

The variety of phenotypes behind 'double outlet right ventricle': clinical and imaging presentations in four dogs and a cat

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1 The variety of phenotypes behind "double outlet right ventricle": clinical and 2 imaging presentations in four dogs and a cat

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- 17

18 Short title

- 19 Double outlet right ventricle in small animals
- 20
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- 23
- 24

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32 Abstract

This report describes five cases of double outlet right ventricle (DORV) in four dogs (3 to 18 months old; two males and two females) and a domestic shorthair cat (6month-old, female) presented with various clinical signs including tachypnea (n=5), exercise intolerance (n=5), mucous cyanosis (n=3), delayed growth (n=2) and/or lethargy (n=2). The represented canine breeds were Poodle, Yorkshire Terrier, Samoyed, and Shetland Sheepdog.

For all animals, echocardiography revealed marked aortic dextroposition with both 39 arterial trunks totally arising from the right ventricle, associated with a ventricular 40 41 septal defect (VSD) and various other congenital abnormalities, including subvalvular aortic stenosis (n=2), minor aortic insufficiency (n=5), subvalvular pulmonic stenosis 42 with pulmonary trunk hypoplasia (n=1), patent ductus arteriosus (n=1), minor mitral 43 44 and/or tricuspid dysplasia (n=3). Subsequent cardiac remodeling was characterized by marked right ventricular hypertrophy for all patients, associated with right 45 46 ventricular and right atrial dilation for most of them (4/5).

47 Two dogs died soon after the initial DORV diagnosis (i.e., after 24 hours and two months). A surgical correction attempted for another dog confirmed the presence of a 48 DORV associated with patent ductus arteriosus, but the animal died during the 49 procedure from sudden cardiac arrest. The fourth dog underwent a contrast-50 enhanced retrospective electrocardiogram-gated multidetector computed tomography 51 under general anesthesia, which confirmed the conotruncal 52 angiography 53 malformation. Despite episodes of exercise intolerance, this dog is still alive, at the age of 48 months, as is the cat at the age of 16 months. 54

55

56 Key words: Canine; Congenital; Doppler; Feline; Heart

57 Abbreviations

bpm	beats per minute
СТ	computed tomography
DORV	double outlet right ventricle
ECG	electrocardiographic
LV	left ventricle
LVPWs	left ventricular posterior wall thickness at end-systole
PCV	packed cell volume
RA	right atrium
RBC	red blood cells
RV	right ventricle
RVFWs	right ventricular free wall at end-systole
RVDd	right ventricular dimension at end-diastole
TGA	transposition of the great arteries
VSD	ventricular septal defect

59 Case 1

A 17-month-old male Poodle dog, weighing 5.0 kg, was referred to the Alfort Cardiology Unit (UCA) at the Veterinary Hospital (CHUVA) of the Alfort National Veterinary School (ENVA) for exercise intolerance and intermittent tachypnea, which had both worsened over the past three months. The physical examination showed congested mucous membranes and a grade I/VI right basilar systolic heart murmur.

Two- and three-dimensional echocardiographic examination^d coupled with color- and 65 continuous-wave Doppler modes (Figs. 1A and 1B), revealed a large doubly 66 committed ventricular septal defect (VSD; maximal diameter: 16 mm) characterized 67 68 by an intermittent bilateral low-velocity shunt (left-to-right in systole and right-to-left in early-diastole, with a maximal velocity of 1.1 and 0.9 m/s respectively, and absent at 69 end-diastole). Severe aorta dextroposition was also observed, with the aorta entirely 70 71 originating from the right ventricle. Minor aortic insufficiency was also noted. Based on these echocardiographic findings, the dog was diagnosed with double outlet right 72 73 ventricle (DORV). The peak systolic transpulmonary flow velocity assessed using 74 continuous-wave Doppler mode was 2.0 m/s. End-systolic right ventricular free wall and left ventricular posterior wall thicknesses (RVFWs and LVPWs, respectively) 75 were measured using the two-dimensional guided M-mode from the right parasternal 76 77 ventricular short-axis view, and confirmed marked right ventricular (RV) wall thickening (RVFWs:LVPWs ratio = 130%, reference interval: 24-50%, [1]), associated 78 with RV dilation (RV end-diastolic diameter (RVDd) to left ventricular end-diastolic 79 diameter ratio of 79%, reference interval: 7-33% [1]), and a paradoxical septal 80 motion. The right atrium (RA) was also markedly dilated, with a right to left atrium 81 ratio (measured at end-diastole at the level of the atrioventricular valves using the 82 right parasternal 4-chamber view) of 1.4 (reference range: 0.6-1.0 [2]). The caudal 83

vena cava was unremarkable. Minor mitral and tricuspid insufficiencies, associated
with mild leaflet remodeling, were also observed. Concomitant electrocardiographic
(ECG) tracing showed a normal sinus rhythm with right axis deviation (heart rate
between 80 and 100 beats per minute (bpm)).

To better describe the cardiac and vessels malformations, a contrast-enhanced 88 multidetector computed tomography (CT) angiography with retrospective ECG-gated 89 reconstructions^{e,f} was subsequently performed under general anesthesia (Figs. 1C 90 and 1D; Videos 1 to 3). This confirmed the association of severe aortic dextroposition 91 and a large doubly committed VSD, with secondary marked RA and RV dilation 92 93 associated with RV hypertrophy. A subpulmonary infundibulum was observed without any subaortic infundibulum. An aberrant coronary artery emerging from the 94 pulmonary trunk was also detected (Fig. 1D). Hematology revealed mild 95 96 polycythemia (red blood cells (RBC) = 8.9×10^{12} /L, reference range: $5.5-8.5 \times 10^{12}$ /L; hemoglobin = 21.3 g/dL, reference range: 10.0-18.0 g/L; packed cell volume (PCV) = 97 98 55.2%, reference range: 35.0-55.0%). Biochemistry was unremarkable.

99 An oral medical treatment (spironolactone^g 0.5 mg/kg/day and omega-3 fatty acids^h) 100 was prescribed. At the time of writing, this dog is still alive at the age of 48 months 101 and appears in good physical condition, despite some episodes of exercise 102 intolerance.

103

104 Case 2

105 A 1.5-year-old female Yorkshire Terrier dog, weighing 3.9 kg, was referred for 106 chronic seizures, which had intensified over the previous week. Episodes of 107 weakness, exercise intolerance and dyspnea had been reported since birth. An oral 108 medical treatment that included benazeprilⁱ 0.3 mg/kg/day and spironolactone^g 1.3

mg/kg/day with furosemide^j (1.3 mg/kg) in the case of dyspnea, was prescribed at 2
months of age by the referral veterinarian,

111 Upon admission, the animal was lethargic, cyanotic and tachypneic. Cardiac 112 auscultation revealed a grade V/VI right mediothoracic systolic heart murmur and a 113 grade IV/VI left basilar systolic heart murmur.

Two-dimensional and Doppler echocardiography^d led to diagnosis of a DORV 114 characterized by an aorta emerging entirely from the RV, along with a small (3.7 mm) 115 116 non-committed muscular VSD, causing a left-to-right shunt (peak velocity of 5.6 m/s; Figs. 2A and 2B). A subpulmonary infundibulum was clearly observed without any 117 118 subaortic infundibulum. The pulmonic flow assessed using color-flow Doppler mode was laminar. Conversely, the aortic flow was turbulent and showed an increased 119 peak velocity (5.52 m/s; peak systolic pressure gradient = 126 mmHg) due to the 120 121 presence of subvalvular aortic stenosis, with concomitant mild aortic insufficiency. 122 Right ventricular hypertrophy (RVFWs:LVPWs ratio = 81% [1]), along with left 123 ventricular (LV) hypertrophy and a secondary decrease of LV internal diameters, 124 were also noted (normalized LV end-diastolic and end-systolic internal diameters of 1.0 and 0.7 [3]). Dilation of the pulmonary trunk, associated with a "notched" Doppler 125 flow pattern of the pulmonary artery flow and decreased peak systolic 126 127 transpulmonary flow velocity (0.49 m/s [4,5]), suggestive of pulmonary arterial 128 hypertension, were also observed. Concomitant ECG tracing displayed a sinus rhythm without any significant abnormality (heart rate between 90 and 110 bpm). 129

130 Complete blood count revealed severe polycythemia (RBC = 12.09×10^{12} /L and PCV 131 = 84%). The dog died suddenly 24 hours after diagnosis.

132 The postmortem examination (Fig. 2C) confirmed the presence of all the 133 echocardiographic abnormalities (DORV, VSD and subvalvular aortic stenosis).

Histopathological examination of the lungs (Fig. 2D) revealed severe remodeling of
the pulmonary arteries, with plexiform arterial networks, hypertrophy of the media,
intimal fibrosis and thrombosis, all of which were consistent with chronic pulmonary
arterial hypertension.

138

139 **Case 3**

A 4-month-old male Samoyed puppy (8.1 kg) was referred for exercise intolerance,
lethargy, shortness of breath, mucous cyanosis, and occasional coughing, that had
worsened over the last two weeks.

On presentation, the main physical examination findings included tachypnea with mild mucous cyanosis and delayed growth. Cardiac auscultation revealed a grade II/VI right mediothoracic systolic murmur and a grade II/VI left basilar diastolic murmur.

147 Two- and three-dimensional echocardiographic examination, coupled with color-flow Doppler mode (Fig. 3A),^d revealed a DORV, with complete aorta dextroposition 148 149 associated with mild aortic insufficiency. A subpulmonary infundibulum was clearly 150 observed without any subaortic infundibulum. The peak systolic transpulmonary flow velocity assessed using continuous-wave Doppler mode was 1.4 m/s. A large 151 152 perimembranous subaortic VSD (up to 1.4 cm) with bilateral low-velocity shunt 153 (mostly left-to-right: 1.69 m/s, gradient = 11 mmHg) was also observed. The RV was 154 severely hypertrophied and dilated (RVFWs:LVPWs ratio = 206%; RVDd to LV enddiastolic diameter ratio = 103% [1]), leading to a paradoxical septal motion and 155 remarkably decreased LV diameters (normalized LV end-diastolic and end-systolic 156 157 internal diameters of 0.9 and 0.6, respectively [3]). The RA was also markedly dilated (right to left atrium ratio = 1.8 [2]), with secondary dilation of the caudal vena cava. 158

The pulmonary trunk and RV outflow tract were also dilated. A high velocity 159 160 pulmonary insufficiency was observed (4.41 m/s at end-diastole, gradient = 78 mmHg), suggesting the presence of severe diastolic pulmonary arterial hypertension. 161 Additionally, using color-flow Doppler mode from the right parasternal transaortic 162 163 short-axis view, an ascending non-aliased low velocity flow was visualized within the pulmonary trunk, consistent with a patent ductus arteriosus providing a left-to-right 164 low velocity shunt (Fig. 3B). Lastly, a mild mitral insufficiency accounting for minor 165 166 mitral dysplasia, was found. The concurrent electrocardiogram showed a sinus rhythm with right axis deviation (heart rate between 130 and 175 bpm). The PCV was 167 54%. 168

Spironolactone^g (1.2 mg/kg/day per os) was initially prescribed. A surgical correction under cardiopulmonary bypass was planned with the main objective to suture a patch in order to isolate the connection between the aorta to the LV while leaving the pulmonary trunk connected to the RV. Unfortunately, the patient died when starting cardiopulmonary bypass owing to ventricular fibrillation. The view of the open heart confirmed a DORV associated with a subaortic VSD and a patent ductus arteriosus. A histopathological examination was declined by the owners.

176

177 Case 4

A 3-month-old female Shetland Sheepdog (5.5 kg) was referred for suspected congenital heart disease. The owners reported exercise intolerance and mucous cyanosis. Physical examination findings included delayed growth, mucous cyanosis, tachypnea, and grade IV/VI left basilar systolic heart murmur.

Two-dimensional and Doppler echocardiography^d was subsequently performed and
revealed a DORV, with the aorta emerging completely from the RV without any well-

defined subaortic infundibulum. A severe subvalvular aortic stenosis was also 184 observed (trans-stenotic peak pressure gradient = 127 mmHg) with mild aortic 185 insufficiency. A small-diameter (3.1 mm) subaortic VSD, leading to a bidirectional 186 interventricular shunt, was also noted. Assessment of the pulmonary outflow tract 187 revealed pulmonary trunk hypoplasia, associated with severe infundibular pulmonary 188 stenosis (peak systolic transpulmonary flow velocity assessed using continuous-189 wave Doppler mode = 6.4 m/s; trans-stenotic peak pressure gradient = 165 mmHg). 190 191 Consequently, the RV was severely hypertrophied and dilated (RVFWs:LVPWs ratio = 169%; RVDd to LV end-diastolic diameter ratio = 89% [1]), as was the RA (right to 192 193 left atrium ratio = 1.4 [2]). Hypertrophy of the interventricular septum was also observed, as well as decreased LV internal diameters (normalized LV end-diastolic 194 195 and end-systolic internal diameters of 0.8 and 0.3, respectively [3]). Minor mitral 196 insufficiency resulting from mild leaflet remodeling was also found. The concomitant 197 electrocardiogram showed a respiratory sinus arrhythmia (heart rate between 90 and 198 120 bpm). Hematology confirmed polycythemia (RBC = $9.82 \times 10^{12}/L$; PCV = 74%). 199 No medication was prescribed according to the owner's request. The animal died 200 suddenly at home two months after the initial diagnosis, and no necropsy could be performed. 201

202

203 Case 5

A 6-month-old 2.5 kg domestic shorthair female cat was referred for exercise intolerance and episodic dyspnea. The cat exhibited tachypnea after minimal manual restraint, and auscultation revealed a grade IV/VI right mediothoracic systolic heart murmur.

Transthoracic echocardiography was subsequently performed,^d and confirmed that 208 209 the aorta emerged entirely from the RV and was even located on the right side of the 210 pulmonary trunk (Figs. 4A and B). A short bilateral infundibulum emerging from the RV was seen. A large muscular VSD (Fig. 4C) mainly committed with the pulmonary 211 212 trunk was also found, so that the LV ejection flow passed through the VSD towards both arteries, but mostly into the pulmonary trunk. A mild aortic insufficiency was 213 detected. The peak systolic transpulmonary flow velocity assessed using continuous-214 215 was Doppler mode was 1.3 m/s. A pulmonary insufficiency of high velocity (3.14 m/s) was also present suggesting diastolic pulmonary arterial hypertension. Accordingly, 216 217 the RV was both hypertrophied and dilated, with a mild RA dilation. The interventricular flow was mostly left-to-right and was characterized by a low peak 218 219 velocity (1.2 m/s), indicating a very low-pressure gradient (6 mmHg) owing to high 220 RV systolic pressure, and was rarely right-to-left at end systole and early diastole. 221 Concomitant ECG tracing showed a normal sinus rhythm (150 bpm) along with right 222 axis deviation. Hematology confirmed polycythemia (RBC = 9.82×10^{12} /L; PCV = 223 74%). A cardiac CT scan was proposed but the owner preferred a more conservative approach and consented only to medical palliation. This consisted of spironolactone^g 224 (1 mg/kg/day). The cat is still alive at the age of 16 months and the owners report an 225 226 improvement of tachypnea.

227

228

229 Discussion

Double outlet right ventricle is a congenital heart disease encompassing a wide spectrum of anatomic abnormalities and pathophysiologic disturbances, explaining many controversies in human cardiology regarding its definition, nomenclature, and

classification as well as optimal timing for surgical repair [6]. According to the 233 234 consensus definition issued by the Congenital Heart Surgery Nomenclature and Database Project, DORV is a rare conotruncal anomaly corresponding to a particular 235 ventriculo-arterial connection, in which the two arterial trunks emerge entirely or 236 predominantly from the RV [6]. In most cases, viability is provided by the presence of 237 a VSD associated with various structural connections to both arteries and allowing 238 blood to flow from the LV to the RV, and then into the pulmonary and systemic 239 240 circulations. In humans, DORV only accounts for approximately 1 to 3% of congenital cardiac defects [7]. Veterinary retrospective studies suggest that DORV is also a rare 241 congenital malformation in both cats and dogs, accounting for 0.5% to 1% of 242 congenital cardiac defects in cats [8,9], with no DORV cases in retrospective reviews 243 performed on 105 to 976 dogs with congenital heart diseases [8,10,11]. A few 244 245 isolated case reports, relying essentially on postmortem examinations, have 246 documented this condition in dogs and cats [12-15], horses [16,17], alpacas [18] and 247 calves [19,20]. To the best of our knowledge, the present report is the first case 248 series with an antemortem DORV diagnosis in small animals, i.e., four dogs and one cat. In all five animals, the echocardiographic diagnosis of DORV was based on the 249 presence of a VSD associated with complete commitment of the aorta to the RV, 250 251 which could be confirmed by CT scan (Case 1) and by necropsy in two cases (Cases 2 and 3). The diagnosis of DORV in Cases 4 and 5 was solely based on 252 echocardiography, which represents a limitation of the present report. 253

The first DORV description was done in 1898, but the term "DORV" was not introduced until 1957, as the defect was initially considered as a particular form of transposition of the great arteries (TGA) [21]. The double outlet left ventricle, another abnormal type of ventriculoarterial connection in which both great arteries arise

entirely or predominantly from the LV, was then described several years later [22]. In 258 259 humans, DORV is a complex cardiac malformation characterized by great heterogeneity among affected patients, depending on the VSD characteristics, the 260 spatial relationship between the VSD and the arterial trunks and between the arterial 261 trunks themselves, and the presence or absence of pulmonic or aortic stenosis, and 262 of other concomitant malformations [6.7.23]. The VSD characteristics include its 263 264 restrictive or unrestrictive features and also its location, with subaortic VSD being the 265 most common type in human patients with DORV, accounting for approximately 50% of surgical patients [6]. Thus, as illustrated in the present case series, DORV 266 267 encompasses a wide spectrum of anatomic defects, with a great variability of clinical presentations and pathophysiologic features, including extreme forms mimicking 268 269 either tetralogy of Fallot or TGA in the case of respectively associated pulmonic 270 stenosis and subpulmonary VSD [6,23]. Various classifications of DORV taking into 271 account these anatomic variations have been proposed in an attempt to standardize 272 surgical approaches in human patients [6,23,24]. One of the most commonly used 273 DORV classification systems identifies four DORV types, i.e., the VSD type (including DORV with subaortic or doubly-committed VSD and without pulmonic stenosis), the 274 tetralogy of Fallot type (including DORV with subaortic or doubly-committed VSD and 275 276 pulmonic stenosis), the TGA type (including DORV with subpulmonary VSD) and the 277 remote VSD type characterized by a non-committed VSD (i.e., a VSD distant from the arterial trunks, observed in the muscular interventricular septum) [6]. According to 278 279 this classification, two DORV types were diagnosed in the present report, and 280 confirmed by either necropsy or CT scan, i.e., two VSD types for Cases 1 and 3 281 (characterized by a doubly-committed or subaortic VSD in the absence of pulmonic stenosis) and one remote VSD type for Case 2. Case 4 can be classified as a 282

tetralogy of Fallot type DORV, owing to the concomitant presence of severe infundibular pulmonary stenosis, and Case 5 as a TGA type owing to the echocardiographic visualization of a large muscular subpulmonary VSD. However, since the latter two descriptions were limited to echocardiographic examinations, the exact location of the VSD, especially for Case 5, could not be confirmed with certainty.

Beside its above-mentioned extreme anatomic variability, DORV in human patients is 289 290 also characterized by its common association with multiple cardiac malformations [6,25]. In this context, CT scan has been shown to be an excellent diagnostic tool for 291 detecting DORV-associated malformations, and a combination of transthoracic 292 echocardiography with CT scan allows excellent diagnostic performance (100 % 293 294 sensitivity for detecting intracardiac anomalies versus 91% for echocardiography 295 alone) [25]. Only one dog in the present case series was able to benefit from these 296 combined imaging techniques (Case 1), as the other dogs were presented before 297 acquisition of the CT-scan technique in our facility, while the cat's owners declined 298 the investigation. Nevertheless, various congenital abnormalities were found associated with DORV in all the animals, including subvalvular aortic stenosis (n=2), 299 aortic insufficiency (n=5), subvalvular pulmonic stenosis with pulmonary trunk 300 301 hypoplasia (n=1), patent ductus arteriosus (n=1), and minor atrioventricular dysplasia (n=3). Understandably, the variety of both the anatomic DORV phenotypes and 302 303 associated malformations explains the wide range of clinical presentations, as 304 illustrated in the present report.

305

306 In conclusion, this short case series illustrates the wide spectrum of congenital 307 anatomic anomalies, associated defects, and clinical presentations embodied by

308 DORV in small animals. Echocardiography remains adequate to reach a diagnosis.

However, owing to the complexity and heterogeneity of DORV, a cardiac CT scan

310 appears to be a wise option before attempting any surgical approach.

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312

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388 Footnotes

391

- ^d Vivid 7, Vivid E9; General Electric Medical System, Waukesha, WI, USA.
- ^e Brilliance 64-slice CT scanner, Philips Medical Systems, Cleveland, OH, USA.
- ^f 3mensio, PIE Medical Imaging, Maastricht, The Netherlands.
- ⁹ Prilactone, CEVA, Libourne, France.
- ³⁹⁵ ^h Agepi Oméga 3, MP Labo, Grasse, France.
- ⁱNelio, CEVA, Libourne, France.
- ^j Dimazon, Intervet, Beaucouze, France.

398 Figure legends

Figure 1: Representative color-flow Doppler (A, B) and three-dimensional retrospective ECG-gated (phase 30%) computed tomography (CT) reconstruction of the heart and great vessels (C and D) from a 17-month-old Poodle dog with double outlet right ventricle (Case 1).

- A and B Color-flow Doppler images obtained from the right parasternal 5-chamber
 view confirming a left-to-right shunt through the ventricular septal defect during the
 early systolic phase (A) and the presence of right ventricle-aortic flow during the
 whole systolic phase (B)
- 407 **C** This CT image confirms a large ventricular septal defect (VSD) with the aorta
 408 exiting from the hypertrophied right ventricle.
- 409 **D** Note on this CT image the presence of an aberrant coronary artery exiting from
 410 the pulmonary trunk.
- 411 Ao: aorta; LA: left atrium; LV: left ventricle; RV: right ventricle.
- 412

413 Figure 2: Images from a 1.5-year-old Yorkshire Terrier dog diagnosed with 414 double outlet right ventricle (Case 2).

A and B - Two-dimensional (A) and color-flow-Doppler mode (B) images respectively obtained from the right parasternal transaortic short-axis view and a right parasternal long-axis view, showing that both arteries arise from the right ventricle. Other abnormalities (B) include a turbulent left-to-right shunt through a small-sized defect within the muscular part of the interventricular septum and a subaortic stenosis (white arrows). The connection between the right ventricle and the aorta is show by an asterisk in both figures.

422 **C** - Necropsy examination (after oblique incision of the right ventricle) confirming that

the aorta and the pulmonary trunk both emerge from the right ventricle. Note also thesmall muscular non-committed ventricular septal defect (arrow).

D - Histopathological examination showing a major remodeling of the pulmonary
arteries with marked obliterative muscular hypertrophy (arrow) and intimal thickening.
These plexiform lesions are consistent with pulmonary arteriopathy due to severe
pulmonary arterial hypertension. Hematoxylin, eosin and saffron stain (Anatomical
Pathology department, BioPôle Alfort, Ecole Nationale Vétérinaire d'Alfort)

Ao: aorta; LA: left atrium; LVPW: left ventricular posterior wall; PT: pulmonary trunk;
RA: right atrium; RV: right ventricle; RVW: right ventricular wall; VSD: ventricular
septal defect.

433

Figure 3: Two-dimensional (A) and color-flow-Doppler mode (B) images recorded in a 4-month-old male Samoyed dog diagnosed with double outlet right ventricle (Case 3), respectively from the right parasternal 5-chamber view and the right parasternal transaortic short-axis view. Both images confirm that the aorta arises from the right ventricle. Figure 3A additionally shows a large ventricular septal defect (arrow) and Figure 3B a non-aliased ascending flow within the pulmonary trunk (arrow) owing to the presence of a patent ductus arteriosus.

441 Ao: aorta; LA: left atrium; LV: left ventricle; PT: pulmonary trunk; RV: right ventricle.

442

Figure 4: Two-dimensional (A) and color-flow Doppler mode (B and C) systolic images recorded in a 6-month-old female cat diagnosed with double outlet right ventricle (Case 5), obtained from a left cranial parasternal short-axis view (A) and two right parasternal long-axis views (B and C). Note the abnormal right lateral position of the aorta (A and B) and the systolic left-to-right shunt through a

large defect within the interventricular septum (C). The larger diameter of the
pulmonary trunk, as compared with that of the aorta, may be due to the increased
flow coming from the ventricular septal defect.

451 Ao: aorta; LA: left atrium; LV: left ventricle; PT: pulmonary trunk; RA: right atrium; RV:

452 right ventricle; RVW: right ventricular wall.









Table 1: Video captions

Video number	Description
Video 1	Contrast-enhanced retrospective ECG-gated multidetector computed
	tomography scan with two- and three-dimensional orthogonal views
	showing the large ventricular septal defect and the aorta emerging
	from the right ventricle. The aberrant coronary artery exiting from the
	pulmonary trunk is also seen in the two movies on the left.
Video 2	Contrast-enhanced retrospective ECG-gated multidetector computed
	tomography scan with two- and three-dimensional orthogonal views
	showing the aorta and the pulmonary trunk both emerging from the
	right ventricle, with an aberrant coronary artery from the pulmonary
	trunk. Note the absence of left ventricular outflow track.
Video 3	Contrast-enhanced retrospective ECG-gated multidetector computed
	tomography scan: volume rendering video.