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Common arterial trunk in a cat: a high-resolution morphological analysis with micro-computed tomography --Manuscript Draft--

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Abstract:	<p>A 6-month-old female cat presented with respiratory distress. Physical examination showed a grade 5/6 holosystolic murmur with prominent precordial impulse over the left cranial chest wall. Echocardiography revealed bilateral hypertrophy of the ventricular walls, a dilated ascending aorta overriding the interventricular septum, a membranous ventricular septal defect and no obvious pulmonary trunk or pulmonary artery branches. Turbulent blood flow was detected around the ventricular septal defect and ascending aorta. Follow-up assessment, 12 months later, revealed marked and progressive biatrial dilation and biventricular hypertrophy. Four months after that, the cat died of severe congestive heart failure. To make a definitive post-mortem diagnosis, we performed contrast enhanced micro-computed tomography (CT) on the ex vivo heart with micron-scale spatial resolution imaging and three-dimensional reconstruction. Micro-CT analysis confirmed a common arterial trunk that bifurcated into the left pulmonary artery and aorta 5 mm distally from the truncal valve. The pulmonary trunk was absent. Slightly distal to the first branching, the common arterial trunk further branched into the right pulmonary artery and ascending aorta, indicating the aortic dominant form. Although CT angiography would be a preferred imaging modality for living animals, micro-CT is a valuable tool for the ex vivo diagnosis of complex cardiac anomaly, such as presented in this cat.</p>
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**Title: Common arterial trunk in a cat: a high-resolution morphological analysis
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Abstract

A 6-month-old female cat presented with respiratory distress. Physical examination showed a grade 5/6 holosystolic murmur with prominent precordial impulse over the left cranial chest wall. Echocardiography revealed bilateral hypertrophy of the ventricular walls, a dilated ascending aorta overriding the interventricular septum, a membranous ventricular septal defect and no obvious pulmonary trunk or pulmonary artery branches. Turbulent blood flow was detected around the ventricular septal defect and ascending aorta. Follow-up assessment, 12 months later, revealed marked and progressive biatrial dilation and biventricular hypertrophy. Four months after that, the cat died of severe congestive heart failure. To make a definitive post-mortem diagnosis, we performed contrast enhanced micro-computed tomography (CT) on the *ex vivo* heart with micron-scale spatial resolution imaging and three-dimensional reconstruction. Micro-CT analysis confirmed a common arterial trunk that bifurcated into the left pulmonary artery and aorta 5 mm distally from the truncal valve. The pulmonary trunk was absent. Slightly distal to the first branching, the common arterial trunk further branched into the right pulmonary artery and ascending aorta, indicating the aortic dominant form. Although CT angiography would be a preferred imaging modality for living animals, micro-CT is a valuable tool for the *ex vivo* diagnosis of complex cardiac anomaly, such as presented in this cat.

Key Words

Congenital heart disease; diagnostic imaging; echocardiography

Abbreviation table

<i>Abbreviations</i>	<i>Definition</i>
3D	three-dimensional
Ao	aortic arch
CAT	common arterial trunk
CT	computed tomography
LA	left atrium
LPA	left pulmonary artery
LV	left ventricle
RA	right atrium
RPA	right pulmonary artery
RV	right ventricle
VS	interventricular septum
VSD	ventricular septal defect

1 51 A 6-month-old, 2.6 kg, intact female domestic short-haired cat was referred for further
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4 52 evaluation of a cardiac murmur detected incidentally prior to ovariohysterectomy. The
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7 53 cat exhibited normal activity and appetite at home according to the owner, but
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10 54 displayed heavy breathing with mild cyanosis at the hospital. A strong precordial
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13 55 impulse was palpable over the left cranial chest wall. Auscultation detected a regular
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16 56 heart rhythm with 140-150 beats per minute, a 5/6 grade holosystolic murmur and clear
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19 57 lung sounds. Complete blood count and serum biochemical profile were within a
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27 59 Transthoracic echocardiography was performed with gentle restraint in the
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30 60 standing position to alleviate stress. The two-dimensional right parasternal short-axis
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33 61 ventricular view revealed bilateral ventricular hypertrophy, especially in the right
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36 62 ventricular wall with a flattening of the interventricular septum. The right parasternal
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40 63 long-axis four-chamber view revealed significant right atrial dilation (17 mm in
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43 64 diameter) and a 5 mm membranous ventricular septal defect (VSD) (Fig. 1A and B,
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46 65 see also Videos 1 and 2 (available in Supplemental Material on-line)) [1]. The left atrium
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49 66 was also dilated (12 mm in diameter) [2]. Moreover, the aorta was significantly dilated
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52 67 and overriding the VSD. Colour-flow Doppler revealed turbulent flow in the area
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55 68 surrounding the VSD in diastole and the ascending aorta in systole, although the
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58 69 direction could not be specified due to the brief nature of the examination (Fig. 1A and

B). The velocity of the regurgitant flow at the aortic valve was 3.0 m/s, estimating the pressure gradient to be 36 mm Hg using the simplified Bernoulli equation [3]. Normal morphology of the right ventricular outflow tract including the pulmonary valve and the pulmonary trunk could not be identified. Although the left pulmonary artery was detected, there was no obvious communication between the pulmonary artery and aorta. Based on the echocardiographic findings, a putative diagnosis of tetralogy of Fallot was made at this point and enalapril hydrochloride (0.25 mg/kg, PO, q 12 hr) was initiated.

A follow-up assessment was performed 12 months after the initial evaluation. The cat's body weight had increased to 4.0 kg. A grade 5/6 holosystolic murmur and normal lung sounds were auscultated. The precordial impulse was more obvious than at the initial presentation. Thoracic radiography revealed marked cardiomegaly, a shifted cardiac axis with a rounded apex and distended pulmonary arteries (Fig. 2A and B). Incomplete expansion of the caudal lung lobes and increased radiopacity in the left cranial lung region were also observed (Fig. 2B), suggesting the existence of pleural effusion. An abbreviated echocardiographic examination was performed due to severe respiratory distress. The imaging revealed progression of right ventricular hypertrophy and right atrial dilatation (23 mm in diameter). The left atrial diameter had also increased to 16 mm (Fig. 2C and D). Turbulent blood flow through the VSD was

1 89 detected only in systole (Fig. 2E and F). The pulmonary valve and artery were not
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4 90 visualised. With enalapril as only medication, the cat remained stable for 4 months until
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7 91 it succumbed to respiratory failure.
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11 92 The post-mortem examination was performed on the day the cat died (16 months
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14 93 after the initial evaluation). The body condition score was 4 out of 5. Approximately 50
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17 94 mL of a modified transudate pleural effusion was present. The lungs and liver were
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21 95 swollen, and they displayed irregularly reddish colour indicating moderate to severe
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24 96 congestion. The heart was markedly enlarged, and the weight of the heart with some
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27 97 great vessel tissue (Fig. 3A and B) was 55 g. The pericardium was slightly thickened
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30 98 with a scant amount of pericardial effusion. The ventricles were concentrically
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33 99 hypertrophied. The right and left auricles were markedly dilated and thickened (Fig.
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36 100 3A). The pulmonary trunk and aortic arch were not anatomically separated from each
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40 101 other (Fig. 3B). Our findings indicated the presence of a common arterial trunk. The
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43 102 detailed morphology and communication between the ventricles and the great vessels
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46 103 were still not evident.
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51 104 To preserve the three-dimensional (3D) morphology of the heart with complex
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54 105 anomaly, we performed micro-computed tomography (CT) analysis. The heart was
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57 106 removed from the cat's chest and fixed in 10% phosphate buffered formalin for further
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1 107 examination. The heart was stained in iodine-potassium iodide contrast agent^h for 1
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4 108 week and washed in water for 1 day to remove excess stain prior to micro-CT scanningⁱ
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7 109 [4]. The heart was scanned at 161 kV, 136 μ A, with a 200 μ m copper filter, and a voxel
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10 110 resolution of 37 μ m was achieved. An imaging software^j was used to visualise the
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13 111 cardiac morphology and segment structures. A micro-CT image revealed the left and
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16 112 right pulmonary arteries separately branched directly from the common arterial trunk
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20 113 (Fig. 3C). As observed with echocardiography, there was a prominent VSD and
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23 114 absence of the normal communication from the right ventricle to the pulmonary trunk
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26 115 (Fig. 3D). The common arterial trunk was the only outflow tract from the right ventricle
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29 116 to the pulmonary arteries. The anatomical location of the mitral and tricuspid valves
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33 117 was normal (Fig. 3E).

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36 118 Further segmentation and visualisation of the micro-CT dataset revealed in detail
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39 119 the abnormal connection between the great vessels. Five mm distal from the truncal
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42 120 valve, the common arterial trunk branched into its distal portion and the left pulmonary
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45 121 artery. A few mm distal to the first bifurcation, the trunk further branched into the right
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48 122 pulmonary artery and the ascending aorta. Notably, this branching portion of the
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51 123 common arterial trunk was markedly dilated and is the area where turbulent blood flow
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55 124 was detected on echocardiography. Moreover, three distinctive arteries seem to
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58 125 directly branch from the same region of the aortic arch. These united vessels were
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probably composed of the right subclavian and left and right common carotid arteries
with a lack of/or remarkably shortened brachiocephalic artery. The origin of the left
subclavian artery was isolated from this abnormal branching by a few mm. Figure 4
and Videos 3 to 5 (available in Supplemental Material on-line) illustrate these findings.
In addition, the origin of the coronary arteries at the sinus Valsalva were clearly evident,
and normal branching of the coronaries was observed. Based on the micro-CT findings,
the feline heart was finally diagnosed as common arterial trunk with VSD.

Discussion

Common arterial trunk is a rare congenital heart disease which has been reported in dogs, cats and large animals [5-15]. It can be defined as a single great vessel that arises from the cardiac base, forming the outlet of both ventricles [16]. This abnormality appears to be due to complete failure of the separation of left and right ventricular outflow tracts in the developing heart. The 180° spiralling aorticopulmonary septum normally partitions the embryonic truncus arteriosus into the pulmonary artery and ascending aorta [17, 18]. This septum creates the normal spiral relationship between the aorta and pulmonary artery. The semilunar valves and their related sinuses are created by the absorption and hollowing out of subendocardial tissue swellings at the distal side of the truncal ridges. An absence of septation of the embryonic truncus arteriosus by the conotruncal ridges leads to a single large artery that begins from the base of the heart and supplies the systemic, pulmonary and coronary blood flows from both ventricles through the VSD [16]. The Collet and Edwards classification of common arterial trunk as persistent truncus arteriosus is based on the origin of the pulmonary artery in humans [19]. According to this classification, the present case exhibiting pulmonary arteries separately branched from close portions of the truncus would be classified as type II. More recently, the Congenital Heart Surgery Nomenclature and Database Project has published a simplified classification that subclassifies common

arterial trunk hearts into aortic or pulmonary dominance, emphasising the principal morphological determinant of surgical outcome [20]. The present feline case of common arterial trunk would be assigned in the aortic dominant form.

Feline common arterial trunk cases have been reported previously. Some cases were diagnosed as persistent truncus arteriosus, which is an older term of common arterial trunk as mentioned above. In a case report from 1968, a 3-week-old cat was diagnosed with common arterial trunk using radiography and confirmed at necropsy [5]. Chuzel et al. (2007) reported a case of common arterial trunk that was also confirmed at necropsy in a 5-month-old cat displaying a single pulmonary trunk which divided into left and right pulmonary arteries and a single aorta arising from the common arterial trunk [7]. Nicolle et al. (2005) reported a common arterial trunk case in a 6-year-old cat [12]. In this case, echocardiographic examination revealed no aorta arising from the left ventricle. The animal had a stenosis of the common arterial trunk. Furthermore, in a recent case report, echocardiography was used to reveal a dilated arterial trunk overriding the interventricular septum with a large VSD in two young cats. On further examination of these two animals using CT angiography, it became apparent that one cat had common arterial trunk and the other had pulmonary atresia with VSD [21]. Some animals, including the one presented in this study, can survive for years with common arterial trunk, but generally the prognosis for these animals is

1 171 poor [5, 7, 12, 21].
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5 172 To make a diagnosis of common arterial trunk, typical findings include: a large
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8 173 VSD, a great vessel overriding the VSD or the right or left ventricle, absence of a
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11 174 pulmonary trunk [16, 22]. Excluding the presence of a hypoplastic pulmonary artery is
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14 175 also needed to differentiate common arterial trunk from tetralogy of Fallot or pulmonary
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17 176 atresia (an extreme form of tetralogy of Fallot in which the main pulmonary artery is
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21 177 present but severely stenotic [19, 23, 24]). Transthoracic echocardiography is a non-
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24 178 invasive and widely accessible technique. Unfortunately, detailed echocardiographic
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27 179 visualisation of the structure of the heart with complex anomalies is usually time
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30 180 consuming and frequently limited due to intolerance for restraint particularly in patients
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33 181 with respiratory distress [25]. Advanced imaging modalities and post-mortem
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36 182 examination are thus important for definitive diagnosis of common arterial trunk. A
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40 183 recent report described the utility of using CT angiography to diagnose a complex
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43 184 congenital cardiac disease in cats similar to the case we are presenting [21]. Using CT
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46 185 angiography, which enables the rapid visualisation of vascular and tissue morphology,
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50 186 would be a preferred technique to be applied in living animals, in order to provide *in*
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53 187 *vivo* differential diagnosis of complex congenital disease.
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57 188 Micro-CT allows the detailed 3D visualisation of cardiac morphology. It offers high
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spatial resolution, low noise imaging compared to clinical CT scanners due to longer scan times (ranging from 30 min to 12 hr), higher frame averaging and the ability to adjust the geometric magnification by adjusting the distance between the source and sample. Micro-CT is well suited to the analysis of *ex vivo* tissues, but antemortem *in vivo* micro-CT imaging is currently limited due to the X-ray dose required for high resolution imaging and toxicity of the iodine contrast agent [26]. Furthermore, the 3D micro-CT datasets can be freely explored and virtually segmented using 3D visualisation software^l [4]. Micro-CT data can also be explored immersively with the use of virtual reality visualisation technology. Moreover, micro-CT data can be used to create detailed 3D-printed organ models reproducing the gross anatomy and including tissue density and fiber orientation. This can be a valuable tool for education. It must be emphasized that micro-CT with iodine has become the gold standard in our laboratory for studying healthy and diseased hearts. In addition, detailed 3D anatomical models of these complex congenitally malformed hearts have the potential to be used to perform computational modelling of cardiac blood flow [27, 28]. By improving our understanding of the complex morphology in congenital heart diseases, micro-CT can contribute to precise disease classification and treatment decision making for future cases.

Footnote

^h 15% I₂KI solution containing 5% iodine (Sigma Aldrich, 207772) and 10%

potassium iodide (Sigma Aldrich, 221945), Merck Life Science UK, Gillingham, UK

ⁱ Nikon XT H 320/225 kV High Flux equipped with PerkinElmer XRD 1611 xP flat

panel X-ray detector, Nikon Metrology NV, Leuven, Belgium

^j Amira version 6.5, Thermo Fisher Scientific, Waltham, MA, USA

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Figure legends

Fig.1 The right parasternal long-axis views of echocardiography from the cat with common arterial trunk (CAT) at the first presentation. (A) and (B) Long-axis views with a colour flow doppler mode in the diastolic (A) and systolic (B) phases. (A) Markedly dilated CAT, thickened myocardial wall of the right ventricle (RV) and interventricular septum and a membranous ventricular septal defect (*) accompanied by turbulent flow are observed. There is regurgitation from the CAT into the right and left ventricles through the ventricular septal defect (*). (B) Turbulent flow is broadly observed in the dilated CAT. See also Videos 1 and 2 (available in Supplemental Material on-line). CAT: common arterial trunk; LA: left atrium; LV: left ventricle; RV: right ventricle.

Fig. 2 Follow-up imaging from the cat with common arterial trunk (CAT) 12-month after the initial presentation. (A) and (B) left-to-right (A) and dorsal-to-ventral (B) images of thoracic radiography display severe cardiac enlargement, prominent pulmonary arteries and vessels and moderate pleural effusion. (C) and (D) The right parasternal long-axis four-chamber views of echocardiography in the diastolic (A) and systolic (B) phases visualise markedly dilated right atrium (RA) and thickened

myocardial wall of the right ventricle (RV). Pleural effusion (†) is also indicated. (E) and (F) The right parasternal long-axis views of the left ventricular outflow tract in the diastolic (E) and systolic (F) phases show that markedly dilated CAT. Turbulent blood flow around the CAT and ventricular septal defect (*) is observed. CAT: common arterial trunk; LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.

Fig. 3 Postmortem findings of the heart from the cat with common arterial trunk (CAT). (A) The appearance of the feline heart shows enlarged right atrium (RA) and hypertrophied right (RV) and left (LV) ventricles with the rounded apex. (B) An enlarged photograph of the boxed area in A shows CAT alongside the right (RPA) and left (LPA) pulmonary arteries which seem to originate from the root of the CAT. No evidence of the right ventricular outflow tract (*) suggesting hypoplasia. (C) A longitudinal-axis micro-computed tomography (CT) image of the feline heart represents separately branching into LPA and RPA from CAT. (D) A micro-CT image of the left ventricular outflow tract shows CAT which overrides a prominent ventricular septal defect (VSD). Markedly hypertrophied ventricular walls are also visualised. (E) A micro-CT image of a four-chamber view of the feline heart shows the atrioventricular valves located at the normal positions (arrowheads). Ao: aortic arch; CAT: common arterial trunk; LA: left

atrium; LPA: left pulmonary artery; LV: left ventricle; RA: right atrium; RPA: right pulmonary artery; RV: right ventricle; VS: interventricular septum; VSD: ventricular septal defect.

Fig. 4 The detailed morphology of communication among the great vessels in the feline heart with common arterial trunk (CAT). (A) Three-dimensional reconstructed feline heart with CAT. (B) A segmented image of the CAT, right (RPA) and left (LPA) pulmonary arteries and aortic arch (Ao) visualising the communication among the great vessels. (C) An intracavity view of B. (D) An intracavity view from the other side of B. (E) A short axis view illustrating the branching from CAT into RPA and LPA. The arrows indicate arteries abnormally branching from the Ao. Scale bars = 5 mm. See also Videos 3 to 5 (available in Supplemental Material on-line). Ao: aortic arch; CAT: common arterial trunk; LA: left atrium; LPA: left pulmonary artery; LV: left ventricle; RA: right atrium; RPA: right pulmonary artery; RV: right ventricle.

Video table

Video 1	Video1_CDI_CAT.avi	A colour-flow doppler echocardiography cine of a long-axis view of the ventricular outflow tract showing turbulent flow in the common arterial trunk.
Video 2	Video2_CDI_VSD.avi	A colour-flow doppler echocardiography cine of a long-axis view of the ventricular outflow tract showing turbulent flow around the ventricular septal defect.
Video 3	Video3_micro-CT1.avi	A three-dimensional (3D)-reconstruction micro-computed tomography (micro-CT) image of the appearance of the great vessels of the cat with common arterial trunk.
Video 4	Video4_micro-CT2.avi	A 3D-reconstruction micro-CT image of a cut-surface of the great vessels of the cat with common arterial trunk showing the

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origin of the pulmonary arteries.		
Video 5	Video5_micro-CT3.avi	A 3D-reconstruction micro-CT image of the great vessels of the cat with common arterial trunk with a sweeping cut-surface showing the origin of the pulmonary arteries.

Fig. 1

Fig. 2

Fig. 3

Fig. 4

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Video

Video1_CDI_CAT.avi

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Video

Video4_microCT2.avi

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Video

Video5_microCT3.avi