



**PATIENT**

Jezebel Meyer

**SPECIES**

Canine

**BREED**

Doberman Pinscher

**SEX**

Female Spayed

**AGE**

11 years

**WEIGHT**

70lbs

**INTERPRETED BY**

Maggie Machen Lamy,  
DVM, DACVIM  
(Cardiology)

**IMAGING PERFORMED BY**

Sarah Pender, CVT

**HOSPITAL NAME**

SVS Imaging QC

**REFERRING VET**

Dr. Neumeister

**INVOICE**

26675

**DATE**

10/3/22

**PRESENTING CLINICAL SIGNS**

History: Labored breathing. Laying down in sternal recumbency for a week. Coughing.  
-Current medications: Placed on Benazepril 20mg: 1/2 pill PO SID, Vetmedin 5mg: 1.5 pills PO BID, Furosemide 20mg: 2pills PO BID. Owners reported that Jezebel is 100% improved today 10/3 from when she started meds on 9/27/22.

**RADIOGRAPHIC FINDINGS** \*NOTE: Images submitted for supplemental cardiac information only.  
Mild cardiomegaly with concern for CHF.

**ELECTROCARDIOGRAPHIC FINDINGS** \*Note: Single lead ECGs are evaluated as a rhythm strip. Morphology/MEA cannot be definitively commented on.

A single lead ECG is available; 25mm/s, 10mm/mV. The average heart rate is 130bpm (range 120-150bpm). The QRS is low voltage and P waves are difficult to visualize; however, a sinus origin is suspected. Isolated VPCs are seen throughout; singles only and monomorphic in appearance. No supraventricular premature beats, pauses or other dysrhythmias observed. ECG diagnosis: Normal sinus rhythm with frequent isolated VPCs.

**ECHOCARDIOGRAM FINDINGS**

2D, m-mode, color flow and doppler imaging is available. Diffuse thickening of mitral valve leaflets with no prolapse into the left atrial lumen. Moderate eccentric mitral regurgitation with moderate left atrial dilation. Normal MR velocity. Mild LV dilation with mildly depressed myocardial function. The tricuspid valve appears normal, with trace TR. Mildly elevated velocity. Normal right atrial and ventricular diameter and morphology. The pulmonic and aortic valves are normal in morphology and mobility. Normal aortic and pulmonic outflow velocities with laminar flow. No AI/PI. No pericardial or pleural effusion noted. No obvious cardiac masses.

**CARDIAC CHART**

CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base; Swe)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
PATIENT	4.8	3.0	2.9	1.9	22	40	0.8
CANINE CARDIAC PARAMETERS	HR (BPM)	AV VMAX (m/s)	PV MAX (m/s)	BODY WEIGHT (kg)	LA 2D short axis Base view (cm)	LVIDd Avg; 2D and m-mode short axis (cm)	LVIDs Avg; 2D and m-mode short axis (cm)
NORMAL PARAMETER	50-100	0.7-1.7	0.7-1.6	BELOW	BELOW	BELOW	BELOW
PATIENT	87	1.2	1.6	31.8	3.8	4.6	3.6
*Normal chamber parameters expressed as a mean value (SD)				3	1.27 (5.3)	2.46 (2.46)	1.36 (5.5)
<b>BODY WEIGHT DEPENDENT PARAMETERS</b>				5	1.40 (4.5)	2.74 (5.2)	1.60 (4.7)
<i>*Note: All measurements based upon multi-modal images and methods. An average value is reported.</i>				10	1.50 (3.8)	3.27 (3.5)	2.06 (3.1)
				15	1.83 (2.0)	3.71 (2.4)	2.43 (2.1)
				20	2.02 (1.9)	4.14 (2.2)	2.80 (2.0)
				25	2.18 (2.4)	4.48 (2.9)	3.10 (2.5)
				30	2.33 (3.3)	4.83 (3.9)	3.39 (3.4)
				35	2.48 (4.3)	5.17 (5.0)	3.69 (4.5)
				40	2.62 (5.2)	5.48 (6.1)	3.96 (5.4)
				50	2.88 (7.1)	6.07 (8.3)	4.46 (7.4)

Adapted from June Boon, Veterinary Echocardiography, 1998  
Rishniw M and Hollis NE, J Vet Intern Med 2000; 14:429-435  
Hansson et al, Vet Rad and Ultrasound 2002  
Bonagura et al. Echocardiography: principles of interpretation, Vet Clin North Am 15:1177, 1995

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

The cause of the murmur is chronic degenerative valve disease causing moderate mitral and trace tricuspid regurgitation. What is unexpected is the degree of left heart enlargement is moderate, while severe would be expected based upon the history. Moderate left atrial enlargement indicates the risk for spontaneous congestive heart failure is elevated in the future. Mild LV dysfunction is common in this breed secondary to valve disease. Early pulmonary hypertension is noted, which is likely secondary to chronic LA pressure elevation. No additional issues are identified.

In light of the clinical signs, chest radiograph findings and response to diuretic therapy, the diagnosis of congestive heart failure is supported and continued medications are warranted lifelong as below. As is mentioned previously, this is somewhat unexpected; however, if the patient is doing well, it is certainly reasonable to continue medications going forward. Monitoring of sleeping respiratory rates will be paramount to screen for congestive heart failure at home. Cough suppression to improve QOL can also be considered (hydrocodone, 0.2-0.4mg/kg up to q4-6h PRN) for any residual mechanical cough in the face of normal sleeping respiratory rates. The average survival time of canine patients with active pulmonary edema is 8-9 months on medications, however they generally are able to maintain a good quality of life for that period. Patient will always be at risk for recurrent CHF, development of arrhythmias/LA tear, syncope and/or sudden death in the future.

The ECG confirms frequent VPCs. VPCs are generated from abnormal conductive or fibrotic tissue in the ventricles of the heart muscle, and even frequent single VPCs will often cause no clinical signs in dogs. When sustained however, ventricular tachycardia can lead to symptoms such as lethargy and collapse.

When addressing arrhythmias, two things must be considered; 1. Is an underlying cause evident or is this primary disease? And 2. Is anti-arrhythmic therapy warranted?

VPCs are a very non-specific finding. They can be due to significant cardiac disease or be extra-cardiac in origin; i.e., due to pain, stress, inflammation, cancer, GI disease, DIC/sepsis, etc. In a Doberman with severe structural disease, this is the likely cause.

Electing to treat arrhythmias is based upon clinical signs and amount/degree of arrhythmia identified. Unfortunately, there is always an elevated risk for collapse and sudden death in any arrhythmic patient, and even on medications this risk unfortunately still persists.

Based strictly upon the amount of arrhythmia present on the available ECG, anti-arrhythmic therapy is not indicated (single beats only). Markers of malignancy (such as polymorphism, sequential VPCs, tight coupling interval, etc.) are low, although the frequency is concerning. Frequency may however decrease significantly outside of the stressful hospital environment, and a holter monitor would be the gold standard approach to this type of rhythm, to ensure a high-risk drug like sotalolol or mexiletine is indicated. This would allow monitoring of the rhythm throughout 24 hours of a normal day and help determine if treatment is indicated. If this is not a possibility for the owner, erring on the side of treatment may be reasonable as below. Fish oil supplementation is recommended for dogs with arrhythmias (500-1000mg of omega 3 and 6 once to twice daily). Monitor at home for collapse, exercise intolerance, and/or cough. Mild activity restriction is advised in arrhythmic patients.

If a holter monitor is elected, this will dictate whether therapy is needed and follow up protocol.

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Omega fatty acid supplementation and mild salt restriction may also be of some long-term benefit. Monitor for acute progression of the cough, labored breathing, exercise intolerance or collapse episodes in the future.

**SPECIES**

Canine

**PLAN**

Continue Pimobendan 0.3mg/kg PO q12h. Continue Furosemide 1-2mg/kg PO q12h. Institute Spironolactone 1-2mg/kg PO q12h. Pending BP >130mmHg, continue Benazepril 0.5m/kg PO q12h. Consider holter monitor as discussed. If a holter is declined, institute Sotalol 40mg PO q12h. Reassess ECG or ideally holter monitor in 1-2 weeks.

**BREED**

Doberman Pinscher

Monitor SRRs at home. Monitor renal values and BP every 3-4 months lifelong. Consider hydrocodone if needed for QOL.

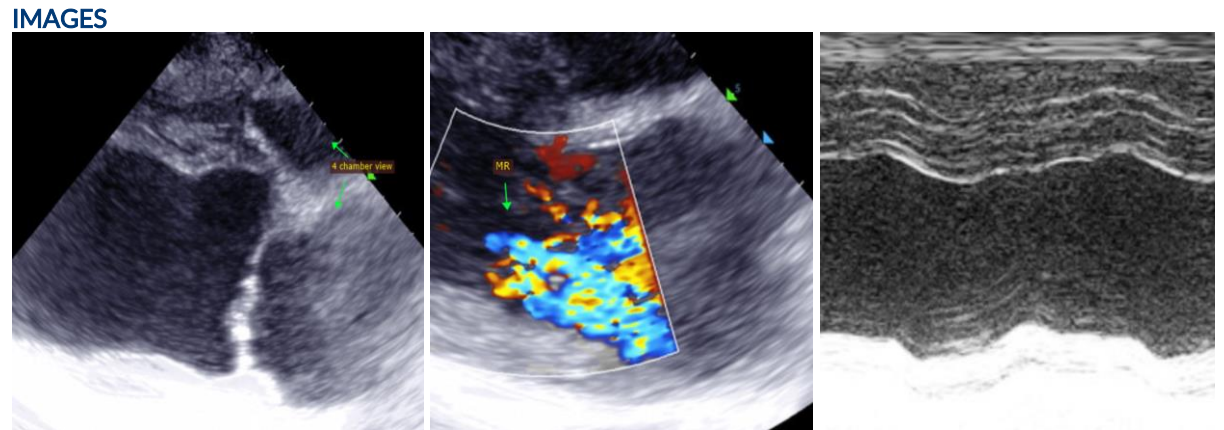
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Recommend conservative monitoring with a recheck echocardiogram in 6 months, sooner if any development of associated clinical signs occurs in the interim.

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**WEIGHT**

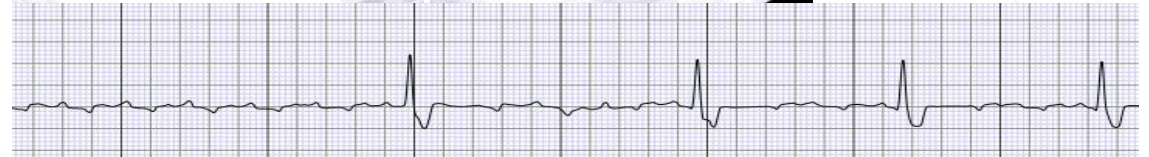
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

**REFERRING VET**

Dr. Neumeister

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance, please contact me.

**Maggie Machen Lamy, DVM**  
Diplomate of the American College of Veterinary Internal Medicine (Cardiology)  
info@sonopath.com

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