries and dental prophylaxis procedures.

• Mild SAS has a good prognosis, but cases of moderate SAS tend to develop CHF later in life.

• Dogs with severe SAS are at significant risk of sudden death before 3–5 years of age.



• Pulmonic Stenosis (PS):

Narrowing of the right ventricular outflow tract at the pulmonic valve.

• Levels of Stenosis:

Subvalvular, valvular, or supravalvular

Common Manifestation in Dogs: Valvular stenosis

• Less common in cats

Heritability of Pulmonic Stenosis (PS)

PS is heritable in Beagles.

Pathophysiology of Pulmonic Stenosis:

Increased pressure in the right ventricle leads to **concentric right ventricular hypertrophy and myocardial fibrosis**.

Possible dilation of the right atrium.



Breed Predisposition

Hereditary in Beagle, Boykin Spaniel

Very common in Boxer, Bulldog breeds

TABLE 3 | Odds ratio by breed with ≥10 cases for pulmonic stenosis (PS).

Breed	No. of cases	Prevalence (%)	Odd ratio	95% Confidence interval	p-value
Bulldog	56	4.80	13.32	9.42-18.84	<0.0001
French buildog	23	2.70	7.52	4.71-12.02	< 0.0001
Pitbull terrier	35	1.30	3.53	2.37-5.26	< 0.0001
Chihuahua	17	0.52	1.45	0.86-2.45	0.1677
German shepherd	12	0.43	1.19	0.65-2.19	0.5682
Mixed breed	80	0.38	n/a	n/a	n/a

Ontiveros et al. Frontier Vet Sci 2019

Table 2. Breed distribution and predisposition-

	Pulmonic Stenosis (363 cases)			
	Ν	%	Odds Ratio	P
Boxer	116	31.9	5.27	< .0001
Mongrel	35	9.6	0.32	< .0001
English Bulldog	27	7.4	3.16	< .0001
French Bulldog	21	5.8	19.1	< .0001
Pinscher	14	3.8	3.1	.0001
German Shepherd	11	3.0	0.44	.0085
Beagle	10	2.7	2.66	.003
West Highland White Terrier	9	2.5	2.91	.003
American Staffordshire Terrier	8	2.2	16.9	< .0001
Chihuahua	8	2.2	3.11	.003
Cavalier King Charles Spaniel	6	1.6	1.62	NS
Cocker Spaniel	6	1.6	1.1	NS
Pitbull Terrier	6	1.6	4.48	.0009
Rottweiler	6	1.6	0.91	NS
Newfoundland	5	1.4	0.81	NS
Golden Retriever	5	1.4	0.97	NS
Shih-Tzu	5	1.4	0.78	NS
Yorkshire Terrier	5	1.4	0.23	.0013
Italian Mastiff	4	1.1	2.1	NS
Poodle	4	1.1	0.23	.0062
Standard Schnautzer	4	1.1	16.7	< .0001
Others	48	13.2		

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Clinical Manifestations:

• Murmur: Turbulent blood flow through the stenotic valve results in **a left basilar systolic murmur.**

• Right-Sided Heart Dysfunction: Severe PS can lead to right-sided diastolic dysfunction and risk of right-sided congestive heart failure (CHF) from chronic pressure overload.

• **Tricuspid Valve Dysplasia (TVD):** Occasionally concurrent with PS, exacerbating right heart dysfunction and clinical severity.

• Symptom Severity:

Most animals exhibit no to mild clinical signs.

• Common Clinical Signs (in approximately one-third of affected dogs): Dyspnea, Lethargy, Exercise intolerance, Syncope

• Advanced Clinical Signs:

Right-Sided Heart Failure:

Signs such as ascites and hepatomegaly may be present in later stages.

• Physical Examination Findings:

Left-sided systolic basilar murmur

Concurrent right apical murmur if tricuspid regurgitation is present Jugular distension linked to increased right atrial pressure

- Differential Diagnosis for Pulmonic Stenosis (PS)
 Subaortic Stenosis (SAS)
 Physiologic Murmur
 Tetralogy of Fallot (TOF)
 Ventricular Septal Defect (VSD)
- Thoracic Radiographs:

May show post-stenotic dilation of the pulmonary trunk and right-sided cardiomegaly.

• Echocardiography:

Assesses the structure of the pulmonic valve and severity of ventricular concentric hypertrophy.

Detects presence of tricuspid insufficiency.

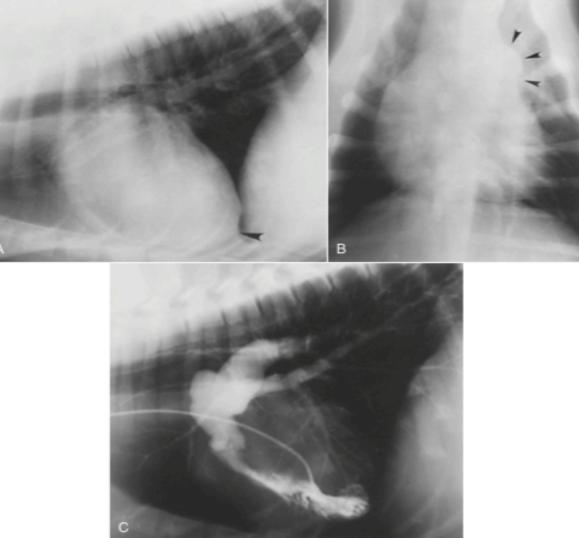
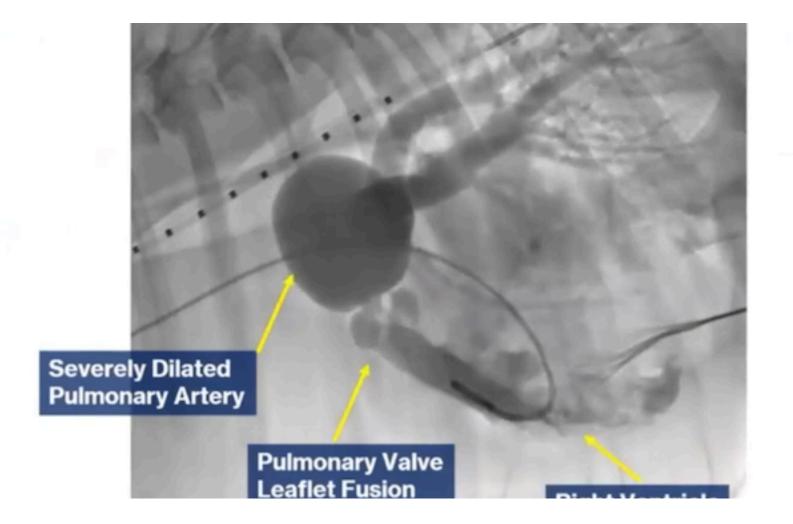
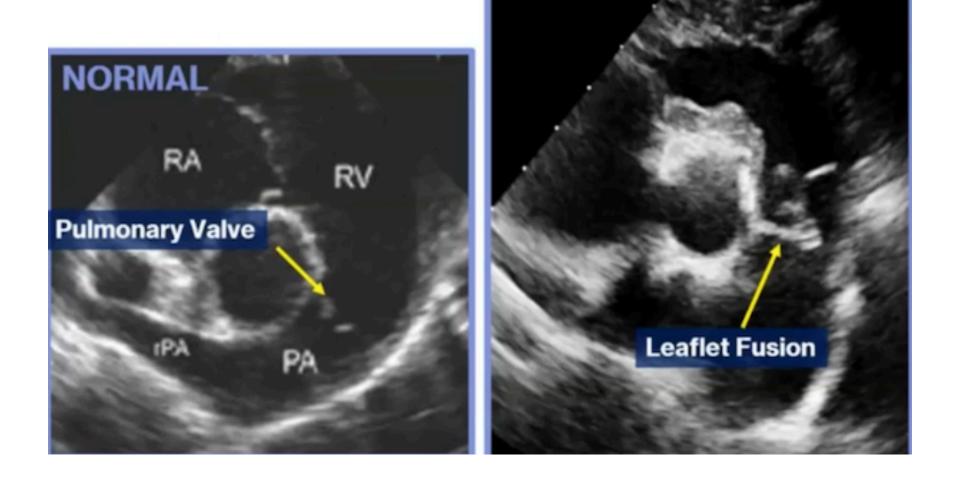
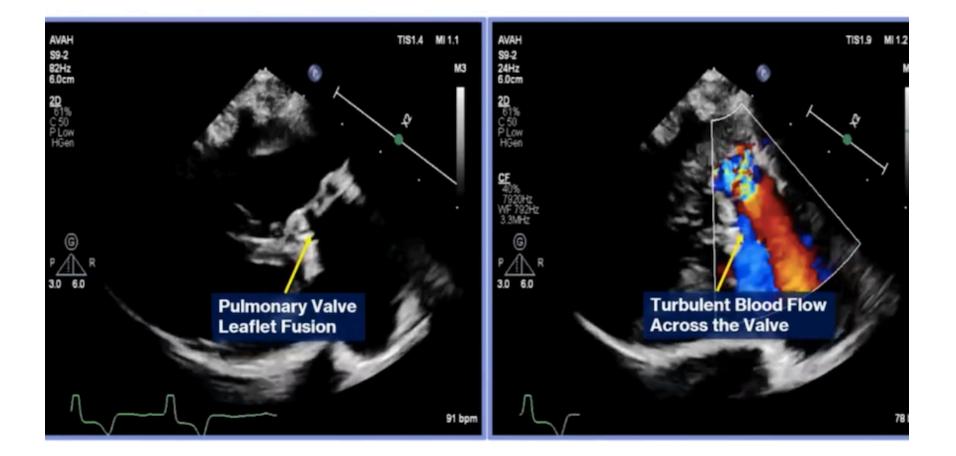


FIG 5.6

Lateral (A) and dorsoventral (DV) (B) radiographs from a dog with pulmonic stenosis, showing right ventricular enlargement (apex elevation on lateral view [arrowhead in A] and reverse D configuration on DV view) along with a pulmonary trunk bulge (arrowheads in B) seen on a DV view. (C) Angiocardiogram using a selective right ventricular injection demonstrates poststenatic dilation of the main pulmonary trunk and pulmonary arteries. The thickened pulmonic valve is closed in this diastolic frame.







• Doppler Echocardiography

Essential for determining stenosis severity.

Continuous-wave Doppler assesses peak systolic pressure gradient.

• The differences pressure between systolic pressure of right ventricular and pulmonary artery:

Mild: 20-49 mmHg (velocity of 2.25–3.5 m/s)

Moderate: 50-80 mmHg (velocity of 3.5–4.5 m/s)

Severe: Above 80 mmHg (velocity above 4.5 m/s)

Treatment Goals:

• Reduce systolic right ventricular pressure overload.

Interventional Procedures

• Balloon Valvuloplasty: Most effective in valvular stenosis without valve annulus hypoplasia. Typically results in a 40%–50% reduction in pressure gradient.

Medical Management:

- Beta-Blockers: Used in mild PS or cases with mild clinical signs.
- Reduce myocardial oxygen demand.
- Provide antiarrhythmic properties.

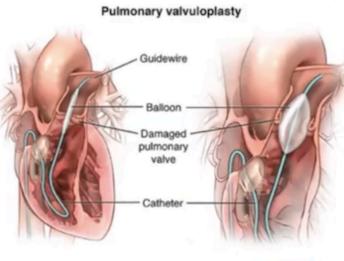
Prognosis of Pulmonic Stenosis

• Varies based on severity of the stenosis.

PS Catheter-Based Intervention

Balloon Pulmonary Valvuloplasty (BPV)

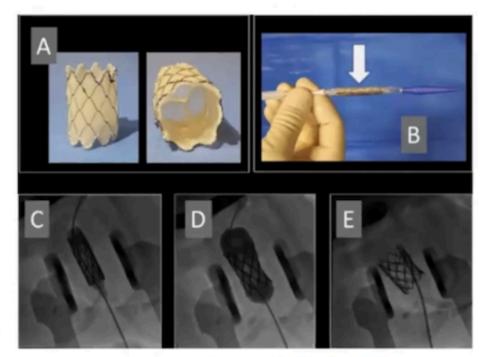
- Minimally invasive catheter-based surgery
- Access heart via small incision in jugular vein (or femoral vein)
- Deflated balloon positioned across pulmonary valve and inflated
- · Goal:
 - Tear valve leaflets apart
 - Reduce pressure gradient across valve
 - Reduce workload of right ventricle
 - Stop/reverse right ventricular remodeling
 - Mitigate/resolve risk of arrhythmias and heart failure



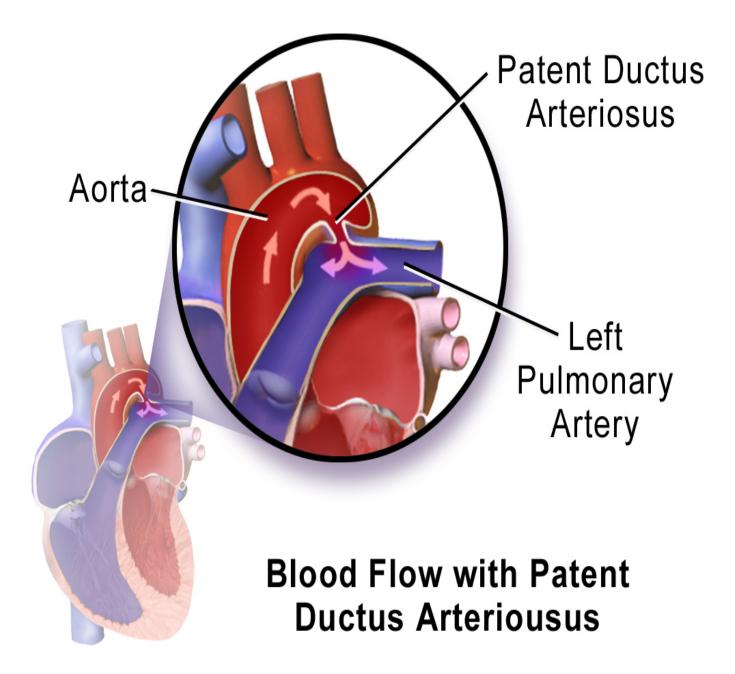
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Future of Intervention for PS

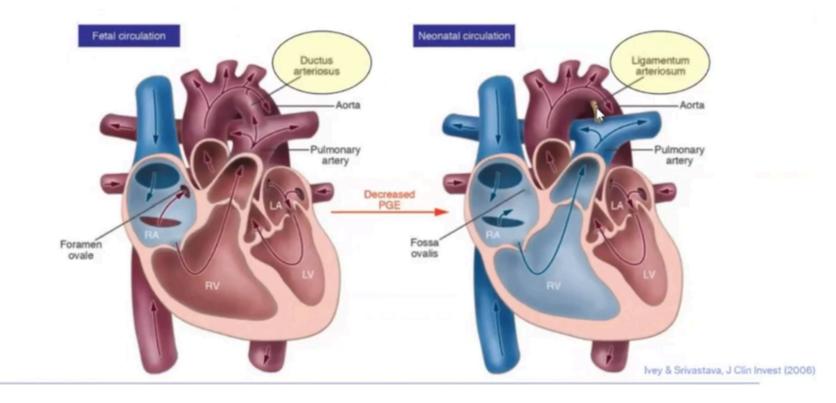
- Transpulmonary Stents
 - Becoming more widely available
 - High success rates
 - Requires more advanced training
 - Risk of stent fracture or migration
- Valve Implantation
 - One case report
 - \$\$\$\$\$



Borenstein et al. J Vet Cardiol 2019



Patent Ductus Arteriosus (PDA)



- PDA allows blood to flow from systemic circulation (aorta) into pulmonary circulation (left to right shunting)
- Can lead to severe complications:
 - Volume overload of lungs and left heart
 - Severe left heart dilation
 - Congestive heart failure (64% of dogs before 1 year of age)
 - Arrhythmias (e.g., atrial fibrillation)
 - Shunt reversal (right to left)

Patent Ductus Arteriosus (PDA) :

Shunts oxygenated blood from maternal circulation away from fetal lungs to systemic circulation.

Postnatal Changes and PDA:

At Birth: Lung inflation reverses blood flow, sending oxygenated blood from the aorta through the ductus. Stimulates constriction of ductus smooth muscle. Normal Closure: Typically closes by 7–10 days after birth.

Patent Ductus Arteriosus:

Occurs when the ductus remains open beyond this period. Allows blood to shunt according to the pressure gradient.

Etiology of PDA: Heritability: PDA is considered heritable in Poodles.



Which Breeds are Predisposed to PDA?

- Small breeds are over-represented
 - Bichon Frise, Maltese, Pomeranian, Chihuahua, Collie, Toy Poodle, Keeshond, Yorkshire Terrier
 - German Shepherd, Shetland Sheepdog, English Springer Spaniel
- Females > Males
- Cats, sheep, horses, many species
- Documented genetic component in toy and miniature poodles



cocothemaltesede

Typical Shunt Direction:

Left-to-right shunt: blood moves from the aorta to the pulmonary artery.

Left-Sided Volume Overload: Blood returns to the left side of the heart, causing dilation of the left ventricle and left atrium.

Shunt Reversal and Eisenmenger Physiology: Large shunts and severe pulmonary hypertension (PH) can cause shunt reversal.

Right-to-Left Shunt:

Blood flows from the pulmonary artery to the aorta. Clinical Manifestations: cyanosis Hypoxemia Polycythemia • Timing: Occurs in puppies a few days to weeks old, not typical in adult dogs.

• Factors Influencing Severity: Size of the patent vessel Presence of other defects

 Clinical Signs of Patent Ductus Arteriosus (PDA): dependent on Shunt Size and Direction: Exercise intolerance, Syncope, Cough, Dyspnea
 Reverse Shunt: Hind limb weakness and Seizures

Physical Examination Findings:

Left-to-Right PDA:

Continuous (machinery) murmur in the left axillary region.

Bounding femoral pulses due to diastolic runoff.

Reverse PDA:

Absence of audible murmur.

Differential cyanosis: Cyanosis limited to caudal mucous membranes.

Diagnosis of Patent Ductus Arteriosus (PDA):

- Thoracic Radiographs: May reveal left-sided cardiomegaly. Signs of congestive heart failure (CHF)
- Electrocardiography (ECG):

Tall R wave in lead II, indicative of left ventricular enlargement.

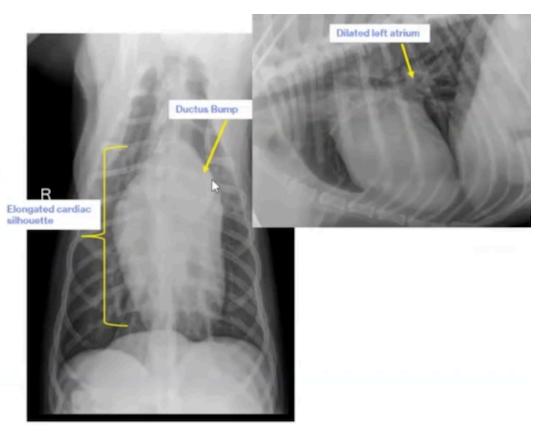
• Echocardiography:

Confirms presence of the shunt.

Evaluates severity of left heart chamber enlargement.

Chest X-rays

- Useful to evaluate anatomic changes
- Dilated left heart, aorta, pulmonary arteries
- Overcirculation of lungs
- Elongated cardiac silhouette
- Aneurysmal bulge of aorta at level of ductus (ductus bump)



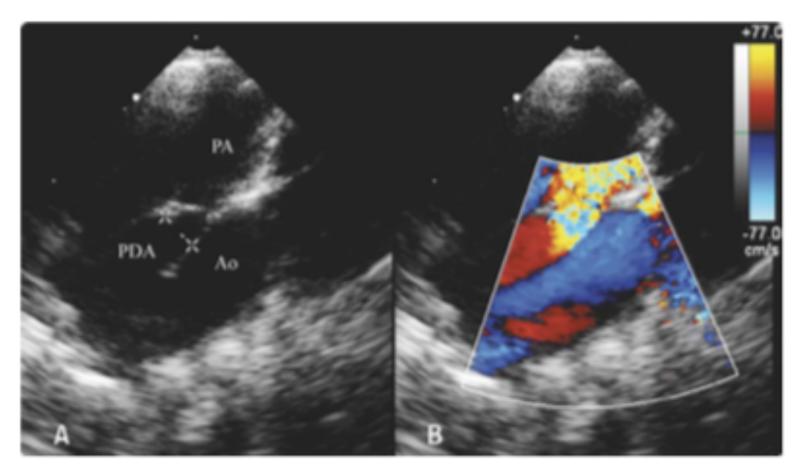
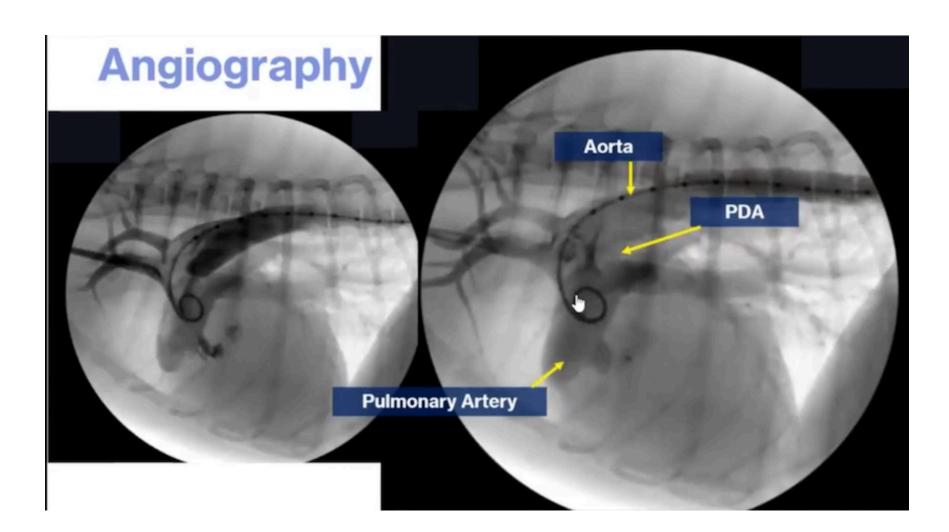


FIGURE 8.26 (a) Transthoracic echocardiographic view of a patent ductus arteriosus (PDA). Measurements of the PDA diameters are obtained. (b) Color Doppler reveals turbulence (mosaic of colors) in the pulmonary artery. Ao, aorta; PA, pulmonary artery; PDA, patent ductus arteriosus.



Management of Patent Ductus Arteriosus:

• Early Closure:

Best achieved through surgical or cardiac catheterization procedures.

• Pre-Closure Medical Treatment:

Medical treatment is necessary for patients in congestive heart failure (CHF) prior to closure.(phlebotomy, Indometastin: NSAID)

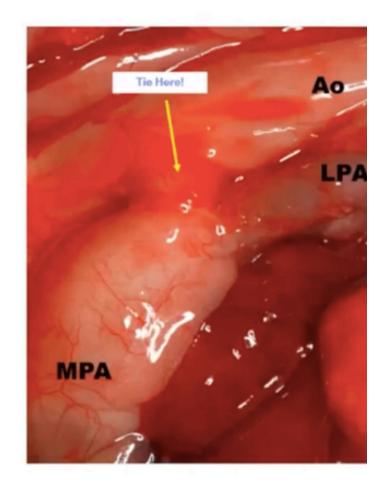
• Surgical Closure:

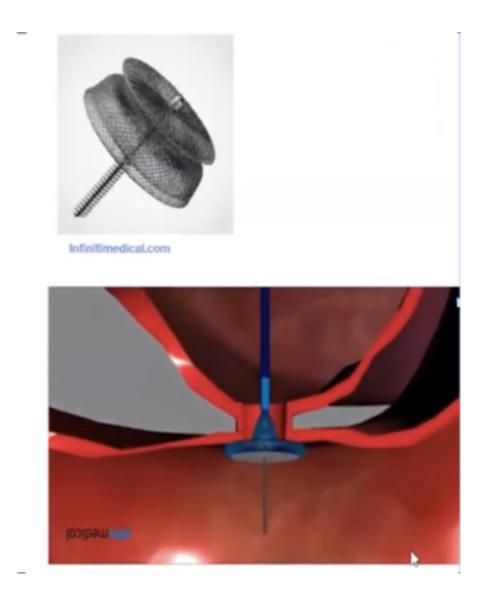
Up to 95% success rate when performed by experienced surgeons.

PDA Therapies

Surgical Ligation

- More invasive method
- Higher risk for bleeding
- Overall very high success rate
- May be only option for very small patients or PDA with unfavorable morphology (shape)





• Infection Risk Management

Unknown risk of infection for implants.

Antibiotic Recommendations: Recommended during surgery and for dental prophylaxis for 1 year post-implant.

• Reverse PDAs: Closure is contraindicated.

• Secondary changes can reverse with early and successful closure of the PDA, and in these cases, prognosis is excellent. Animals with a PDA that is not closed rarely live past 1 year of age, although rarely some can live well into adulthood with an uncorrected PDA.

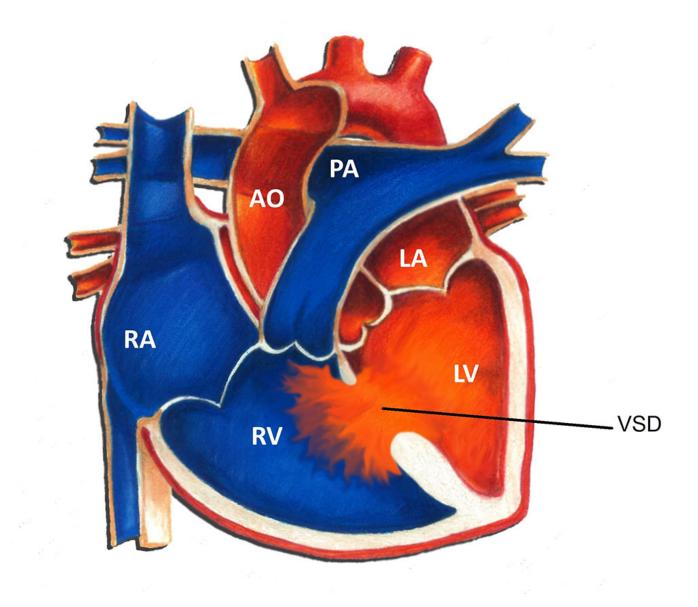
Prognosis for PDA After Intervention

- With treatment, outstanding prognosis for long-term survival
- Considered to be curative
- However clinical signs at presentation, concurrent congenital heart disease, and severe mitral regurgitation after PDA closure are negatively associated with survival



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- VSDs are defects that allow communication between the left and right ventricles.
- Typically occur in the membranous or upper portion of the septum.
- Prevalence: Most common congenital cardiac defect in cats.
- Etiology of VSDs: Heritability: VSDs are heritable defects in Keeshonds and English Springer Spaniels.

Pathophysiology of Ventricular Septal Defects (VSDs)

- Blood shunts from left to right during systole.
- Increased blood flow enters the pulmonary artery and returns to the left heart.

Determines physical examination findings:.
 Murmur Characteristics

Left-to-Right Shunts: May present as a right-sided, sternal, or left basilar holo systolic murmur.

Location is dependent on the site of the septal defect.

Small VSDs

High velocity of blood flow can result in a louder murmur.

Potential Complications:

Blood overflow may damage aortic valve cusps: Leads to aortic insufficiency and a left basilar diastolic murmur.

Large Ventricular Septal Defects :

Large VSDs may allow ventricular pressures to equalize.

Murmur Intensity: Can result in a softer murmur or absence of an audible murmur.

Associated Clinical Signs:

- Congestive heart failure (CHF)
- Hypoxemia

Differential Diagnosis for VSDs

- Mitral regurgitation
- Tricuspid regurgitation
- Subaortic stenosis (SAS)
- Pulmonic stenosis (PS)

Diagnosis of Ventricular Septal Defects (VSDs)

- Color-flow Doppler echocardiography used for visualization of the defect or shunt.
- Echocardiographic:

VSD Size Comparison: Compared with the diameter of the aortic annulus. Small VSDs: Less than 25% of aortic diameter.

Moderate VSDs: Between 25% and 75% of aortic diameter.

Large VSDs: More than 75% of aortic diameter.

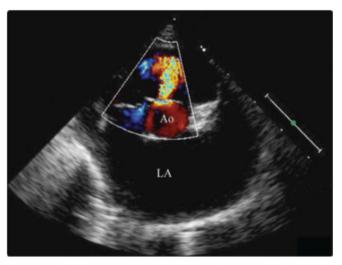


FIGURE 8.29 Right parasternal short-axis echocardiographic view confirming the presence of a ventricular septal defect with color-flow Doppler. Turbulent blood flow is seen entering the right ventricle from a defect located just below the aorta in the perimembranous portion of the interventricular septum. Ao, aorta; LA, left atrium.

• Asymptomatic Patients:

No treatment is indicated for small restrictive VSDs.

• Management of Larger Nonrestrictive VSDs:

Medical Management: Left-sided congestive heart failure (CHF) is managed medically as needed.

Definitive Therapy: Cardiopulmonary bypass for open-heart surgery with patch grafting is typically required.

Role of VSD Size: Size of restrictive VSDs influences the risk of left ventricular volume overload and congestive heart failure (CHF).

Long-Term Monitoring:

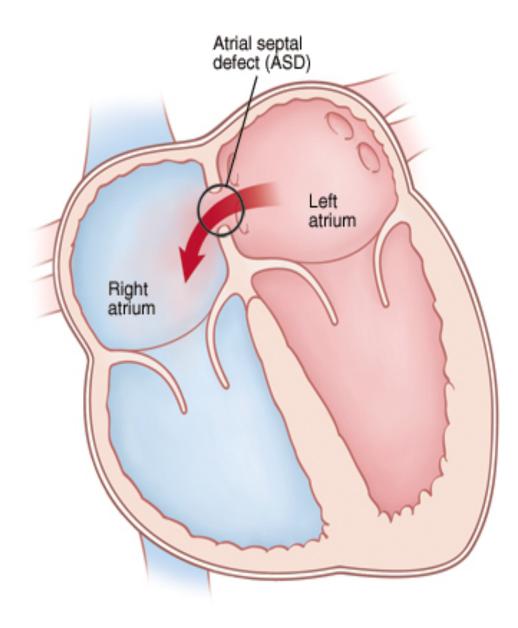
significant left-to-right VSDs are managed medically when signs of left-sided CHF occur.

The cyanosis and polycythemia that result from right-to- left VSDs may improve with sildenafil, which targets PH.

Interventional and Surgical Options

Transcatheter Occlusion Devices: Suitable for medium- to large-breed dogs with muscular VSDs.

Other VSD locations are less amenable to this approach due to proximity to aortic or pulmonic valves.



- ASDs are defects resulting from malformation of the septum primum and septum secundum.
- Pathophysiology of Atrial Septal Defects (ASDs):

Shunting Dynamics: Typically results in a left-to-right shunt of blood.

• Effects:

Dilation of the right heart. Pulmonary overcirculation. Right heart failure.

Clinical Presentation of Atrial Septal Defects:

- Murmur Characteristics: ASDs rarely cause a murmur due to low-volume, low-pressure gradients.
- Occasionally, a soft left basilar systolic murmur may be auscultated due to increased blood volume in the right ventricular outflow tract.
- Most ASDs remain asymptomatic.

- Severe Cases: Large defects or those with concurrent congenital defects may lead to right heart failure or shunt reversal.
- Signs include cyanosis and hypoxemia.

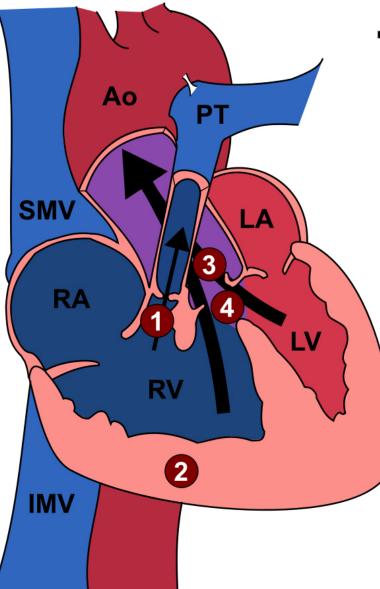
• Definitive diagnosis is made through echocardiography to visualize the defect.

• Management of Atrial Septal Defects:

Small ASDs: Often do not require treatment. Large ASDs: May result in right heart failure if untreated.

- Surgical intervention is challenging due to limited access and costs.
- Medical management of right heart failure if it develops.

• Prognosis varies depending on the size of the defect. Small ASDs: Generally have an excellent prognosis.



Tetralogy of Fallot

Major Defects



Pulmonary Stenosis



2 Right Ventricular Hypertrophy

3 Overriding Aorta



Ventricular Septal Defect



Component Defects:

- Ventricular septal defect (VSD)
- Dextroposition of the aorta
- Pulmonic stenosis (PS)
- Right ventricular concentric hypertrophy

Etiology of Tetralogy of Fallot (TOF)

• TOF is heritable in Keeshonds.



- Impact of Pulmonic Stenosis: Increases right ventricular pressures. Leads to various degrees of right-to-left shunting across the VSD.
- Cyanosis
- Hypoxemia
- Polycythemia

- Concurrent Conditions: Other congenital heart defects (CHD) may be present.
- Clinical Presentation of Tetralogy of Fallot :

Murmur Characteristics: Systolic murmur originating from PS and/or VSD.

Respiratory Difficulties: Episodes of respiratory difficulty occur when right-to-left shunting worsens, leading to systemic hypoxemia.

Differential Diagnosis for Tetralogy of Fallot:

- Pulmonic stenosis (PS)
- Ventricular septal defect (VSD)

Diagnosis of Tetralogy of Fallot

Thoracic Radiographs: Show right heart enlargement and pulmonary stenosis.

Echocardiography:

Visualizes the VSD and quantifies the severity of PS. Identifies displacement of the aortic root toward the right side. Assesses right ventricular hypertrophy.

Color-Flow Doppler

Management of Tetralogy of Fallot:

Medical Management:

- Aimed at limiting right-to-left shunting and hypoxemia.
- Use of oral nonselective beta-blockers (propranolol) to blunt decrease in systemic vascular resistance during exercise.
- Phlebotomies: Indicated when polycythemia is severe (PCV above 70%).

Prognosis

Prognosis varies based on the severity of the shunt and right ventricular pressure overload. Most dogs with TOF reach the age of 5 years.

Mitral Valve Dysplasia (MVD)

• VD results from malformation of the mitral valve apparatus, including valve leaflets, chordae tendineae, or papillary muscles.

• Predisposition: Large breed dogs are predisposed to MVD.

• Impact of Severe MVD: Causes significant mitral regurgitation. May lead to secondary changes similar to degenerative valvular disease.

• Long-Term Complications: Development of left-sided congestive heart failure (CHF).

Clinical Presentation of Mitral Valve Dysplasia (MVD)

- Murmur Characteristics: Left apical systolic murmur with intensity correlating to severity of regurgitation.
- Associated Signs: May include signs of arrhythmias or CHF.

• Differential Diagnosis for Mitral Valve Dysplasia (MVD)

Mitral regurgitation from degenerative valve disease Dilated cardiomyopathy (DCM)

Diagnosis of Mitral Valve Dysplasia (MVD)

• Thoracic radiographs and echocardiography may indicate varying levels of left-sided heart enlargement.

Signs of CHF: Pulmonary edema in dogs and Edema and/or pleural effusion in cats

• Echocardiographic Findings: Visualizes abnormalities in the mitral valve apparatus.

• Medical Management:

• Focus on treating congestive heart failure (CHF) and arrhythmias.

• **Prognosis:**

Generally poor due to early onset of arrhythmias and/or CHF.

Mitral Valve Dysplasia (MVD)

- MVD results from malformation of the mitral valve apparatus, including valve leaflets, chordae tendineae, or papillary muscles.
- Large breed dogs are predisposed to MVD.
- Severe MVD: Causes significant mitral regurgitation. And may lead to secondary changes similar to degenerative valvular disease.
- **Long-Term Complications:** Development of left-sided congestive heart failure (CHF).

Clinical Presentation of Mitral Valve Dysplasia (MVD)

• Murmur Characteristics: Left apical systolic murmur with intensity correlating to severity of regurgitation. May include signs of arrhythmias or CHF.

Differential Diagnosis for Mitral Valve Dysplasia (MVD)
 Mitral regurgitation from degenerative valve disease
 Dilated cardiomyopathy

Diagnosis of Mitral Valve Dysplasia:

- **Imaging Techniques:** Thoracic radiographs and echocardiography may indicate varying levels of left-sided heart enlargement.
- Pulmonary edema in dogs
- Edema and/or pleural effusion in cats
- Echocardiographic Findings: Reveals abnormalities in the mitral valve apparatus.

Management of Mitral Valve Dysplasia (MVD)

• Medical Management: Focus on treating congestive heart failure (CHF) and arrhythmias.

Prognosis:

• Generally poor due to early onset of arrhythmias and/or CHF.

• Tricuspid Valve Dysplasia:

- TVD results from malformation of the tricuspid valve apparatus, including valve leaflets, chordae tendineae, or papillary muscles
- Breed Predisposition:

TVD is the most common congenital heart defect in **Labrador Retrievers** and is heritable in this breed.

• TVD causes significant tricuspid regurgitation and can cause elevations in right atrial pressures and development of right-sided CHF.

• Clinical Presentation a right apical systolic murmur

Signs associated with arrhythmias or CHF may be present.

A common arrhythmia in dogs with TVD is atrial fibrillation (AF).

• Differential Diagnosis

Tricuspid regurgitation from degenerative valve disease, VSD, DCM.

Diagnosis

- **Radiographically**, dogs with TVD often have dramatic right atrial enlargement, a dilated caudal vena cava, and hepatomegaly.
- Cats have an enlarged right atrium and a dilated, tortuous caudal vena cava.
- Right heart failure in dogs may cause signs of jugular venous distension, hepatomegaly, and ascites.

• Echocardiography reveals structural abnormalities of the tricuspid valve apparatus



FIGURE 8.30 ECG from a dog with tricuspid dysplasia. There is a sinus rhythm with splintered QRS complexes (arrows) (amplitude 10 mm/mV, speed 50 mm/s).

• Management

TVD is treated medically by managing CHF and arrhythmias.

• Prognosis

Prognosis is poor when tricuspid valve regurgitation is severe due to the early onset of arrhythmias and/or CHF. Dogs with mild TVD have a normal life expectancy.

Breed Predispositions for Congenital Heart Disease

DISEASE	BREED
Patent ductus arteriosus	Maltese, Pomeranian, Shetland Sheepdog, English Springer Spaniel, Keeshond, Bichon Frise, Toy and Miniature Poodles, Yorkshire Terrier, Collie, Cocker Spaniel, German Shepherd Dog, Chihuahua, Kerry Blue Terrier, Labrador Retriever, Newfoundland, Welsh Corgi; female > male
Subaortic stenosis	Newfoundland, Golden Retriever, Rottweiler, Boxer, German Shepherd Dog, Great Dane, German Short-Haired Pointer, Bouvier des Flandres, Samoyed (valvular aortic stenosis: Bull Terrier)
Pulmonic stenosis	English Bulldog (male > female), Mastiff, Samoyed, Miniature Schnauzer, West Highland White Terrier, Cocker Spaniel, Beagle, Labrador Retriever, Basset Hound, Newfoundland, Airedale Terrier, Boykin Spaniel, Chihuahua, Scottish Terrier, Boxer, Chow, Miniature Pinscher, other terriers & spaniels
Ventricular septal defect	English Bulldog, English Springer Spaniel, Keeshond, West Highland White Terrier; cats
Atrial septal defect	Samoyed, Doberman Pinscher, Boxer
Tricuspid dysplasia	Labrador Retriever, German Shepherd Dog, Boxer, Weimaraner, Great Dane, Old English Sheepdog, Golden Retriever; other large breeds (male > female?); cats
Mitral dysplasia	Bull Terrier, German Shepherd Dog, Great Dane, Golden Retriever, Newfoundland, Mastiff, Dalmatian, Rottweiler (?); cats (male > female)
Tetralogy of Fallot	Keeshond, English Bulldog
Persistent right aortic arch	German Shepherd Dog, Great Dane, Irish Setter

