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IDEXX 1

1

WVC Vegas

DIAGNOSING CARDIAC DISEASES

Augusta Pelosi, DVM,
Dipl. ACVS and ACVIM (Cardiology)

2

CONFLICT OF INTEREST DISCLOSURE

I have financial interest, arrangement or affiliation with:

IDEXX

WVC Vegas

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OVERVIEW

PART 1. CONGENITAL CARDIAC CONDITIONS

Disease Presentation and Progression
Diagnostics

PART 2. ACQUIRED CARDIAC CONDITIONS

Disease Presentation and Progression
Diagnostics

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PERSISTENT EMBRYOLOGIC ANATOMY

- PDA
- PFO
- TRUNCUS
- COR TRIATRIATUM
- DOUBLE CHAMBER RIGHT VENTRICLE

ABERRANT DEVELOPMENT

- VASCULAR RINGS
- PERSISTENT LEFT CRANIAL VENA CAVA
- PS
- SAS
- AV VALVE DYSPLASIA
- ASD/VSD/AVSD
- AP WINDOW
- DOUBLE OUTLET VENTRICLES
- DOUBLE INLET
- TOF
- TGA
- COARTATION/INTER. ARCH

5

PATENT DUCTUS ARTERIOSUS (PDA)

BREEDS

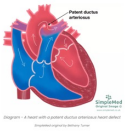
Chihuahua, Bishon frise, Collie, CKCS, English Springer Spaniel, German shepherd, Kerry Blue Terrier, Keeshond, Maltese, Pomeranian, Poodle, Shetland sheepdog, Welsh Corgi, Yorkshire terrier

SEX ~80% are female

6

THE FACTS AND THE CHRONOLOGY

- It is a communication between Ao and PA
- In the fetus, it shunts right to left shunt (to bypass lungs)
- ↓ pulmonary resistance → left to right shunt
- It should close postnatally by constriction of wall muscles by 3 weeks
- Lack of muscle allows for patency



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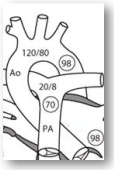
What's the normal sys and dia pressure in the

AORTA?

PULMONARY ARTERY?



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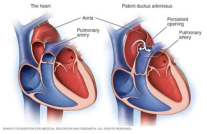
PHYSIOLOGY

Continuous shunting from left to right as in "normal" conditions the pressure in Ao > pressure PA throughout cycle

- Loads PA, lungs, left side
- Causes left sided **VOLUME** load and CHF



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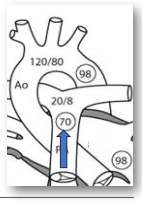
The "reversed" shunt

The direction of blood flow reverses due to severe pulmonary hypertension.

PH allows reversal of shunt → R to L PDA
PRESSURE load on RIGHT side



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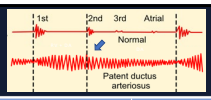
PHYSICAL EXAM



	LEFT-TO-RIGHT		RIGHT-TO-LEFT
	Restrictive	***Nonrestrictive with low pulmonary resistance***	***Nonrestrictive with suprasystemic pulmonary vascular resistance***
SIGNS	Asymptomatic	CHF	Differential Cyanosis
ARTERIAL PULSE	Normal, wide, bounding	**Wide, bounding**	Normal (+++)
AUSCULTATION	**Continuous** murmur at heart base Thrill throughout sys-dia	Murmur throughout sys-dia Murmur and thrill may be just in sys	Murmur **absent**

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CLINICAL PRESENTATION

- **Asymptomatic -** Identified due to presence of murmur
- **CHF -** Pulmonary edema, respiratory distress
- **Reversed PDA**
  - Cyanosis
  - Polycythemia: PCV (70-80%)

Dogs → happens over short time
May miss the "reversal" phase
Cats → can occur over longer period
Witness the switch
Continuous → Systolic → absent

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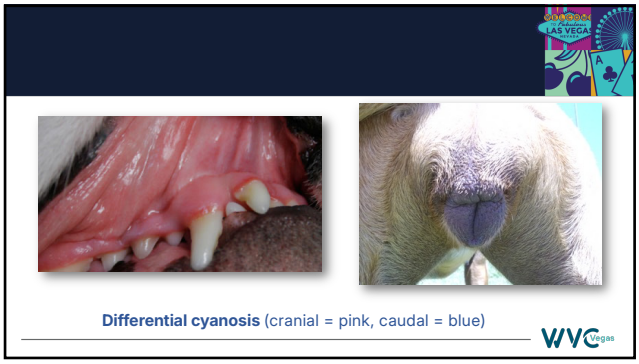

Differential cyanosis (cranial = pink, caudal = blue)

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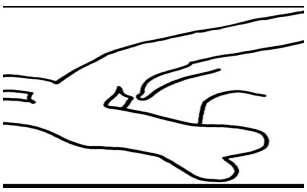
Why in PDA we see differential cyanosis?



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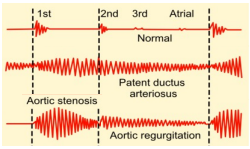
DIFFERENTIAL DIAGNOSES for PDA

- **Conditions producing To-and-fro murmurs instead of continuous (combo systolic and diastolic)**
- Aortopulmonary fistula
- Aortopulmonary windows



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DIAGNOSTICS - RADIOGRAPHS & ECG

Left-to-Right		Right-to-Left
Moderately Restrictive	*Nonrestrictive with low pulm. resistance*	*Nonrestrictive with suprasystemic pulm. resistance*
Left heart enlargement Increased pulmonary vascularity Enlarged pulmonary trunk Prominent ascending Ao	Enlargement of all four chambers CHF	Reduced pulmonary vascularity Dilation pulmonary trunk Hypertrophy RV Normal or near normal left heart

ECG

- Non-specific
- Left sided chamber enlargement with left-to-right shunt
  - Wide P waves → LAE, Tall R waves →LVE
- Right ventricular hypertrophy and peak P wave with right-to-left shunt

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VASCULAR RINGS

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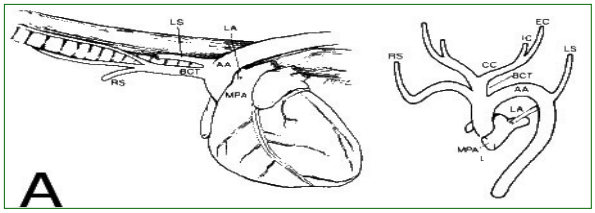

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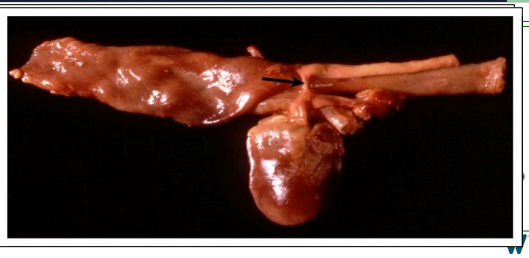
PERSISTENT RIGHT AORTIC ARCH (PRAA) WITH LEFT LIGAMENTUM ARTERIOSUM



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DIAGNOSTICS - RADIOGRAPHS

J Vet Intern Med 2004;18:510–514

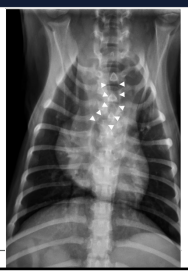
Tracheal Signs and Associated Vascular Anomalies in Dogs with Persistent Right Aortic Arch

James W. Buchanan

Table 2. Tracheal position in 120 dogs.

	Controls	Megaesophagus	PRAA
Marked rightward	3	4	
Moderate rightward	11	9	
Mild rightward	24	14	
Midline	24	3	
Mild leftward	1		
Moderate leftward			7
Marked leftward			20
Total	63	30	27

PRAA, persistent right aortic arch.



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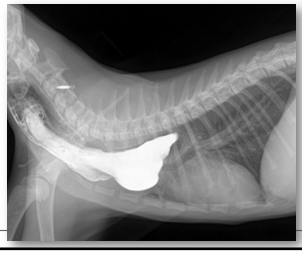
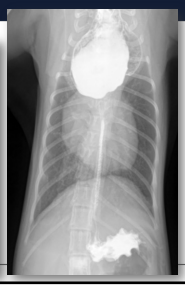
DIAGNOSTICS - ESOPHAGRAM





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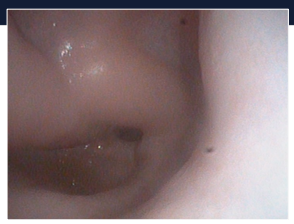
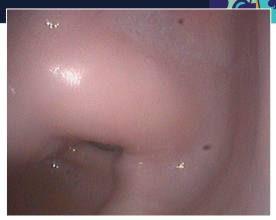
DIAGNOSTICS - ESOPHAGOSCOPY





Look for extraluminal compression, try to detect pulsations from adjacent artery

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PULMONIC STENOSIS

BREEDS

English bulldog, French bulldog, Mastiff, American pit bull terrier, Miniature schnauzer, West Highland white terrier

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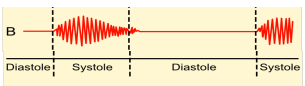
PHYSICAL EXAM

- Ejection quality, systolic left heart base murmur
- Normal pulse quality and lung sounds
- Persistent foramen ovale is often present
- Dog may be asymptomatic or exhibit signs such as: exercise intolerance, dyspnea, cyanosis



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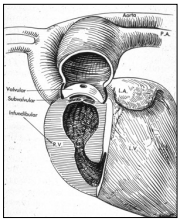
VALVULAR STENOSIS
- With or without annulus stenosis
- With or without aberrant coronary

SUBVALVULAR STENOSIS
- Fibrotic or muscular stenosis
- Aberrant coronary artery

SUPRAVALVULAR STENOSIS
- Rare



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DIAGNOSTICS - RADIOGRAPHS & ECG

ECG
- RVH

RADIOGRAPHS
- Right ventricular enlargement; cardiac apex may be lifted off the sternum
- Post-stenotic dilation
- Lung fields normal or hypoperfused

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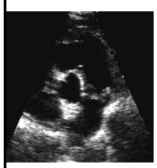

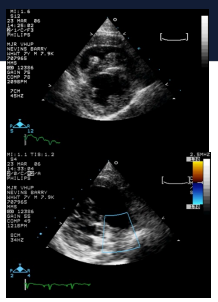

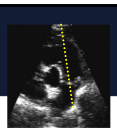

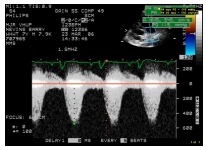

Modified Bernoulli equation
$V=5.45$ m/s $PG=4V^2$
$PG=119$ mmHg

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SUBAORTIC STENOSIS

BREEDS

Newfoundland, Golden Retriever, Dogue de Bordeaux, Rottweiler, Bouvier de Flandres, Bullmastiff

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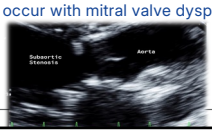
Fixed Subaortic Stenosis: fibrotic band with possible concurrent malformation of the mitral valve

Subvalvular stenosis is most common in dog, but supravalvular may be more common in cats

Sometimes tubular fixed stenosis

Usually isolated but may occur with mitral valve dysplasia



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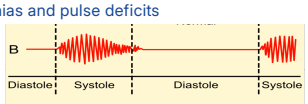
PHYSICAL EXAM

- Common in dog, rare in other species
- Often asymptomatic, but may have history of fatigue, syncope or sudden death
- Ejection quality systolic murmur at the left heart base
- Murmur or bruit may extend up the carotid arteries
- Poor pulse quality with moderate to severe disease
- +/- arrhythmias and pulse deficits



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DIAGNOSTICS - RADIOGRAPHS & ECG

ECG
- Normal or LVH, ST segment deviation and arrhythmias with severe disease

RADIOGRAPHS
- Often unremarkable, but left ventricular enlargement is possible
- Aortic root dilation

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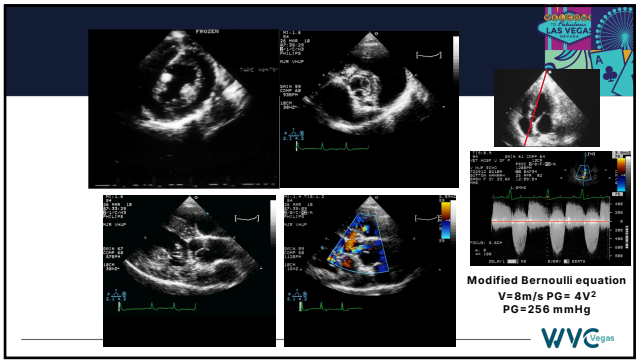

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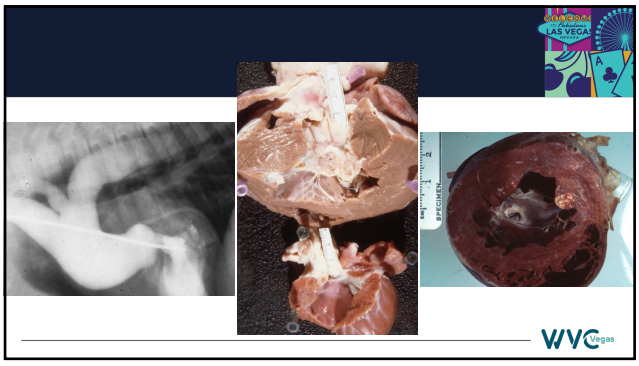

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CONDITIONS - Acquired

DEGENERATIVE VALVE DISEASE

ALTERATION OF ANATOMY

DCM

HCM

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DCM

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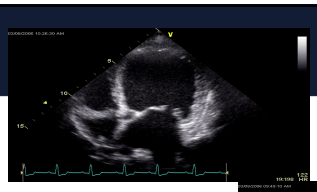

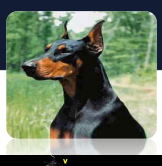

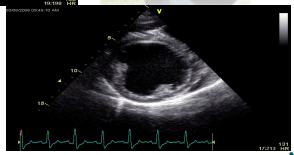

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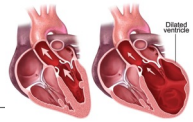
DEFINITION

A heterogeneous group of diseases whose commonalities are substantial morphologic and/or functional abnormalities of the *myocardium*

Though it could involve any heart chamber, it usually refers to the *ventricular* myocardium



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Fairly common problem in small animal medicine

- 0.5% of the dogs evaluated at U.S. referral-hospitals were diagnosed with DCM from 1986 to 1991 (Sisson et al., 2000)
- 0.4% of the dogs evaluated in a US hospital (90,004) from 1995 and 2010; subset of dogs with an inherited disease (27,254 cases) presented an incidence rate of 1.3% (Bellumori et al. 2013)
- 1.1% of the dog population seen at veterinary hospitals (Fioretti and Delli, 1988)

Substantial impact on morbidity & mortality

Though only rarely curable (except for some secondary cardiomyopathies), effective "palliative" therapy is possible in many cases

GENETIC COUNSELING for familiar forms

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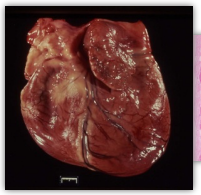
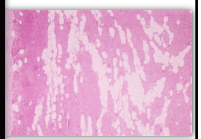
- Dilated chambers
  - Left atrium/ventricle
  - Right side also affected
- Histopath changes
  - Fibrous tissue
  - Fatty infiltration





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TYPES CARDIOMYOPATHIES	Classification
ISCHEMIC CARDIOMYOPATHY (hypothyroidism ?)	Etiologic
TOXIC CARDIOMYOPATHY [alcoholic, drug-induced ex. doxorubicin]	Etiologic
NUTRITIONAL/DEFICIENCY CARDIOMYOPATHY ▪ BEG (Boutique, exotic meats & grain free) diets ▪ L-carnitine ▪ Taurine deficiency	Etiologic
MYOCARDITIS (autoimmune & infectious)	Etiologic
TACHYCARDIA-INDUCED CARDIOMYOPATHY	Etiologic
INFILTRATIVE CARDIOMYOPATHY ▪ amyloid ▪ neoplastic	Morphologic
HYPERTROPHIC CARDIOMYOPATHY	Morphologic
DILATED CARDIOMYOPATHY	Functional
Subset: familial Cardiomyopathy (genetic) [Ex. Dobermann, "Boxer" cardiomyopathy, Portuguese Water dog]	Genetic

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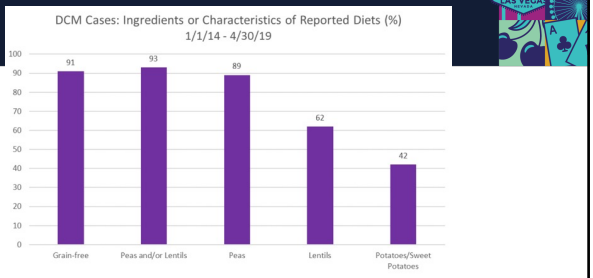

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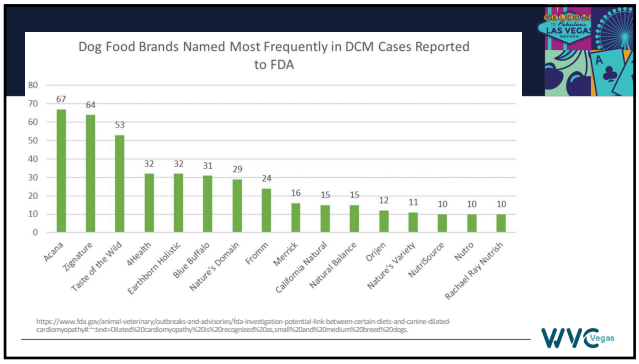

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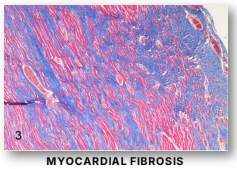
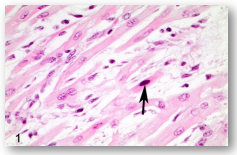
Parvovirus Infection Is Associated With Myocarditis and Myocardial Fibrosis in Young Dogs

Jordan Ford[1], Laura McEndaffer[1], Randall Renshaw[2], Alex Molesan[1], and Kathleen Kelly[1]



MYOCARDIAL FIBROSIS

INCLUSION BODIES



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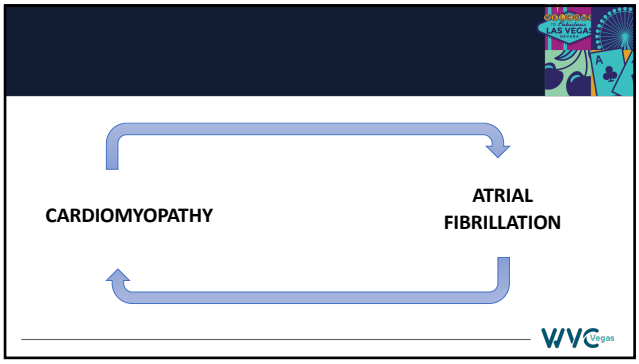
HYPERTROPHIC CARDIOMYOPATHY	Morphologic
DILATED CARDIOMYOPATHY	Functional
Subset: familial Cardiomyopathy (genetic) [Ex. Dobermann, "Boxer" cardiomyopathy, Portuguese Water dog]	Genetic

63



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TYPES CARDIOMYOPATHIES	Classification
ISCHEMIC CARDIOMYOPATHY (hypothyroidism ?)	Etiologic
TOXIC CARDIOMYOPATHY [alcoholic, drug-induced ex. doxorubicin]	Etiologic
NUTRITIONAL/DEFICIENCY CARDIOMYOPATHY ▪ BEG (Boutique, exotic meats & grain free) diets ▪ L-carnitine ▪ Taurine deficiency	Etiologic
MYOCARDITIS (autoimmune & infectious)	Etiologic
TACHYCARDIA-INDUCED CARDIOMYOPATHY	Etiologic
INFILTRATIVE CARDIOMYOPATHY ▪ amyloid ▪ neoplastic	Morphologic
HYPERTROPHIC CARDIOMYOPATHY	Morphologic
DILATED CARDIOMYOPATHY	Functional
Subset: familial Cardiomyopathy (genetic) [Ex. Dobermann, "Boxer" cardiomyopathy, Portuguese Water dog]	Genetic

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- Large & giant breed dogs preferentially affected
- 58% of Doberman Pinschers affected by DCM [*Wess et al. 2010 and Dutton et al. 2018*] and up to ~25% of Boxers affected by ARVC [*Stern et al. 2010*].

BREEDS

- Suggests a familial / genetic basis
- Great Dane, Doberman Pinscher, Portuguese Water Dog, Irish Wolfhound, Newfoundland, Boxer, Welsh Springer Spaniels, and Cocker Spaniel

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Name	Breed affected	Disease	Gene involved	Mutation type	Biological result	Mode of inheritance	Penetrance
DCM1	Doberman Pinscher	Dilated cardiomyopathy	Pyruvate dehydrogenase kinase 4 (PDK 4)	16 Base pair deletion	Altered cardiomyocyte metabolism with preferential glucose oxidation	Autosomal dominant	68%
DCM2	Doberman Pinscher	Dilated cardiomyopathy	Titin	Single base pair (missense) change from C to T	Incompletely understood; hypothesized changes to secondary structure resulting in titin unfolding and degeneration	Autosomal dominant	47%
Striatin	Boxer	Arrhythmogenic right ventricular cardiomyopathy	Striatin	[illegible] pair	Altered electrical conduction and structural integrity between myocytes	Autosomal dominant	72%

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Name	Breed affected	Disease	Gene involved	Mutation type	Biological result	Mode of inheritance	Penetrance
Striatin	Boxer	Arrhythmogenic right ventricular cardiomyopathy	Striatin	8 Base pair deletion	Altered electrical conduction and structural integrity between myocytes	Autosomal dominant	72%

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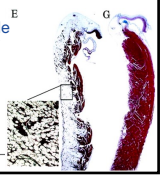
"BOXER CARDIOMYOPATHY"

ARVC Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Present with ventricular arrhythmias
- No MR (no murmur)
- ECG (especially Holter exam) shows VPCs - 'left bundle branch block morphology' because originate in the RV
- In U.S., the echo is usually normal (>90 %)
- Sometimes have dilated LV with systolic impairment
- In rare cases have marked RV dilation and dysfunction



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CLINICAL MANIFESTATIONS

- Ventricular arrhythmias can be the first (but often missed) manifestation (**OCCULT**)
- Heart enlargement and poor systolic function are the final morphologic and clinical forms
- Syncope and/or CHF signs are the usual trigger to seek veterinary advice

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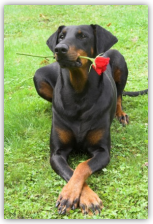
OCCULT PHASE

ASYMPTOMATIC (DEFINITION)

Echocardiographic changes
- LV dilation
- Systolic dysfunction
- Mitral regurgitation

May have arrhythmias
- Usually ventricular
- Identify by Holter



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From the OWNER's Perspective

- ✓Fast breathing when resting or sleeping (> 30-35 breaths per minute)
- ✓Increased effort associated with breathing
- ✓Restless sleeping, moving around a lot and changing positions
- ✓Coughing or gagging
- ✓Weakness
- ✓Reduced ability to exercise
- ✓Collapse or fainting
- ✓Decreased appetite
- ✓Weight loss
- ✓Distended abdomen
- ✓Depressed attitude or quiet and not interactive
- ✓Sudden death

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From the CLINICIAN's Perspective

Usually starts with an "Index of Suspicion"

- **Detecting environmental causes / triggers**
  - Nutrition (strange diets in cats – taurine deficient)
  - "Toxins" – doxorubicin
- **Family history and signalment**
  - Dobermans, Boxers, Main Coon cats, Ragdoll cats, etc
- **Clinical history & Physical Exam**
  - Syncope
  - CHF (left or right or both)
  - Murmur (left apical systolic) or Gallop sounds (S3)
  - Arrhythmias: VPCs or Atrial fibrillation

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DIFFERENTIAL DIAGNOSES for DCM

- Acutely Depressed systolic function caused by:
  - Drugs (anesthesia, high dose beta blockers, etc)
  - Myocardial depressant factor
    - Liberated by severe abdominal visceral disease
- Chronically depressed systolic function caused by:
  - Atrial fibrillation

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DIAGNOSTICS

- Genetic testing
- Bioassays
  - Plasma taurine, troponin, proBNP
- ECG
- Holter
- Radiographs
- Echo

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NT-PROBNP ASSAY

SCREENING TOOL

Usefulness of cardiac biomarker screening to detect dilated cardiomyopathy in Dobermanns

J. Dukes-McEwan[1]*, K. E. Garven[1], J. Lopez Alvarez[1], P. Oliveira[1], P. F. Mottershead[1] and R. Willis[1]

FOLLOW UP

PREDICTION OF SURVIVAL

Prospective Evaluation of NT-proBNP Assay to Detect Occult Dilated Cardiomyopathy and Predict Survival in Doberman Pinschers

G.E. Singletary, N.A. Morris, M. Lynne O'Sullivan, S.G. Gordon, and M.A. Oyama

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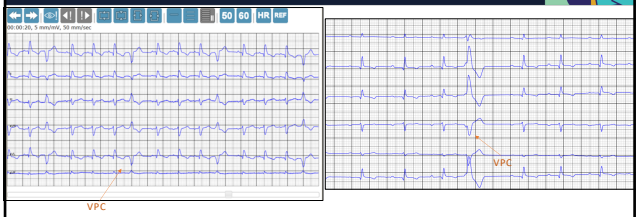
IDENTIFICATION VPCs



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Note: Upright, wide and bizzare QRS complexes of a different configuration of the normal sinus beats without associated P waves.

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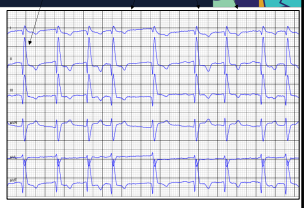
IDENTIFICATION ATRIAL FIBRILLATION



- Very common arrhythmia
- Chaotic, disorganized atrial activity
- Three classic criteria:
  1. No P waves (fibrillation waves)
  2. 'Irregularly irregular'
     - Tennis shoes in a dryer
  3. Rapid rate
- Often associated with significant underlying structural heart disease

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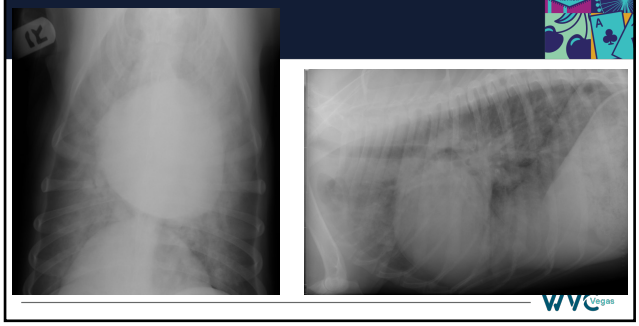

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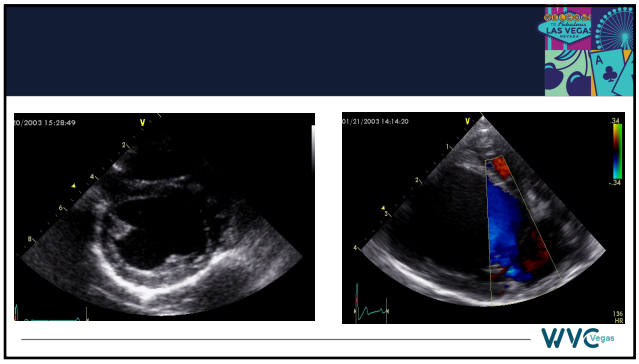

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DEFINITION

A heterogeneous group of diseases whose commonalities are substantial morphologic and/or functional abnormalities of the *myocardium*.

- **Electrical** right ventricular arrhythmia
- **Mechanical** Systolic dysfunction → DCM or Diastolic dysfunction → HCM/RCM
- **Those that don't quite fit** Unclassified cardiomyopathy (UCM)

INCIDENCE:
Hypertrophic cardiomyopathy is the most common form identified in humans [*Maron et al. 1995*] and felines [*Payne et al. 2015*], with prevalence 10-15% in felines [*Payne et al. 2015*,6].

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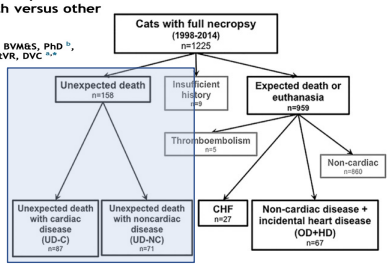
Cardiac pathology findings in 252 cats presented for necropsy; a comparison of cats with unexpected death versus other deaths[☆]

L.J. Wilkie, BSc, BVetMed [a,*], K. Smith, BVM&S, PhD [b], V. Luis Fuentes, MA, VetMB, PhD, CertVR, DVC [a,*]



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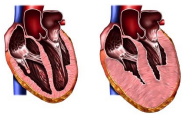
HCM

- Usually a familial / genetic condition that we see primarily in cats, & rarely in dogs or other species
  - Most common acquired adult cat heart disease
- Variable, but potentially serious effects on morbidity and mortality



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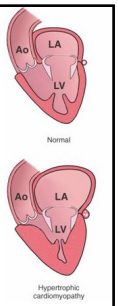
Forms of HCM

GENERALIZED HCM
- Free wall, septum, papillary muscles

OBSTRUCTIVE HCM
- Systolic anterior motion (SAM) of mitral valve
  - MR with LVOT turbulence
- *HOCM* = HCM + SAM



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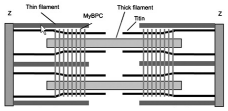
HCM – Known Etiology

Mutation in myosin binding protein C

- Different mutation for M. Coon, and Ragdoll cats
  (two known mutations, one suspected, others likely >400 known for humans)
- Male predilection (in some studies 3:1)



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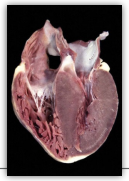
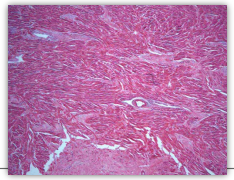
- Thickened muscle, particularly LV septum
- Fibrosis, intramural coronary artery disease
- Marked myofiber disarray





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CLINICAL MANIFESTATIONS

- Normal systolic function
- Variable degrees of diastolic failure (none to severe)
- Asymptomatic
  - Common, especially if heart rate is consistently below 200 bpm in exam setting
- Left sided CHF
- Sudden death (~5-15%)
- Aortic thromboembolism (5-15%)

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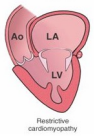
RESTRICTIVE (RCM)

A less common form of cardiomyopathy of unknown etiology, seen almost exclusively in cats.

What is it?
- "Diastolic failure that isn't HCM"
- Irregular but not overtly thickened ventricular walls
- Sometimes have mild to moderate systolic failure



- Similar features as HCM (physical exam)

- Echo is key to diagnosis
  - Usually massive and disproportionate left atrial dilation with no other explanation (not severe MR)
  - Not HCM

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UNCLASSIFIED CARDIOMYOPATHY (UCM)

- Muscle wall becomes fibrotic or stiff
  - Not hypertrophic
- Diastolic dysfunction
  - Biatrial enlargement
- Also has systolic dysfunction
  - Not as bad as DCM
- Commonly manifests as heart failure

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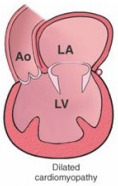
DILATED CARDIOMYOPATHY (DCM)

- Diet cardiomyopathy:
  - Taurine deficiency in diet (historical and uncommon)
  - Diet (unknown cause): low and high pea/lentil diets (Karp et al 2021)

- Eccentric hypertrophy: Left ventricular dilation & Systolic dysfunction

- Very poor prognosis
  - Unless it is reversible (taurine deficiency)



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CLINICAL PRESENTATION

Hiding from owners for a couple days
Then, dyspnea and/or tachypnea
Can present with pleural effusion or pulmonary edema
May or may not have a previous (or current) murmur

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Signalment; breeds at risk (Maine coon, Ragdolls, Sphynx), males predisposed, increased age
Owner's perspective: often no signs, exercise intolerance, increased respiratory rate

Physical Exam
- Gallop "rhythm" or sound
- Adult onset murmur (particularly if III-IV/VI or greater)
- Signs of respiratory distress (tachypnea, dyspnea)

Thromboembolism Lameness / paralysis, pain, poor or no pulse

Echocardiography – usually the key to diagnosis

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DIFFERENTIALS

Other causes for hypertrophy
- Systemic hypertension
- Hyperthyroidism

- Often cannot definitively distinguish HCM from these other possibilities by echo (LVH looks similar for all), but gallop sounds, and clinical signs of CHF are very rare with these other causes of LVH and strongly suggest HCM
- **Severe** LVH is likely to be HCM
- BNP assay?
- Genetic testing ?

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Available tools
- Blood work
- proBNP
- Electrocardiography
- Blood pressure
- Radiography
- Echocardiography

- Variable availability
- Non-uniform quality

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NT-PROBNP ASSAY
Cardiac vs non-cardiac respiratory distress

201 cats: 99 normal, 9 equivocal HCM, 15 mild HCM, 17 moderate HCM, 61 severe HCM	No difference in NTproBNP between equivocal and healthy cats.[13] Severe HCM had significantly higher NTproBNP than other groups. Cut-off for mild HCM detection 100 pmol/L, AUC 0.96	Wess[13]
227 cats: 114 normal, 87 HCM, 22 UCM, 3 UCM, 1 DCM	NTproBNP effectively discriminated between normal cats and those with occult cardiomyopathy.[14] Cut-off 99 pmol/L, AUC 0.92. Correlation of NTproBNP with LV wall thickness and LA size.	Fox[14]
146 cats: 43 normal, 16 equivocal, 50 mild heart disease, 37 moderate/severe	NTproBNP SNAP test can be used to help exclude moderate to severe occult cardiomyopathy; negative predictive value 94%[19]	Machen[19]

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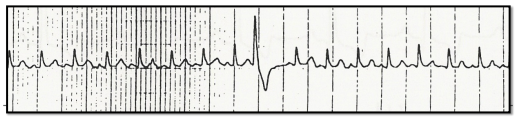
DIAGNOSTICS - ECG

- Limited value as a screening tool
  - Arrhythmias are not common
  - Detected on physical examination
- Useful when arrhythmia ausculted
- Many non-cardiac causes for arrhythmias
- More useful when combined with imaging



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DIAGNOSTICS - RADIOGRAPHS

- Good screening tool
  - Readily available, cost effective, noninvasive
- Best way to diagnose CHF
- Abnormal radiograph suggestive of disease
  - Prompt additional diagnostics - Echocardiogram
- Limitation with feline cardiomyopathy
  - Lacks ability to assess function
  - Cannot measure wall thickness
  - Cannot discriminate between types of CM

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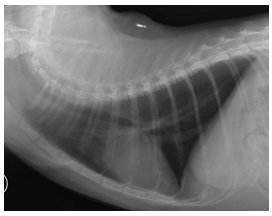
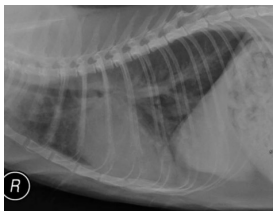

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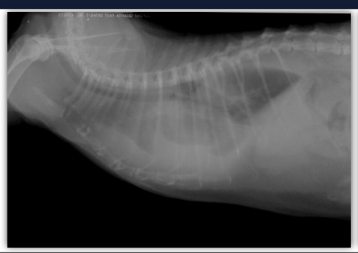
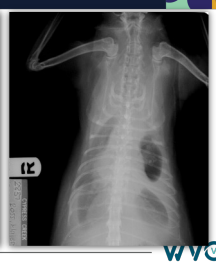

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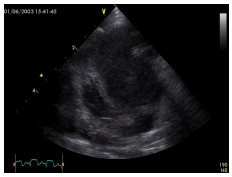
- Definitive diagnostic test
  - Essential for cardiomyopathy
- Cardiac function and chamber dimensions
- Gold standard
  - Establish diagnosis/estimate severity
- Drawbacks
  - Limited availability
  - Cost considerations
  - ***Tremendous*** user dependence



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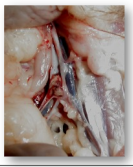
SEQUELAE: ARTERIAL THROMBOEMBOLISM

- Uncommon, but devastating
- Aortic bifurcation (usually)
  - Acute rear limb paralysis
  - Right forelimb
- Intensely painful



May develop clots in other organs
Brain
Kidney
GI tract

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DMVD

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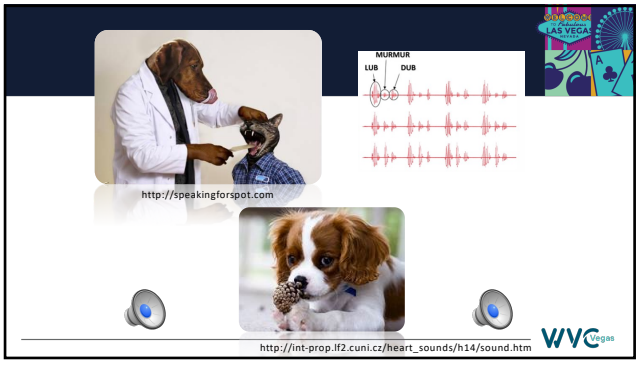

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Synonymous with: Degenerative mitral valve disease, Myxomatous valvular degeneration, Atrioventricular valvular insufficiency (AVVI)

Mitral valve
- Most common valve affected
- Mitral regurgitation → volume load

Tricuspid valve
- Rarely alone
- Often in conjunction with mitral valve
- Tricuspid regurgitation → volume load

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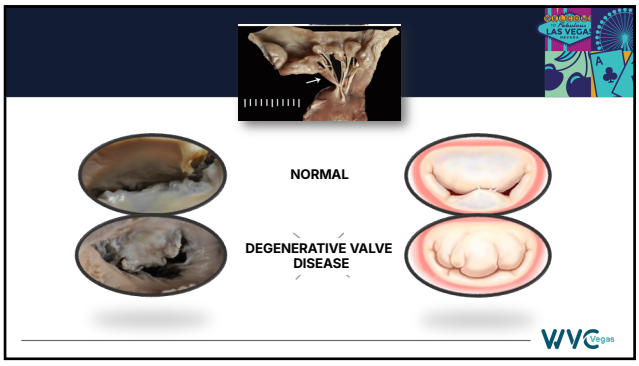

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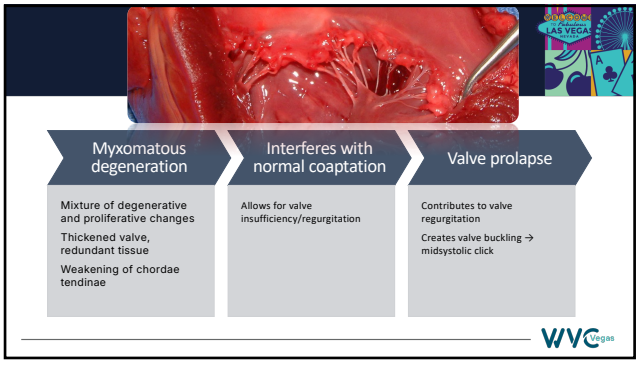

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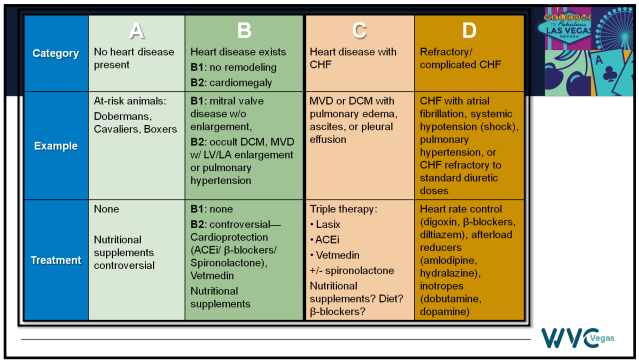

Category	A	B	C	D
	No heart disease present	Heart disease exists **B1**: no remodeling **B2**: cardiomegaly	Heart disease with CHF	Refractory/ complicated CHF
Example	At-risk animals: Dobermans, Cavaliers, Boxers	**B1**: mitral valve disease w/o enlargement, **B2**: occult DCM, MVD w/ LV/LA enlargement or pulmonary hypertension	MVD or DCM with pulmonary edema, ascites, or pleural effusion	CHF with atrial fibrillation, systemic hypotension (shock), pulmonary hypertension, or CHF refractory to standard diuretic doses
Treatment	None Nutritional supplements controversial	**B1**: none **B2**: controversial—Cardioprotection (ACEi/ β-blockers/ Spironolactone), Vetmedin Nutritional supplements	Triple therapy: • Lasix • ACEi • Vetmedin +/- spironolactone Nutritional supplements? Diet? β-blockers?	Heart rate control (digoxin, β-blockers, diltiazem), afterload reducers (amlodipine, hydralazine), inotropes (dobutamine, dopamine)

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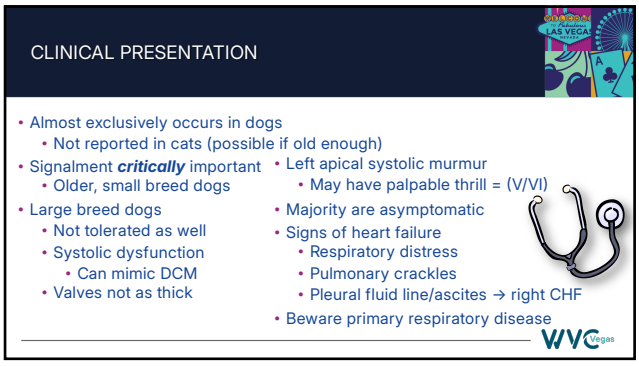

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Mitral valve disease in dogs (SMALL BREEDS)

Annular dilation occurs secondary to mitral regurgitation and causes lack of coaptation

Anterior leaflet prolapse is seen more commonly (48 % anterior, 7% posterior and 44% bileaflet) [Terzo et al, 2009]

16 % ruptured chordae in DMVD (most commonly seen with severe) [Serres et al. 2007]

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Mitral valve disease in dogs (LARGE BREEDS)

PRIMARY VS. SECONDARY?

Mitral valve disease features

- absent or low intensity murmur,
- less pronounced morphologic changes of the valve,
- more pronounced and earlier onset systolic dysfunction,
- frequent rhythm abnormalities (atrial fibrillation)

[Haggstrom and Borgarelli et al. 2004]

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CLINICAL PRESENTATION

Initial stage
- Small volume
- Not hemodynamically significant

Compensatory phase
- Decreased stroke volume
- Compensate with RAAS/SNS
- Cardiac enlargement, increased contractility

Decompensation
- Regurgitation becomes excessive
- Fluid retention to maintain CO
- Volume overload → CHF

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COMPENSATED

- Murmur generally loud
  - May be heard on right
- Coughing
  - Not in heart failure
- Radiographic changes
  - Left atrial and left ventricular enlargement
  - Evidence of airway compression
  - **No** pulmonary edema
- Treatment dilemma

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DECOMPENSATED

- Congestive heart failure
- Respiratory signs
  - Coughing or respiratory difficulty
  - May present in acute distress
- Pulmonary crackles
- Unequivocally require therapy
  - Acute treatment
  - Chronic therapy

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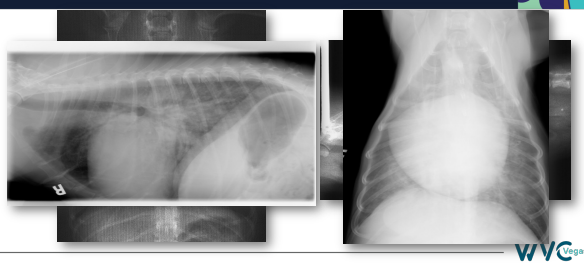

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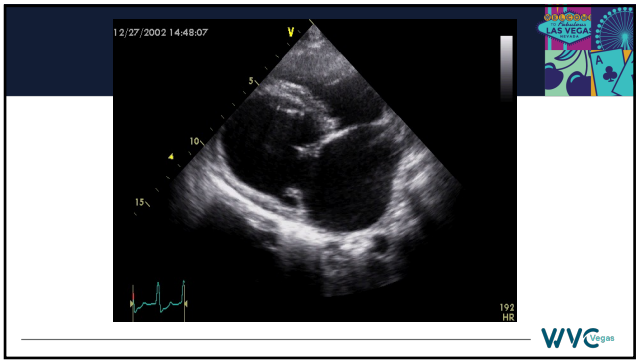

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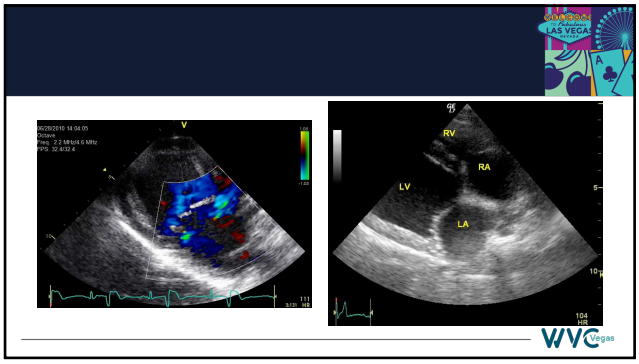

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SEQUELAE

Left atrial split
- Hemorrhagic pericardial effusion
- Acute presentation → collapse

Pulmonary hypertension
- Not uncommon
- Can cause respiratory distress and syncope

Chordae tendinae rupture
- Acute respiratory distress → fulminant CHF
- Rare

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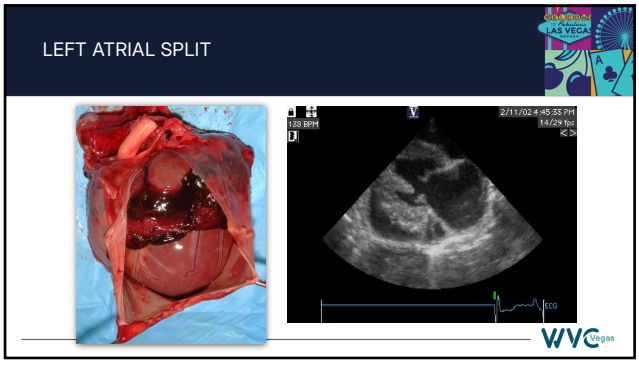
LEFT ATRIAL SPLIT
WVC Vegas

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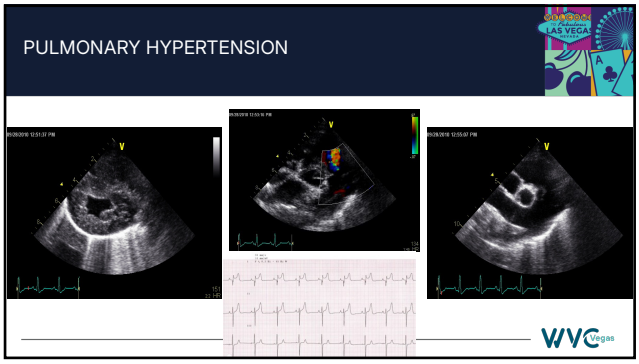
PULMONARY HYPERTENSION
WVC Vegas

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THANK YOU

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