



Large Animal Cardiology

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History and Exam

History and Observation

- Information that may indicate a cardiovascular problem
 - Reduced performance: more often will be attributed to musculoskeletal or respiratory issue
 - Cough
 - o Dyspnea
 - Stunted growth
 - Lethargy
 - Collapse
- Observe from a distance prior to physical exam. Note the following -
 - Animal's disposition
 - Alert, dull, nervous, or quiet
 - Dyspnea present?
 - Any peripheral edema?
 - Jugular pulsation or distension?
 - Some degree of pulsation = normal in large animals
 - If animal lowers head to graze → head is lower than right atrium → distension of entire jugular occurs
 - This is NOT pathologic
 - To determine pathologic distension: observe jugular vein when animal's head is in normal position
 - Pathologic = jugular vein pulsating beyond lower 1/3 of the neck
 → jugular distension. Consider pericardial or cardiac disease

Cardiovascular Exam

- Mucous membrane color and capillary refill time (CRT)
 - Normal = moist, pink, CRT < 2 seconds
- Peripheral pulse
 - Facial artery: rostroventral border of the mandible
 - Transverse facial artery: caudal to the lateral canthus of the eye, ventral to the zygomatic arch
 - Brachial artery: high on the medial aspect of the forelimb (also used for pulse detection)
 - Cardiac apex beat palpate thorax wall with flat of hand. Useful in detecting thrills
 - Thrill: a palpable vibration caused by turbulent blood flow. Present in murmurs graded V-VI/VI
- Auscultation of all regions of heart on left and right side of thorax





Cardiac Auscultation

- Entire precordium should be auscultated during exam
- Point of maximum intensity (PMI) of valves
 - Mitral valve: left 5th ICS, dorsal to olecranon (*cardiac apex*)
 - Aortic valve: left 3rd ICS, below point of shoulder and medial to triceps muscle mass (*heart base*)
 - o Pulmonic valve: slightly cranioventral to aortic valve at left heart base
 - Tricuspid valve: right 3rd 4th ICS medial to triceps just above olecranon



Valve locations for auscultation (P = pulmonic, A = aortic, M = Mitral, T = Tricuspid)

- Heart rate and rhythm assessment
 - Horse: 44 BPM (23-48 range)
 - Cow: 30 BPM (60-80 range)
 - Sheep: 75 BPM (60-120 range)
 - Goat: 90 BPM (70-120 range)
 - Swine: 68 BPM (58-86 range)
- It is normal to hear 2, 3, or 4 heart sounds in horse & cow!
 - S1 = closure of mitral and tricuspid valve
 - Systole occurs between S1 & S2
 - S2 = closure of aortic and pulmonic valve
 - S3 = vibrations resulting from termination of rapid ventricular filling in early diastole
 - S4 = late diastole, during atrial contraction

Diagnostic Procedures

Electrocardiography (ECG)

- Use in large animals is limited to determination of heart rate and rhythm





- CANNOT get accurate information about chamber enlargement or mean electrical axis because of the "category B" distribution pattern of Purkinje fibers in ventricles of horse/cow – depolarization happens in single "burst" of activation, so multiple lead tracings are not accurate
- Can use single lead system (base-apex lead)
 - Right forelimb (white) electrode \rightarrow along jugular groove on right side of neck
 - Ground (green) electrode \rightarrow right base of neck
 - Left limb (black and red) electrodes → over left precordium behind olecranon (left apex)

Echocardiography

- Non-invasive diagnostic technique allowing for real-time evaluation of blood flow and movement of walls/valves of heart
- Can measure wall thickness and luminal dimensions
- Best non-invasive diagnostic test for congenital and acquired heart disease
- When is an echo indicated?
 - New/loud murmur
 - Arrhythmia detected
 - To rule out CHF
 - Impaired athletic performance after musculoskeletal and respiratory disease are excluded
 - Fever of unknown origin

Radiography

- Most helpful in foals: lateral AND dorsoventral views possible
- Angiography to identify congenital lesions
- Provides limited information from large adult horses
 - o Can only obtain lateral views
 - o Cardiac enlargement must be significant to detect
 - o +/- information about pulmonary infiltrates to aid in diagnosis of CHF

Bloodwork

- CBC: helpful to look for signs of infectious disease
 - o Endocarditis
 - Bacterial pericarditis
- Serum chemistry: electrolytes, liver and kidney values, etc.
- Arterial blood gas: information about oxygenation and intracardiac shunts
- Serum cardiac Troponin I (cTnI): evaluate myocardial damage

Blood Pressure

- Dinamap indirect BP measurement on coccygeal artery (OR metatarsal in foals)
- Direct BP measurement = gold standard
 - o Requires arterial catheterization





Useful for monitoring patient on cardioactive drug (i.e. Hydralazine – arterial vasodilator)

Cardiac Catheterization

- Not common in large animals
- Useful for critical care patients or diagnostic challenges
- Can demonstrate...
 - Cardiac output
 - Pulmonary hypertension
 - Pressure profiles
 - Oxygen contents of different cardiac chambers
- Pacemakers sometimes needed in donkeys & horses with symptomatic high grade AV block

Cardiac Murmurs

- Clinical causes include
 - Valvular disease
 - Stenosis
 - Insufficiencies
 - Intra- or extra-cardiac shunts
 - Septal defects
 - PDA
 - Sinus of Valsalva rupture
 - Conditions \rightarrow increased cardiac output
 - \circ Conditions \rightarrow increased velocity of blood flow
 - Anemia
 - Fever
 - High adrenergic tone
- Diastole = longest phase of cardiac cycle, therefore, longer murmurs are more likely to be diastolic
 - Diastolic murmurs = MUCH more common in horses than small animals
 - Aortic insufficiency = common finding in older horses

Murmur Description

Grade I: Soft murmur, heard while listening in a quiet room/stall after listening for a prolonged period of time

Grade II: Soft murmur that can be heard almost immediately

Grade III: Low - moderate intensity murmur

Grade IV: Moderately intense murmur without a palpable thrill

Grade V: Loud murmur with the presence of a palpable precordial thrill





Grade VI: Loud murmur, palpable thrill. The murmur is audible with the stethoscope removed from the thorax



Congenital Murmurs

- Clinical signs of congenital defects = dependent upon severity of hemodynamic derangement
 - $\circ~$ Asymptomatic, reduced exercise tolerance \rightarrow cyanosis and death
 - VSD = most common defect seen in foal & calf
- Valvular defects = next in frequency
 - 1. Ventricular Septal Defect (VSD)
 - a. Most common cardiac defect in horse/cattle
 - Harsh, holosystolic murmur overriding S2; PMI = right side, +/- additional murmur over pulmonic region (= relative pulmonic stenosis)
 - c. Cardiac cath elevated RV pressures and PO2
 - d. Echo may visualize defect; agitated saline injection may show bubbles in BOTH R & L ventricles (bubble study)
 - 2. Patent Ductus Arteriosus (PDA)
 - a. Continuous, machinery murmur; PMI = L base of heart, murmur radiates to manubrium of sternum & R cardiac base
 - b. Uncommon to auscult continuous murmur in foal (systolic murmur = more common). If a murmur does NOT disappear by day 4 of life = pathologic/abnormal murmur
 - 3. Tetralogy of Fallot
 - a. Systolic murmur transmitted widely over R & L thoracic wall
 - b. Radiographs RVE
 - c. Cardiac cath elevated RV pressure, lower pulmonary arterial pressure
 - d. Stunting of growth, cyanosis, dyspnea
 - 4. Misc. defects





- a. Persistent truncus arteriosus
- b. Atrial septal defects
- c. Atrioventricular septal defects
- d. Valvular dysplasia or stenosis
- e. Other complex congenital lesions

Acquired Murmurs (valvular disease)

- Most valvular lesions do not halt athletic horses, however, it's possible performance may decrease over time
 - \circ Exception: ruptured chordae tendinae \rightarrow profound effect on performance
- Survival prognosis = good
- 1. Functional (physiologic) murmur
 - a. Benign, typically high frequency, low intensity, grade I-II/VI at rest (intensity can increase > III/VI if horse is excited or w/ colic)
 - b. Usually crescendo-decrescendo; PMI = pulmonic/aortic valve (heart base)
 - c. Common in young/athletic horses
 - d. Functional murmurs disappear at rest
- 2. Aortic Stenosis (AS)
 - a. Crescendo-decrescendo (ejection quality) systolic murmur; PMI = Ao valve region
 - b. Most outflow tract murmur in horse = Ao valve > pulmonic valve
 - c. MOST likely of clinical significance if...
 - i. Grade III/VI or louder
 - ii. Heard on both sides of chest
 - d. Acquired AS is more common than congenital (subaortic) AS in horses
- 3. Mitral insufficiency/regurgitation (MR)
 - a. Holosystolic, plateau murmur; PMI = mitral area, may radiate towards aortic valve
 - b. Second most common acquired valvular disease in horses, cattle, pigs
 - c. Severe MR can \rightarrow LCHF w/ coughing, exercise intolerance, dyspnea
 - d. Mild MR may not be associated w/ clinical signs
 - e. Acutely ruptured chordae tendinae \rightarrow CV collapse w/ fulminant pulmonary edema
- 4. Ruptured chordae tendinae
 - a. Results in marked prolapse of MV leaflets w/ severe regurgitation
 - b. Widely radiating pansystolic murmur
 - c. Increased LA pressure \rightarrow more pronounced/prolonged S3
 - d. +/- Acute dyspnea, frothy nasal discharge due to pulmonary edema
- 5. Tricuspid Insufficiency
 - a. Harsh, holosystolic plateau murmur; PMI = right AV valve
 - b. <u>Most common acquired murmur in cattle</u>, pigs, sheep (secondary to endocarditis)





- c. TR occasionally produces murmur in horse (<u>common</u> in standardbred racehorses)
- d. +/- Prominent jugular pulsations
- e. May be associated w/ high altitude disease in cattle or other causes of PHT
- 6. Aortic Insufficiency
 - a. Long diastolic decrescendo murmur; PMI = left hemothorax. Musical sounding
 - b. The most common acquired valvular defect in aged horses
 - c. Severe AI may \rightarrow bounding, "waterhammer" arterial pulses due to <u>diastolic</u> runoff
 - d. Echo: guarded poor prognosis if...
 - i. Fractional shortening < 30%
 - ii. LA > 16 cm diameter
 - iii. Pulmonary artery > aorta
- 7. Pulmonic insufficiency and stenosis: rare in large animals

Cardiac Arrhythmias

NORMAL Equine ECG

- Heart rate = 30 45 BPM at rest
- P Wave: bifid in lead II (can be single/polyphasic, too). Maximum amplitude ~ 0.5 mV.
 Duration 0.08 0.2 seconds
- PR Interval: duration 0.22 0.56 seconds
- QRS Complex: lower amplitude and more variation than in the dog. Not enough definitive diagnostic criteria to diagnose ventricular enlargement patterns
- T Wave: usually positive (leads III & aVF) or biphasic (lead II, aVR, aVF). Changes in T waves in normal horses with exercise or excitement
- T wave changes & ST segment changes: hypoxia, shock, septicemia, toxemia Normal VARIATIONS in Equine ECG
 - Sinus Arrhythmia and Wandering Pacemaker: occur in <30% normal horses at rest. Associated with high vagal tone, disappear with exercise or atropine. Not associated with respiration (like in dog)
 - Low grade AV block: 1st degree AV block (PR interval > 0.40 sec) & Mobitz Type I (Wenckebach) 2nd degree AV block occurs in <20% of normal resting horses
 - Are considered normal and due to high vagal tone if there are NO associated clinical signs and the rhythm returns to sinus rhythm <u>with exercise</u>, <u>excitement</u>, <u>or atropine</u>
 - High-grade AV block or AV block that persists with exercise = pathologic.
 Dropping of every other beat/ 2+ beats in succession is NOT normal
 - Sinus arrest and Sinoatrial Block: occasional pauses or breaks in normal sinus rhythm (NSR). Pauses usually disappear with exercise or atropine and do NOT require therapy
 - Prolonged sinus arrest can → syncope, sometimes necessitating pacemaker placement





ABNORMAL Rhythms

Atrial Fibrillation: common in horses, develops in two different populations of horses: (1) young horses of racing age without evidence of other cardiovascular disease, and (2) older horses with loud murmurs and significant cardiac pathology found upon necropsy



- Idiopathic atrial fibrillation: young horses, racing age. Present with sudden (an unexplained) loss of stamina
 - Slow and irregular heart rate may be confused with 2nd degree AV block or sinus arrest on auscultation
 - Absent soft murmur, usually without signs of heart failure early on
 Good prognosis for return to NSR with treatment
- Atrial fibrillation with concurrent heart disease: older horses, usually with loud
- murmurs of MR or TR
 - Often evidence of heart failure
 - Heart rate tends to be fast (70 100 BPM)
 - May present for exercise intolerance, dyspnea, edema, weakness, or collapse
- Diagnosis: based on physical exam, detection of an irregular heartbeat & pulse.
 Confirmed by ECG. Evaluate for presence of underlying heart disease via echo
- o Treatment
 - When heart disease is <u>absent mild</u>: conversion of atrial fibrillation to NSR via <u>Quinidine</u>
 - Decreases automaticity, slows conduction, prolongs effective refractory period of atrial cells
 - Negative inotrope with anti-vagal effects (like atropine)
 - Well absorbed via PO administration through a nasogastric tube
 - New techniques of transvenous cardioversion of atrial fibrillation in horses are beginning to replace oral cardioversion with Quinidine
 - Possible SE: tachycardia, depression, urticaria, edema of nasal mucosa, anorexia, colic, diarrhea, laminitis, seizures, ventricular arrhythmias
 - ONLY give Quinidine if animal has no systemic illness or CHF present





- When heart disease is <u>present</u>: initiate treatment to relieve signs of CHF and control the ventricular response rate to the fibrillating atria
 - Administer Furosemide IV or IM (0.5 1.0 mg/kg) to control edema
 - Maintenance Digoxin BID
- Prognosis: fair good when onset is recent and loud murmurs/signs of CHF are absent
 - 75 85% of horses with atrial fibrillation will convert to NSR
 - 20 30% of converted cases may revert back to atrial fibrillation
 - Prognosis is dependent upon underlying heart disease
 - The longer a horse has been in atrial fibrillation, the greater chance of recurrence (even after cardioversion)
- Atrial and ventricular arrhythmias
 - If consistent or frequent signs of heart disease (i.e. myocarditis). Can also be associated with...
 - Septicemia
 - Toxemia
 - Acute GI disease
 - Viral or bacterial diseases specific to horses
 - Disease associations in horses
 - <u>Atrial tachycardia</u> develops with <u>myocardial</u> disease
 - <u>Ventricular tachycardia</u> develops with septicemia, toxemia, GI disturbances (<u>non-cardiac causes</u>)
- Atrial Tachycardias
 - Usually managed by digitalization supplemented with Quinidine in refractory cases
 - Lidocaine: causes convulsions in horses so ONLY used at very low doses (0.25-0.5 mg/kg slow IV) if necessary, for <u>ventricular tachycardia</u> (VT)
 - Ensure serum potassium levels are normal prior to administration







Ventricular tachycardia to third degree AVB in a donkey

- Heart block in Horses
 - Advanced (high grade) 2nd or 3rd degree block = occasional cause of syncope in horses
 - High grade AV block is more common in donkeys/mules (syncope is often mistaken for seizures)



Third degree AV block in a donkey, lead II

Congenital Heart Defects - EQUINE

Ventricular Septal Defect

- Varies in size, often located in the upper, membranous septum
- Is NOT typically accompanied by another congenital defect (different than bovine VSD)
- Clinical signs are dependent on the severity of the defect
 - o Stunted growth
 - o Exercise intolerance
 - Heart failure
 - +/- Normal longevity (small defects)
- Physical findings
 - o Harsh, holosystolic murmur. Usually accompanied by a thrill
 - PMI = Right 2-4 ICS, radiates
 - +/- Signs of LV failure
 - A high, large VSD can undermine the support of the aortic valve \rightarrow Aortic Regurgitation \rightarrow <u>diastolic murmur in addition to the systolic murmur of the VSD</u>
- ECG: normal or suggestive of LVE
- Radiographs
 - Variable, often WNL
 - +/- Generalized cardiac enlargement
 - +/- Enlarged MPA
 - +/- Vascular pattern from pulmonary overcirculation associated with left-to-right shunting
- Cardiac Catheterization
 - Significant shunting
 - Oxygen saturation of the pulmonary artery blood >>> oxygen saturation of the RA blood
 - Severe cases: RV and pulmonary artery pressures elevated (reactive PHT)

Tetralogy of Fallot

- Clinical presentation





- Stunting of growth
- o Cyanosis
- o Dyspnea
- Exercise intolerance
- Physical findings
 - Systolic murmur radiating widely over cardiac area
- ECG: RVE
- Radiographs: RVE
- Cardiac catheterization
 - Elevated RV pressure
 - Normal low pulmonary artery pressure
 - Low aortic blood oxygenation

Patent Ductus Arteriosus

- Foals: grade II-III/VI continuous murmur, PMI = 3rd 4th left ICS at level of point of shoulder
 - Heard until 4 5 days post birth

Other defects

- Complex cardiac defects reported in Arabian foals
- Atrial septal defect
- Endocardial cushion defects

Congenital Heart Defects – BOVINE

Ventricular Septal Defect

- Often coexists with another heart defect (i.e. truncus arteriosus) unlike horses
- Two-dimensional and Doppler echo = diagnostic
- Cardiac cath is rarely performed

Other defects

- Ectopia cordis: the heart is located most often in the neck
- PDA: usually diagnosed based on characteristic murmur

Acquired Cardiac Diseases

Aortic Valve Insufficiency in the Horse

- Pathology
 - o Diffusely thickened aortic valve OR nodules/bands affecting valve
 - Nodules = most common, loose fibrous connective tissue, fibroblast abundant
- Clinical Signs
 - Diastolic decrescendo murmur, PMI = L heart base
 - +/- Bounding pulses





- CHF & cardiomegaly in <u>older</u> animals (after peak working years)
- Diagnosis
 - Based on physical exam
 - ECG suggestive of L heart enlargement
 - Echo to document aortic insufficiency
 - Presence of fever/leukocytosis/peripheral embolization → <u>consider bacterial</u> <u>endocarditis</u>
- Treatment
 - o Most affected horses are NOT symptomatic, are old and not being worked
 - No adequate therapy
 - \circ $\;$ If CHF Digitalis and diuretics
- Aortic valve more commonly affected than mitral valve

Mitral Valve Insufficiency in the Horse

- Pathology
 - Localized or diffuse thickening, nodular thickening OR combination of lesions
 - Diffuse fibrous thickening = most common
- Clinical Signs
 - Most affected horses are asymptomatic
 - \circ Middle older age: can be severe enough to \rightarrow exercise intolerance +/- CHF
 - Grade III+/VI plateau-shaped holosystolic murmur, PMI = mitral area, radiating to right side
- Diagnosis
 - Based on physical exam
 - Echo to support: L heart volume overload in absence of AI
 - o Normal Thoroughbreds: small amount of MR/TR present, especially after training
 - Mitral valve prolapse can occur with MR (less severe than degenerative valvular disease)
- Prognosis
 - Good: mitral insufficiency rarely \rightarrow CHF in horse
 - If progression to CHF occurs = poor prognosis
 - Poor oral absorption of diuretics & ACE-inhibitors

Ruptured Chordae Tendinae in the Horse

- Chordae tendinae: anchor valve cusps to papillary muscles
- Rupture \rightarrow grossly incompetent valve
- Large volume of blood regurgitated into LA during systole → decreased CO, increased
 LVEDV & LVEDP → pulmonary congestion & fulminant life-threatening edema = LCHF
- +/- Sudden death
- Causes of ruptured MV chordae
 - o Blunt trauma
 - Severe physical exertion
 - Underlying primary MVD





- Clinical signs
 - Acute dyspnea with white foamy discharge from nostrils
 - Loud pansystolic murmur, PMI = MV, usually with palpable thrill
 - Decreased CO \rightarrow signs of low output heart failure or CV collapse
- Diagnosis
 - o Echo to visualize severe MR and a flail MV leaflet
 - o Radiographs may show pulmonary infiltrates from LCHF
- Prognosis
 - o Grave: most animals will be euthanized for fulminant CHF

Aorto-Cardiac Fistula (Ruptured Sinus of Valsalva) in the Horse

- Acquired cardiac fistula
- Middle-aged, breeding stallions
- Occurs suddenly \rightarrow rapid death or severe distress
- Clinical signs
 - Acute collapse
 - Respiratory distress
 - Severe exercise intolerance
 - Continuous (PDA-like) murmur, PMI = R 4th ICS
- Diagnosis
 - Rupture should be suspected in breeding stallions with <u>characteristic continuous</u> <u>murmur</u> and <u>appropriate clinical signs</u>
 - Echo to confirm diagnosis: visualize fistula from R aortic sinus with continuous left \rightarrow right shunting from LVOT \rightarrow RV or RA
 - o ECG may show unifocal ventricular tachycardia
- Treatment
 - Supportive therapy complete rest
 - o Furosemide
 - Ventricular antiarrhythmics or digoxin
- Prognosis
 - Usually grave: survival time = 24 hr. 4 yr. (if able to stabilize)
 - Sudden death possible

Bacterial Endocarditis in Ruminants

- Degenerative valvular disease = uncommon in ruminants
- In ruminants: bacterial endocarditis appears as vegetative lesions, mostly on the <u>right</u> side of circulation Tricuspid and Pulmonic valves
- Pathogenic Organisms (vary w/ species)
 - Horse: *Streptococcus, Actinobacillus, Pasturella*
 - o Cattle & Goats: Arcanobacterium pyogenes, Streptococci
 - Swine: Erysipelothrix rhysiopathiae, Streptococci
 - Lambs: Enterococci
- Clinical Signs





- Recurrent fever
- o Anorexia
- Weight loss
- Poor milk production
- o Shifting leg lameness
- o Tachycardia
- o Tachypnea
- o History of traumatic reticuloperitonitis or pneumonia
- 1/6 cases admitted with primary complaint of heart disease
- Physical Examination: nonspecific and non-localizing
 - Systolic murmur *may* be present over TV and PV
 - Diastolic murmur of PI may be present
 - Jugular distension & jugular pulses together with distension of mammary veins may be observed \rightarrow development of ventral edema, ascites, cachexia
- Diagnosis
 - History, physical signs (murmur, fever), positive blood cultures
 - Echo visualization of vegetative lesions
 - o Leukocytosis
 - Neutrophilia
 - o Lymphopenia
 - Hyperglobulinemia
 - Anemia due to chronic infection
- Treatment
 - High dose antibiotics 4-6 wk. (penicillin or ampicillin) unless c/s indicate otherwise
- Prognosis
 - Varies with stage of disease
 - Early (no heart failure): fair
 - Heart failure present: guarded poor

Myocardial Disease in the Horse

- Primary myocardial disease = cardiomyopathy, not well documented in horses
- Myocardial failure in the horse = most commonly from ingestion of monensin
- Myocarditis (w/o CHF) = most frequently recognized myocardial disease in the horse
- Myocarditis
 - Inflammatory or degenerative myocardial lesions found in 2-15% necropsied horses
 - Bacterial, viral, or parasitic causes (*Streptococcus equi*, influenza, purpura hemorrhagica, equine infectious anemia, strongyle larvae)
 - Strongyle larvae damage aortic root → microemboli → arteriosclerosis & myocardial necrosis
 - Other causes: endocarditis, thoracic/abdominal abscessation, guttural pouch infections, colic, toxemias





- \circ EHV-1 \rightarrow myocarditis = well documented in aborted equine fetuses
- Clinical signs = often absent
 - Decreased performance
 - Dyspnea on exertion
 - Tachycardia disproportionate to fever
 - Arrhythmias
 - Murmurs
 - Loud gallop sounds
 - Fainting/collapse
 - Sudden death: reported in horses recently recovered from strangles/influenza
 - Development of CHF
- Treatment: directed at primary illness (cause of myocarditis)
 - Stall rest for 4 6 mo.
 - Antiarrhythmic therapy if serious arrhythmia is detected
 - Digitalis & diuretics if CHF present
- Monensin toxicosis
 - Horses = more sensitive to monensin than cattle, develop cardiac signs if ingest treated cattle feed
 - Clinical signs
 - Acutely affected
 - Abdominal pain
 - Diarrhea
 - Acute circulatory failure
 - Death
 - Delayed syndrome: 3 6 mo. post-ingestion
 - Necropsy
 - Myocardial degeneration
 - Pericardial effusion
 - Evidence of heart failure
 - Treatment: supportive, myocardial damage is irreversible
- White snakeroot intoxication
 - \circ Ingestion \rightarrow severe myocardial degeneration, hemorrhages, infarcts
 - Arrhythmias & sudden death = frequent sequelae

Myocardial Disease in Cattle

- Cardiomyopathy is rare in cattle
 - Bovine DCM reported in some breeds
- Myocardial disease in cattle is typically secondary to...
 - Systemic infection
 - Pneumonia
 - Traumatic reticulopericarditis





- Neonatal infection
- Mastitis
- Nutritional deficiency
 - Vitamin E
 - Selenium
- $\circ \quad \text{Neoplastic infiltration}$
 - Lymphosarcoma
- Ingestion of toxins
 - Monensin
 - Gossypol
 - White snakeroot
 - Cassia occidentalis
- Clinical syndromes will vary (sudden death chronic CHF)

Traumatic Pericarditis in Cattle

- Most common cause of CHF in cattle
- Perforation or migration of a foreign body (i.e. wire) into the pericardial sac. Disease course varies from few d. few wk./mo.
- Pathology
 - Significantly thickened pericardial sac
 - Adhesions between pericardium & diaphragm and pericardium & epicardium
 - +/- Visualize foreign body
 - Variable amounts of pus, fibrin, gas
- Clinical signs: signs reflect presence of infection due to reticuloperitonitis with evidence of cardiac tamponade
 - Depression, anorexia, weight loss
 - Diarrhea, constipation, rumen atony
 - Decreased milk production
 - Arched back, abducted elbows, pain
 - o Fever, tachycardia, tachypnea
 - o Venous distension of the jugular and mammary veins
 - Ventral and cervical edema, ascites later in disease course
- Physical findings
 - o Early in disease: pericardial friction rub may be present
 - Later in disease
 - Pericardial effusion develops → muffled heart sounds
 - Gas and fluid accumulate → splashing, tinkling, gurgling sounds synchronous with heartbeat
- Diagnosis
 - o Rule outs
 - Heart failure from congenital defects
 - Infective endocarditis
 - Altitude sickness (PHT)





- Lymphosarcoma
- Other myocardial diseases
- To confirm diagnosis
 - Echo
 - Pericardiocentesis at 4th 5th ICS on Left side (idiopathic effusions can occur and will often resolve)
- Treatment
 - Drain pericardial sac when heart failure is present
 - Pericardiocentesis
 - Instillation of antibiotics, debriding enzymes, placement of pericardial drain/surgical exposure and drainage
- Prognosis = poor

Pericardial Disease in the Horse

- An "epidemic" of pericarditis and mare reproductive loss in Spring-Summer of 2001 in Kentucky (previously very rare). *Actinobacillus* sp. were the principal isolates
 - Cause of outbreak: "Septic penetrating setal emboli (SPSE)" from infestation of eastern tent caterpillars
- Clinical signs
 - Fever, anorexia, lethargy
 - Muffled heart sounds +/- pericardial friction rub
 - Signs of RCHF
 - Jugular distension
 - Jugular pulses
 - Ventral edema
 - Ascites
 - Pleural effusion
 - Tachycardia
- Diagnosis
 - Rule out other causes of RCHF
 - Rule out pleural effusion due to pleuritis
 - Echo or successful pericardiocentesis to confirm diagnosis
 - o Can measure RA pressure before and after pericardiocentesis
 - +/- Low voltage QRS complexes or electrical alternans on ECG (not as helpful in diagnosing as in small animals)
- Treatment
 - Signs of systemic congestion: drain and lavage pericardial sac
 - Systemic antibiotics, sometimes even if negative culture
 - $\circ~$ If large number of eosinophils OR if infectious etiology is excluded \rightarrow consider corticosteroid therapy

Neoplasia

- Lymphosarcoma = most common neoplasia of the heart in cattle





- Consider as a differential diagnosis for most cardiac diseases in cattle
- Less frequent in horse if it occurs, may include heart