Case Log for the European Master of Small Animal Veterinary Medicine / Cardiology

Case log explanations and instructions:

The case log for the European Master of Small Animal Veterinary Medicine / Cardiology shall contain at least 200 cases mostly compiled in the second half of the program. Among the 200 cases, not less than 60 cases in cats and 60 cases in dogs. Other species are possible but not mandatory.

The following cases categories are mandatory (individual cases may include more than one category)**:**

1. 40 cases of acquired cardiac diseases (DCM, pericardial effusion, MMVD, heartworm, endocarditis etc) (code AD).
2. 20 cases of congenital diseases (for example PDA, pulmonic stenosis or aortic stenosis, tetralogy of Fallot, VSD, ASD etc. (code CD).
3. 20 cases of feline cardiomyopathy (code FC).
4. 10 cases of heart failure (can be from categories above)
5. 5 cases of arrhythmias (code AR).
6. 3 cases of syncope (SY)

For each case in Cardiology, the following information is mandatory (information is provided as an example)

1. **Date :** 10 February 2017.
2. **Name or file number** : Dougal, File 538743.
3. **Signalment :** Dog, crossbred, 10y, neutered female'
4. **Major complaint/ Problem :** heart murmur and dyspnea
5. **Examinations (ECG, ECHO, Radiographs, Holter, …) :** Radiographs show a left ventricular enlargement, LA is severly enlarged, VHS 12, alveolar lung pattern causdodorsally, pulmonray veins dilated. Echocardiography shows reduced systolic function LVIDs 36 (normal 25), FS 18 % (normal 25 %). The LV is also volume overloaded (M-Mode LVIDd 65 (normal 50). Moderate secondary mitral insufficiency. ECG shows hypervoltage (3.5 mV) and atrial fibrillation with average heart rate of 240/min
6. **Diagnosis :** DCM and atrial fibrillation
7. **Treatment**: initial stabilisation with Furosemide 5 mg/kg IV every hour until respratory rate was 40/min. Pimobendan IV. Long term management with Pimobendan, Furosemide (3 mg/kg TID), ACE-I BID. Atrial firbillation was treated with digoxin 0.022 mg/BSA.
8. **Complications/Results :** after one week atrial firbrillation rate was 140/min. Doígoxin level was measured.
9. **Follow-up :** dogs was stable for 1 months, then again pulmonary edema- Resolved with additional diuretic (Hydrochlorothiazide 1 mg/kg BID)
10. **Your comment :** owner shall count respiration rate
11. **Code :** AD

The case log needs to be compiled as an excel file using the template in the appendix
Abbreviations may be used but must be explained at the beginning of the case log table
List the cases in chronological order

Abbreviations:

LA: Left atrium
LLA:LA in measured right parasternal long-axis
RA RV
Right atrium and ventricle
Ao: diameter aorta

LVOT/RVOT
Left and Right ventricular outflow tract
LVPWd or s
Left ventricular posterior wall thickness (d: diastole s: systole)
IVSd or s
Interventricular septal thickness (d: diastole s: systole)
LVIDd or s
Left ventricular end diastolic or systolic diameter
EPSS
E-point septal separation
FS EF
Fractional shortening Ejection fraction
ESV EDV
End systolic or diastolic volume
PA LPA RPA
Pulmonary artery (main, left or right)
TV MV AV PV
Tricuspid, mitral, aortic or pulmonary valve
A P
Aortic or pulmonic velocity
MI TI AI PI
Mitral, tricuspid, aortic or pulmonary insufficiency.
LAA
Left atrial appendage blood flow.

DLVOTO: Dynamic left ventricular outflow tract obstruction
SAM: Systolic anterior motion
HCM HCOM: hypertrophic cardiomyopathy (o: obstructive)
RCM: restrictive cardiomyopathy// eRCM: endomyocardial RCM

nspCM: nonspecific phenotype (cardiomyopathy)
VSD ASD: Ventricular or atrial septal defect
L-R or R-L PDA: Left to right or vice versa patent ductus arteriosus
PS SAS: Pulmonic and Subaortic stenosis
ARVC : arrhythmogenic right ventricular cardiomyopathy
PH: pulmonary hypertension
MMVD: myxomatous mitral valve disease
DCM: Dilated cardiomyopathy
SVT : supraventricular tachycardia
R or L BBB: Right or left bundle bunch block
LAD: Left atrial dimension (right longitudinal)

SBP: Systemic blood pressure
ddx : differential
RRR: resting respiratory rate
BCh: biochemistry CBC: complete blood count
XR: X-rays

Case #	Date	Name or file number	Signalment, including body weight	Major complaint/Problem	Tests: ECG	Radiographs	Echocardiography	other tests (blood work, Holter etc.)	Diagnosis	Treatment	Outcome	Your comments	Code
1	08.12.2021	Name, File NXXX	Dog, Border Collie, 4 months, female intact, 4 kg	Presented for investigation of a heart murmur (continuous, left cranial 5/6).	Not performed. One lead ECG during echocardiographic examination suggested sinus rhythm.	Not performed.	Echocardiographic examination showed presence of moderate systolic function [LVIDsN 1.71 (normal < 1.14); LVESV 24.08 mL (normal < 11 mL); FS 22.9%; EF 58%], severe LV volume overload [LVEDV 61.6 mL (normal < 25.8 mL); LVIDdN 2.33 (normal < 1.66)], moderately dilated LA [LAD 39.3 mm (normal < 32 mm); LA/Ao 1.75 (normal < 1.6)]. normal RV and RA dimensions, normal valve morphologies and presence of a persistent ductus arteriosus with an ostium of 3.6 mm and an ampulla of 12 mm. Color doppler showed presence of a continuous turbulent blood flow through the PDA with a normal pressure gradient (5.12 m/s). Aortic and pulmonic velocity are within normal limits. Mitral inflow profile was slightly restrictive (1.2 m/s; E/A ratio 2.05). TDI measurements were normal (E' 14.25 cm/s; A' 10.76 cm/s). Lung ultrasound revealed absence of a significant amount of B-lines.	Biochemistry, CBC and clotting times were within normal limits.	PDA with severe secondary volume-overload of the left heart and left-sided congestive heart failure.	The dog was treated by interventional PDA occlusion via ACDO placement (Amplatz Canine Duct Occluder).	One month postoperative, echocardiographic examination showed marked decrease of the cardiac size. The dog was clinically doing well without medication. Six months postoperative, echocardiographic examination showed presence complete reverse remodelling of the heart with still mild systolic dysfunction. The next control is planned in 6 months.	The owner was instructed to monitor the dog for signs of weakness.	CD
2	12.09.2020	Name, File NXXX	Dog, Shar-pei, 5 yo , MN, 8 kg	During first consult in our centre because of painful tail a 4/6 systolic murmur over the left heart base was auscultated.	Right side axis deviation vs RBB, HR 144/min, sinus rhythm.	dilation over the PA and suspicion of enlarged RV.	RV both hypertrophy and dilation (pressure+volume overload) indicating chronic pressure overload /afterload mismatch. Also LV septal flattening. PV thickened and distorted with some fusion and a significant post-stenotic dilation but AV/Ao ratio was 1,55 . P velocity was 5,09 m/s (103,6 mmHg) with PI of 1,89 m/s. Left side dimensions and A and M inflows were normal. TV didn't show any signs of dysplasia. No TAPSE was recorded.	not performed	severe PS (type B)	atenolol 0,8 mg kg SID and then, after 1 week increased to BID. We recommended referral to an interventionist nearby to evaluate its fitness for the procedure. Owners refused initially referral due to financial reasons	control 1 and 4 month afterwards, pressure gradient 100 mmHg (velocity 5,0 m/s). On November he suffered a syncope and now owners agreed for ballooning of valve. After ballooning dog had no more syncope and a pressure gradient of 45 mmHg	Although there was some degree of dooming I considered it as type B (or at least, mixed).	CD
3	30.08.2019	Name, File NXXX	Dog, Spanish Mastiff, 7 yo, M 70kg	Dog referred by dermatology for arrhythmia. History of CKD.	AF with LBBB (160-180 bpm) - really calm dog-	not performed	Moderate enlargement of LV: LVIDD-n 1.9 (LVID 84 mm (N< 65), LVIDs-n 1.4, LVIDs 65 mm (max 48 mm)) and LA (LA/Ao 1,9) but apparently normal RV/RA. Reduced systolic function EPSS 14 (<6mm), and FS (average of 5) : 21-24%	CBC mild anemia, BC creatinine 2,7, BUN 90, normal electrolytes. Normal SBP.	DCM; DD: Possible tachycardia induced cardiomyopathy with AF + LBBB.	Digoxin 0,12 mg m2 every 12 h, Diltiazem delayed 4 mg kg every 12 h, pimobendan 0,25mg kg BID,	At 1 and 3 weeks control AF was at 100-130. Then 4 month afterwards, he developed ascites, lost muscular/weight, LV still enlarged and FS lowered to 19% so Digoxin levels were controlled (1,5 ng/ml). Spironolactone 2mg kg SID and furosemide 0,5 mg kg BID were added. Dog stabilized but died suddenly on April 2020.	It was 80 kg dog so really demanding echographic examinations	AR/AD/CHF
4	12.10.2022	Name, File NXXX	Cat, Maine Coone, 13 yo, male intact, 5.7 kg	Presented with signs of weight loss and for investigation of a heart murmur (systolic 3/6 left and right parasternal). Thyroid gland was enlarged on palpation. Furthermore no abnormalities were detected on physical examination.	Six lead 5 minute ECG showed normal sinus rhythm (184 bpm) without signs of arrhythmias.	Not performed.	Echocardiographic examination showed normal systolic function, normal ventricle dimensions and normal valve morphologies. Mild hypertrophy of the left ventricular free wall was present [LVPWd: 6.2 mm (normal < 5.5 mm)]. LA and RA were normal in size [LAD 14 mm (normal < 16 mm)]. Papillary muscles were prominent and false tendons were present in the left ventricle. Color doppler showed a mild mitral insufficiency due to systolic anterior motion of the mitral valve causing dynamic left ventricular outflow tract obstruction and turbulence in the RVOT. Spectral doppler showed an mildly increased velocity in the LVOT of of 3.2 m/s and a dynamic pattern in the RVOT with a velocity of 2.0 m/s suggesting DLVOTO and DRVOTO, respectively. Mitral inflow profile was suggestive of impaired relaxation (E<A). IVRT was slighlty increased [59 ms (normal < 55 - 60 ms)].	T4 values were markedly increased (72 nmol/L; nomal < 48 nmol/L).	HCM phenotype ACVIM stage B1 (possibly secondary to hyperthyroidism) with mild DLVOTO and DRVOTO.	Radio-iodine therapy.	One month after radio-iodine therapy the T4 values were well-controlled. Repeated echocardiography showed mild decrease in LV wall thickness compared to the pre-treatment echocardiogram. Control echocardiography was advised in 3 months, however the cat was lost to follow-up.	None.	FC, AD
5	15.11.2022	Name, File NXXX	Dog, Shih Tzu, 15 yo, male neutered, 4.2 kg	Presented with signs of syncope, coughing and exercise intolerance. Physical examination revealed a systolic heart murmur of 5/6 over the left apical region.	Six lead ECG showed the presense of sinus tachycardia. Sinus p waves were associated with narrow QRS complexes at a frequency of 160 - 180 bpm.	Thoracic radiographs showed an enlarged cardiac silhouette [VHS = 13.5 (normal < 11.5); VLAS = 4.0 (normal < 3)] with marked bulging of the left atrium. The trachea is markedly displaced dorsally and compressed by the left atrium. The pulmonary vessels are enlarged (both arteries and veins) and an ill-defined increased opacity is seen in the perihilar region. Some very thin fissure lines are visible bilaterally. Radiographs were consistent with marked left-sided and mild right-sided cardiomegaly with left atrial dilation and assiciated vascular congestion but no clear pulmonary oedema.	Echocardiographic examination showed presence of severe LV volume overload [LVIDdN 2.21 (normal < 1.7); LVEDV 28.9 mL (normal < 13.3)] and LA volume overload [LAD 31.3 mm (normal < 24.6mm); LA/Ao 1.92 (normal < 1.6)] and mild systolic dysfunction [LVIDsN 1.32 (normal < 1.09)]. Additionaly, mild RA dilation and RV volume overload were detected, a marked thickening of the mitral and tricuspid valve leaflets with marked prolaps and flail of the septal leaflet of the mitral valve. Color doppler revealed a severe mitral insufficiency with excentric jet and coanda effect with a normal pressure gradient of 109 mmHg on spektral doppler. Also severe tricuspid insufficiency with a mildly increased pressure gradient (38 mmHg) was detected, suggesting mild pulmonary hypertension. Mitral inflow profile is pseudonormal with a markedly increased E wave velocity of 1.53 m/s (normal < 1.1 m/s). Right ventricular systolic function was mildly decreased [TAPSE 6.3 mm (normal > 7.2 mm); S' 5.2 cm/s (normal > 5.9 ms/s)]. PV/PA ratio was markedly enlarged [2.1 (normal < 1.7)] and RPAD index was mildly decreased [26% (normal > 29%)]. Caval vein collapsability was at the lower reference range [34% (normal > 33%)].	Biochemistry showed a mild increased ALT value (probably due to early congestion) and the presence of moderate azotemia. Electrolytes incl. calcium and phosphor and complete blood count did not show significant abnormalities. Urine examination showed a decreased specific gravity. UPC value was normal. Blood pressure measurement was normal (systolic pressure of 140 mmHg). 24 hour Holter monitoring showed episodes of acute onset of sinus arrest with pauses of > 4 seconds) followed by a junctional escape rhythm before going back to normal sinus rhythm and episodes of sinus tachycardia suggesting sinus node dysfunction (ddx Sick sinus syndrome, however only 2 criteria fulfilled on 24h ECG).	Mitral- and tricuspid valve disease ACVIM stage C1 with severe left-sided volumeoverload, mild signs of pulmonary hypertension (most likely post-capillary, class 2) and sinus node dysfunction. Chronic kidney disease stage 2.	The dog was treated with pimobendan (0.3 mg/kg PO q12h), furosemide (2mg/kg/d PO), sildenafil (1.5 mg/kg PO q12h), benazepril (0.3 mg/kg PO q12h) and spironolactone (2.4mg/kg PO q12h). Omega 3 fatty acids were supplemented and food was changed to a cardiac diet. Additionally, theophylline (10mg/kg PO q12h) was started.	The dog experienced less syncopes after starting oral treatment. A control consult was planned in 1 month. However, the dog died suddenly 1 month after initial presentation.	Pacemaker implantation was proposed as a possible treatment option. Because of the poor prognosis of the structural heart disease the owner declined pacemaker implantation.	AR, SY
6	25.11.2020	Name, File NXXX	Dog, Maltese, 11 yo, female neutered, 5 kg	Presented with signs of syncope, partial anorexia, weakness and exercise intolerance. Syncopes were progressively worsening. Physical examination revealed marked bradycardia (40 bpm).	Regular bradycardia (44 bpm) with presence of complete dissociation between p waves and QRS complexes. Ventricular escape rhythm (44 bpm) dissociated from atrial activity (p wave frequency of 140 bpm). ECG diagnosis: third degree atrioventricular block.	The cardiac silhouette has normal dimensions (VHS 10.2), mild increased sternal contact and D-reverse shape on the VD projection. The trachea is mildly dorsally displaced; a soft tissue band is seen superimposed to the dorsal aspect of the trachea at the level of the thoracic inlet. the lung fields have normal dimensions and radiolucency.	Echocardiographic examination showed a normal systolic function, normal LV and RV volumes and normal LA and RA volumes. Valve morphologies were normal. Color doppler showed a moderate mitral insufficiency especially during diastole, a mild pulmonic insufficiency and tricuspid insufficiency with normal pressure gradients. Mitral inflow profile was abnormal showing more A waves than E waves, dissociated from eachother (typical for 3rd degree AV block).	Abdominal ultrasound revealed a hypoechoic splenic nodule (ddx nodular hyperplasia, extramedullary haematopoesis, hematoma or primary/secondary neoplasia). Fine needle aspirates of the nodule were indicative of nodular hyperplasia). CBC, biochemistry, T4, TSH and electrolytes were within normal limits. Snap 4Dx (Anaplasma, Erlichia, Dirofilaria, Borellia) was negative. Toxoplasma titers were negative. Cardiac troponin I levels were increased [0.258 µg/L (normal < 0.07 µg/L)].	Third degree atrioventricular block without structural cardiac changes. Most likely degenerative / fibrotic disease (ddx myocarditis not fully excluded).	Pacemaker implantation (single lead, right ventricular apex, active fixation, VVI 60 bpm bipolar).	One month after pacemaker implantation the dog did not show clinical signs and pacemaker position and function were normal. Settings were changed to VVIR with hysteresis of 40 bpm. Six months after pacemaker implantation, the pacemaker function and position was normal. Echocardiography revealed mitral valve disease ACVIM stage B1. One year after pacemaker implantation the dog was doing well, the splenic nodule was stable, the pacemaker funtion and position was normal and MMVD was stable.	The owner was asked to keep the dog calm for 1 month after the surgery. The owner was advised to not let the dog waer a collar, but replace it by a harnas.	AR, SY
7	29.09.2020	Name, File NXXX	Dog, Jack Russel Terrier, 11 months, female intact, 4.2 kg	Presented for investigation of a systolic heart murmur (left cranial, 4/6).	Normal sinus rhythm (112 bpm) with marked right axis deviation.	Not performed because thoracic CT scan was performed.	Echocardiography revealed moderate to severe right ventricular hypertrophy with moderate right atrial dilation and slight underfilling of the left heart (mild afterload mismatch). Normal RV systolic function [(TAPSE 10.2 mm (normal 7.7 - 12.3 mm); FAC 47.3% (normal 39.6 - 65.8 %)]. Presence of systolic paradoxical motion of the interventricular septum. The pulmonic valve appeared dome-shaped with partial attachment of the leaflet to eachother. The aorta to pulmonary artery ratio (AO/PA) was 1.08. Caval vein colapsability was normal ((45% (normal > 33%)] Furthermore, no significant abnormalities were detected on 2D images. A bubble study was performed, showing the absence of a right-to-left shunt (and absence of a persistent foramen ovale). Color doppler flow showed a turbulent flow over the pulmonary artery and a moderate to severe pulmonic regurgitation. Spectral doppler revealed a severely increased pressure gradient over de pulmonary artery of 110 mmHg. Aortic velocity was within normal limits, as well as mitral inflow profile profile.	Thoracic angigraphic CT-scan was performed showing thickening of the pulmonic valves with an AO/PA ratio of 0.9 with moderate post-stenotic dilation. Coronary arteries were normal. Right ventricular hypertrophy and right atrial dilation was confirmed. Furthermore, no significant abnormalities were detected. The lung parenchym did not show significant abnormalities. Complete blood count, biochemistry panel and electrolytes were measured and did not reveal significant abnormalities. Clotting times were within normal reference range.	Severe valvular pulmonic stenosis (type A) with a pressure gradient of 110 mmHg. Presence of moderate to severe right ventricular hypertrophy and moderate RA dilation. No signs of right sided heart failure.	Atenolol was started (titrated up till 1.4mg/kg PO q12h). Balloon valvuloplasty was performed one month after starting atenolol	One month balloon valvuloplasty, the dog did not have complaints and was doing well. Repeated echocardiography revealed a reduction in pressure gradient of 30%. Six months after balloon valvuloplasty, the dog was clinically doing well and did not have clinical signs. Repeated echocardiography revealed 42% of reduction in pressure gradient over the pulmonic valve. A reduction in RA dilation and RV hypertrophy was detected. Mild RV hypertrophy and mild RA dilation was still present. One year after balloon valvuloplasty repeated echocardiography revealed a 47% reduction in pressure gradient with stable measurements of RA and RV. Atenolol therapy has been continued.	A yearly control visit has been advised after the last control.	CD