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PDA is the most common congenital heart defect of dogs; it also occurs in cats. PDA causes a left-to-right shunt that results in volume overload of the left ventricle and produces left ventricular dilation and hypertrophy. Progressive left ventricular dilation distends the mitral valve annulus causing secondary regurgitation and additional ventricular overload. This severe volume overload leads to left-sided congestive heart failure and pulmonary edema, usually within the first year of life. Atrial fibrillation may occur as a late sequel due to marked left atrial dilation.

Diagnosis

CLINICAL PRESENTATION
Signalment - PDA is seen more commonly in purebred, female dogs. Maltese, Pomeranians, Shetland sheepdogs, English springer spaniels, keeshonds, biscion frise, miniature and toy poodles, and Yorkshire terriers are at increased risk to develop PDA. A genetic basis has been established in poodles.

History - Most young animals with PDA are asymptomatic or have only mild exercise intolerance. The most common complaint in symptomatic animals with left-to-right shunts are cough or shortness of breath (or both) due to pulmonary edema. Animals with right-to-left or reverse PDA may be asymptomatic or have exercise intolerance and hindlimb collapse on exercise.

Physical examination findings
The most prominent physical finding associated with PDA is a characteristic continuous (machinery) murmur heard best at the left heart base. The left apical cardiac impulse is prominent and displaced caudally and a palpable cardiac A "thrill" often is present. Femoral pulses are strong or hyperkinetic (water hammer pulse) due to a wide pulse pressure caused by diastolic runoff of blood through the ductus. Tall R waves (> 2.5 mV) or wide P waves on a lead II electrocardiogram are supportive of the diagnosis, but not always present. Atrial fibrillation or ventricular ectopy may be present in advanced cases.

The physical examination findings in animals with right-to-left or reverse PDA differ from those with left-to-right shunts. “Differential” cyanosis is typically present (i.e., cyanosis is most apparent in the caudal mucous membranes), but cyanosis may also be noted in the cranial half of the body in some animals. Cyanosis occurs because there is admixture of non-oxygenated blood (from the pulmonary artery) with the oxygenated aortic blood. Femoral pulses are normal. A systolic cardiac murmur, rather than a machinery murmur, is often present. However, a murmur may not be ausculted if polycythemia is present or if left and right sided pressures are nearly equal and shunting of blood through the ductus is minimal.

Radiography/Echocardiography
Thoracic radiographs typically show left atrial and ventricular enlargement, enlargement of pulmonary vessels, and a characteristic dilation of the descending aorta on the dorsoventral view. Echocardiography provides information that further confirms PDA and helps rule concurrent cardiac defects, but is not invariably required to establish the diagnosis. Echocardiographic findings that support a diagnosis of PDA include left atrial enlargement, left ventricular dilation and hypertrophy, pulmonary artery dilation, increased aortic ejection velocity, and a characteristic reverse turbulent Doppler flow pattern in the pulmonary artery.

With right-to-left PDA, thoracic radiographs show evidence of biventricular enlargement and marked enlargement of the pulmonary artery segment. Pulmonary arteries may also appear tortuous. A right-to-left PDA can be documented by performing a saline bubble contrast echocardiogram. Observing bubbles in the descending aorta, but not in any left sided cardiac chamber, is diagnostic.
Medical management
Animals with pulmonary edema should be given furosemide for 24 to 48 hours prior to surgery. If atrial fibrillation is present, the ventricular response rate should be controlled using digoxin (with or without α-adrenergic blockers or calcium channel blockers) prior to surgery. If hemodynamically significant arrhythmias are present they must be controlled. Complete resolution of clinical signs of congestive heart failure may be difficult with medical management alone.

Surgical treatment
Surgical correction of PDA is accomplished by ligation of the ductus arteriosus. Ligation of PDA is considered curative and should be performed as soon as possible after diagnosis. Secondary mitral regurgitation usually regresses after surgery due to reduction in left ventricular dilation. Inadvertent ductal rupture during dissection is the most serious complication associated with PDA repair. The risk of this complication decreases as the surgeon's experience increases. Small ruptures, especially those on the back side of the ductus, often respond to gentle tamponade, but will enlarge and worsen if dissection is continued. Large ruptures must be controlled immediately with vascular clamps and then repaired with pledget-buttressed mattress sutures. Once bleeding is controlled, a decision must be made whether to continue surgery, or to abandon surgery in favor of repair at a later time. Second surgeries are more difficult due to adhesions at the surgical site, so complete occlusion should be attempted during the initial procedure, if possible. Often, simple ductal ligation is not possible after a rupture has occurred. In such instances, surgical alternatives include ductal closure with pledget-buttressed mattress sutures or ductal division and closure between vascular clamps. The divided ductal ends are closed with a continuous mattress suture oversewn with a simple continuous pattern. Ductal closure without division is safer than surgical division, but re-cannulation of the ductus may occur. Because ductal division requires added technical expertise, it should be undertaken only by experienced surgeons.

SURGICAL TECHNIQUE
Perform a left 4th space intercostal thoracotomy. Identify the left vagus nerve as it courses over the ductus arteriosus and isolate it using sharp dissection at the level of the ductus. Place a suture around the nerve and gently retract it. Isolate the ductus arteriosus by bluntly dissecting around it without opening the pericardial sac. Pass a right-angle forceps behind the ductus, parallel to its transverse plane, to isolate the caudal aspect of the ductus. Then, dissect the cranial aspect of the ductus by angling the forceps caudally approximately 45 degrees. Complete dissection of the ductus by passing forceps from medial to the ductus in a caudal to cranial direction. Grasp the suture with right-angle forceps. Slowly pull the suture beneath the ductus. If the suture does not slide easily around the ductus, do not force it. Regrasp the suture and repeat the process, being
careful not to include surrounding soft tissues in the forceps. Pass a second suture using the same maneuver. Alternatively, the suture may be passed as a double loop and the suture cut so that you have 2 strands. Slowly tighten the suture closest to the aorta first. Then, tighten the remaining suture.

VASCULAR RING ANOMALIES
The most common type of vascular ring anomaly is a persistent fourth right aortic arch, right dorsal aortic root, and rudimentary left ligamentum arteriosum (left sixth arch). The left pulmonary artery and the descending aorta are connected by the ligamentum arteriosum. The esophagus is encircled by the ligamentum arteriosum (or patent ductus arteriosus) on the left, the base of the heart and pulmonary artery ventrally, and the aortic arch on the right. The esophagus is constricted by this vascular “ring” and begins to dilate cranially as food accumulates. Food not passing beyond the constriction is intermittently regurgitated. Chronic regurgitation predisposes to aspiration pneumonia. Approximately 95% of those diagnosed with vascular ring anomalies will have a persistent right aortic arch (PRAA). Persistent left vena cava occurs in conjunction with PRAA in about 40% of the cases.

Diagnosis
Signalment. Vascular ring anomalies occur in both dogs and cats, but are more common in dogs. German shepherds, Irish setters, and Boston terriers are the most commonly affected dog breeds. Siamese and Persian cats have been diagnosed more often than other cat breeds. Males and females are equally affected. The condition may affect multiple animals in a litter. Vascular ring anomalies are present at birth. Clinical signs are usually evident at the time of weaning, most being diagnosed between 2 and 6 months of age. The condition may not be recognized until later in life if obstruction is partial and signs are mild. Early diagnosis and treatment of PRAA may improve the prognosis.

History. The classic history is acute onset of regurgitation when solid or semisolid food is first fed. Regurgitation of undigested food occurs soon after eating early in the disease; later it may occur at variable times (minutes to hours). Affected animals may grow slower than litter mates and appear malnourished. They often have a voracious appetite, some immediately eating the regurgitated food. Coughing with respiratory distress may be a result of aspiration pneumonia and/or tracheal stenosis secondary to a double aortic arch.

Physical Examination Findings
Affected animals are often thin and small. An enlarged esophagus may sometimes be palpated at the thoracic inlet and neck. The thoracic inlet and caudal neck area may bulge when the chest is compressed. Murmurs are rare; an occasional patient may have a continuous murmur associated
with concurrent patent ductus arteriosus. Pneumonia may be suggested by auscultating coarse crackles or finding fever.

**Radiography/Ultrasonography/Endoscopy**
Thoracic radiographs may reveal a dilated esophagus cranial to the heart containing air, water, or food. The trachea may be displaced ventrally and the esophagus may overlap it. Signs of pneumonia may be identified. Positive contrast radiography using a barium suspension or barium with food will demonstrate esophageal constriction at the base of the heart with varying degrees of esophageal dilatation extending cranially. The caudal esophagus is usually a normal size, although sometimes it is dilated. Fluoroscopy is beneficial in evaluating esophageal motility. The dilated esophagus does not usually demonstrate normal peristaltic contractions. Although not routinely performed, angiography is beneficial in preoperatively identifying the type of vascular ring anomaly and other cardiac anomalies. Echocardiography may also be beneficial. Endoscopic examination of the esophagus helps rule out other causes of esophageal stricture or obstruction and may reveal esophageal ulceration. Tracheoscopy is not routinely performed, but may document tracheal lumen narrowing secondary to external compression.

**SURGICAL TREATMENT**
Surgical treatment of PRAA is described below. Other types of vascular ring anomalies can be managed in a similar fashion. A persistent left vena cava often covers the left ventral area of the vascular ring. A persistent right ligamentum arteriosum and some aberrant right subclavians should be approached from the right side. Angiograms are helpful in patients with double aortic arches to determine which arch is dominant and if adequate circulation can be maintained after transection of the other arch. It may not be possible to relieve constrictions caused by a double aortic arch. If the animal is severely debilitated, place a gastric feeding tube for several days before surgery.

Some surgeons attempt to decrease esophageal lumen size if the esophagus is severely dilated and not expected to return to normal size. This is accomplished by placing a series of nonpenetrating “plication” or “gathering” sutures in the accessible lateral esophageal wall. Alternatively a portion of the esophagus may be resected. These techniques are not recommended routinely because they increase the risk of complications.