Patent Ductus Arteriosus in the older Dog

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Abstract

Objectives: The objectives of this study were to describe the clinical course of Patent Ductus Arteriosus (PDA) in older canine patients (defined as patients greater than 24 months at the time of presentation) with and without intervention. This study was also undertaken to evaluate if adult dogs with persistent flow across the ductus should still be closed, what the benefits are from late closure, and to hypothesise why some animals still continue to deteriorate despite closure.

Background: Currently accepted therapies for patent ductus arteriosus are surgical ligation and transcatheter coil embolisation. The majority of dogs are diagnosed and treated at young age. Some older dogs survive with few clinical signs and live a normal life span without intervention. Some dogs deteriorate despite intervention.

Methods: The case records of 24 dogs that had reached 24 months of age before diagnosis were reviewed and those animals that had no concurrent congenital cardiac diseases were included in the study (n = 21). Those animals that were still alive were requested to participate in a long-term follow-up study.

Results: After closure of the ductus (n = 16), the clinical signs disappeared in all but one animal. On follow-up of these animals, there was echocardiographic evidence of left ventricular systolic and diastolic dysfunction in many. Late cardiac death was recorded in 3 animals. In dogs where there was no intervention congestive heart failure was a common, but not an inevitable sequel. Development of mitral valve endocardiosis was a common feature.

Conclusions: Older animals with PDA follow an individual course, independent of pre-existing heart failure. Irreversible left ventricular dysfunction is common, however it does not seem to affect the clinical course. These data show a favourable outcome in a high percentage of adult dogs diagnosed with PDA that undergo closure. Outcome without intervention was less favorable.

Key Words

Patent ductus arteriosus - dog - adult - clinical features - outcome.

The ductus arteriosus is a normal foetal structure that shunts blood from the pulmonary artery to the aorta. In pups, the ductus arteriosus functionally closes within days after birth[1]. Patent Ductus Arteriosus (PDA) is due to failure of closure and is one of the most commonly recognised congenital cardiac lesions in dogs[2,3,4]. Currently accepted therapies for PDA are surgical ligation and transcatheter coil embolisation[5].

Many dogs with a left-to-right shunting PDA have no history of clinical problems. However, several do present in left heart failure and, if left untreated, up to 65% may die of left-sided heart failure within the first year of life[6]. In some dogs, clinical signs are not apparent until they are mature, but there usually are signs before the dog is 3 years old[7]. The appearance of signs in older dogs is unusual[7]. Occasional dogs survive with few clinical signs and may live a normal life span[8, 9, 10, 11, 12]. However, available information has been based on single case reports or case studies with little long-term information.

The outcome of dogs with PDA referred to the Hospital for Small Animals of Edinburgh University between January 1990 and December 2000, with and without ductal closure, was evaluated. The results were correlated with earlier reports in an attempt to increase knowledge of the clinical course and improve the management of these older patients. This study was undertaken to evaluate if adult dogs with persistent flow across the ductus should still be closed, what

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the benefits are from late closure, and to hypothesise why some animals still continue to deteriorate despite closure.

Method and Patients

The case records of 24 dogs that had reached 24 months of age before diagnosis of their PDA, presented at the Hospital for Small Animals of the Royal (Dick) Veterinary School of Edinburgh (Scotland) from 1990 to 2000, were reviewed retrospectively. Diagnosis was based on demonstrating a PDA by Colour Doppler echocardiography in all 24 cases. Only those animals diagnosed with a left-to-right shunting PDA as isolated lesion were included in the study (n = 21).

Owners were initially contacted by telephone to determine survival rates and reason of death in animals that were deceased. Those animals that had survived were requested to participate in a long-term follow-up study.

Retrospective study

The history, signalment, clinical examination and Doppler flow echocardiographic findings were obtained from the case records. The electrocardiograms and thoracic radiographs were evaluated by one author (NVI). The six-lead electrocardiographic strips (ECG) were assessed and measured by the recommendations of Tilley[13]. Thoracic right-lateral and dorsoventral radiographs were reviewed in couplets. Cardiomegaly was assessed using the Vertebral Heart Sum (VHS)[14]. Pulmonary perfusion was assessed in the dorsoventral view by comparing the width of the pulmonary artery and the width of the 9th rib where they crossed each other[15]. Data from the echocardiograms and Doppler studies obtained by multiple cardiologists were retrieved and compared with previously published normal values[16]. The ultrasound studies were all performed on a Vingmed 700 (BMS, Scotland) or Esaote Challenge 3000 (Esaote, Italy) machine. M-Mode measurements were obtained from the standard right-parasternal short-axis view[4]. Left atrial size was assessed subjectively by combination of the two-dimensional and M-Mode findings. The diameter of the ductus arteriosus was estimated at the pulmonary artery side from the right parasternal short-axis view via echocardiography, or at surgery or via angiography depending on the technique used for closure.

Follow-up study

Animals represented to the Cardiology clinic were fully assessed (including clinical examination, electrocardiography, thoracic radiography and Colour flow Doppler echocardiography). The six-lead electrocardiographic (ECG) strips (Schiller, Cardiovit 60, Switzerland) were assessed according to the recommendations of Tilley.[13] Dorsoventral and right lateral radiographs were obtained in those animals that could be positioned without sedation. Radiographs pre-and post-closure were blinded and assessed independently of each other. The follow-up Doppler echocardiographic examinations (including transthoracic 2-D, M-mode, spectral, and colour-flow echocardiographic evaluations) were performed and analysed by one author (NVI) and compared with values recorded in the retrospective study. The follow-up ultrasound studies were all performed on a Vingmed CFM 800 (BMS, Scotland) machine using a transducer array of 2.25 to 5 MHz, depending on the size of the dog. Echopac, was used to analyse the data (average of 5 consecutive heart cycles) and videotapes were reviewed for the presence of flow across the ductus. M-Mode measurements were obtained from the standard right-parasternal short-axis view[4]. Left ventricular systolic function was interpreted as being compromised if one or more of the following were identified: fractional shortening (FS) < 25%[17], ejection fraction (EF) calculated by Simpson's rule < 40%[16] and systolic time intervals (PEP/ET ratio > 0.44 sec measured from spectral Doppler of aortic flow)[16]. Left atrial size was assessed by combination of the two-dimensional (left atrial diameter parallel to the mitral valve annulus before opening of the mitral valve 18 and 2-D left atrium/aorta ratio)[19] and M-Mode findings (left atrium/aorta ratio)[16]. Pulmonary hypertension was considered to be present if the pulmonary insufficiency jet exceeded 2.2 m/s or if the tricuspid regurgitation jet exceeded 2.8 m/s[20].

Comparing data and statistical analysis

Statistical analysis of the comparative data was performed with Sigmastat, (v. 2.03, SPSS Inc.) software. Echocardiographic data pre- and post-closure were compared via a paired Student's t-test. For correlation between age of presentation and radiographic signs the Mann Whitney Rank test was used. Significance for all tests was obtained if $P < 0.05$.

Results

Between January 1990 and December 2000, of a total population of 101 dogs that were diagnosed with PDA, twenty-four animals were 24 months of age or older at initial presentation (Table 1). In 1 of these older dogs the PDA was associated with pulmonary hypertension, tricuspid valve dysplasia and an atrial septal defect, resulting in right-to-left shunting. One dog was diagnosed with concurrent pulmonic stenosis and another dog with additional aortic stenosis. The progress of the remaining 21 patients, in whom the shunt was from left to right, is the subject of the present study. Full clinical records were available in all 21 cases. ECG- strips were retrieved and analysed in 17 dogs. Thoracic radiographs were available in 16 dogs. Echocardiographic data were available in 20 animals. Fourteen animals had their ductus surgically ligated. In 2 animals closure was by coil embolisation. A further 5 owners declined intervention for their animals. Follow-up data were obtained in all 21 animals. Eleven of the 21 dogs returned for complete cardiac assessment.

Epidemiology

Initially, there were 12 breeds represented with German Shepherd Dogs (n = 4), Labradors (n = 3), Cavalier King Charles Spaniels (n = 2), mongrels (n = 2), Border Collies (n = 2) and West Highland White terriers (n = 2) being the most common (Table 1). The age range at the time of diagnosis was 24-108 months (mean 51, median 48). There were 17 females and 4 males. All animals over 6 years of age at initial presentation were females.

Findings at initial presentation and at follow-up

Medical history (Table 1 and 2)

Eleven dogs (52 %) had no clinical signs at initial presentation. Age of onset of clinical signs coincided mostly with age of presentation, only one animal, where the murmur was picked up at routine vaccination, had experienced exercise intolerance for some months prior to presentation.

After closure of the ductus, the clinical signs disappeared in all but one animal, which continued to cough until it deceased, in a road traffic accident, at the age of 14. The episodes of anxiety, observed in one dog, disappeared despite only partial closure of the PDA by coil embolisation. One dog redeveloped, after initial improvement with the help of medication, biventricular heart failure 24 months after successful surgical ligation of the ductus. However, this dog had developed severe mitral and tricuspid valve endocardiosis.

Clinical findings (Table 1)

A left basal continuous murmur was detected in all animals and was graded equal to or higher than IV/VI in 19/21 dogs. The remainder 2 dogs had a grade III/VI continuous murmur. Additional systolic murmurs with point of maximum intensity over the mitral valve were audible in 33% of the cases. The majority of the dogs were in sinus rhythm or had sinus arrhythmia, only 2 dogs were in atrial fibrillation. Ascites, consistent with right-sided heart failure, was noticed in one of these dogs (dog 33). The right-sided heart failure had been secondary to initial failure of the left ventricle.

At follow-up, only 1 of the dogs that underwent a transcatheter intervention to close the ductus had a persisting left basal continuous murmur (grade III/VI). The two non-occluded dogs had a grade IV/VI (III/VI at initial presentation) and V/VI (V/VI 12 months earlier) continuous murmur respectively. Additional systolic murmurs were audible in 5/11 dogs and they all appeared mitral in origin. Of the mitral murmurs, three were new. One mitral murmur present at initial presentation reduced two grades and another disappeared completely. One animal developed a murmur with point of maximum intensity over the tricuspid valve.

One dog had a chaotic heart rhythm, increased respiratory effort and ascites at representation.

Electrocardiography (Table 3)

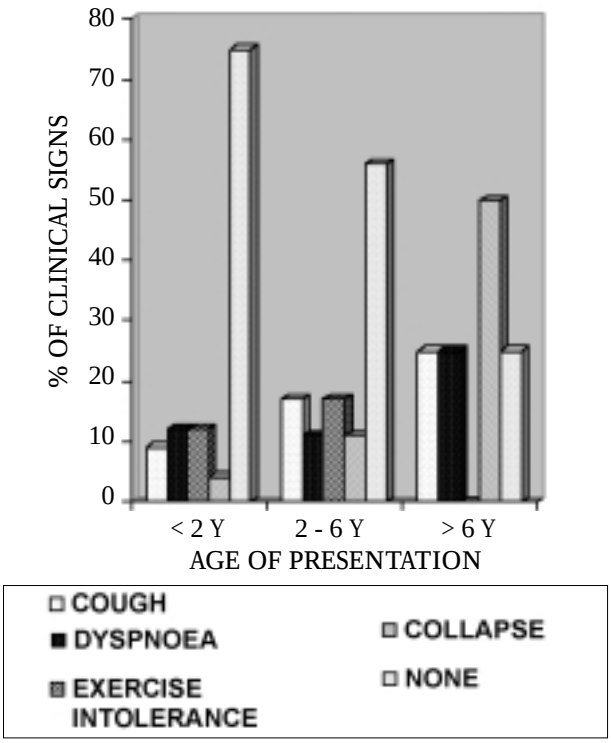
The majority of the animals was in sinus rhythm (53%) or had a sinus arrhythmia (12%). Atrial or ventricular premature complexes were noticed in 4 cases, one of which was in heart failure. Atrial fibrillation was present in 2 animals, both in heart failure. All animals had a normal mean electrical axis.

Table 1 - Epidemiology, clinical signs and findings at initial presentation and at follow-up examination in 21 adult dogs with a left-to-right shunting PDA.

Dog	Breed	Sex	Age in months		Clinical signs			Clinical findings			
No			Dx	Fu	Dx	POST	Fu	Cont Murmur		MV Murmur	
								Dx	Fu	Dx	Fu
78	CORGI	F	24	48	NCS	NCS	NCS	V	N	N	N
20	CKCS	M	24	132	EI	NCS	NCS	III	N	N	III
27	WHWT	FN	24	132	COL	NCS	NCS	V	N	III	N
28	X	F	24		C,EI	NCS		V		IV	
96	JRT	FN	24		NCS			VI		N	
102	OESD	F	24	84	D,L	NCS	NCS	V	N	IV	II
26	X	FN	30		NCS			VI		IV	
3	GSD	M	36		NCS			IV		N	
16	WEIM	FN	36		NCS			V		N	
73	NF	MN	36	60	C,EI,L	NCS	C,EI	V	N	N	II
7	ESS	F	48		C,D	C	C	V		N	
80	GSD	M	48	72	NCS	NCS	NCS	V	N	N	N
85	BC	F	48	84	NCS	NCS	NCS	V	N	N	N
30	LAB	M	60		NCS			V		N	
87	LAB	F	108	120	ANX	NCS	NCS	V	III	N	N
75	LAB	F	108	132	COL	NCS	NCS	VI	N	N	N
68	CKCS	F	48	72	NCS		NCS	III	IV	N	II
49	BC	FN	54		COL			V		III	
82	WHWT	F	60	72	NCS		NCS	V	V	II	III
33	GSD	FN	96		C,D			+		II	
36	GSD	F	120		NCS			V		N	

GSD: German Shepherd Dog; ESS: English Springer Spaniel; WEIM: Weimaraner; CKCS: Cavalier King Charles Spaniel; X: cross-bred; WHWT: West Highland White Terrier; LAB: Labrador Retriever; BC: Border Collie; NF: Newfoundland; CORGI: Welsh Corgi; JRT: Jack Russell Terrier; OESD: Old English Sheepdog; SHEL: Shetland Sheepdog; I SET: Irish Setter
M: male; F: female; FN: female neutered;MN: male neutered
Dx: at the time of diagnosis; POST: the months following ductal closure; Fu: at follow-up examination
NCS: no clinical signs; C: cough; D: dyspnoea; EI: exercise intolerance; L: lethargy; COL: collapse; ANX: anxiety
MV: mitral valve, N: none

Table 2 - Comparative incidence of clinical features in left-to-right shunting PDA in age groups under 2 years of age (n=67) [42], between 2-6 years of age (n=18) and over 6 years of age (n=4).



Follow-up electrocardiograms were available in 11 animals. The follow-up time ranged from 16 to 116 months (mean 45, median 26). Seven animals had a normal cardiac rhythm at representation, two exhibited occasional ventricular premature complexes. One animal had developed atrial fibrillation 12 months post-closure of its ductus. Neither of the 2 animals that were in atrial fibrillation at initial presentation had converted into sinus rhythm.

Radiography (Table 3)

At initial presentation, cardiomegaly was evident on thoracic radiographs in 13/16 (81%) of the cases when using the VHS system (VHS mean 12.0; median 12.0; range 10.0-14.3). All (100%) animals above 6 years of age had radiographic evidence of cardiomegaly. At follow-up (mean 40 months, median 25, range 2.5-107 months) there were radiographs available from 9 animals to compare pre- and post-data. The size of the cardiac shadow remained exactly the same in 2 animals, reduced in 6 animals and increased in 1 animal. The latter never had its ductus closed. One of the dogs whose cardiac shadow remained equal in size had developed significant mitral valve endocardiosis. Despite a reduction in heart size, 7 of the 8 occluded dogs with available radiographic data had a VHS above 10.5 vertebrae at follow-up.

The left atrium appeared enlarged in 8/16 (50%) of the radiographs at initial presentation, with tracheal elevation noted in 5/16 (31%) of the cases. The typical 3 bulges (aortic, pulmonic and auricular) on dorsoventral radiographs were

Table 3 - Electrocardiographic and radiographic findings at initial presentation and at follow-up examination in 21 adult dogs with a left-to-right shunting PDA.

Dog No	Breed	ECG THYTHM		RADIOGRAPHY			
		Dx	Fu	VHS		Pulmonary œdema	
				Dx	Fu	Dx	Fu
78	CORGI	S	S				
20	CKCS	S	SA	11.3	11.3	NONE	NONE
27	WHWT	S	S	13.5	11.7	HILAR	NONE
28	X	S		12.0		NONE	
96	JRT	S		12.5		NONE	
102	OESD	S/VPC	S	12.5	12.0	NONE	NONE
26	X	S		11.7		NONE	
3	GSD		AF	12.0		NONE	
16	WEIM	S/VPC		13.5	13.3	HILAR	NONE
73	NF	AF	AF	14.3	14.0	HILAR	GENERAL
7	ESS	SA		12.3	12.3	NONE	NONE
80	GSD	SA	S	10.0	9.5	NONE	NONE
85	BC		SA				
30	LAB	S					
87	LAB	S/VPC	SA				
75	LAB	S	S	11.0	10.8	NONE	NONE
68	CKCS			10.5		NONE	
49	BC	S/VPC		13.0		NONE	
82	WHWT	S	S	10.5	10.8	NONE	NONE
33	GSD	AF					
36	GSD	S		12.0		NONE	

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Dx: at the time of diagnosis; Fu: at follow-up examination
S: sinus rhythm; SA: sinus arrhythmia; AF: atrial fibrillation; VPC: ventricular premature complexes
VHS: vertebral heart sum

Table 4 - Echocardiographic findings at initial presentation and at follow-up examination in 21 adult dogs with a left-to-right shunting PDA.

Dog No	ECHOCARDIOGRAPHY						Endocardiosis	Systolic function	LA size	
	LVd		LVs		FS (%)			FS EF PEP/ET	Dx	Fu
	Pre	Post	Pre	Post	Pre	Post	Fu	Fu		
78	I	N	I	I	38	29		NNN	++	N
20	I	I	I	I	27	34	MVE	NNN		+
27	I	I	I	I	22	15		DDN	++	+
28	I		I		45				++	
96	I		I		27				N	
102	I	I	I	I	24	19	MVE	DDI	++	N
26	I		I		30				N	
3	I		I		32					
16	I		I		24				+	
73	I	I	I	I	38	34	MVE	NNI	+++	+++
7	I		I		19					
80	I	N	I	I	34	19	MVE	DDN	N	N
85	I	I	I	I	32	16		DNN	++	N
30	I		I		35					
87	I	I	I	I	38	29		NNN	+	+
75	I	I	I	I	27	22		DDN	++	N
68	I	I	I	I	-	30	MVE	NNN		+
49	I		I		15					
82	I	I	I	I	36	42	MVE	NNI		++
33	I		I		35					
36	I		I		32					

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FS: fractional shortening; EF: ejection fraction; PEP: pre-ejection period; ET: ejection time
N: normal; D: decreased; I: increased; /: not measurable
LA: left atrial size; N: within normal limits; + mildly; ++ moderately, +++ severely enlarged

Table 5 - Classification, ductal size, treatment and outcome in 21 adult dogs with a left-to right shunting PDA.

Dog No	Breed	Buchanan's classification[5]	Pouchelon's Classification[9]	Additional treatment	Intervention	Ductus diameter (mm)	Outcome (death/Alive)/Reason (Cardiac /Non Cardiac)	Survival in months
78	CORGI	2	4		C	4.7	A	54
20	CKCS	3a	4	enalapril	S		D/NC	136
27	WHWT	3b	4	furosemide	J		A	132
28	X	3a	4		J		D/NC	132
96	JRT	2	4		S	10.0	A	36
102	OESD	3a	4	furosemide	S		A	90
26	X	2	4		J		D	30
3	GSD	2	4		J		D/C	57
16	WEIM	3b	4		J	17.0	D/C	46
73	NF	3b	5		S	20.0	D/C	60
7	ESS	3a	4		S		D/NC	168
80	GSD	2	4		S	8.0	A	78
85	BC	2	4		S	5.0	A	72
30	LAB	2	4		J		D/NC	96
87	LAB	2	4		C	10.0	A	132
75	LAB	2	4		S	9.0	A	138
68	CKCS	1	4		N	3.5	A	84
49	BC	3a	5		N		D/C	114
82	WHWT	2	4		N	4.0	A	72
33	GSD	3b	4	digoxin	M		D/C	101
36	GSD	2	4		M		D	

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LA: left atrial size; N: within normal limits; + mildly; ++ moderately, +++ severely enlarged
J: Jackson-Henderson technique; S: standard ductal ligation; M: medical; N: none; C: coil embolisation
D: dead; A: alive; C: cardiac; NC: non-cardiac

visible in 32% of the cases while an aortic bulge, pulmonary artery bulge and auricular bulge were seen in 88%, 75%, and 50% of the cases respectively. There was no significant statistical correlation between the age of the animals at initial presentation and the occurrence of an aortic and/or, pulmonary artery and/or auricular bulge. An aortic bulge disappeared only in one animal. A pulmonary artery bulge disappeared in 3 animals (dogs 20, 80, 102) after ductal occlusion, all less than 4 years of age at the time of ductal occlusion. One animal (dog 73) acquired a pulmonary artery bulge, but was in left and right-sided heart failure at representation. After ductal closure, the auricular bulge disappeared in all but one animal (dog 73), the same one in biventricular congestive heart failure.

Overperfusion of the pulmonary vasculature was evident on 88% of the initial dorsoventral views. The radiographic signs of pulmonary overperfusion disappeared in all animals that had their ductus closed. Pulmonary oedema was only evident in 3 cases (19%) at initial presentation. These 3 animals were all between 24 and 36 months of age.

Doppler flow echocardiography (Table 4)

Colour Doppler Echocardiography revealed the presence of concurrent defects in 3/24 cases. These cases were excluded from the analysis of all the data. The changes in myocardial wall thickness' and left ventricular diameters as seen on M-Mode echocardiography are listed in Table 4. All animals had an increased left ventricular diameter in diastole and systole. The fractional shortening values (FS) (range 19-45%) are shown in Table 4.

The left atrium appeared enlarged in 52% of the cases, with concurrent mitral regurgitation in 64% of these animals. The ductus was clearly visualised in 18 of the 21 cases. The ductus was respectively measured 17 and 20 mm at the pulmonary artery side in two animals that were in congestive heart failure. There appeared no correlation between ductus size and age of onset of clinical signs.

Echocardiographic data at follow-up were available in 11 animals, 2 of which had no intervention. The follow-up time ranged from 16 to 108 months (mean 42, median 26). The left atrium was still enlarged in 3 of the 9 dogs (dogs 27, 73, 87) that had their ductus occluded. Two of these 3 dogs had no echocardiographic evidence of mitral regurgitation (but one had a marked residual shunt, dog 87), and the third dog (dog 73) had developed severe mitral valve endocardiosis. The mitral valve appeared markedly thickened in 6/11 animals, consistent with mitral valve endocardiosis. There was no correlation between the presence of mitral endocardiosis and age of these animals. The 2 non-occluded animals had developed moderate endocardiosis when reassessed at 6 years of age.

Systolic function, assessed by combination of FS, EF and PEP/ET ratio, showed that all parameters were normal in only 3/9 (33%) occluded animals. From the 2 animals with an unoccluded ductus the only systolic abnormality was increased PEP/ET ratio in one of them. When the animals with mitral regurgitation and/or residual shunting were excluded, some evidence of systolic dysfunction was still present in all. Of the 4 dogs with FS < 30% at initial presentation, subsequent systolic function described as all parameters normal, occurred in only one dog. The other 3 dogs had evidence of persistent systolic dysfunction. One of these had developed endocardiosis (dog 102).

There was no significant difference in left ventricular diastolic and systolic diameter pre-and post-occlusion, although there was an obvious trend towards decreased inner ventricular chamber size (Table 4). The left ventricular diameter in diastole returned within normal reference range in 2 animals (dogs 78, 80) but the left ventricular diameter in systole was enlarged in all occluded animals at follow-up. In animals where the shunt was not occluded, the left ventricular diameter in diastole and systole was[11] much above the normal limits, indicating severe volume overload.

There was no echocardiographic evidence of pulmonary hypertension in any of the animals. However dog 73, who was in left and right-sided heart failure, had a pulmonic insufficiency jet velocity of 2.1 m/s.

Persistent flow across the ductus was noticed in 5/9 (56%) animals. This was considered trivial in 3 animals, mild in 1, moderate in 1.

Treatment and complications (Table 5)

Fourteen animals had their ductus surgically ligated, 8 using the standard technique[21] and 6 via the Jackson-Henderson approach[22] (Table 5). One of them had recanalised after earlier ligation when 4.5 years old and had a second surgery 12 months later (dog 7). In 2 animals closure was achieved by trans-arterial coil embolisation 23, and a further 5 owners declined intervention for their animals. Of the 16 animals that had their ductus occluded, 4 animals (dogs 16, 27, 73, 102) were in heart failure at the time of intervention.

Complications occurred in 6/14 of the surgeries and in 1/2 of the interventional procedures (dog 87, haemolysis). The surgical complications included haemorrhage (total 3/14) (dogs 16, 26, 102), cardiac arrest (1/14) (dog 16), wound seroma formation (1/14) (dog 96), pronounced Branham's reflex 24 (1/14) (dog 75), laryngeal paralysis (1/14) (dog 16) and transient ventricular arrhythmias (dog 73) in 1 case.

All haemorrhaging animals were less or equal to 3 years of age. One dog died of intra-operative haemorrhage (dog 26) making the total short-term (1 month post-intervention) successful surgical survival rate 93%. Persistence of flow across the ductus was identifiable on Colour flow echocardiography in 44% of the animals scanned before discharge. Late closure of the ductus occurred in 1 dog (dog 27). There was echocardiographic evidence of late recanalisation (trivial) in 2 dogs (dogs 73, 78). Dog 7 which had two surgeries because of complete recanalisation was not included.

Survival (Table 5)

From those animals that were discharged after closure, 7 had died at the time of writing. Time of survival after surgery ranged from 10-112 months (median 36 months) and age of death ranged from 46 months to 168 months (median 96 months). From the 4 dogs that were in heart failure at initial presentation, 2 dogs had died and two were still alive. Those that had died, lived 10 months and 24 months after surgery and both were euthanased because of refractory cardiac failure (in one case due to severe mitral and

tricuspid valve endocardiosis). Two other dogs, that also were in heart failure initially, were still alive at 66 months and 108 months post-intervention. The first one is currently on treatment for left-sided heart failure secondary to mitral valve endocardiosis. The latter, although having signs of mild systolic dysfunction without the presence of mitral regurgitation or residual shunting, and having developed interstitial pulmonary fibrosis, was doing well clinically.

Three of the 5 non-treated animals had died. Two of these 3 dogs lived respectively 5 months and 60 months after diagnosis and died secondary to heart failure at the age of 101 and 114 months. The cause of death of the third dog remains unknown. The two alive non-treated dogs are currently 84 and 72 months old. Although they both have developed moderate mitral valve endocardiosis, and have echocardiographic evidence of severe volume overload, they are both doing very well clinically.

The 2 animals who had their ductus ligated at 108 months of age, both having severe cardiomegaly and clinical signs of collapse and anxiety episodes at initial presentation, remained completely without clinical signs 24 months and 30 months post-intervention.

Discussion

Older dogs with a persistent left to right shunting PDA are probably more common than generally believed. There was an incidence of 24% in our population which agrees to what previously has been reported[12, 25]. In this group of older dogs only one dog had right-to-left shunting PDA. Buchanan[26] stated that development of pulmonary hypertension and flow reversal later in life in dogs with left to right shunt is very uncommon. When older dogs are recognised with right to left PDA it is very likely that they have had unrecognised pulmonary hypertension since the neonatal period. The prevalence of right-to-left shunting PDA in this population (4%) is similar to previous reports[4, 27]. There was no higher prevalence of concurrent defects in this older population, compared with previous reports (10%) in younger groups[2, 28].

The time of diagnosis coincided in many cases with the first observation of clinical signs, suggesting that veterinary attention is often only sought when clinical signs are present and that even loud continuous murmurs are often overlooked if there are no clinical signs. However, other than an increased percentage of animals that suffer syncope, their clinical behaviour does not differ considerably from the well- recognised patterns of cardiac decompensation in younger dogs[6, 28]. The cause of the collapsing episodes remains uncertain but different explanations can be considered. It is difficult to exclude intermittent right-to-left shunting, however the absence of polycythaemia in any of these animals would make this aetiology unlikely. Poor cardiac output by coronary steal[29] or paroxysmal arrhythmias have been suggested in human medicine[30]. Only one of the collapsing dogs had ventricular premature complexes on its baseline ECG. Twenty-four hour ECG or event recording data was not available to determine if prolonged malignant arrhythmias could be accounted for the collapsing episodes in any of these animals. Sudden death, reported in younger human patients with persistence of the ductus and a history of syncope[31], was not observed in this older canine group.

Anxiety was seen in one of the animals. It has been associated with poor cardiac output and different type of arrhythmias in people[32], and also with reverse shunting[32], but the reason remains unknown in this dog.

In younger dogs, most continuous murmurs are loud[33]. In this older population 90% of the murmurs were grade 4 or above, similar to what is seen in younger dogs. Mitral valvular incompetence, attributed to mitral valvular dilation and stretch[12] and papillary muscle displacement[4] secondary to left ventricular volume overload, is a frequent finding in PDA[11]. This explains the presence of a concurrent systolic murmur in 33% of the cases at initial presentation. However mitral valvular endocardiosis was responsible for most mitral murmurs at follow-up.

Atrial fibrillation, known to be a late clinical finding and normally associated with a grave prognosis[11], was only seen in 2 animals. Both these animals died, one 2 years later (dog 73) and for the other dog the time and reason of death remains unknown. One animal (dog 3) developed atrial fibrillation one year post successful ligation of the ductus and the reason for this development is uncertain. In humans irreversible left ventricular dysfunction and deterioration have been postulated as a possible explanation[30].

There was radiographic evidence of cardiomegaly in the majority of animals after successful closure of the ductus. This indicates that the radiographic cardiac size changes are rarely reversible in older dogs and this was confirmed echocardiographically. Where severe radiographic cardiomegaly persisted (dog 7), it did not affect lifespan, although clinical signs of cough, probably attributed to left main stem bronchi compression by the cardiomegaly, persisted until death (at the age of 14).

In this group of adult dogs an aortic bulge was a common, but rarely reversible feature, as previously reported by Buchanan[34]. The animal (dog 73) that developed a pulmonary artery bulge after ductal closure had evidence of pulmonary hypertension on post-mortem, explaining the appearance of this radiographic sign. In this study, radiographic evidence of pulmonary oedema was more commonly seen in the younger adults (24-36 months).

It is not known why some animals with persistence of the ductus survive over 6 years of age or why the prognosis varies so widely among those who do. It has been natural to assume that small size of the ductus is the determining factor. However, in this study, this could not be confirmed as a ductus > 8 mm was not always associated with congestive heart failure or with poor survival. It has been postulated in human medicine[30] that the maintenance of normal pulmonary vascular resistance is probably an important factor in the long survival of the older patient with a persistent ductus. Mitral valve endocardiosis was the most common complicating factor in our study population and it appeared at younger age and in different breeds than are normally predisposed to this acquired cardiac condition[35]. It is possible that the turbulence and

regurgitation created by mitral valvular leakage are a predisposing factor for the development of degenerative mitral valve disease. Chesler and Gornick[36] previously documented mechanical stress as a predisposing factor for the development of mitral valve prolapse in humans. Mitral valve prolapse has been identified in dogs and it has been postulated that it might be an early sign of development of myxomatous valve disease[37]. Similarly, a pathological study in humans found a high incidence (18%) of myxomatous valves amongst patients with a ventricular septal defect[38], another aetiology of left ventricular volume overload, but careful comparison with other information in that report suggested that they were unrelated occurrences. Degenerative valve disease did not appear to be a feature in any of the adult dogs in Goodwin's study[12], however only echocardiography and no pathology data were available. Weirich[11] did detect in his pathological study of 6 dogs with PDA, the presence of mitral valve endocardiosis in a 10 month old Springer Spaniel.

There was obvious echocardiographic evidence of left ventricular systolic dysfunction in many animals before and after closure of their ductus. The reversibility of systolic dysfunction appears uncommon in this older population, however the clinical significance remains questionable because many animals have a very good quality life without any signs of exercise intolerance.

Reluctance to operate on older animals with PDA has been based on the higher complication rate (fatal haemorrhage) due to increased ductus friability in the older dog[39]. However, in the hands of experienced surgeons, as here, the complication rate appeared not to be higher than in the younger group[40]. In human medicine the risk of endocarditis is one of the main indications to close the ductus, independent of its size[30]. Endocarditis was not recognised in our study population and this contrasts with previous case reports in adult dogs[12].

The majority of animals with clinical signs prior to closure now had returned to a symptom free good quality life. In case of owners' reluctancy for thoracotomy, interventional coil embolisation remains a less invasive alternative[23].

Buchanan's clinical classification[5] and Pouchelon's echocardiographic classification[9] do not appear useful with regards to decision making for treatment in the older dog with PDA. There appears to be no obvious course in survival of this older age group, but heart failure at initial presentation does not always predict a poor prognosis. Mitral valve endocardiosis is the main complicating factor and compromises outcome. Since degeneration of the mitral valve might be accelerated by turbulence created by mitral insufficiency due to stretch and dilation of the mitral annulus, measurements to reduce this pre-existing period of valve damage, should be shortened by early ligation of the ductus.

Conclusion

Older animals with PDA follow an individual course, independent of pre-existing heart failure. Late closure does not appear to carry a higher risk in the hands of experienced surgeons and it relieves clinical signs. In cases of reluctancy for invasive thoracotomy, interventional coil embolisation appears a good alternative.

Since in veterinary medicine quality of life (absence or lessening of clinical signs) without a negative impact on mortality remains a primary end-point, closure of a patent ductus arteriosus, independent of its size and age of presentation, remains indicated. Early closure is advisable to avoid mechanical stress on the mitral valve, that might predispose to development of mitral valve endocardiosis.

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