



**PATIENT**

Timber Hale

**SPECIES**

Canine

**BREED**

Jack Russell Terrier

**SEX**

Male Neutered

**AGE**

15 years

**WEIGHT**

7.8lbs

**INTERPRETED BY**

Maggie Machen  
Lamy, DVM  
DACVIM (Cardiology)

**IMAGING PERFORMED BY**

Pamela Harrigan,  
RDCS

**HOSPITAL NAME**

Wignall Animal  
Hospital

**REFERRING VET**

Dr. Colella

**INVOICE**

24549

**DATE**

6/2/22

**PRESENTING CLINICAL SIGNS**

History: Recheck echo. History normal cardiac structure and function with trivial MR noted on prior echocardiogram. Current presentation: Timber has episodes of open-mouthed breathing where the tongue and gums turn purple. These episodes are noted when he is stressed. Signs resolve quickly when he calms down. Continues to have a good appetite; activity level appropriate for his age and arthritis. On exam, murmur is louder, now grade II/VI parasternal. BP: 144, 150, 150mmHg. Current medications: Cyclosporine 25 mg SID; Pentoxifylline 100 mg TID; Ursodiol 30 mg BID; Gabapentin 25 mg BID; Levothyroxine 0.2 mg BID; Tylosid 50 mg SID; Denamarin 8,75 mg SID; Omeprazole. \*No sedation for study.  
-Pertinent previous echo measurements (1/2021 MML): LA 1.4 cm; LA:Ao 1.2 cm; LV 2.0; normal chamber sizes; trace MR; no TR; normal RA/RV dimensions.

**ECHOCARDIOGRAM FINDINGS**

2D, m-mode, color flow and Doppler imaging is available.  
**Left ventricle:** The LV diameter is normal with adequate myocardial function. LV wall thicknesses are normal.  
**Left atrium:** The left atrium is normal in diameter.  
**Mitral valve:** The mitral valve is normal with no prolapse into the left atrial lumen. Trivial mitral regurgitation.  
**Aortic valve/Aorta:** The aortic valve is normal in morphology and mobility. Normal aortic outflow velocity; laminar flow. No aortic insufficiency.  
**Right ventricle:** Moderate RV dilation with hypertrophy consistent with pressure overload. Septal flattening in end-systole.  
**Right atrium:** Moderate to severe RA dilation.  
**Tricuspid valve:** The tricuspid valve appears thickened with moderate tricuspid regurgitation; velocity consistent with severe pulmonary arterial hypertension.  
**Pulmonic valve/Pulmonary artery:** The pulmonic valve is mildly thickened with normal mobility No pulmonic insufficiency. Normal RVOT velocity; laminar flow. Significant MPA and branch dilation.  
**Pericardium/other:** No pericardial or pleural effusion noted. No obvious cardiac masses.  
**Heart rhythm:** ECG reveals a sinus rhythm with an average HR of 140bpm.

**2-Dimensional Measurements**

Ao diam (cm)	1.2
LA diam (cm)	1.37
LA:Ao (Swe)	1.1
IVS thickness (cm)	0.6
LVID diastole (cm)	1.9
PW thickness (cm)	0.6
LVID systole (cm)	0.9
FS (%)	54

**Doppler Measurements**

PV Vmax (m/s)	0.51
AoV Vmax (m/s)	1.1
MR Vmax (m/s)	
TR Vmax (m/s)	4.7
TR PG (mmHg)	88

**INTERPRETATION OF THE FINDINGS**

Interesting case. This study shows evidence of severe pulmonary hypertension (PAH), with an estimated systolic pulmonary arterial pressure >90mmHg (normal being <25mmHg). This is causing severe hypertrophy and dilation of the right heart and MPA (indicating severe right-heart pressure overload) and moderate TR. The left heart appears essentially normal, with normal dimensions. This is in stark contrast with the prior study, albeit nearly 1.5 years ago. No TR was noted at the time and no right heart enlargement.



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The underlying genesis of PAH is poorly understood in cases other than prior or active heartworm infestation, though it occurs with increased frequency in a variety of forms of chronic lung disease and in patients with idiopathic pulmonary fibrosis. Without a chronic respiratory history, the cause remains open. Clinical signs of weakness, heavy breathing, cyanosis, and exertional syncope are attributed to severe PAH as is seen here. Patients with this degree of PAH can eventually develop right-sided congestive heart failure (ascites/pleural effusion), debilitating cyanosis and labored breathing/exertional syncope if poorly controlled.

Going forward, medical management of PAH is recommended utilizing Sildenafil and Pimobendan therapy. Primary respiratory therapy is recommended if symptoms are present, as primary respiratory disease is a common underlying cause particularly given the breed.

Unfortunately, this tends to be a progressive issue with a guarded to poor prognosis, particularly once syncope and CHF develop. Patient will always be at risk for right-sided CHF, worsening syncope/dyspnea and or sudden death going forward.

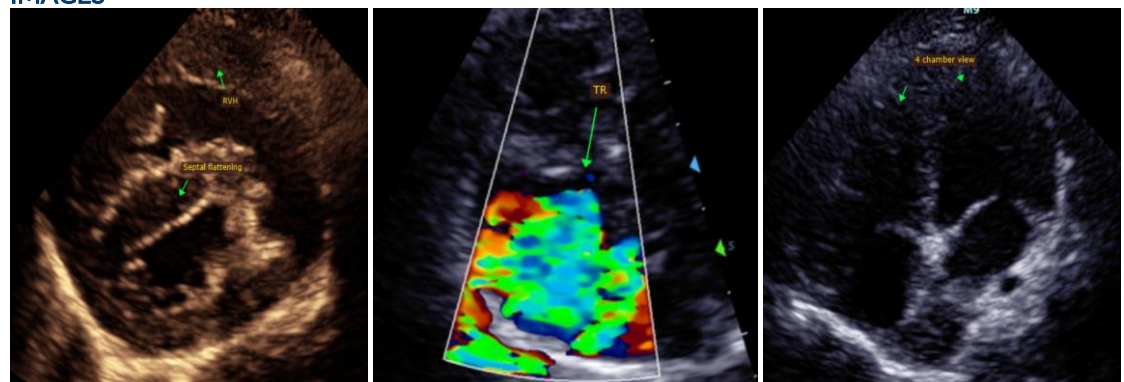
**RECOMMENDATIONS**

- Institute sildenafil 1-2mg/kg PO q8h.
- Institute Pimobendan 0.3mg/kg PO q12h.
- Baseline CXR, heartworm test, etc. recommended.
- If indicated, institute Hydrocodone with homatropine for QOL; 0.2-0.4mg/kg up to q4-6h PRN.
- Pending response, consider ancillary options including theophylline, anti-inflammatory prednisone, inhaled fluticasone, home flow by O2, course of Baytril for cough flares, etc.
- Omega fatty acid supplementation and mild salt restriction may be of some long-term benefit.
- Elective anesthesia is not advised.
- Lifelong activity/stress restriction is advised.
- Monitor for development of a cough, labored breathing, exercise intolerance or collapse episodes.

**PLAN**

- Recommend conservative monitoring with a recheck echocardiogram in 6 months, sooner if any development of clinical signs.

**IMAGES**





**PATIENT**

Timber Hale

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.

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Thank you for this referral. This report was generated using transcription software, and minor dictation errors may be present. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance, please contact me.

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Jack Russell Terrier

Maggie Machen Lamy, DVM  
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