10

Congenital Heart Diseases

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Introduction

Congenital disease of the heart can often be complex and confusing. Understanding normal circulation can be challenging, but when it gets deranged in development, a new level of confusion can ensue. Congenital heart disease typically changes the loading conditions in the cardiac chambers in one of two ways: by increasing the pressure load or by increasing the volume load in one or more heart chambers. Combinations of the two loads can exist. By understanding if the condition presents a volume load or a pressure load will aid in the diagnosis and therapy of patients with congenital heart disease.

The prevalence of congenital cardiac disease in dogs was reported to be approximately 0.5–0.85% in 1992 [1]. The Veterinary Medical Database (VMD) is a central storage database for diagnosis information of cases submitted by five veterinary teaching institutions. The author reviewed the VMD from 1965 to 2003 which revealed that of all dogs entered, 0.09% had a diagnosis of some type of congenital cardiac defect. The most commonly reported congenital heart defect was the patent ductus arteriosus (PDA), followed by subvalvular aortic stenosis (SAS) and pulmonic valve stenosis (PS). Some breeds of dogs are overrepresented with certain congenital defects, such as SAS in the golden retriever. Table 10.1 shows the most common congenital defects and the dog breeds in which they are most frequently seen.

Classifications of Congenital Heart Disease

A thorough knowledge of normal cardiac anatomy is essential in understanding congenital defects. Grouping cardiac congenital defects by the hemodynamic consequences they cause aids in understanding their development. Valvular dysplasias are malformations of the cardiac valve apparatus that result in either a stenosis, regurgitation, or both. Stenosis is seen in a valve orifice that is too small for the workload required of it. It is analogous to a narrowing in a river that causes the increased speed of flow and turbulence that are rapids. Dysplasias can be seen at any of the four cardiac valves and tend to create increased pressure in the cardiac chamber upstream.

Shunts are abnormal hemodynamic communications between two portions of the cardiovascular anatomy. The most commonly seen shunts are patent ductus arteriosus

Cardiology for Veterinary Technicians and Nurses, First Edition. Edited by H. Edward Durham, Jr.

Table 10.1 The commonest congenital heart defects and the most susceptible dog breeds for each disease.

Patent ductus arteriosus	
	Bichon Frise
	Chihuahua
	Cocker Spaniel
	Collie
	English Springer Spaniel
	German Shepherd
	Keeshond
	Labrador Retriever
	Maltese
	Newfoundland
	Poodle Breeds
	Pomeranian
	Shetland Sheepdog
	Yorkshire Terrier
Subaortic stenosis/aortic stenosis	
	Bouvier de Flandres
	Boxer
	Bull Terrier
	English Bulldog
	German Shepherd
	German Shorthair Pointer
	Golden Retriever
	Great Dane
	Newfoundland
	Rottweiler
	Samoyed
Pulmonic stenosis	
	Airdale Terrier
	Beagle
	Boykin Spaniel
	Boxer
	Chihuahua
	Cocker Spaniel
	English Bulldog
	Mastiff

Table 10.1 (*Continued*)

Pulmonic stenosis (*continued*)	
	Samoyed
	Schnauzer Breeds
	Terrier Breeds
	West Highland White Terrier
Ventricular septal defect	
	English Bulldog
	English Springer Spaniel
	Keeshond
	West Highland White Terrier
Tricuspid valve dysplasia	
	Boxer
	German Shepherd
	Golden Retriever
	Great Dane
	Labrador Retriever
	Old English Sheepdog
	Weimaraner

(PDA), ventricular septal defects (VSD), or atrial septal defects (ASD). Most shunts flow from the systemic circulation to the pulmonary circulation due to the greater pressure of the systemic circulation, and tend to create an increased volume load on the heart. Cyanotic heart diseases are typically shunts in which hypoxic blood from the pulmonary circulation enters the systemic circulation, leading to systemic hypoxia and cyanosis. Congenital cardiac malformations do not always present as a single defect. The term complex congenital defect simply refers to abnormalities that may include more than one of the above classifications, such as a PDA with a PS. Virtually all significant congenital cardiac defects cause a murmur that can be heard during auscultation, but exceptions do occur. Generally, the loudness of the murmur does not correlate well with the severity of the disease. Echocardiography has become the most common modality for diagnosing congenital heart defects. An algorithm has been proposed to aid is sorting out the complex cardiac abnormalities commonly seen with congenital heart disease [2].

Subaortic Stenosis

Subvalvular aortic stenosis or subaortic stenosis (SAS) is an important cardiac congenital defect seen in Newfoundlands, golden retrievers, boxers, Rottweilers and other large-breed dogs. It is notable that the defect is rarely diagnosed in Labrador retrievers. This defect is a fibromuscular ring that develops in the left ventricular outflow tract (LVOT)

between the left ventricle (LV) and the valve proper. The fibrous ring creates an obstruction to blood flow from the LV through the aortic valve. The severity of SAS is determined by degree of pressure load added to the LV to eject blood. Subaortic stenosis is present at birth in affected animals, and can progress in severity up to approximately one year of age, at which time a final diagnosis of severity can be made.

The increased pressure in the LV can lead to ventricular hypertrophy, decreased coronary perfusion, myocardial ischemia, and fibrosis [3]. The amount of these changes is related to the severity of the stenosis and the amount of pressure overload it exerts on the LV. Severe obstructions lead to marked LV hypertrophy and ischemia, and mild obstructions showing little or no obvious hypertrophy.

Clinical Presentation

Often this condition has no outward clinical manifestations until the onset of heart failure, or in the cases of severe stenosis, sudden death may be the first sign. Typically, SAS is first suspected when a murmur is heard during a routine veterinary examination in breeds that are predisposed. This usually occurs during the well-puppy visit. The physical examination may reveal an audible, left basilar systolic murmur, as well as hypokinetic femoral pulses. The murmur often is a crescendo or diamond-shaped murmur, a so called "ejection" murmur. The loudness of the murmur does not always coincide with the severity of the stenosis, but generally very loud murmurs (grades 4–6/6), are often associated with severe disease, and may radiate cranially through the carotid arteries making the murmur audible on the dog's head.

Diagnostics

Radiographs typically show a normal-size cardiac silhouette in the lateral projection with occasional loss of the cranial cardiac waist. From the ventrodorsal (VD) view there is widening of the mediastinum caused by a poststenotic dilation of the aortic arch, with a bulge over the cranial heart projecting toward the patient's left side (Figure 10.1). Left ventricular and left atrial (LA) enlargement may rarely be seen with severe SAS that also affects the mitral valve as a result of mitral regurgitation.

Echocardiography is the standard of care in diagnosing and prognosticating for a SAS. Echocardiographic findings correlate with severity. Echocardiography of mild to moderate SAS typically shows that left ventricular dimensions typically remain within normal limits. In severe SAS some degree of LV hypertrophy may be appreciated and the fibrous ring that creates the obstruction of a narrowed LVOT is sometimes visible. This "imaging lesion" appears as a ridge of tissue on the septal and posterior wall on the LVOT (Figure 10.2). Some dogs will also have abnormalities in their mitral valve leading to mitral regurgitation and potentially LA enlargement.

Using Doppler echocardiography with color flow mapping, an image of the aorta will show turbulent blood flow during systole in the aorta (Figure 10.3). During diastole, aortic insufficiency is commonly seen [3,4] (Figure 10.4).

Definitive diagnosis is acheived by measuring the velocity of blood moving across the aortic valve recorded with spectral Doppler imaging (Figure 10.5) (see Chapter 6). Doppler-derived velocities over 2.25 m/s are considered abnormal in all breeds of dog [1, 3]. The velocity is translated into a pressure gradient between the LV and the aorta

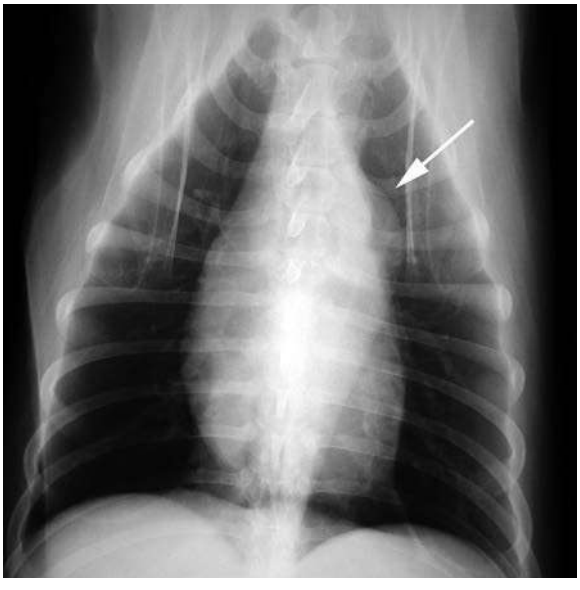

Figure 10.1 A ventrodorsal radiograph of a dog with subaortic stenosis: note the aortic bulge in the 1–2 o'clock region indicated by the arrow. This is a result of the poststenotic dilation of the aortic arch. The remaining cardiac silhouette is normal size and shape. The pulmonary parenchyma is normal, as are the other thoracic vascular structures.

by use of the modified Bernoulli equation, which states that four times the velocity squared will equal the pressure difference or *pressure gradient* (PG) between two chambers (Equation 10.1). Doppler assessment of the aortic outflow velocity is done from the left apical five-chamber view and the subcostal view. The subcostal view has been shown to provide the best alignment for measuring the PG across the aorta [5].

$$4^{*}V^{2} = \Delta P \tag{10.1}$$

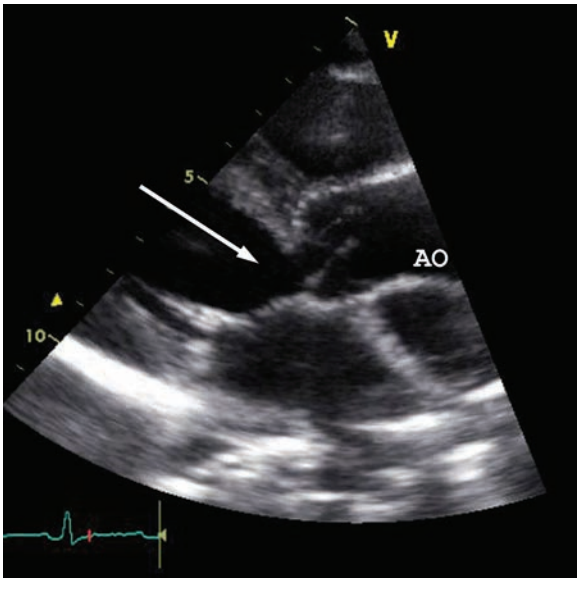

Figure 10.2 Long axis echocardiographic view of subaortic stenosis lesion: the arrow points to the narrowed left ventricular outflow tract. The fibrous obstruction protrudes downward in the image. The aortic valve cusps are seen centrally between the arrow and the aorta (AO) label. They are extending upwards and towards the right. The left atrium is in the far field, and the right atrium in the near field.

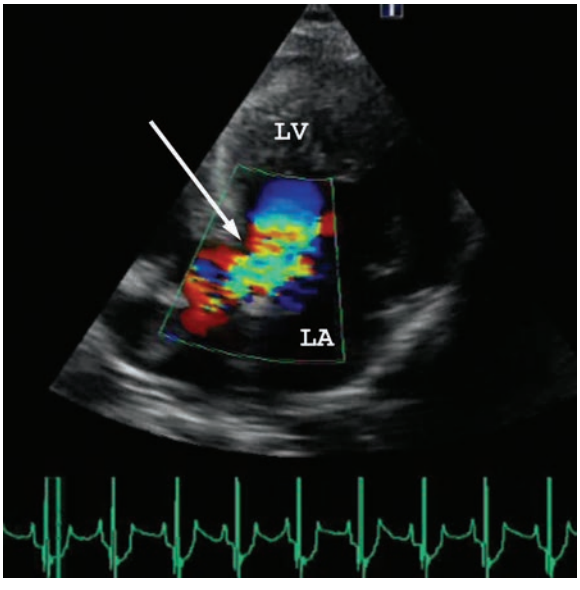

Figure 10.3 Left apical long axis view of turbulence in the aorta: the yellow–green mosaic of high velocity turbulent blood flow by color-flow Doppler in the aorta during ejection is typical for subaortic stenosis. The left atrium (LA) and left ventricle (LV) are labeled. Notable in the color mapping is the blue shell of downward flow nearest the top of the image, which as blood accelerates changes from flat blue to ice blue to high velocity variant flow.

where V = Doppler-measured blood velocity expressed in m/s and ΔP = the PG between the two cardiac chambers that the blood was moving between during the velocity measurement.

Although the exact classification of SAS severity is not uniform among veterinary cardiologists, most agree that dogs with a PG of 25–50 mmHg are classified as having mild SAS, those with 50–80 mmHg as moderate, and those with 80 mmHg or above as having severe SAS [4; 6, pp. 477–525] in nonanesthetized dogs. Some authors classify severe

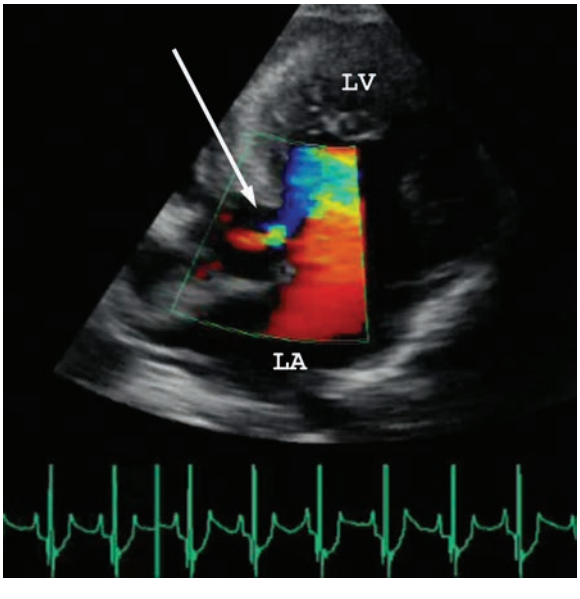

Figure 10.4 Left apical long axis view of aortic insufficiency: the arrow points to the red retrograde flow through the aortic valve during diastole of aortic insufficiency commonly seen in subaortic stenosis cases. The left atrium (LA) and left ventricle (LV) are labeled. The red Doppler signal of blood filling the LV from the LA can be seen to the right of the aortic valve.

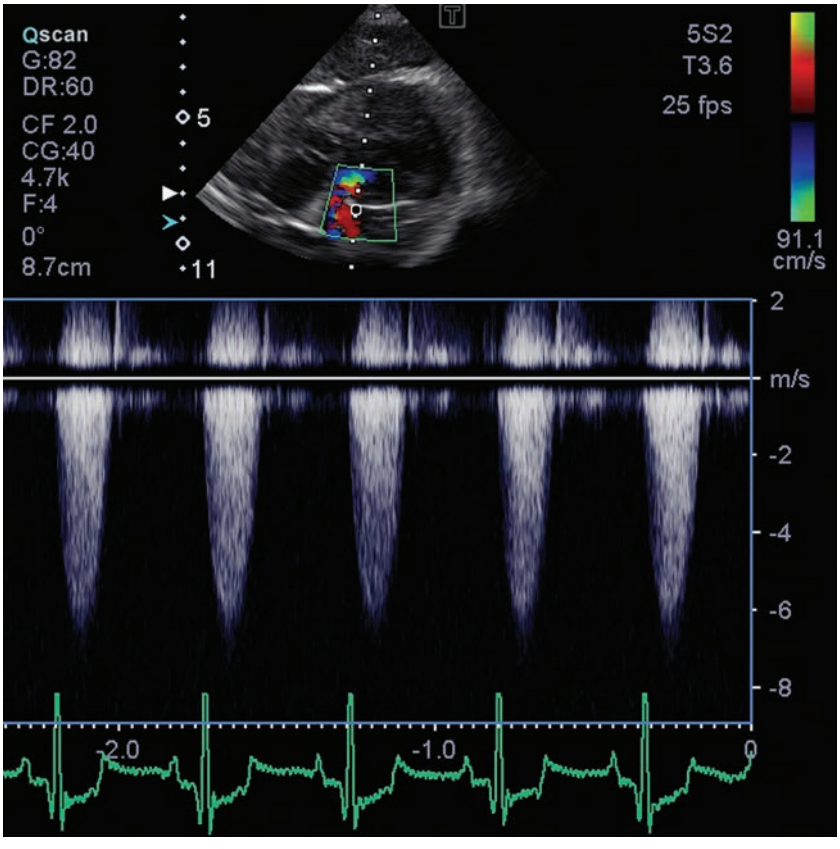

Figure 10.5 Continuous wave spectral Doppler of severe subaortic stenosis. The spectral Doppler image here shows the peak velocity across the stenotic aortic valve to be greater than 6 m/s, which corresponds with a peak pressure gradient of greater than 144 mmHg. Dogs with pressure gradients of greater than 130 mmHg have an increased risk of morbidity and mortality. The velocity scale is on the right of the spectral graph. An ECG is provided for timing of the cardiac cycle. The cursor line can be seen aligned with the color of turbulent blood flow in the reference image above.

SAS as a PG above 100 mmHg [1,3]. Research from 2014, however, demonstrated that dogs with a PG equal to or less than 133 mmHg, were at no greater risk of mortality than those with a PG between 80 and 130 mmHg, suggesting that reclassification of severe SAS is warranted [7].

It must be remembered that the transaortic velocity is dependent on many factors. Increased sympathetic tone or cardiac shunts may falsely elevate velocity, whereas negative inotropic drugs, anesthesia, and myocardial failure will decrease transaortic velocities. Normal velocities are generally considered to be less than 1.7 m/s, thus an equivocal zone exists between the normal 1.7 m/s and the clearly abnormal of 2.25 m/s [3]. Owners with breeding dogs that fall in this category must counsel with the veterinary cardiologist carefully to determine the dog's potential to pass on an inheritable heart condition to their offspring.

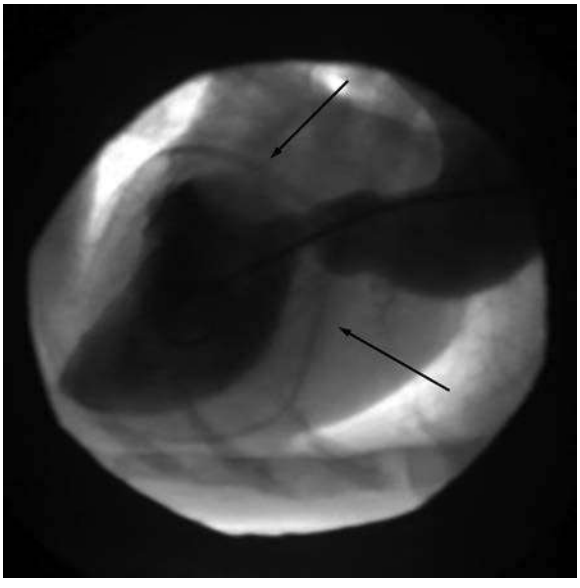

Figure 10.6 Angiogram of subaortic stenosis: the pigtail catheter is entering the image from the right side. The catheter is placed in the carotid artery and advanced through the aortic root past the aortic valve, and into the left ventricle. An injection of contrast is made into the left ventricle. This frame is taken at end diastole. The narrowing of the stenosis can be seen between the left ventricle and the aorta. The arrows point to the coronary arteries as they wrap around the ventricles. The left ventricle should have an opening into the aorta as wide as the aortic root. The poststenotic dilation can be seen to the right of the image.

Prior to the widespread use of echocardiography to diagnose SAS, many cases were diagnosed via cardiac catheterization. During cardiac catheterization a PG could be measured directly by means of intracardiac catheters and pressure transducers. Anesthesia will reduce the PG by approximately half as compared with Doppler derived PG [8]. Additionally, radiographic contrast imaging of the left ventricular outflow tract could be performed (Figure 10.6).

Therapy

Currently, there is no curative treatment for SAS. Several methods have been attempted to partially relieve the degree of obstruction for severe cases in an attempt to move dogs to the moderate category including: balloon valvuloplasty (Figure 10.7), open-heart surgical intervention [9], interventional catheters with cutting balloons [10] and medical therapy with beta-blockers. None of these treatment options have been shown to extend life better than another. At this time beta-blocker therapy is the most widely accepted treatment due to the low-risk-to-benefit ratio [3]; however, the benefits may not be as hopeful as initially thought [7]. If signs of congestive heart failure (CHF) and/or arrhythmias become present then traditional therapy with diuretics, angiotensin-converting enzyme inhibitors and anti-arrhythmic therapy is warranted (see Chapter 15).

Patients with mild stenosis generally live normal lives with no clinical signs. Dogs with moderate SAS may develop syncope, CHF and/or ventricular arrhythmias; however, they may live a full life with the disease. Dogs with severe SAS may show syncope or will often die of CHF or of ventricular arrhythmias. Any degree of SAS predisposes the patient to bacterial endocarditis. Because the obstruction is actually not the valve leaflets themselves but "below" the valve, the high velocity jet impacts the valve leaflets at the onset of systole. Over time the damage from this impact damages the valve leaflet

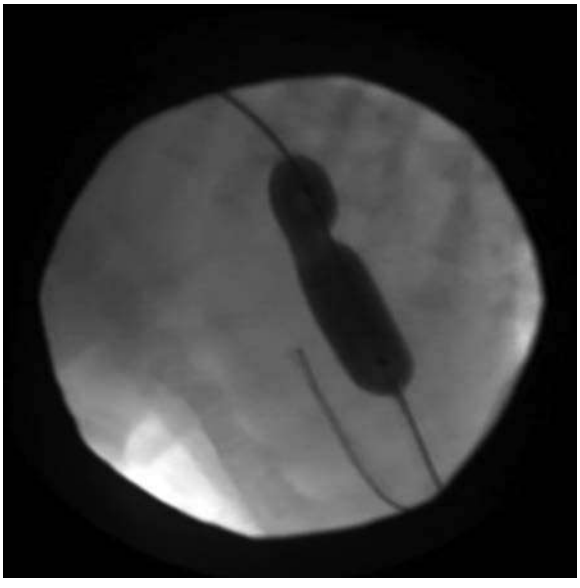

Figure 10.7 Fluoroscopic view of aortic balloon valvuloplasty: a cardiac guidewire has been passed from the carotid artery to the aorta and across the aortic valve into the left ventricle. It can be seen curling back towards the top of the heart. The interventional balloon can be seen inflated with contrast agent across the stenosis. A visible waist is noted in the balloon where the stenosis is located.

surface, roughening them which allows for adherence of bacteria present in the bloodstream from normally occurring bacteremia (see Chapter 11). The general recommendation for any dog suspected or diagnosed with subaortic stenosis undergoing surgical or invasive procedures (including dental prophylaxis, spay or neuter, endoscopy, or severe infections) is to be provided with an extended course of prophylactic antibiotics.

Pulmonic Stenosis

Pulmonic stenosis is a malformation of the pulmonic valve. Unlike subaortic stenosis, PS occurs in the valve itself. This anomaly can present in different forms. One variation is a narrowed annulus with fairly normal valve leaflets. A second version appears as a normal valve annulus with tethered and fused leaflets. Pulmonic stenosis is most often seen in terrier breeds, bulldogs, and other small breed dogs. Although PS can occur in any breed, it is not commonly seen in large-breed dogs except for the boxer and to a lesser extent, the Labrador retriever. Bulldogs and boxers present an unusual challenge for the veterinary cardiologist since they can develop a third type of pulmonic stenosis that involves the coronary arteries. In dogs, two coronary arteries typically leave the aortic root, one to the left heart and one to the right. In 1990, four variations of the origin of the right coronary artery were described, subcategorized as R1 and R2. The R1 anomaly is a single coronary artery that circumnavigates the entire heart with no major branches. The R2 anomaly is an artery of single aortic origin, but branches into right and left main branches. Three different variations have been reported: R2a, R2b, and R2c. The R2b and R2c do not cause any clinical sign, so the prevalence in the general population is hard to predict. The R2a anomaly is a cause of pulmonic stenosis (Figure 10.8). The R2a anomaly occurs mostly in bulldogs and boxers in which the right coronary artery may be absent,

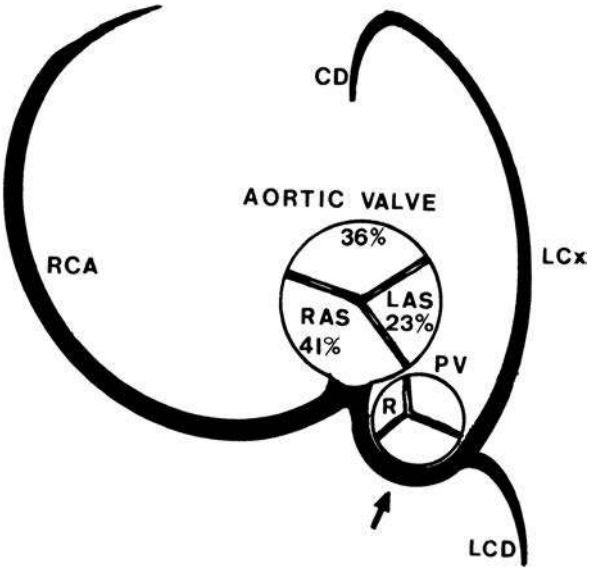

Figure 10.8 A single coronary artery arrangement in pulmonic stenosis: the single left coronary artery (arrow) exits the aorta towards the bottom of the image and courses over the pulmonic valve (PV) narrowing the orifice. The pulmonic cusps and sinuses (R) are hypoplastic. The mean portion of aortic circumference associated with each cusp for a small group of dogs are indicated by the percentages. The left aortic sinus (LAS) does not have a coronary sinus and represents only ~23% of aortic circumference. The right cusp and sinus (RAS) is nearly twice the size of the LAS. The left cranial descending coronary artery (LCD) arises from the anomalous left main coronary ostium. The right coronary artery (RCA) traverses the right ventricle. The caudal descending coronary artery (CD) is a terminal branch of the left circumflex coronary artery (LCx). Source: Buchanan (1990) [11]. Reproduced with permission of JAVMA.

causing a branch of the left coronary artery to literally wrap around the pulmonary artery creating the stenosis [11].

Clinical Presentation

Similar to SAS, PS is often suspected after routine examination when a left basilar systolic murmur is heard. The intensity of the murmur correlates somewhat with the severity of the stenosis, but loud murmurs may be present with moderate stenosis. This may be due to the relatively small size of the most common patients seen with PS vs SAS, creating loud murmurs with even moderate PG. Similar to SAS, the quality of the murmur often is an ejection quality (crescendo or diamond-shaped) murmur loudest at the left heart base. Other clinical manifestations of PS include exercise intolerance, syncope, and ascites from right ventricle (RV) failure. A palpable thrill is often felt in cases with severe PS in the left axillary region.

Diagnostics

Radiographic findings will show evidence of right ventricular and right atrial enlargement with a prominent poststenotic dilation of the main pulmonary artery visible in

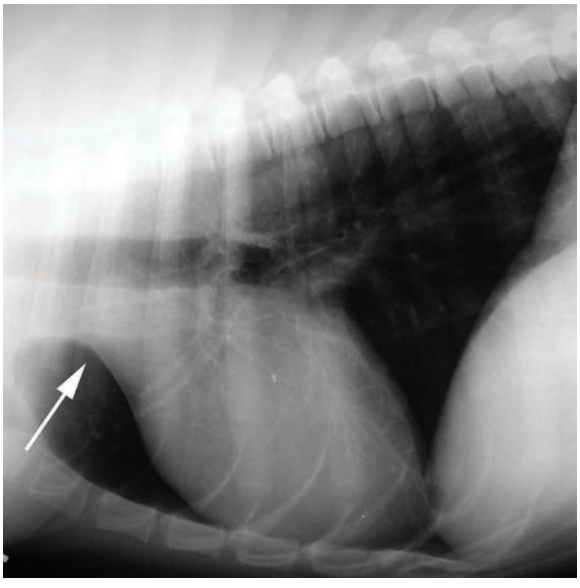

Figure 10.9 Lateral radiograph of a dog with pulmonic stenosis: the arrow in this image points to the enlarged pulmonary artery. It is noticed as a loss of the cranial waist in the cardiac silhouette. The overall cardiac silhouette is normal in shape and size. A normal reference image is available in Chapter 5.

both the lateral and VD views (Figures 10.9 and 10.10). The remaining pulmonary vascular may appear to be under circulated. If signs of right-heart failure are present then a dilated caudal vena cava may also be seen.

An electrocardiogram can be useful in distinguishing SAS from PS by calculating the mean electrical axis (MEA) (see Chapter 4). Right ventricular hypertrophy will cause the MEA to deviate toward the right axis in PS, while MEA remains normal in SAS, even

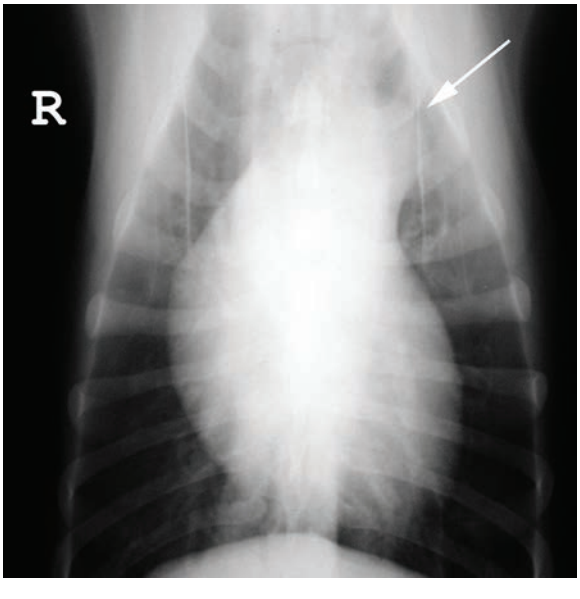

Figure 10.10 Ventrodorsal radiograph of a dog with pulmonic stenosis: the arrow in this image indicates the poststenotic dilation creating a noticeable bulge at the left cranial aspect of the cardiac silhouette. Using the clock-face analogy, the bulge is at 1 o'clock.

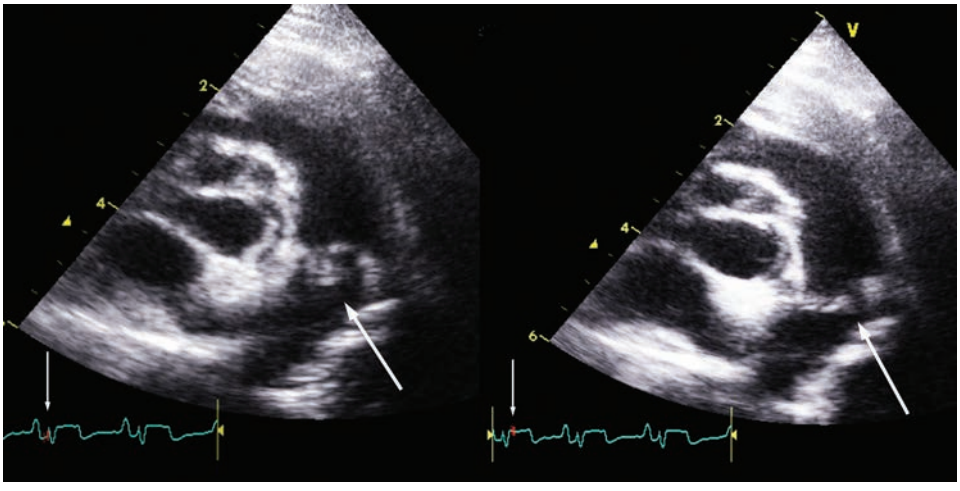

Figure 10.11 Echocardiographic view of a stenotic pulmonic valve: this short axis view of the heart base shows a stenotic pulmonic valve. The twin image shows the pulmonic valve at the start of systole. The small arrows at the bottom shows the electrocardiogram (ECG) indicating the red line in the early QRS complex. The ECG demonstrates a right axis shift in the predominantly negative QRS complex. The left image is just before the valve should open. The valve cusps (large arrows) can be seen as thickened and billowing upwards towards the right ventricle. The right image shows the cusps as they try to open. In a normal pulmonic valve, the cusps should not be visible during systole, but in this image they are prominent and thick.

in the presence of LV hypertrophy. The "P" wave amplitude can measure greater than normal ("P" pulmonale) if right atrial enlargement is present.

Echocardiography is the best method for making the definitive diagnosis. Echocardiographic features of PS are right ventricular hypertrophy, enlargement of the right atrium (RA), poorly mobile, tethered pulmonic cusps, and/or a hypoplastic valve annulus and poststenotic pulmonary artery dilation (Figure 10.11). The increase in pressure and lack of forward flow through the pulmonic valve (PV) also affects the LV. In severe PS, a decreased LV internal dimension and flattening of the interventricular septum (IVS) is usually present (Figure 10.12). Evaluation with Doppler color mapping can show tricuspid regurgitation (TR) and due to the elevated pressure in the RV being transmitted through the tricuspid valve to the RA, a patent foramen ovale (PFO) is sometimes noted. An air-contrast echocardiogram is useful in confirming the suspected diagnosis of a PFO (Figure 10.13) (see Chapters 1 and 6).

Color Doppler of the pulmonic valve will demonstrate highly turbulent blood flow and insufficiency of the pulmonic valve is also noted [1] (Figure 10.14). Spectral Doppler interrogation of transpulmonic flow reveals elevated velocities across the pulmonic valve (Figure 10.15). Pressure gradients greater than 80–100 mmHg are considered to be severe, pressures between 50–80 mmHg are moderate and less than 50 mmHg are mild pulmonic stenosis [1, 3]. However, a 2011 study suggests that dogs with a PG greater than 60 mmHg are at increased risk of cardiac mortality [12]. The transpulmonic valve velocities can be acquired from the right parasternal short axis basilar view, or from

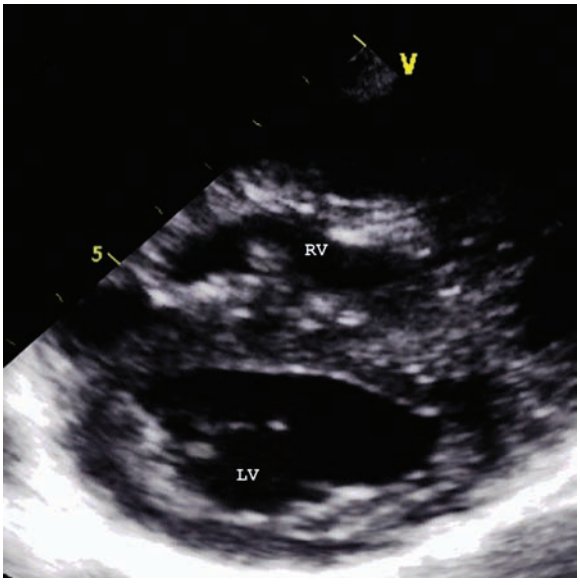

Figure 10.12 Echocardiographic short axis view of ventricles in canine pulmonic stenosis: the right ventricle (RV) at the top of the image exhibits RV hypertrophy. The RV should be approximately one-third as thick as the left ventricle (LV). The LV is approximately normal thickness, while the RV is seen as the same as the LV. The intraventricular septum is flattened rather than rounding towards the RV due to the excessive RV pressure. A normal reference image is available in Chapter 6.

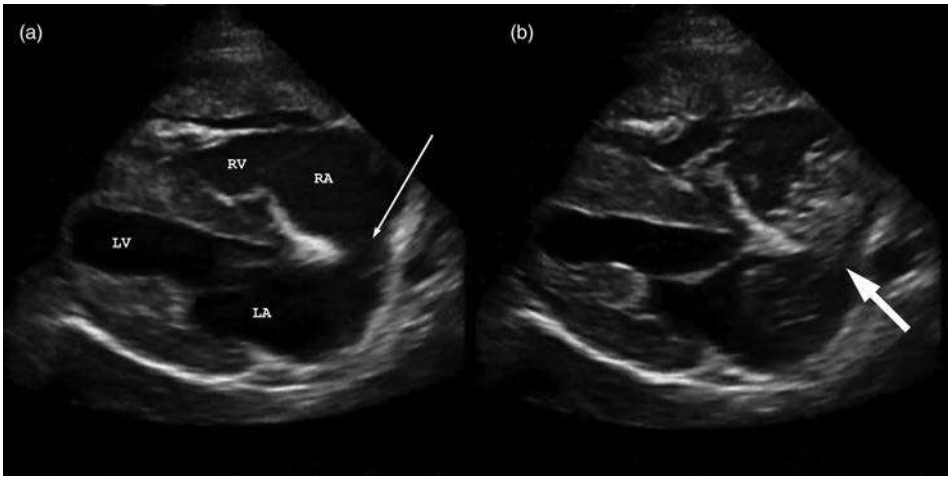

Figure 10.13 Echocardiographic image of a patent foramen ovale (PFO): this twin image shows a long axis four-chamber of a canine heart with pulmonic stenosis. The chambers are labeled as: LV, left ventricle; LA, left atrium; RA, right atrium; RV, right ventricle. (a) The heart just before a microbubble air-contrast echocardiogram. The PFO is apparent by the hypoechoic area in the interatrial septum (arrow). The right ventricle is hypertrophied in response to the increased work added to it by the stenotic valve. The left ventricle is decreased in chamber dimension due to poor cardiac output from the right ventricle. The RA appears slightly larger than the LA and the RV papillary muscle and tricuspid chordae tendineae are visible as the hyperechoic structure in the RV. (b) The microbubbles entering the RA from the right of the image during later diastole frame. An injection of agitated saline was made into a cephalic vein. As the microbubbles enter the RA, increased pressure in the RA from tricuspid regurgitation as a result of a pressure overload of the RV pushes the bubbles into the LA through the PFO (wide arrow). The microbubble air-contrast echocardiogram is diagnostic for a right-to-left shunting septal defect.

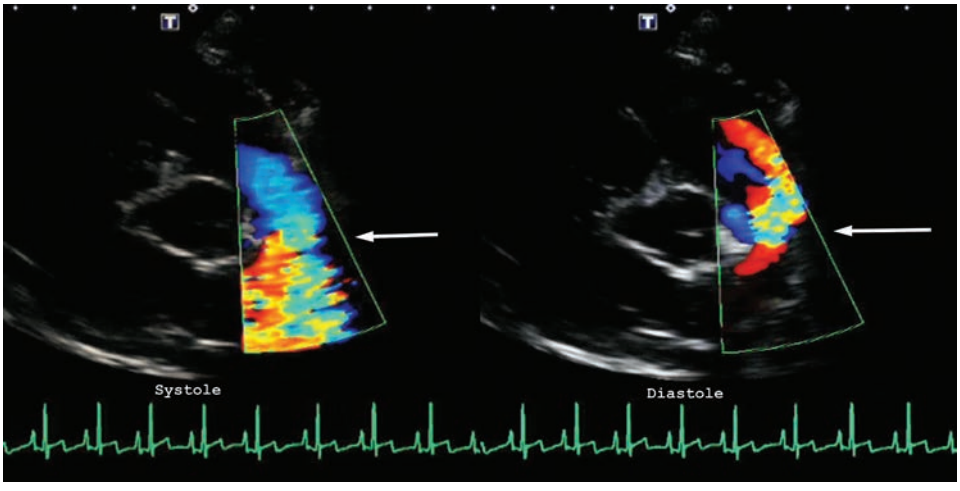

Figure 10.14 Doppler color-flow mapping of pulmonic stenosis: this twin image shows turbulent blood flow across the pulmonic valve in systole (left image) and diastole (right image). The arrows indicate the level of the valve for reference. During systole, blood moves downward in this view and is coded in blue, until it reaches the valve, where it increases in velocity and becomes turbulent. The yellow–green mosaic of color beyond the valve indicates turbulence. In diastole, the incompetent valve allows blood to flow retrograde into the right ventricle or upwards in this view and is coded as red. This pulmonic insufficiency also is somewhat turbulent.

the left cranial right ventricular outflow tract view [6, pp. 37–99] with transthoracic echocardiography.

Cardiac catheterization of the RV allows for recording of elevated RV pressure. Angiography demonstrates the narrow orifice of the pulmonic valve and the prominent poststenotic dilation (Figure 10.16).

Therapy includes medications, surgery or balloon valvuloplasty via cardiac catheterization. Cases of mild to moderate PS can often be very successfully managed with medical therapy alone or may not require any treatment. These patients may show no clinical signs other than exercise intolerance after periods of heavy activity. Patients with severe PS often benefit from an interventional procedure to reduce their PG in the mild to moderate range. Negative inotropic drugs such as beta-blockers or calcium channel blockers are used to treat PS when definitive therapy is not an option or as a bridge to intervention.

Several surgical procedures have been tried in dogs with PS, but most require extracorporeal perfusion and are not cost-effective for most clients. Surgeries to open the pulmonic orifice can be performed using a tubular graft to create a conduit around the stenosis [11] (Figure 10.17). Placement of an overlaying patch graft to create a structurally sound aneurysm around a bisected valve or similar techniques have been used [13, 14]. Valve replacement requires cardiopulmonary bypass.

Balloon valvuloplasty is a widely utilized treatment for dogs since open heart surgery is often risky and cost prohibitive. Balloon valvuloplasty is a cardiac catheterization procedure which can be performed via a transvenous catheter introducer whereby catheters are advanced into the right heart from the jugular vein following flow [15, 16]

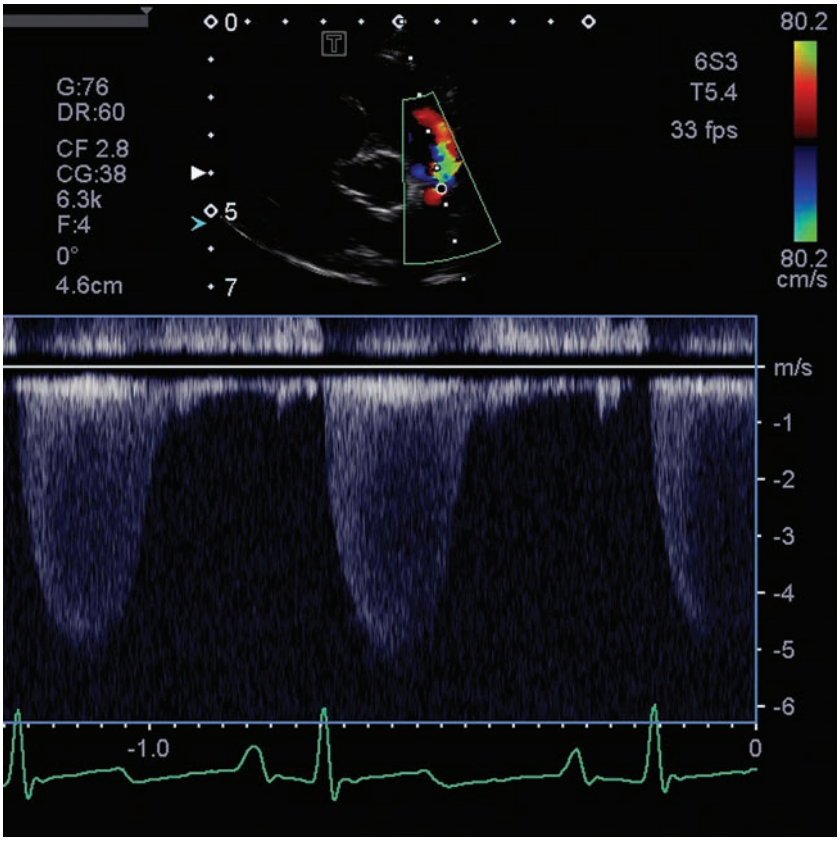

Figure 10.15 Spectral continuous wave Doppler of transpulmonic blood flow: the top portion of the image shows the reference echocardiographic image, and the Doppler cursor can be seen passing through the pulmonic valve area. The aorta is seen in cross-section in the center of the heart. The graph is a recording of the transpulmonic blood velocity. Normal blood velocity across the pulmonic valve is typically equal or less than 1.5 m/s. In this example the velocity scale on the right indicates a velocity of approximately 5 m/s which corresponds with a pressure gradient (see Chapter 6) of approximately 100 mmHg. This would equate to the right ventricle generating approximately 125 mmHg or five times greater than normal right ventricular pressure to eject blood though the stenotic valve.

(see Chapters 8 and 16). A special interventional balloon catheter is inflated across the valve to dilate it (Figure 10.18). The goal is to open the valve leaflets and subsequently reduce the transpulmonic PG. During this procedure it is possible to measure the PG directly with catheterization before and after the procedure. Balloon valvuloplasty is particularly suited to dogs with valve fusion and a normal valve annulus. Dogs with a hypoplastic valvular annulus may still benefit, but the outcome is not as positive and these dogs may see their PGs rise again over time. Evidence indicates that for dogs with high, moderate and severe PS, some intervention does improve their quality and quantity of life [17].

The R2a anomaly eliminates balloon valvuloplasty as a treatment option, as inflation of the balloon would rupture the coronary artery causing immediate death (Figure 10.19).

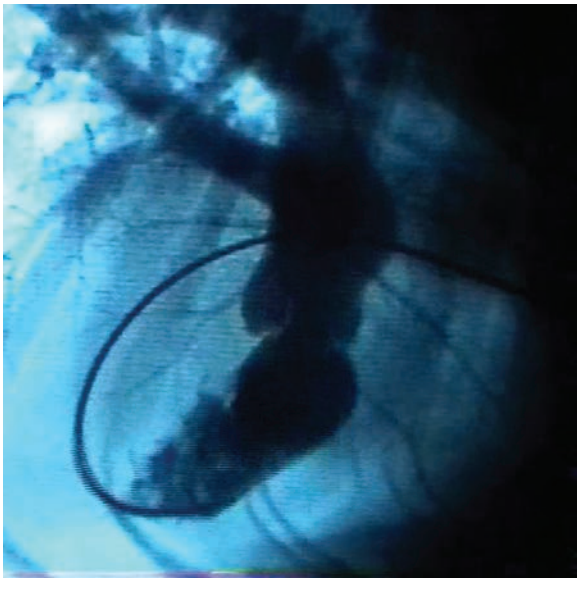

Figure 10.16 Fluoroscopic angiogram of canine pulmonic stenosis. The catheter is entering the image on the right from the cranial aspect of the patient; the spine is at the top. The catheter runs from the jugular vein, cranial vena cava, right atrium, then finally through the right ventricle and out of the pulmonic valve. A radiopaque contrast is injected into the right ventricle. A prominent narrowing separates the pulmonic root (upper) and right ventricular outflow tract (lower). In the center of the stenosis is a dark contrast jet of maximal flow. The subtle noncontrasted light lines to either side of the central jet are the restricted valvular cusps. The poststenotic dilation is above the pulmonic root. The two pulmonary arterial branches and some of the pulmonary vasculature can also be seen.

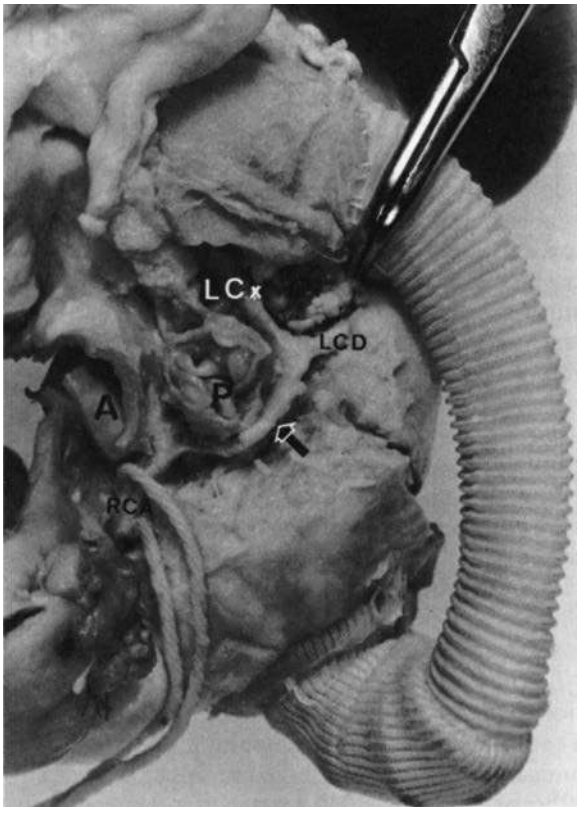

Figure 10.17 Pulmonic bridging conduit postmortem in a dog with R2a coronary artery anomaly: the anomalous left main coronary artery (arrow) runs cranial and adjacent to the constricted pulmonic valve (P) before dividing in the left circumflex (LCx) left cranial descending (LCD) coronary arteries. The right coronary artery (RCA) courses in a normal fashion after bifurcating a few millimeters away from the large right aortic sinus (A). Source: Buchanan (1990) [11]. Reproduced with permission of JAVMA.

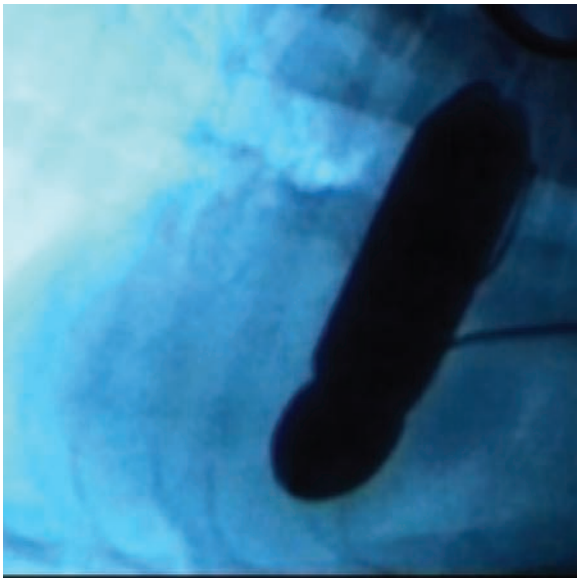

Figure 10.18 Fluoroscopic image of pulmonic balloon valvuloplasty. This image shows an interventional dilation balloon for pulmonic stenosis being inflated. The narrowed waist is at the point of the valvular stenosis. The balloon is inflated for several seconds, then deflated; this is repeated two to three times to achieve maximal effect. The catheter portion of the interventional balloon is entering the image on the right. The catheter runs from the jugular vein, cranial vena cava, right atrium, then finally through the right ventricle and out of the pulmonic valve.

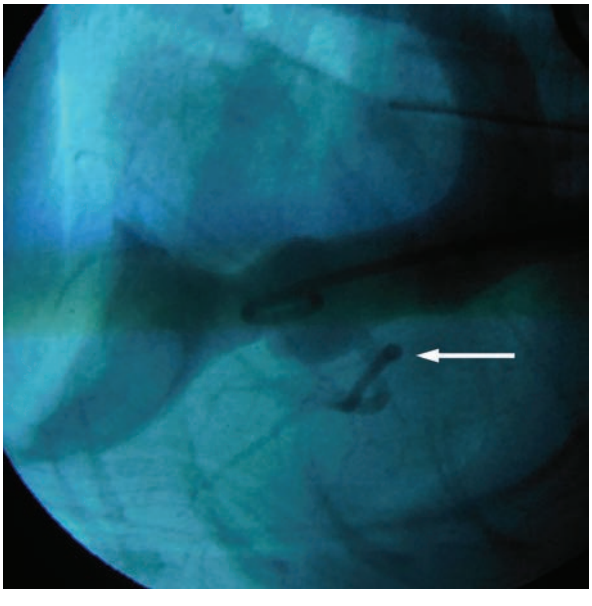

Figure 10.19 Fluoroscopic angiogram of canine single coronary aortic root: the pigtail catheter entering from the right is being advanced from the carotid artery and is positioned atop the aortic valve. A radiopaque contrast is injected to record this diastolic angiogram. The single coronary artery can be seen exiting the aorta to the lower right, and traverses over the pulmonary trunk (arrow). Smaller coronary branches may be appreciated coursing around the outer surface of the heart. There is aortic regurgitation, most likely due to the catheter deforming the aortic valve. Figure 8.14 demonstrates normal coronary arterial courses. A catheter may also be seen in the upper right quadrant of the image. The catheter is being advanced through the jugular vein, to the cranial vena cava, and sits resting in the right atrium in preparation for a right ventriculogram.

Atrioventricular Valve Dysplasia

The most common dysplasia of the atrioventricular (AV) valves seen in veterinary medicine is tricuspid valve dysplasia (TVD) which is a breed concern in the Labrador retriever. Also seen in Old English sheepdogs, Great Danes and boxers, TVD may take many shapes and leave the valvular apparatus incompetent and/or stenotic [18, 19]. Ebstein's anomaly is a particular variation of TVD seen in humans that may be analogous to forms of TVD in dogs, specifically Labrador retrievers [20]. Ebstein's anomaly is characterized by the location of the tricuspid valve leaflets below the valve annulus, and tethering of these leaflets to the RV walls causing them to have poor mobility.

Likewise mitral valve dysplasia (MVD) presents with a wide variation of deformities to the valve apparatus. Mitral valve dysplasia has been reported to occur in cats, Great Danes, German shepherds and bull terriers [1].

Clinical Signs

Clinical manifestations of MVD or TVD range from none to rapid onset of CHF. Mitral valve dysplasia can lead to pulmonary edema and TVD to ascites associated with right-heart CHF. Cases with moderately functional valves may only exhibit signs of exercise intolerance or lethargy. A murmur may not always be present on physical examination, but when noted, MVD is most often associated with a left apical systolic murmur of mitral regurgitation, and TVD with a right-sided systolic murmur. Murmur intensity can be variable, ranging from grade 2 to 5/6 and generally does not correlate well with the severity of the dysplasia. Examination of the neck in patients with TVD may reveal jugular pulses associated with tricuspid regurgitation. A positive hepato-jugular reflux test (see Chapter 3) indicates elevated RA pressures.

Diagnostics

Radiographs show enlargement of the atria if there is significant regurgitation in the corresponding AV valve of the heart. Some ventricular enlargement may also be noted for the corresponding valve. If the valves are stenotic, the engorgement of the "upstream" vessels will be seen as enlargement of the cranial or caudal vena cava in TVD, and enlargement of the pulmonary veins in MVD (Figure 10.20).

Electrocardiography in patients with AV dysplasia may show atrial arrhythmias such as atrial fibrillation, atrial flutter, or atrial premature complexes (APC). A left axis shift with LA and/or LV enlargement pattern, such as "P" mitrale, can be noted in MVD cases. In dogs with TVD, "P" pulmonale, or a deviation of the MEA toward the right, is likely. Conduction defects through the ventricle, such as right bundle branch blocks or "splintering" of the QRS complex (Figure 10.21) are common findings on an ECG. If the stage of decompensation is advanced ventricular premature complexes (VPC) are also possible.

Echocardiography is the most useful modality for diagnosing AV dysplasia. The morphology of the valve itself can be viewed for abnormal placement, abnormal movement and abnormal connection of the papillary muscles and chordae tendineae. The atria associated with the dysplastic valve are generally enlarged, and in some cases severely.

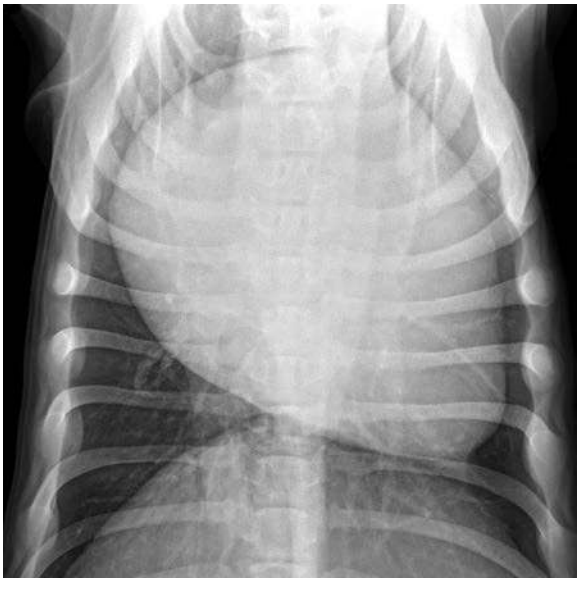

Figure 10.20 Ventrodorsal radiograph of canine tricuspid dysplasia showing marked enlargement of the cardiac silhouette. In this example it is the right ventricle and atrium that are enlarged. The dysplastic valve allows for severe tricuspid regurgitation.

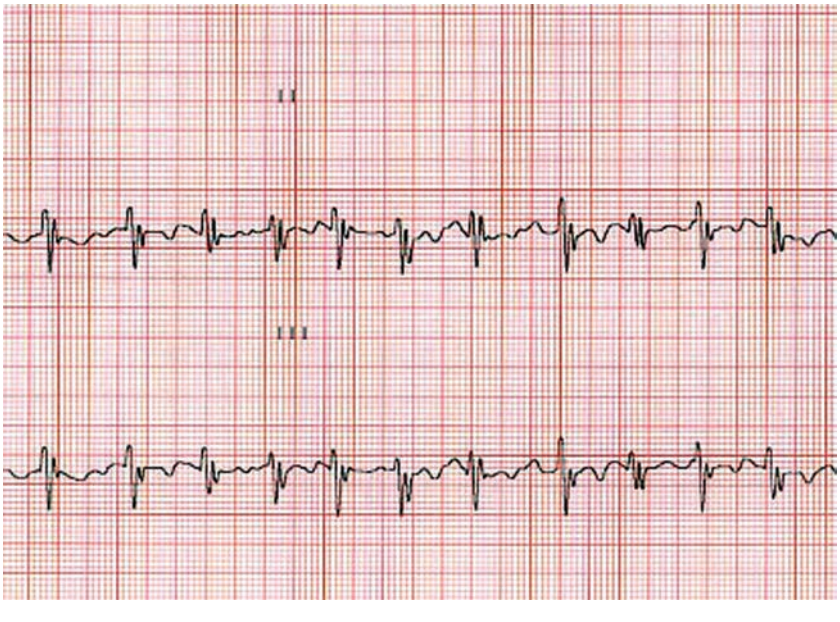

Figure 10.21 Electrocardiogram of QRS splintering recorded at 25 mm/s and standard calibration and shows leads II and III. The baseline is uneven, but clear P waves can be identified before each QRS complex. The QRS has an extra positive deflection after the S wave, but before the T wave. This "splintering" of the QRS complex is a characteristic commonly seen with tricuspid dysplasia.

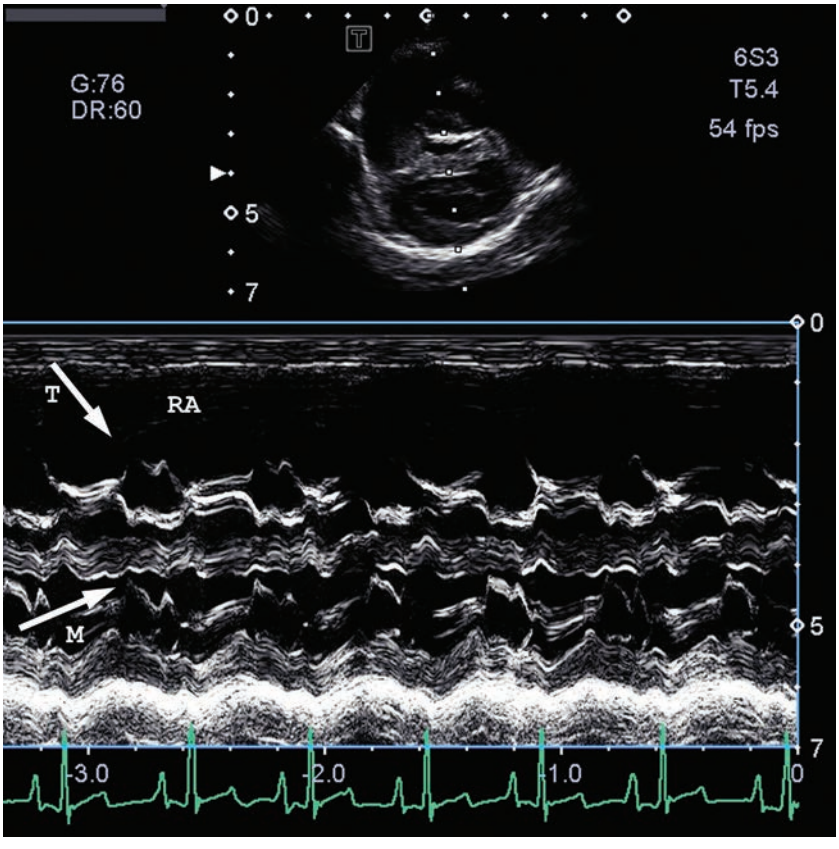

Figure 10.22 Short axis M-mode echocardiogram image of tricuspid dysplasia showing the characteristic "double fish-mouth" of right atrial enlargement (RA). This is recorded in the echocardiographic plane used to record the mitral valve motion. The upper arrow (T) points to the tricuspid valve and the lower arrow (M) points to the mitral valve. The interventricular septum is between the two valves.

Commonly, the chordae tendineae may be absent with the valve leaflet directly attached to the papillary muscle. Ventricular function and size can be abnormal, usually dilated, with poor contractility. M-mode of the valve motion may show poor movement of the leaflets in diastole, especially with MVD.

Dogs with TVD may have such severe RA dilation that a short-axis image of the tricuspid valve can be appreciated. This so called "double fish-mouth" is only seen with RA dilation (Figure 10.22). The IVS may develop an abnormal motion also. If RV pressure is very elevated the IVS will be flattened; if the RV is volume overloaded then paradoxical IVS motion may be seen in which the IVS moves away from the LV posterior wall in systole (Figure 10.23). Three-dimensional echocardiography can provide view of the valve, which allows for planemetry measurements, as well as permits visualization previously uncommon in veterinary medicine (Figure 10.24).

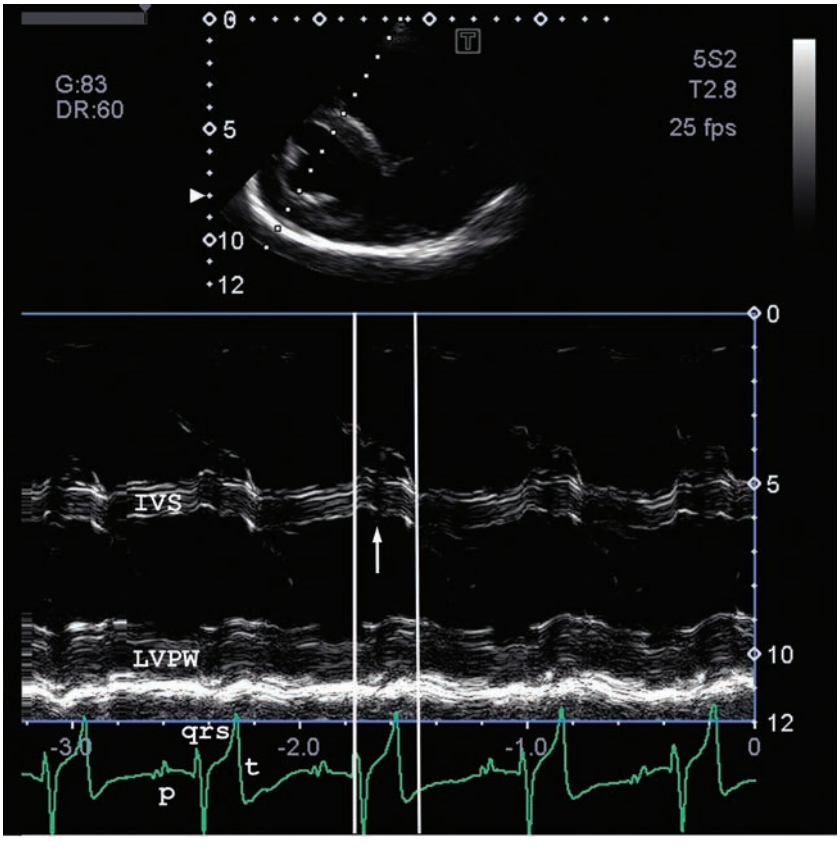

Figure 10.23 M-mode echocardiographic image of paradoxical septal motion: in the presence of right ventricular volume overload, the interventricular septum (IVS) will move away from the center of the left ventricular lumen rather than towards it as usual during systole. The parts of the electrocardiogram are labeled P (p), QRS (qrs), and T (t) along with the left ventricular posterior wall (LVPW). Note the negative QRS complex in lead II typical of a mean electrical axis shift towards the right. The vertical lines indicate the duration of systole in the third cardiac contraction shown. The IVS can be seen moving upwards (arrow) away from the left ventricle between the lines (see Chapter 6 for normal reference).

Using Doppler modalities, PGs across the valves can be estimated and regurgitation diagnosed with color flow mapping (Figures 10.25 and 10.26). An increase in velocity of blood from the atria to the ventricles is indicative of a stenotic valve.

Unfortunately, short of open heart surgery, there is little that veterinary cardiology has to offer. Valve replacement surgery is costly, and has rarely been performed in AV valve dysplasia. As the patient ages the long-term hemodynamic consequences are virtually identical to acquired valve disease. Medical management of the hemodynamic effects with standard heart failure therapy is the most common treatment. In the case of a stenotic tricuspid valve, cardiac catheter balloon valvuloplasty is possible, but more often TVD lesions are only regurgitant [18].

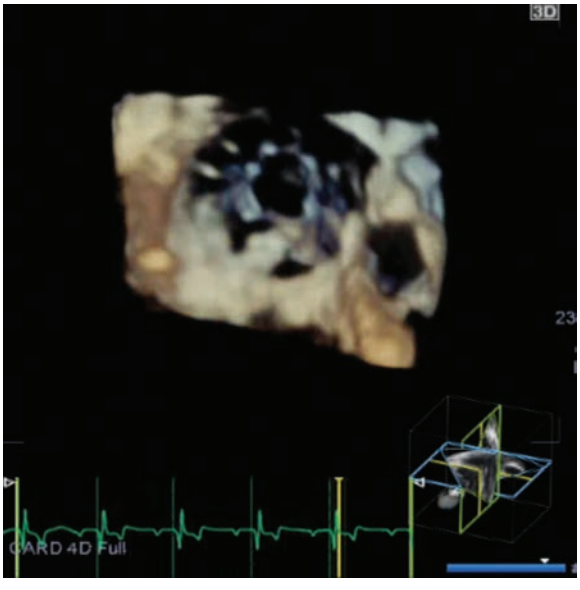

Figure 10.24 Three-dimensional echocardiographic image of a dysplastic tricuspid valve (larger dark circular shape) from the right ventricular side of the valve looking towards the right atrium. The area of the valve can be measured. There is a large hole (smaller dark circle within the larger one) in the center of the valve where the valve would be incompetent. The area of the defect could also be measured from this view.

Intercirculatory Shunts

Intercirculatory shunts are abnormal hemodynamic communications between the pulmonic and systemic circulations. They can occur at any level of the circulation; in the heart itself, in the central vasculature, or in the peripheral vessels. Shunts between the

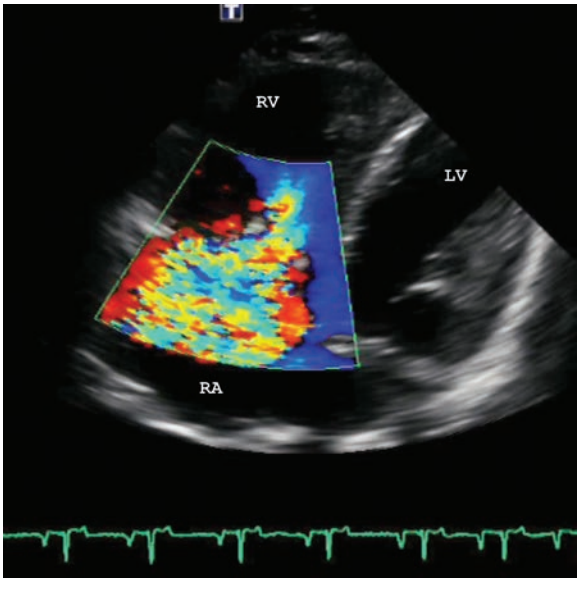

Figure 10.25 Color-flow mapping Doppler image of tricuspid regurgitation: this is a left apical four-chamber view of a canine heart with tricuspid dysplasia. The right ventricle (RV) is towards the top and the right atrium (RA) is at the bottom. The left ventricle (LV) is to the right of the image. The presence of tricuspid regurgitation is demonstrated by the large turbulent color-flow signal in this image. On the RV side of the valve, the blue color signal represents blood flow away from the transducer, or down in this example. As the blood reaches the tricuspid regurgitant orifice, it accelerates to become the high velocity turbulent color image shown. The red color on the edges of the yellow–green mosaic is aliasing of the color signal.

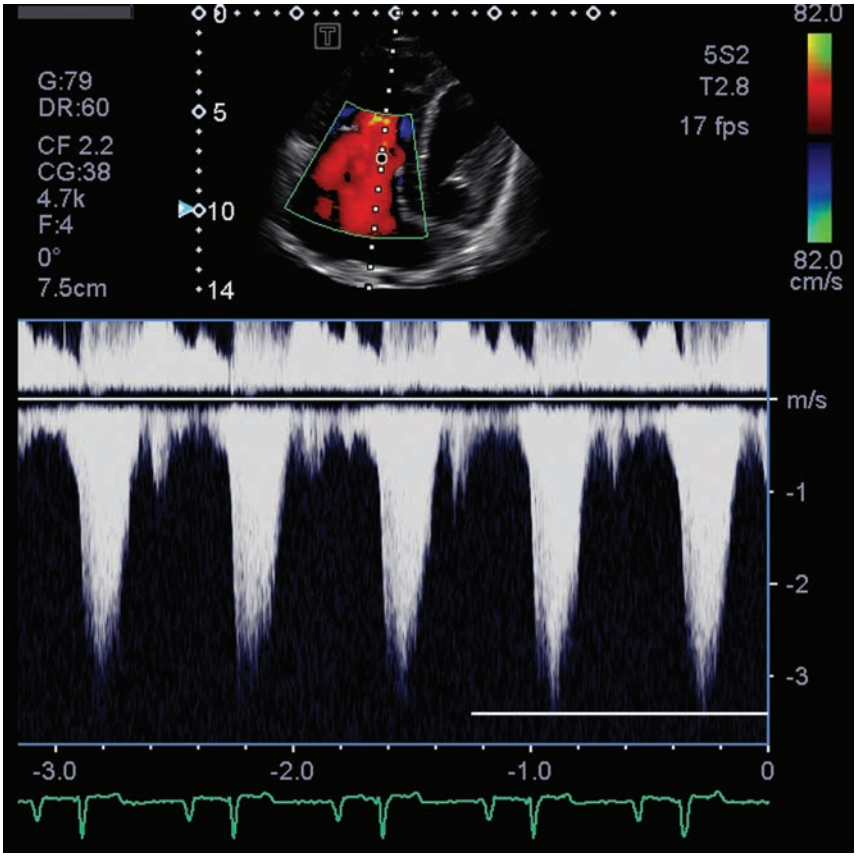

Figure 10.26 Spectral continuous-wave Doppler of tricuspid regurgitation: the reference image near the top of the image shows the Doppler cursor aligned with regurgitant blood flow across the tricuspid valve. The velocity of greater than 3 m/s is consistent with slightly elevated right ventricular pressure. This could be due to pulmonary hypertension of an obstruction of the right ventricular outflow tract such as pulmonic stenosis. Note also the negative QRS complex in the lead II electrocardiogram across the bottom consistent with a mean electrical axis shift towards the right.

cardiac chambers are termed *septal defects* and can be atrial or ventricular. Central vasculature anomalies may be persistent flow through the fetal ductus arteriosus or aortopulmonary windows [1]. Aortopulmonary windows are a failure of the spiral septum to completely develop with the *truncus arteriosus* of the embryo. They are extremely rare and create an ostium or "window" between the aorta and the main pulmonary artery. Shunts that occur out in the peripheral vasculature are termed an *arteriovenous fistula.* Shunts can flow from the arterial circulation to pulmonary (so called *left to right* shunts) or pulmonary to arterial (*right to left*). Due to the greater blood pressure in the arterial system, most shunts flow from left to right. Shunts provide an abnormal volume load to one or more parts of the circulation. The size of the defect and the load it represents determines the severity of cardiac effects.

Atrial Septal Defects

Atrial septal defects are very uncommon and often have little or no clinical significance unless the patient is an athlete, which could explain a lack of peak performance in an otherwise perfect specimen. They are often incidental findings on echocardiogram that was performed for some other reason. Unless increased RV and RA pressures are present, ASDs will shunt from the LA to the RA during diastole. ASDs rarely present with murmurs; however, when a murmur is heard, they are often soft, left basilar systolic murmurs due to relative pulmonic stenosis from increased flow through the right ventricular outflow tract [1].

Diagnostics

Radiographs may show some enlargement of the RA and some pulmonary overcirculation from the increased volume load. Generally radiographs are nondiagnostic.

Echocardiography may reveal a "drop out" lesion on the interatrial septum, but this area is fairly nonechogenic anyway and overinterpretation must be avoided. Color-flow Doppler can be used to demonstrate flow across the defect. This will be noted as a low-velocity flow generally coded in red and is best appreciated during longer diastolic periods (Figure 10.27).

Patent foramen ovale is the result of nonclosure of a fetal physiological shunt that passes blood from the placental circulation entering the RA to the LA then LV. This is not a true ASD because the atrial septum is anatomically correct. Typically after birth, a flap of tissue that was held open by the flow from the maternal circulation through the foramen ovale closes when the right atrial pressure drops (see Chapter 1). If RA pressures never drop below LA then the ovale may remain patent. When PFO is present,

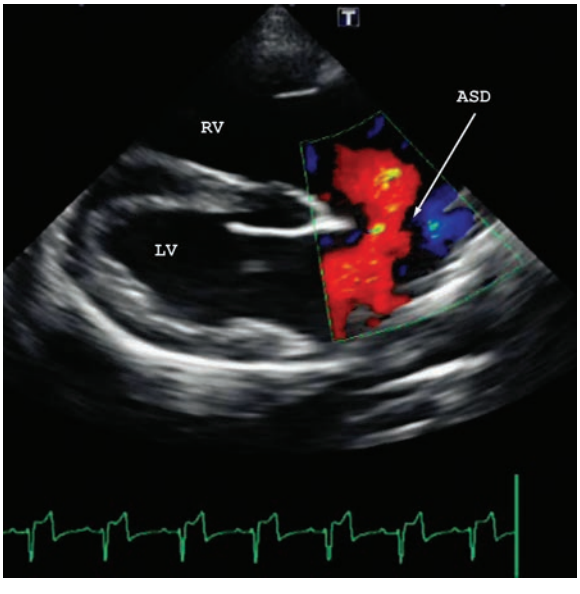

Figure 10.27 Long axis four-chamber view of an atrial septal defect (ASD): the color-flow mapping in this image shows blood moving from the left atrium to the right atrium (coded red). The left ventricle (LV) and right ventricle (RV) are labeled for reference. The arrow indicates the opening between the two atria filled with the Doppler color signal. This portion of the interatrial septum often will be seen as hypoechoic, and a false diagnosis of ASD is possible. The color-flow mapping assists in determining a true ASD from a normal interatrial septum with echo "drop-out".

PS or pulmonary hypertension and TR are almost always present as well. As previously stated, a contrast echocardiogram is very helpful in diagnosing a PFO.

Treatment

Typically, no treatment is needed for the ASD since they are small and have minimal hemodynamic consequences. In dogs with large ASDs, transcatheter placement of closure devices can be attempted [21].

Ventricular Septal Defect

Shunting resulting from a hole in the IVS is known as a VSD. These defects most often occur in membranous or perimembranous regions of the heart just below the aortic valve, tricuspid valve or pulmonic valve of the RV and near the aortic valve as viewed from the LV. In the embryological development of the heart this area is the last to be closed by union of the IVS and the spiral septum of the truncus arteriosus. The proximity of the VSD to the aortic valve may lead to prolapse of an aortic valve leaflet. Ventricular septal defects can occur in the muscular portion of the IVS in horses and cattle, but rarely occur in small animals. The size of a VSD can be quite variable ranging from 1 to 2 millimeters to several centimeters, and can affect the cardiac architecture as seen with tetralogy of Fallot.

Clinical Presentation

Patients will present with a loud right-sided basilar systolic murmur (grade 5–6/6) as juveniles. They may exhibit exercise intolerance or syncope during exercise, but are otherwise normal. If the defect is very small or *restrictive*, the patient may never develop cardiac signs. If a diastolic murmur is present, then aortic prolapse and regurgitation should be suspected.

Diagnostics

Radiographs generally show enlargement of the LA and LV with pulmonary overcirculation with enlargement of the complete pulmonary vasculature. If the shunt volume is large and cardiac output compromised, evidence of CHF may also be present.

Electrocardiography is typically within normal limits with the exception of potential LV and LA enlargement patterns. In late-stage disease, atrial or ventricular arrhythmias may be present.

Echocardiography demonstrates an enlarged LV and LA as rounded, volume-loaded chambers (Figure 10.28). Systolic function may be impaired. The defect itself may be hard to visualize unless it is large, but color-flow Doppler will reveal a high-velocity jet moving from the LV to the RV during systole near the aortic valve in the long axis five-chamber view and the short axis heart base view [6, pp. 437–475] (Figure 10.29).

Spectral Doppler interrogation of this jet indicates the PG between the LV and RV. This yields information on prognosis as an assessment of chamber pressures. Assuming the pressure in the ventricles is within normal limits, the PG should be approximately 100 mmHg at peak systole. Using the modified Bernoulli equation, the velocity measured should be near 5 m/s (Equation 10.2) (Figure 10.30).

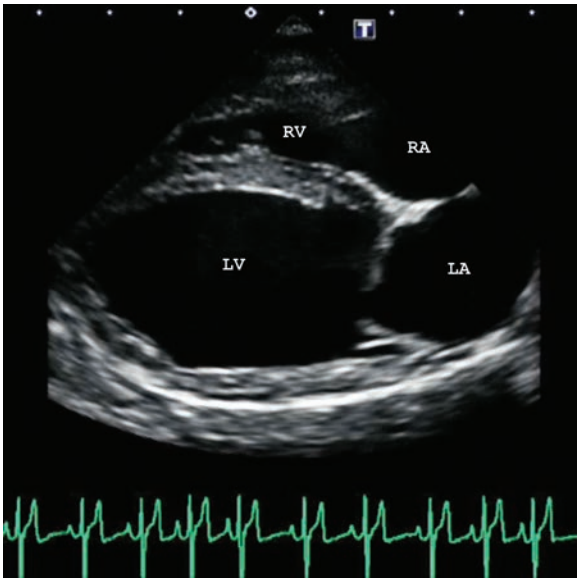

Figure 10.28 Long axis four-chamber view of a canine heart with volume overload. The chambers are labeled as: LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium. The LV shows an enlarged rounded shape and the LA is also enlarged. This is a typical-looking LV in dogs with ventricular septal defects due to the overcirculation of blood through the pulmonary vasculature. As blood shunts from the LV to the RV the additional volume from the LV is carried to the lung and return directly to the LV causing the pictured changes in the heart.

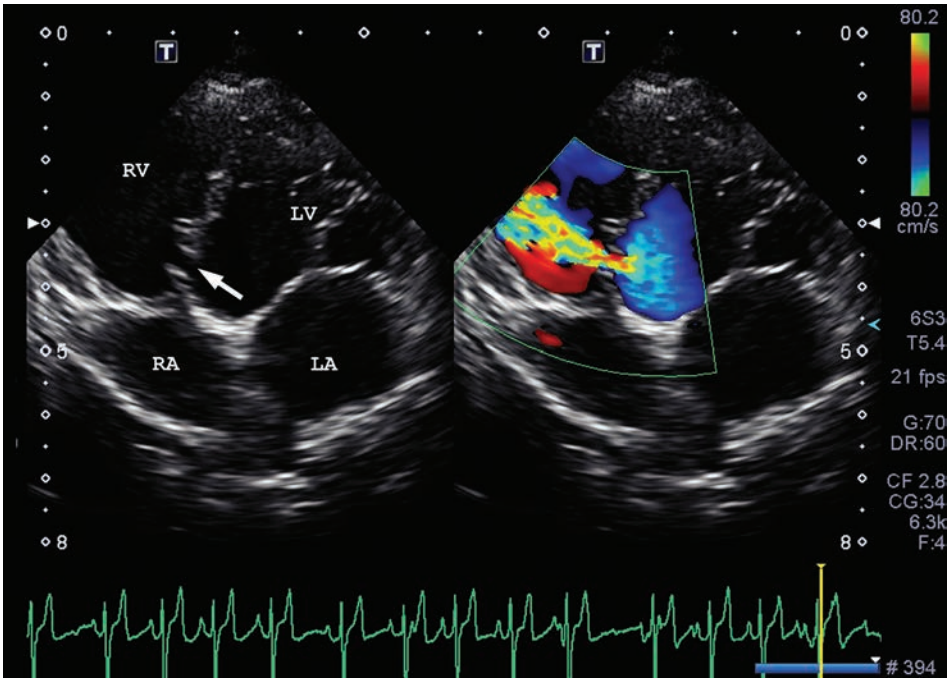

Figure 10.29 Twin image of a ventricular septal defect (VSD) with and without color-flow mapping: this image is from the left apical four-chamber projection. The left image shows the VSD (arrow) as a hypoechoic space in the interventricular septum. The chambers are labeled as: LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium. The right portion shows the exact same image with the Doppler color-flow mapping added, demonstrating the trans-septal flow. The large blue signal is blood being ejected from the LV as it heads out the aorta. A yellow–green mosaic high-velocity turbulent blood flow jet can be seen crossing through the VSD into the RV.

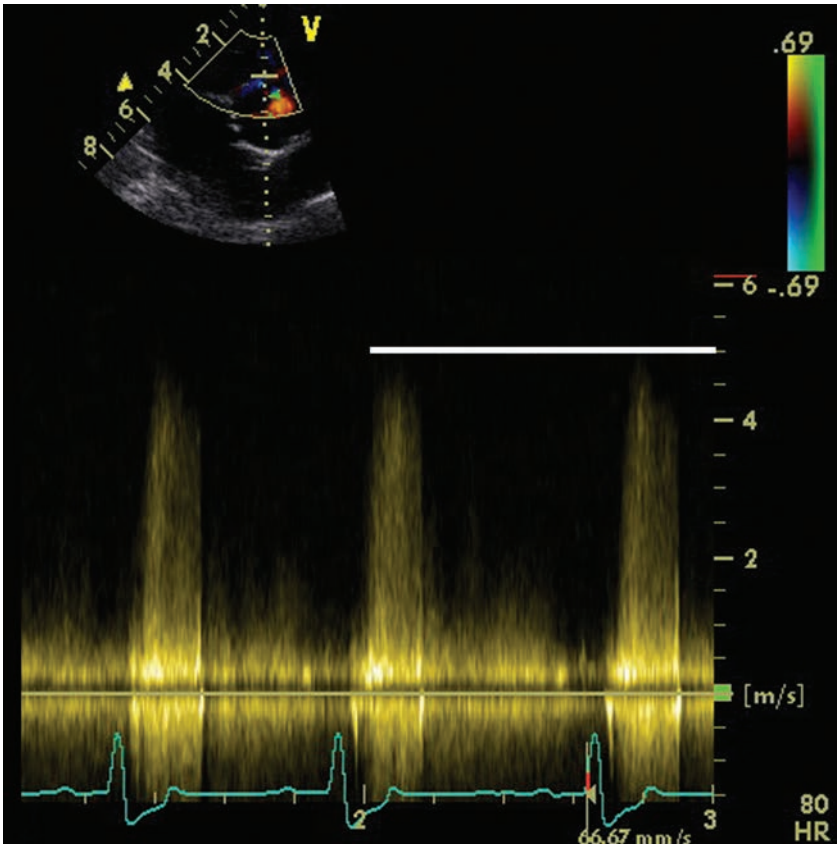

Figure 10.30 Spectral Doppler velocity image of blood flow through a ventricular septal defect: the blood flow from the left ventricle to the right ventricle recorded in this spectral Doppler image is moving towards the transducer, thus is displayed upwards from the baseline. It also is seen as the red signal within the reference image at the top. The velocity scale indicates the velocity to be approximately 5 m/s (line). Using the modified Bernoulli equation, the calculated pressure gradient would be approximately 100 mmHg between the ventricles. This implies normal ventricular chamber pressures associated with a heart in compensation for the condition.

RV peak systolic pressure = ~25 mmHg. LV peak systolic pressure = ~125 mmHg. Thus LV pressure – PV pressure = ~100 mmHg.

$$
\begin{aligned}
&4 * V^2 = \Delta P \\
&4 * V^2(\mathrm{m/s}) = 100\,\mathrm{mmHg}.\ \text{Solve for } V^2 \\
&100\,\mathrm{mmHg}/4 = 25 \\
&\sqrt{25} = 5\,\mathrm{m/s}
\end{aligned}
\tag{10.2}
$$

If cardiac catheterization is performed, an injection of contrast into the LV will be seen passing through the IVS into the RV outflow tract (Figure 10.31). With the advent of echocardiography, cardiac catheterization is uncommon.

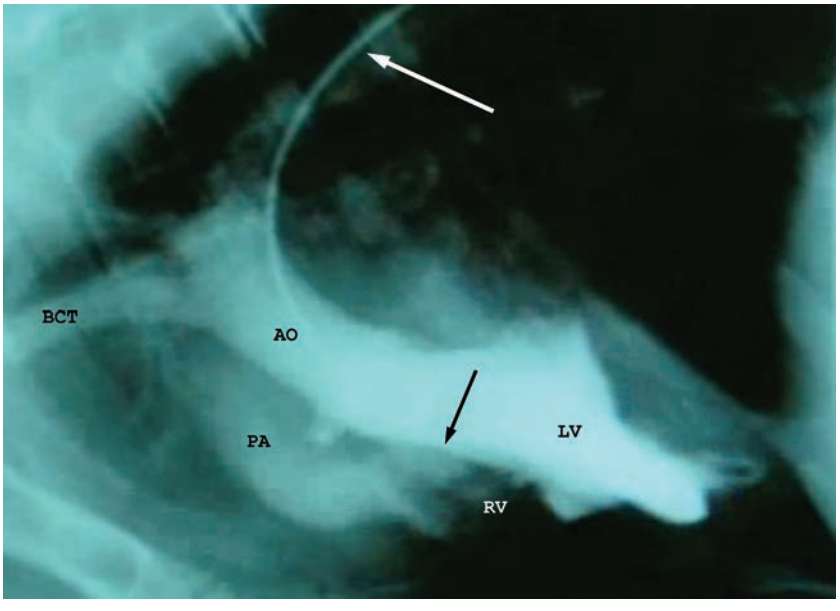

Figure 10.31 Fluoroscopic ventriculogram of a ventricular septal defect: the heart is seen here in a right lateral projection with the patient's head to the left. The major anatomical features are labeled: AO, aorta; LV, left ventricle; RV, right ventricle; PA, pulmonary artery; BCT, brachiocephalic trunk. A pigtail catheter (white arrow) has been advanced from the femoral artery retrograde through the descending aorta, around the aortic arch, through the aortic valve, then into the apex of the left ventricle. The contrast was injected into the LV, and is seen passing through a ventricular septal defect (VSD) (black arrow). The contrast is simultaneously highlighting the LV and RV outflow tracts, PA and proximal AO. Although the contrast jet of trans-VSD flow is difficult to appreciate, the reader should understand that for the highlighted structure to be illuminated from a LV injection, a VSD must be present. This image also demonstrates the VSD flow heading directly out the PA, and not filling the RV with contrast. This is typical for the VSD since the shunt occurs during systole, and the defect is located in proximity to pulmonic valve.

Treatment

The pathophysiology of the VSD generally leads to volume overload of the LA and LV. The shunt occurs during systole which protects the RV from the additional volume. The shunt volume travels from the LV directly out of the pulmonary artery in most cases, circulating through the lungs and back to the LA then LV. Added to the shunt volume is the returning volume from the body to the RA. If the shunt volume adds 2.5 times the normal volume to the LV, then LV failure is likely [1]. Most patients with large VSDs eventually succumb to left-heart CHF as the volume of the LV increases by the shunt volume returning to the left heart through the lungs. Consequently, the systolic function can be depressed and the heart goes into low output failure. As LV end-diastolic pressure rises and systolic function decreases, the PG between the LV and the RV can drop, as well as the LV to RV VSD velocity, providing a clue to the decompensation of the LV. During this time, the loudness of the systolic murmur may decrease as compared with when the diagnosis was first made. Surgical closure of VSD has been reported [22]. but because of cost, surgery is not generally practiced. Transcatheter occlusion has been performed

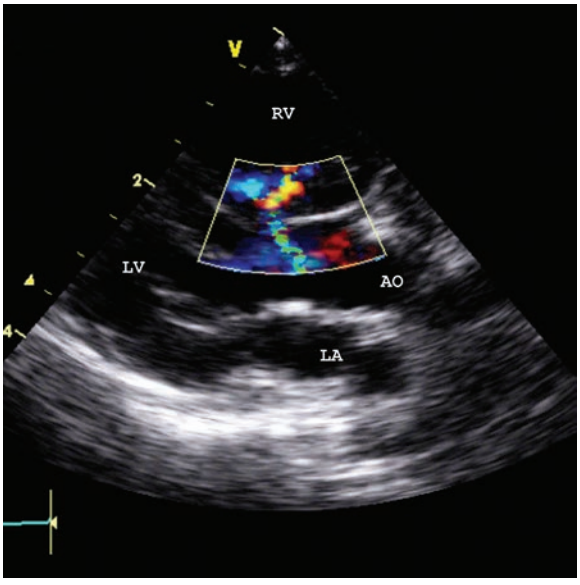

Figure 10.32 Long axis four-chamber image of a right-to-left shunting ventricular septal defect. The chambers are labeled as: LV, left ventricle; RV, right ventricle; AO, aorta; LA, left atrium. The color-flow signal in the center of the images demonstrates blood flowing from the RV to the LV, as indicated by the predominately blue color of flow away from the transducer. The yellow–green mosaic superimposed on the blue indicates turbulence as the blood crosses the defect.

with various occlusion devices [23–25]. Transcatheter occlusion for muscular VSD is more promising than for membranous and perimembranous VSD. Deployment of an occlusion device in the membranous and perimembranous regions comes with the risk of entrapping a portion of the aortic valve and inducing significant aortic insufficiency. Management of left-sided CHF is carried out with standard therapy with diuretics, ACE inhibitors, and positive inotropes.

In rare cases, the overcirculation of the pulmonary vasculature makes the vessels hyperreactive and they become stiff, leading to increased pulmonary vascular pressures. If they should rise to the point of exceeding systemic pressure, then the shunt will reverse from left to right to right to left (Figure 10.32 and 10.33). This condition, known as Eisenmenger's syndrome, leads to cyanosis and hypoxemia and is often fatal. It can also be confirmed by a contrast echocardiogram. Right-to-left shunting VSDs also show dilation of the large pulmonary arteries but undercirculation to the lung periphery, and the size of the heart generally remains normal on a radiograph [1]. Some newer drugs can be used to lower pulmonary hypertension [26] increasing blood flow to the lungs, and to combat secondary polycythemia, but a heart–lung transplant is the only curative treatment.

Patent Ductus Arteriosus

Patent ductus arteriosus is considered the most common cardiac defect in dogs, especially poodles, Shetland sheepdogs, collies and Maltese (see Table 10.1). Fortunately, it is rare in cats and may normally persist for a week in foals prior to closing. This shunt is formed by a failed closure of the ductus arteriosus (DA) of the fetal circulation which allows blood to bypass the lungs *in utero*. The DA is formed from the left sixth aortic

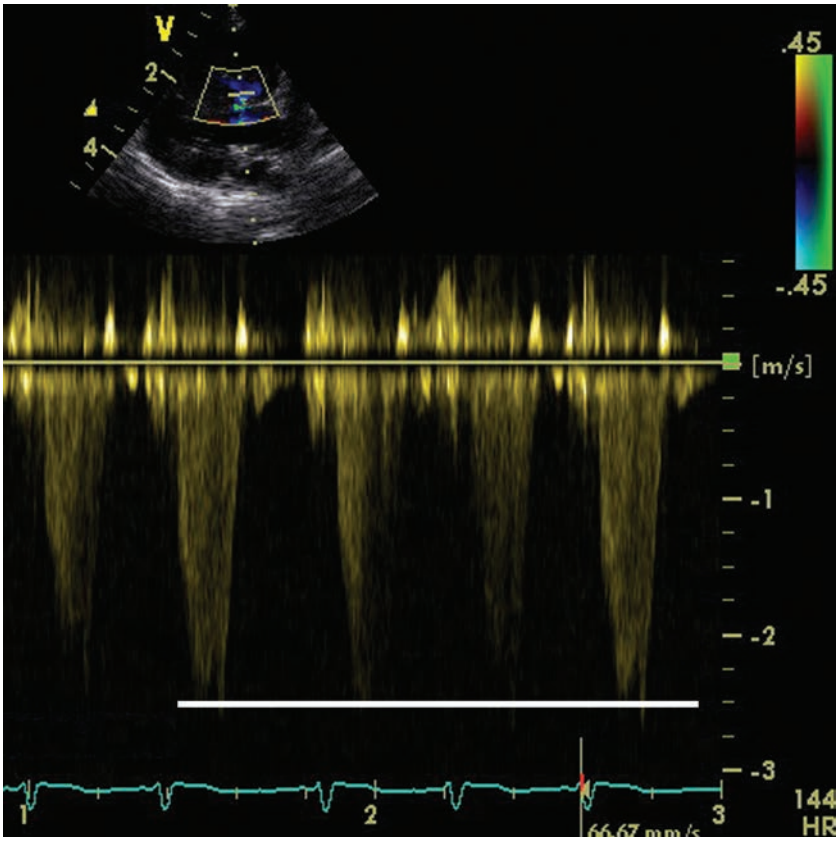

Figure 10.33 Spectral Doppler image of a right-to-left shunting ventricular septal defect: the Doppler cursor is aligned with the right-to-left shunt in the upper reference image. The velocity is recorded below the baseline as blood moves away from the transducer. The velocity seen here is approximately 2.5 m/s. Initially this velocity may seem insignificant, but when applied to the clinical context a grim picture emerges. Using the modified Bernoulli equation, this velocity represents a 25 mmHg pressure gradient (see chapter 6) between the RV and LV. Because the shunt is right to left, the RV pressure must be greater than LV pressure. If LV pressure is even approximating normal peak systole pressure of 120 mmHg, this implies the RV pressure is nearly 145 mmHg, or roughly six times normal. Unless the patient has concurrent pulmonic stenosis, this elevated pressure is due to the pulmonary hypertension of Eisenmenger's physiology.

arch in the embryo and allows blood from the RV to cross directly into the descending aorta. The DA normally closes once the pulmonary vascular resistance drops postparturition. Once the pulmonary vascular pressure dips below systemic pressure, if the DA does not close and the DA remains patent, blood flows from the descending aorta to the main pulmonary artery distal to the pulmonic valve. This arrangement keeps the right heart protected from the extra volume. Similar to the VSD, the extra blood is shunted to the main pulmonary artery then travels to the left heart through the lungs creating a volume overload of the LA and LV. Two-thirds of dogs with a PDA will develop CHF by one year of age [1].

Clinical Presentation

Puppies with a PDA are often diagnosed during the initial veterinary examination by the characteristically loud (grade 4–6/6) continuous murmur in the axillary region of the left cardiac base. The murmur is very characteristic; sometimes described as a "machinery" murmur because the sound resembles the sound of a washing machine agitating clothes. If auscultation is performed at the left apex, typically only a grade 4–6 systolic murmur is appreciated. By moving to the craniodorsal axillary region the diastolic portion of the continuous murmur becomes apparent. They may appear as "poor doers" or runts compared with littermates or are normal with only exercise intolerance noted.

During diastole the blood shunting from the aorta to the pulmonary artery lowers the diastolic arterial blood pressure. This results in a greater than normal pulse pressure (see Chapter 3) and palpation of the femoral arteries is described as *hyperkinetic,* or bounding pulses.

Diagnostics

Radiographs show LV enlargement patterns in both lateral and VD views and a multiple bulge effect at the 12–3 o'clock position on the VD view indicating dilation of the aorta, pulmonary artery, and the left auricular appendage (Figure 10.34). Pulmonary overcirculation is present. If the patient is diagnosed later in life, then signs of CHF may also be present.

Electrocardiography shows a normal MEA with dramatic enlargement of the QRS complex in lead II indicating LV enlargement (Figure 10.35). Advanced cases may have atrial fibrillation, APCs or VPCs.

Echocardiography confirms the LV and LA enlargement and allows for assessment of systolic function. Patients with PDA may have marked to severe LV and LA dilation. Systolic function may be normal, but depending on the age at diagnosis, can

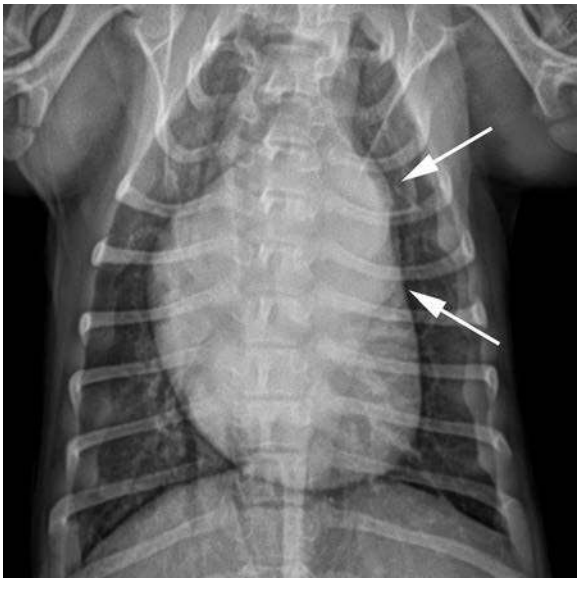

Figure 10.34 Ventrodorsal radiograph of canine patent ductus arteriosus: the upper arrow indicates the location of a bulge in the aorta. The lower arrow marks the location of a second bulge made by the pulmonary artery. The luminary vasculature is also prominent due to the additional blood flow created by the shunt.

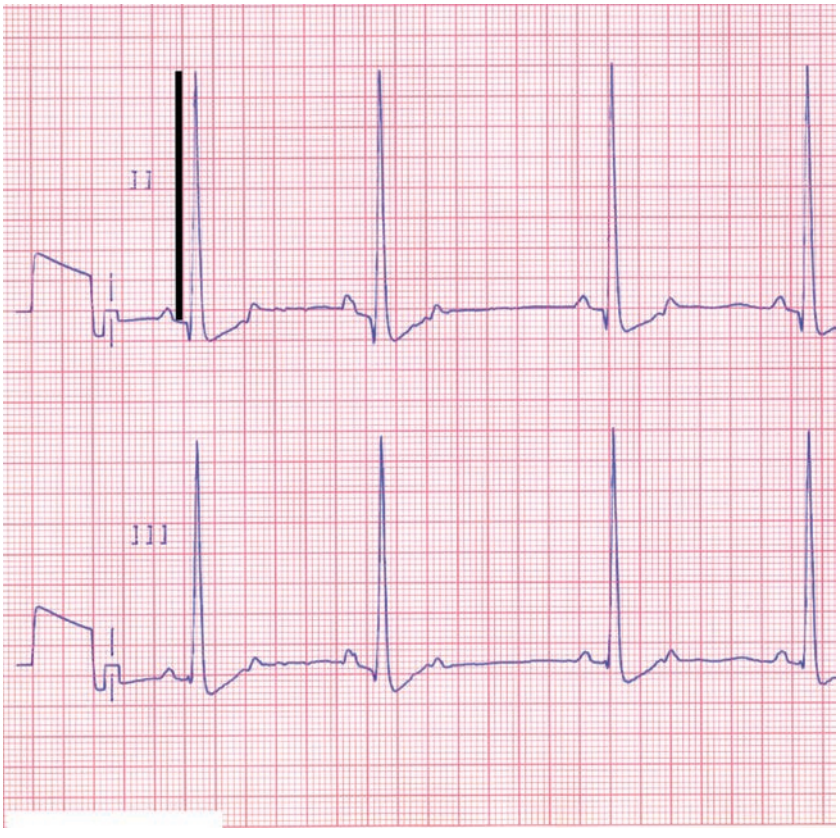

Figure 10.35 Electrocardiogram showing a left ventricular enlargement pattern: the calibration in this electrocardiogram (ECG) is 1 cm/mV, and the paper speed is 50 mm/s. The black line marks the height of the R wave of this patient. The amplitude of the R wave measures 4.1 mV. This is 1.1 mV greater than normal and is typical of dogs with patent ductus arteriosus.

be compromised. Earlier diagnosis generally provides for persevered contractility. The RV and RS are usually within normal limits. Color-flow mapping of the pulmonary artery from the right short axis heart base view shows continuous turbulent blood flow [6, pp. 437–475]. During the Doppler examination of PDA, a continuous turbulent color-flow pattern will be seen circulating in the main pulmonary artery. This color-flow pattern is virtually pathognomonic for PDA. The inflow jet of blood coursing from the aorta can often be appreciated around the 5 o'clock region of the image (Figure 10.36). Because a PDA is not in the heart itself, the actual DA can be difficult to visualize. An alternative echocardiographic view is from the left heart base view of the pulmonary artery. The PDA appears as a small tubular structure entering the distal main pulmonary artery (Figure 10.37). Spectral Doppler of the transductal flow is expected to reveal peak systolic velocities of 4–5 m/s providing pulmonary artery and aortic pressures are normal (Figure 10.38). Velocities less than 4 m/s imply poor cardiac output and a failing LV.

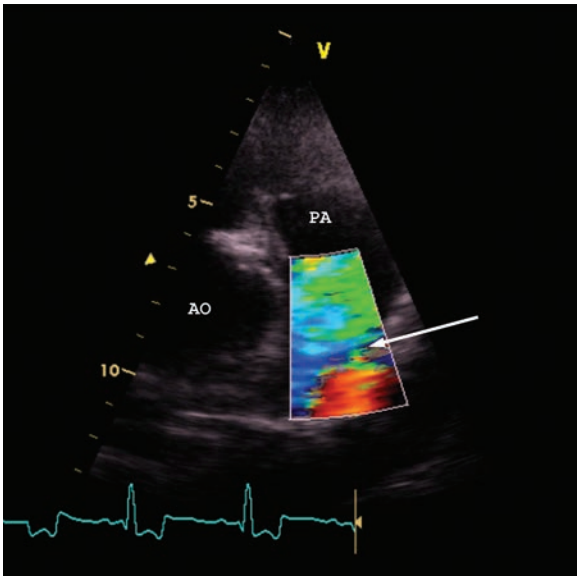

Figure 10.36 Color-flow Doppler of a patent ductus arteriosus (PDA) in the short axis heart base view: the aortic root (AO) and the main pulmonary artery (PA) are labeled. The arrow points to the narrow jet of yellow turbulent blood flow through the aperture of the PDA. The red signal below is blood flowing towards the transducer from the aorta (a section out of the view) through the PDA towards the aperture into the PA. The blue signal is pulmonic outflow moving away from the transducer. Above the arrow is the yellow–green mosaic of turbulent blood flow where the ductal flow mixes with the right ventricular outflow.

Treatment

Treatment of PDA is best accomplished by closure of the shunt. This has traditionally been accomplished with surgical ligation [27, 28] for which results have been excellent [29]. Surgical ligation is an open thoracic procedure but not open heart. Entering through a left hemithoracotomy, the surgeon isolates and then ligates the PDA, in some cases bisecting the PDA after placement of two ligatures. Bisecting the PDA eliminates

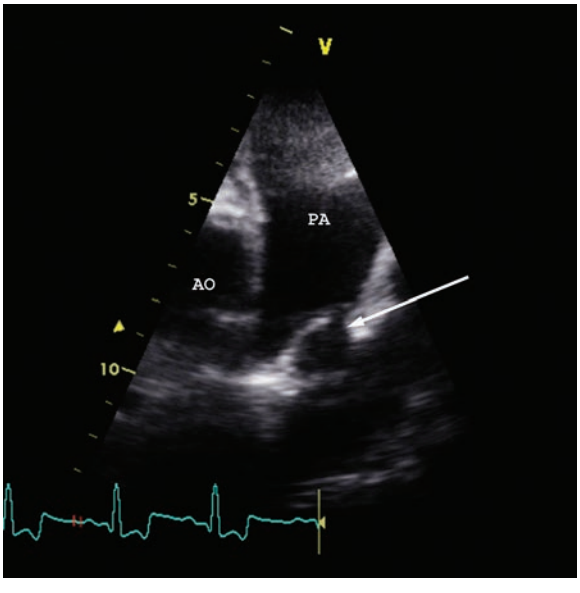

Figure 10.37 Echocardiographic short axis heart base view of a patent ductus arteriosus: this is similar to Figure 10.35 without the color Doppler added. The aorta (AO) is central, with the main pulmonary artery (PA) on the right. In the lower right quadrant of the image is the ampulla of the ductus arteriosus (arrow). The aperture of the ampulla into the PA is just at the tip of the arrow.

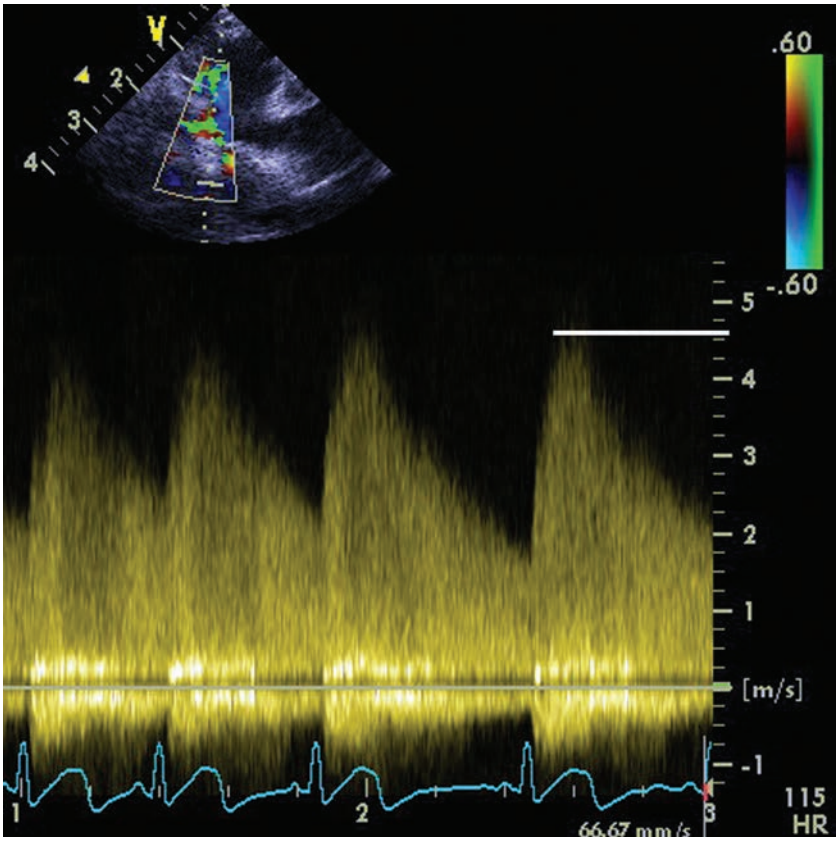

Figure 10.38 Spectral Doppler recording of transductal flow: the Doppler cursor is aligned with blood flow through the patent ductus arteriosus. The velocity trace in the lower portion demonstrates a peak velocity of approximately 4.5 m/s during systole, and velocity at end diastole of near 2.0 m/s. The exact end diastolic velocity is determined by the length of diastole. Longer diastolic periods allow for more shunting of flow out of the aorta continually lowering the pressure. The peak systolic velocity of 4.5 m/s represents a pressure gradient of 81 mmHg between the aorta and the pulmonary artery. This difference is only slightly abnormal and generally implies a heart compensating for the abnormal blood flow.

the possible complication of recanalization of the PDA, which can occur [30]. Generally, surgery should be as soon as possible once the diagnosis has been made. Delaying surgery can potentially allow time for permanent remodeling of the myocardium leading to persistent systolic dysfunction. The risk of tearing the aorta, pulmonary artery, or the PDA is always present, but more so in older dogs. Surgery should ideally be performed in dogs under the age of 2 years [1].

Transcatheter occlusion is an alternative to surgery; both treatments are curative. Transcatheter occlusion is a method of deploying a device in the PDA from inside the vasculature and occurs in the cardiac catheterization laboratory (see Chapter 16). A variety of devices have been developed including [31, 32]: embolization coils, vascular plugs, and most recently the Amplatz Canine Ductal Occluder® (Infiniti Medical Supply,

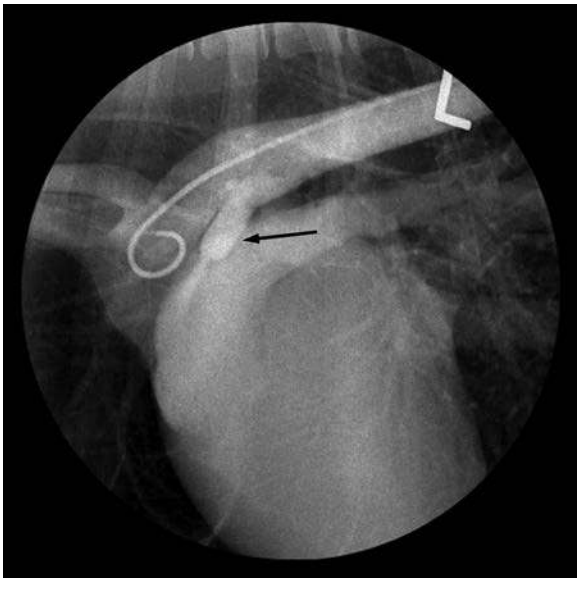

Figure 10.39 Fluoroscopic angiogram of patent ductus arteriosus (PDA) in a dog: the catheter was advanced from the femoral artery retrograde through the descending aorta to the cranial portion of the aortic arch in this right lateral view. The radiopaque contrast is injected in the proximal aorta to highlight the structure of the aortic arch. The PDA is indicated by the arrow. A small jet of contrast can be seen streaming into the main pulmonary artery through the PDA. The brachiocephalic and common carotid arteries can be seen cranial to the pigtail catheter. The arrow is sitting inside the main pulmonary artery.

Menlo Park, CA, USA, http://infinitimedical.com/index.html). These devices are introduced into the PDA through arterial vasculature usually via the femoral artery through the abdominal aorta to the descending aorta and the PDA. Occasionally this procedure is done via the pulmonary artery [32]. Initially, an angiogram of the PDA is performed (Figure 10.39) and the anatomy positively identified. A cardiac catheter is then directed through the vasculature to the PDA where the cardiologist can then place an occlusion device in the lumen of the PDA (Figure 10.40). The occlusion devices are self-expanding

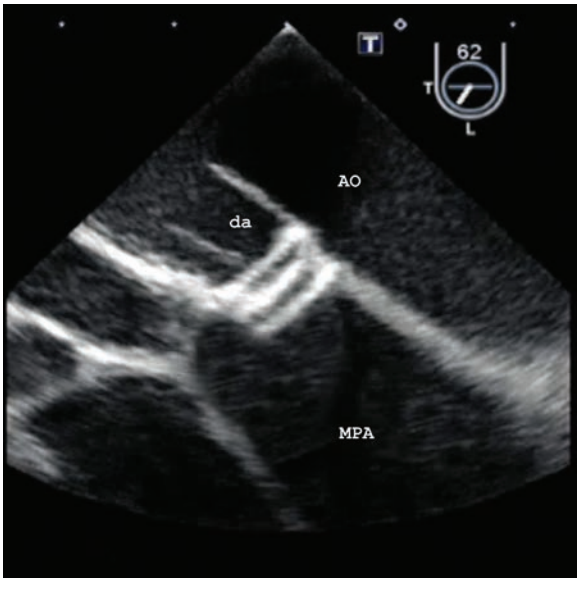

Figure 10.40 Transesophageal echocardiographic image of an Amplatz® Canine Ductal Occluder. The central three-lined hyperechoic structure is an Amplatz® Canine Ductal Occluder being positioned with the patent ductus arteriosus (da). The bright line below the "da" label is the deployment cable. AO, aorta. MPA, main pulmonary artery.

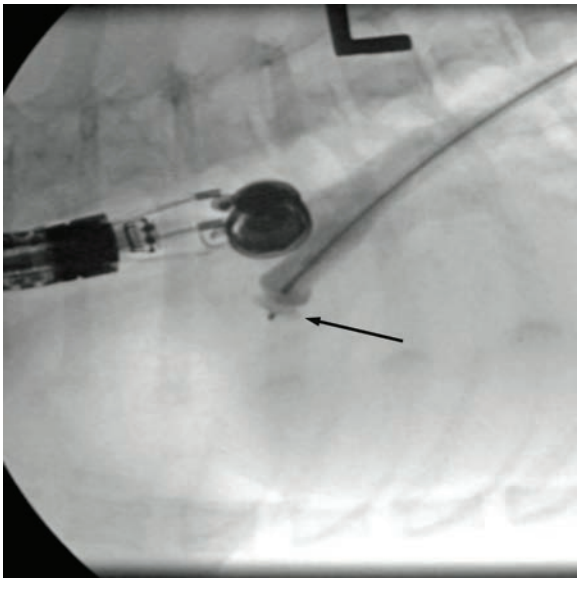

Figure 10.41 Fluoroscopic angiogram of patent ductus arteriosus in a dog after closure: the catheter was advanced from the femoral artery retrograde through the descending aorta to the cranial portion of the descending aorta in this right lateral view. The catheter contains the Amplatz® Canine Ductal Occluder (arrow) and deployment cable. The instrument above the ductus arteriosus is a transesophageal ultrasound probe. An injection of radiopaque contrast is made through the catheter directly into the ductus arteriosus. No contrast can be seen flowing past the Amplatz® Canine Ductal Occluder.

and can be collapsed to pass through the cardiac catheter. Once positioned in the PDA, the occlusion device is released from its deployment catheter. The devices are thrombogenic and usually will take 10–20 minutes to completely occlude blood flow. A second angiogram will verify complete occlusion (Figure 10.41). Surgery and transcatheter occlusion have excellent success rates and long-term outcomes [29, 31] although, rarely, complications such as embolization device dislodgement or infection do occur [33].

Cyanotic Conditions

The complex congenital and cyanotic heart conditions are extremely rare in small animal medicine. It would be beyond the scope of this text to delve too deeply into their pathophysiology. Congenital cardiac conditions that cause a mixing of blood from the pulmonary circulation into the systemic circulation can cause the patient to be cyanotic. Conditions, such as tetralogy of Fallot (TOF), Eisenmenger's syndrome, reversed PDA, right-to-left ASDs, and endocardial cushion defects can all cause deoxygenated blood from the pulmonic circulation to mix with systemic blood. However, these conditions are rare. Some patients do adapt to these conditions and can live normal lifespans; however, they rarely exercise well due to poor oxygenation. The low oxygen saturation of the blood perfusing the kidneys stimulates the release of erythropoietin, which in turn creates a secondary polycythemia.

Tetralogy of Fallot is a combination of four defects typically seen together; a large VSD, dextropositioning of the aorta, PS and RV hypertrophy (Figures 10.42 and 10.43). Tetralogy of Fallot was first described by Niels Stensen in 1672, but was named for Etienne-Louis Arthur Fallot [34] who linked the defect to "blue baby syndrome" in 1888. The

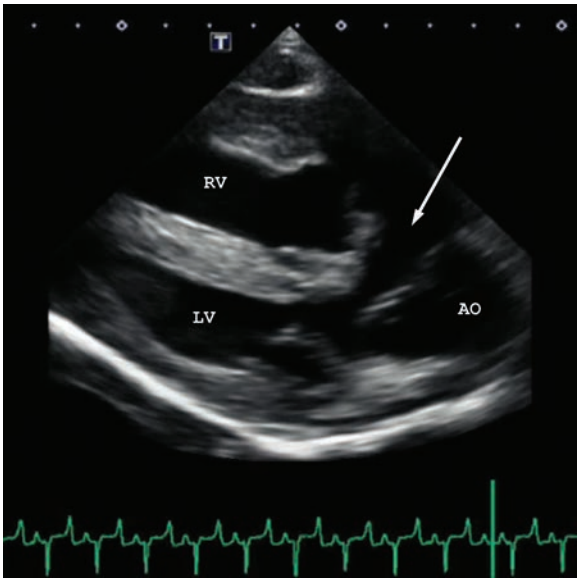

Figure 10.42 Long axis five-chamber echocardiographic view of a heart with tetralogy of Fallot. The chambers are labeled as: LV, left ventricle; RV, right ventricle; AO, aorta. A large interventricular septal defect is indicated by the arrow. The aorta has shifted over the mid-septum and receives blood flow from both ventricles. The aortic valve is just above the "AO" label. Portions of the mitral and tricuspid valves can be seen as thickened hyperechoic linear structures in their respective ventricles. Hypertrophy of the RV can be seen just above the RV label.

large VSD obliterates the support structure for the aorta, so it shifts to a position centered over where the IVS would be effectively pushing it toward the RV, known as dextroposition, or "overriding" aorta. Because of the PS, the blood returning to the RV is shunted into the overriding aorta mixing with the oxygenated blood returning from the lungs as it heads toward the body. Dogs with TOF may have a systolic murmur related to the PS, but often are silent.

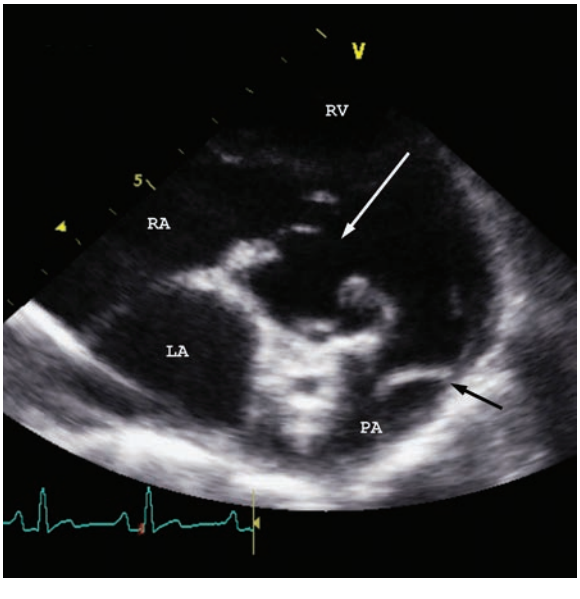

Figure 10.43 Short axis basilar echocardiographic view of a heart with tetralogy of Fallot. The chambers are labeled as: LA, left atrium; RA, right atrium; RV, right ventricle; PA, pulmonary artery. The aorta is in the center of the image. The white arrow points to the interventricular septal (IVS) defect just under the aortic valve. The aortic valve is distorted by the lack of support from the IVS. The black arrow indicates the thickened leaflets and narrow orifice of the pulmonic valve.

A thoracic radiograph of a dog with TOF will show a bulging aorta, enlarged right heart and pulmonary undercirculation from the PS. The mixed venous and arterial blood is then sent to the body causing peripheral cyanosis. The PS also causes the RV to hypertrophy. Surgical correction has been performed in dogs during cardiopulmonary bypass [35]. Due to cost, most patients receive medical management for blood hyperviscosity, and ultimately succumb to complications from hypoxemia, hyperviscosity and/or cardiac arrhythmia [1].

The so called "reverse" PDA or more properly *persistent fetal flow* is also extremely rare and has a similar pathophysiology to Eisenmenger's syndrome, which is a right-to-left shunting VSD due to pulmonary hypertension. In such cases, pulmonary hypertension (Eisenmenger's physiology) can also be present with a PDA causing the flow to shunt from the pulmonary artery to aorta; opposite of the common PDA. The development of pulmonary hypertension happens very early in life, perhaps even such that after parturition, the pulmonary vascular resistance does not reduce, and the fetal path of blood flow is maintained. Typical presentation is an animal showing exercise intolerance, lethargy, syncope, and/or abnormal blood work. These cases usually have a normal auscultation, but have the unique physical examination finding of *differential cyanosis*. Differential cyanosis is noted by observing pink mucus membranes cranially (e.g. the gums or eyes) and cyanotic mucus membranes caudally (e.g. vulva or prepuce). Since the

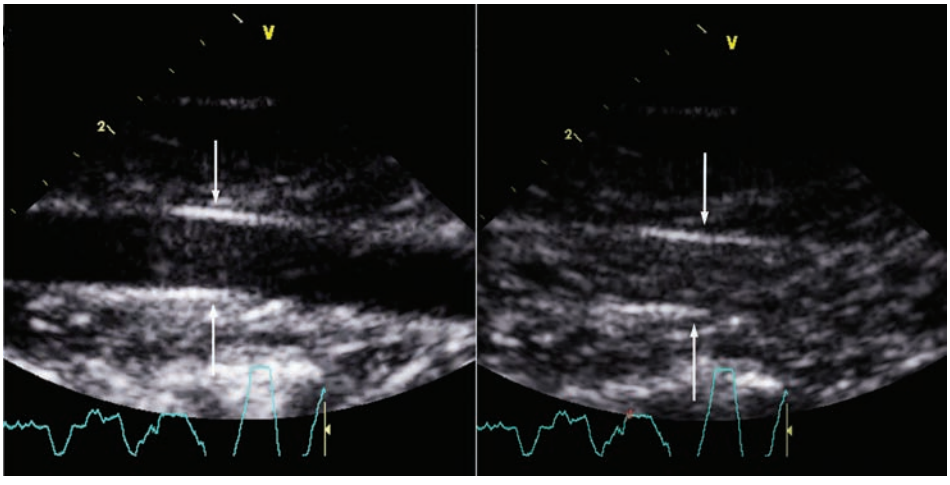

Figure 10.44 Ultrasonic view of the abdominal aorta with positive air-contrast diagnosis of right-to-left shunting patent ductus arteriosus: the left image shows the abdominal aorta before the microbubble injection. The arrows indicate the walls of the aorta seen as two bright lines. The lumen of the aorta is black. The grey area in the center of the aorta is an ultrasound artifact of the highly reflective aortic vascular walls. The right image is the aorta filled with the microbubbles coursing through. The only path for microbubbles to enter this section of the aorta is through a shunt distal to the brachiocephalic and carotid arteries. The only common communication between the pulmonary circulation and the systemic circulation is the ductus arteriosus. If the right-to-left shunting of fetal flow persists after birth, then the so-called "reverse" patent ductus arteriosus is present. Consequently, microbubbles injected into the venous system will travel to the right atrium, right ventricle, and out of the pulmonary artery. The microbubbles will then pass through the ductus arteriosus from the pulmonary artery to the descending aorta where they are visualized in the abdominal aorta.

shunt occurs in the descending aorta, distal to the brachiocephalic and common carotid arteries, only blood caudal to the shunt is mixed with hypoxic blood from the pulmonary artery.

Radiographs of a reverse PDA typically shows dilation of the main pulmonary artery, and lobar branches and a "ductal bump" can sometimes be seen in the VD projection. Echocardiography can be used to diagnose a reverse PDA. Typical findings are PV hypertrophy, dilation of the pulmonary artery, and color-flow Doppler of the main pulmonary artery demonstrates flow through the DA. An air-contrast echocardiogram of the abdominal aorta is positive if microbubbles are seen in the aorta after injection into a peripheral vein. The air contrast microbubbles travel from the venous return, to the RV, to the pulmonary artery, across the DA and into the aorta where they travel caudally (Figure 10.44).

Treatment of reverse PDA entails management of polycythemia and hyperviscosity with phlebotomy or drugs. Surgical closure in dogs with reverse PDA is not recommended. The supersystemic pulmonary vasculature pressure will overburden the RV if the shunt is closed, leading to sudden death. Medications, such as sildenifil can be used to lower pulmonary arterial pressures in other right-to-left shunts [6, pp. 437–475] but their use is still novel in reverse PDA.

Cor triatriatum dexter and sinister are a developmental defect of the atria (right=dexter, left=sinister) in which a third atria is created by an abnormal extra membrane restricting flow into the atria and on to the ventricle (Figure 10.45). Cor triatriatum dexter has been reported in dogs, but not in cats. It may cause signs of right heart failure and has been palliated with balloon dilation of the abnormal membrane [36, 37].

Cor triatriatum sinister has only been reported in cats, along with supervalvular mitral stenosis [38, 39] (Figures 10.46 and 10.47). This condition leads to left-sided CHF. In the case of cor triatriatum sinister, standard cardiac catheterization is difficult due to the anatomical arrangement preventing passage of catheters from the arterial circulation

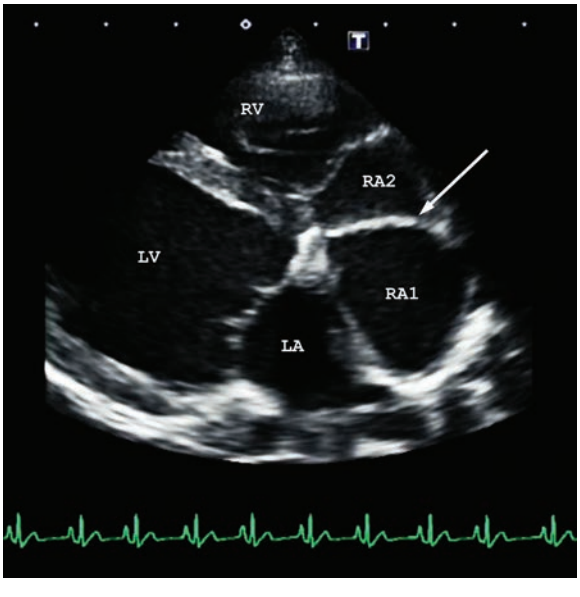

Figure 10.45 Long axis echocardiographic view of cor triatriatum dexter: the chambers are labeled as: LA, left atrium; LV, left ventricle; RA1, distal right atrial chamber; RA2, proximal right atrial chamber; RV, right ventricle. The arrow indicates the membrane separating the two portions of the right atrium. A small non-valved ostium allows blood flow to enter RA2 from RA1. The severity of disease is related to the opening; larger orifices leading to better outcomes.

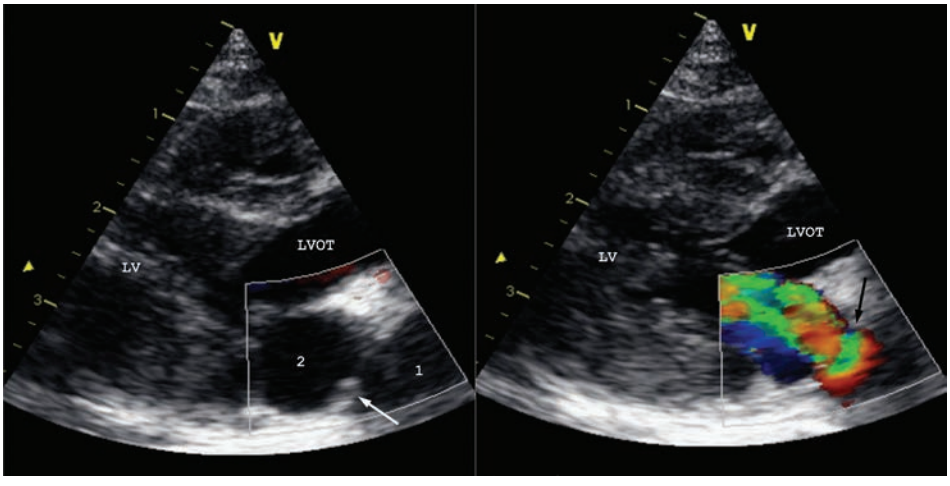

Figure 10.46 Long axis echocardiographic view of cor triatriatum sinister (CTS): this twin image shows the two chambers of the left atrium in CTS. The left image chambers are labeled as: LV, left ventricle; LVOT, left ventricular outflow tract; 1, distal left atrial chamber; 2, proximal left atrial chamber. A slight hyperechoic area (arrow) between 1 and 2 is the membrane separating the two atria. The right image is the same view with color-flow Doppler to demonstrate high-velocity blood flow. The arrow marks the location of the ostium through the membrane at the narrowing of the color Doppler signal. The dome shape color to the lower right shows the point at which blood become turbulent as it enters the opening. The blood then streams directly through the proximal chamber 2, and into the LV. The severity of disease is related to the opening; larger orifices leading to better outcomes.

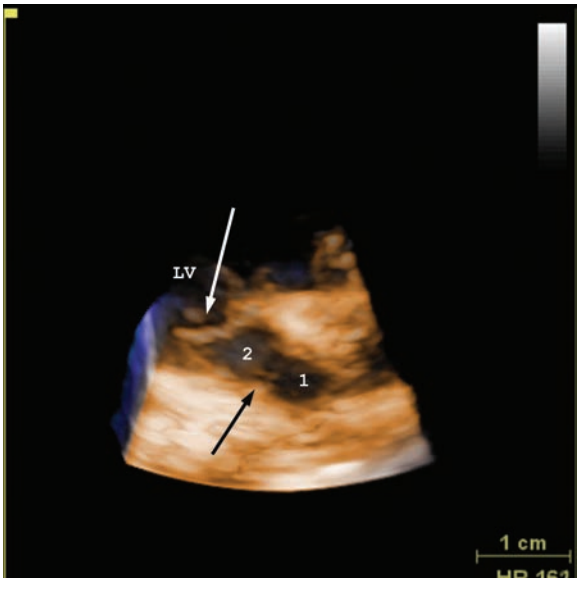

Figure 10.47 Three-dimensional image of cor triatriatum sinister. It should be viewed as one does a block suspended in space, with one corner visible. The viewer sees mostly one side (yellowish) and only a portion of a side going away on the left (blue). The distal left atrial chamber is labeled 1 and the proximal chamber labeled 2. The left ventricle (LV) is back and away from the viewer. The white arrow points to the mitral valve and the black arrow to the membrane separating 1 and 2.

retrograde to the LA. A hybrid open heart surgery and balloon dilation procedure has been reported with some success [40]. Medical management of CHF is most commonly elected by clients.

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