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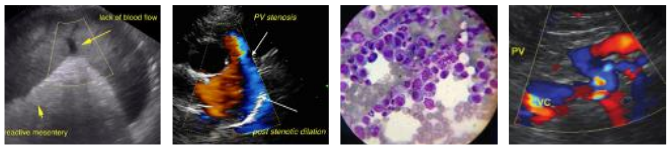

SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT

Sundae Bergqvist

SPECIES

Canine

BREED

French Bulldog

SEX

Spayed Female

AGE

10 Years

WEIGHT

N/A

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Jessica Miller

HOSPITAL NAME

Cresskill AH

REFERRING VET

Dr. Khodari

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PRESENTING CLINICAL SIGNS

History: Collapsing episode. Grade III/VI heart murmur. X-rays at outside facility: prominent pulmonary artery and RA Current meds: cerenia, clavamox, gabapentin, apoquel

Abnormal PE/Chem/CBC/UA Results: ALT 248, PCV 64%, BUN 28

ULTRASONOGRAPHIC EXAMINATION OF THE HEART

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	5.0	3.4	1.3	1.5	24	51	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	157	.99	.80	--	3.57	1.79	--

Cardiac Presentation

The cranial and caudal **mitral** valve leaflets presented vegetative thickening consistent with endocardiosis. Doppler indicated measurable insufficiency. The **left ventricle** presented thicknesses with linear contour and was not dilated nor restricted. The **myocardium** presented normal echogenicity without subjective evidence of significant fibrotic or ischemic disease. **Contractility** of the ventricular walls was adequate and in normal range for this patient evidenced by the fractional shortening measurement and subjective evaluation of the different regions of the myocardium. 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 or chamber overload. **Tricuspid** insufficiency noted at 3.4 m/s. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonic** tract assessment revealed normal valve structure, laminar flow, and diameter (approx.1:1 pa/ao ratio). A 4.0 cm x 2.8 cm heart base mass was noted in this patient deriving from the aorta and deviating the aorta superimposing upon the left atrium. No pericardial effusion was noted. Hepatic veins were dilated in this patient suggestive for potential emerging right sided failure.

ULTRASONOGRAPHIC FINDINGS

- Heart base mass, likely chemodectoma/aortic body tumor
- concurrent mitral and tricuspid insufficiency
- Early pulmonary hypertension

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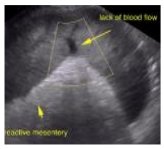
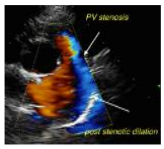
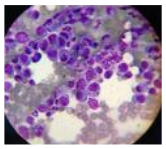
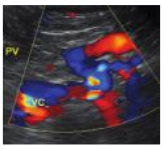

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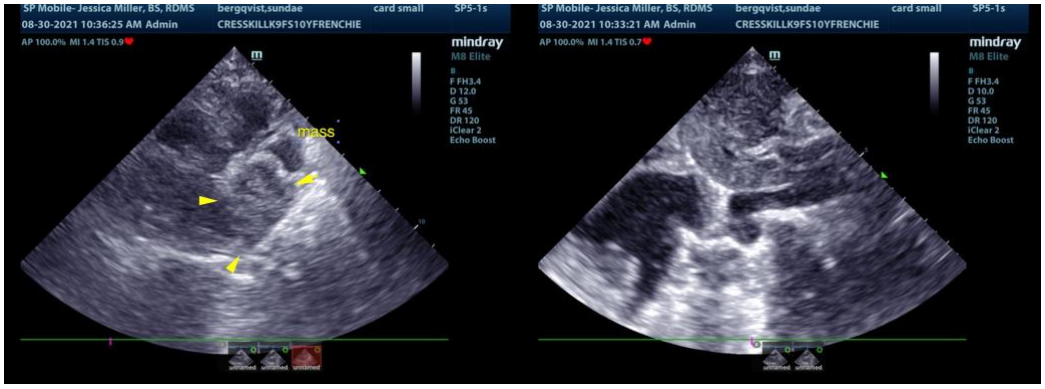
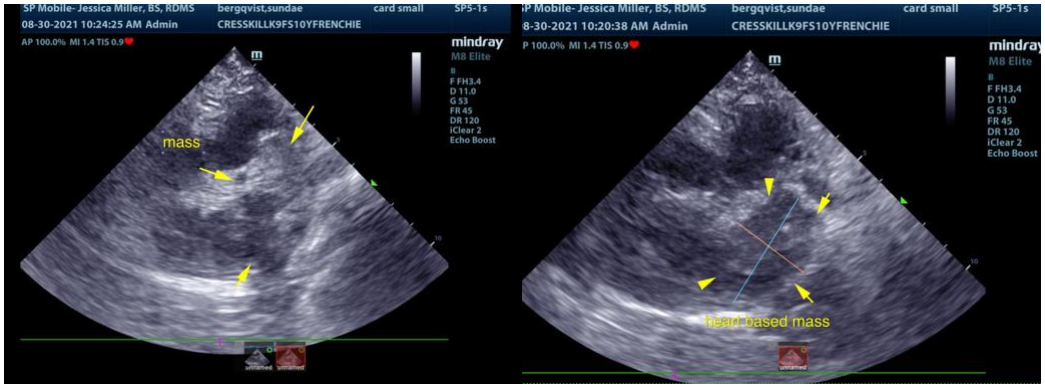
INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

No specific therapy in this patient, unless exercise intolerance is an issue as pulmonary hypertension may develop into a clinical issue. The mass is expansive, yet no associated pericardial effusion was present. Oncological referral recommended, however, opportunities for treatment are minimal. These are typically slow growing masses and initiate to cause clinical signs when deviation of the aortic outflow or venous inflow occurs. Given the collapsing episode, pulmonary hypertension may be the underlying issue, paroxysmal arrhythmia or systemic hypertension. I recommend blood pressure measurements and holter monitor would be ideal to assess and control any arrhythmogenic activity. If neither of these are abnormal then sildenafil trial could be considered at 1 mg per kg BID increasing to 1.5 mg per kg BID.





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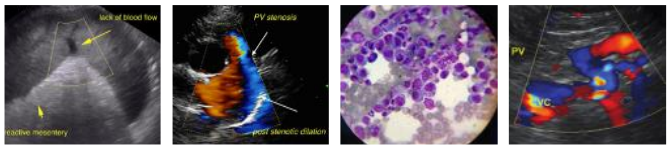

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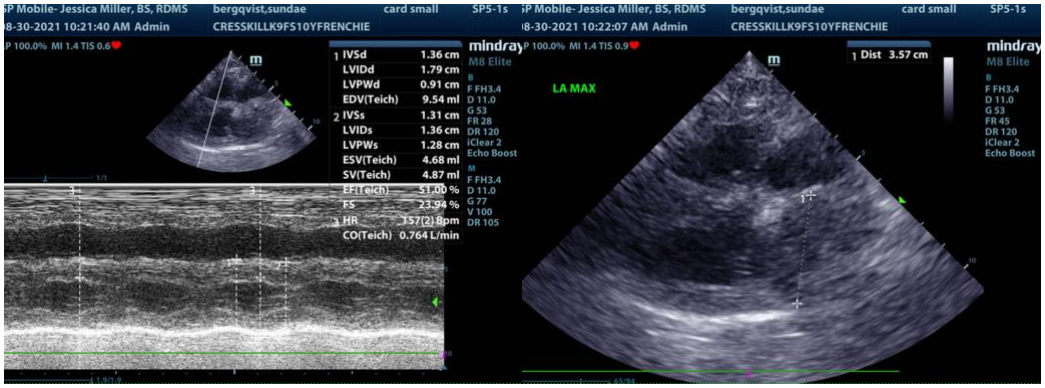
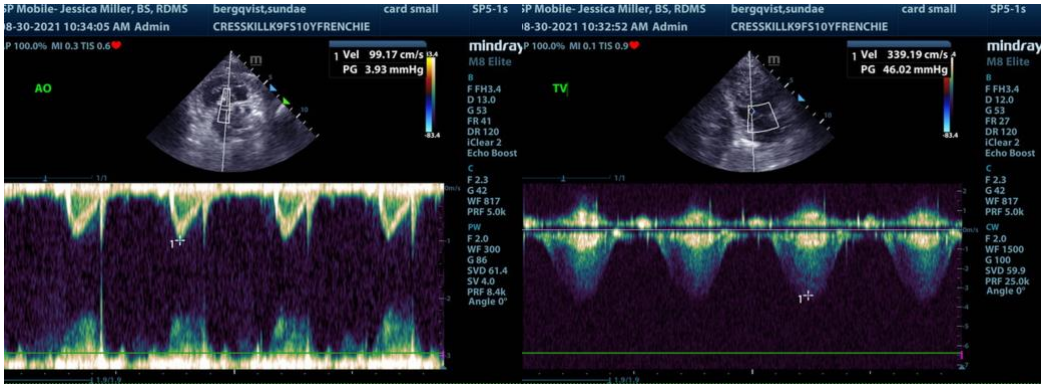




The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

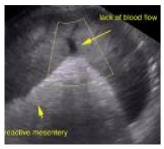
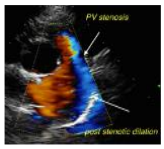
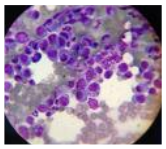
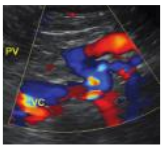
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

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

Pericardial Effusion and Cardiac Neoplasia

http://www.sonopath.com/CardiacNeoplasiaEffusion

Description: The pericardium is a fibrous sac that encloses the heart and the great vessels—aorta, pulmonary artery, proximal pulmonary veins, and vena cava—located at the heart's base. It is attached caudally to the diaphragm and under normal circumstances contains 1-15 mL of fluid. The latter is comprised of phospholipids that lubricate the heart and allow it to expand and contract without generating friction. The pericardium also fixes the heart, prevents excess motion, and links the diastolic distensibility of the ventricles, thus limiting the degree to which either the left or the right ventricle will distend during diastole. When there are acute changes in venous return (i.e., during exercise), the pericardium plays a critical role in limiting ventricular filling. In cases of chronic cardiac enlargement, the pericardium also becomes distended, and its ability to limit ventricular filling, especially when the heart is at rest, becomes

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compromised. Pericardial tamponade occurs when there is a rapid accumulation of fluid and the pressure inside the pericardium increases significantly. With tamponade, ventricular filling is restricted and cardiac output is decreased. The right atrium and ventricle are the most vulnerable to this condition as these compartments have thinner walls and a lower pressure.

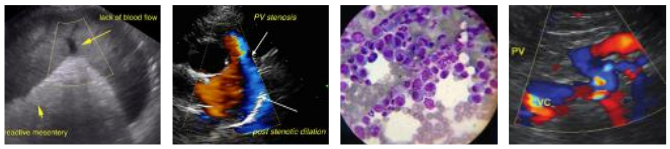
Etiology: Causes of pericardial effusion include:

- Neoplasia
  - Right atrial (RA) hemangiosarcoma
  - Heart base (aortic body) tumors
  - Mesothelioma
  - Rhabdomyosarcoma
  - Ectopic thyroid carcinoma
  - Metastatic neoplasia
- Idiopathic
- Congestive heart failure
- Peritoneal-pericardial diaphragmatic hernia
- Pericardial cyst
- Hypoalbuminemia
- Infectious pericarditis (bacterial, *Coccidioides immitus*)
- Feline infectious peritonitis
- Left atrial tear secondary to valvular disease
- Coagulopathy

The majority of neoplastic masses consist of hemangiosarcoma and heart-based tumors (chemodectomas or ectopic thyroid adenocarcinoma). Idiopathic pericardial effusion is a diagnosis of exclusion; the effusion is typically hemorrhagic. Approximately 50% of dogs will be cured with a single pericardiocentesis, while some dogs will require multiple pericardiocenteses as well as surgery. A peritoneal-pericardial diaphragmatic hernia is a congenital hernia seen in dogs and cats in which the abdominal contents (i.e., liver, small intestine, spleen, stomach) herniate into the pericardial sac. Constrictive pericarditis is an uncommon condition in which a non-distensible, thickened, fibrotic pericardium develops over time.

Clinical Signs: One will observe the following clinical signs, which often present in combination: ascites, lethargy, exercise intolerance, pale mucous membranes, weak pulses, *pulsus paradoxus*, and respiratory distress.

Diagnostics: Survey radiographs will reveal hepatomegaly, cardiomegaly (generalized or sectorial globoid), and small pulmonary vessels. Pulmonary edema is typically not found, although one may discover concurrent pulmonary metastatic disease. An ECG will show electrical alternans or small complexes, but often the changes are very subtle and difficult to detect.



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Echocardiography is usually considered the gold standard for diagnosing pericardial effusion. Findings include:

- Anechoic space between the heart and the pericardium.
- Abnormal side-to-side cardiac motion.
- Decreased chamber size (right ventricle [RV] and left ventricle [LV]).
- Presence of a pericardial or cardiac mass.
- Tamponade with early diastolic RA and RV collapse.

Cytology is helpful in the diagnosis of lymphoma, septic pericarditis, and idiopathic effusion, but not in cases of neoplasia.

According to a study that found troponin I levels to be higher in dogs with neoplastic pericardial effusion, the cardiac troponin I assay can be helpful in the diagnosis hemangiosarcoma.

Prognosis:

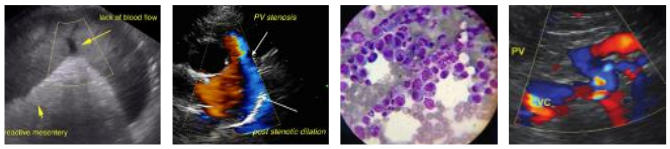
- Cardiac hemangiosarcoma: < 8 months with surgical debulking and chemotherapy.
- Chemodectoma (aortic derived): MST 730 days post pericardectomy.
- Idiopathic: 50% complete resolution post cardiocentesis; curative with pericardectomy, which can be done via thoracotomy, or thorascopy, or using a balloon to tear the pericardium.
- Mesothelioma: Poor.
- Restrictive pericarditis: Poor, especially when the pericardium has not been surgical stripped.

References:

Cagle LA, Epstein SE, Owens SD, et al. Diagnostic yield of cytology analysis of pericardial effusion in dogs. *J Vet Int Med* 2014;28:66-71.

Feigenbaum H. Pericardial disease. In: Feigenbaum H, ed. *Echocardiography, 5th ed.* Philadelphia, PA: Lippincott, Williams & Wilkins; 1994:556-588.

Jackson J, Richter KP, Launer DP. Thorascopic partial pericardectomy in 13 dogs. *J Vet Int Med* 1999;13:529-33.



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Johnson MS, Martin M, Binns S. A retrospective study of clinical findings, treatment and outcome in 143 dogs with pericardial effusion. *J Small Anim Prac* 2004;45:546-52.

Kienle RD, Thomas WP. Echocardiography. In: Nyland TG and Mattoon JS, eds. *Small Animal Diagnostic Ultrasound, 2nd ed*. Philadelphia, PA: WB Saunders; 2000:354-423.

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Shaw SP, Rozanski EA, Ruhs JE. Cardiac troponins I and T in dogs with pericardial effusion. *J Vet Int Med* 2004;18:322-24.

Sidley JA, Atkins CE, Keene BW, et al. Percutaneous balloon pericardiotomy as a treatment for recurrent pericardial effusion in 6 dogs. *J Vet Intern Med* 2002;16:541.

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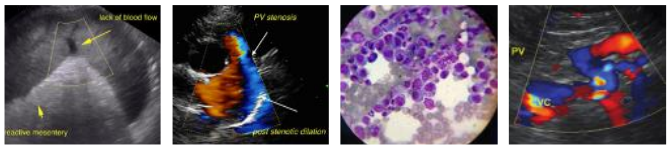
Sisson D, Thomas WP, Reed J, et al. Intrapericardial cysts in the dog. *J Vet Int Med* 1993;7:364-69.

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.



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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.

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.

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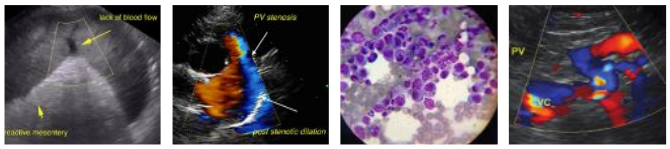

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