



**PATIENT**

Spock Amodeo

**SPECIES**

Feline

**BREED**

DSH

**SEX**

Female

**AGE**

5 Months

**WEIGHT**

5.12 Pounds

**INTERPRETED BY**

Eric Lindquist, DMV,  
 DABVP (Canine & Feline), Cert. IVUSS

**IMAGING PERFORMED BY**

Shari Reffi, CVT

**HOSPITAL NAME**

Heart & Paw Lk  
 Hopatcong

**REFERRING VET**

Dr. Caitlin Verhalen

**INVOICE**

35044

**DATE**

12/22/25

**PRESENTING CLINICAL SIGNS**

History: Pre-anesthetic heart evaluation for spay. Grade V/VI heart murmur. Asymptomatic. Meds: (Torb on board for scan).

**ULTRASONOGRAPHIC EXAMINATION OF THE HEART**

FELINE CARDIAC PARAMETERS	BODY WEIGHT (lbs)	HR (BPM)	IVSd (cm)	LVIDd (cm)	LVWd (cm)	FS (%)	EF (%)
<b>NORMAL PARAMETER</b>	-----	150-240	0.3-0.6	1.0-2.1	0.25-0.6	35-67	80-100
<b>PATIENT</b>	5.12 lbs	167	0.33	1.77	0.33	28	57
FELINE CARDIAC PARAMETERS	LA/AO (M-mode)	LA/AO HEART BASE (Sisson)	LAD LA MAX 4 Chamber		LVOT VEL. (m/s)	RVOT VEL. (m/s)	IVRT (m/)
<b>NORMAL PARAMETER</b>	<1.5	1.6	0.7-1.7		<1.6	<1.3	40-60
<b>PATIENT</b>	1.3	1.3	--		1.00	1.00	NM
Adapted from June Boon, Veterinary Echocardiography, 1998 Sisson D et al. JVIM 1991; 5: 232, Jacobs et al. Am J Vet Res 1985; 46:1705							

E-wave velocity: 1.0

**Cardiac Presentation**

The echocardiogram in this patient demonstrated normal left atrial size based on 3 separate LA measurements. The cranial and caudal mitral valve leaflets presented normal linear structure and kinetics. Left and right ventricular septal defect was noted in this patient. This appears to be compensated at this time. The right atrium was slightly enlarged. Minor right ventricular eccentric hypertrophy was noted, with minor volume overload of the right ventricle and pulmonary artery. Minor tricuspid insufficiency was noted, compensated. No visible pericardial or free pleura fluid was noted or extra cardiac pathology in the visible planes. The cranial mediastinum and pericardial regions were free of masses in the visible window.

**ULTRASONOGRAPHIC FINDINGS**

- Ventricular septal defect, compensated
- Minor tricuspid insufficiency
- Slightly enlarged right atrium
- Minor right ventricular eccentric hypertrophy with minor volume overload of the right ventricle and pulmonary artery

**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**



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No overt contraindication to anesthetic procedure at this time. Recheck echo at full 1 year of age. Torbutrol (premed), propofol (induction), and isoflurane (maintenance) is recommended.

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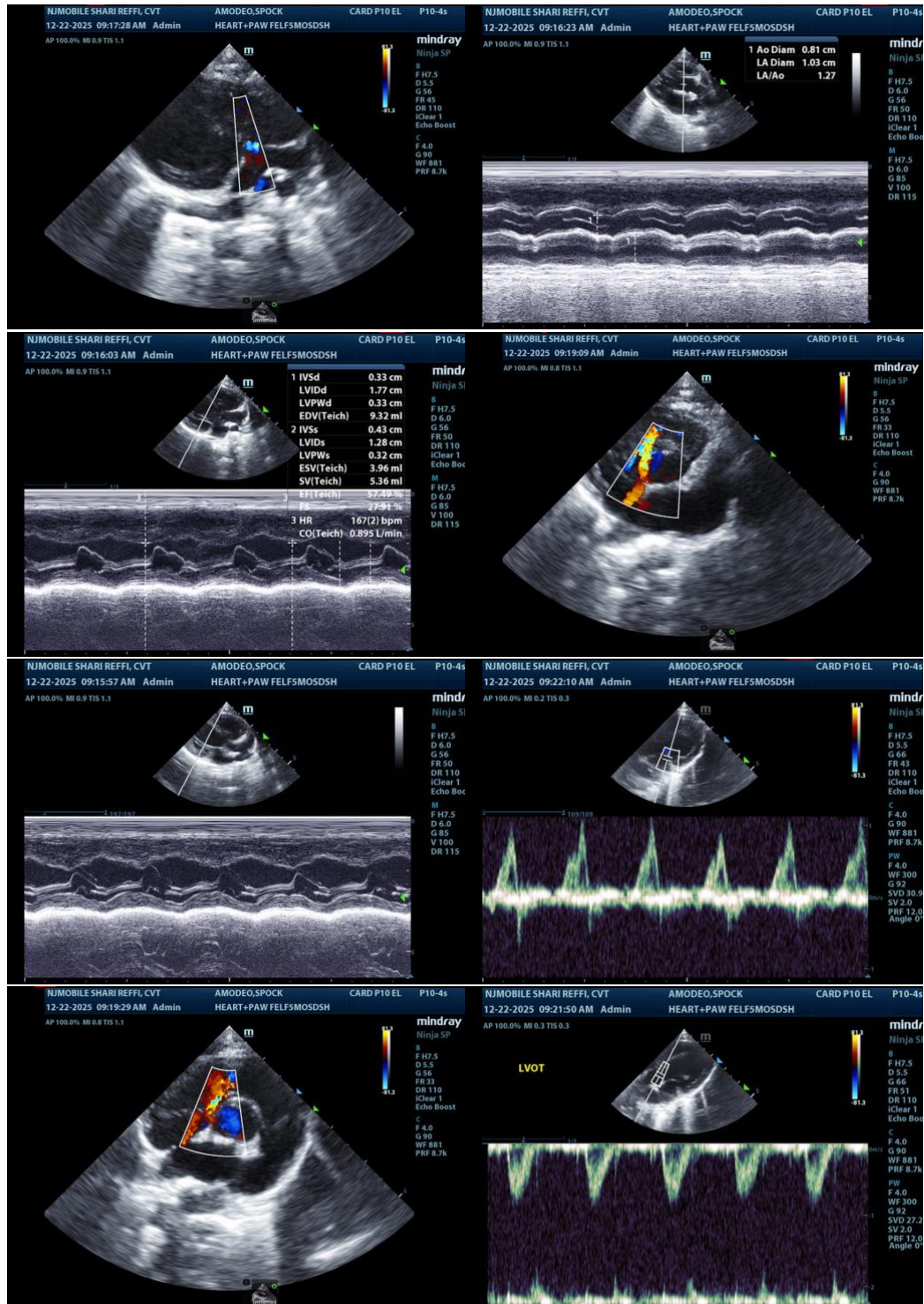
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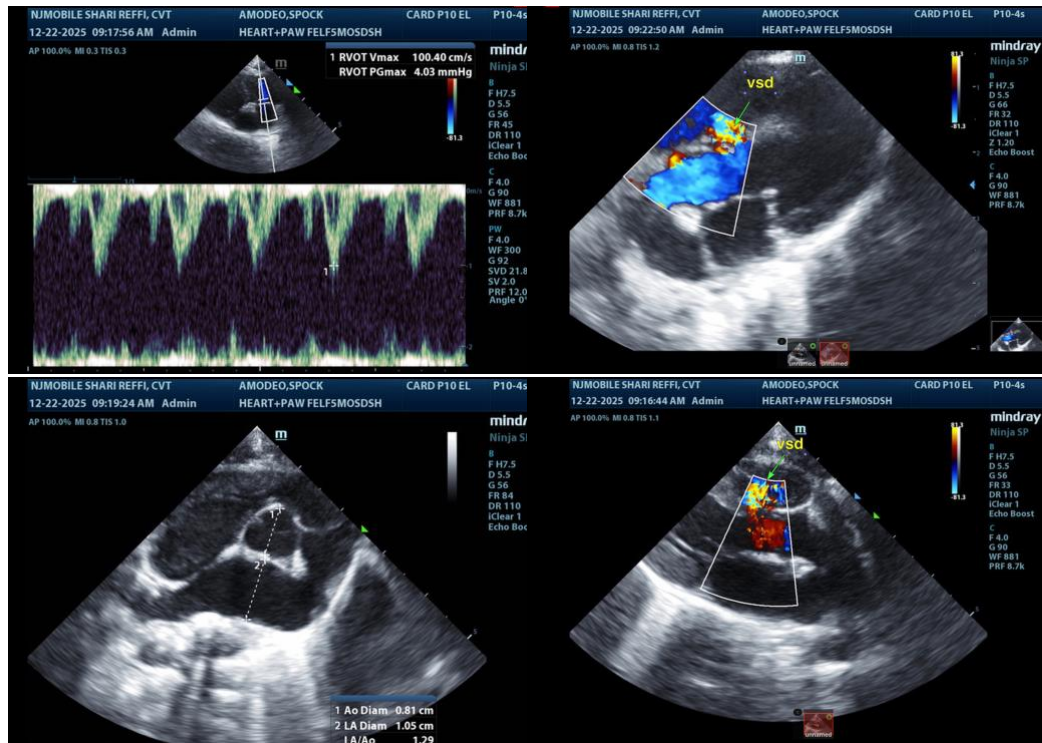
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**The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.**

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(CFM), Cert. IVUSS,**  
 CEO, Owner, Founder -- SonoPath.com  
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**Ventricular Septal Defect**

<http://www.sonopath.com/VSD>

<http://www.sonopath.com/EchoModler>

**Description:** Although ventricular septal defects (VSDs) account for less than 10% of congenital cardiac defects in dogs, they are far more common in cats, accounting for 56% of all cardiac defects. In dogs, the most frequently affected breeds



**PATIENT**

are the West Highland White Terrier, Keeshound, Mongrel, Pinscher, French Bulldog, German Shepherd, Labrador Retriever, English Springer Spaniel, and Basset Hound.

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The most common VSDs in dogs and cats are located in the basal septum. The left side of the defect is typically located just below the aortic valve, while the right side of the defect is located either in the inflow tract ("subcrystal" or "perimembranous") or the outflow tract ("supracrystal"). VSDs can occur in combination with other congenital abnormalities or as part of an endocardial cushion defect. Muscular defects are less common. The resulting hemodynamic consequence depends mainly on the size of the VSD. In small defects, the pressure difference between the left and right heart remains normal and the shunt flow moves at a high velocity. Depending on the shunt volume, VSDs can lead to volume overload in the left heart and pulmonary artery. A large shunt will typically result in a pressure equilibrium across both chambers; however, if pulmonary hypertension develops, then reverse shunting can occur. The latter occurs mainly in cats and only rarely in dogs. VSDs that develop in combination with other defects will have a completely different pathophysiology.

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**Clinical signs:** Only a minority of patients with VSDs will show clinical signs. If the VSD is not too large (i.e., still resistive), a (usually loud) heart murmur can be auscultated on the right hemithorax. Large, non-resistive defects are not necessarily associated with a murmur.

**WEIGHT**

5.12 Pounds

**Diagnostics:** Perimembranous VSDs are easily seen on a right-sided apical five-chamber view just below the aortic valve. They can also be clearly seen on short axis views of the heart base. Supracrystal defects are easier to see on short axis views than on regular five-chamber views. To identify muscular defects, careful tracking of the interventricular septum using multiple short axis views is necessary. They can also be seen on left apical views when the transducer is placed a bit further dorsally (i.e., not perfectly apical). In small, resistive defects, the shunt flow will be observed as a high-velocity systolic jet. The typical maximal flow velocity (Vmax) is approximately 5 m/s if normal pressure differences between the chambers are still present. Large defects do not show high-velocity flows. Slow left-to-right, right-to-left, or bidirectional flow can be displayed on a color Doppler ultrasound. In the latter case, the flow velocities are best displayed by PW-Doppler. Reversal of flow may also be demonstrated with a bubble study.

**INTERPRETED BY**

Eric Lindquist, DMV,  
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**Treatment:** The treatment depends on the pathophysiological status of the patient. Patients that have small defects with an insignificant flow do not require therapy; they can lead normal lives. Larger defects with marked left-sided and pulmonary arterial volume overload can be treated medically with the following: pimobendan (0.25-0.3 mg/kg PO BID); angiotensin-converting enzyme (ACE) inhibitors (enalapril at 0.5 mg/kg PO Q12-24hr or benazepril 0.25-0.5 mg/kg PO Q24hr); furosemide (1-2 mg/kg PO Q12-24hr, which can be increased incrementally, as needed); and/or spironolactone (0.25-2 mg/kg PO BID). Alternatively, they can be treated surgically via one of two options: pulmonary artery banding to increase right ventricular pressures and reduce shunting volumes, or closure of the defect via open heart surgery or a minimally invasive VSD closure.

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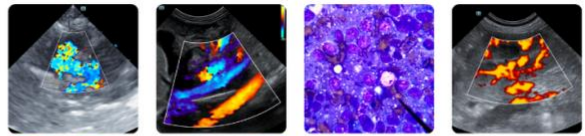
Bonagura JD, Lehmkuhl LB. Congenital heart disease. In Fox PR, Sisson D, Moise NS, eds. Canine and Feline Cardiology. Philadelphia, PA: WB Saunders; 1999:471-535.

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Oliveira P, Domenech O, Silva J, et al. Retrospective review of congenital heart disease in 976 dogs. *J Vet Int Med* 2011;25:477-83.

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Riesen S, Kovacevic A, Lombard C, Amberger C. Prevalence of heart disease in symptomatic cats: an overview from 1998 to 2005. *Schweiz Arch Tierheilk* 2007;149:65-72.

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Saunders AB, Carlson JA, Nelson DA, et al. Hybrid technique for ventricular septal defect closure in a dog using an Amplatz Duct Occluder II. *J Vet Cardiol* 2013;15:217-24.

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