

Pankatz CA-#4

IVUSS Cardiology Case#4

Aortic Stenosis

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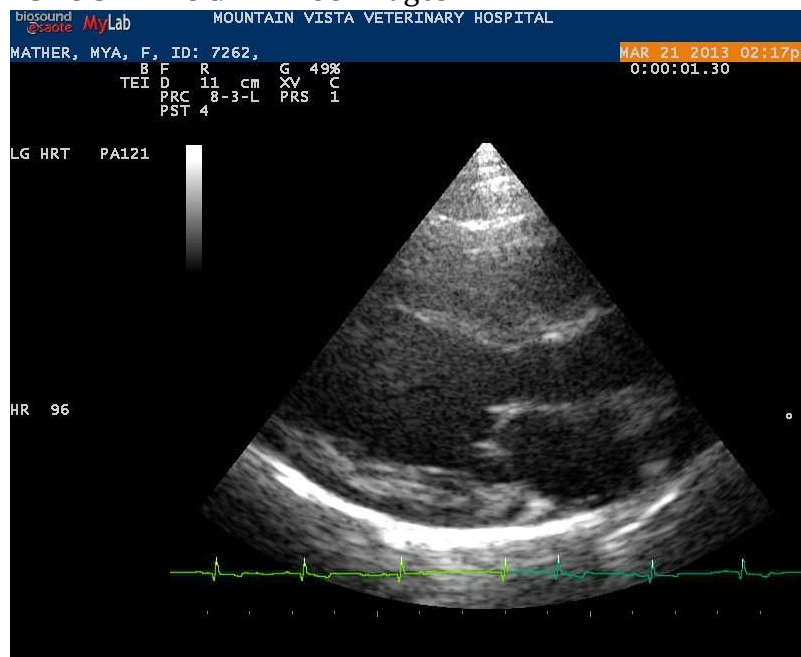
HISTORY:

“Mya” a 3-year-old FS Golden Retriever was presented to our hospital for a routine examination. She had a history of a systolic heart murmur in her medical history since a young puppy. Mya was spayed at 6 months of age without any anesthetic complications. Clinically Mya was doing very well with no clinical symptoms of heart disease or other problems. An echocardiogram was recommended in order to determine the cause of the heart murmur.

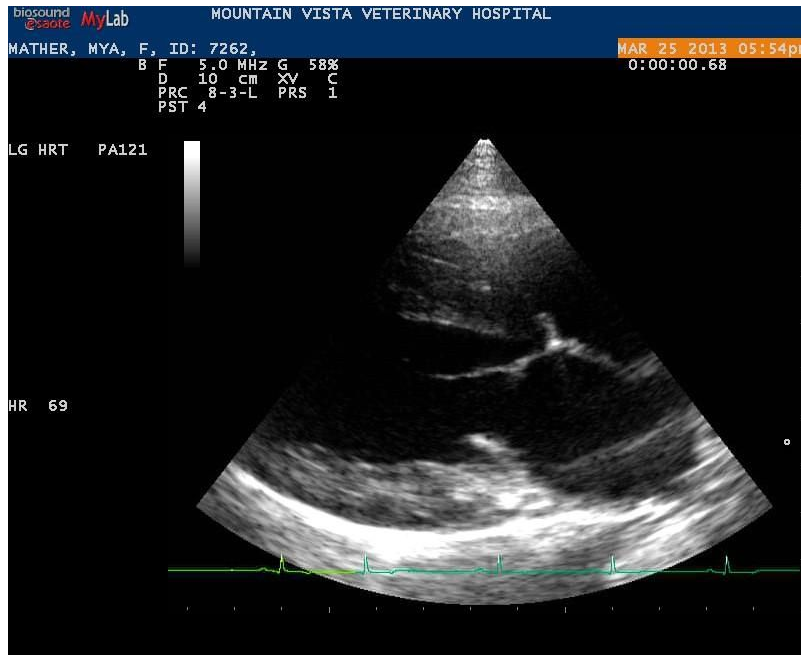
PHYSICAL EXAMINATION:

HR=96 RR=36 T=38.2 C pink mm CRT<2 sec. A grade 4/6 systolic heart murmur was clearly heard on both sides of the chest with PMI at the left heart base. No cardiac arrhythmias were appreciated and the lung fields were clear. No pulse deficits were noted. BCS 3/5 Good coat quality and eyes/ears/teeth appeared to be normal. No masses or organomegaly were noted on abdominal palpation and no lymph nodes were palpable. Overall Mya presented as a very healthy patient.

ECHOCARDIOGRAM: 35 images



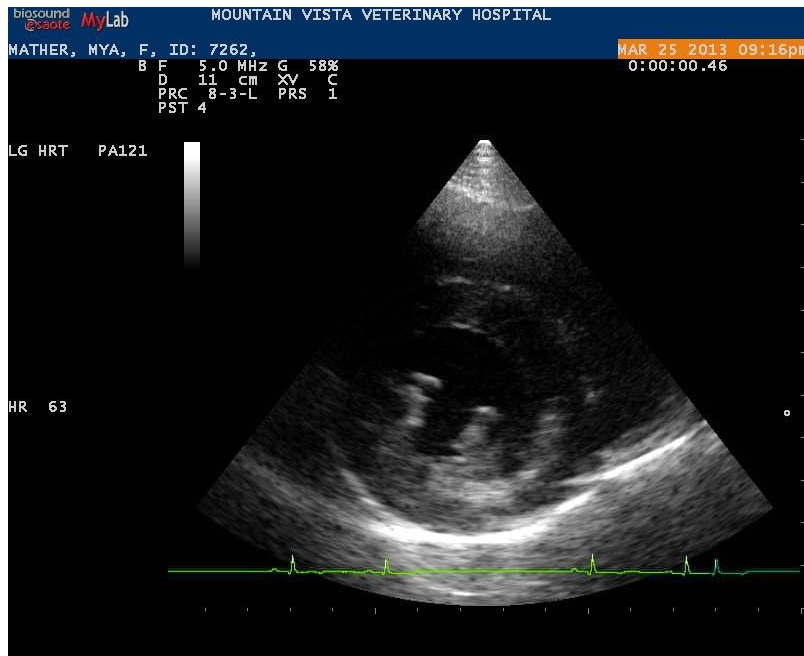
5- chamber right parasternal long-axis view of the LVOT.



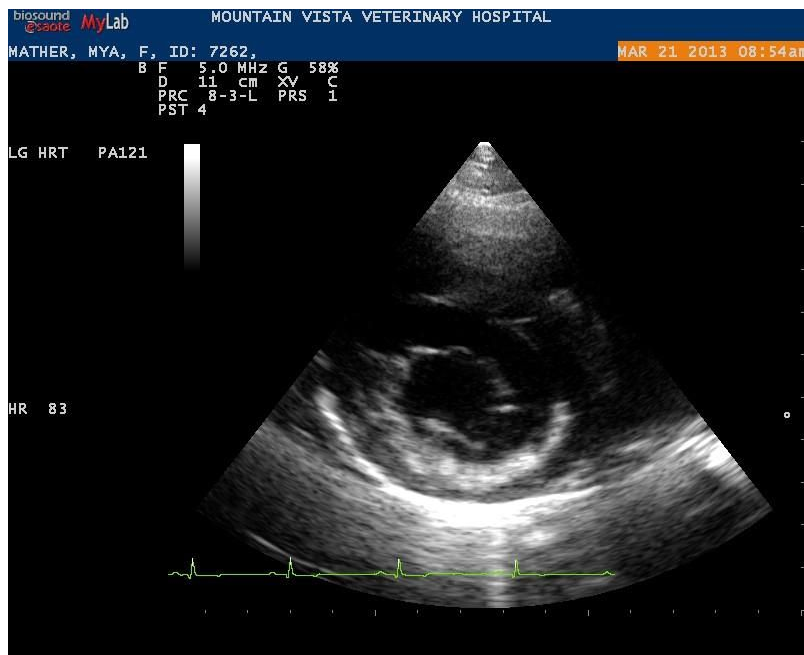
4-chamber right parasternal long-axis view. Mitral and tricuspid valves appear to be normal.



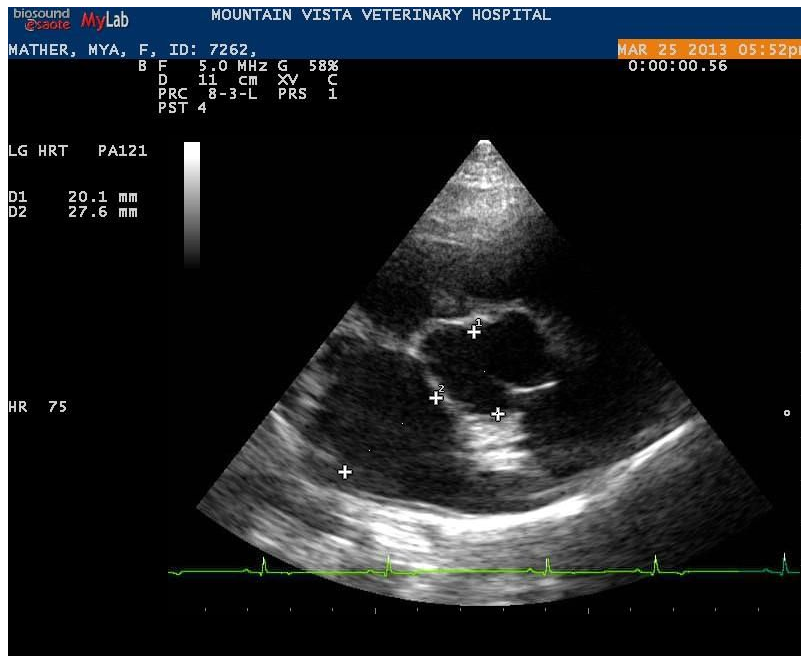
Right parasternal transverse view at the level of the papillary muscles.



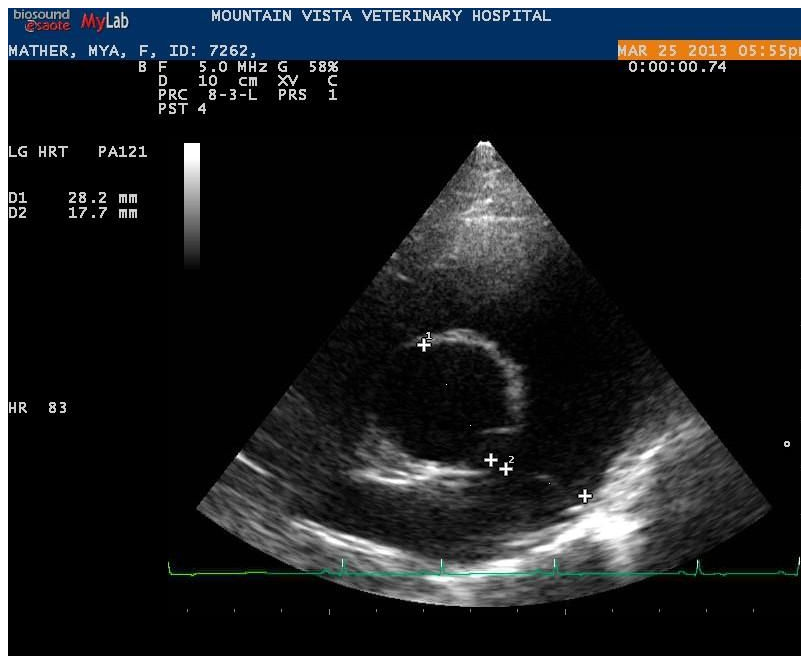
Right parasternal transverse view at the level of the chordae tendinae.



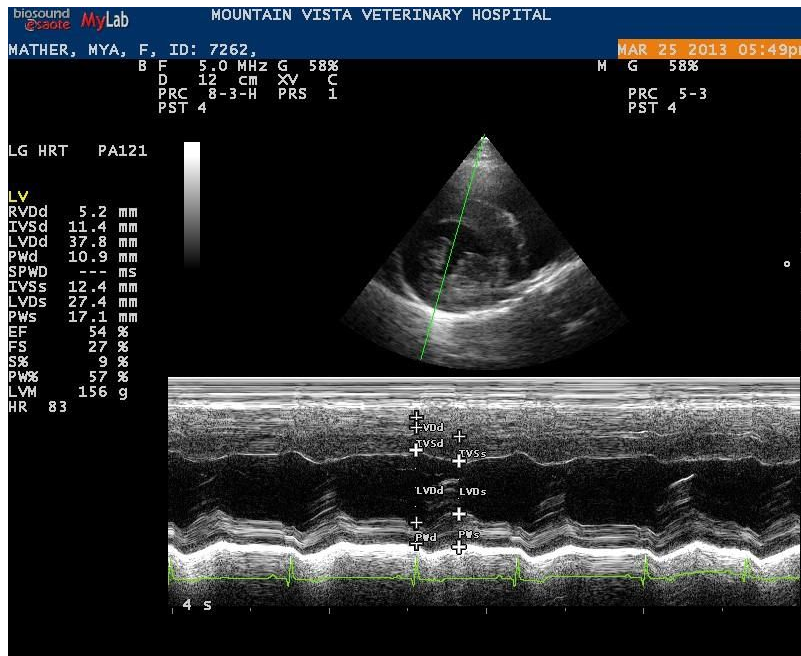
Right parasternal transverse view at the level of the mitral valves.



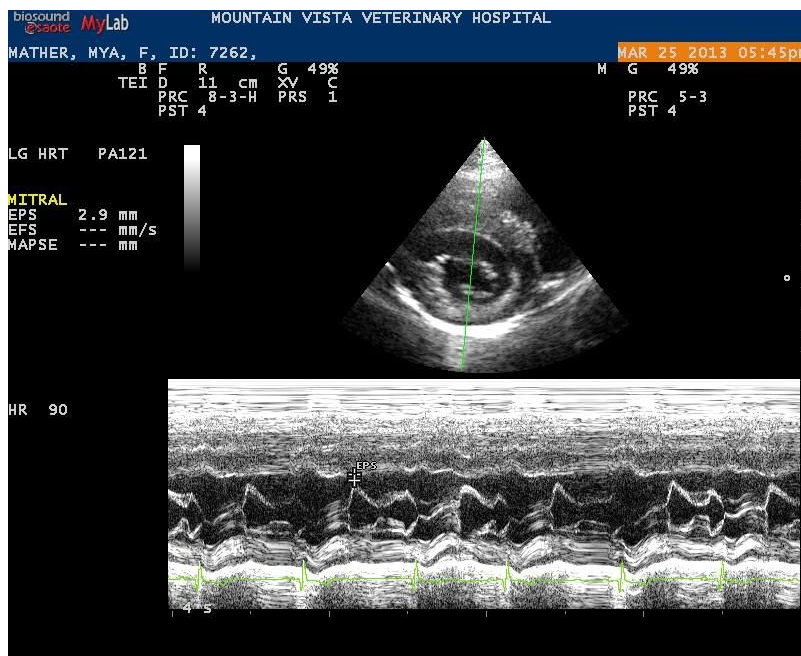
Aortic base transverse view LA/Ao=1.37



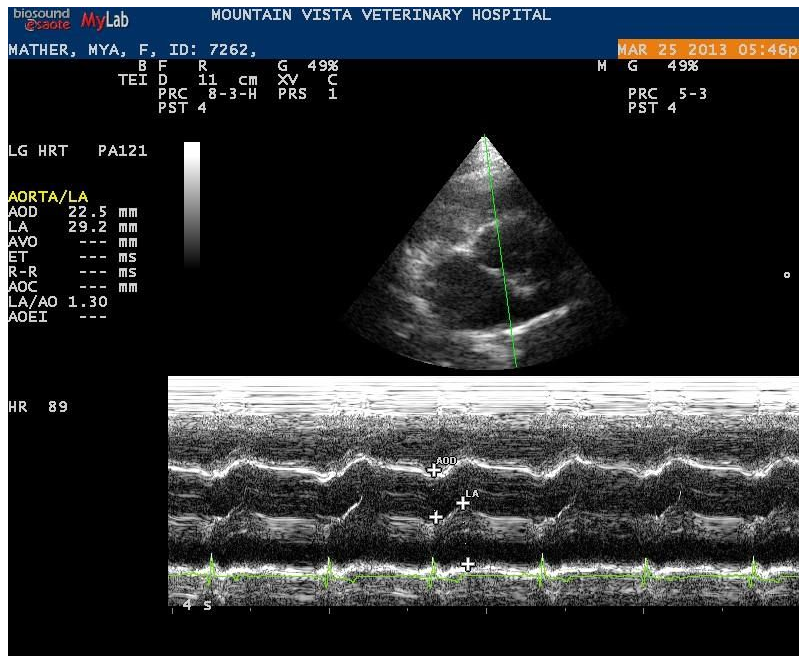
Right parasternal transverse view at the level of the pulmonary artery. MPA/Ao=0.62. (N=0.88-1.15) The aorta is dilated at this level compared to the aortic root causing the MPA/Ao ratio to be low.



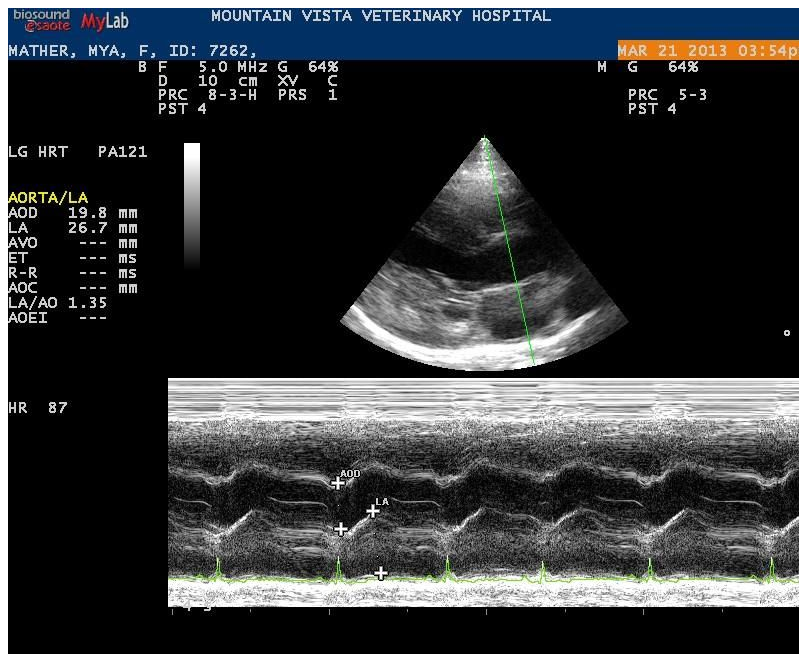
M-mode of the left ventricle. There is mild thickening of the interventricular septum and the left ventricular free wall in diastole. The left ventricular chamber size is normal. Systolic function is good shown by a normal left ventricular chamber size in systole.



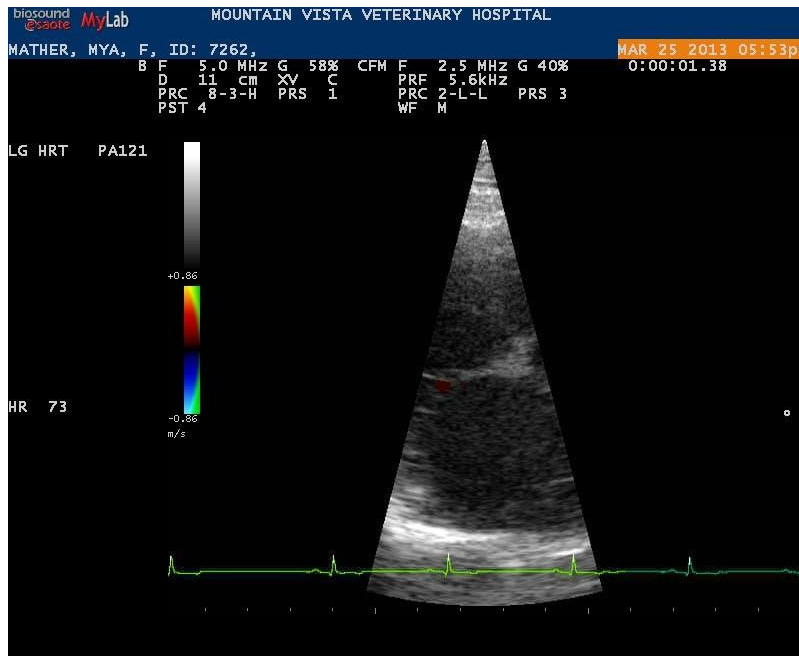
M-mode of the mitral valves. EPSS 2.9mm (N<7.7mm). There is no evidence of systolic anterior motion (SAM) of the mitral valve.



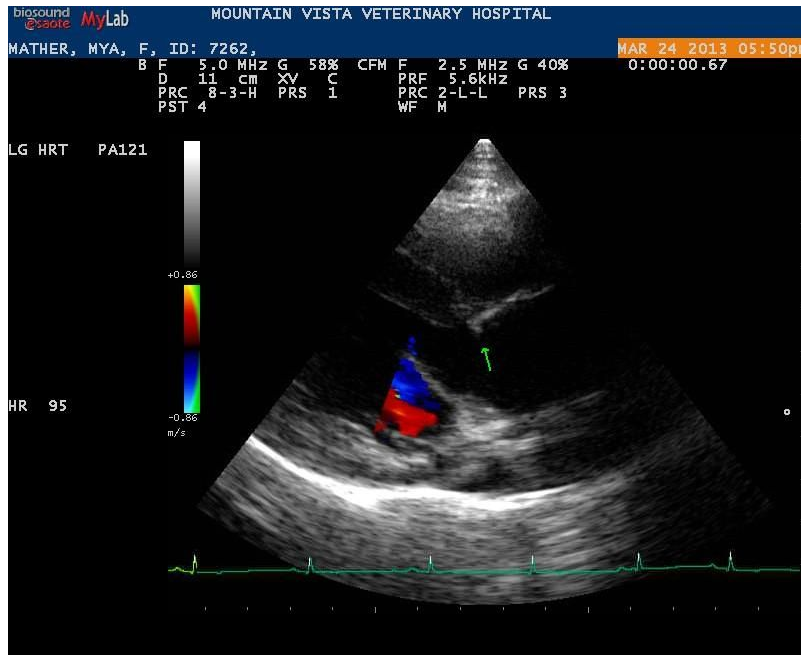
M-mode in short axis showing LA/Ao=1.30.



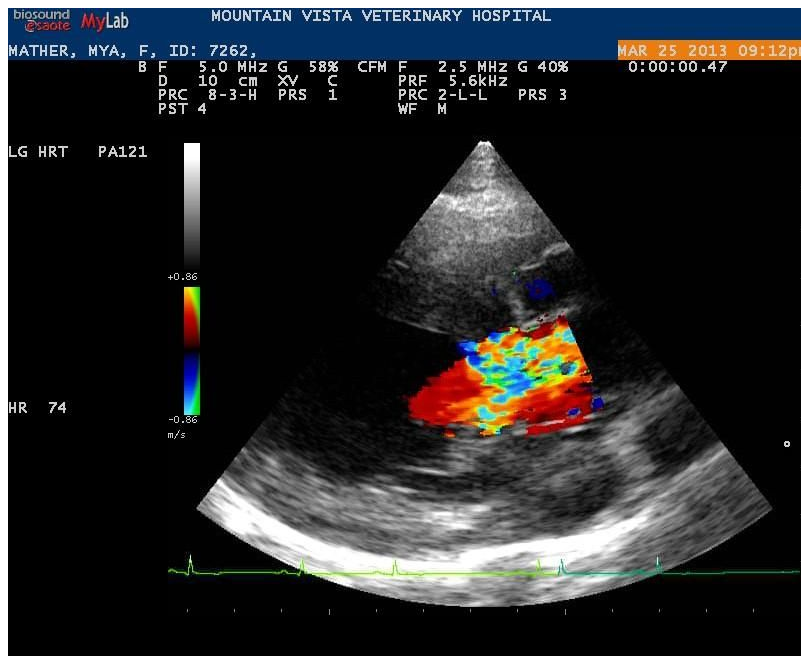
M-mode in long-axis showing LA/Ao=1.35.



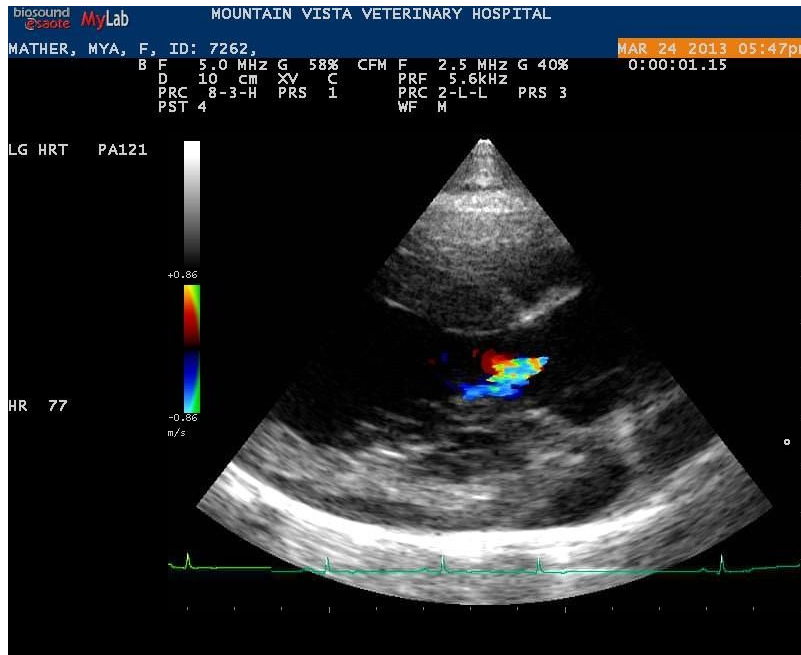
Colour Doppler analysis of the mitral valve in a tipped 4-chamber right parasternal long axis view. Because of the depth needed to evaluate this region, the scan sector was decreased to optimize the colour Doppler signal. No mitral regurgitation is noted in systole.



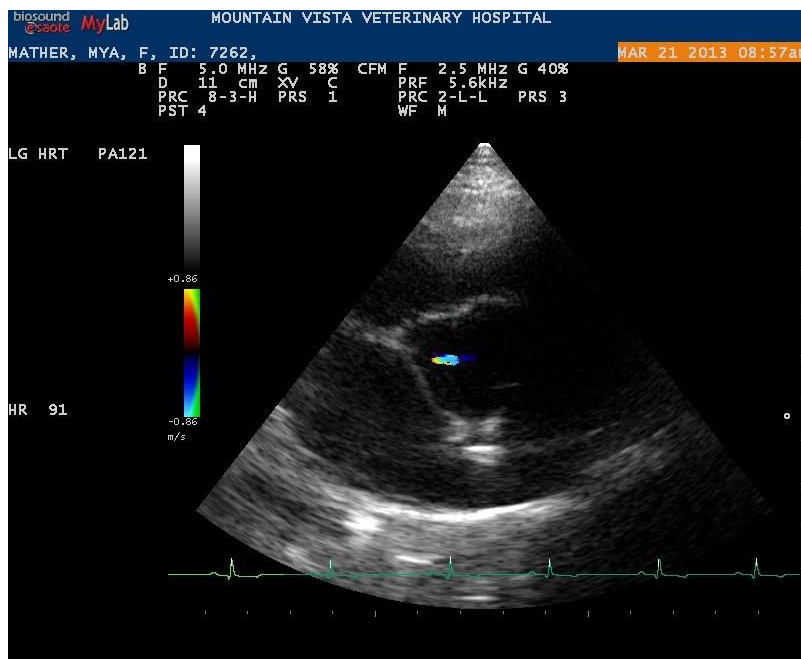
Shortened 5-chamber right parasternal long-axis view showing the anterior mitral valve in diastole. Note the small ridge of tissue just proximal to the aortic valve (green arrow) which likely represents the stenotic lesion.



Colour Doppler analysis of the LVOT showing turbulent blood flow.



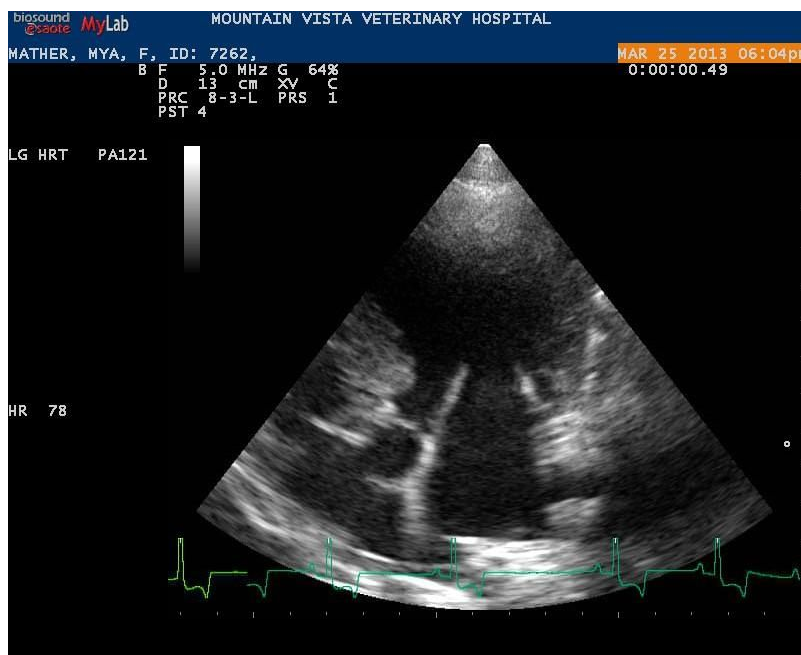
Mild aortic insufficiency (AI) with a regurgitant jet originating from the centre of the aortic valve noted on colour Doppler in diastole.



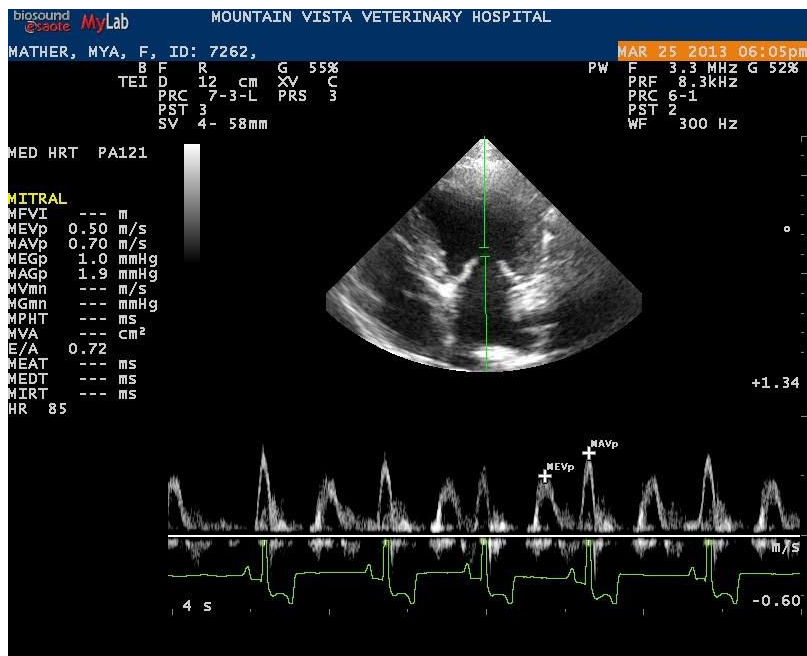
AI also noted in the heart base view.



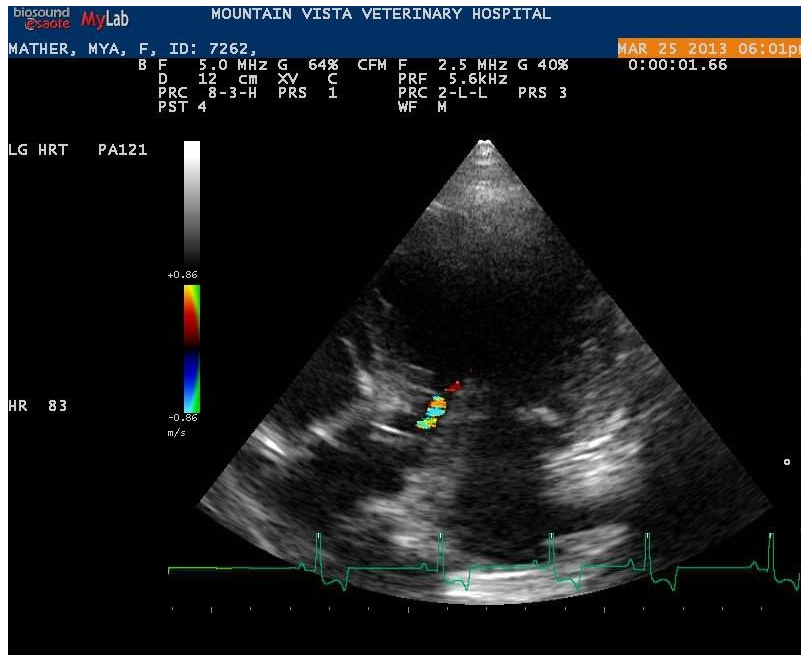
Left apical 4-chamber view.



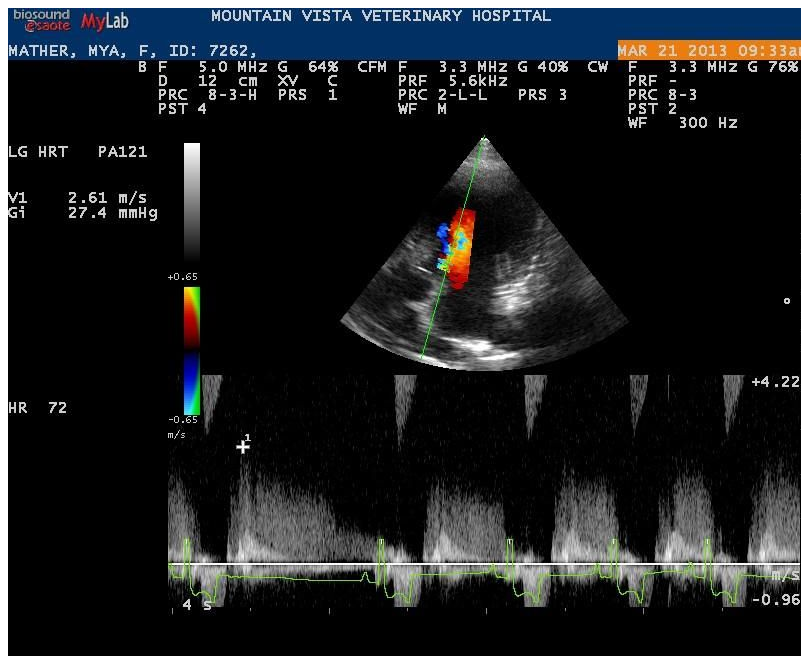
Left apical 5-chamber view.



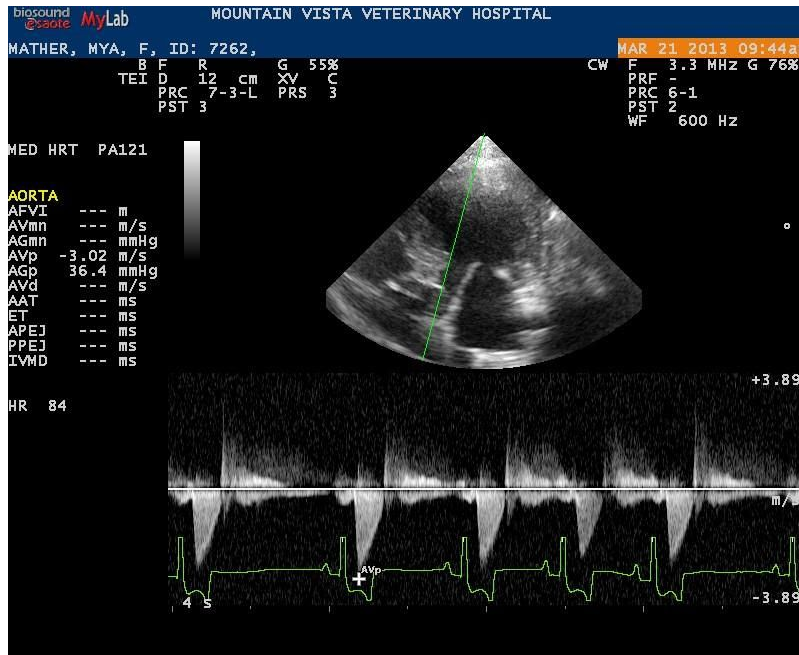
PW Doppler of the mitral valve in diastole shows a reversal in peak velocities of the ME and MA waves. A ME:MA ratio of <1 indicates grade one diastolic dysfunction which is impaired left ventricular relaxation.



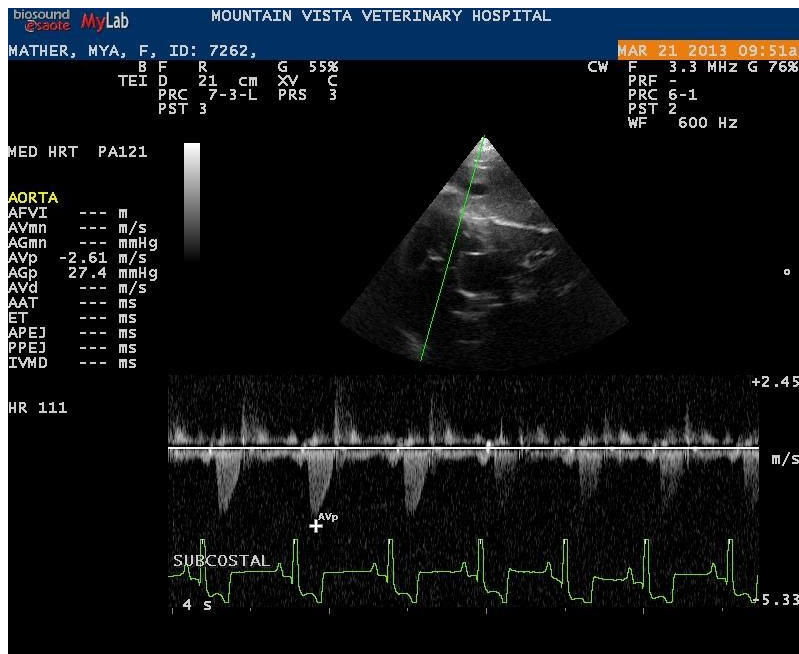
Colour Doppler analysis of the aorta in the left apical 5-chamber view showing mild AI in diastole



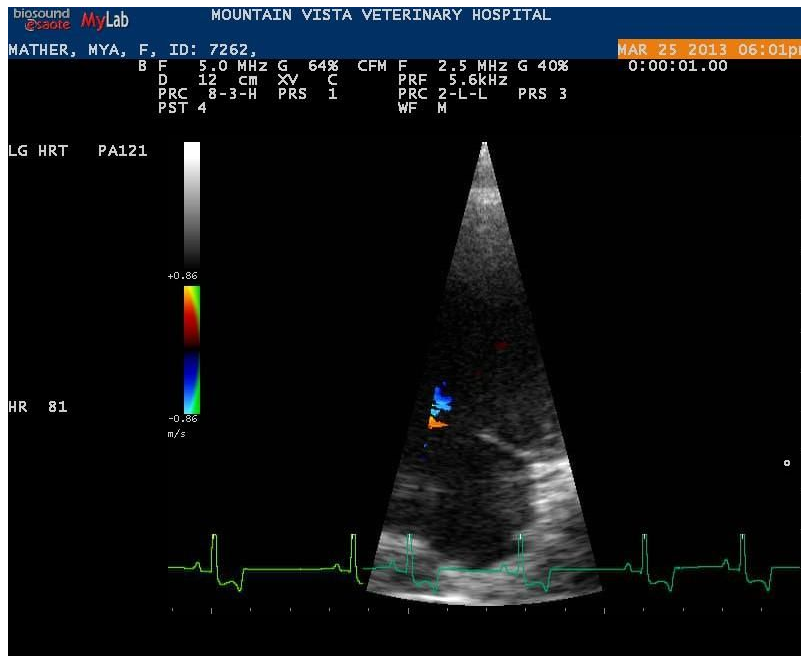
AI documented on CW Doppler. The peak velocity is recorded at 2.61m/s.



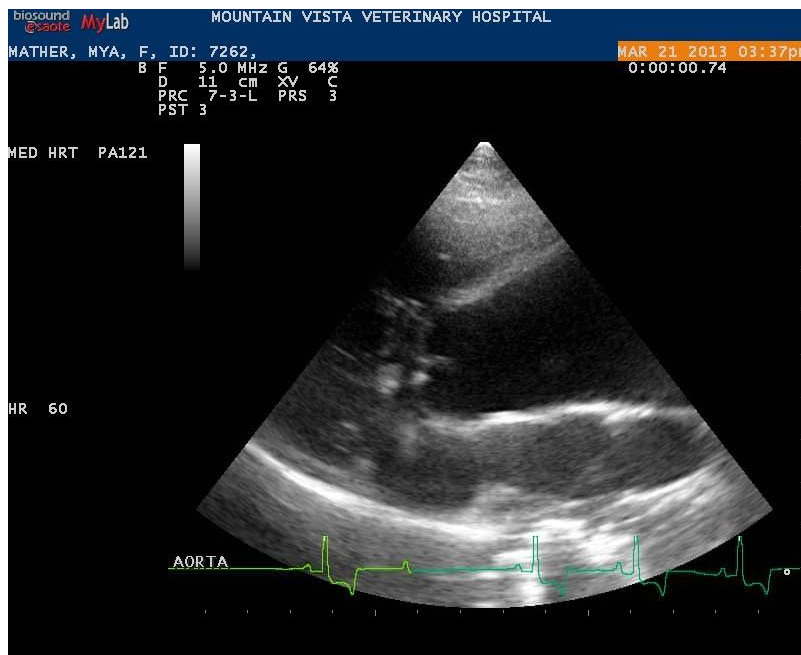
Aortic maximum velocity recorded on CW Doppler 3.02m/s PG=36.4 which is consistent with a diagnosis of mild aortic stenosis.



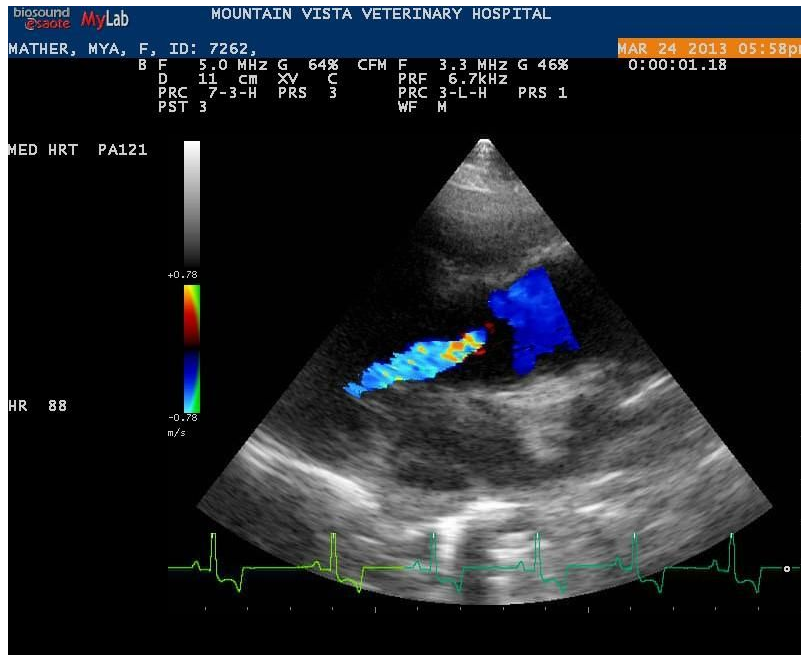
Subcostal view of the aorta and CW Doppler. A maximum velocity of 2.61m/s is measured. In this case, better alignment and maximum velocity was measured in the left apical parasternal view above.



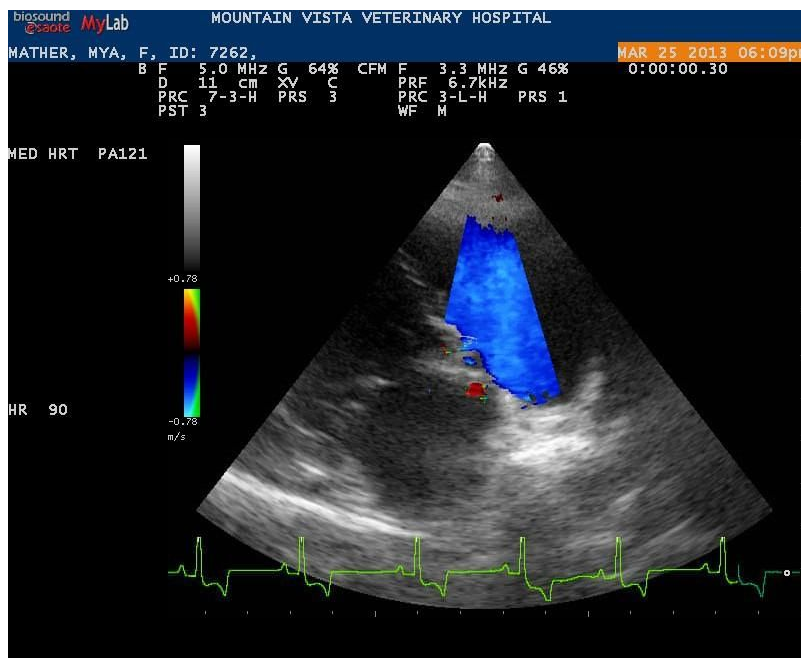
Colour Doppler analysis of the mitral valve in this 4-chamber left apical view. No MR is noted.



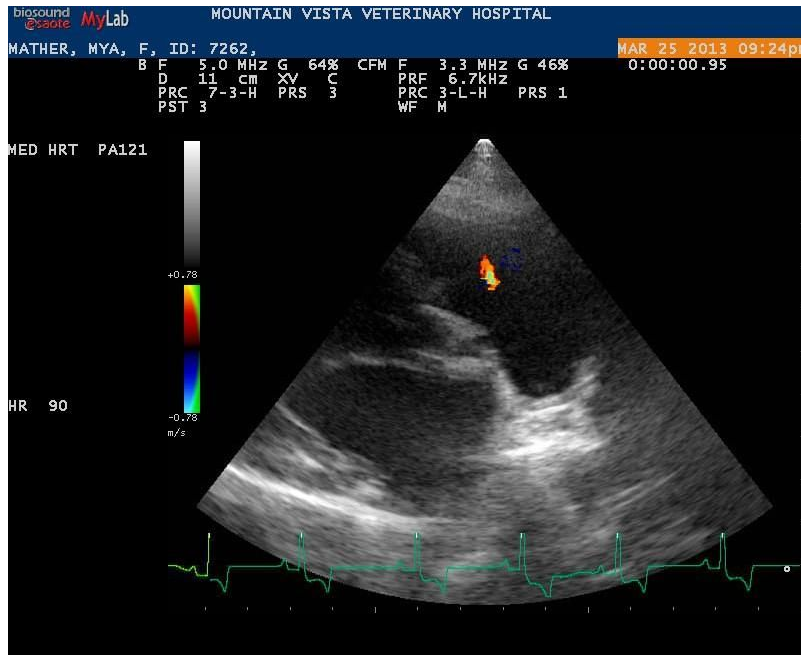
Left parasternal long-axis view of aortic valve and the aorta. The aorta dilates after the aortic valve with its walls diverging in opposite direction.



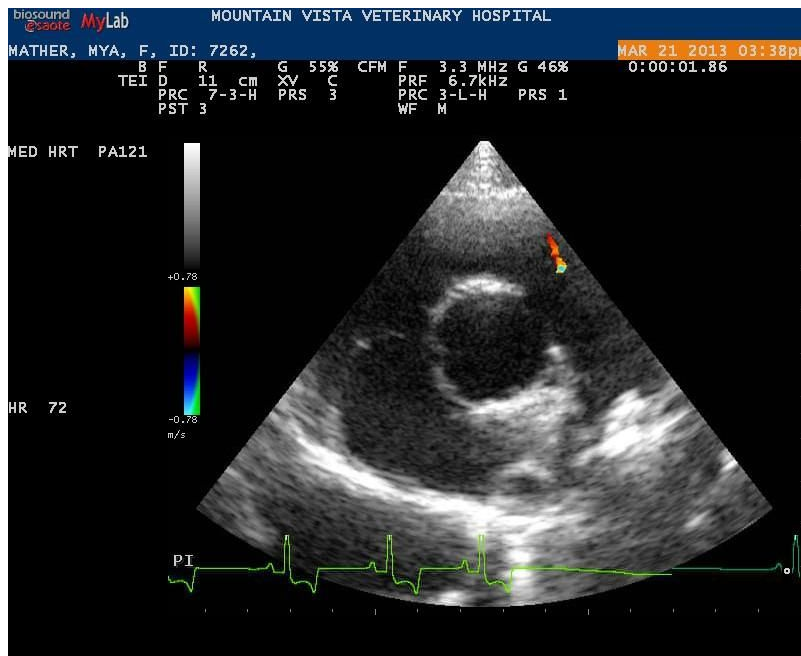
Moderate AI is noted on colour Doppler in this left parasternal long axis view.



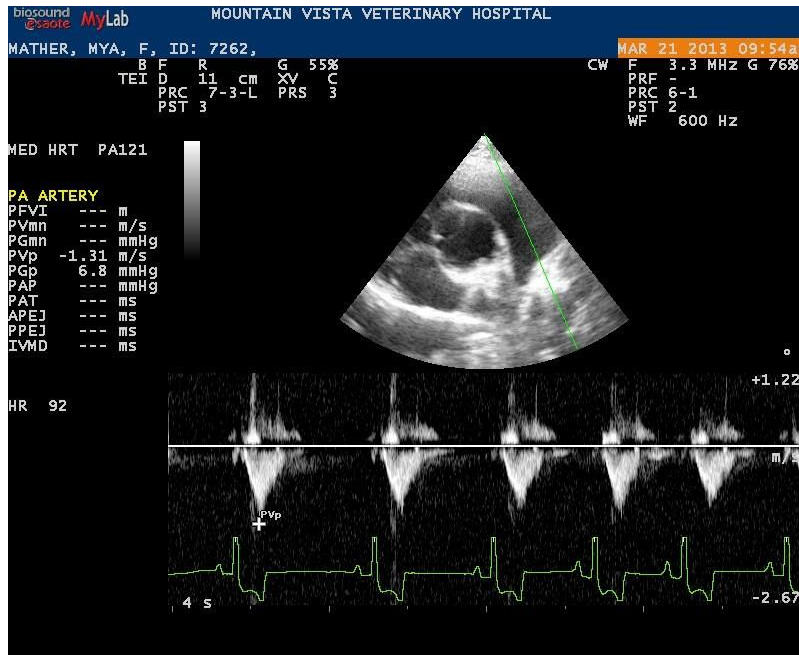
Colour Doppler analysis of the pulmonary artery in the left parasternal long-axis view. The flow is laminar.



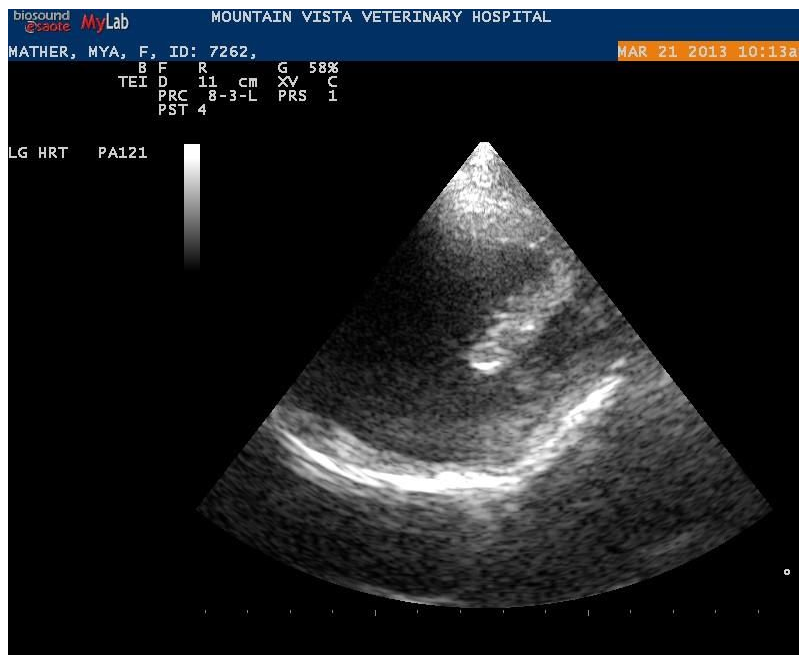
Mild pulmonic insufficiency (PI) noted in the left parasternal view of the pulmonary artery.



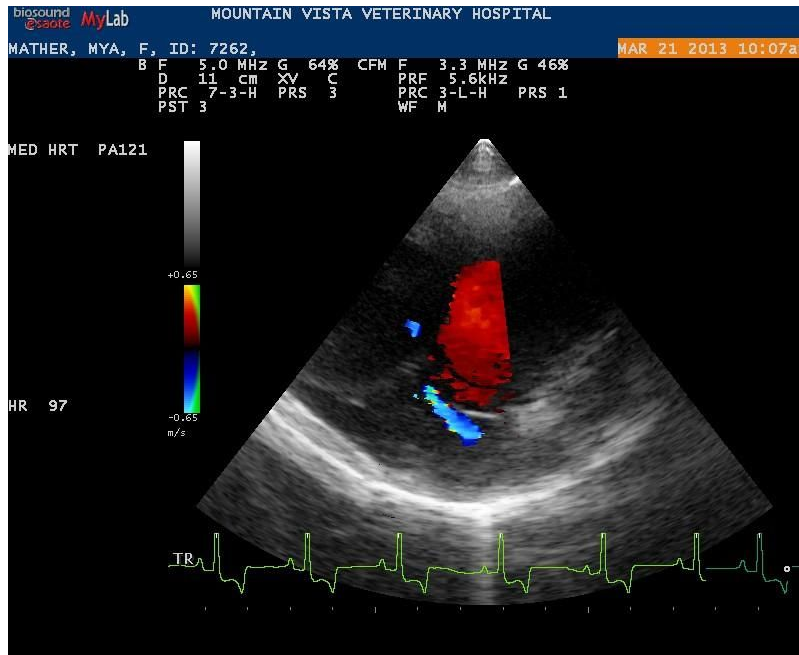
Mild PI also noted in the left parasternal transverse view of the pulmonary artery.



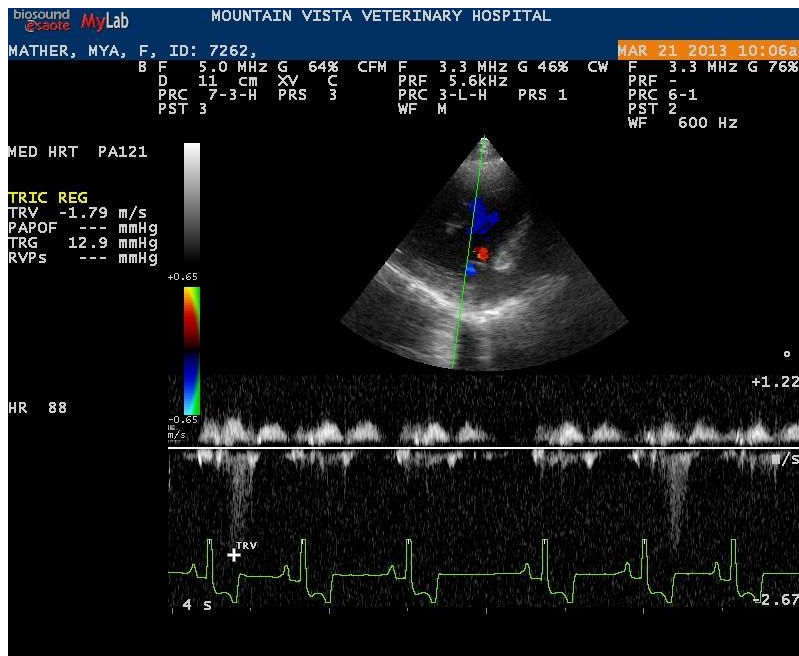
Pulmonary maximum velocity on CW Doppler is 1.31ms which rules out pulmonic stenosis in this patient.



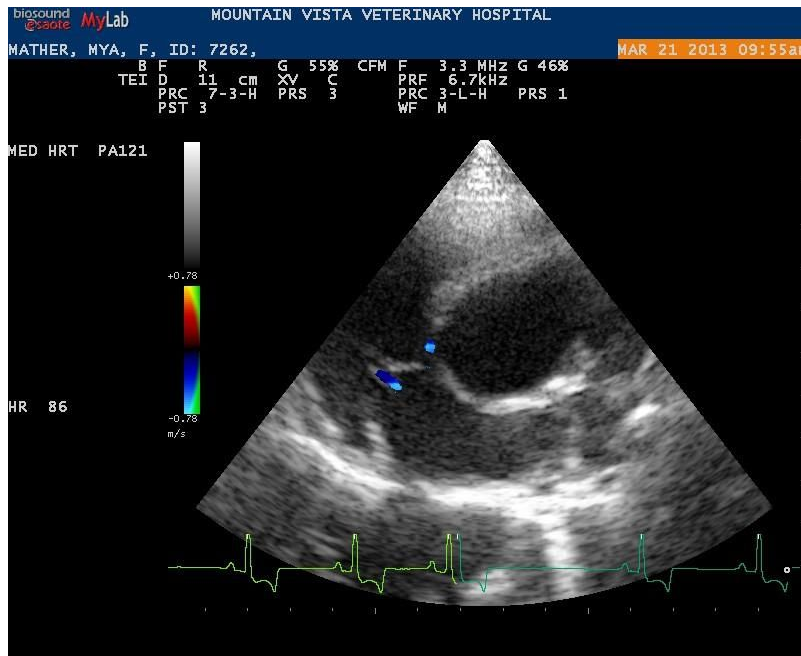
Left parasternal view of the right atrium and auricle. No masses are noted.



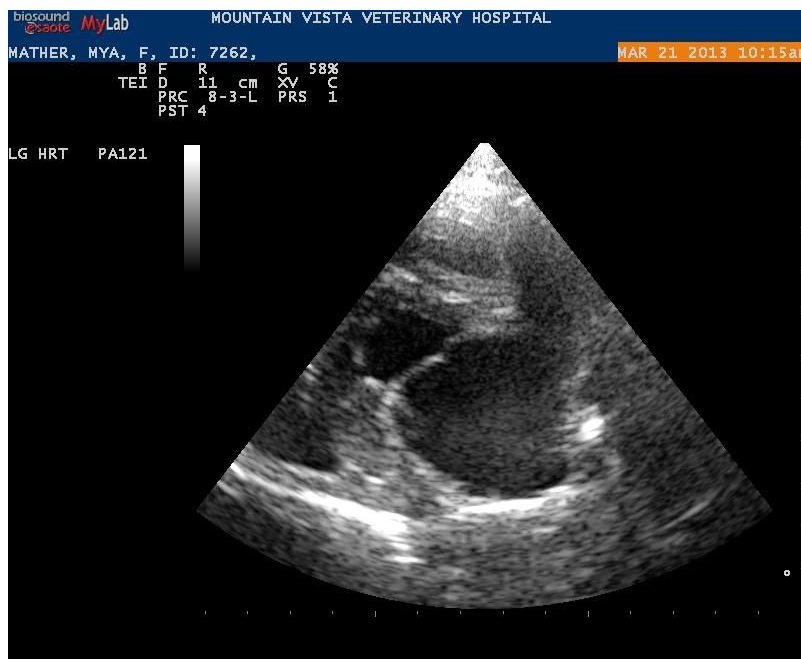
Mild tricuspid regurgitation noted at the left parasternal view of the right atrium and auricle. No masses are seen.



The maximum velocity of the TR jet is recorded at 1.79m/s on CW Doppler. Using the Bernoulli equation, this translates into a pressure gradient of 12.9 mmHg between the right ventricle and atrium. This value estimates the pulmonary artery systolic pressure in the absence of pulmonic stenosis. Since this was the highest velocity recorded, pulmonary hypertension is ruled out in this patient.



Trace TR is present in this left parasternal transverse view of the tricuspid valve.



Left parasternal view of the left atrium and auricle.

MEASUREMENTS: wt. 30.4kg

M-mode	#1	#2	#3	June Boon 95% Prediction Intervals mm	Golden Retriever Breed Specific Ref. Ranges mm	Cornell, Kittleson VIN Echo Calculator mm
IVSd	12.9	11.9	10.9	10.2-11.37	8-13	8-10
LVDD	38.3	38.3	38.8	37.1-38.5	37-51	38-46
PWd	13.5	11.9	12.4	8.2-9.2	8-12	8-10
IVSs	18.1	12.4	16.1	15.5-16.8	10-17	
LVDs	25.9	25.4	24.9	18-29	18-35	25-31
PWs	18.1	16.6	19.2	13.27-14.5	10-19	
FS%	32	34	36	33-46	27-55	35-40
LA	29.2	26.7		21.3-36.5	16-32	22-27
Ao	22.5	19.8		16.8-30.5	14-27	22-27
LA/Ao	1.3	1.34	N<1.5			
EPSS	2.9	N<7.7				
2-D						
IVSd	12.6					
LVDD	36.7					
PWd	12.1					
IVSs	13.7					
LVDs	28.8					
PWs	18.4					
FS%	21.5					
LA	27.6					
Ao	20.1					
LA/Ao	1.37	Rishniw N<1.6				
MPA/Ao	0.62	N=.88 -1.15				

ME:MA 0.72 (N=1-2)

Aortic Maximum Velocity 3.02m/s=36.4mmHg

Pulmonary Artery Maximum Velocity 1.31m/s=6.8mmHG

TR Maximum Velocity 1.79m/s=1.79mmHG

ECHOCARDIOGRAM REPORT:

The interventricular septum and left ventricular wall are mildly thickened in diastole (except when using the Golden Retriever breed specific reference range where the IVS was considered to be normal). There is good systolic function shown by normal FS% and normal left ventricular chamber size in systole. There is a pattern of decreased left ventricular relaxation on the ME and MA profiles on PW Doppler indicating grade one diastolic dysfunction. There is no left atrial enlargement. The mitral valves and the tricuspid valves appear normal. Mild tricuspid regurgitation and mild pulmonic insufficiency are present and considered to be physiologic. There is mild-moderate aortic insufficiency. There is mild aortic stenosis shown by a pressure gradient of 36mmHG. Two-dimensional echocardiographic findings support a diagnosis of subaortic stenosis. There is no pulmonic stenosis shown by a pulmonary artery peak velocity of 1.31m/s. Pulmonary hypertension is ruled out using the TR jet to estimate pulmonary artery systolic pressure. The right heart is subjectively normal. There are no cardiac tumors or pericardial effusion present.

DDX: Mild subaortic stenosis with mild-moderate aortic insufficiency

DISCUSSION:

Echocardiography is a non-invasive diagnostic tool used to screen for congenital heart disease in veterinary patients. Subaortic stenosis (SAS) is one of the most common forms of congenital heart disease in the dog (1,2). Breeds that are known to be at increased risk include the Golden Retriever, Rottweiler, Boxer, German Shepherd and Newfoundland (1,2,3). Congenital aortic stenosis is rare in the cat but has been reported in the literature (4,5). As in our patient, most dogs with aortic stenosis are noted to have a heart murmur as an incidental finding at the time of physical examination or vaccination (6). A systolic ejection-type murmur heard best over the left fourth intercostal space that radiates up the carotid artery is usually present and may be accompanied by a diastolic murmur of concurrent aortic insufficiency (7).

Anatomically, aortic stenosis is classified as supra-ventricular, valvular, or subvalvular with subvalvular stenosis representing more than 95% of the lesions identified (1,2). The classic description of SAS in the dog is that of a discrete fibrous ridge or collar that completely or partially encircles the LVOT immediately below the aortic valve (8). Three classes of fixed subaortic stenosis are described: Class one involves nodules within the left ventricular outflow tract (LVOT). Class two involves a fibrous ridge of tissue in the LVOT and class three involves a tunnel type of stenosis where the mitral valve is stiff and may be dysplastic forming the posterior wall of the obstructive subvalvular tunnel (1). It is therefore important to evaluate the mitral valves when screening patients for SAS. Mild cases of SAS may not show a discrete subvalvular lesion at all (6). In our patient, a small ridge of tissue was noted on the ventricular septum just proximal to the aortic valve in the right parasternal

long-axis 5-chamber view. The mitral valves were subjectively normal and no mitral regurgitation was documented.

Dynamic SAS may also occur as the result of hypertrophy at the base of the interventricular septum or systolic anterior motion of the mitral valve (1,2). Dynamic SAS is more commonly associated with systolic anterior motion (SAM) of the mitral valve in cats with hypertrophic cardiomyopathy, but has also been reported in dogs both as an isolated lesion and concurrently with SAS (9,10). In our patient, the ventricular septum proximal to the aortic valve did not show evidence of hypertrophy and mitral valve motion appeared to be normal on both on two-dimensional and m-mode analysis ruling out SAM. Furthermore, SAM is associated with mitral regurgitation which was not detected in our patient.

Several other two-dimensional echocardiographic features are associated with SAS. Aortic valve anatomy may be affected in patients with SAS (1). Partial fusion of the cusps or asymmetric opening of the cusps may be seen on long or short-axis views of the valves (11). Areas of increased echogenicity can be seen within the myocardium indicative of myocardial fibrosis (2). Neither of these findings were seen in our patient however poststenotic dilation of the ascending aorta was detected on two-dimensional analysis. This is commonly seen in patients with SAS and is best appreciated in the left parasternal cranial left ventricular outflow view (1). Assessment of the interventricular septum (IVS) and left ventricular free wall (LVW) are determined using two-dimensional and m-mode assessment. In patients with SAS, the IVS and LVW are hypertrophied, however wall thickness can be normal in mildly affected patients (1,2). Left ventricular concentric hypertrophy occurs secondary to aortic outflow obstruction which creates excessive work on the ventricle (12). Our patient showed mild concentric hypertrophy in response to the SAS. It is important to note that the degree of concentric hypertrophy cannot be used as an indicator of severity (1). An inadequate degree of hypertrophy in response to obstruction can result in early development of myocardial failure and congestive heart failure (1).

Systolic function was normal in our patient. In young animals with aortic stenosis, systolic function is generally preserved however older surviving dogs may suffer progressive decline in myocardial contractility eventually leading to congestive heart failure (13). The ME:MA ratio obtained on pulse wave Doppler indicated that our patient had grade one diastolic dysfunction. The ME wave was larger than the MA wave in diastole with a ME:MA ratio of <1 . This represents impaired myocardial relaxation (14). Decreased peak E velocity occurs because left ventricular diastolic pressure does not decrease as much as in normal hearts (14). Diastolic dysfunction has been documented in Boxers with SAS where LV relaxation, elastic recoil, filling and stiffness have been found to be abnormal (14). It is thought that compensatory left ventricular hypertrophy in aortic stenosis may lead to impaired diastolic dysfunction (1).

Colour Doppler is used to evaluate valvular leaks and can provide information about abnormal flows within the heart. A turbulent colour flow signal in the LVOT indicates the presence of a high velocity flow in this region. Both mitral and aortic insufficiencies can be seen with aortic stenosis (3). Mitral insufficiency may be secondary to mitral valve dysplasia or systolic anterior motion (11,16). This was not seen in our patient however a mild-moderate aortic insufficiency was noted on colour Doppler analysis. Aortic insufficiency has been reported in anywhere from half to three-quarters of dogs with aortic stenosis (17). Valvular insufficiencies can add volume to the concentrically hypertrophied heart resulting in increased diastolic left ventricular chamber size (1).

Continuous wave Doppler analysis of the LVOT is required to make a diagnosis of aortic stenosis. High velocity flow obtained through the left ventricular outflow tract or aorta is the defining feature of obstruction to flow and the Bernoulli equation can be applied in order to calculate the pressure gradient (1). The subcostal imaging plane has been shown to be most accurate in obtaining aortic maximum velocity measurements as it may provide better alignment for the CW Doppler cursor however the left and right parasternal apical five-chamber views can also be used (1,11). It is important to note that the outflow tract may not be in line with the aorta when SAS exists so the Doppler beam should be aligned along the outflow tract as opposed to being parallel the walls of the aorta. Colour Doppler can assist in achieving this (1). In this case, a higher aortic peak velocity was obtained using the left parasternal apical five-chamber view compared to the subcostal view. Ideally, both views should be attempted but the subcostal view can be challenging in some patients due to the pressure created by the probe on the patient in order to visualize the heart.

The degree of aortic stenosis is graded depending upon the pressure gradient obtained through the obstructed pathway. There is some controversy as to what velocity must be exceeded in order to make a diagnosis of aortic stenosis however it is generally accepted that aortic velocities $>2\text{m/s}$ are abnormal (1). When the pressure gradient is calculated using the Bernoulli equation grading of the stenosis is $<50\text{mmHg}$ mild, >50 but $<80-90\text{mmHg}$ moderate and $>80-90\text{mmHg}$ severe (1, 2). The information obtained from two-dimensional and m-mode analysis should also be considered when making a diagnosis of subaortic stenosis. It is important to screen for other concurrent congenital defects as pulmonic stenosis, patent ductus arteriosus, ventricular septal defect, and mitral dysplasia have been reported to occur with aortic stenosis (11). Our patient falls into the category of mild SAS and had several echocardiographic features that helped support the diagnosis.

Many dogs with mild aortic stenosis can be expected to enjoy long full lives in the face of this congenital defect (2,6). Dogs with severe obstruction often die either suddenly or due to complications created by concurrent defects such as mitral valve dysplasia and left sided congestive heart failure within the first three years of life (3). Dogs with mild-moderate SAS are at increased risk for developing infective

endocarditis as they tend to live long enough to be susceptible to this condition (2). As the disease is known to progress until 12 months of age, screening for dogs prior to breeding should be performed after their first year of life (18).

There is no ideal treatment protocol for the management of SAS. Both surgical and medical management have been described (13). Surgical excision of abnormal tissue in the LVOT and balloon dilatation have been attempted but often with unrewarding results or short-lasting effects (13). Beta-adrenergic blockers have been used to reduce myocardial oxygen consumption and ventricular arrhythmias in moderate to severe cases (2,13). Prophylactic use of antibiotics prior to dental surgery or other types of surgery can be considered to reduce the risk of bacteremia and endocarditis however the benefits of this remains unproven in the veterinary literature (2).

CONCLUSION: No specific treatment for Mya's SAS was recommended. Her prognosis is considered to be very good, however due to mild changes noted on her echocardiogram, a follow-up exam was recommended in 1-2 years.

REFERENCES:

1. Boon JA. Stenotic Lesions In:Boon JA.Veterinary Echocardiography 2nd. Ed Iowa:Blackwell Publising 2011:477-525.
2. Kienle RD. Aortic Stenosis In:Kittleson MD, Kienle RD. Small Animal Cardiovascular Medicine 1st. Ed. VIN online textbook 1998.
3. Kienle RD, Thomas WP, Pion PD. The natural clinical history of canine congenital subaortic stenosis. J Vet Intern Med. 1994;8:423-431.
4. Stepien RL, Bonagura JD: Aortic stenosis: clinical findings in six cats, J Small Anim Pract 1991;32:341.
5. Lui S: Supravalvular aortic stenosis with deformity of the aortic valve in a cat, J Am Vet Med Assoc 1968;152:55.
6. O'Grady MR, Holmberg DL, Miller CW, Cockshut JR. Canine congenital aortic stenosis: a review of the literature and commentary. Can Vet J. 1989;30:811-815.
7. Goodwin JK. Congenital Heart Disease In:Tilley LP, Goodwin JK. Manual of Canine and Feline Cardiology 3rd. Ed. Philadelphia:WB Saunders 2001:273-293.
8. Patterson DF: Congenital defects of the cardiovascular system of dogs: studies in comparative cardiology, Adv Vet Sci Comp Med. 1976; 20:1.
9. Sisson DD, Thomas WP: Dynamic subaortic stenosis in a dog with congenital heart disease, J Am Anim Hosp Assoc 1984;20:657.
10. Liu SK, Maron BJ, Tilley LP: Canine hypertrophic cardiomyopathy, J Am Vet Med Assoc. 1979; 174:708.
11. Bussadori C, Amberger C, Le Bobinnec G, et al. Guidelines for the echocardiographic studies of suspected subaortic and pulmonic stenosis. J Vet Cardiol 2000;2:15-22.

12. Oyama MA, Thomas WP. Two-dimensional and M-mode echocardiographic predictors of disease severity in dogs with congenital subaortic stenosis. *J Am Anim Hosp Assoc* 2002;38:209-215.
13. Fuentes VL. Aortic Stenosis In: Abbott JA. *Small Animal Cardiology Secrets*. Philadelphia: Hanley and Belfus Inc 2000:300-305.
14. Boon JA. Evaluation of size, function, and hemodynamics In: Boon JA *Veterinary Echocardiography 2nd*. Ed. Iowa: Blackwell Publishing 2011:153-255.
15. Schober KE, Fuentes VL. Doppler echocardiographic assessment of left ventricular diastolic function in 74 boxer dogs with aortic stenosis. *J Vet Cardiol* 2002;4:7-16.
16. Buoscio D, Sisson D, Zachary J, et al. Clinical and pathological characterization of an unusual form of subvalvular aortic stenosis in four golden retriever puppies. *J Am Anim Hosp Assoc* 1994;30:100-110.
17. O'Grady M. The incidence of aortic valve insufficiency in congenital canine aortic stenosis: a Doppler echocardiographic study. *J Vet Int Med* 1990;4:129.
18. Pariaut R. Heart In: Barr F, Gaschen L *BSAVA Manual of Canine and Feline Ultrasonography*. Gloucester: BSAVA 2011:37-70.