



**PATIENT PRESENTING CLINICAL SIGNS**

Ralphie Eisner

History: 11 yo mn boxer mix, long history of chronic skin issues/pododermatitis. Recently-reverse sneezing, unilateral nasal discharge-resolved on antibiotics, panting, tachypnea with increased effort, lethargy, hypertension-systolic >200 (in hospital/stressed) on Amlodipine 5mg SID. Also on Apoquel and Levothyroxine.

**SPECIES**

Canine

Abnormal PE/Chem/CBC/UA Results: Mild ALP elevation.

**BREED ULTRASONOGRAPHIC EXAMINATION OF THE HEART**

Boxer Mix

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. Trivial **mitral** valve insufficiency was noted. The **left ventricle** presented concentric hypertrophy with **myocardial** remodeling. However, **contractility** and internal diameter was normal. The **left ventricular outflow** tract demonstrated normal laminar flow and subjective structural integrity. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted. **Tricuspid** valvular assessment demonstrated adequate linear morphology and kinesis. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonary outflow** tract assessment revealed normal valve structure, laminar flow, and diameter (approx.1:1 pa/ao ratio). No visible **pericardial** or free pleura fluid was noted. The cranial **mediastinum** and **pericardial** and **extra-cardiac** regions were free of masses in the visible window.

**SEX**

Neutered male

**AGE**

11 years

**WEIGHT**

79 lbs

**INTERPRETED BY**

Eric Lindquist, DMV  
DABVP, Cert. IVUSS

**IMAGING PERFORMED BY**

Dr. Petrone

**HOSPITAL NAME**

Long Branch AH

**REFERRING VET**

Dr. Petrone

**INVOICE**

93096

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CANINE	MR	TR	LA/AO	LA/AO	FS	EF	EPSS
<b>CARDIAC PARAMETERS</b>	<b>VMAX</b> (m/s)	<b>VMAX</b> (m/s)	(Boon method)	(Heart Base; Swe)	(%)	(%)	(cm)
<b>NORMAL PARAMETER</b>	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
<b>PATIENT</b>			1.2	1.4	45		0.1
CANINE	HR	AV	PV	BODY WEIGHT	LA	LVIDd	LVIDs
<b>CARDIAC PARAMETERS</b>	(BPM)	<b>VMAX</b> (m/s)	<b>MAX</b> (m/s)		2D short axis Base view (cm)	Avg; 2D and m-mode short axis (cm)	Avg; 2D and m-mode short axis (cm)
<b>NORMAL PARAMETER</b>	50-100	0.7-1.7	0.7-1.6				
<b>PATIENT</b>			1.2	79 lbs	3.4 max	3.0	

**ULTRASONOGRAPHIC FINDINGS**

Concentric hypertrophy of the left ventricle with myocardial remodeling. However, contractility and internal diameter was normal.

Mitral insufficiency with centralized jet, compensated.

Compensated stage B1 valvular disease.



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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

There was no evidence of volume overload noted. There was mild to moderate filling of the left atrium. Systemic hypertension may be playing a role in the left ventricular hypertrophy. The heart is currently compensated. However, if the patient can undergo blood pressure measurements in a non-white coat effect environment or after Torbutrol therapy and reassessment if systolic pressure is greater than 160 then further adjustment of the hypertension is indicated. EKG is warranted. No primary cardiac therapy is recommended at this time. Causes of hypertension such as adrenal and renal disease should be ruled out with abdominal sonogram. Recheck echocardiogram in 6 months.

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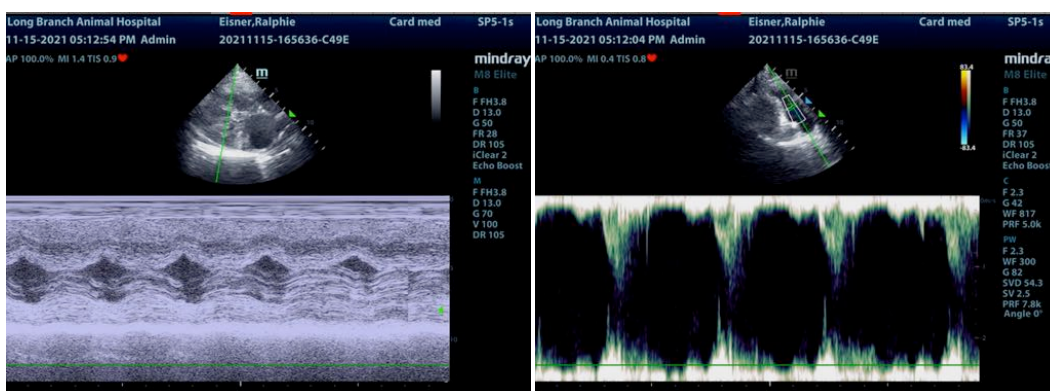
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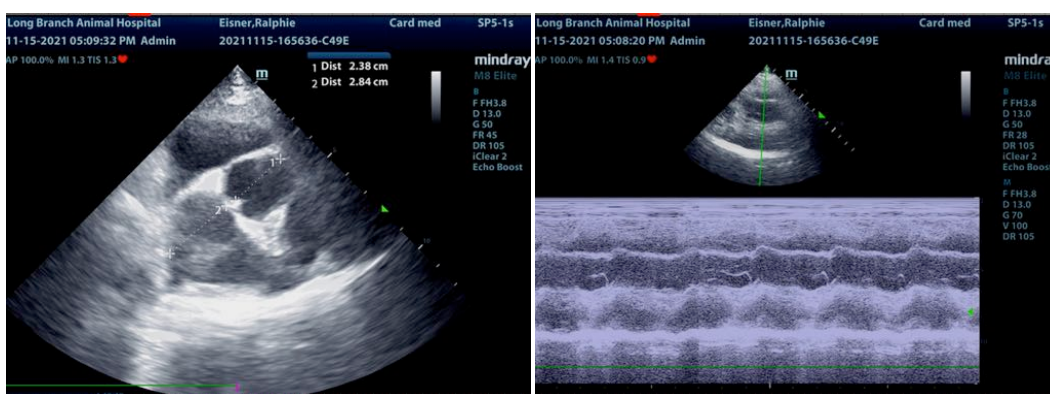
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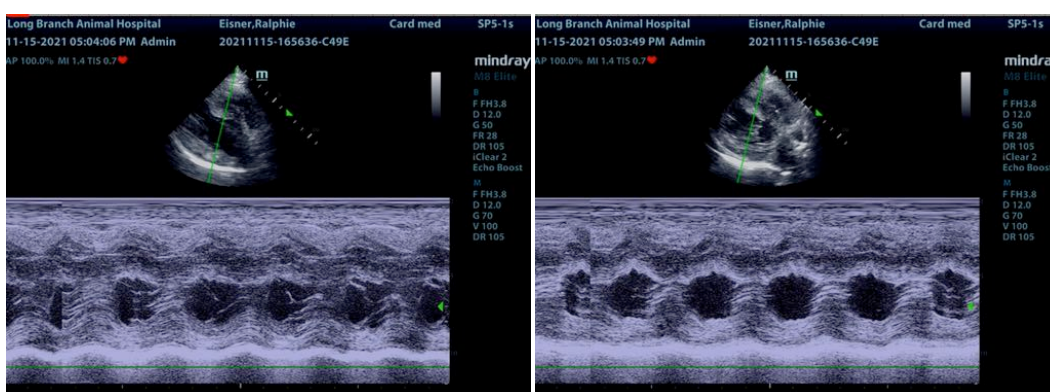


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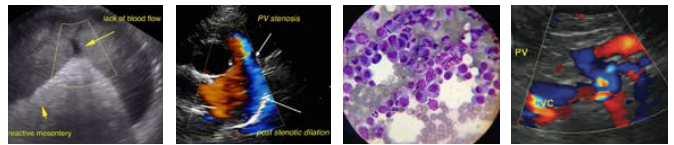
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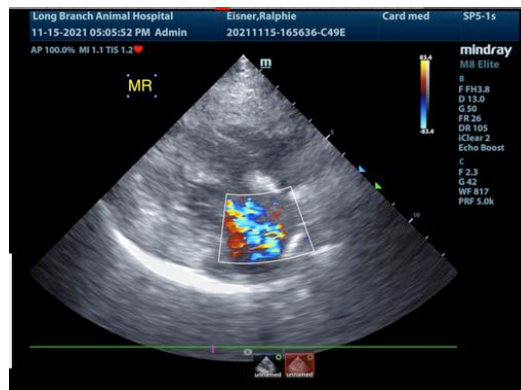
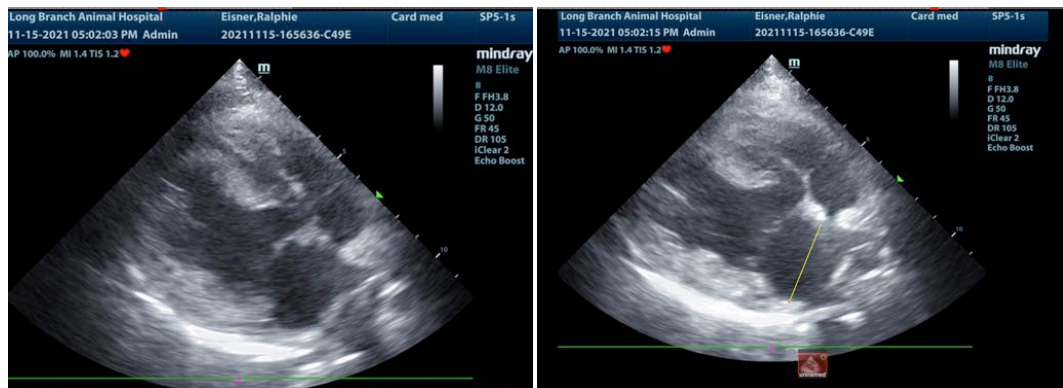
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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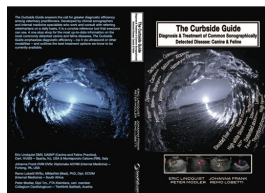
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Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

**Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com**  
[info@SonoPath.com](mailto:info@SonoPath.com)

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The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by [SonoPath.com](http://sonopath.com) Lindquist, Frank, Lobetti, and Modler.

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An essential quick guide for every general practitioner and sonographer.

<https://sonopath.com/products/curbside-guide-editing-due-release-12012015>

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**Systemic Hypertension**

<http://www.sonopath.com/Hypertension>

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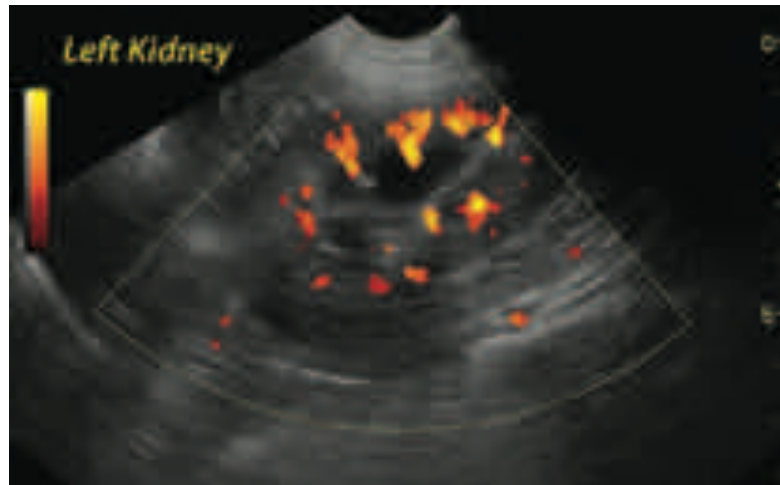
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Long axis of the left kidney in a hypertensive dog with nephritis and protein losing nephropathy. The Power Doppler shows a high perfusion intensity within the interlobar and arcuate vessels. Also note the abnormal presence of excessive renal cortical perfusion. Acute PLN cases often have unremarkable structural renal changes sonographically.

**Description:** Systemic hypertension refers to the persistent elevation of systemic blood pressure (BP) and is classified as either primary (essential or idiopathic) or secondary. Persistent damage caused by hypertension is typically noted in effector organs such as the eyes, central nervous system (CNS), heart, and kidneys. In cats, the most common diseases associated with systemic hypertension are chronic kidney disease and hyperthyroidism, whereas the same condition in dogs more typically results in chronic kidney disease (especially proteinuric renal disease), hyperadrenocorticism, diabetes mellitus, and pheochromocytoma. Less typical causes of systemic hypertension in both species include hyperaldosteronism, acromegaly, and the use of hypertensive medications (e.g. phenylpropanolamine and excessive thyroxine supplementation).

**Clinical Signs:** Although hypertension is often clinically silent, ophthalmologic changes are usually the most obvious signs. Ophthalmologic changes secondary to hypertension are better described in cats than in dogs, and include intraocular hemorrhage, hypertensive retinopathy, hypertensive choroidopathy, and hypertensive optic neuropathy. CNS signs can include seizures, vascular accidents, and changes in mentation. Congestive heart failure secondary to hypertensive cardiac changes is rare, but affected animals may exhibit increased sensitivity to fluid administration.

**Diagnostics:** Blood pressure values obtained during diagnostic evaluation must be assessed in conjunction with other clinical findings, such as retinal detachment as well as a history of polyuria and polydipsia, as these associated findings may point to the cause of the hypertension or indicate hypertension-associated organ damage. A single high blood pressure value should never be used to diagnose systemic hypertension in the absence of other clinical data, as patient distress or stress may artificially elevate blood pressure. Current recommendations suggest that a systolic blood pressure greater than 160 mm Hg is indicative of hypertension; however, the use of antihypertensive medications may not be warranted if blood pressure elevations are mild and underlying etiologies addressed. The minimum database should include physical examination, fundoscopic examination, CBC, serum chemistry profile, urinalysis with urine protein quantification, and serum T4 assay in cats. Measuring the blood pressure in separate moments throughout the day in a quiet, non “white coat effect” environment, in a home environment, or potentially with light opioid sedation (Buprenorphine



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0.01 mg/kg) can also be considered when looking for hypertension. Empirically, in our experience, this opioid approach tends to be an effective approach when assessing for hypertension in excited patients. However, a complete comparative study is warranted.

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Canine

**Treatment:** First, the use of any hypertensive medications should be discontinued immediately. Drug groups that can be used to control hypertension include angiotensin-converting enzyme (ACE) inhibitors (enalapril or benazepril 0.5 mg/kg PO BID), calcium channel blockers (amlodipine 0.625 mg PO Q24hr for cats and 0.1 mg/kg PO Q24hr for dogs), and beta blockers (atenolol 6.25-12.5 mg PO BID for cats and 0.25-1 mg/kg PO BID for dogs). Medications from different groups may be additionally administered if the original medication is not sufficiently controlling the condition.

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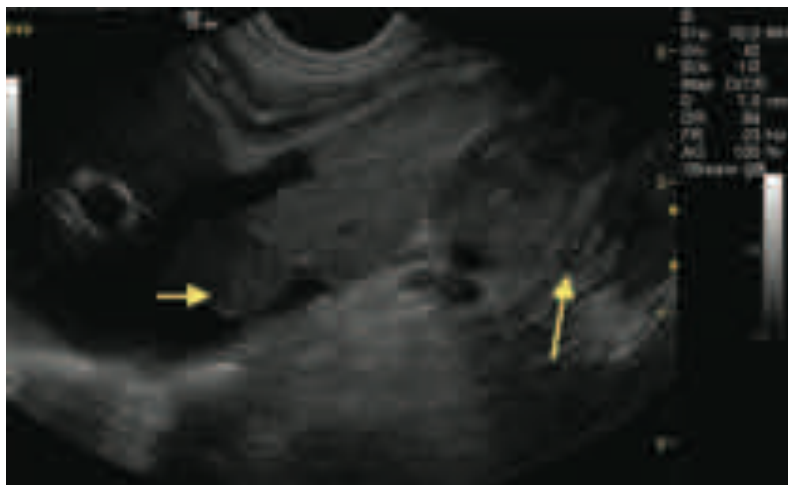
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Long axis of the right adrenal gland and caudal vena cava in a dog with a pheochromocytoma and hypertension. The enlarged heterogenous adrenal gland is seen on the image right (large arrow). There is tumoral invasion (small arrow) into the caudal vena cava via the phrenicoabdominal vein. The echogenic tumor material occupies the caval lumen entirely. Note the significant centrifugal mass effect of the intravascular tumor on the vessel wall as indicated by focal widening of the caval diameter. Part of the right liver lobes and descending duodenum are seen on the image left and in the near field.



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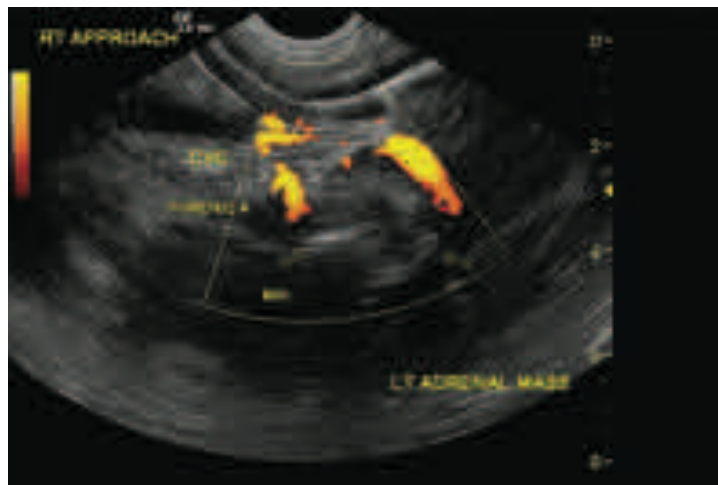
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Long axis of a left-sided adrenal mass as imaged using a right lateral sonographic approach in a hypertensive dog. The enlarged heterogenous left-sided adrenal tumor is seen deep to the descending duodenum and caudal vena cava. Note the presence of multifocal mineralizations (MIN) within the tumor and the left phrenicoabdominal artery passing atop of the mass lesion.

**References:**

Brown S, et al. Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. *J Vet Intern Med* 2007;21:542-58.

Michell AR, Bodey AR, Caulfield M. Evolution, essential hypertension and the high arterial pressures in certain athletic breeds of dogs. *Vet J* 2011; 88:125-27.

Jepson RE, Elliott J, Brodbelt D, Syme HM. Effect of control of systolic blood pressure on survival in cats with systemic hypertension. *J Vet Intern Med* 2007;21:402-09.

Steele JL, Henik RA, Stepien RL. Effects of angiotensin-converting enzyme inhibition on plasma aldosterone concentration, plasma renin activity and blood pressure in spontaneously hypertensive cats with chronic renal disease. *Vet Ther* 2002;3:157-66.