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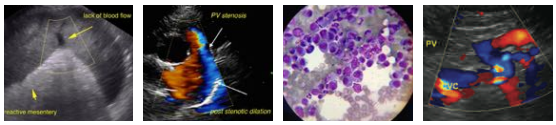

ANIMAL SOUNDS NW
MOBILE VETERINARY ULTRASOUND
IMAGING PERFORMED BY
AnimalSoundsNW.com 541-357-9423

PATIENT

Scooby Doo Kennedy

SPECIES

Canine

BREED

German Shepherd

SEX

Spayed Female

AGE

6 months

WEIGHT

34 lbs

INTERPRETED BY

Eric Lindquist, DMV, DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Sara Hansen

HOSPITAL NAME

West Eugene AH

REFERRING VET

Dr. Sundholm

DATE

5/31/22

Invoice
30811

PRESENTING CLINICAL SIGNS

History: Scooby Doo was examined in April 2022 and had a grade 3/6 heart murmur. The owners said that a heart murmur was detected on a puppy exam at 8 weeks of age. The dog is doing well clinically.

ULTRASONOGRAPHIC EXAMINATION OF THE HEART

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. Trivial **mitral** valve insufficiency was noted with a centralized jet. 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 aortic valve was thickened with a subvalvular ridge or lambus. This is consistent with subaortic stenosis. The **aortic outflow** velocity was excessive in this patient and measured 3.5 m/sec. This is also consistent with subaortic stenosis. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted. Mild **tricuspid** insufficiency was noted at 2.5 m/sec. The septal leaflet appears thickened and somewhat shortened. A minor form of tricuspid dysplasia is likely. 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.

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	<1.3	28-40	40-100	<0.6
PATIENT			1.0	1.08	40	85	0.14
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT**	**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	110	3.5	1.71	34 lbs	3.26 max	2.77	

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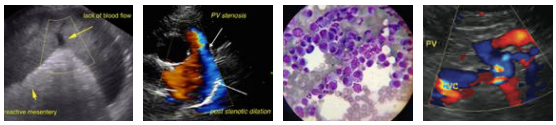

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ULTRASONOGRAPHIC FINDINGS

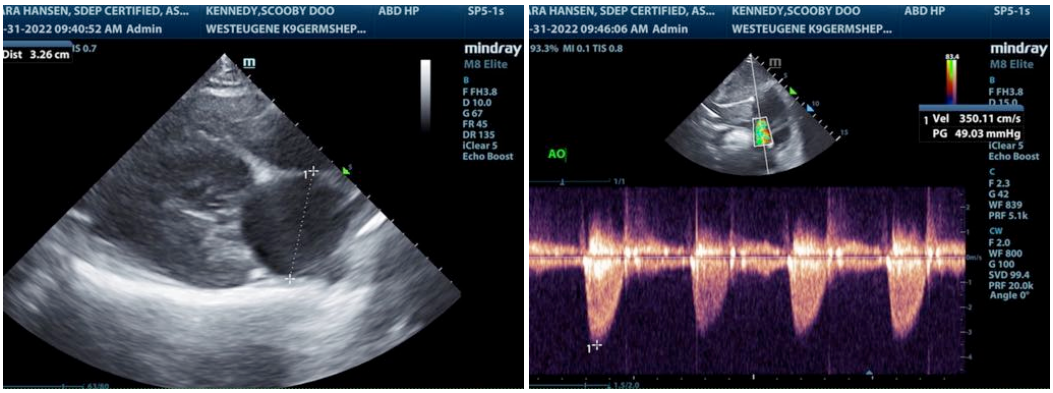
Subaortic stenosis, compensated.

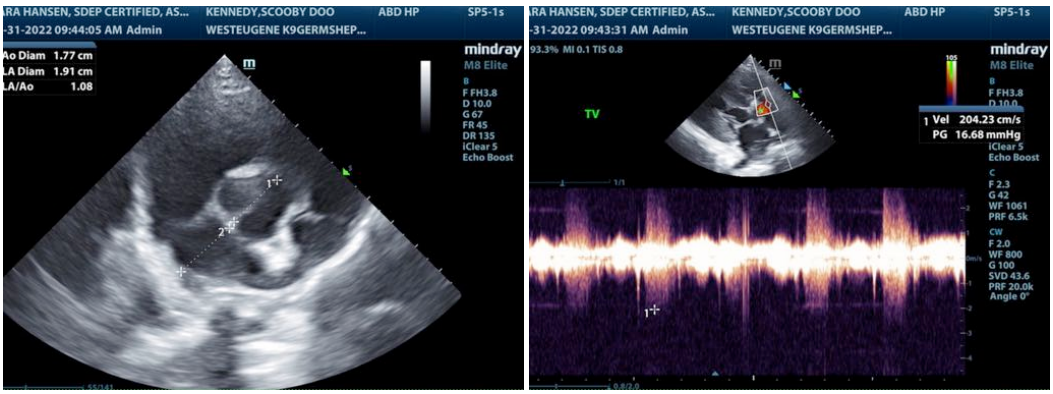
Minor mitral valve insufficiency.

Minor form of tricuspid dysplasia.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The congenital lesions are compensated at this time. No overt contraindication to anesthetic procedure. However, I do recommend prophylactic antibiotics prior to any anesthetic procedure. No treatment is necessary at this time. A recheck echocardiogram is recommended in 3-6 months or earlier if clinical signs such as exercise intolerance occur. Prognosis long term is guarded.





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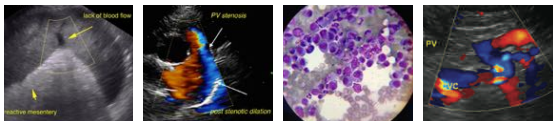

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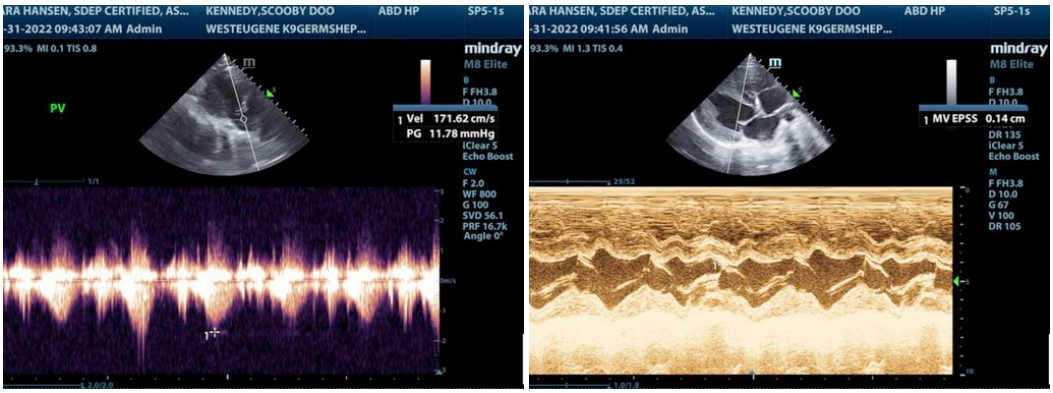

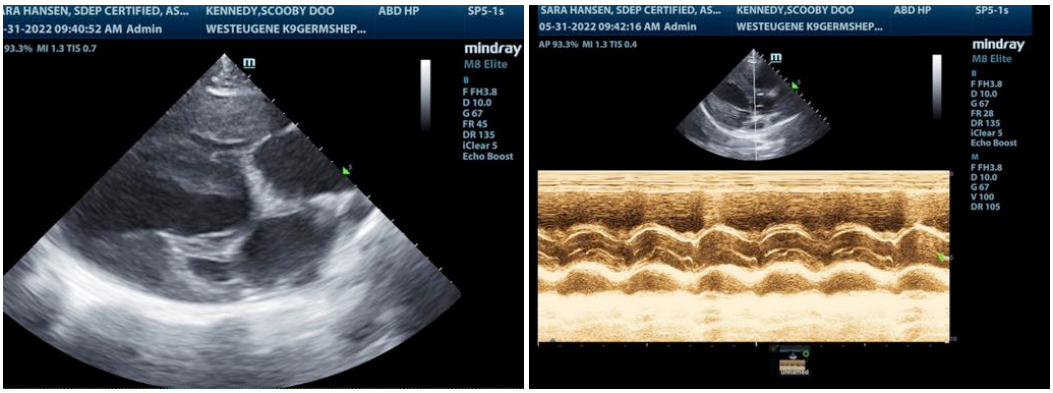

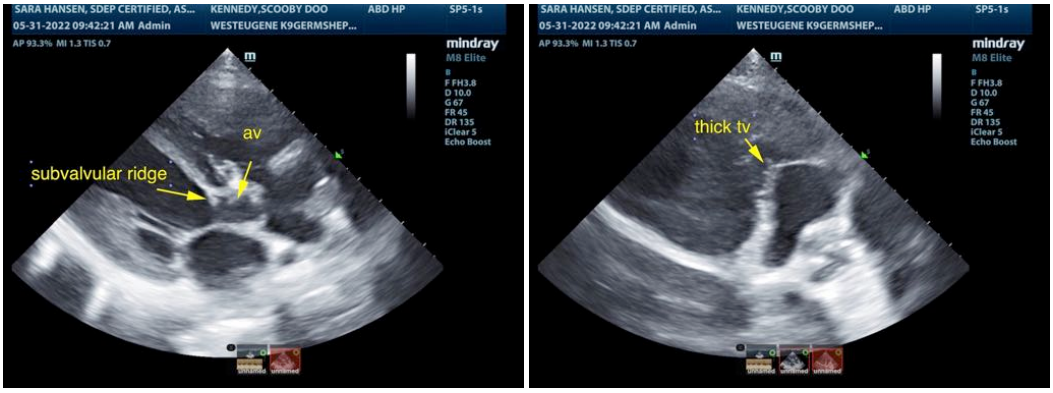

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

Eric.Lindquist@SonoPath.com

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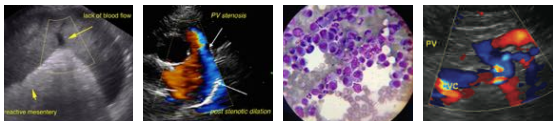

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Subvalvular Aortic Stenosis

http://www.sonopath.com/SAS

http://www.sonopath.com/EchoModler

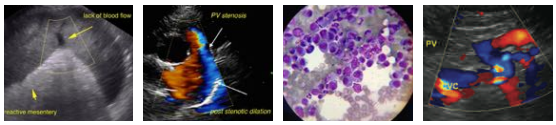
Description: Subvalvular aortic stenosis (SAS) is the second most common heart defect in dogs and is more common in larger breeds, such as the Newfoundland, Golden Retriever, Boxer, and Rottweiler.

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



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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 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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Kienle RD, Thomas WP, Pion PD. The natural clinical history of canine congenital subaortic stenosis. *J Vet Intern Med* 1994;8:423-31.

Oyama MA, Sisson DD. Cardiac troponin-I concentration in dogs with cardiac disease. *J Vet Int Med* 2004;18:831-39.

Reist-Marti SB, Dolf G, Leeb T, et al. Genetic evidence of subaortic stenosis in the Newfoundland dog. *Vet Rec* 2012;170:597.