COMMON HEART DISEASES IN DOGS AND MANAGEMENT

Cardiorespiratory Center
Thonglor Pet Hospital Rama IX

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PGFVS (Cardiorespiratory Med.)

HEART DISEASE IN DOGS

Cardiorespiratory Center
Thonglor Pet Hospital Rama IX

SITHA S. DVM,
PGFVS (Cardiorespiratory Med.)

CONGENITAL HEART DISEASE : 5%

ACQUIRED HEART DISEASE : 95%

OF ALL HEART CONDITIONS IN DOGS.

1. Degenerative mitral valve disease (DMVD) : >75%
2. Dilated cardiomyopathy (DCM) : 8-10%
3. Cardiac mass : 5%

OUTLINE

1. Identification of the diseases **(DMVD).**

- Staging of the severity

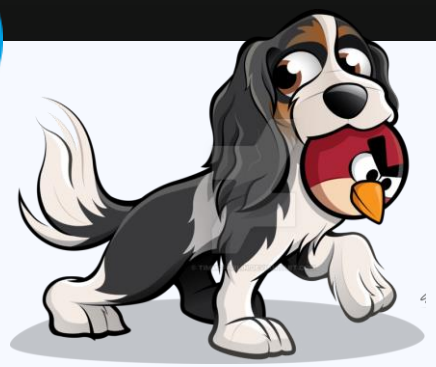
2. Management.

- Pre-clinical (heart disease)
- Clinical (heart failure)
- Applying "**evidence-based medicine**"

OUTLINE

1. Identification of the diseases (DMVD / MMVD).

- Staging of the severity

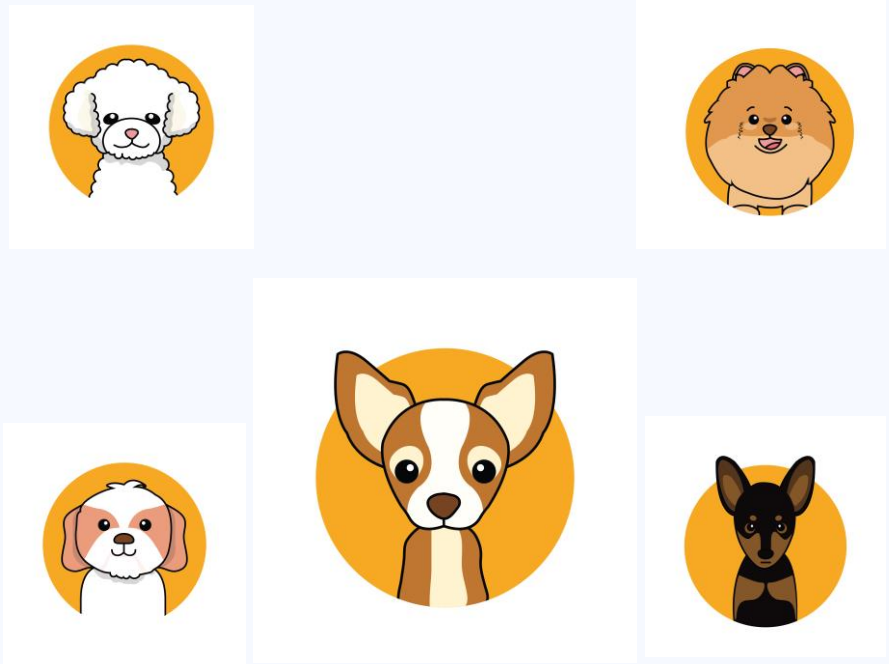


DEGENERATIVE MITRAL VALVE

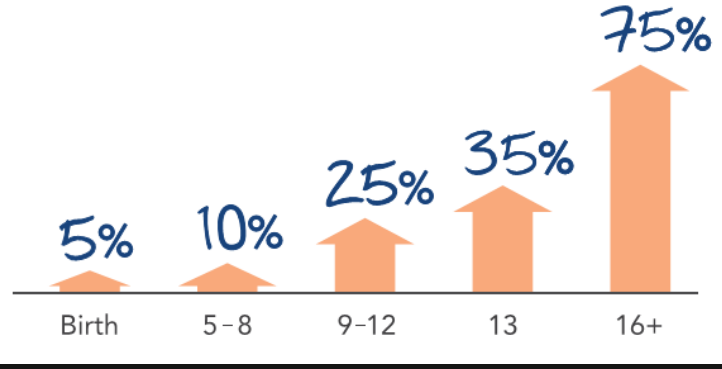
SLOW PROGRESSIVE DISEASE

AGING-ASSOCIATED DISEASES
SMALL TO MEDIUM-SIZED BREEDS,
MALE (1.5X) > FEMALE

- 62% MITRAL VALVE ALONE
- 32.5% MITRAL & TRICUSPID VALVES

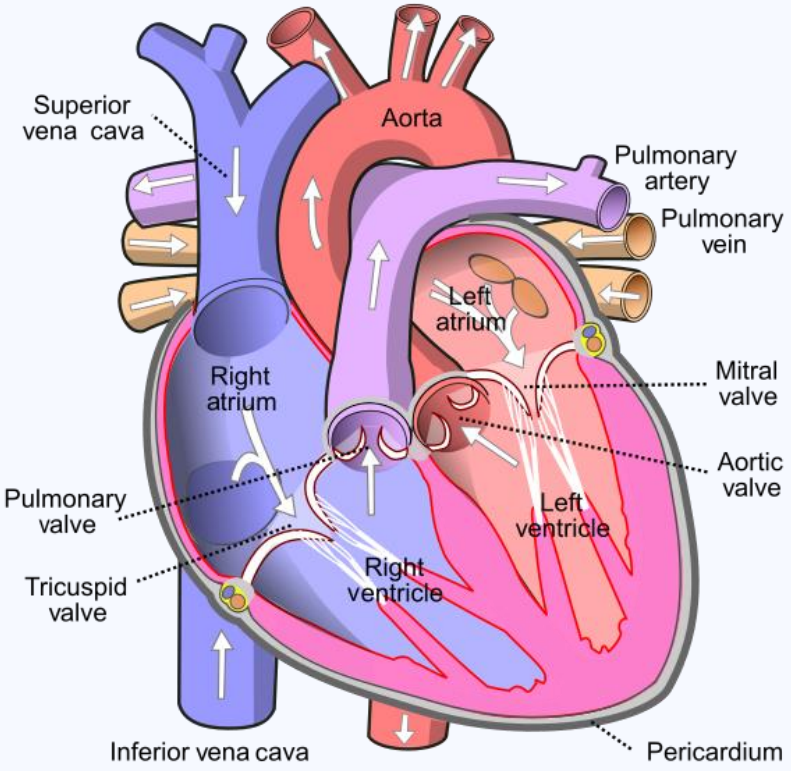


THE LIKELIHOOD OF HEART DISEASE INCREASES DRAMATICALLY WITH AGE.[1,2]



MITRAL VALVE APPARATUS

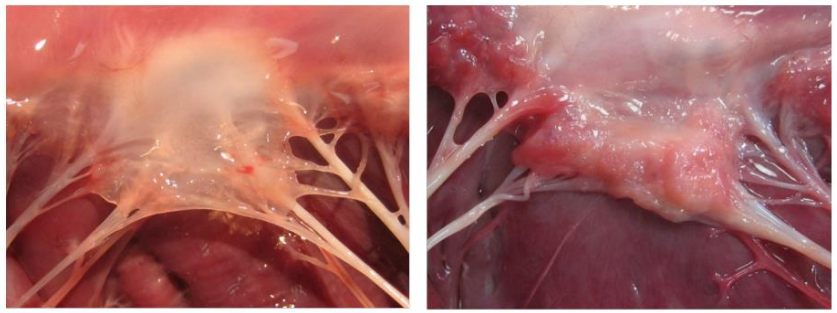
- ♥ Valve leaflets (Ant./ Post.)
- ♥ Annulus
- ♥ Chordae tendinae
- ♥ Papillary muscles



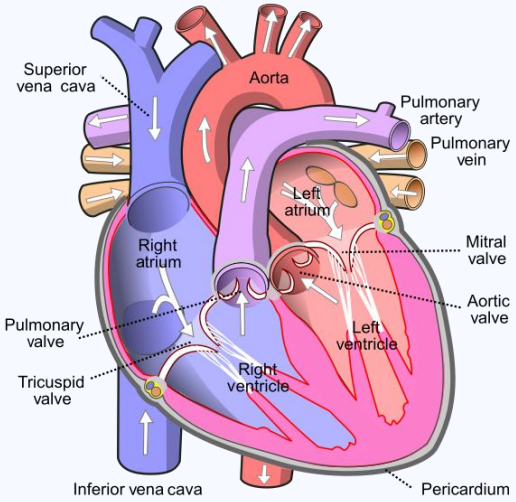
DEGENERATIVE MITRAL VALVE

ANTERIOR MITRAL VALVE
POSTERIOR MITRAL VALVE

- ♥ Mucopoly-saccarides
- ♥ Collagen degeneration
- ♥ Endothelial proliferation



Normal DMVD



- **Thickened** leaflets / chordae
- **Mitral valve regurgitation**
- **LA & LV Chamber dilation**

- Increase LA pressure
- Pulmonary venous congestion
- Pulmonary edema

DMVD SLOW PROGRESSIVE DISEASE

mitral regurgitation (**murmur HS**) ----------> B1

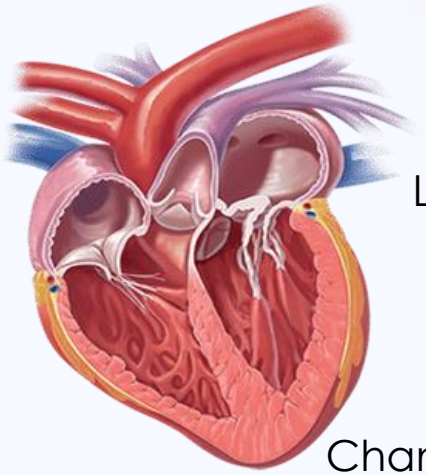
- Volume overload

Chamber(LA , LV) dilation ----------> B2

- Increase LA pressure
- Pulmonary congestion

Pulmonary edema ----------> C

LV systolic dysfunction (end stage) ----------> D



DCM

LV systolic dysfunction

Chamber dilation / Arrhythmia

Functional mitral regurgitation ➡ lung edema
Arrhythmia

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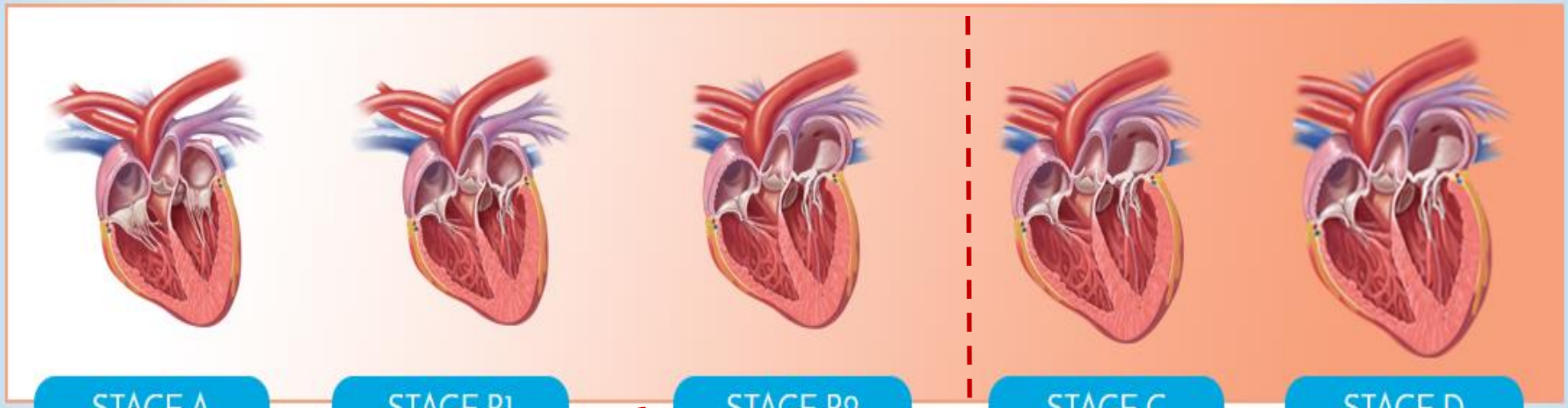
CONSENSUS STATEMENT

Journal of Veterinary Internal Medicine ACVIM Open Access

Consensus Statements of the American College of Veterinary Internal Medicine (ACVIM) provide the veterinary community with up-to-date information on the pathophysiology, diagnosis, and treatment of clinically important animal diseases. The ACVIM Board of Regents oversees selection of relevant topics, identification of panel members with the expertise to draft the statements, and other aspects of assuring the integrity of the process. The statements are derived from evidence-based medicine whenever possible and the panel offers interpretive comments when such evidence is inadequate or contracictory. A draft is prepared by the panel, followed by solicitation of input by the ACVIM membership which may be incorporated into the statement. It is then submitted to the *Journal of Veterinary Internal Medicine*, where it is edited prior to publication. The authors are solely responsible for the content of the statements.

ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

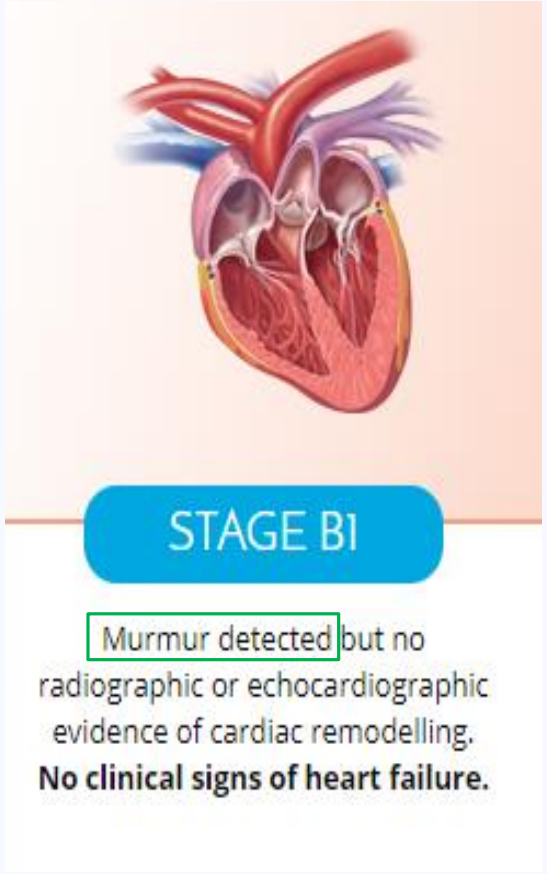
ACVIM CONSENSUS (2009 → 2019)



No evidence of cardiogenic lung edema on chest-XR

+ Evidence of cardiogenic lung edema on chest-XR

MMVD STAGE B1



HEART MURMURS

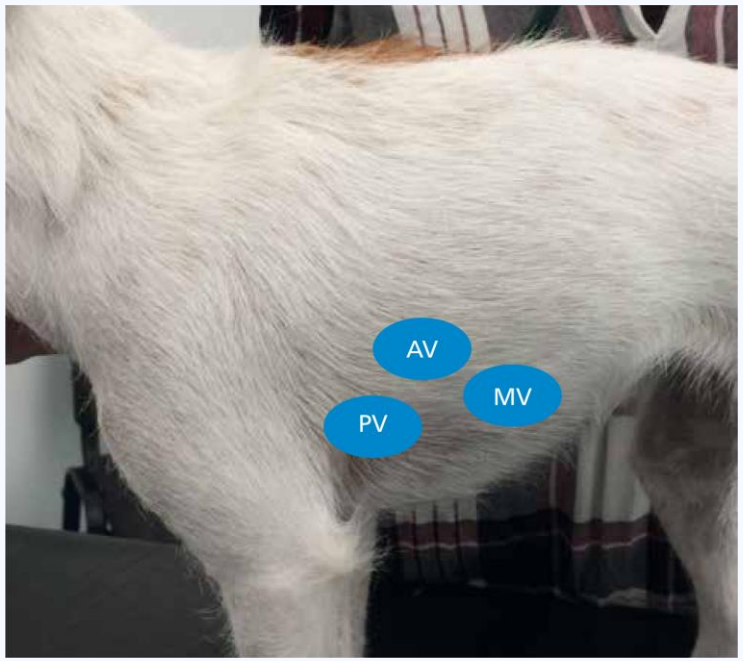


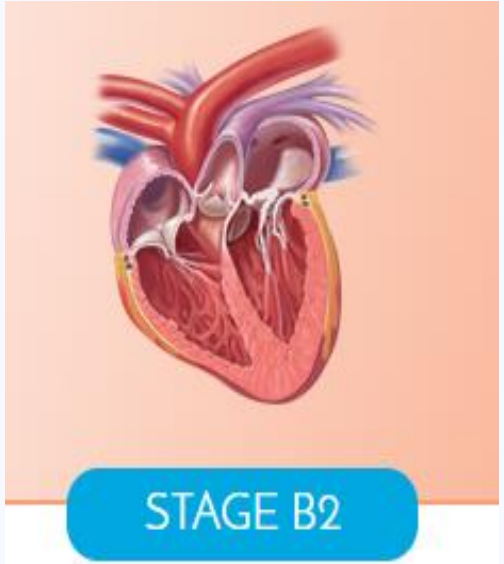
Table 2. Heart murmur grading

Intensity	Grade	Loudness
Low intensity	I	Low intensity murmur heard in a quiet environment only after careful auscultation over a localised cardiac area
	II	Low intensity murmur heard immediately when the stethoscope is placed over the PMI
Moderate intensity	III	Murmur of moderate intensity **localized to a single area**
	IV	High intensity murmur that can be auscultated over several areas without any palpable precordial thrill
High intensity	V	High intensity murmur with a palpable precordial thrill
	VI	High intensity murmur with a palpable precordial thrill that may even be heard when the stethoscope is slightly lifted off the chest wall

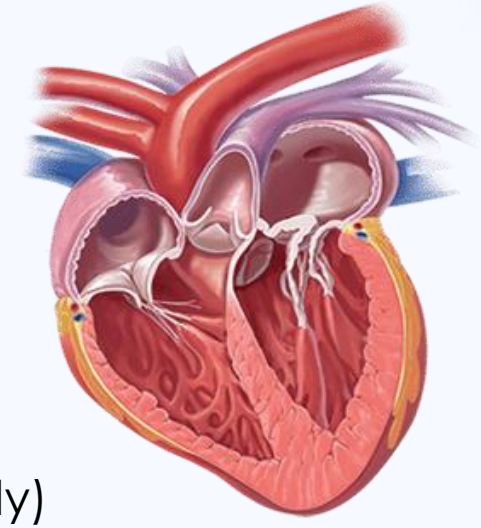
Adapted from: Kvart, 2010: 22

Dogs—left side, 5th intercostal space at costochondral junction
Cats—left side, 5th–6th intercostal space, near sternum

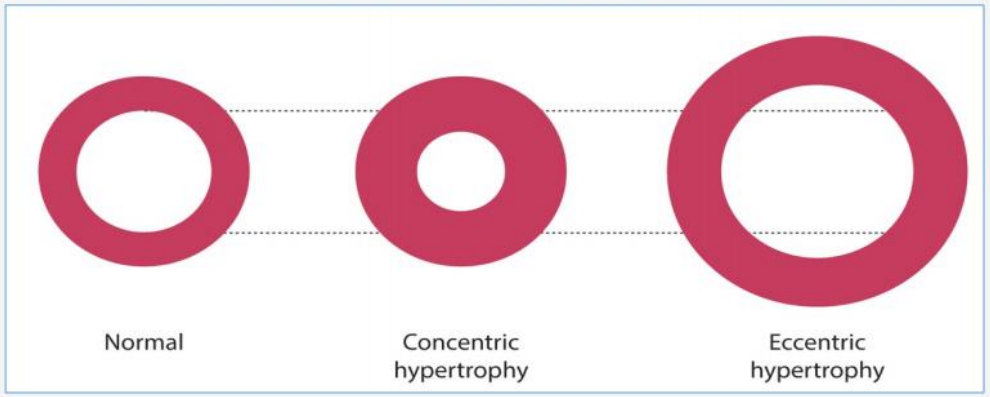
MMVD STAGE B2

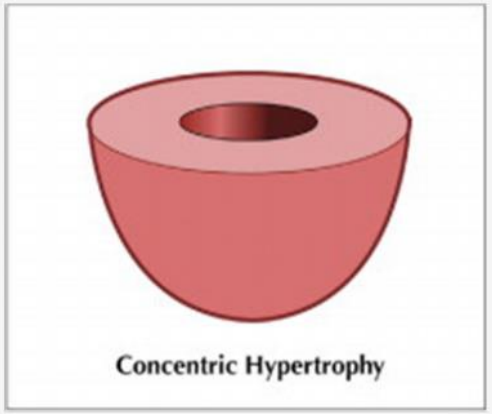


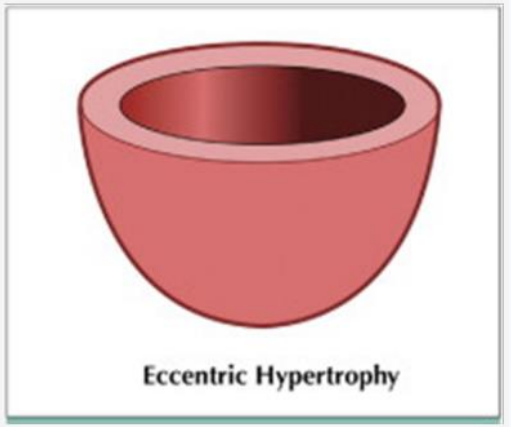
No evidence of cardiogenic lung edema on chest-XR



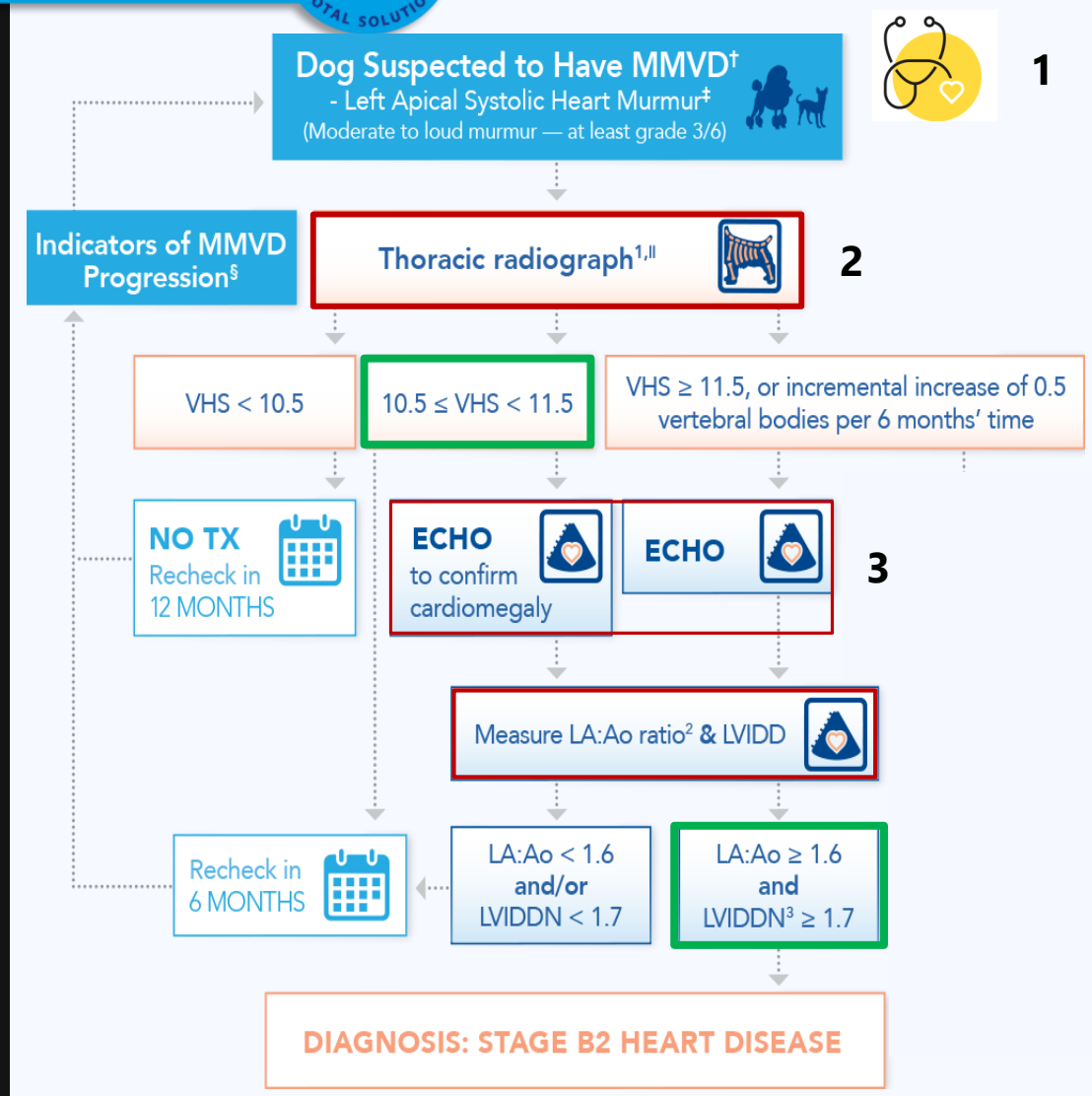
Heart enlargement (Cardiomegaly)







- May be usually thick or **dilated**, permanent or temporary depending on the causes.



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ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

Bruce W. Keene[1] | Clarke E. Atkins[1] | John D. Bonagura[1,2] | Philip R. Fox[3] | Jens Häggström[4] | Virginia Luis Fuentes[5] | Mark A. Oyama[6] | John E. Rush[7] | Rebecca Stepien[8] | Masami Uechi[9]

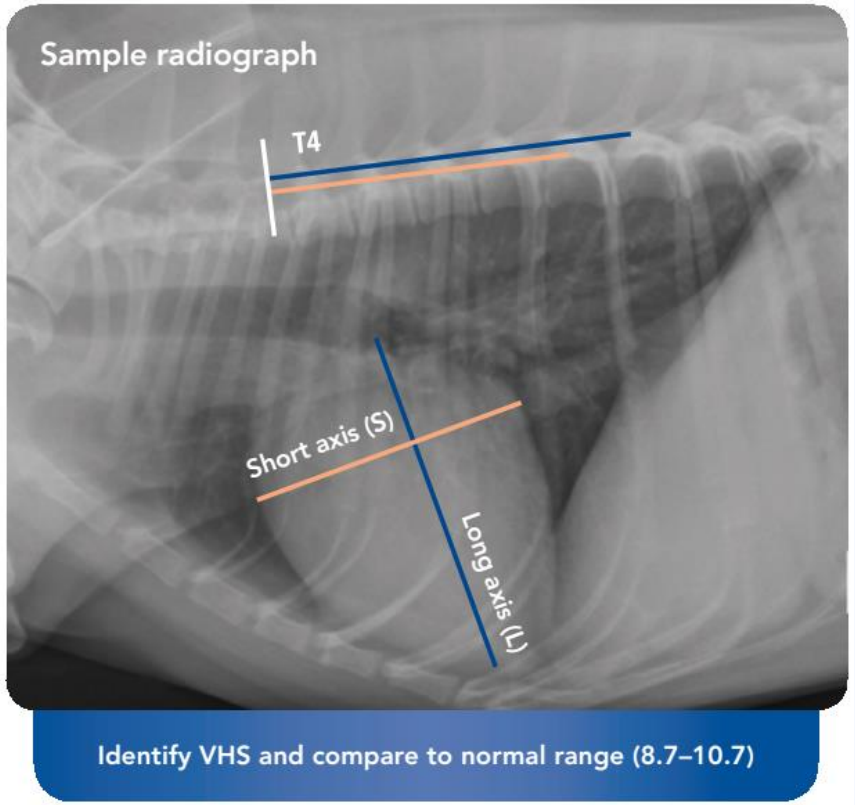
- Stage B2 criteria for heart enlargement identify dogs that are likely to benefit substantially from treatment before the onset of clinical signs of heart failure. (Class I, LOE: Strong):
  - murmur intensity ≥3/6;
  - echocardiographic LA : Ao ratio in the right-sided short axis view in early diastole ≥1.6 (Figure 1)[45];
  - Left ventricular internal diameter in diastole, normalized for body weight (LVIDDN) ≥1.7 (Table 1)[46];
  - breed-adjusted[47–53] radiographic vertebral heart score (VHS) >10.5.

Left side heart enlargement **(Stage B2)** criteria

1. **PE:** Murmur intensity **≥3/6**
2. **Radiographic** vertebral heart score (VHS) **>10.5**

3. **Echocardiographic**
- LA:Ao ratio in the right-sided short axis view in early diastole **≥1.6**

- Left ventricular internal diameter in diastole, normalized for body weight (LVIDdN) **≥1.7**



SAMPLE VHS CALCULATIONS FROM RADIOGRAPH ABOVE

This example: **Long axis line = 5.2,** Short axis line = 4.4

VHS = L + S
= 5.2 + 4.4
= 9.6
= in normal range

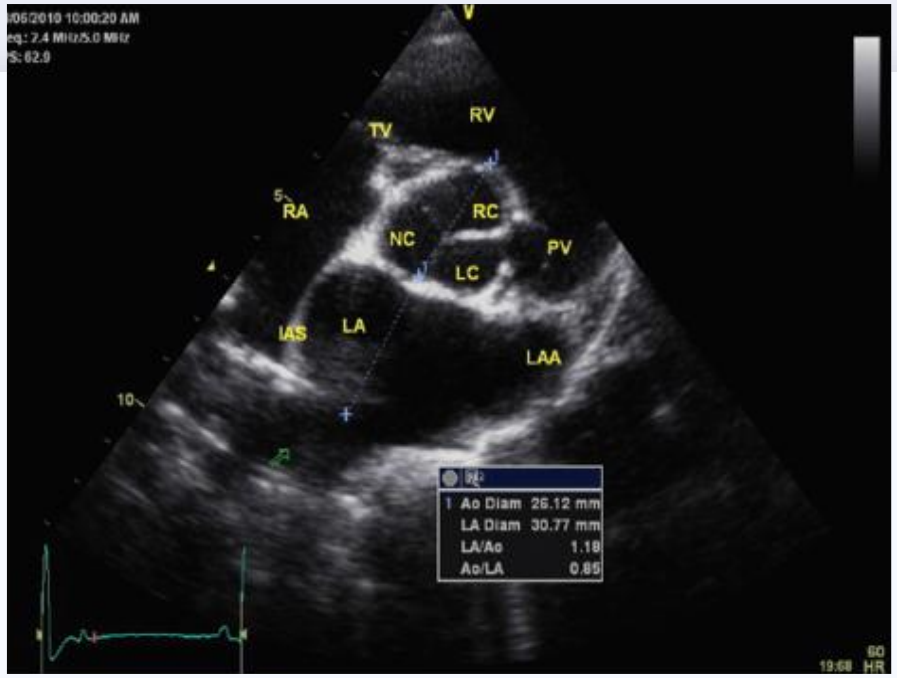
Left side heart enlargement **(Stage B2)** criteria

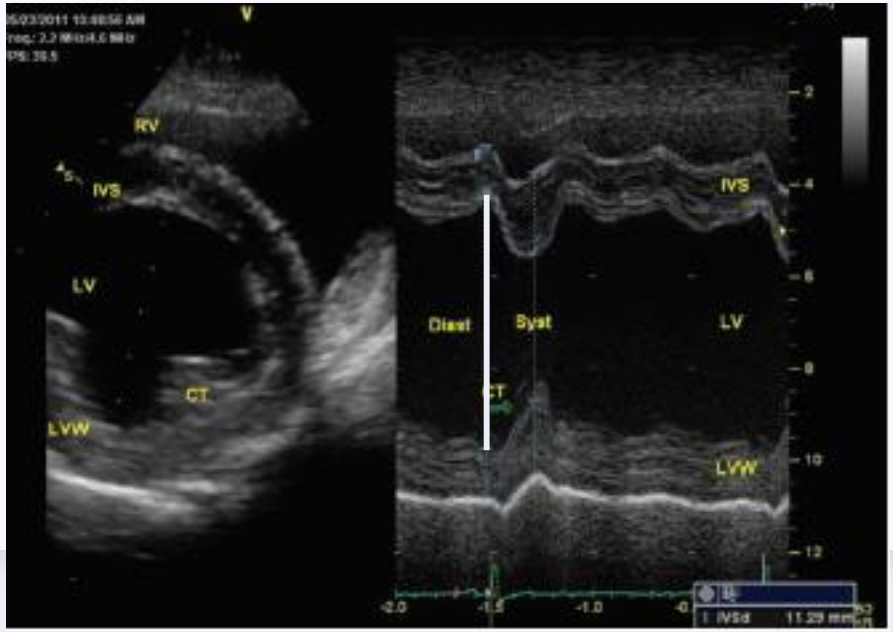
1. **PE:** Murmur intensity **≥3/6**
2. **Radiographic** vertebral heart score (VHS) **>10.5**

3. **Echocardiographic**
- LA:Ao ratio in the right-sided short axis view in early diastole **≥1.6**

- Left ventricular internal diameter in diastole, normalized for body weight (LVIDdN) **≥1.7**

$$LVIDDN = \frac{\text{measured LVIDd (cm)}}{\text{Weight (kg)}^{0.294}}$$





Dog Suspected to Have MMVD†
- Left Apical Systolic Heart Murmur‡
(Moderate to loud murmur — at least grade 3/6)

1

Indicators of MMVD Progression§

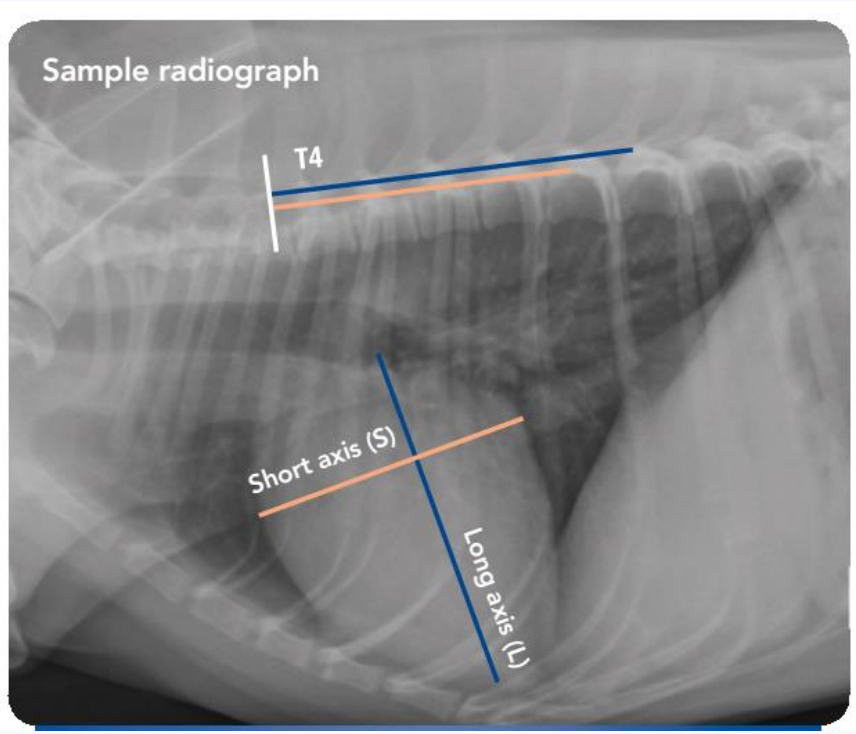
Thoracic radiograph[1,‖]

2

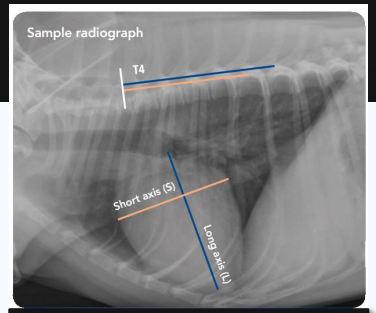
VHS < 10.5	10.5 ≤ VHS < 11.5	VHS ≥ 11.5, or incremental increase of 0.5 vertebral bodies per 6 months' time
NO TX Recheck in 12 MONTHS	Recheck in 6 MONTHS	**ECHO UNAVAILABLE**

DIAGNOSIS: STAGE B2 HEART DISEASE

Echo unavailable

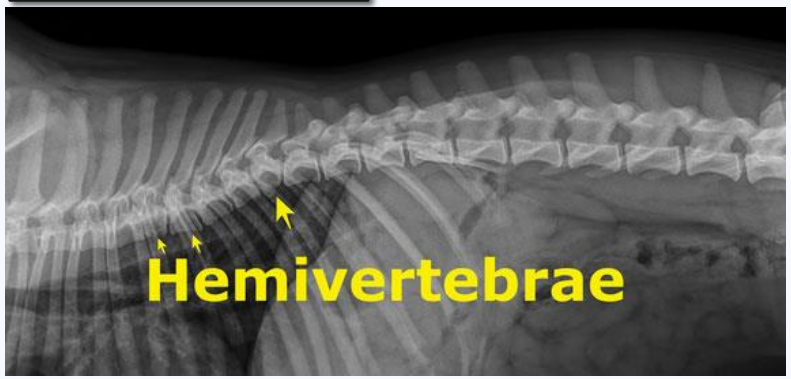


An increase in VHS of more than 0.07/month is a significant risk factor for the development of congestive heart failure within the next 6 months.



VHS limitation

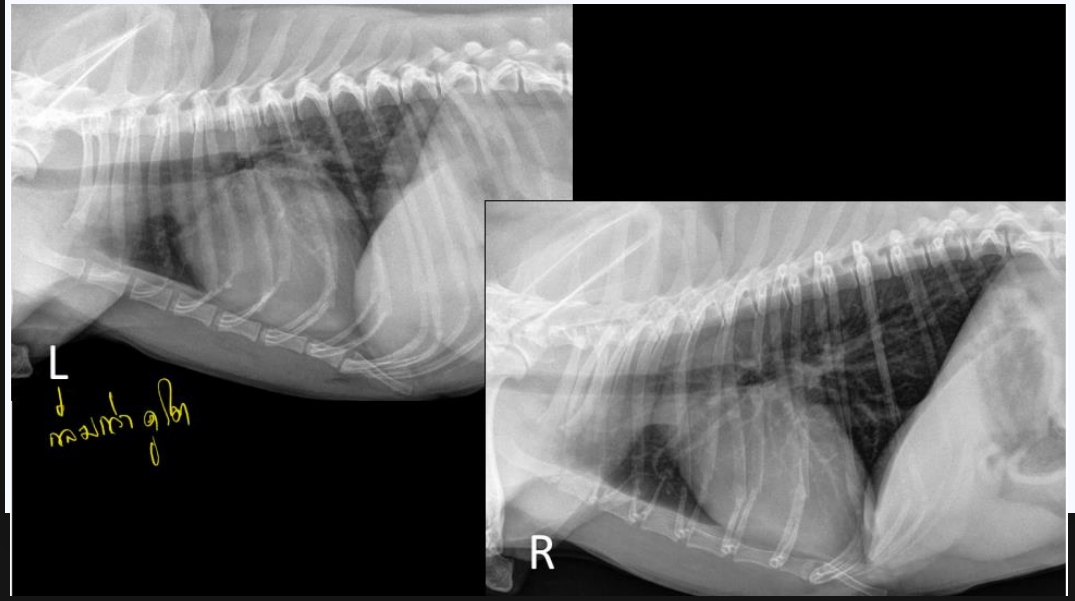
VHS also **varies** with breed, positioning hemivertebrae, cardiac cycle, heart rate, sedation…

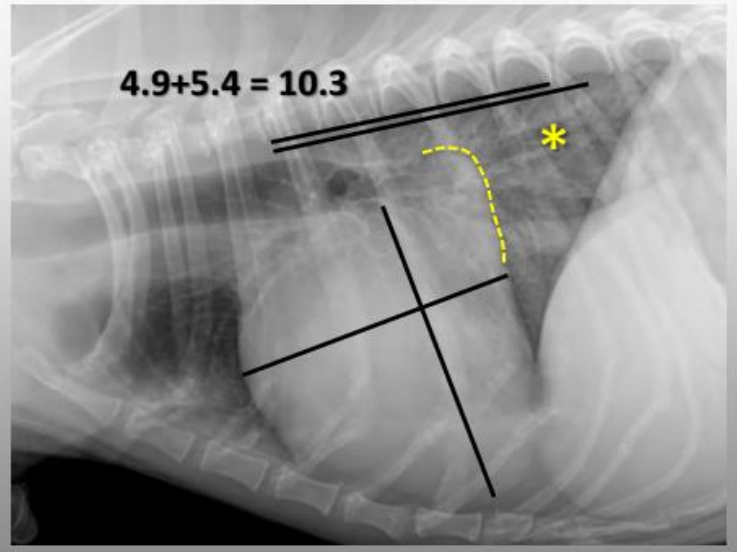


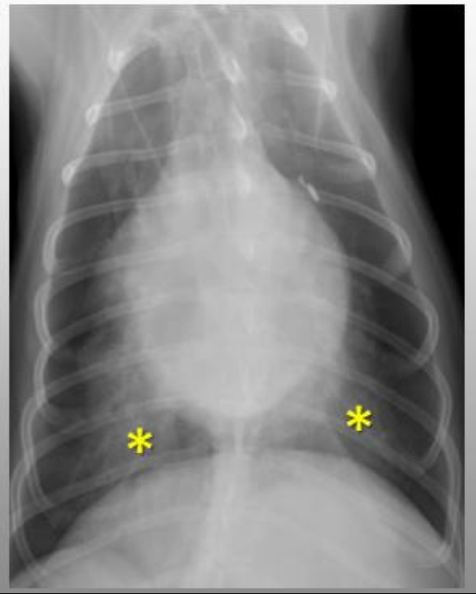
VHS
Cutoff: **≥12.25** vertebrae
Sensitivity: 30%
Specificity: 96%

Table 2: Vertebral heart score (VHS) normal values

	VHS range		VHS range
Average normal for most dogs	9.2-10.2	Greyhound	10.4-10.6
Beagle	10.1-10.9	Labrador	10.2-10.6
Boxer	10.8-12.4	Lhasa Apso	8.8-10.4
Boston Terrier	10.3-13.1	Pug	9.8-11.6
Bulldog	11.0-14.4	Poodle	9.6-10.6
Cavalier King Charles	10.1-11.1	Rottweiler	9.7-9.9
Cocker Spaniel	11.0	Shih Tzu	8.9-10.1
Doberman	9.4-10.6	Dachshund	9.3-11.6
German Shepherd	9.0-10.4	Whippet	10.8-11.8
Pomeranian	9.6-11.4	Yorkshire Terrier	9.3-10.5







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VLAS (Vertebral Left Atrial Size)

- A newer index of radiographic left atrial enlargement, the VLAS, provides a quantitative method of estimating left atrial size. Measured on either the right or left lateral radiograph by drawing a line from the center of the most ventral aspect of the carina to the most caudal aspect of the LA where it intersects with the dorsal border of the caudal vena cava, that line then is transposed to the cranial edge of the 4th thoracic vertebral body.[54] Studies are ongoing to determine a VLAS value that accurately predicts B2 remodeling, but in the absence of echocardiography, VLAS values of ≥3 likely identify Stage B2 MMVD. (Class 1, LOE: moderate)

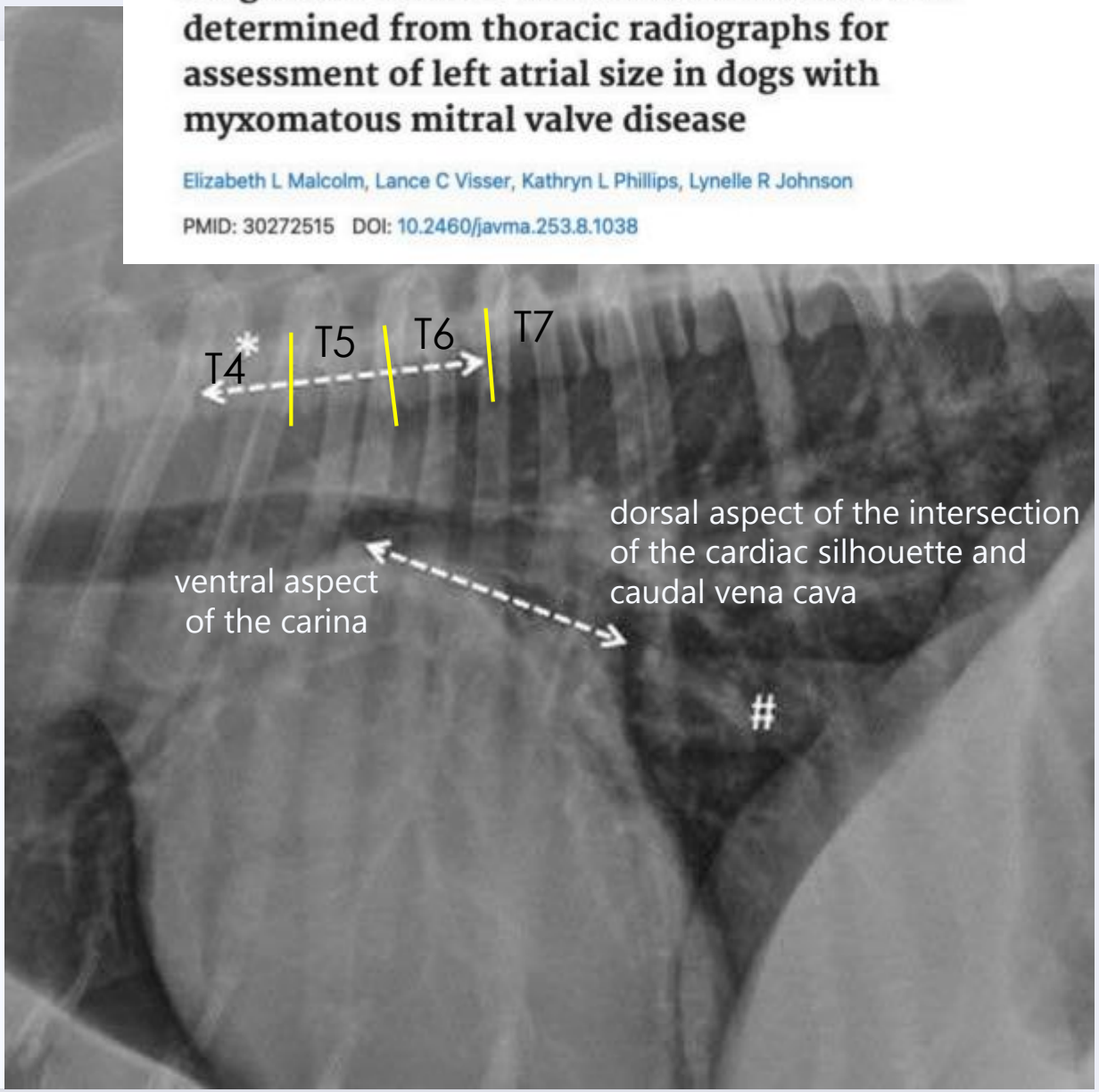
VLAS in predicting LA/Ao ≥1.6
Cutoff: **≥2.5** vertebrae
Sensitivity: 67%
Specificity: 87%

https://www.imv-imaging.co.uk/veterinary-learning/journal-club/vertebral-left-atrial-score-vlas/

Diagnostic value of vertebral left atrial size as determined from thoracic radiographs for assessment of left atrial size in dogs with myxomatous mitral valve disease

Elizabeth L Malcolm, Lance C Visser, Kathryn L Phillips, Lynelle R Johnson

PMID: 30272515 DOI: 10.2460/javma.253.8.1038



VLAS (ACVIM 2019) **≥3** : **Stage B2**

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RLAD (**R**adiographic **L**eft **A**trial **D**imension)

RLAD was obtained by
drawing a line bisecting the 90 degrees angle defined by the long and short cardiac axes lines of the VHS, up to the dorsal edge of the left atrium and comparing its length to T4's vertebral body length.

RLAS in predicting LA/Ao ≥1.6
Cutoff: **≥1.8** vertebrae
Sensitivity: 96.8%
Specificity: 93.5%

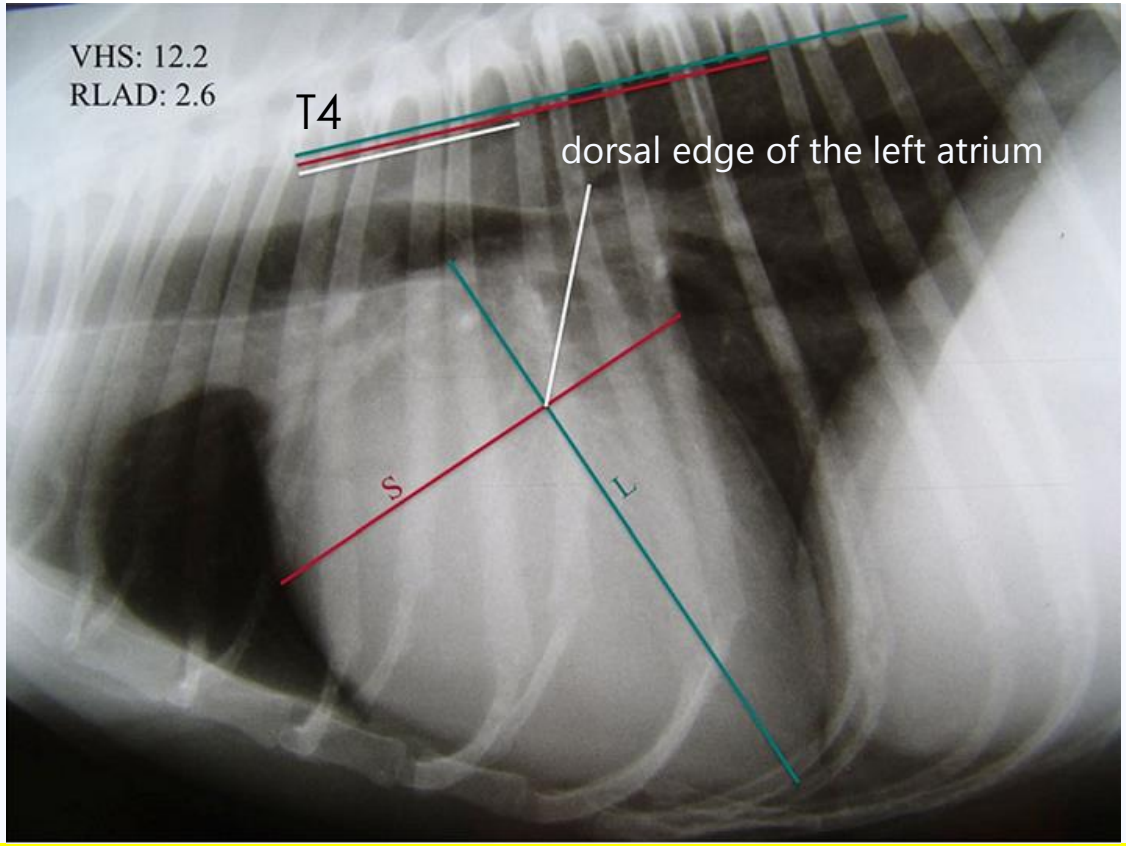
Sánchez Salguero *et al. Irish Veterinary Journal* (2018) 71:25
https://doi.org/10.1186/s13620-018-0137-x

Irish Veterinary Journal

RESEARCH Open Access

A radiographic measurement of left atrial size in dogs

Xavier Sánchez Salguero[1], David Prandi[2,3], Francisco Llabrés-Díaz[4], Edgar Garcia Manzanilla[5,6*] and Claudio Bussadori[7]



RLAD (Vertebral Left Atrial Dimension) **≥1.8** : **LA enlargement**

Echo unavailable

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Radiographic quantification of left atrial size in dogs with myxomatous mitral valve disease

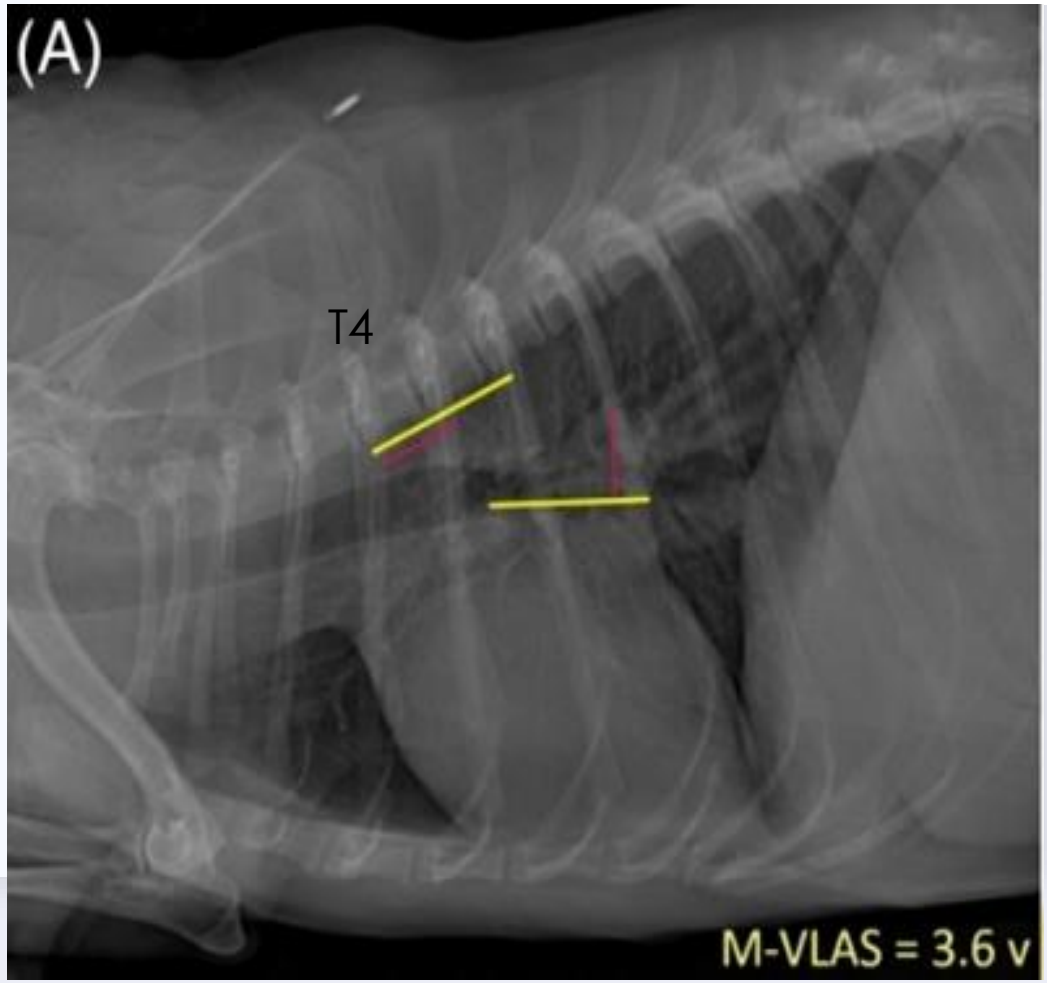
Christopher Lam | Brad J. Gavaghan | Fiona E. Meyers

M-VLAS (Modified Vertebral Left Atrial Size)

1 line: VLAS

2 line, at the most distal LA border excluding the pulmonary vein orifice, and extended to perpendicularly intersect with the first line(VLAS).

M-VLAS in predicting LA/Ao ≥1.6
Cutoff: **≥3.4** vertebrae
Sensitivity: 93%
Specificity: 93%



Radiographic quantification of left atrial size in dogs with myxomatous mitral valve disease

Journal of Veterinary Internal Medicine Open Access ACVIM American College of Veterinary Internal Medicine

Christopher Lam | Brad J. Gavaghan | Fiona E. Meyers

TABLE 1 Descriptive data for control healthy dogs and dogs with various stages of myxomatous mitral valve disease

Variable	Control	Stage B1	Stage B2	Stage C	*P* value
No. of dogs	6	22	21	21	-
Female	5	11	8	7	-
Male	1	11	13	14	-
Bodyweight (kg)	7.57 ± 2.13	8.37 ± 3.63	9.41 ± 2.93	6.96 ± 3.02	.09
Age (year)	8.72 ± 5.89	11.07 ± 2.77	10.65 ± 2.93	10.96 ± 2.13	.33
LA/Ao	1.26 ± 0.18	1.18 ± 0.14	2.07 ± 0.42[a]	2.39 ± 0.52[a]	< .001
LVIDdN	1.32 ± 0.15	1.51 ± 0.22	2.00 ± 0.18[a]	2.09 ± 0.35[a]	< .001
M-VLAS	2.60 ± 0.30	2.75 ± 0.55	4.10 ± 0.57[a]	4.57 ± 0.86[a]	< .001
VLAS	1.83 ± 0.29	1.93 ± 0.33	2.66 ± 0.36[a]	2.92 ± 0.54[a]	< .001
VHS	9.28 ± 0.77	10.28 ± 0.76	11.61 ± 0.93[a]	12.04 ± 1.52[a]	< .001
RLAD	1.22 ± 0.24	1.54 ± 0.42	2.34 ± 0.43[a]	2.71 ± 0.66[a]	< .001
Medications at time of assessment	n/a	Frusemide (2/22)[b], pimobendan (2/22)[b], codeine (1/22), doxycycline (1/22)	Pimobendan (6/21), trilostane (2/21), clopidogrel (1/21), rivaroxaban (1/21)	Frusemide (13/21), pimobendan (12/21), benazepril (6/21), trilostane (1/21), insulin (1/21), amoxycillin-clavulanate (1/21), diltiazem (1/21)	-

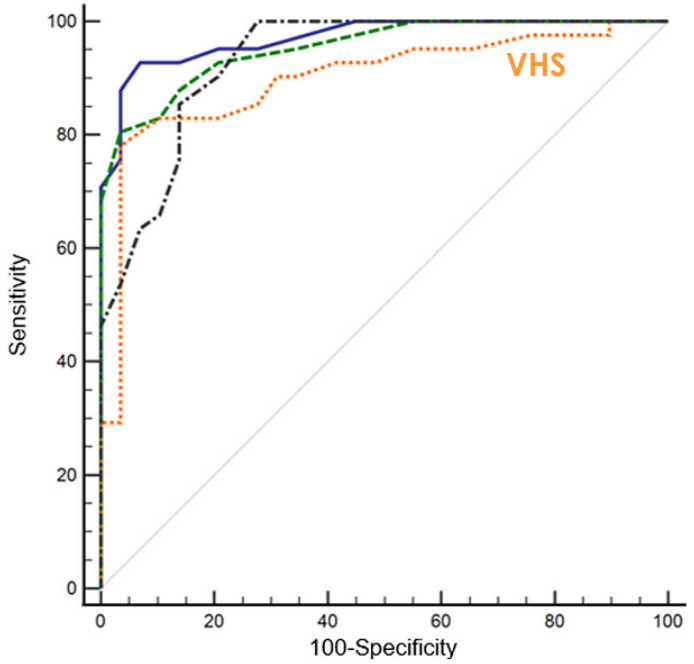


FIGURE 2 Receiver operating characteristic (ROC) curves of M-VLAS, VLAS, VHS, and RLAD measured in 29 dogs with LA/Ao < 1.6, and 41 dogs with LA/Ao ≥1.6. Significant difference in AUC was observed between M-VLAS (AUC 0.97, 95% CI 0.90-1.00) and VHS (AUC 0.90, 95% CI 0.80-0.96) (*P* = .03). Blue solid—M-VLAS, green dashed—VLAS, orange dotted—VHS, and black dot-dashed—RLAD

The results of this study showed a positive correlation between M-VLAS, VLAS, VHS and RLAD with LA/Ao in dogs with MMVD. Significantly higher M-VLAS, VLAS, VHS, and RLAD were observed in stage B2 and C dogs than stage B1 and control healthy dogs.

Comparison of ROC curves indicated VLAS, RLAD, and M-VLAS was superior to VHS in identifying this LA enlargement.

Use of radiographic measurements to diagnose stage B2 preclinical myxomatous mitral valve disease in dogs

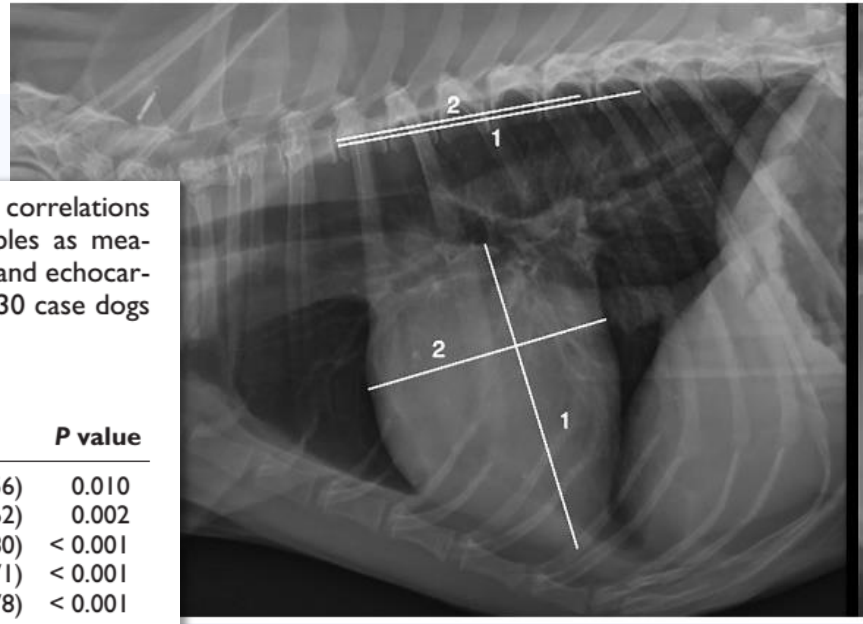
Rebecca L. Stepien DVM, MS
Mariola B. Rak DVM
Lauren M. Blume BVSc

Table 2—Results of analysis to identify potential correlations between results for selected radiographic variables as measured on left lateral thoracic radiographic images and echocardiographic variables for the 25 control dogs and 30 case dogs described in Table 1.

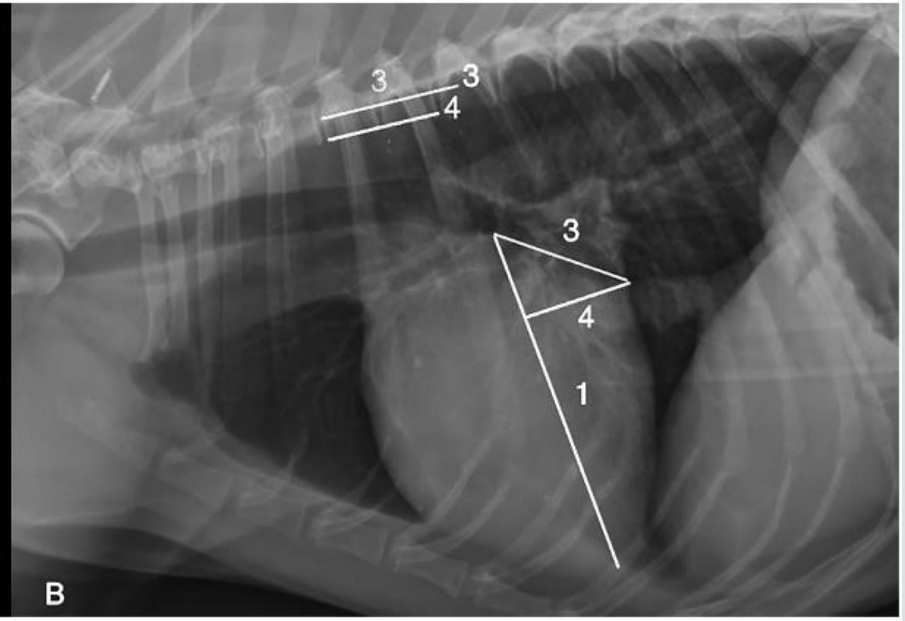
Echocardiographic variable	Radiographic variable*	Correlation coefficient (95% CI)	*P* value
LVIDDN	VHS_{length}	0.34 (0.09 to 0.56)	0.010
	VHS	0.42 (0.17 to 0.62)	0.002
	VLAS	0.68 (0.51 to 0.80)	< 0.001
	LA_{width}	0.54 (0.31 to 0.71)	< 0.001
	LA_{total}	0.65 (0.46 to 0.78)	< 0.001
LA:Ao	VHS_{length}†	0.23 (–0.05 to 0.47)	NS
	VHS†	0.24 (–0.03 to 0.48)	NS
	VLAS†	0.46 (0.21 to 0.65)	< 0.001
	LA_{width}†	0.54 (0.32 to 0.70)	< 0.001
	LA_{total}†	0.47 (0.23 to 0.66)	< 0.001

The Pearson correlation coefficient (*r*) is reported unless otherwise noted.
*Reported as VBUs to the nearest 0.25 vertebral body as measured on left lateral radiographic images of 25 control dogs and 30 case dogs.
†Spearman rank correlation coefficient (ρ) is reported.
NS = Not significant.

Echo unavailable



VHS



3 = VLAS
4 = **LA width**
VLAS + 4 = **LA total**

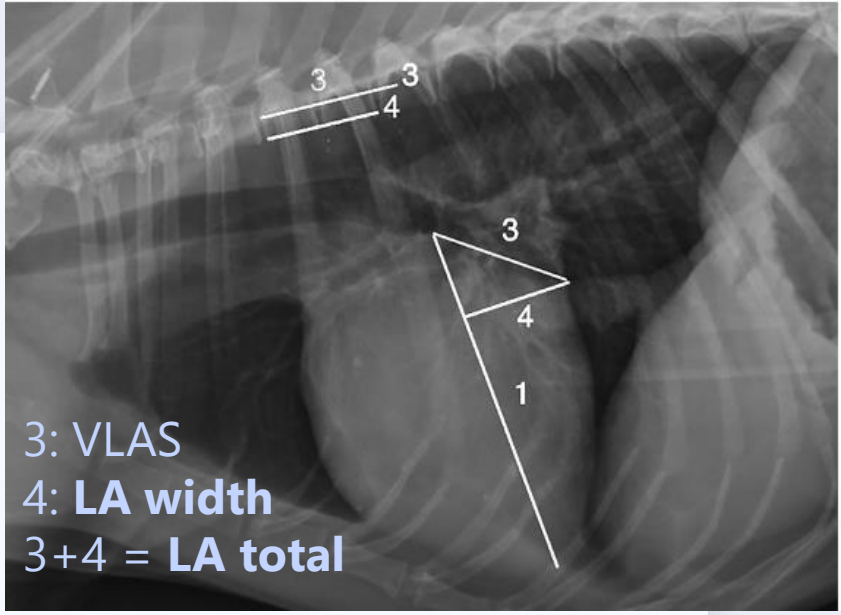
Use of radiographic measurements to diagnose stage B2 preclinical myxomatous mitral valve disease in dogs

Table 3—Results of ROC curve analysis to assess diagnostic accuracy of radiographic variables to predict group status (case group vs control group) as described in Table 1.

Radiographic variable	ROC curve analysis		Cutoff value*		Percentage sensitivity (95% CI)	Percentage specificity (95% CI)	Percentage PPV (95% CI)	Percentage NPV (95% CI)	LR	*P* value
	AUC (95% CI)	*P* value	Type	VBUs						
VHS	0.68 (0.54–0.82)	0.023	Optimal sensitivity†	12.00	37 (22–54)	92 (75–99)	85 (58–97)	55 (40–69)	4.58	0.024
			Maximum specificity‡	12.25	30 (2–48)	96 (80–99)	90 (60–99)	53 (39–67)	7.50	0.016
VLAS	0.79 (0.67–0.91)	< 0.001	Optimal sensitivity†	2.50	70 (52–83)	84 (65–94)	84 (65–94)	70 (52–83)	4.38	< 0.001
			Maximum specificity‡	3.00	40 (25–58)	96 (80–99)	92 (67–99)	57 (42–71)	10.0	0.003
LA_{width}	0.78 (0.66–0.90)	< 0.001	Optimal sensitivity†	2.00	63 (46–78)	76 (57–89)	76 (57–89)	63 (46–78)	2.64	0.006
			Maximum specificity‡	2.25	27 (14–44)	100 (87–100)	100 (68–100)	53 (39–67)	NA	0.006
LA_{total}	0.81 (0.69–0.92)	< 0.001	Optimal sensitivity†	4.50	70 (52–83)	84 (65–94)	84 (65–94)	70 (52–83)	4.38	< 0.001
			Maximum specificity‡	5.00	47 (30–64)	96 (80–99)	93 (70–99)	60 (45–74)	11.67	< 0.001
VHS + VLAS	0.74 (0.66–0.87)	0.003	Optimal sensitivity†	14.50	47 (30–64)	96 (80–99)	94 (70–99)	60 (45–74)	11.67	< 0.001
			Maximum specificity‡	14.75	43 (27–61)	96 (80–99)	93 (69–99)	59 (43–72)	10.83	0.001
VHS + LA_{total}	0.75 (0.62–0.88)	0.002	Optimal sensitivity†	16.25	53 (36–70)	92 (75–99)	89 (67–98)	62 (46–76)	6.67	< 0.001
			Maximum specificity‡	16.50	50 (33–67)	96 (80–99)	94 (72–99)	62 (46–75)	12.5	< 0.001

*Results reported for measurements obtained on left lateral thoracic radiographic images of 25 control dogs and 30 case dogs. †Cutoff value determined with the Youden index. ‡Cutoff value with near 100% specificity (least false-positive categorization).

AUC = Area under the curve. LR = Likelihood ratio.



Three radiographic variables of left atrial size
1.) Vertebral left atrial size [VLAS]
2.) Left atrial width.
3.) Combined variable of VLAS + left atrial width) "LA total" most accurately distinguished control dogs from case dogs, and the **VLAS was the simplest and fastest to perform in a clinical setting**.

CONCLUSIONS AND CLINICAL RELEVANCE

Results indicated that when echocardiography is unavailable, radiographic **VLAS ≥3** VBUs could be used with minimal risk of false-positive diagnosis of stage B2 MMVD in dogs.

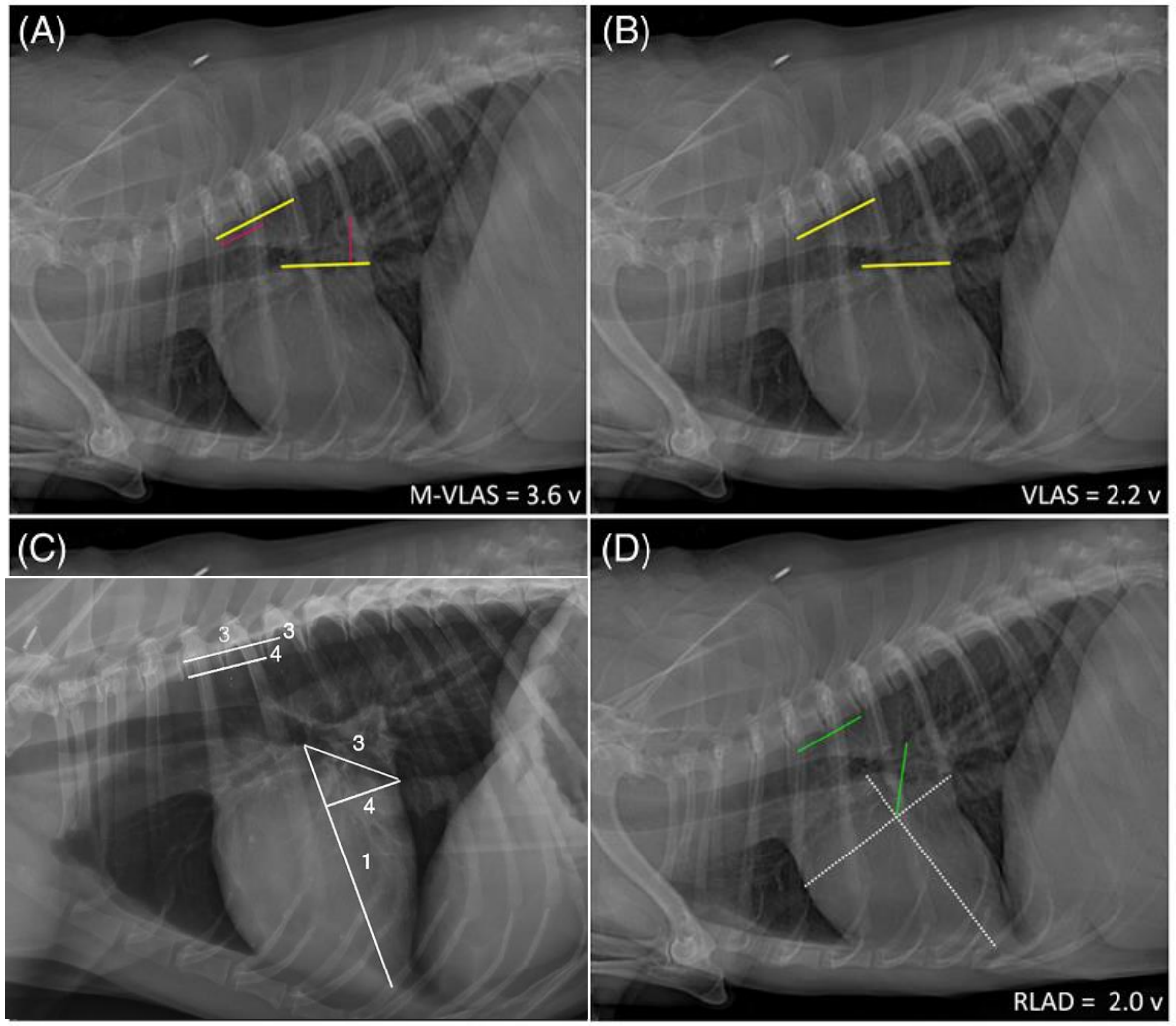
Use of radiographic measurements to diagnose stage B2* / LA

Echo unavailable

M-VLAS
Cutoff: **≥3.4** vertebrae
Sensitivity: 93%
Specificity: 93%

LA width* (4)
Cutoff: **≥2.25** vertebrae
Sensitivity: 27%
Specificity: 100%

LA total* (VLAS(3)+4)
Cutoff: **≥5** vertebrae
Sensitivity: 47%
Specificity: 96%



VLAS*
Cutoff: **≥2.5** vertebrae
Sensitivity: 67%, 70%*
Specificity: 87%, 84%*

Cutoff: **≥3.0** vertebrae
Sensitivity: 40%*
Specificity: 96%*

RLAD
Cutoff: **≥1.8** vertebrae
Sensitivity: 96.8%
Specificity: 93.5%

* = **diagnose stage B2**

MMVD STAGE C

+ Evidence of cardiogenic lung edema on chest-XR

Dyspnea breathing pattern

Obstructive: Deep & slow

- Inspiratory
  - - Stertor/Snoring
  - - Stridor
  - **Dynamic Upper** airway obstruction
    - - Nasal dz.
    - - Pharyngeal dz.
    - - BOAS
    - - Laryngeal dz.
    - - Extra-Tho-TC
- Expiratory
  - **Dynamic Lower** airway obstruction
    - - Intra-Tho-TC.
    - - Bronchial collapse
    - - Asthma
    - - Eosi. Broncho-pneumopathy
- Mixed
  - **Fix** airway obstruction
    - Inlet-TC.
    - Gr.4 TC.
    - Tracheal tumor

Restrictive: short, rapid, shallow

- Adventitious Lung sound
  - Parenchymal disease
    - **- Pneumonia**
    - **- Pulmonary edema**
    - **- Pulmonary fibrosis**
    - **- Infiltrative lung dz.**
    - **- ARDS**
- Quiet or Absent Lung sound
  - Pleural space disease
    - - Pneumothorax: Dorsal
    - - Pleural effusion: Ventral

*Tho: Thoracic
*TC: tracheal collapse
*Dz: disease

CLINICAL SIGNS OF HEART FAILURE

Healthy dog breathing normally at rest

Healthy dog panting

CLINICAL SIGNS OF HEART FAILURE

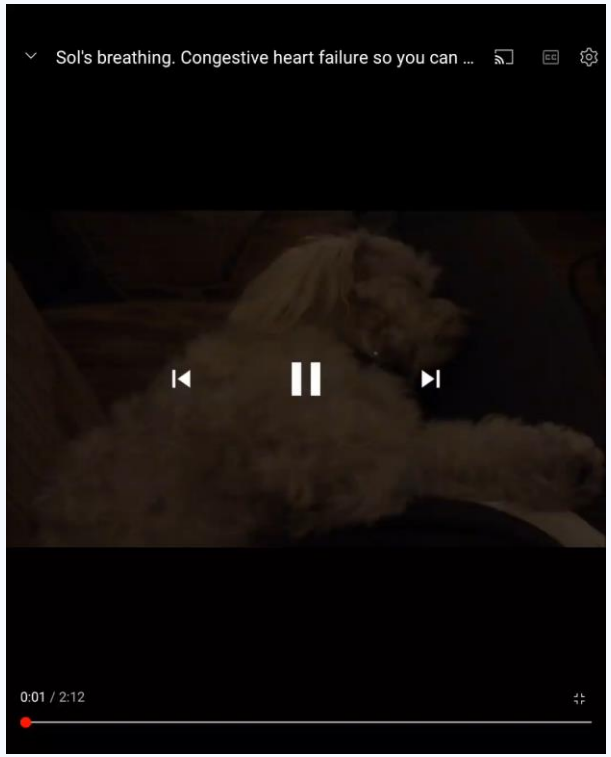
- Early Signs of CHF
  - Exercise intolerance
  - Rapid SRR (> 30 tpm)
  - Tachycardia
  - +/- Coughing (MSB. compression)

- Late Signs of CHF
  - Rapid breathing (RRR >80 tpm)
  - Tachycardia
  - Coughing up pink foam (severe pulmonary edema)

Postures

- Exaggerated chest and abdominal motion to respiration
- Extended head and neck
- Stand with adducted elbow
- Open mouth breathing / flaring nostrils
- Change in gum and/or tongue color to a bluish gray (poor oxygen flow)
- Minimize extraneous activities or action that exacerbate airway narrowing (swallow, bark)

CLINICAL SIGNS OF HEART FAILURE



Rapid SRR

Video of a dog with mild dyspnea

CLINICAL SIGNS OF HEART FAILURE

Chihuahua showing mild to moderate difficulty breathing (dyspnea)

CLINICAL SIGNS OF HEART FAILURE

Video of a dog with marked dyspnea

CLINICAL SIGNS OF HEART FAILURE

Dogs with heart failure often have difficulty breathing combined with cough

CLINICAL SIGNS OF HEART FAILURE

Problem	Location
Sneezing/discharge	Nasal cavity/oropharynx
Coughing with minimal dyspnea	Tracheobronchial
Dyspnea with minimal coughing	Laryngeal/bronchial/lung/pleural/ mediastinum
Coughing and dyspnea	brochoalveolar

Recognition of CHF. in MMVD

- Older than 7 yrs and < 15 kg
- Loud murmur (IV/VI or >)
- sinus arrhythmia absent ----(respi > cardiac)
- Tachycardia (HR >160 bpm)
- Dyspnea (sleeping RR > 30)

"The most common clinical sign of congestive heart failure is persistent coughing accompanied by difficulty breathing."

Cough on its own is not considered a sign of congestive heart failure

* Beijerink, Campbell, Gavaghan, Singh and Wooley. Published online via Vetforum, Boehringer Ingelheim, 2015.

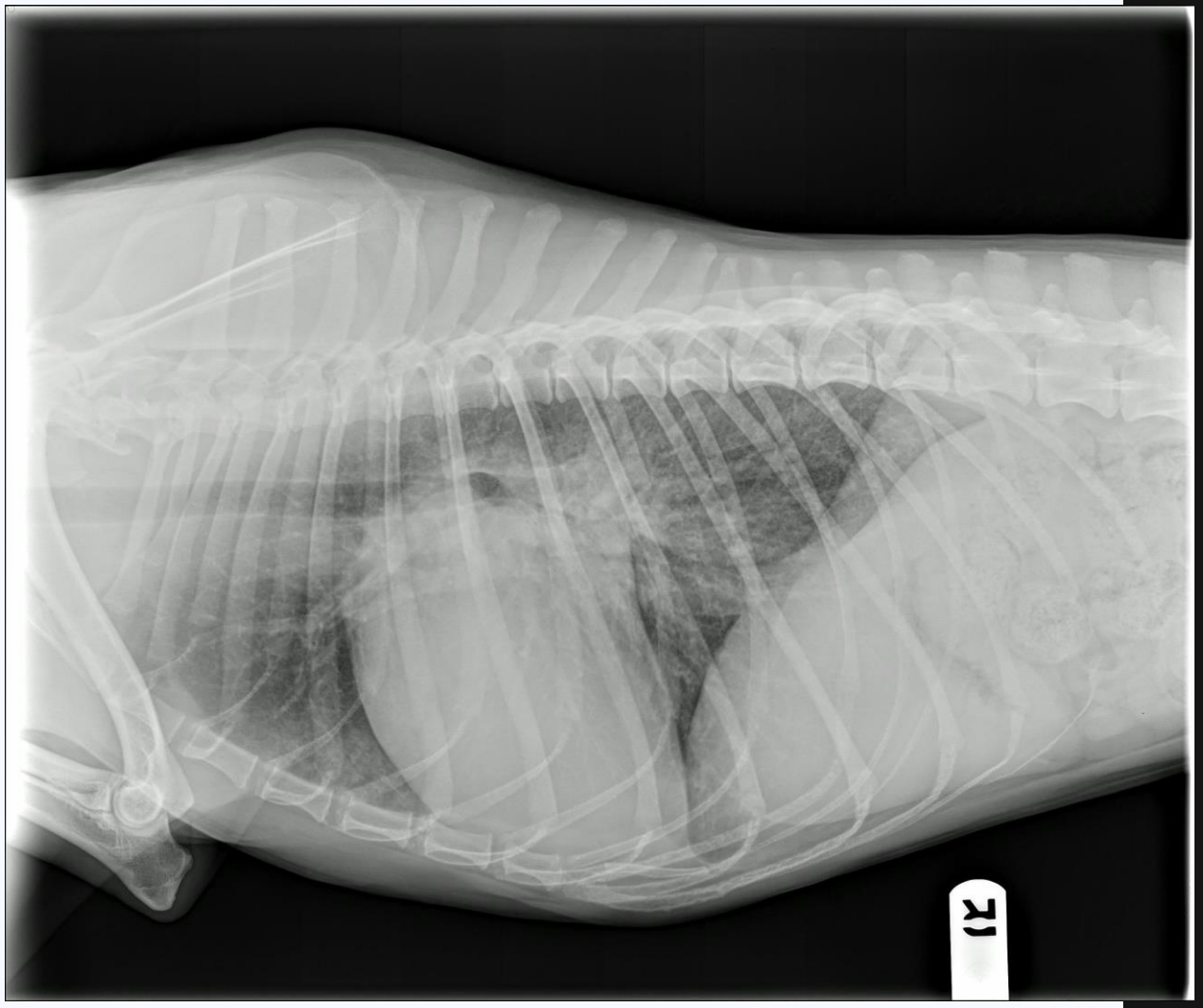
EVIDENCE OF CARDIOGENIC PULMONARY EDEMA

- Significant left-sided heart enlargement manifested as:
  – Increased VHS, VLAS, M-VLAS
  – Increased height of the cardiac silhouette / loss of the caudal cardiac waist on lateral projection
  – Left auricular enlargement at the 2- to 3-o'clock position on the DV or VD projection

- Caudal mainstem bronchi were compressed on lateral projections 2nd to the cardiomegaly and left atrial enlargement.

- Enlarged pulmonary veins
- Perihilar and/or caudodorsal unstructured interstitial or alveolar pattern

Sequence of pulmonary edema: Perihilar → Cd-D (R then L) → Cr-V Interstitial → Alveolar pattern



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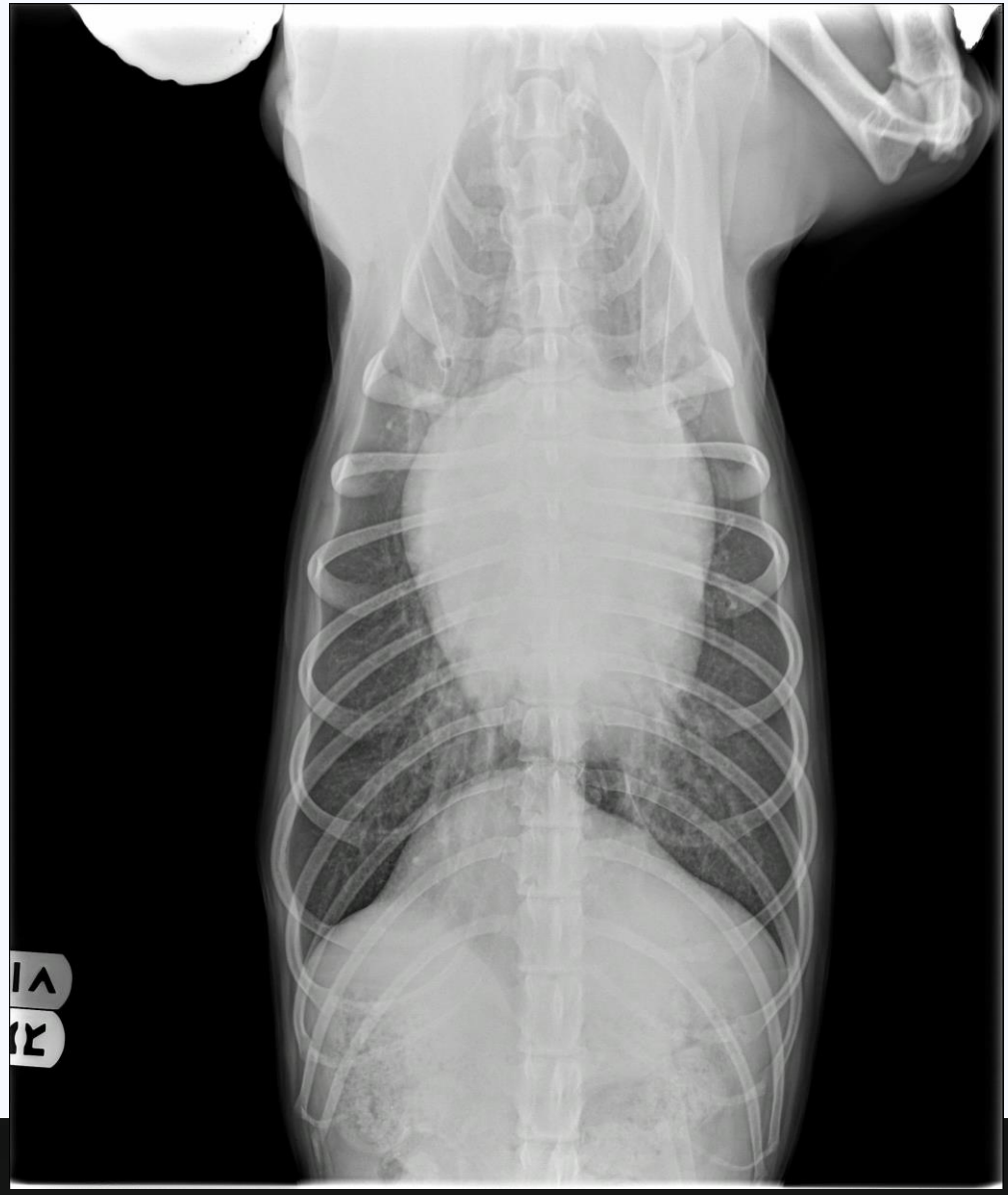
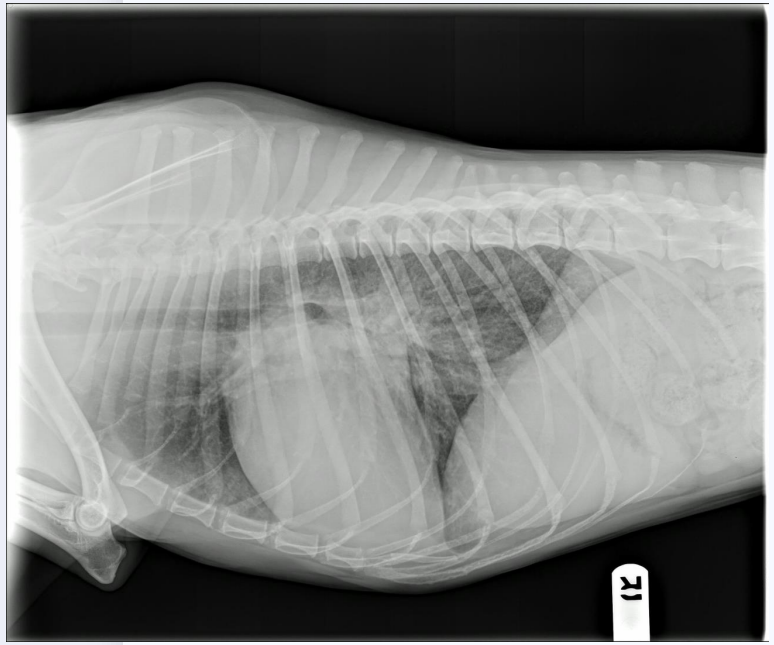
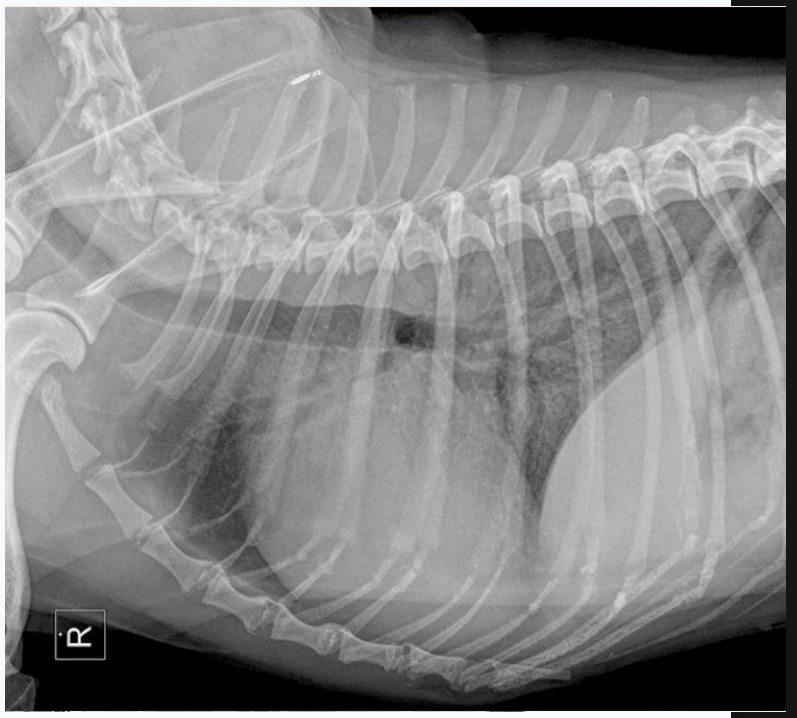
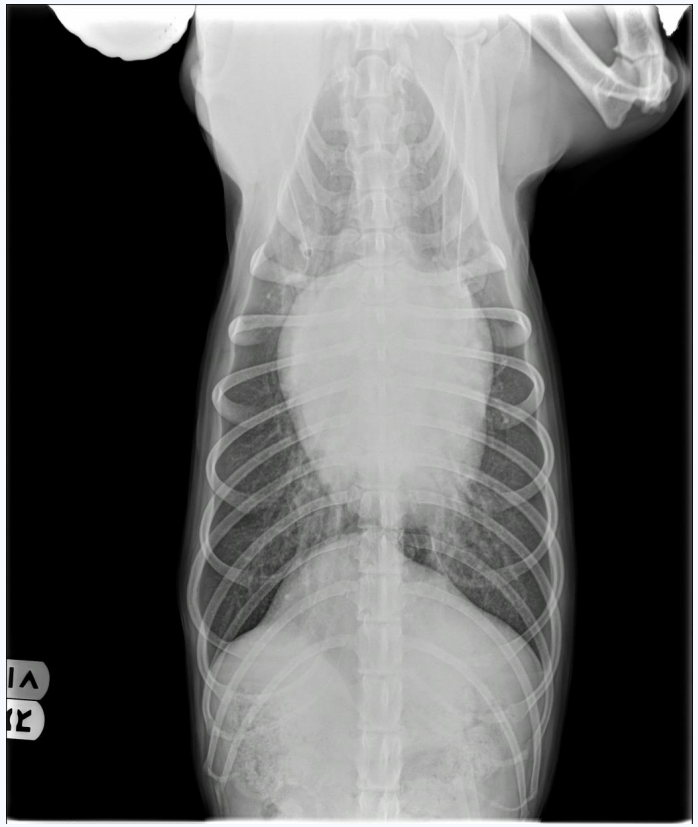


EVIDENCE OF CARDIOGENIC PULMONARY EDEMA





EVIDENCE OF CARDIOGENIC PULMONARY EDEMA



EVIDENCE OF CARDIOGENIC PULMONARY EDEMA

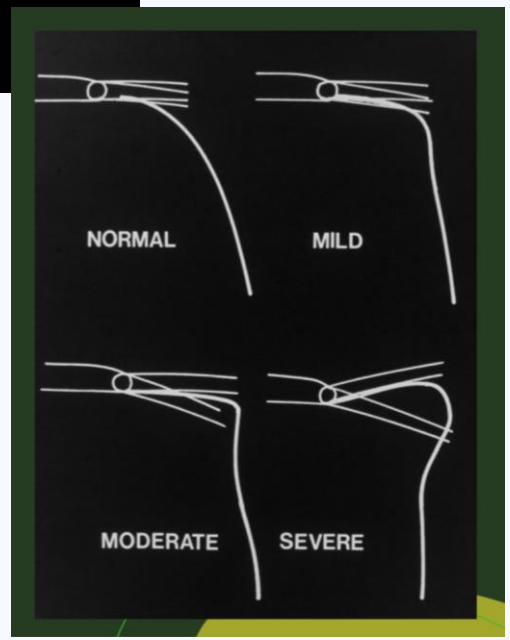
J Vet Intern Med 2010;24:1358–1368

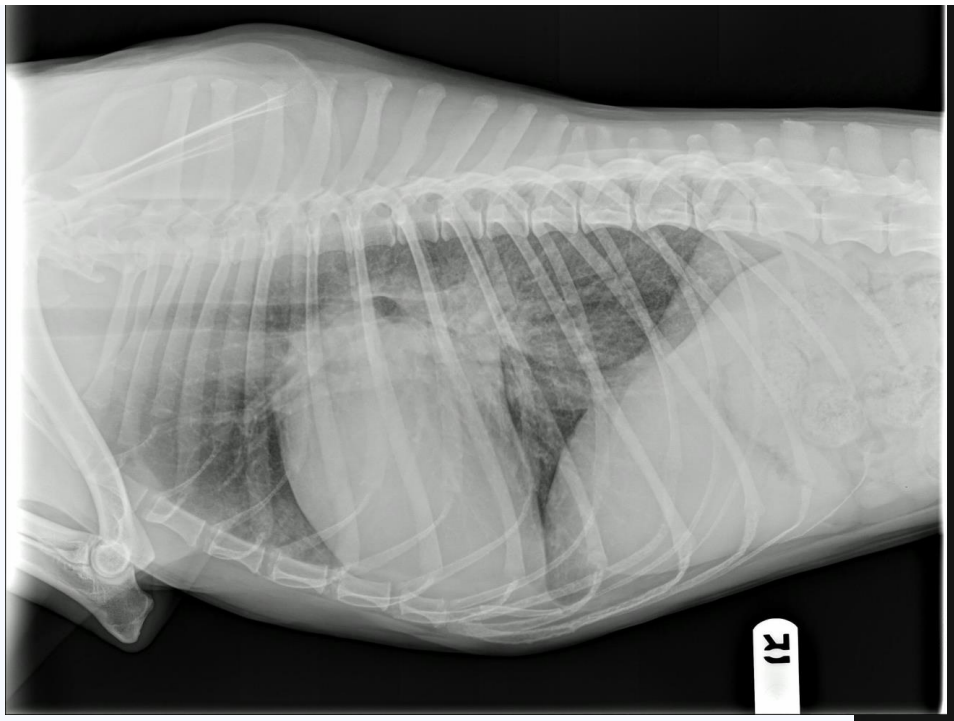
Detection of Congestive Heart Failure in Dogs by Doppler Echocardiography

K.E. Schober, T.M. Hart, J.A. Stern, X. Li, V.F. Samii, L.J. Zekas, B.A. Scansen, and J.D. Bonagura

Appendix 1. Radiographic composite score on congestive heart failure (CHF).

Variable	Assessment	Points
Left atrial enlargement	None	0
	Mild	1
	Moderate to severe	3
Pulmonary venous congestion	None	0
	Present	3
Pulmonary infiltrates compatible with cardiogenic edema	None	0
	Mild interstitial	1
	Diffuse interstitial	2
	Alveolar	3
Pleural effusion	None	0
	Yes	1
Final assessment	Score 0–2	CHF not likely
	Score 3–4	CHF possible
	Score >4	CHF likely





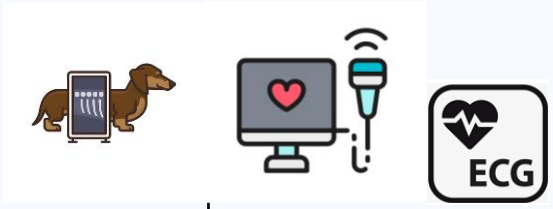
Score 3 + 3 + 3 = 9 (Left CHF. likely)

Sequence of pulmonary edema: Perihilar → Cd-D (R then L) → Cr-V
Interstitial → Alveolar pattern

Aging small to medium-sized breeds

Dog Suspected to Have MMVD†
- Left Apical Systolic Heart Murmur‡
(Moderate to loud murmur — at least grade 3/6)

No clinical signs of heart failure

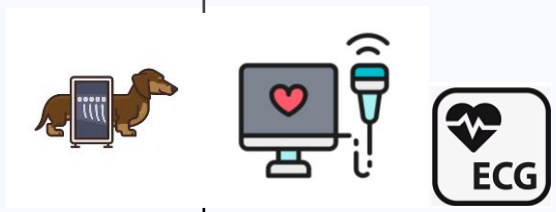


Evidence Lt. side heart enlargement

NO → Stage B1

YES → Stage B2

Clinical signs of heart failure



Evidence Lt. side heart enlargement & cardiogenic lung edema

→ Stage C

OUTLINE

2. Management.

- Pre-clinical (heart disease)
- Clinical (heart failure)
- Applying "**evidence-based medicine**"

Classes of recommendation	
CLASS I	BENEFIT>>>RISK
CLASS IIA	BENEFIT>>RISK
CLASS IIB	BENEFIT>RISK
CLASS III	BENEFIT=RISK
CLASS IV	RISK>>BENEFIT

Levels of Evidence (LOE)	
STRONG	The highest level of evidence based on high quality studies generating a clear and statistically valid result.
MODERATE	This level of evidence is based on well designed, controlled studies for interpretation by the reader.
WEAK	Based on quality studies which leave room for observation and discussion.
EXPERT OPINION	Considered the weakest LOE based on experience of the panel.

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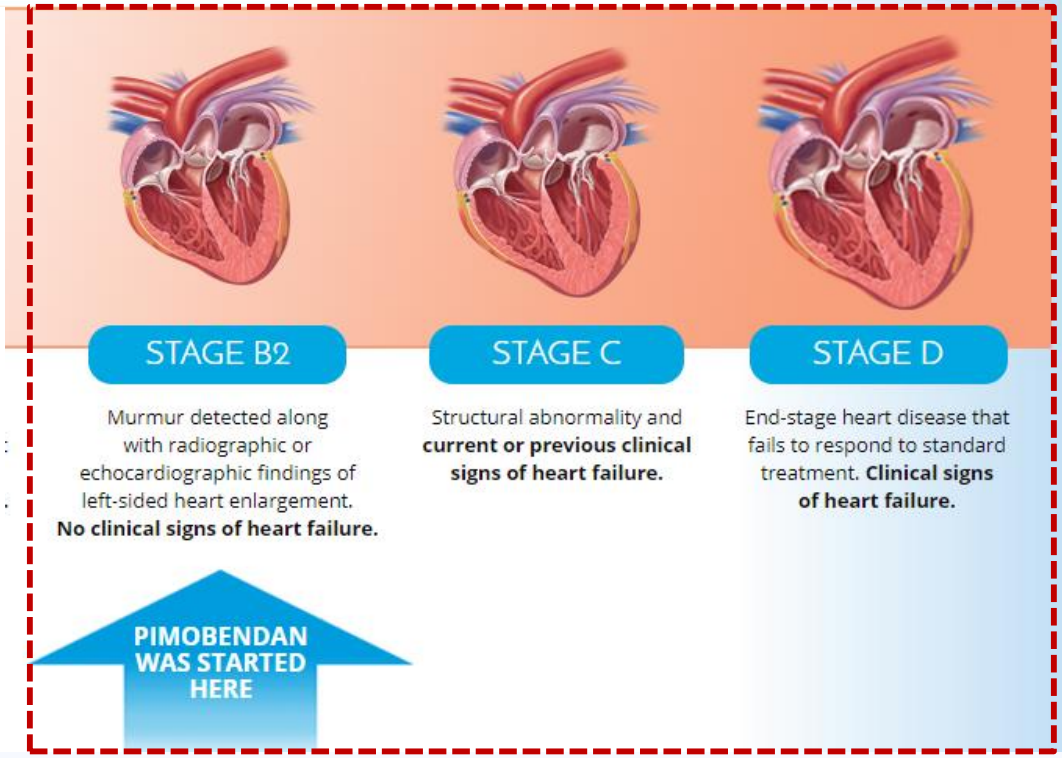
CONSENSUS STATEMENT

Journal of Veterinary Internal Medicine Open Access
ACVIM American College of Veterinary Internal Medicine

Consensus Statements of the American College of Veterinary Internal Medicine (ACVIM) provide the veterinary community with up-to-date information on the pathophysiology, diagnosis, and treatment of clinically important animal diseases. The ACVIM Board of Regents oversees selection of relevant topics, identification of panel members with the expertise to draft the statements, and other aspects of assuring the integrity of the process. The statements are derived from evidence-based medicine whenever possible and the panel offers interpretive comments when such evidence is inadequate or contradictory. A draft is prepared by the panel, followed by solicitation of input by the ACVIM membership which may be incorporated into the statement. It is then submitted to the *Journal of Veterinary Internal Medicine*, where it is edited prior to publication. The authors are solely responsible for the content of the statements.

ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

Bruce W. Keene[1] | Clarke E. Atkins[1] | John D. Bonagura[1,2] | Philip R. Fox[3] | Jens Häggström[4] | Virginia Luis Fuentes[5] | Mark A. Oyama[6] | John E. Rush[7] | Rebecca Stepien[8] | Masami Uechi[9]



The **2009 guidelines** from (ACVIM) : **recommend that treatment of heart failure begin when the dog shows clear clinical signs of disease (stage C in the visual shown).**

The **2019 guidelines** from (ACVIM) : administered to dogs in **stage B2—*before* clinical signs of heart failure appear**

Stage B2	Asymptomatic MMVD causing MR severe enough to result in cardiac remodeling, dogs in this category should meet the following criteria:		
RECOMMENDATIONS FOR DIAGNOSES	- Murmur intensity >3/6; - Echocardiographic LA : Ao ratio in the right-sided short axis view in early diastole >1.6 - Left ventricular internal diameter in diastole, normalized for body weight (LVIDDN) >1.7 - Breed-adjusted radiographic vertebral heart score (VHS) >10.5 Ideally, all of these criteria should be met. However, in the absence of echocardiographic measurements, clear radiographic evidence of cardiomegaly (VHS > 11.5) or evidence of increasing interval change can be used to identify Stage B2.	CLASS I	LOE: STRONG
	VLAS values of >3 likely identify Stage B2 MMVD.	CLASS I	LOE: MODERATE
RECOMMENDATIONS FOR TREATMENT	Pimobendan at a dosage of 0.25-0.3 mg/kg PO q12h.	CLASS I	LOE: STRONG
	Surgical intervention in advanced Stage B2 is possible and recommended by some panelists.	CLASS IIA	LOE: MODERATE
	Dietary treatment.	CLASS IIA	LOE: WEAK
	For patients in stage B2 on either initial examination, 5 (of 10) panelists recommend treatment with ACEI.	CLASS IIA	LOE: WEAK

Journal of Veterinary Internal Medicine
Open Access
ACVIM
Standard Article
J Vet Intern Med 2016;30:1765–1779

Effect of Pimobendan in Dogs with Preclinical Myxomatous Mitral Valve Disease and Cardiomegaly: The EPIC Study—A Randomized Clinical Trial

A. Boswood, J. Häggström, S.G. Gordon, G. Wess, R.L. Stepien, M.A. Oyama, B.W. Keene, J. Bonagura, K.A. MacDonald, M. Patteson, S. Smith, P.R. Fox, K. Sanderson, R. Woolley, V. Szatmári, P. Menaut, W.M. Church, M. L. O'Sullivan, J.-P. Jaudon, J.-G. Kresken, J. Rush, K.A. Barrett, S.L. Rosenthal, A.B. Saunders, I. Ljungvall, M. Deinert, E. Bomassi, A.H. Estrada, M.J. Fernandez Del Palacio, N.S. Moise, J.A. Abbott, Y. Fujii, A. Spier, M.W. Luethy, R.A. Santilli, M. Uechi, A. Tidholm, and P. Watson

STUDY DESIGN

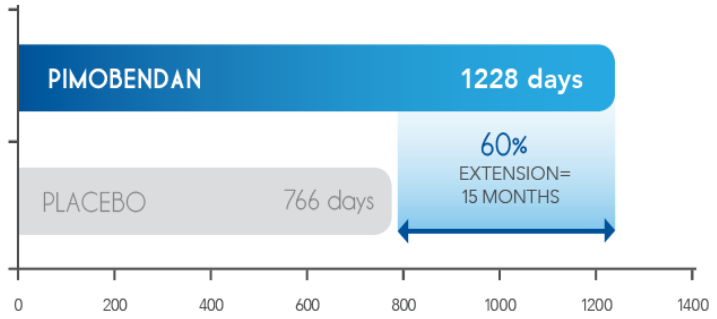
- 36 CENTRES
- 360 DOGS
- DOUBLE-BLINDED PLACEBO-CONTROLLED
- 11 COUNTRIES
- 5+ YEARS

FINDINGS

- 15-MONTH DELAY in time to composite primary endpoint compared with dogs receiving placebo*
- 60% MORE TIME in asymptomatic stage B2 of heart disease*
- 10% MORE LIFE without CHF signs that impact quality of life or cardiac-related death†

RESULTS REVEAL THE PROVEN BENEFITS OF EARLY TREATMENT OF MMVD–BEFORE THE ONSET OF CHF[1]

Dogs who received pimobendan experienced a 15-month delay in onset of clinical signs of CHF, cardiac-related death, or euthanasia versus dogs in the placebo group.



Median time to onset of CHF or cardiac-related death (days)

At 1228 days, there were more than twice as many dogs in the pimobendan group that were still alive and had not reached the primary endpoint versus the placebo group.*

Journal of Veterinary Internal Medicine
ACVIM
Open Access

Standard Article
J Vet Intern Med 2018;32:72–85

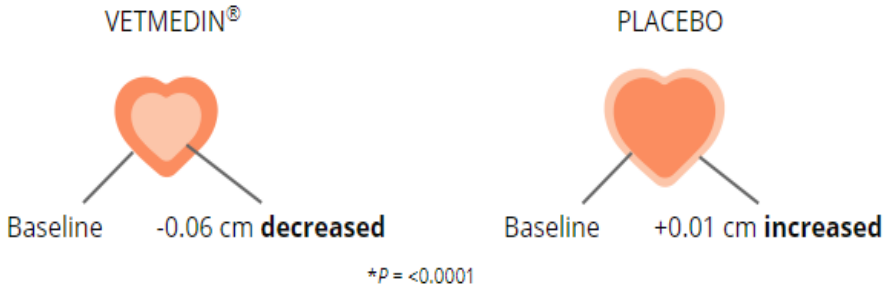
Longitudinal Analysis of Quality of Life, Clinical, Radiographic, Echocardiographic, and Laboratory Variables in Dogs with Preclinical Myxomatous Mitral Valve Disease Receiving Pimobendan or Placebo: The EPIC Study

A. Boswood, S.G. Gordon, J. Häggström, G. Wess, R.L. Stepien, M.A. Oyama, B.W. Keene, J. Bonagura, K.A. MacDonald, M. Patteson, S. Smith, P.R. Fox, K. Sanderson, R. Woolley, V. Szatmári, P. Menaut, W.M. Church, M.L. O'Sullivan, J.-P. Jaudon, J.-G. Kresken, J. Rush, K.A. Barrett, S.L. Rosenthal, A.B. Saunders, I. Ljungvall, M. Deinert, E. Bomassi, A.H. Estrada, M.J. Fernandez Del Palacio, N.S. Moise, J.A. Abbott, Y. Fujii, A. Spier, M.W. Luethy, R.A. Santilli, M. Uechi, A. Tidholm, C. Schummer, and P. Watson

FINDINGS

HEART SIZE ON DAY 35—LVIDDN Measurement

(Normalised left ventricular internal diameter in diastole)



Absolute Change From Baseline Median (Average)

STUDY CONCLUSION

The 2nd EPIC Study publication findings further highlight the benefit of a proactive approach to preclinical MMVD. Results continue to support the case for early treatment of MMVD during Stage B2 to positively impact the life of dogs with MMVD.[1]

1-MONTH IMPACT

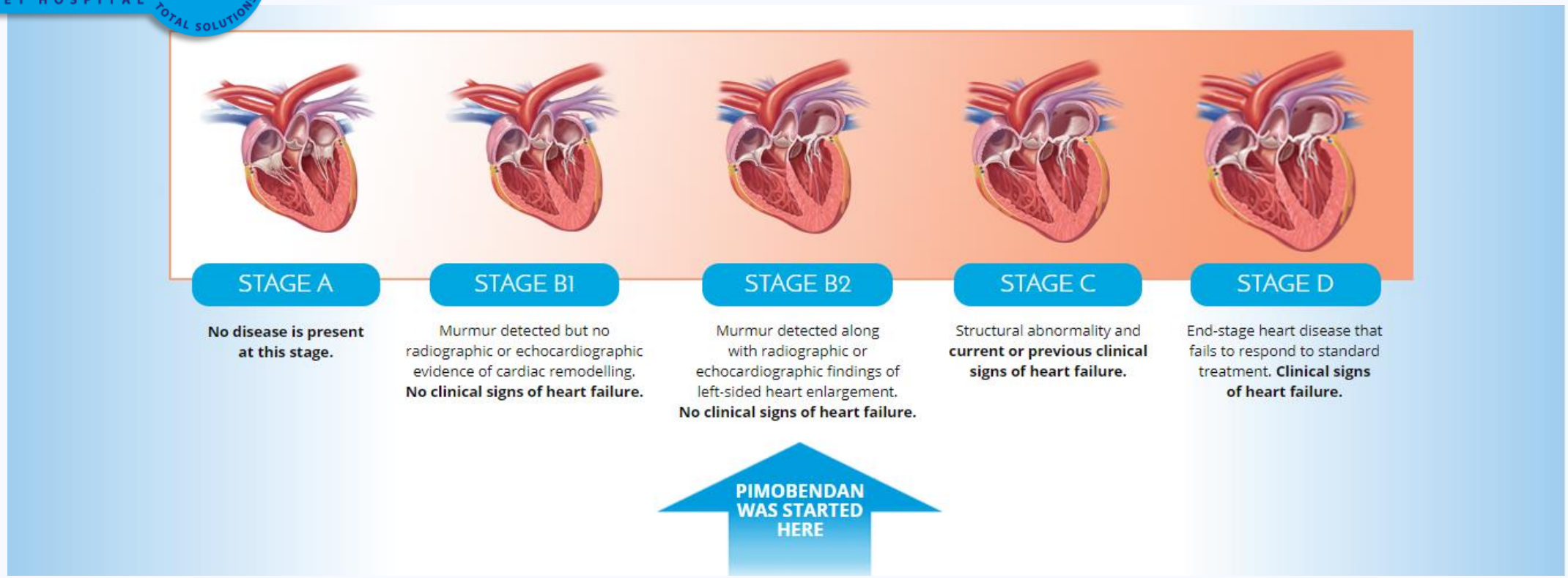
Within the first month of pimobendan treatment, dogs demonstrated significant decrease in heart size.

REDUCTION IN HEART SIZE

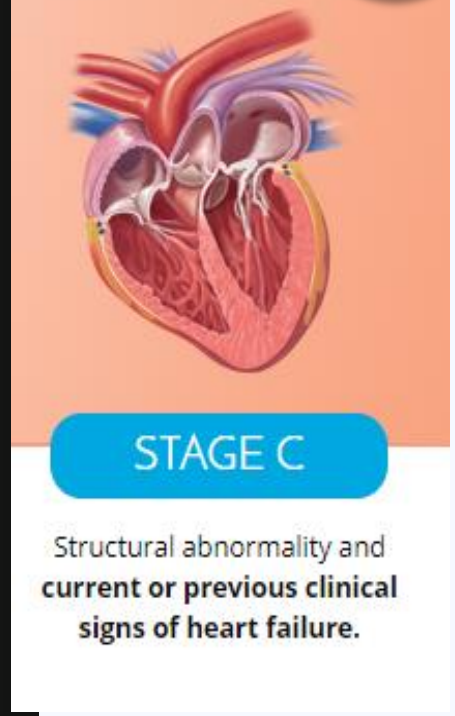
Observed heart size reduction was maintained over study duration, with dogs treated with pimobendan having a smaller average heart size.

MORE TIME

In asymptomatic Stage B2 of heart disease*



- 15 months delay(top up): significantly extend the symptom-free period of those dogs' lives by an average of 15 mth.
- Reduction in heart size (1 month impact)
- Improve quality of life and extend life
- Reevaluation by echocardiography in 4-6 months (May be more frequent in large dogs)



CHF - ER Management

Goals

- **Reduce venous congestion, edema, and effusion formation**

Reduce vascular volume (preload)

- Diuretic: Furosemide

Reduce venous tone (vasodilators - increase venous capacitance)

- Nitroglycerine (Nitroderm patch)
- Morphine (reduce stress and/or improve breathing dynamics.)

- **Increase contractility and CO**
  - Pimobendan PO, IV (Decrease Afterload: " Inodilator ")
  - Dopamine CRI
  - Dobutamine CRI

- **Normalize HR and rhythm**
  - Antiarrhythmic drugs

- Address forward failure signs (hypotension, hypothermia)

Cardiac cachexia : 60kcl/kg BW, Omega-3fatty acid

STAGE C

Structural abnormality and **current or previous clinical signs of heart failure.**

Acute Fulminant Pulmonary edema

+ Handle gently

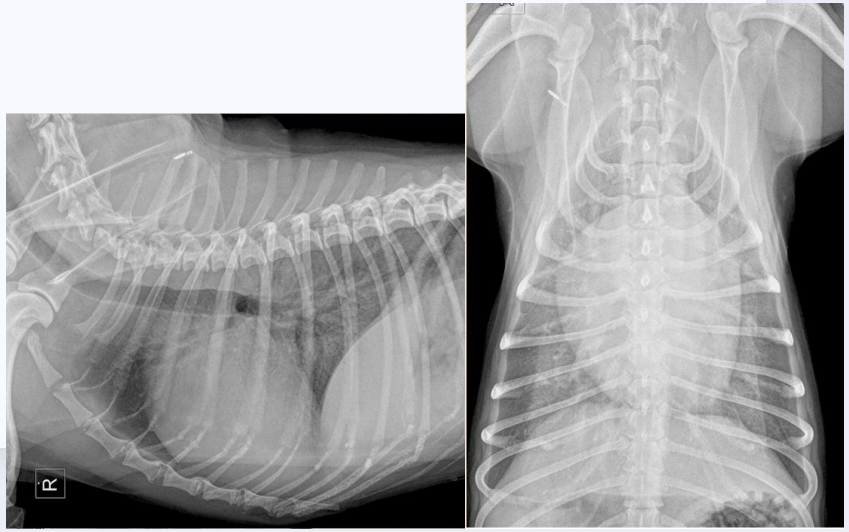
+ **Run a full blood test** to assess for concurrent organ damage due to reduced cardiac output.

+ Delay radiograph / Echo.

+ Anxiolytics agent: Butorphanol 0.2 - 0.25 mg/kg **<u>Good choices in patient with cardiac dz.</u>**

Acepromazine 0.01 - 0.03 mg/kg PO

Morphine 0.1 – 0.3 mg/kg SC



Acute (Fulminant) Pulmonary edema

STAGE C

Structural abnormality and **current or previous clinical signs of heart failure.**

Medical emergency

- **Anxiolytics: Morphine** low dose (0.025 - 0.05 mg/kg IV) morphine dilates the splanchnic vasculature and increases venous capacitance. Additional benefits of allowing slower, deeper respirations, and decreasing anxiety in patients with CHF. *** Side effect = VOMITING ***
- **O2 therapy**

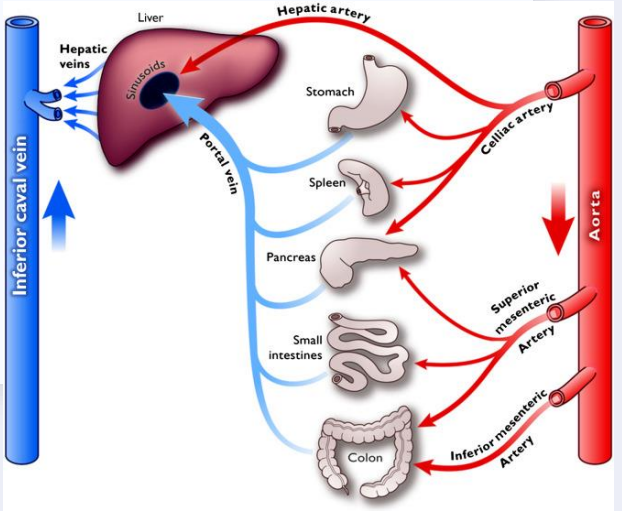
- **Diuretic: Furosemide** 4-6 mg/kg IV or IM q2hrs (or 2mg/kg q1hr) until see reduction in RR & effort
  - Offer water all time once diuresis begins
  - Continue high doses once see a 30% reduction in RR & effort → reduce dose (aim for <25 bpm whilst sleeping)
  - If RR not improving to <40-50/min within 2-3 hours (ie. 2-3 doses) switch to CRI or incorrect Dx?

*Onset 30-45min(Strickland, 2016) , plasma half-life 1-2 hr.

*Spacing of each dosing is too long --> inadequate efficacy

*Efficacy: CRI > intermittent bolus (Ohad, 2018)

- **Pimobendan** 0.25 – 0.3 mg/kg q12hr / q8hr PO, SID IV
- **N**itroglycerine (Topical-venodilator): Nitroderm patch

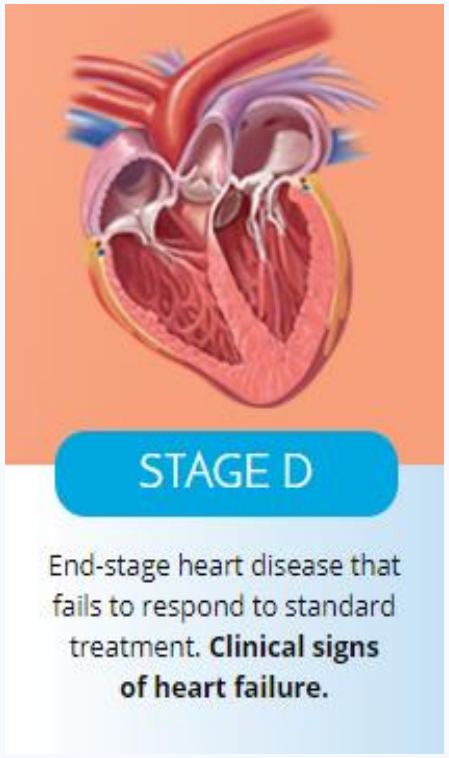


Anxiolytics agent : Butorphanol (0.2-0.25 mg/kg) or acepromazine (0.01-0.03 mg/kg)

An ACE inhibitor and spironolactone can be added at a later time.

Stage C	Denotes dogs with either current or past clinical signs of heart failure caused by MMVD.			
RECOMMENDATIONS FOR DIAGNOSES		Analyze serum NT-proBNP concentration.	CLASS I	LOE: MODERATE
		Obtain basic laboratory tests.	CLASS I	LOE: EXPERT OPINION
		Most symptomatic dogs with MMVD are middle-aged or older, and it is prudent to complete the clinical database.	CLASS I	LOE: EXPERT OPINION
		Complete clinical database (including thoracic radiographs and ideally echocardiogram).	CLASS I	LOE: EXPERT OPINION
RECOMMENDATIONS FOR TREATMENT +	HOSPITAL BASED ACUTE	Torasemide at approximately 5% to 10% of the furosemide dosage (0.1-0.3 mg/kg q24h7).	CLASS I	LOE: MODERATE
		Pimobendan, 0.25-0.3 mg/kg administered PO q12h.	CLASS I	LOE: WEAK
		ACEI (0.5 mg/kg PO q12h).	CLASS IIB	LOE: WEAK
		Furosemide 2 mg/kg IV (or IM) followed by 2 mg/kg IV (or IM) hourly until patients respiratory signs are substantially improved.	CLASS I	LOE: EXPERT OPINION
		Oxygen supplementation, if needed, can be administered.	CLASS I	LOE: EXPERT OPINION
		Furosemide CRI (0.66-1 mg/kg/hour) for life-threatening pulmonary edema.	CLASS IIA	LOE: EXPERT OPINION

Stage C	Denotes dogs with either current or past clinical signs of heart failure caused by MMVD.		
CHRONIC (HOME BASED)	Continue pimobendan, 0.25-0.3 mg/kg PO q12h.	CLASS I	LOE: STRONG
	Continue PO furosemide (2mg/kg q12h).	CLASS I	LOE: MODERATE
	Spironolactone (2.0 mg/kg PO q12 - 24 h).	CLASS I	LOE: MODERATE
	Diltiazem (often combined with digoxin) in cases of complicated atrial fibrillation.	CLASS I	LOE: MODERATE
Chronic	Dietary recommendations: - Ensure adequate protein intake - Modestly restrict sodium intake - Omega-3-fatty acids	CLASS I	LOE: MODERATE
	In centers with low complication rates, patients benefit from surgical intervention to repair their mitral valve apparatus.	CLASS I	LOE: MODERATE
	Continue or start ACEI (0.5 mg/kg PO q12h).	CLASS IIB	LOE: WEAK
	None of the panelists routinely use nitroglycerin.	CLASS III	LOE: EXPERT OPINION



Failure refractory to standard treatment

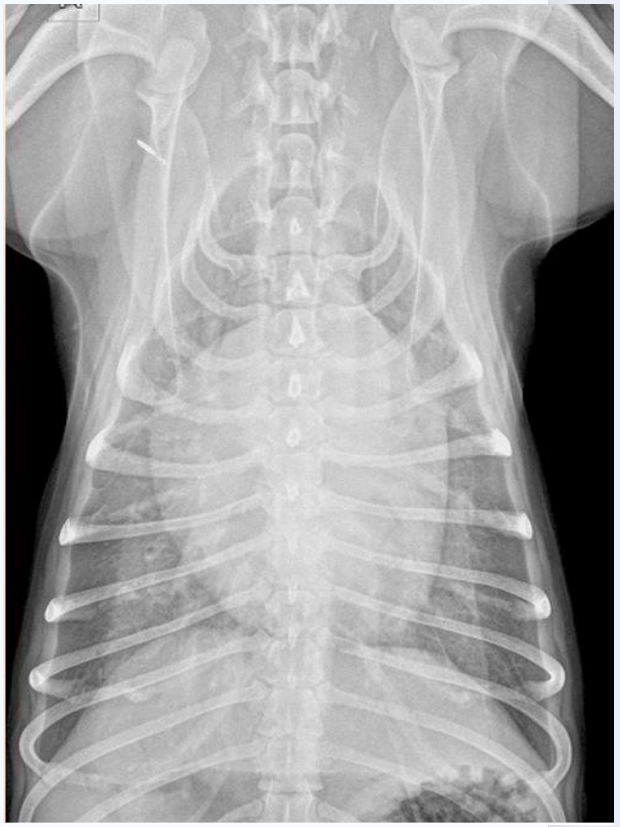
Meds

-> more than 8mg/kg/day of furosemide with

+ Pimobendan 0.3mg/kg q12hr -> q8hr
+ Benazepril 0.25mg/kg q24hr
+ Spironolactone 2mg/kg q12hr

Echo

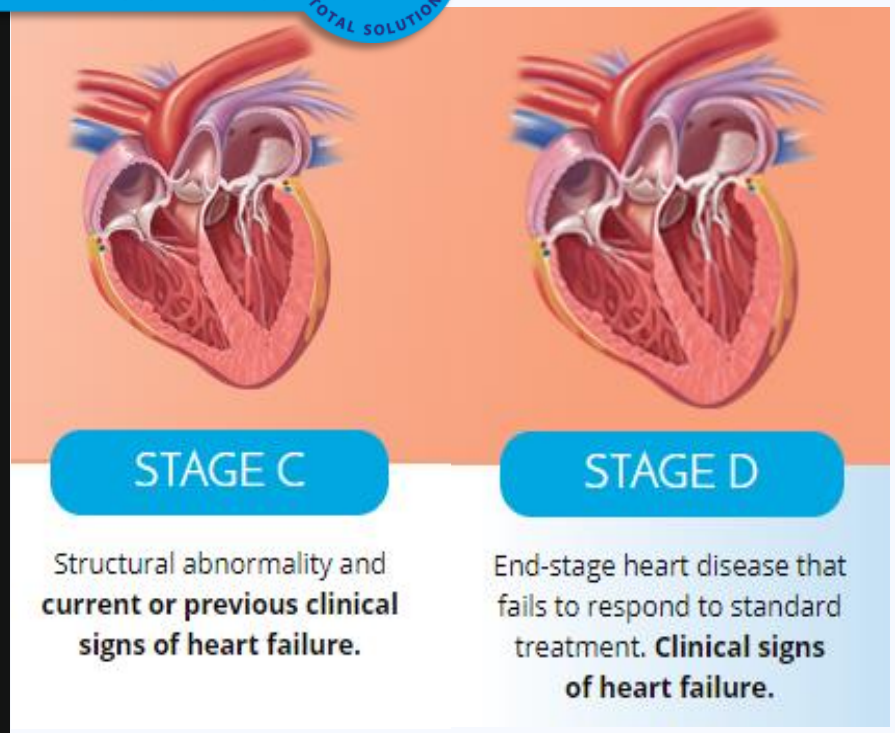
-> LV systolic dysfunction.



THONGLOR PET HOSPITAL TOTAL SOLUTIONS

Stage D	Refers to dogs with end-stage MMVD, in which clinical signs of heart failure are refractory to standard treatment		
RECOMMENDATIONS FOR DIAGNOSES	CHF involves the same diagnostic steps outlined for Stage C plus the finding of failure to respond to treatments outlined in the Stage C guidelines. Chronic PO furosemide dosages -8 mg/kg q24h in any dosing regimen to maintain patient comfort in the face of appropriate dosages of pimobendan, an ACEI, and spironolactone indicate disease progression to Stage D.		
RECOMMENDATIONS FOR TREATMENT	**HOSPITAL BASED**	Pimobendan dosage may be increased (off-label use) to include a third 0.3 mg/kg daily PO dose.	**CLASS IIA** / **LOE:EXPERT OPINION**
	CHRONIC HOME-BASED TREATMENT	Torsemide, a potent and longer-acting loop diuretic, may be used to treat dogs no longer adequately responsive to furosemide.	**CLASS I** / **LOE: MODERATE**
		Furosemide (or torsemide) dosage should be increased as needed to decrease the accumulation of pulmonary edema or body cavity effusions.	**CLASS IIA** / **LOE:EXPERT OPINION**
		Pimobendan dosage is increased by some panelists to include a third 0.3 mg/kg daily dose.	**CLASS IIA** / **LOE:EXPERT OPINION**
		Beta blockers generally should not be initiated at this stage.	**CLASS IV** / **LOE:EXPERT OPINION**
		Dietary requirements: All of the dietary considerations for Stage C (above) apply.	

ยา	กลุ่ม	ขนาดและวิธีการบริหารยาที่แนะนำ
Benazepril	ACE inhibitor	0.25 – 0.5 mg/kg PO q (12)-24h
Enalapril	ACE inhibitor	0.5 mg/kg PO q12-24h
Furosemide	Loop diuretic	*Acute CHF*: Initial bolus, 2 mg/kg IV, IM, or SC Repeat at 1 - 4 mg/kg q1-4h until RR↓, then q6-12h Or, 0.66 – 1 mg/kg/h CRI for (6-12)h *Maintenance*: (0.5-)1-3 mg/kg PO q8-24h Titrate to lowest effective dosage for chronic therapy
Hydrochlorothiazide	Thiazide diuretic	0.5 – 4 mg/kg PO q12-48h
Imidapril	ACE inhibitor	0.25 mg/kg PO q24h
Pimobendan	Positive inotrope / Inodilator	0.2 – 0.3 mg/kg PO q(8-)12h
Ramipril	ACE inhibitor	0.125 - 0.25 mg/kg PO q24h
Spironolactone	Potassium sparing diuretic	0.5 – 2 mg/kg PO q24h (or, divided, q12h)
Torsemide	Loop diuretic	Calculate 1/8 to 1/12 of patient's total daily furosemide dose & give as 2 divided doses



Complications of heart failure

+ Kidney damage or failure : "CvRD-H"
+ Liver damage. Heart failure can lead to a buildup of fluid that puts too much pressure on the liver. This fluid backup can lead to scarring

++ Pulmonary hypertension
> Common in stage C > B2 (Borgarelli et al, 2015)
> Prevalence of PH in dogs with MMVD between 14 and 53%.
> Prevalence of PH was associated with severity of MMVD
> Humans and animals with more severe mitral regurgitation, and therefore higher left-atrial pressure, have an increased risk of developing PH.
> Poorer prognosis with PH. >55 mmHg.

Journal of Veterinary Internal Medicine Open Access

J Vet Intern Med 2015;29:569–574

Prevalence and Prognostic Importance of Pulmonary Hypertension in Dogs with Myxomatous Mitral Valve Disease

M. Borgarelli, J. Abbott, L. Braz-Ruivo, D. Chiavegato, S. Crosara, K. Lamb, I. Ljungvall, M. Poggi, R.A. Santilli, and J. Haggstrom

+ Arrhythmias: AF, VT, VF

DEGENERATIVE MITRAL VALVE DISEASE

PROGNOSIS

+ MMVD B2 may live 2 years or more -> C

+ **MMVD with CHF** survival times can generally be between 12 - 24 months at time of diagnosis
(Some shorter, some longer) related to adequate care, treatment, complication (Lefborn et al, 2016).

+ **Decreased survival time** in dogs with
- Severe MR / chordal rupture
- Syncope
- Tachycardia
- PAH (moderate to severe); PAP >55mmHg
- Severe increased LA size

DEGENERATIVE MITRAL VALVE DISEASE

TABLE 1 Selected echocardiographic cutoffs and relative scores

	Score			
	1	2	3	4
LA/Ao	<1.70	1.70-1.90	1.91-2.50	>2.50
LVIDDn	<1.70	1.70-2.00	2.10-2.30	>2.30
FS (%)	<45	45-50	>50	
E-vel (m/s)	<1.20	1.20-1.50	>1.50	

Abbreviations: E-vel, E-wave transmitral peak velocity; FS, fractional shortening of the left ventricle; LA/Ao, left atrium-to-aorta ratio; LVIDDn, left ventricular end-diastolic diameter normalized for body weight; .

TABLE 2 Severity classification based on the total score obtained from the summation of the single scores obtained with Table 1

Severity classification	Total score
Mild	4-5
Moderate	6-7
Severe	8-12
Late stage	13-14

PROGNOSIS: Survival times **(MINE score)**

Received: 8 July 2020 | Accepted: 7 April 2021
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STANDARD ARTICLE

Journal of Veterinary Internal Medicine ACVIM Open Access
American College of Veterinary Internal Medicine

The Mitral INsufficiency Echocardiographic score: A severity classification of myxomatous mitral valve disease in dogs

Tommaso Vezzosi[1] | Giovanni Grosso[1] | Rosalba Tognetti[1] | Valentina Meucci[1] | Valentina Patata[2] | Federica Marchesotti[2] | Oriol Domenech[2]

Mild

Median survival time = 2,344 days (95% CI; 1,877 - 2,810d)

Moderate

Median survival time = 1,882 days (95% CI; 1,341 - 2,434d)

Severe

Median survival time = 623 days (95% CI; 432 - 710d)

Late stage

Median survival time = 157days (95% CI; 53 - 257d)

CASE 1

Penny: 9y FS CKCS x Maltese, 8 kg

Presenting complaint: **Coughing**

- 1-2x/day for 2 years
- 8-10x/day for 6 months

- Current treatment:
  - Frusemide 20 mg q 12 hrs
  - Pimobendan 2.5 mg q 12 hrs
  - Benazepril 2.5 mg q 24 hrs
  - Spironolactone 25 mg q 12 hrs

Problem	Location
Sneezing/discharge	Nasal cavity/oropharynx
Coughing with minimal dyspnea	Tracheobronchial
Dyspnea with minimal coughing	Laryngeal/bronchial/lung/pleural/ mediastinum
Coughing and dyspnea	brochoalveolar

CASE 1

Penny: 9y FS CKCS x Maltese, 8 kg

Temp: 101.4 F

- HR 100 bpm
- PR 100 bpm
- Grade IV/VI left apical systolic murmur

- Resp. sinus arrhythmia
- RR 24, eupneac
- MM pink, CRT 1 sec, tacky

CASE 1

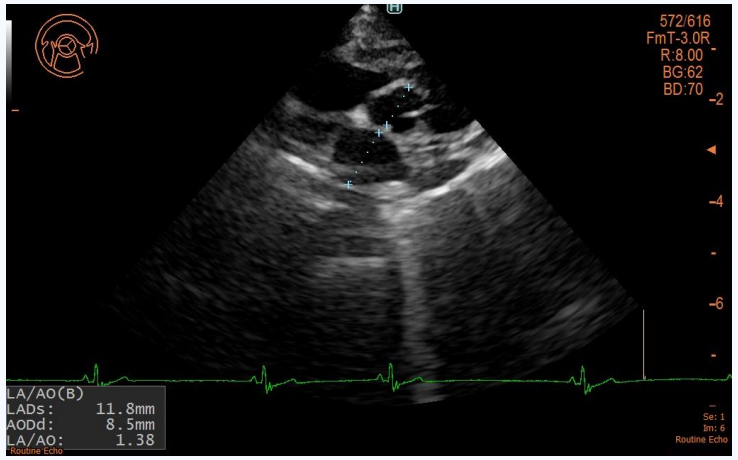
Penny: 9y FS CKCS x Maltese, 8 kg

- Signalment and murmur consistent with MMVD
- III/VI murmur could be consistent with CHF
- HR, sinus arrhythmia, and RR are not consistent with CHF
- Long history of cough suggests chronic airway disease
- Plan: Chest xrays, Echocardiogram, Blood Work

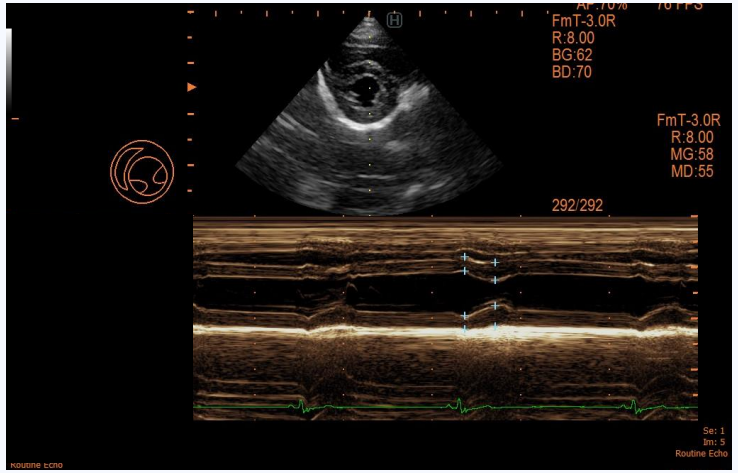
Bronchointerstitial pattern.
Vertebral heart score 10.8 without enlargement of any specific chamber

Penny: 9y FS CKCS x Maltese, 8 kg

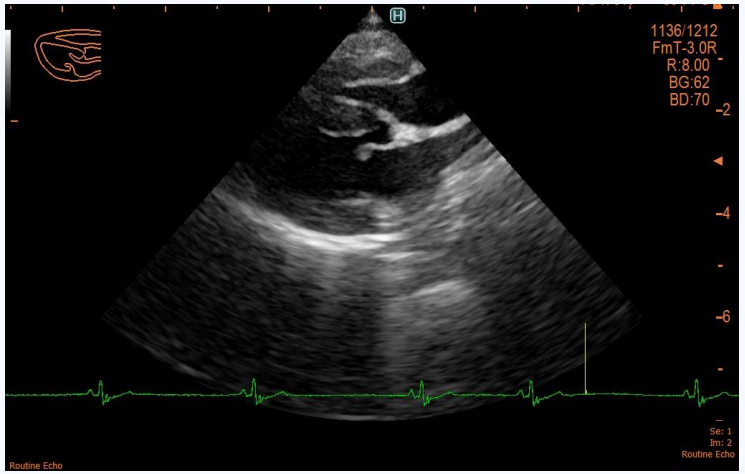
Penny: 9y FS CKCS x Maltese, 8 kg

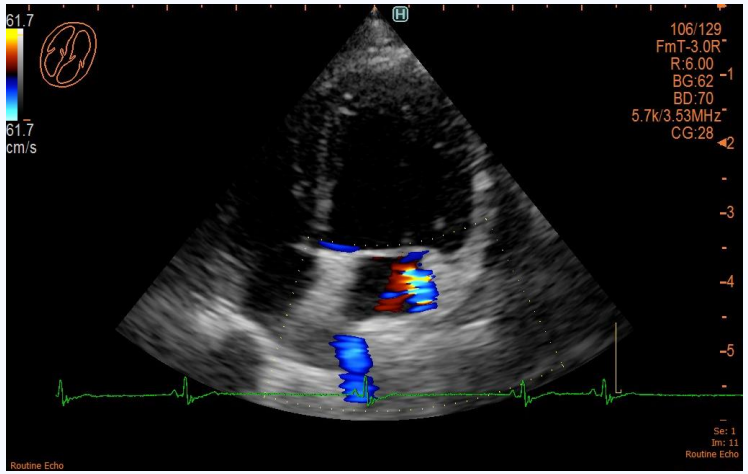


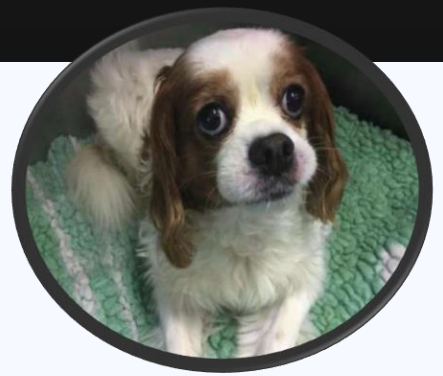
LA/Ao: 1.38 (<1.6)



LVIDd: 28.4 mm
LVIDd**N**: 1.54 (<1.7)







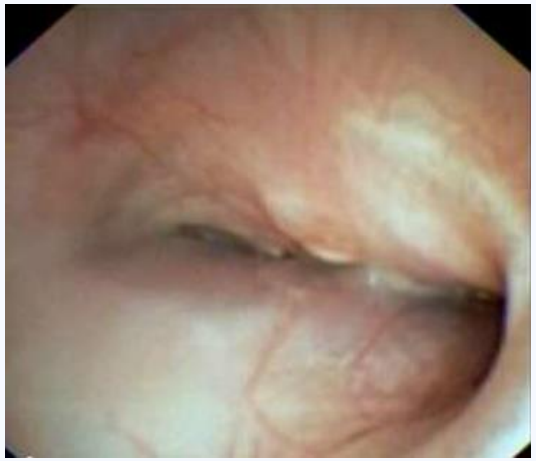
Penny: 9y FS CKCS x Maltese, 8 kg

- Stopped cardiac medications
- Doxycycline 5 mg/kg bid 3 weeks
- Theophylline 10 mg/kg bid 3 weeks

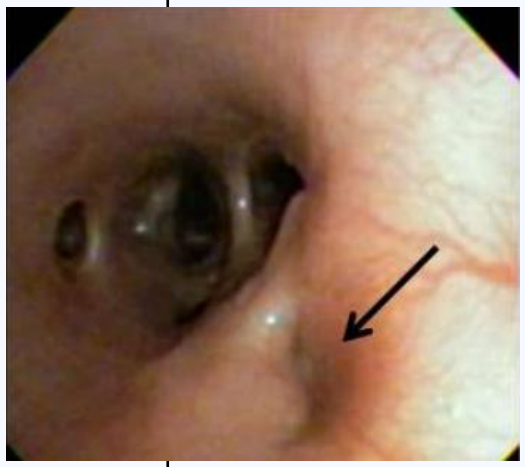
Plan

- Bronchoscopy

100% collapse
left cranial lobar bronchus



100% collapse
left caudal lobar bronchus



100% collapse
Right middle lobar bronchus

Diagnosis of Bronchomalacia with Myxomatous Mitral Valve Degeneration B1

CASE 2

Thung-Ngern: 7y M Chihuahua, 3.8 kg

The referring DVM **ausculted a heart-murmur** during the examination and referred **Thung-Ngern** for cardiac evaluation and echo request.

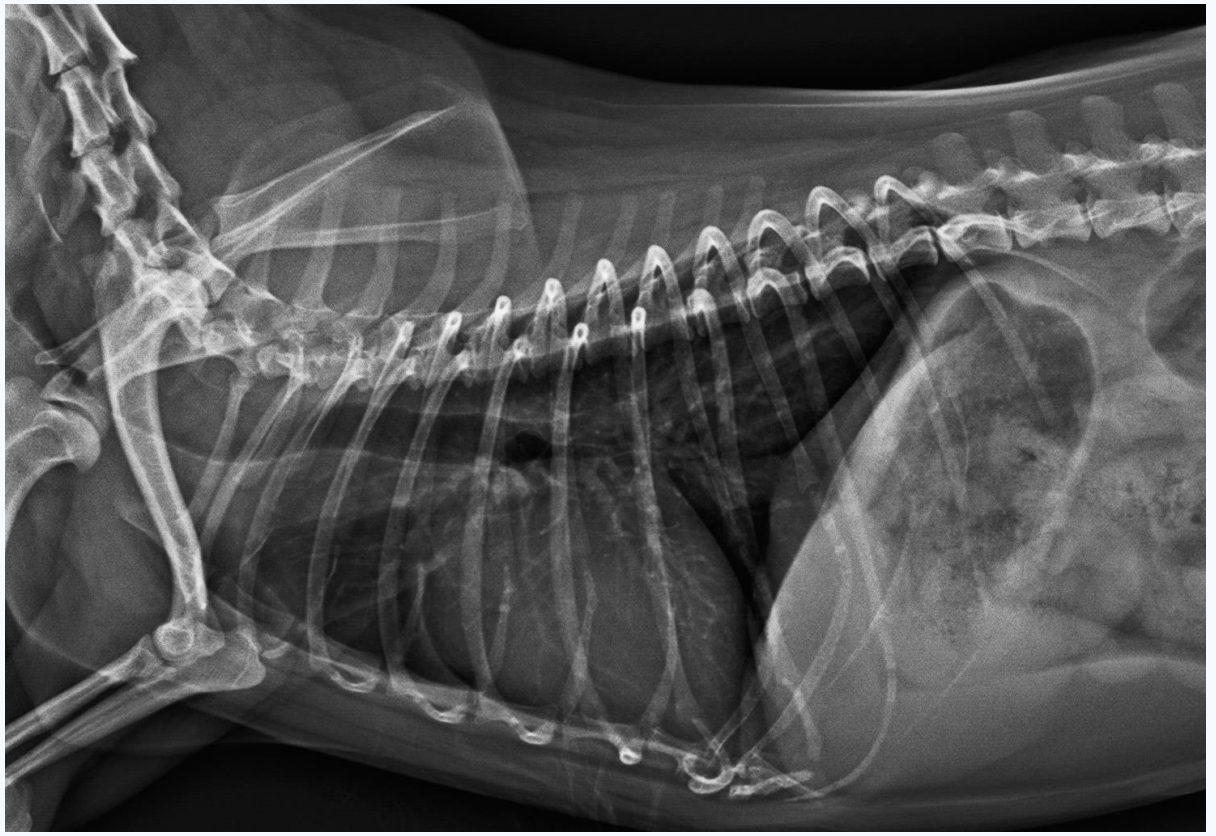
Temp: 100.4 F

- HR 130 bpm
- PR 130 bpm
- Grade IV/VI left apical systolic murmur
- RR 24, eupneac
- MM pink, CRT 1 sec, tacky

no signs associated with Lt. CHF.

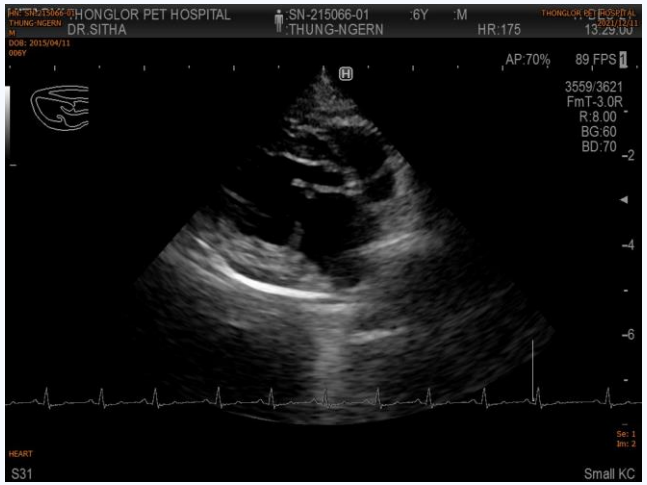
Plan: Chest xrays, Echocardiogram

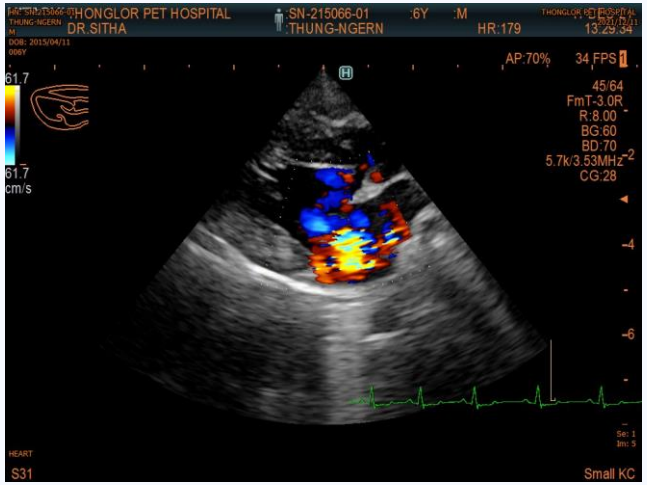
Thung-Ngern: 7y M Chihuahua, 3.8 kg

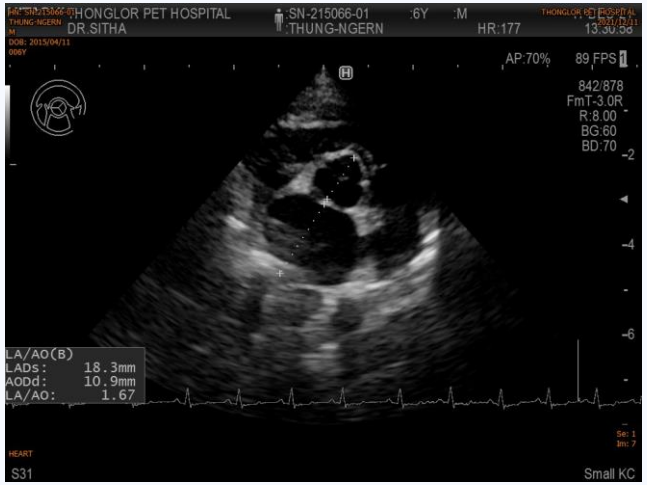


VHS = 10.6
No evidence of cardiogenic pulmonary edema.

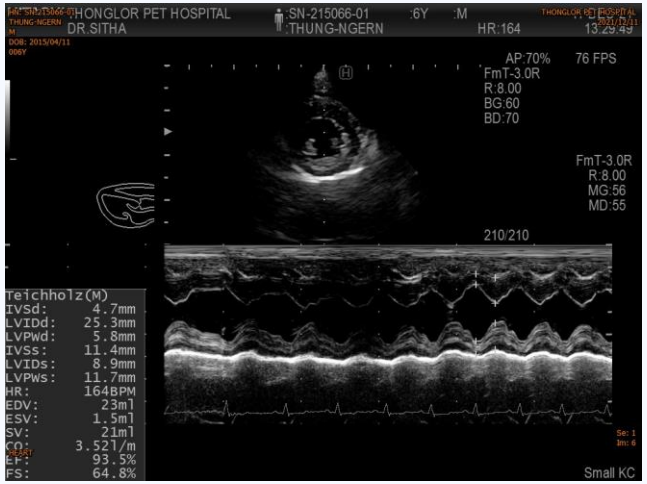
Thung-Ngern: 7y M Chihuahua, 3.8 kg



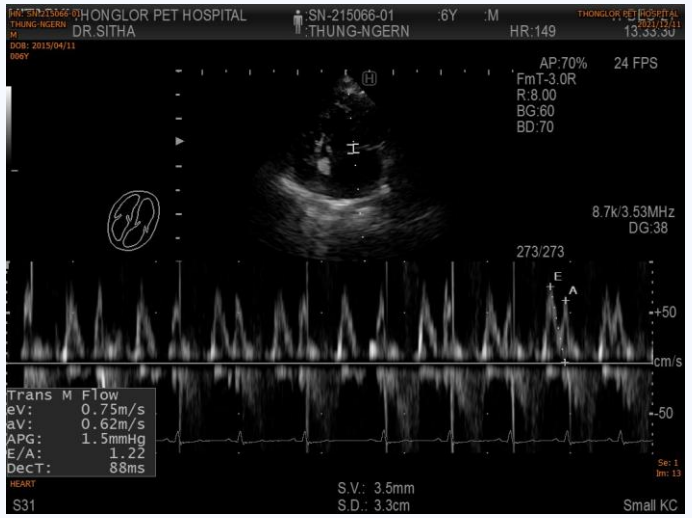




LA/Ao: 1.67 (<1.6)



LVIDd: 25.3 mm
LVIDd**N**: 1.71 (<1.7)



Ev: 0.75 m/s
E/A: 1.22

Received: 6 March 2019 | Accepted: 13 March 2019
DOI: 10.1111/jvim.15488

CONSENSUS STATEMENT

Journal of Veterinary Internal Medicine ACVIM Open Access
American College of Veterinary Internal Medicine

Consensus Statements of the American College of Veterinary Internal Medicine (ACVIM) provide the veterinary community with up-to-date information on the pathophysiology, diagnosis, and treatment of clinically important animal diseases. The ACVIM Board of Regents oversees selection of relevant topics, identification of panel members with the expertise to draft the statements, and other aspects of assuring the integrity of the process. The statements are derived from evidence-based medicine whenever possible and the panel offers interpretive comments when such evidence is inadequate or contradictory. A draft is prepared by the panel, followed by solicitation of input by the ACVIM membership which may be incorporated into the statement. It is then submitted to the *Journal of Veterinary Internal Medicine*, where it is edited prior to publication. The authors are solely responsible for the content of the statements.

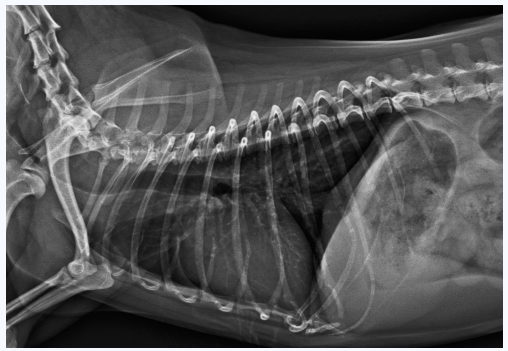
ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

Bruce W. Keene[1] | Clarke E. Atkins[1] | John D. Bonagura[1,2] | Philip R. Fox[3] | Jens Häggström[4] | Virginia Luis Fuentes[5] | Mark A. Oyama[6] | John E. Rush[7] | Rebecca Stepien[8] | Masami Uechi[9]

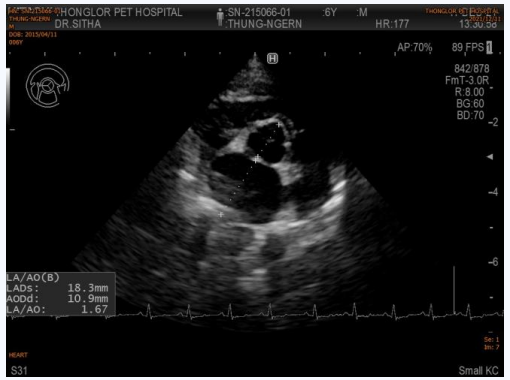
Thung-Ngern: 7y M Chihuahua, 3.8 kg

Diagnosis of MMVD Stage B2

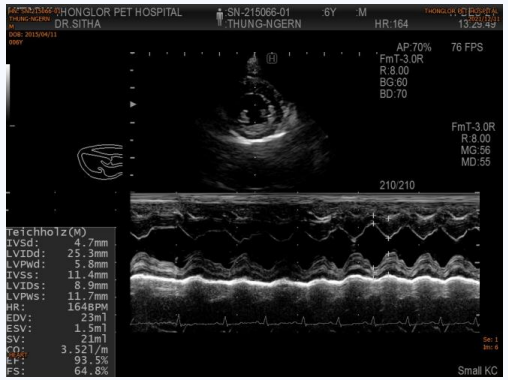
- **Mild to moderate mitral regurgitation.**
- **Normal LV systolic function.**
- **Normal LV diastolic function.**



VHS = 10.6
No evidence of cardiogenic pulmonary edema.



LA/Ao: 1.67 (<1.6)



LVIDd**N**: 1.71 (<1.7)

Pimobendan 0.25mg/kg q12h AC

Received: 8 July 2020 | Accepted: 7 April 2021
DOI: 10.1111/jvim.16131

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STANDARD ARTICLE

The Mitral INsufficiency Echocardiographic score: A severity classification of myxomatous mitral valve disease in dogs

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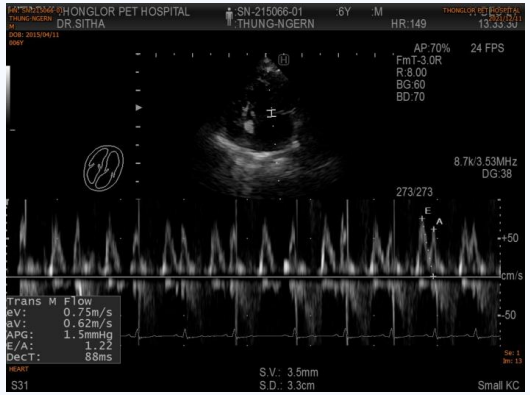
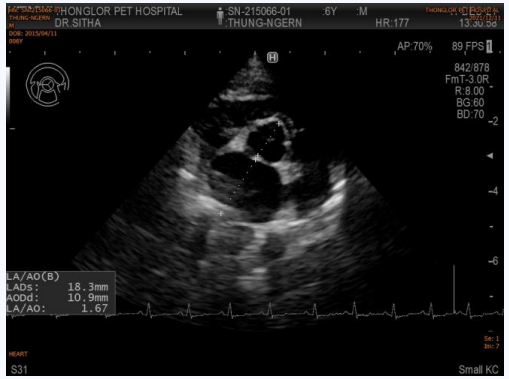
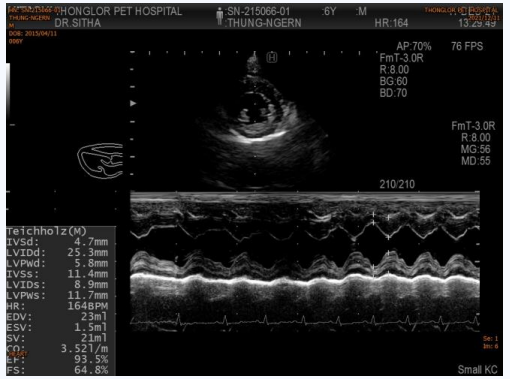


Evel: 0.75 m/s
E/A: 1.22



LA/Ao: 1.67 (<1.6)



LVIDd**N**: 1.71 (<1.7)

Thung-Ngern: 7y M Chihuahua, 3.8 kg

Survival times
MINE score

1.) LA/Ao: 1.67 = 1 score
2.) LVIDd**N**: 1.71 = 2 score
3.) %FS: 64.8 = 3 score
4.) E-vel: 0.75 = 1 score

Total = 7

TABLE 1 Selected echocardiographic cutoffs and relative scores

	Score			
	1	2	3	4
LA/Ao	<1.70	1.70-1.90	1.91-2.50	>2.50
LVIDDn	<1.70	1.70-2.00	2.10-2.30	>2.30
FS (%)	<45	45-50	>50	
E-vel (m/s)	<1.20	1.20-1.50	>1.50	

Abbreviations: E-vel, E-wave transmitral peak velocity; FS, fractional shortening of the left ventricle; LA/Ao, left atrium-to-aorta ratio; LVIDDn, left ventricular end-diastolic diameter normalized for body weight; .

TABLE 2 Severity classification based on the total score obtained from the summation of the single scores obtained with Table 1

Severity classification	Total score
Mild	4-5
Moderate	6-7
Severe	8-12
Late stage	13-14

Mild

Median survival time = 2,344 days (95% CI; 1,877 - 2,810d)

Moderate **(Total score 6 - 7)**

Median survival time = 1,882 days (95% CI; 1,341 - 2,434d)

Severe

Median survival time = 623 days (95% CI; 432 - 710d)

Late stage

Median survival time = 157days (95% CI; 53 - 257d)

CASE 3

Lucky: 14-year-old Mc Pomeranian, 6.4 kg.

Loss of consciousness / Collapse: Syncope, Seizure ??

Pertinent history

- Diagnosed with **MMVD stage B2 with severe mitral regurgitation and mild tricuspid regurgitation 2years ago.**
- **Previous medication history: discontinue Pimobendan**
- **Episode brief with recovery and no tonic-clonic motor signs.**
- **Episode follows stress or excitement.**

Syncope

Lucky: 14-year-old Mc Pomeranian, 6.4 kg.

Physical examination

On presentation, QAR
Respiratory rate was 56 bpm with dyspnea.

Mucous membranes were cyanosis and CRT 2 seconds.

Lung auscultation : **increased respiratory sounds to fine crackle lung field bilaterally**.

Heart rate of 168 beats/minute
Cardiac auscultation 5/6 systolic murmur PMI over the left apical radiates dorsocranially,
4/6 systolic murmur PMI on the right, regular rhythm. Femoral pulses strong and symmetric.

Rectal temperature was not measured initially to minimise stress.

Lucky: 14-year-old Mc Pomeranian, 6.4 kg.

Diagnostic plan and initial stabilization

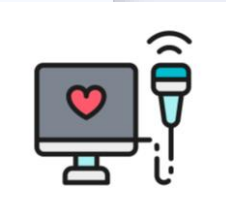
The pertinent history, presence of acute dyspnea & syncope in an asymptomatic MMVD male dog with severe mitral regurgitation raised the suspicion of heart disease and CHF.

Cardiogenic > Non-cardiogenic causes

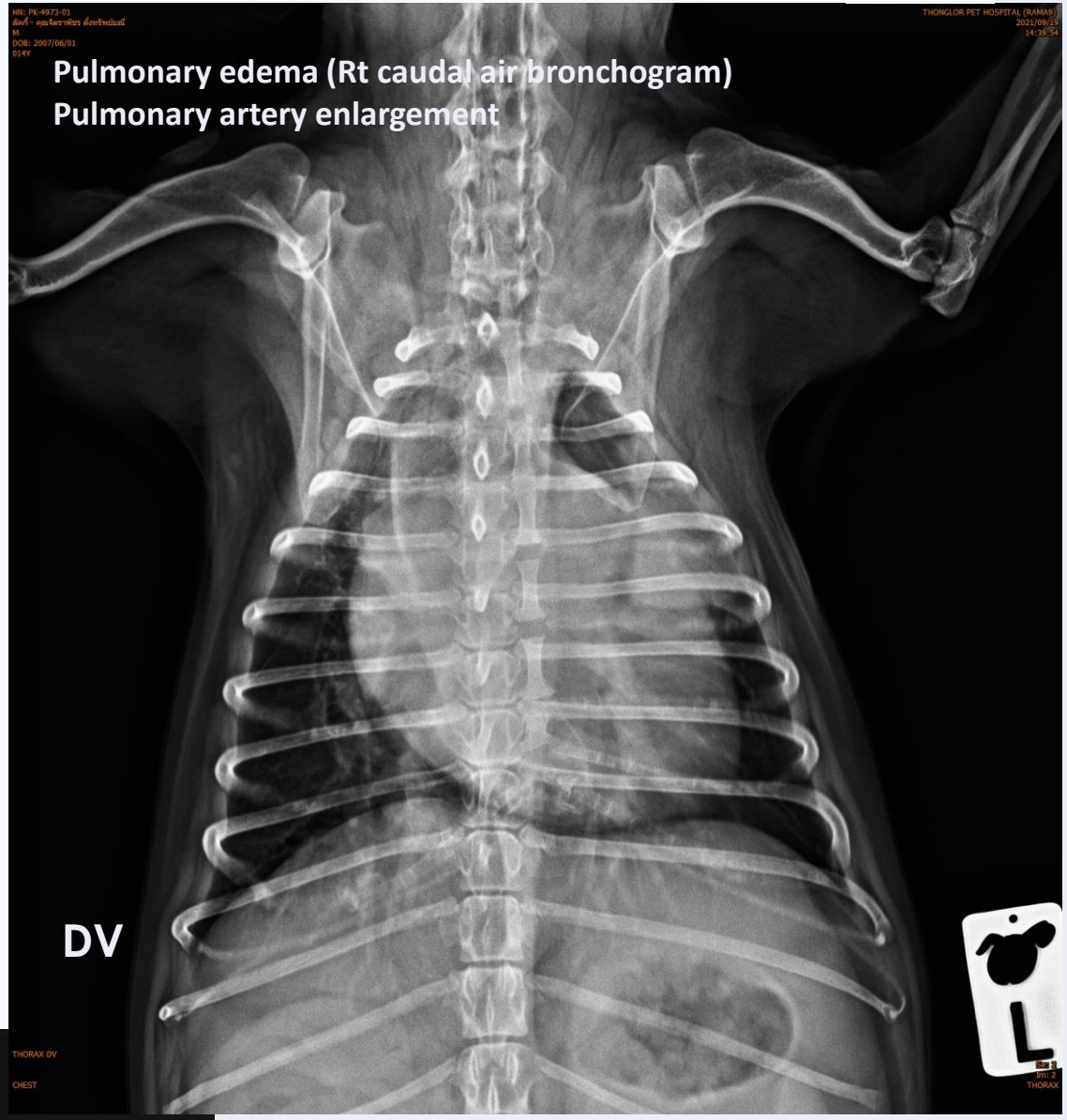
Initial stabilisation: placed in an oxygen-enriched chamber.

Once the patient had relaxed and appeared more comfortable,

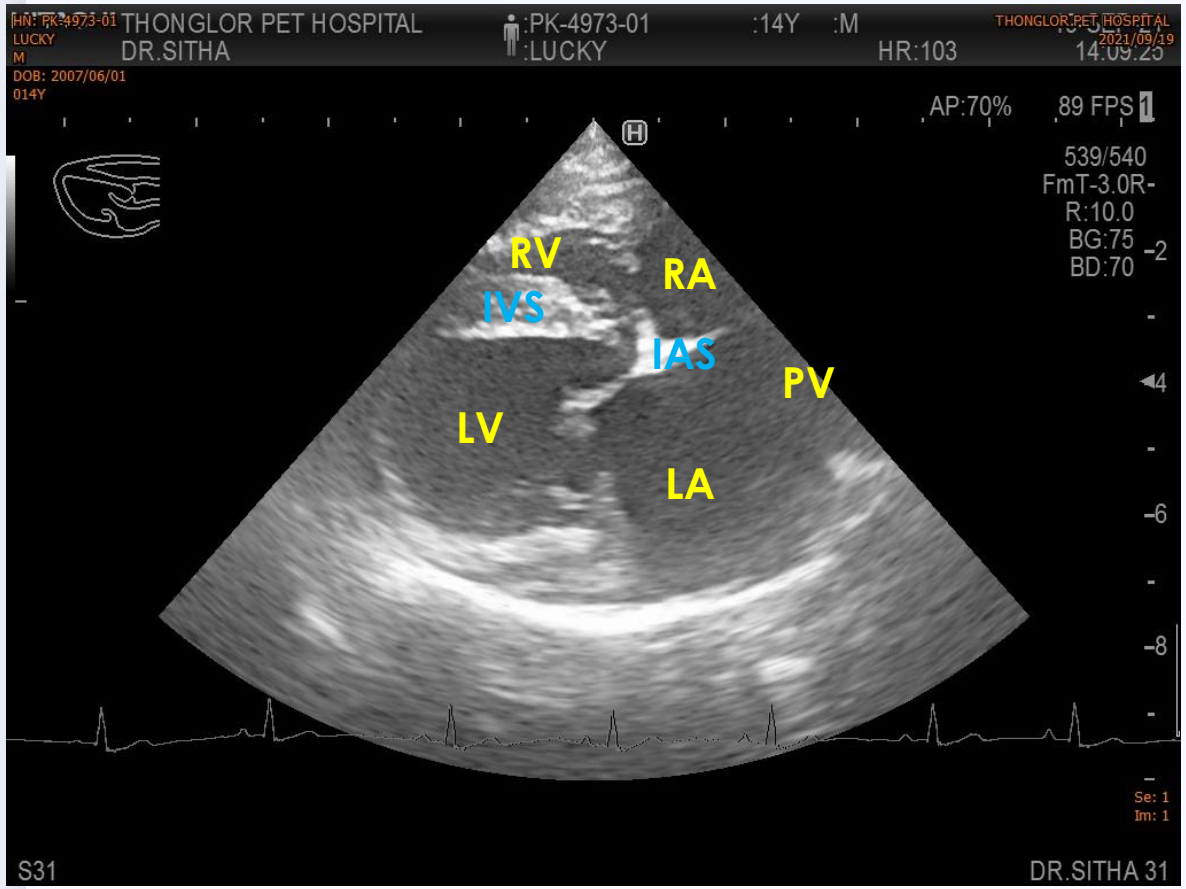
- Thoracic radiography
- Electrocardiogram (ECG)
- Echocardiogram

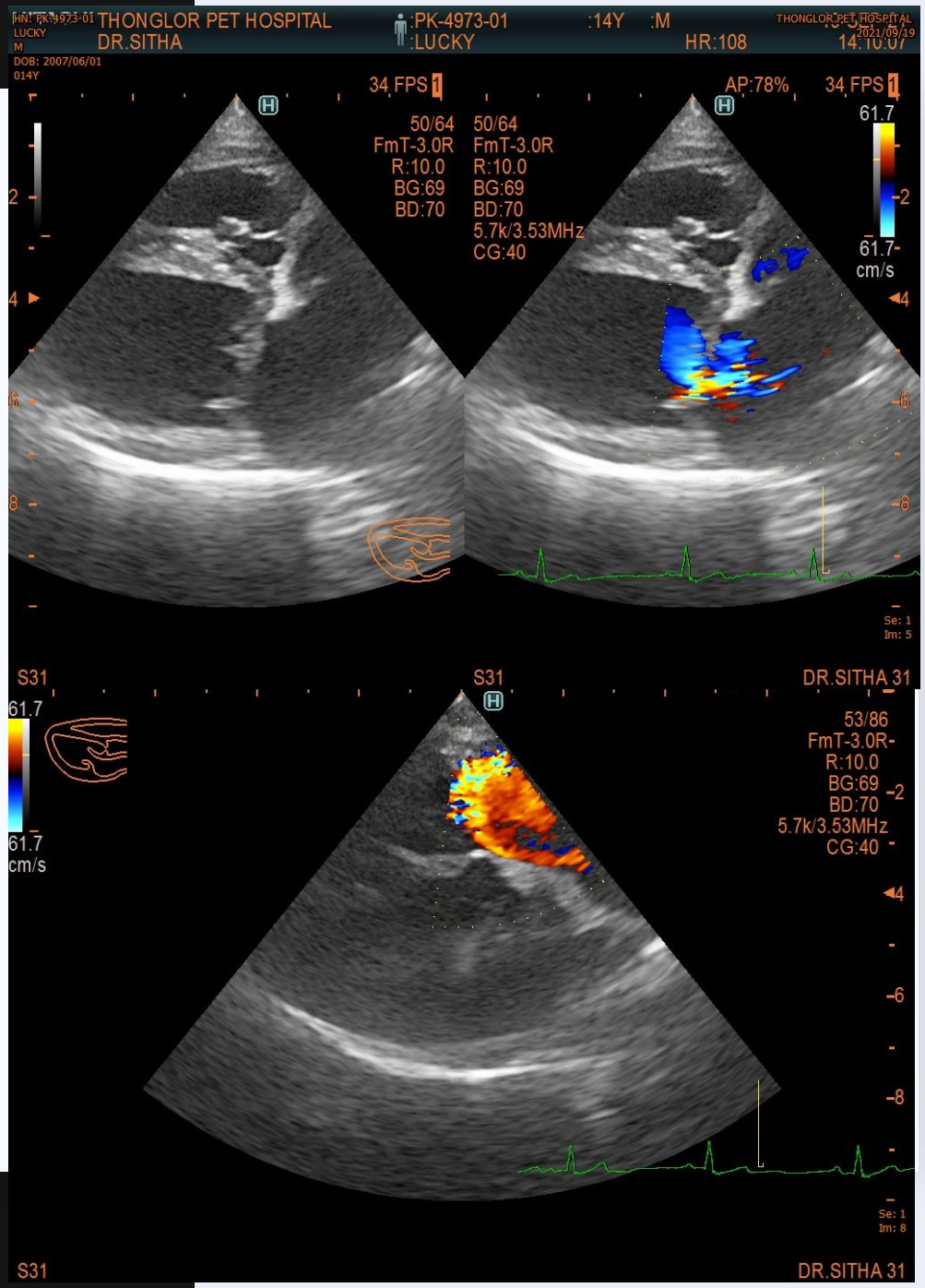


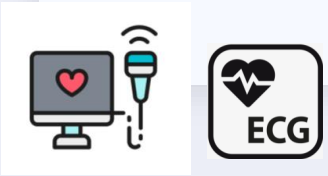
Thoracic radiographs



Echocardiogram

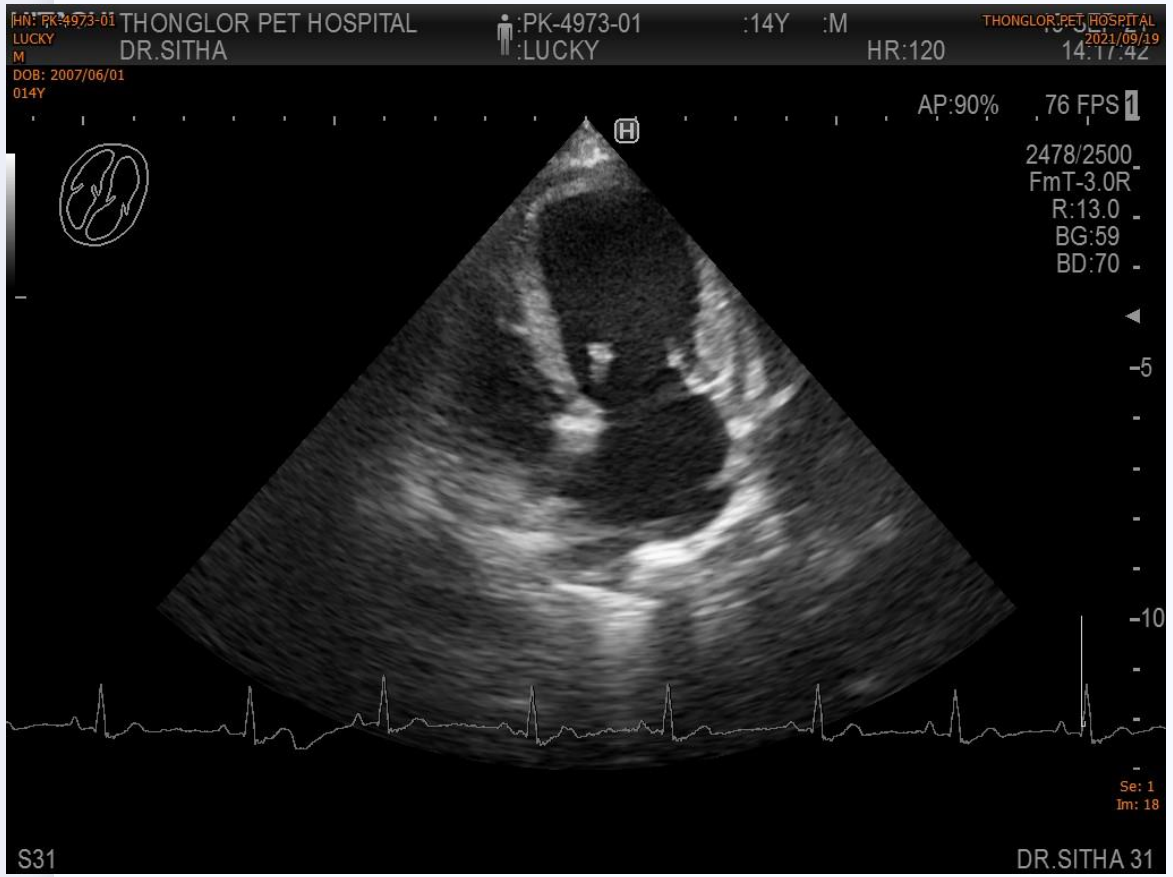


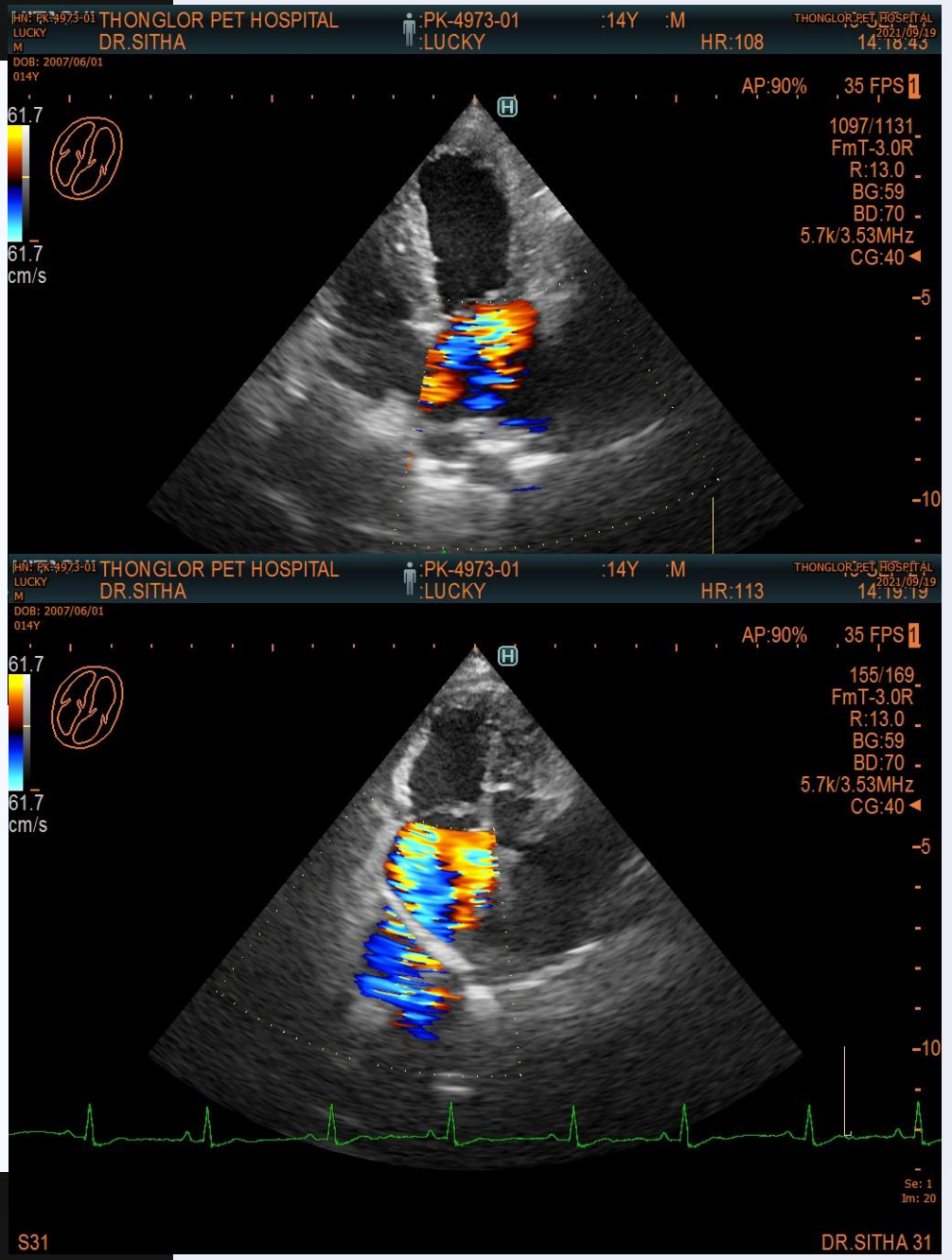




ECG

Echocardiogram





ECG

Echocardiogram

Left atrium assessment

2D: LA/Ao = 2.57 * (<1.6)**

“ Left atrial enlargement ”

Received: 6 March 2019 | Accepted: 13 March 2019
DOI: 10.1111/jvim.15488

Journal of Veterinary Internal Medicine ACVIM

CONSENSUS STATEMENT

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ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

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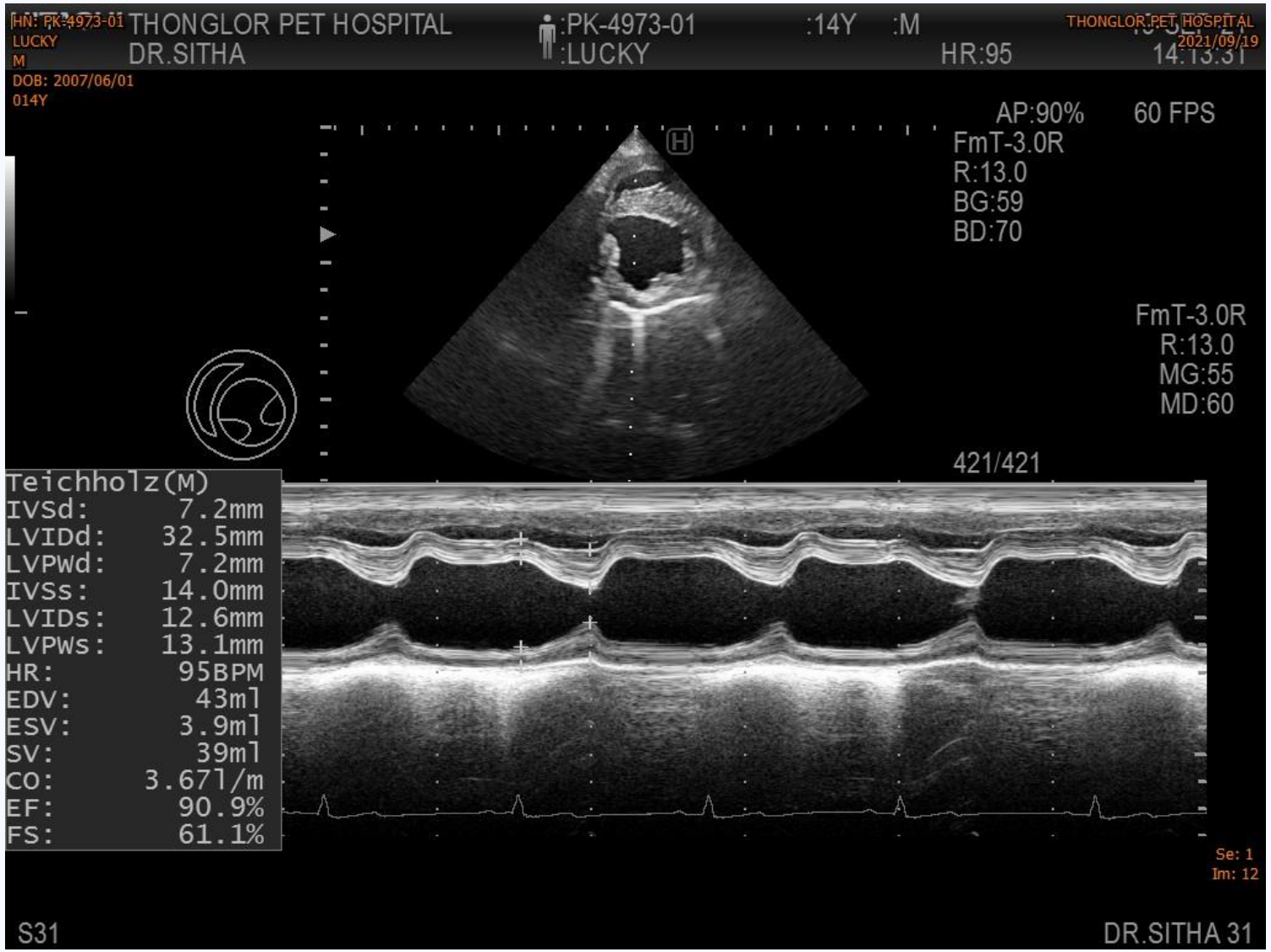
Echocardiogram

Left ventricle assessment

M-mode

- **LVIDd: 32.5 mm**
- **LVIDdN: 1.88 *** (<1.7)**

“ Left ventricular enlargement ”



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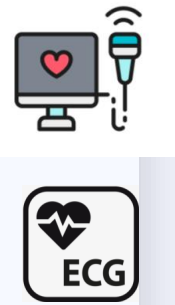
Echocardiogram

Pulmonary artery assessment

2D: MPA/AO = 1.2 (0.8-1.0)

“ Pulmonary artery enlargement ”



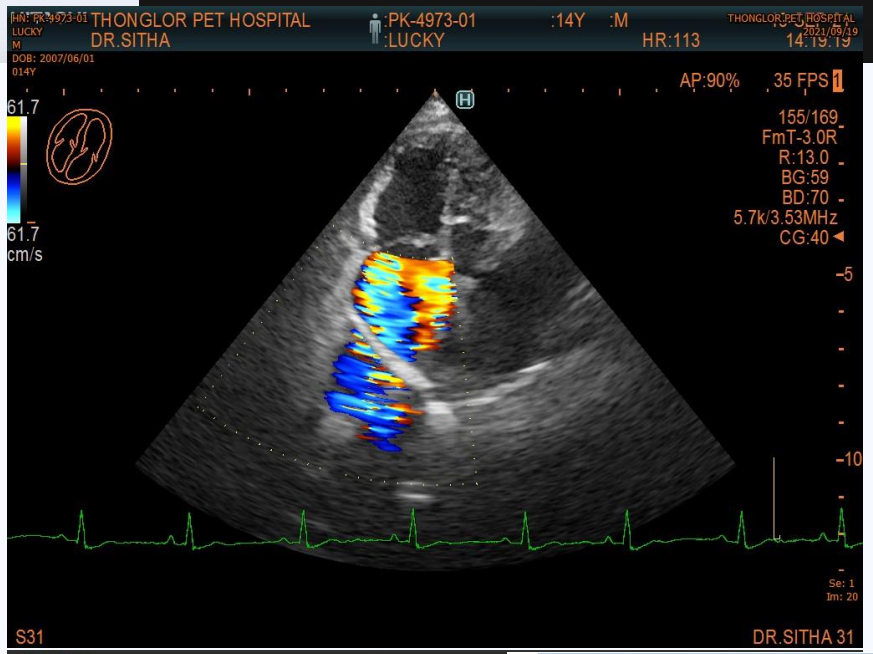
Echocardiogram

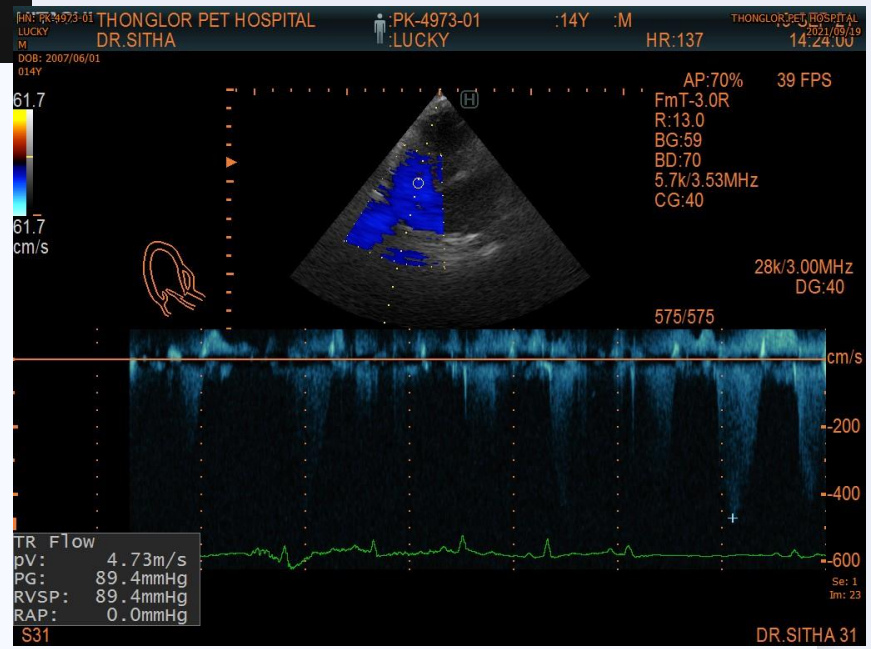
Pulmonary hypertension

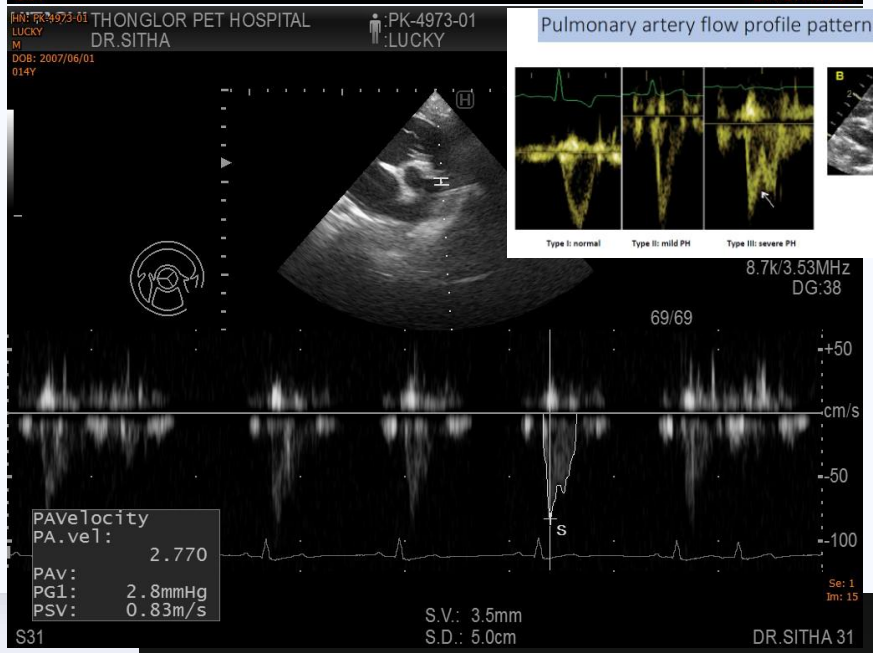
- Severe tricuspid regurgitation
- TR-Vmax: 4.73m/s (<2.5m/sec)
- RVSP: 89.4 mmHg (<25 mmHg)

- MPA/AO : 1.2
- %RPAD: 13.64 (38 - 49)
- Systolic notching of the Doppler RV outflow profile (Type III)

"Severe pulmonary hypertension"







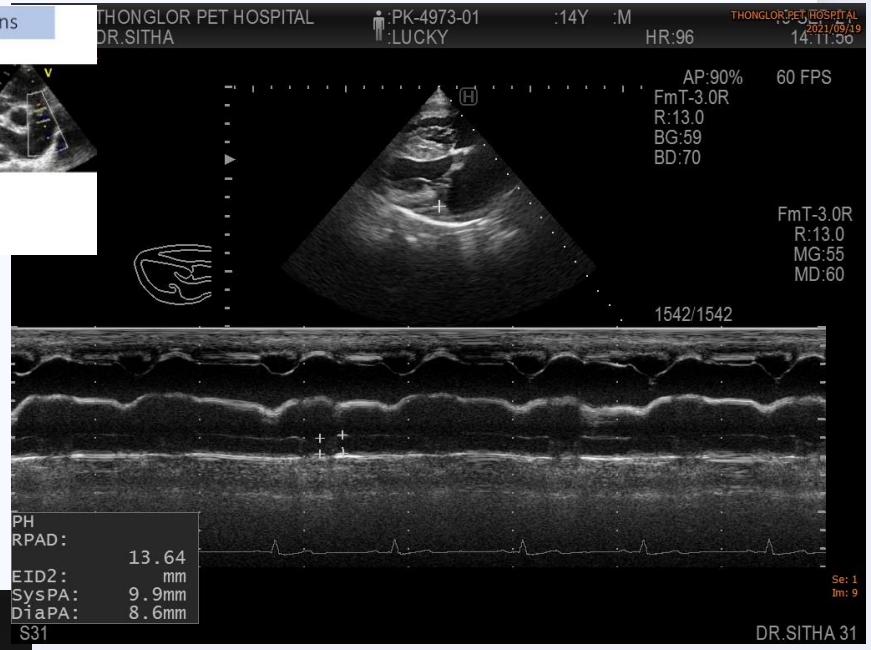


TABLE 2 Echocardiographic probability of PH in dogs

Peak tricuspid regurgitation velocity (m/s)	Number of different anatomic sites of echo signs of PH[a]	Probability of PH
≤3.0 or not measurable	0 or 1	Low
≤3.0 or not measurable	2	Intermediate
3.0 to 3.4	0 or 1	Intermediate
>3.4	0	Intermediate
≤3.0 or not measurable	3	High
3.0 to 3.4	≥2	High
>3.4	≥1	High

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CONSENSUS STATEMENT

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ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs

Carol Reinero[1] | Lance C. Visser[2] | Heidi B. Kellihan[3] | Isabelle Masseau[4] | Elizabeth Rozanski[5] | Cécile Clercx[6] | Kurt Williams[7] | Jonathan Abbott[8] | Michele Borgarelli[9] | Brian A. Scansen[10]

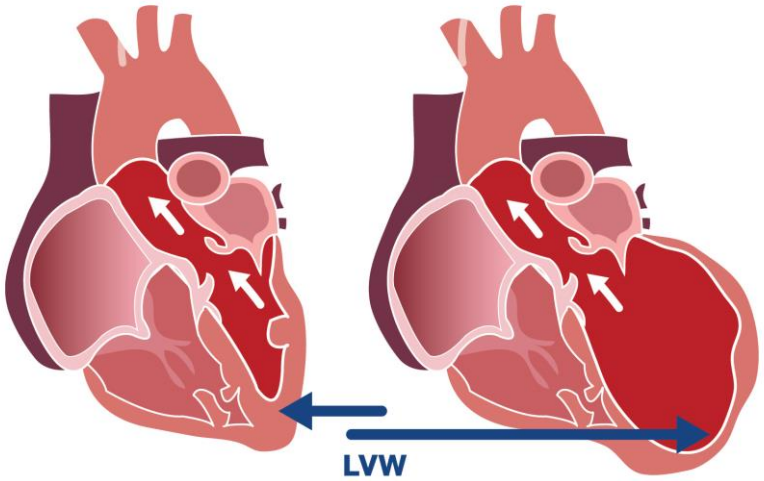
Lucky: 14-year-old Mc Pomeranian, 6.4 kg.

Diagnosis of

MMVD Stage C with Pulmonary hypertension (PH).

- Cardiogenic pulmonary edema.
- High probability of PH
- RVSP: 89.4 mmHg (Severe PH)

CANINE DILATED CARDIOMYOPATHY



Note the thin left ventricular wall (LVW), dilated LV chamber, and depiction of decreased forward blood flow with DCM.

CANINE DILATED CARDIOMYOPATHY

NON-AGING-ASSOCIATED DISEASES
LARGE - GIANT BREEDS

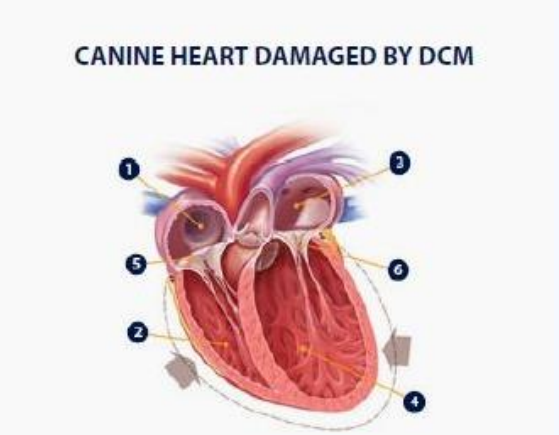
8-10% OF HEART DISEASE IN DOGS
80-90% DOBERMAN PINCHER & BOXER
25% IRISH WOLFHOUNDS

MALE (50%) > FEMALE (33%) (DOBERMAN PINCHER)

• STANDARD SCHNAUZERS • GOLDEN RETRIEVERS
• COCKER SPANIELS • GREAT DANES
• NEWFOUNDLANDS • SCOTTISH DEERHOUNDS

CANINE DILATED CARDIOMYOPATHY

- Loss of myocardial contractility and progressive ventricular dilation.



LV systolic dysfunction

⇩

Arrhythmia / Low CO signs

⇩

Chamber dilation / Arrhythmia

⇩

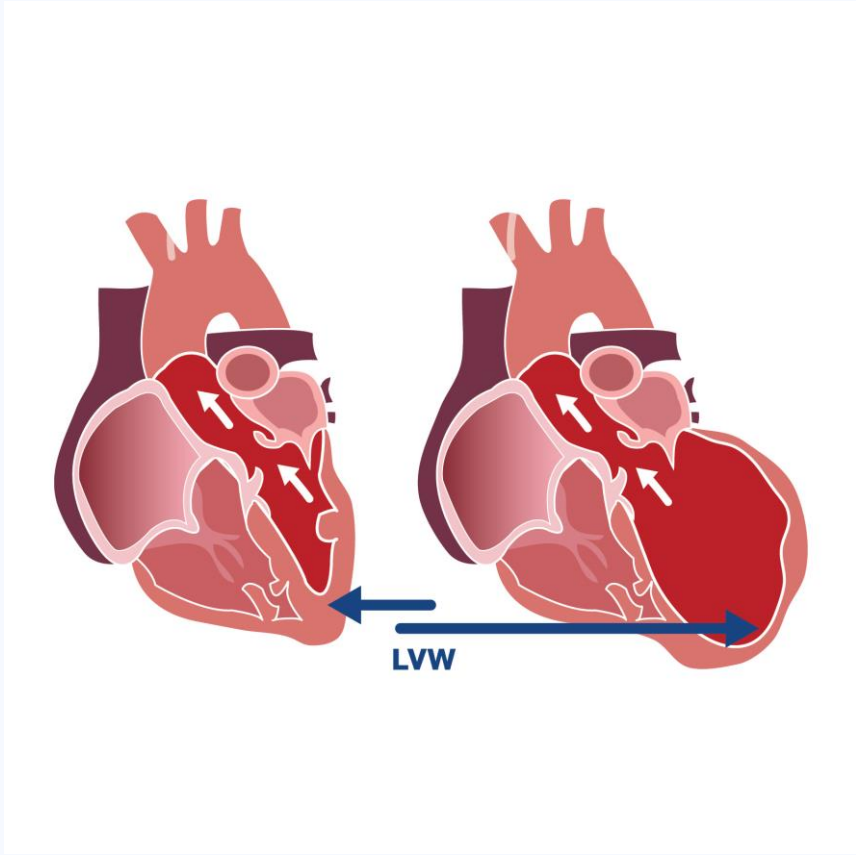
Functional mitral regurgitation ⇨ lung edema

Arrhythmia

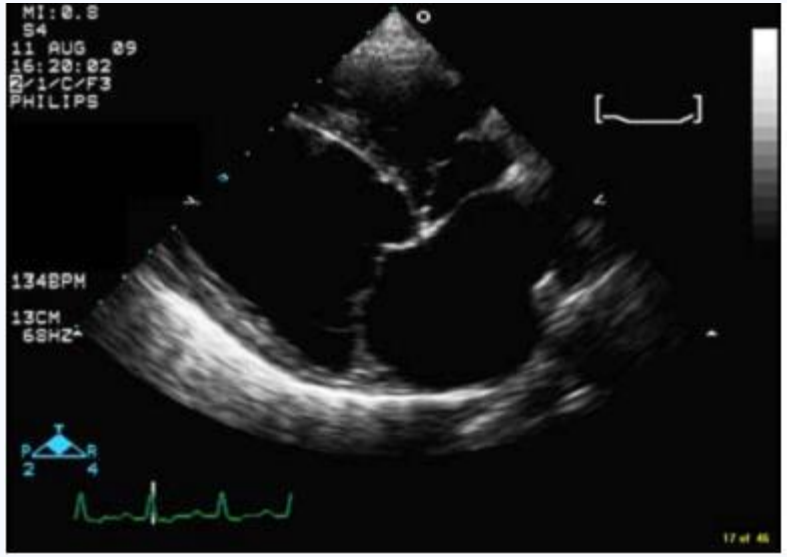
CANINE DILATED CARDIOMYOPATHY

- **Primary**: Disease of the heart muscle of unknown etiology
  + Genetic : Dobermans: PDK4 gene
  : Boxers: STRN gene
  : German Shorthaired Pointers: DMD gene
  : Great Danes: X-lined likely since males predisposed
  - specific gene unknown

- **Secondary**: Disease of heart muscle due to
  + toxins : chemotherapy
  + nutritional deficiencies : taurine, carnitine
  + endocrinopathies : hypothyroidism
  + Arrhythmias : chronic rapid heart rate
  + infection / inflammation : myocarditis



DIAGNOSIS



A

B

Journal of Veterinary Cardiology, Vol.5, No. 2, November 2003

Proposed Guidelines for the Diagnosis of Canine Idiopathic Dilated Cardiomyopathy

The ESVC Taskforce for Canine Dilated Cardiomyopathy

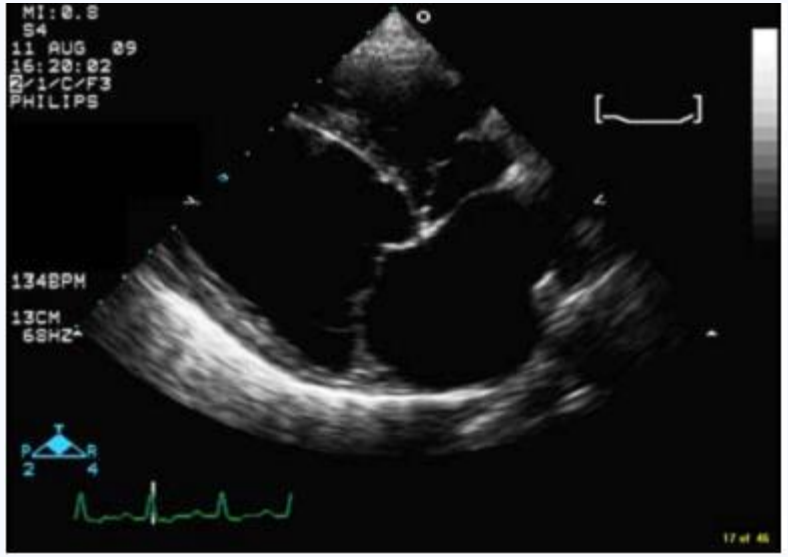
Joanna Dukes-McEwan[1]; Michele Borgarelli[2]; Anna Tidholm[3]; Andrea C Vollmar[4]; Jens Häggström[5]

Abstract

Dilated cardiomyopathy (DCM) is a major cause of morbidity and mortality in various dog breeds. The diagnosis of overt DCM is not normally problematic, although the importance of active exclusion of other causes of the dilated, hypokinetic heart is emphasised. Recent interest in human familial DCM has prompted a number of researchers to investigate the genetic basis of canine DCM. Prospective screening of dogs from lines with familial prevalence of DCM may identify dogs with pre-clinical ("occult") DCM. Dogs with other echocardiographic abnormalities or arrhythmias may also be identified. It is clear that dogs, like humans, have a prolonged pre-symptomatic phase of the disease extending over years. The ESVC DCM taskforce was established to provide the veterinary cardiology community with guidelines for the diagnosis of DCM, predominantly based on 2D and M-mode echocardiography. Diagnosis of DCM requires all of the following: (i) Left ventricular dilatation (ii) Reduced systolic function (iii) Increased sphericity of the left ventricle. We propose a scoring system for the identification of dogs in the pre-clinical stages. These include a number of major criteria and minor criteria. Future prospective longitudinal studies are required to test these in different breed populations to assess their predictive power and further refinements may be required. The importance of post mortem confirmation of disease is emphasised, and the two major histopathological features associated with DCM, the attenuated wavy fibre and the fibro-fatty infiltration-degenerative forms, require further investigation to identify the different aetiopathogenetic factors which may be involved.

Key words: Dog, Dilated Cardiomyopathy, Echocardiography, Histopathology, Familial Dilated Cardiomyopathy

DIAGNOSIS



A

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Journal of Veterinary Cardiology xxx (xxxx) xxx

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Review

Screening for dilated cardiomyopathy in dogs

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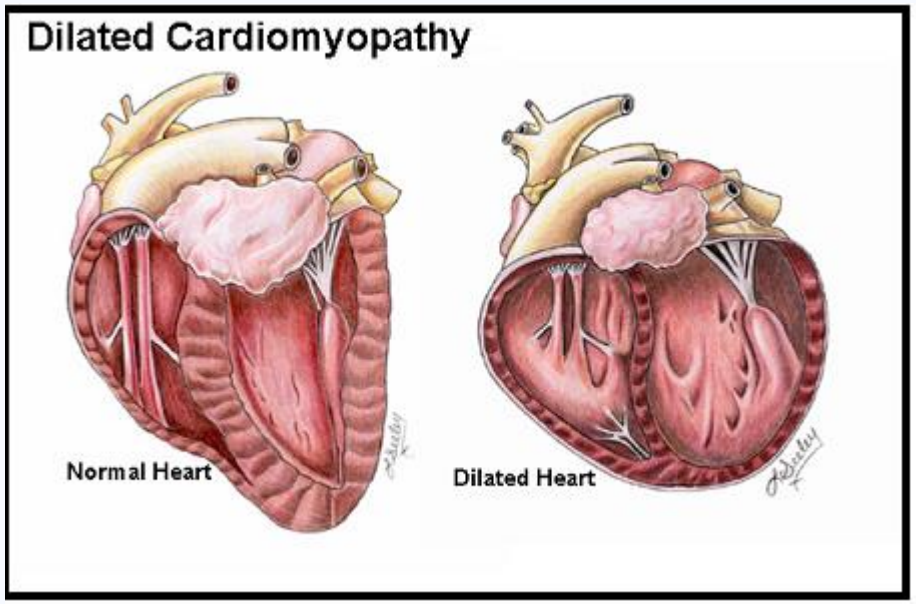
DCM



DCM is generally divided into two phases:

1) An occult phase, during which time disease is present but dogs remain asymptomatic. This phase can last months to years.
2) An overt clinical phase, when clinical signs develop.

EXCELLENT CENTER

THONGLOR PET HOSPITAL

โรงพยาบาลสัตว์ทองหล่อ

TOTAL SOLUTIONS

ศูนย์โรคหัวใจ
Cardiorespiratory center

ปัจจุบันโรคหัวใจเป็นโรคที่พบมากขึ้นในสัตว์เลี้ยง คลินิกหัวใจของโรงพยาบาลสัตว์ทองหล่อ มีเครื่องมือครบครันในการตรวจวินิจฉัยภาวะผิดปกติของระบบหัวใจ และหลอดเลือด ทั้งนี้ การตรวจรักษายังดำเนินการโดยสัตวแพทย์ ผู้มีความชำนาญโดยเฉพาะ เพื่ออายุที่ยืนยาวของสัตว์เลี้ยงที่ท่านรัก


สพ.ญ. ชัญกร กวางรัตน์


สพ.ญ. รติพร ดันดีศักดิ์


สพ.ญ. อุบลรัตน์ นิมิตไตรทิพย์


สพ.ญ. สุชาดา หัทยานานนท์


น.สพ. วิศรุต ช้างพันธ์


สพ.ญ. ออมอุสาห์ ก๊วหา


น.สพ.ศิธา สาริคาร


น.สพ. อุกฤษฎ์กานต์ คงเสียมงคล


น.สพ. พงษ์ระวี แสนมัญญา


สพ.ญ. พลอยไพลิน ปริณดาวงศ์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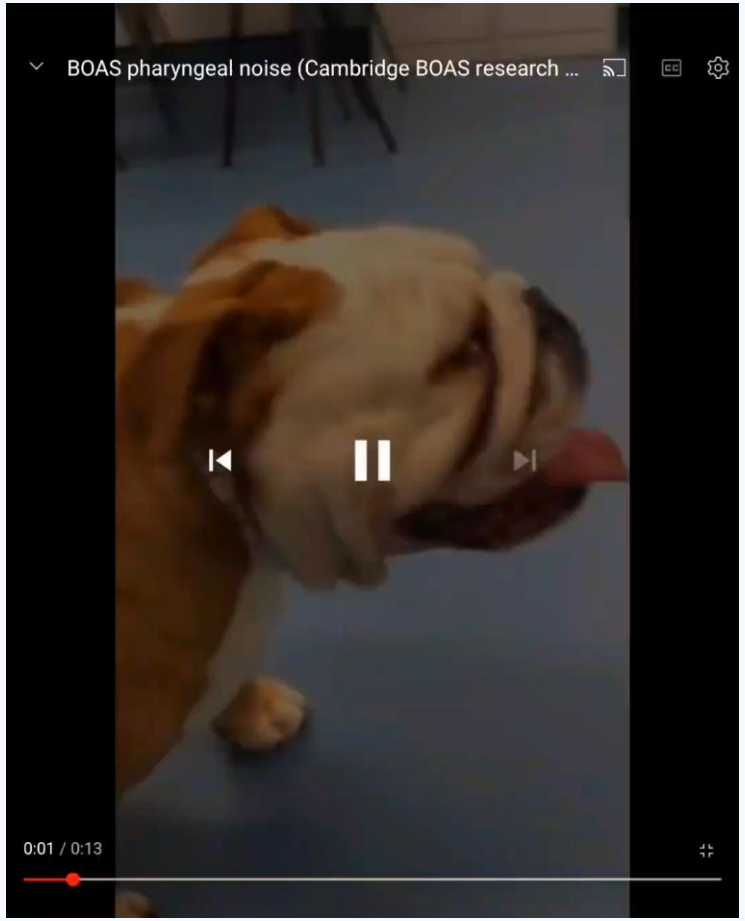
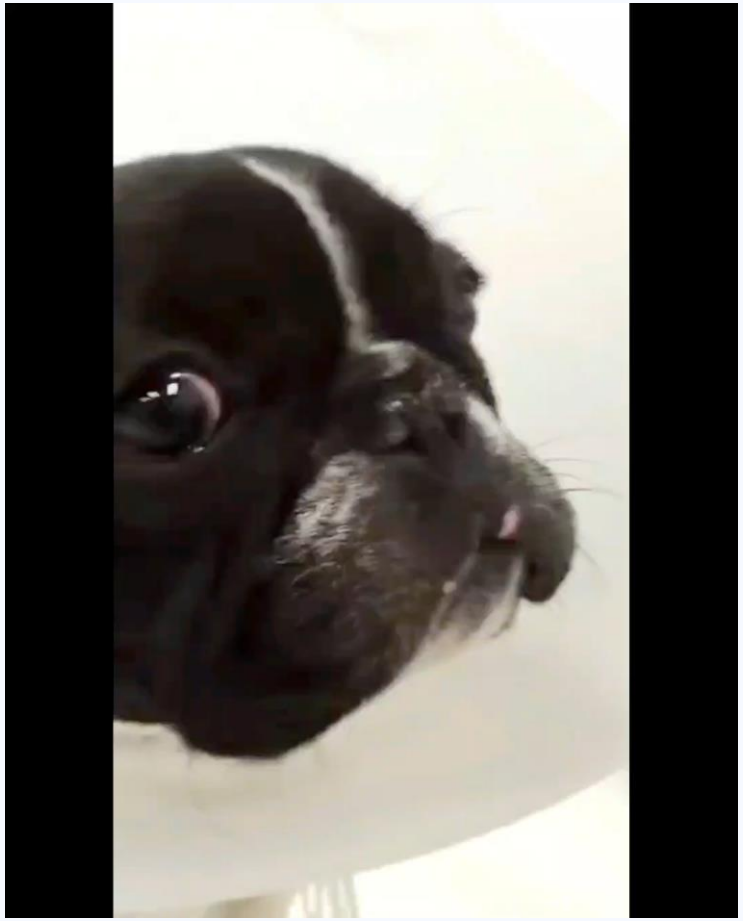
สพ.ญ.วิภาดา งามสงวน

สอบถามข้อมูลเพิ่มเติมได้ที่ 02-079-9999

Thonglor Pet Hospital www.thonglorpet.com @jaothonglor

STERTOR





- Stertor and stridor are two abnormal sounds that result from upper airway partial or complete obstruction.
- Stertor, which sounds like a snore, The low-pitched sound that is associated with stertor suggests that flaccid tissue is vibrating throughout the respiratory cycle. This occurs routinely in brachycephalic patients that have elongated soft palates

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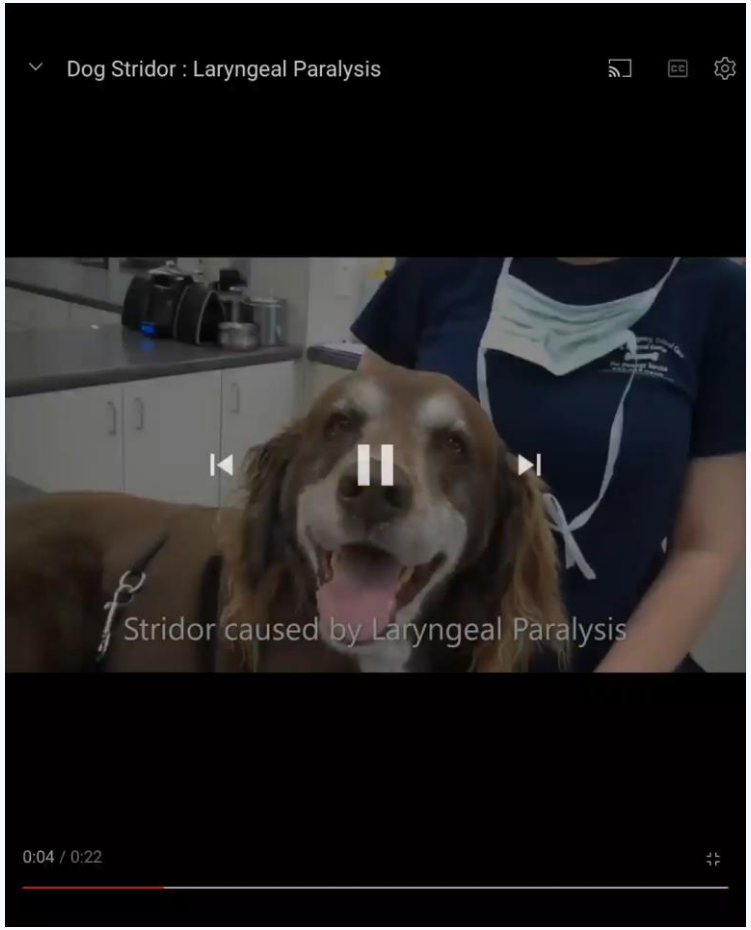
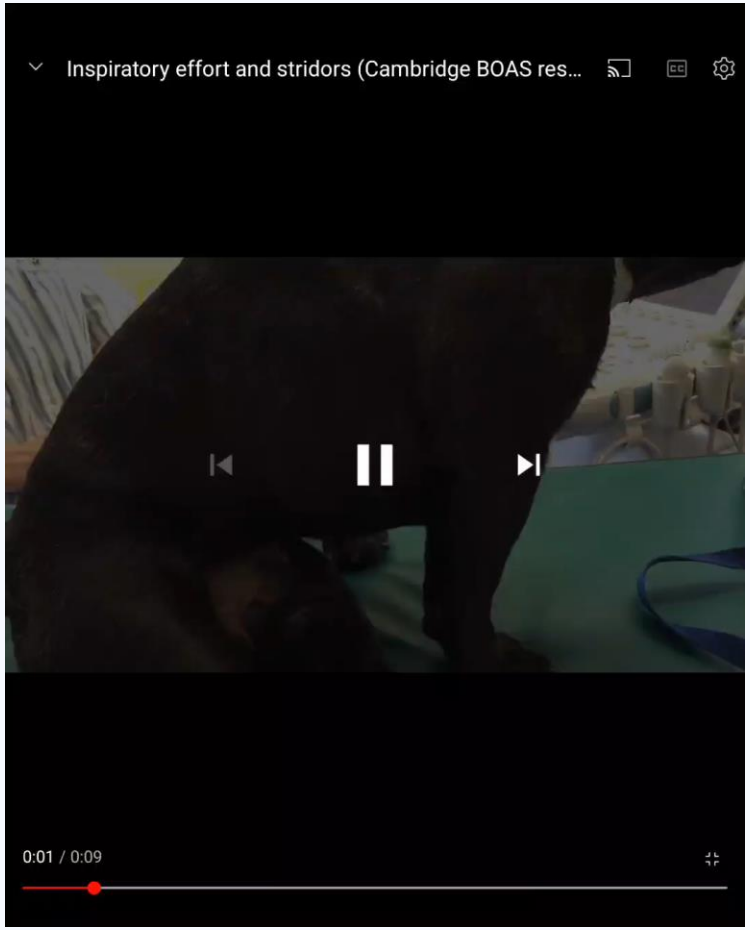
STRIDOR





- Stertor and stridor are two abnormal sounds that result from upper airway partial or complete obstruction.
- Stridor is a high-pitched sound that results from rigid tissue vibrations. It is typically associated with laryngeal or tracheal disease.

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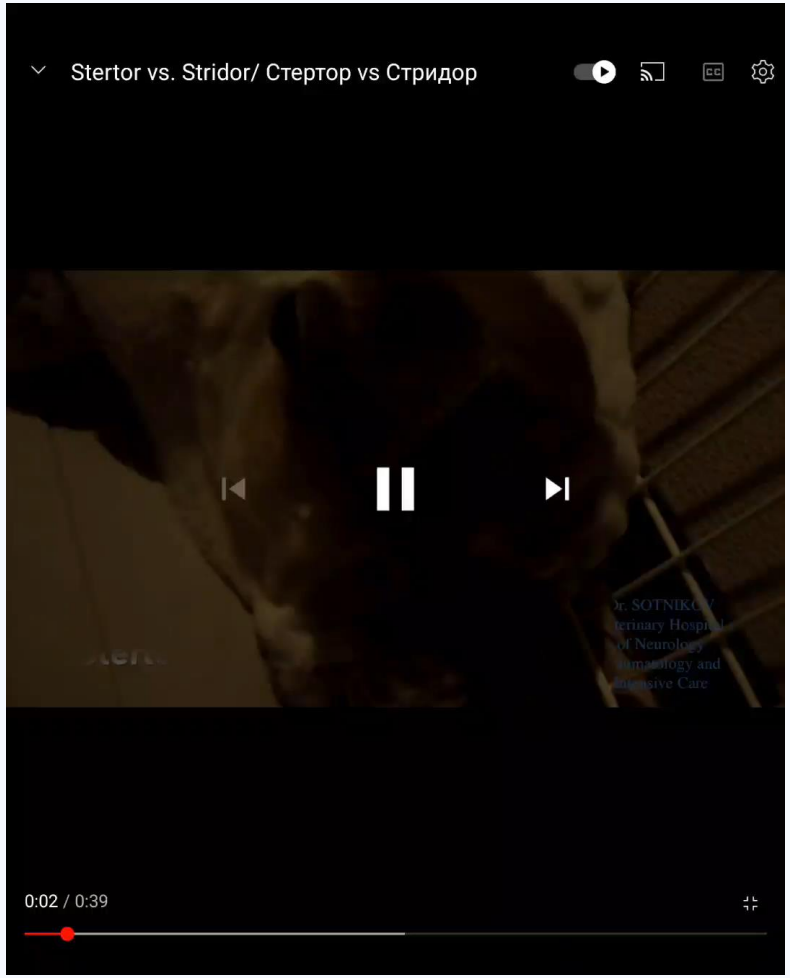
STERTOR VS STRIDOR



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- Stridor is a high-pitched sound that results from rigid tissue vibrations. It is typically associated with laryngeal or tracheal disease.

Back page25

HEART DISEASE IN CATS

SITHA S. DVM,
PGFVS(CARDIORESPIRATORY)