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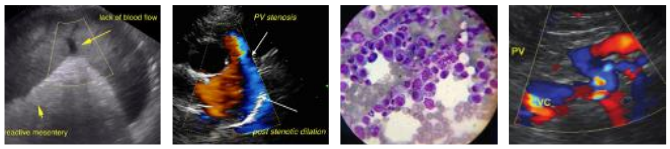

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1-800-838-4268 info@sonopath.com

PATIENT

Greta Wehmeyer

SPECIES

Canine

BREED

English Bulldog

SEX

Spayed Female

AGE

6 Years

WEIGHT

N/A

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Diane McFadden

HOSPITAL NAME

Newton VH

REFERRING VET

Dr. Pierson

INVOICE

12621

DATE

8/20/21

PRESENTING CLINICAL SIGNS

History: peritoneal effusion, vomiting, hepatomegaly (liver values normal), cardiomegaly (no murmur). not on any meds.

Abnormal PE/Chem/CBC/UA Results: glob 4.5

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

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	--	--	1.28	1.27	55	87	NM
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				
PATIENT	142	2.00	1.50	--	2.4	2.71	--

Cardiac Presentation

The cardiac presentation presented normal left atrial and left ventricular volume with paradoxical septal motion, arrhythmogenic activity and severe right sided volume overload with a 2.5:1 right atrial to left atrial ratio and a 1:1 ratio with the right ventricle and left ventricle. Tricuspid insufficiency was noted. No evidence of pulmonic stenosis noted. The left ventricular septum was flattened owing to excessive right sided pressures. Persistent arrythmia noted.

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The left kidney measured 7.5 cm. The right kidney measured 7.41 cm.

Adrenal Glands

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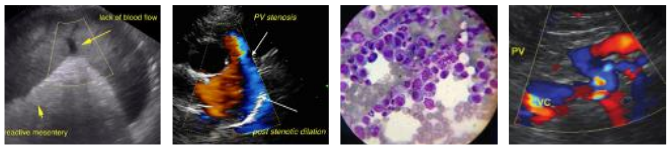

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Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 3.27 cm x 0.76 cm at the caudal pole and 0.84 cm at the cranial pole. The right adrenal gland measured 3.76 cm x 1.45 cm at the cranial pole and 0.86 cm at the caudal pole.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The **liver** was mildly swollen, uniform. The hepatic veins and vena cava were dilated with secondary ascites (owing to right sided heart failure). The vena cava measured 1.6 cm in width at the diaphragm. The gallbladder and common bile duct were unremarkable.

Gastrointestinal

Normal **GI**, yet stomach was deviated caudally owing to hepatomegaly.

Pancreas

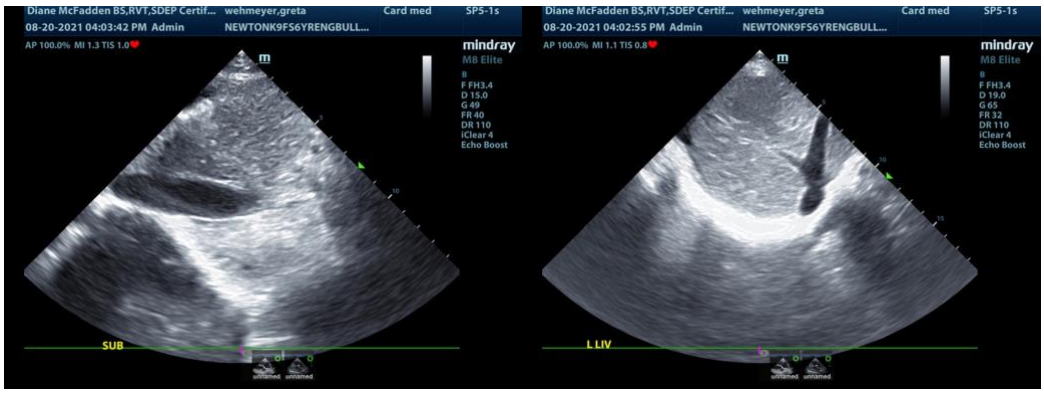
The **pancreas** was enhanced in echogenicity owing to the long-standing ascites.

ULTRASONOGRAPHIC FINDINGS

- Right sided heart failure
- Moderate amount of ascites owing to right sided heart failure
- Swollen liver with dilated hepatic veins and vena cava
- Pancreas, enhanced in echogenicity owing to long standing ascites

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The vomiting is likely owing to gastrointestinal hypoxia. I recommend pimobendane at 0.3 mg per kg BID, Lasix 2 mg per kg BID, spironolactone at 1-2 mg per kg BID and Ace-Inhibitor at 0.5 mg per kg SID. EKG warranted given the arrythmia noted. This patient is at risk for sudden death. Recheck echo in 1 week. I do not recommend exertion in this patient.



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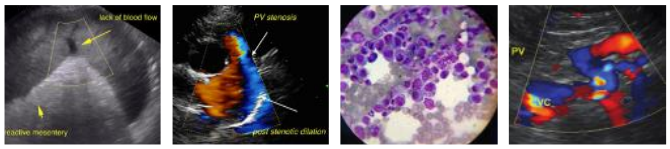

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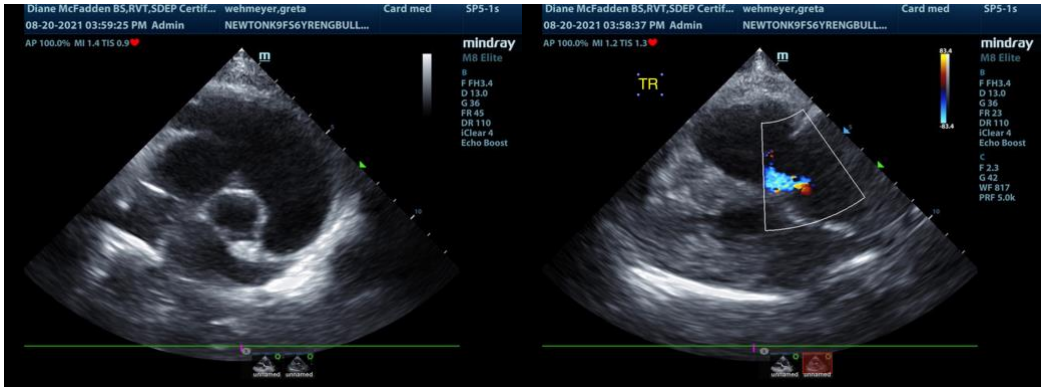

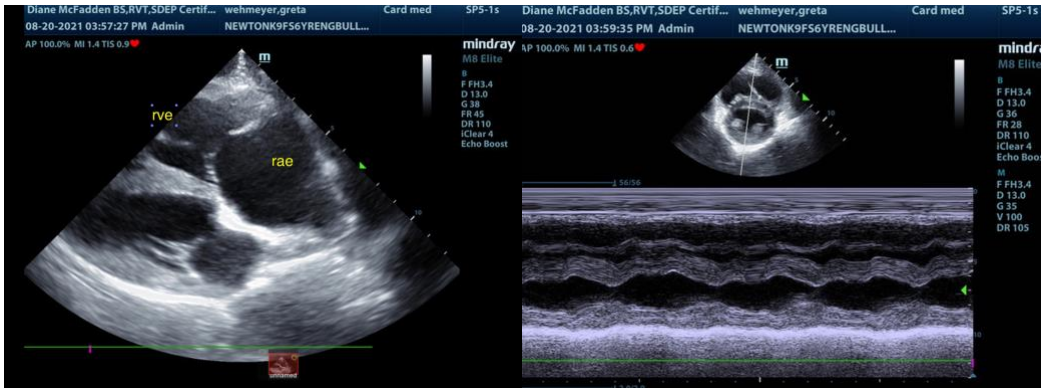

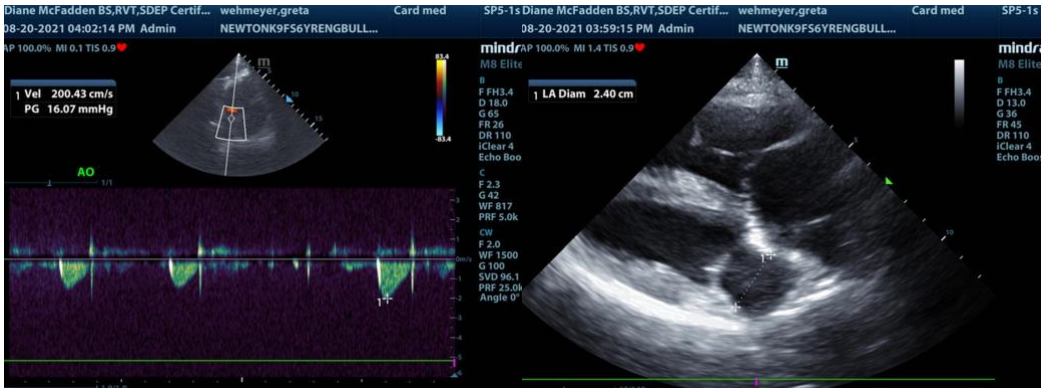

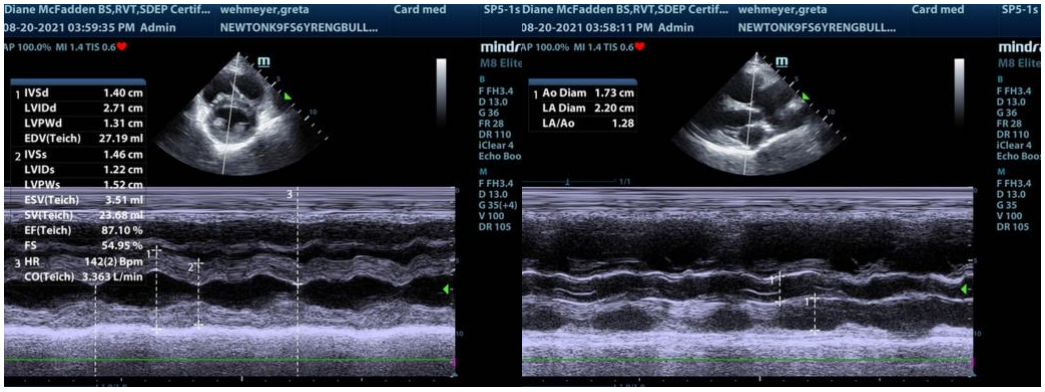

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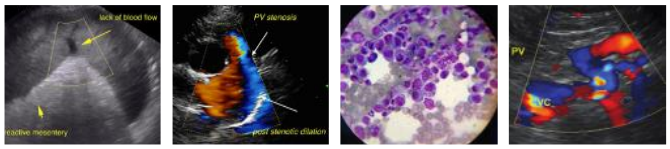

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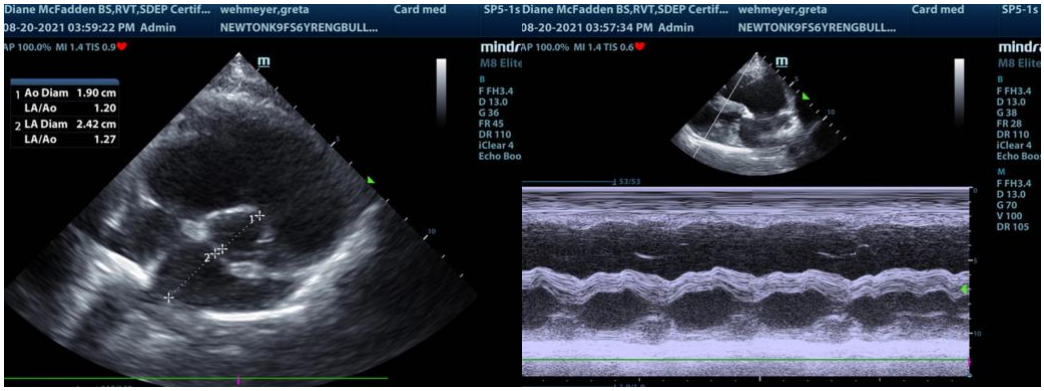


The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

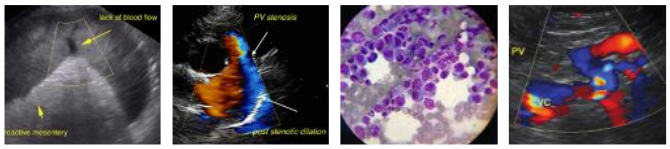
Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com
info@SonoPath.com

Right Heart Disease-General Considerations

http://www.sonopath.com/RightHeartDisease

Description: Right heart disease is often an incidental finding, which can be either cardiogenic or secondary to respiratory or systemic disease. The coughing patient with right heart disease may present with primary respiratory disease (i.e., bronchial collapse, collapsing trachea, pneumonitis) and suffer from secondary pulmonary hypertension (PHT). Concurrent mitral valve disease and chronic left-sided congestive heart failure (CHF) might also lead to PHT. The dyspeic patient with right heart enlargement might have pulmonary hypertension due to airway disease, chronic CHF, parenchymal lung disease (e.g. pulmonic fibrosis), or a cardiac shunt with secondary PHT and shunt reversal.

Primary cardiac causes of right heart enlargement include: tricuspid dysplasia/degeneration; pulmonic stenosis; pulmonic insufficiency; atrial or septal defects; patent ductus arteriosus; right auricular masses; and pericardial peritoneal diaphragmatic hernias. The second most common cause of right-sided enlargement is secondary PHT, which results in high-velocity tricuspid insufficiency (TR vel.>2.8 m/sec) and pulmonic insufficiency due to diseases that cause increased pulmonary vascular resistance or increased pulmonary wedge pressures. The most common cause of secondary PHT is left-sided heart failure (LHF), which presents radiographically as a more globoid-shaped heart with marked left atrial and ventricular enlargement. There are also signs of left-sided CHF as opposed to a simple prominent cranial waist or reverse D radiographic presentation.



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Secondary, non-cardiac causes of PHT include: acute or chronic respiratory disease; pulmonary thromboembolic disease; thoracic neoplasia; excessive thoracic fat deposition (e.g. Pickwickian syndrome, which leads to chronic hypoxia); brachycephalic syndrome; high altitude disease; heartworm disease; and primary vascular disease.

Clinical Signs: The most common presenting symptoms of right heart disease are collapse, syncope, intermittent or constant acute respiratory distress (e.g. thromboembolic disease), and exercise intolerance.

Diagnostics: Physical examination may reveal a right-sided apical heart murmur and/or a cranial left heart murmur, a split S2, jugular distension, ascites, and signs consistent with respiratory disease (i.e., cough, wheeze, tracheal collapse, tachypnea). Radiographic findings may reveal an enlarged right atrium, right ventricle, and/or primary/secondary branches of the pulmonary artery. In cases of PHT, an enlarged or engorged pulmonary artery is often present. Tortuous arteries or those that suddenly terminate can indicate the presence of thromboembolic disease or heartworms. An interstitial pattern might indicate the presence of pulmonary parasitism or primary interstitial lung disease. Pulmonic stenosis is suspected if the pulmonic segment is enlarged. ECG findings include tall P and S waves with a right axis shift.

Treatment: Please refer to the chapter “Pulmonary Hypertension” for therapeutic recommendations.

References:

Oyama MA, Rush JE, Rozanski EA, et al. Assessment of serum N-terminal pro-B-type natriuretic peptide concentration for differentiation of congestive heart failure from primary respiratory tract disease as the cause of respiratory signs in dogs. *J Am Vet Med Assoc* 2009;235:1319-25.

Rozanski E. Interstitial lung disease in small animals. Proceedings from American College of Veterinary Internal Medicine Forum, Denver, CO, June 15-18, 2011.

Zoia A, Augusto M, Drigo M, Caldin M. Evaluation of hemostatic and fibrinolytic markers in dogs with ascites attributable to right-sided congestive heart failure. *J Am Vet Med Assoc* 2012;241:1336-43.