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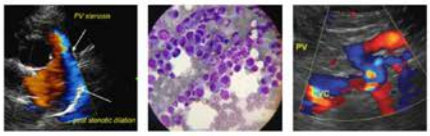

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Clinical Sonography & Telecytology
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PATIENT

Bella Cleppe

SPECIES

Canine

BREED

Rottweiler

SEX

Female

AGE

22 Months

WEIGHT

104 Pounds

INTERPRETED BY

Eric Lindquist, DMV

DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Gromalak

HOSPITAL NAME

SVS Imaging

REFERRING VET

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PRESENTING CLINICAL SIGNS

Came for Spay. No clinical signs notes Exam findings and abnormal lab values: Heart murmur noted on exam. Opted for Presx panel level II (full cbc/chem) all WNL, Heart Murmur grade III No heart murmur listed on any prior exams.

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			1.3	1.3	40	71	0.6
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	97	3.38	1.45		3.4	5.0	

EKG Findings

Sinus arrhythmia at average heart rate of 100/min. Artifacts in baseline. P-wave width borderline, R-wave Amplitude borderline at 2.5 mV.

The rhythm is considered normal, normal heart rate
left heart enlargement possible - still, ECG is unspecific and insensitive for this.

Peter Modler, DVM, Dipl.-Tzt

Cardiac Presentation

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. The cranial and caudal **mitral** valve leaflets presented normal linear structure, extension in systole, and union in diastole with normal kinesis. The **left ventricle** presented thicknesses with linear contour and was not dilated nor restricted. No secondary changes noted in the left venticle owing to the subaortic stenosis. 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 **aortic** valve was thickened and domed. Aortic velocity was excessive at 3.38 m/sec with secondary aortic insufficiency at 5.0 m/sec. 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.

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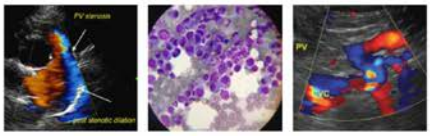

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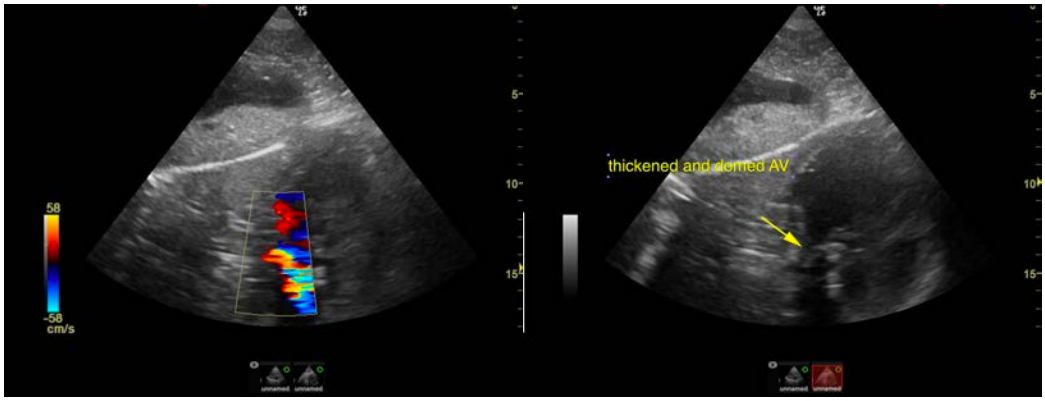
ULTRASONOGRAPHIC FINDINGS

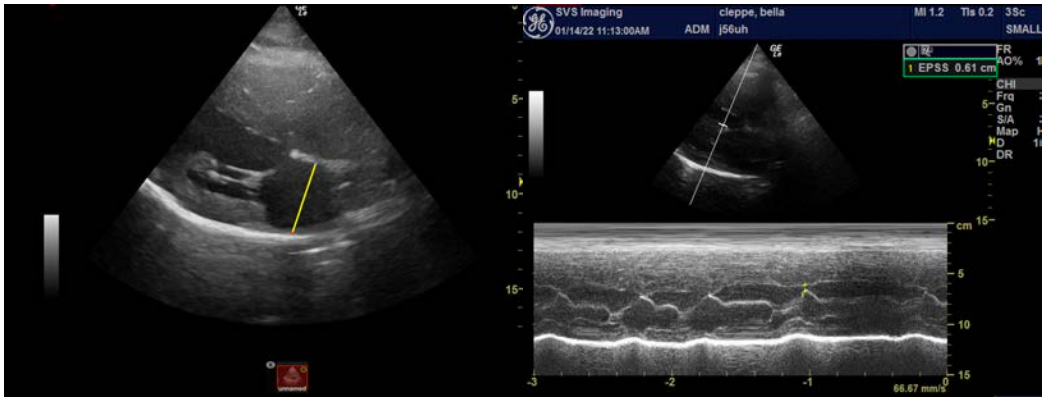
- Subaortic stenosis, mild to moderate without secondary cardiac changes

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The breeding line should be evaluated for similar congenital abnormalities. Even though there is no history of a heart murmur, it is likely that this is still a congenital lesion given the structure. History of an endocarditis is technically possible, yet this lesion is most consistent with congenital valvular dysplasia or subaortic stenosis.

This is not a clinical issue at this time. However, prior to any anesthetic procedure, recommend prophylactic antibiotics 3 days prior and 5 days post procedure. No treatment recommended. Recheck echo in 6 months, earlier if any clinical signs initiate such as exercise intolerance. However, given that this patient is fully grown with these velocities it is unlikely that any clinical signs will develop in this patient unless complicating factors such as endocarditis cause further stenosis of the aortic valve.





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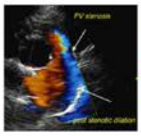
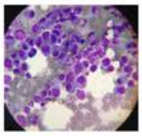
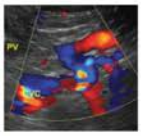

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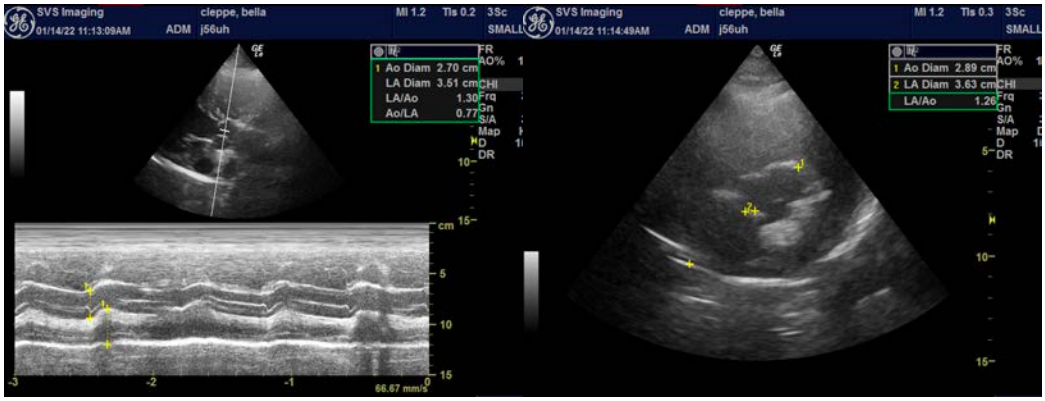

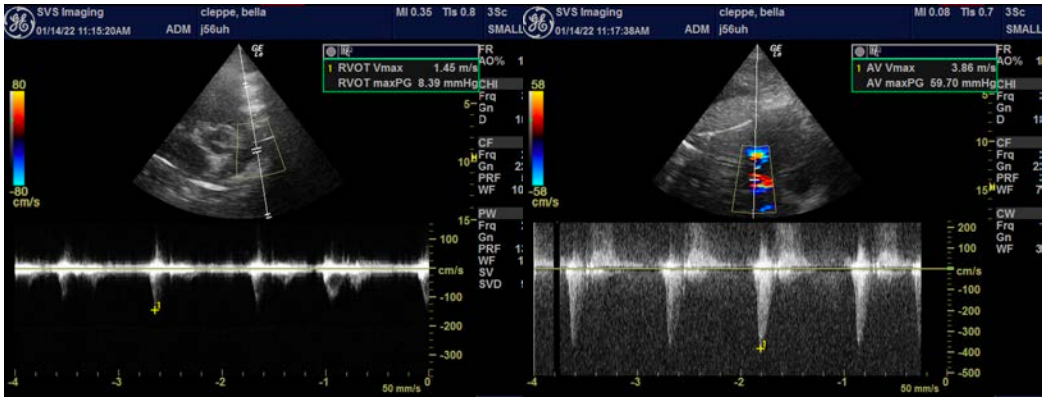

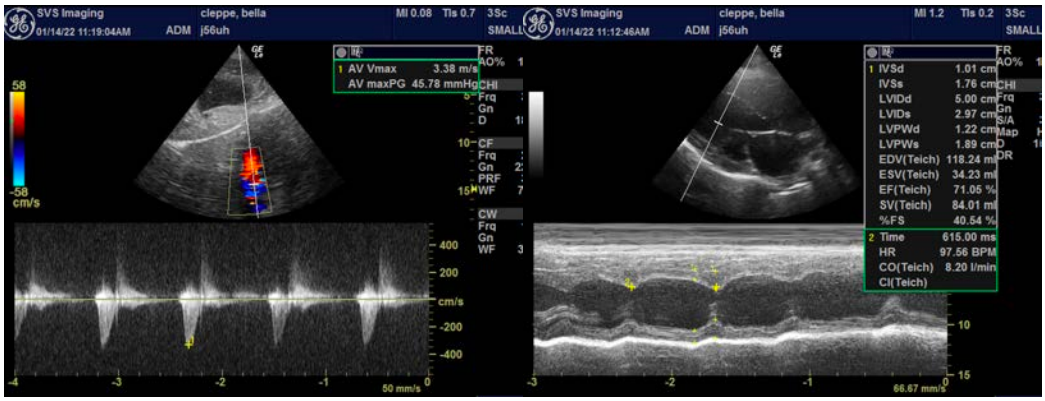

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

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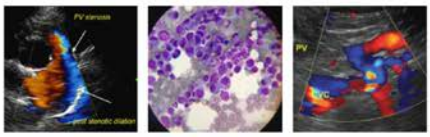

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Subvalvular Aortic Stenosis

http://www.sonopath.com/SAS

http://www.sonopath.com/EchoModler

Description: Subvalvular aortic stenosis (SAS) is the second most common heart defect in dogs and is more common in larger breeds, such as the Newfoundland, Golden Retriever, Boxer, and Rottweiler.

Clinical Signs: Primary clinical signs include an ejection character heart-based murmur, syncope, and occasionally congestive heart failure (CHF). Hypokinetic pulses may be present in more advanced cases. Primary differentials include pulmonic stenosis, ventricular septal defect, mitral regurgitation, patent ductus arteriosus, and an idiopathic flow murmur. SAS patients are predisposed to bacterial endocarditis and necessitate prolonged antibiotic therapy during surgical and dental procedures as well as under circumstances where an infection, such as pyoderma, pyometra, or a UTI, may be present.

Diagnostics: Radiographs may present as completely normal or they can reveal an aortic bulge as well as left atrial and ventricular enlargement. Pulmonary venous congestion and CHF are also sometimes detected.

Ascertaining NT-proBNP levels may be useful in young puppies with murmurs as a means of screening for congenital anomalies. One study showed that median NT-proBNP levels in dogs with SAS were 833 pmol/L compared to 333 pmol/l for normal dogs. Although NT-proBNP is not specific for SAS, an elevated level should prompt additional diagnostic tests and echocardiography in particular.

Moderate to severe SAS is easily confirmed by 2D or Doppler echocardiography. Typical findings include concentric left ventricular hypertrophy, a subvalvular obstructing lesion, and poststenotic dilation of the aorta. The papillary muscles and endocardial surface of the ventricular myocardium often appear hyperechoic, presumably because of myocardial ischemia and replacement fibrosis or calcification. Structural changes in the mitral valve can often be appreciated and abnormal motion of the mitral valve (systolic anterior motion) can be detected in those dogs with coexisting mitral valve dysplasia and dynamic obstruction. Spectral Doppler measures the peak velocity of flow in the left ventricular outflow tract (LVOT). It is performed to assess disease severity; the results correlate closely with those obtained using invasive measures. Although Doppler-estimated pressure gradients between 80 and 100 mm Hg (peak flow velocities ranging from 4.5-5.0 m/s) are taken to indicate moderate LVOT obstruction and higher velocities more severe obstructions, these designations are somewhat arbitrary. Doppler color flow recordings are valuable for detecting and estimating the severity of any coexisting aortic or mitral valve insufficiency. The detection of mild SAS by echocardiography is often not possible as dogs with subtle abnormalities may escape detection by even the most accomplished examiners. A diagnosis of mild SAS is more secure when

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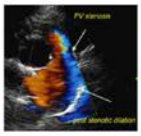
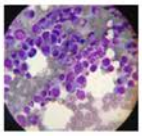
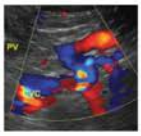

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mildly elevated velocity measures are accompanied by disturbed flow, an anatomic lesion is visible, or velocity flow suddenly accelerates over a discrete region in the LVOT.

Treatment: Both moderate and severe cases of SAS are at risk for sudden death. Medical therapy with a beta blocker (e.g. atenolol at 0.5-1mg/kg BID) is the preferred treatment; mild exercise restriction is also recommended in severe cases. A number of surgical treatment options can be considered for dogs with moderate to severe SAS, but most are of uncertain value. Open resection of the obstructing lesion during cardiopulmonary bypass offers the best opportunity for reducing the systolic pressure gradient. Other surgical procedures employed to dilate or bypass obstructions have either failed to achieve a sustained reduction of the systolic pressure gradient or they entail an unacceptable risk of complication. Balloon valvuloplasty is no longer typically performed in most cases due to a lack of lasting beneficial effects and difficulty in adequately reducing the pressure gradient.

References:

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