Patent Ductus Arteriosus in Dogs

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Patent ductus arteriosus (PDA) is the most common congenital cardiovascular defect in canines.1 PDA represents a failure of the fetal ductus arteriosus to properly close after birth. PDA is usually recognized in young dogs, but is occasionally seen in middle aged to older animals. Four clinical presentations of PDA have been described and likely represent a continuum of severity. They are based on the size of the patency and the direction of the shunt. Many dogs are asymptomatic at the time of diagnosis. Approximately 50% of untreated cases will die of congestive heart failure within the first year of life.2

**Incidence**

PDAs occur more frequently in female dogs (3:1 sex ratio) and in certain breeds.3 The breeds most affected by PDA are: miniature and toy poodle, collie, Pomeranian, Shetland sheepdog, Maltese, English springer spaniel, Yorkshire terrier, German shepherd, and cocker spaniel.3,4,5,6 Affected dogs should not be bred. Inheritance is thought to be polygenic. Phenotypically, normal dogs that are closely related to dogs with PDA may be carriers and also should not be bred.4

**Pathogenesis**

Normal functional ductal closure occurs within hours after birth. Within a few days, massive changes in the ductal wall transform the structure into a fibrous cord, which is referred to as the ligamentum arteriosum.4 Failure of ductal closure results from histologic abnormalities of the ductal wall. Normal ductal wall contains a loose branching pattern of circumferential smooth muscle. Dogs with PDA have a subnormal amount and asymmetric distribution of this ductus specific smooth muscle. A reciprocal increase in amounts of elastic tissues in areas that should be smooth muscle results in a ductal wall that is more like the aorta. These wall changes preclude the normal closure of the ductus.7

**Pathophysiology**

**Left-to-Right Shunt** Since aortic pressure is normally greater than pulmonary artery pressure, blood continuously shunts from the aorta into the main pulmonary artery. This causes a continuous "machinery" cardiac murmur. Increased pulmonary blood flow and venous return to the left heart are the result. The volume overload causes left atrial and ventricular dilation, hypertrophy, and increased ventricular diastolic pressure. Left ventricular failure with pulmonary edema may develop from volume overload, especially with a large defect and relatively normal pulmonary vascular resistance. The left ventricular stroke volume is increased due to increased filling (Frank-Starling principle). Aortic diastolic pressure is low due to run-off of blood through the ductus. This widens the pulse pressure and results in a hyperkinetic or bounding pulse.8

**Right-to-Left Shunt** A small percentage of PDAs exhibit "reversed" flow, with blood shunting into the descending aorta from the pulmonary artery. These dogs have a very high pulmonary vascular resistance, which induces systemic pressures in the right ventricle and pulmonary artery. Decreased pulmonary flow, a small left ventricle and marked hypertrophy of the right ventricle.

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ventricle are seen along with evidence of caudal hypoxemia. The exact mechanism of the increased pulmonary vascular resistance is not completely understood. In these cases shunt reversal is thought to occur at an early age. The ductal lumen in these dogs is anatomically very large.

**Clinical Description**

**Type 1** (small PDA) is characterized by an asymptomatic left to right shunt with a high frequency continuous murmur heard only at the high left heart base. The heart rate and pulse quality are normal in these dogs and a precordial thrill is faint or not present at the left heart base. Radiographs and electrocardiography (ECG) are normal in these dogs, even at two years. Surgery is recommended, but not urgent.3

**Type 2** (medium PDA) is also characterized by an asymptomatic left to right shunt. However, PDAs in this category have a coarse, continuous murmur heard at the left heart base and left apex. The pulse is normal to slightly bounding, and mild to moderate left heart enlargement develops by one year of age. A small-to-medium sized ductus aneurysm may be present, and left ventricular enlargement is usually indicated on the ECG by R waves exceeding 3mV in lead II. Borderline increases in pulmonary vascular markings are present.3

**Type 3** (large PDA) is subdivided into those with (3a) and without (3b) congestive heart failure (CHF). Dogs with Type 3a usually have a reduced exercise capacity, a coarse continuous murmur and thrill over the left thorax, a systolic murmur of mitral regurgitation, bounding pulse due to wide pulse pressure, marked left heart enlargement by six months of age, a medium to large ductus aneurysm, a significant increase in pulmonary vascular markings and ECG lead II R waves possibly exceeding 5mV. Surgery is recommended immediately in these cases. Type 3b is characterized by the above signs plus those of CHF including dyspnea caused by pulmonary edema, cachexia, and possible atrial fibrillation or other arrhythmias. The pulmonary edema must be resolved as much as possible before surgery is performed. This is accomplished by cage rest, furosemide, supplemental oxygen, an angiotensin converting enzyme inhibitor and digitalization.3

**Type 4** (large PDA) is associated with pulmonary hypertension. This type is either a right-to-left or equally balanced shunt and is found in dogs from 2 weeks to 12 years. Prognosis and longevity are poor in dogs with pulmonary pressures high enough to cause right to left shunting. Hindleg weakness or collapse with exercise may be present. Differential cyanosis (caudal, but not oral membranes) may be present and pulses are normal to weak. Usually a murmur or precordial thrill is not present, but a split and/or prominent second heart sound may be detectable. A right axis deviation on ECG, large right heart and main pulmonary artery with normal to decreased pulmonary vessels on radiographs, and polycythemia are also features of this type. Surgery is contraindicated in Type 4 PDAs, and medical management including periodic phlebotomy is indicated.3

An incomplete ductal closure occurs in some dogs. this is the non-patent forme fruste (or ductus diverticulum)3,4, where the ductus is completely closed at one point (usually the pulmonary arterial end), but the remainder of the ductus stays open. There is no murmur nor any other clinical sign associated; diagnosis is made (usually incidentally) by angiography.

**Diagnosis**

The physical examination and history provide clues in the diagnosis and classification of a PDA. Knowing what breeds are predisposed to certain defects may suggest a problem, but assessing mucous membrane color, pulse character, capillary refill time, presence or absence of a precordial thrill, and heart sounds are the most important part of the initial work up and suggest the differential diagnosis.5 It is important to assess both the cranial (oral) and caudal (vaginal or preputial) mucous membranes when exam-
Visualization and isolation of patent ductus arteriosus.

ining a dog with a suspected cardiovascular defect, because a reversed or right-to-left shunting PDA can result in a differential cyanosis. This occurs because the ductus arteriosus is distal to the arteries supplying the head and therefore the cranial mucous membranes appear normal. Dark red mucous membranes suggest polycythemia which may be in response to chronic hypoxia as is seen in dogs with large right to left shunts. Decreased capillary refill time indicates a decrease in the regional blood flow and could signal low cardiac output and heart failure.5

Chest palpation should be done to check for caudal displacement of the cardiac impulse which suggests cardiomegaly and also to check for a precordial thrill. The arterial pulse character is most easily assessed on the femoral artery. A bounding or “water hammer” pulse suggests a PDA and is due to a widened pulse pressure.5 Signs of right heart failure such as jugular venous distension, hepatosplenomegaly and ascites are not expected with defect.5

Thoracic auscultation should include evaluation of all four valvular areas and determining the point of maximal intensity (PMI) of the murmur. The PMI for a PDA is the high left heart base. If an apical systolic murmur is heard, evaluation of the heart base for a diastolic component is warranted as these are features of a PDA. Murmurs should be characterized by grade I through VI, timing (left to right shunting PDAs have a continuous murmur), and radiation of the murmur. Finally, the acoustic quality of the murmur should be assessed. PDAs produce a continuous murmur of a machinery like quality which is loudest during systole.5

Thoracic radiographs taken routinely may reveal cardiomegaly, left atrial and auricular (atrial appendage) enlargement, enlarged pulmonary vessels, aneurysmal dilation of the proximal descending aorta and possibly signs of left heart failure such as
pulmonary venous distension or edema.6,9

Electrocardiography in dogs with PDA usually reveals wide P waves, deep Q waves in lead II and very tall R waves in the caudal leads.6

Echocardiography performed on dogs with PDA reveals dilation of the pulmonary trunk, left atrial and ventricular enlargement and sometimes the ductus itself, can be visualized. Doppler studies document continuous and turbulent flow into the pulmonary artery from the aorta.6

Cardiac catheterization is usually not necessary, but when performed normally reveals a higher oxygen content in the pulmonary artery than the right ventricle and a wide aortic pulse pressure.6 Structural changes in the cardiac chambers and abnormal ductal flow are evident with angiocardiography.

Treatment: Surgery

Once a left-to-right PDA has been diagnosed, surgical ligation is recommended, as discussed earlier. Several surgical techniques are available for PDA surgery. Atropine may be given preoperatively because manipulation of the left recurrent laryngeal and vagus nerves may cause reflex slowing of the heart.3,10 A left lateral thoracotomy is performed at the fourth or fifth intercostal space. Positive pressure ventilation is started when the thoracic cavity is exposed. The left cranial, and sometimes the middle, lung lobes are reflected caudally.3,10,11,12 The ductus is now seen ventral to the aorta and dorsal to the pulmonary artery. Fremitus can be palpated ventral and anterior to the ductus where the blood flow from the shunt is reflected off the pulmonary artery near the pulmonary valve.10

At this point, various options are available to the surgeon. The recurrent laryngeal and vagus nerves are reflected back with a suture placed in a pleural flap or directly with umbilical tape.10,11 The use of gas-sterilized swabs to dissect the ductus has been suggested as being safer and more expedient than traditional methods.11 Others advocate the use of right-angled forceps to reach the ductus.10,12 Once the ductus is dissected, it can be ligated with a variety of suture materials and a variety of methods of suture placement.3,10,11,12 The use of tantalum hemostatic clips for occlusion of a patent ductus was first introduced in 1971.13 While some believe ducts closed in this way recanalize, no published evidence of recanalization was found by the authors.

Another method is division of the ductus between clamps. This is usually reserved for large PDAs and involves placing patent ductus clamps adjacent to the aorta and pulmonary trunk, transecting the ductus, and closing first the pulmonary artery side and then the aortic side in a continuous over-and-over pattern with silk suture.3

After the ductus has been occluded, the pericardial edges are apposed to prevent herniation of the left auricle and a chest tube is placed several rib spaces posterior to the thoracotomy. Rib sutures are then placed, the ribs are drawn together and the sutures are tied. The muscle layers and skin are then closed.10 Negative pressure is placed on the chest tube when the thoracic cavity is no longer open. The chest tube may be removed immediately or whenever it is no longer needed.10,11

Complications of PDA surgery include hemorrhage, congestive heart failure, endocarditis, thromboembolism, recanalization, rupture of the ductal aneurysm, and arrhythmia.3 Postoperative care consists of hospitalizing the dogs for 2 days to 3 days and standard monitoring. A systolic murmur of mitral regurgitation caused by annular dilation may be noticed, but should disappear rapidly.3 Exercise should be restricted for one month and digoxin, if the dog was on it pre-operatively, should be continued until a recheck examination is performed in 2 months. Most dogs live a normal lifespan if the PDA is surgically corrected before the onset of congestive heart failure.3

Medical Management

Medical management is indicated for re-
versed PDA's with secondary polycythemia. Treatment includes enforced rest, exercise limitation, stress avoidance, and maintenance of packed cell volume (PCV) between 62% to 68%. Fluid volume should be maintained during phlebotomy with crystalloid solutions.

References


