Anatomy + Physiology of Normal Heart

Atrium = filling chamber
Ventricle = pumping chamber

**Pressures and Volumes**

![Graph showing pressures and volumes](image)

**Atrial Pressure + Jugular Pulse**

- A wave – atria contract and ↑ intraatrial pressure causes blood to flow up the jugular vein
- C wave – right ventricular pressure pushes tricuspid valves up into atria and increase atrial pressure
  - not normally discernable in jugular pulse
- CV interval – atria slowly fill with blood ontop of closed AV valve (atrial pressure slowly rises)
- V wave – blood backflow as it hits the AV valve

**Ventricular + Aortic pressure**

- AV valves close when pressure in the ventricles (LVP) exceeds pressure in atrium (LAP)

**Isovolumetric contraction** = period where ventricle contract but ventricular pressure (LVP) is still less that aortic pressure (AP)

**Volume does not change until LVP pressure>AP pressure**

**Isovolumetric relaxation** = ventricle relaxes until atrial P > ventricular P and AV valve opens (5)
ECG

P wave – atrial depolarisation
PR interval – AV node delay (allows ventricles to fill completely and prevents atrial and ventricular simultaneous contraction)
QRS complex – ventricular depolarisation (beginning of ventricular systole) – so large it masks atrial repolarisation
QT interval – ventricular systole
Flat point between S and T = rapid ejection (passive, elastic process, no depolarisation)
T wave – ventricular repolarisation
- end of wave = end of ventricular systole

**Automaticity (intrinsic heart rate)**

SA node → Interatrial conduction fibers → AV node → L + R bundle of His → Purkinje fibres

**SA Node**
- dominant pacemaker (creates intrinsic heart rate if heart is healthy)
- most excitable

Location – roof of right atrium at junction of the cranial vena cava and the right auricle

Structure - specialised myocytes – resting potential far less negative than other myocytes to aide depolarisation

**AV Node**
Location – interatrial septum just above the right AV valves

Mechanism - Conduction through the AV node slow
- impulse delay allows atrial systole and ventricular filling to complete before ventricular systole (i.e lets atria contract fully before ventricle does)
Depolarisation ↓ Ventricular contraction ↓ Rapid ↑ intraventricular pressure ↓ AV valves close ↓ isovolumic contraction ↓ Rapid ↑ increase in intraventricular pressure ↓ ventricular pressures exceeds diastolic pressures in arteries ↓ Arterial valves open ↓ ejection period (initially v. rapid - 70% of the emptying in first third of the ejection period) ↓ aortic valves close (end of systole)

**Cardiac Cycle**

**Isovolumic** = volume doesn’t change (eg isovolumic contraction of Left ventricle is the ventricle contracting before the pressure is enough to open the aortic valve – takes a moment)

**Systole** = isovolumic contraction + ejection period  
Electrical systole - precedes mechanical systole (impulse happens before contraction)  
- onset of QRS complex = onset of electrical systole on ECG

**Systole on ECG** = QRS complex → immediately after T wave

**Diastole**

Diastole = isovolumic relaxation + rapid ventricular filling + slow ventricular filling + atrial contraction

Sudden ventricular relaxation ↓  
Rapid ↓ ventricular pressure ↓  
Blood large arteries pushed back ↓  
aortic and pulmonary valves close ↓  
Isovolumic relaxation (ventricles relax w/out altered volume) ↓  
Ventricular pressure falls below atrial pressure ↓  
AV valves open ↓  
Rapid ventricular filling – lasts 1/3 of diastole (during systole blood accumulates in atria from veins and pressure is slightly higher) ↓  
slow ventricular filling – lasts 1/3 of diastole ↓  
Atrial systole – lasts 1/3 of ventricular diastole (25% of ventricular filling

Atrial Systole = after P wave on ECG

**Purkinje Fibres**  
Impulse direction – Apex → Base

Location (spp differences greatly affect ECG waves)

- dog, cat, and man - fibres end in subendocardium  
(tissues just deep to endocardium)  
- ruminants, pigs & horses - fibres end in subepicardial surface  
(tissues just below epicardium)
Heart rate – affected by Nervous supply

Autonomic system effects - ↓↑ HR (chronotropic effects)
- ↓↑ FOC (inotropic effects)

- PNS – vagus n.
  - mainly to SA node and AV node
    Effects - ↓ HR - ↓ rate of SA node depolarisation (↓ intrinsic HR)
    - ↓ rate of transmission through AV node
    - ↓ FOC (only slight as fibres in atria not ventricles)
    Influenced by – vasomotor center – aortic and carotid baroreceptors direct PNS impulse down aortic branches of vagus

Sinus arrhythmia – HR changes reflect respiration
  Mechanism – inspiration ⇒ ↓ vagal tone ⇒ ↑ HR
  - expiration ⇒ ↑ vagal tone ⇒ ↓ HR

- SNS - all parts of the heart, esp. to ventricular myocardium
  Effects - ↑ rate of SA node depolarisation (↑ intrinsic HR)
  - ↑ rate of transmission through AV node
  - ↑ FOC (atria and ventricles)
  - ↑ rate of conduction and excitability in all myocardium
  - ↑ contractility
  Influenced by - α and β adrenoreceptions
  - β1 dominates myocardium - positive inotrope (↑ FOC)
  - β2 dominates in SA node – positive chronotrope (↑ HR)

Stroke volume – blood volume ejected by the ventricle during a contraction

Depends on - pre-load on the ventricles
  - afterload on the ventricles
  - contractility

Preload = tension on the ventricular wall at end diastole (ventricular stretch)
  - Frank-Starling mechanism = ↑ myocardial stretch during filling ⇒ ↑ FOC ⇒ ↑ SV
  Mechanism – optimal muscle stretch causes actin and myosin filaments to have a degree of
  - interdigation that maximises FOC
  **Determines end diastolic volume

Afterload = tension needed to develop in ventricle in order to eject blood
(force needed to open up arterial valves)
  - affected by arterial resistance (arterial diastolic pressure) and ventricular diastolic pressure
  - (force needed for ventricular P>arterial P)
  **determines end systolic volume

Contractility = ability to contract (inherent to myocardial cell)
  - Affected by - extracellular and intracellular calcium concentration
    - SNS
    - inotrope drugs (↑ contractility)
  SNS - mechanism – increase cyclic AMP
    - effects - ↑ calcium influx
    - ↑ calcium release from sarcoplasmic reticulum
    - ↑ troponin-C sensitivity to calcium
    - ↑ rate of interaction between actin and myosin
Myocyte contraction mechanism:

Depolarisation
↓
Calcium influx (moves into myocyte)
↓
interacts with the sarcoplasmic reticulum (intracellular calcium store)
↓
calcium release from sarcoplasmic reticulum
↓
calcium binds to troponin-C (regulates contraction)
↓
actin and myosin chains bind
↓
Myosin shape altered and myofilament shortens
↓
Contraction!!

**Cardiac output** = volume of blood pumped each minute (SV x HR)
- HR has more effect than SV
  - ↑ HR → ↓ duration of cardiac cycle → ventricles fill and empty more rapidly (compensates to maintain SV)
  - if HR > critical level, contractility decreases and diastolic filling insufficient (↓ SV)

**Mean Arterial Blood Pressure (BP)**
  Affected by – Peripheral vascular resistance
  - CO

**Pulse Pressure** = systolic pressure – diastolic pressure

Quality – Strength - ↑ difference btw systolic P and diastolic P = ↑ amplitude (height) = ↑ strength
- equality – high equality = amplitude of each pulse equal
- filling – ability to feel the vessel between each pulse
- shape – peak of wave steep/normal/broad

Regularity – time between each pulse should be constant

Pulse Deficits – pulse should be felt after every apex beat
  Pulse deficit = not enough time for ventricular filling → can feel apex beat and auscultate S1 but no pulse felt

Frequency – beats per minute

Symmetry – same pulse strength on left and right
- asymmetric = vessel occlusion (eg thromboemboli)
Cardiorespiratory Disease

**Syncope** = brief loss of consciousness due to insufficient O2 or glucose delivery to brain
- confused with episodes of weakness or seizures
C/s – normal animal → suddenly falls over → rapid recovery
Cause of episode – true syncope – exertion/excitement
- cough syncope - pulmonary diseases (eg. collapsing trachea or bronchitis)

Non-cardiac causes – hypoglycaemia
- cough syncope – blood flow to brain temporarily obstructed
- ↓ BP eg vasovagal syncope
- neurologic abnormalities
- endocrine derangement eg ↓ glucagon or ↑ insulin - insulinoma

Cardiac causes – severe bradycardia – AV node block
- severe sustained tachycardia
- obstructed inflow/outflow of the heart
  - subaortic stenosis
  - pulmonic stenosis
  - severe pulmonary hypertension
  - intracardiac thrombosis/neoplasia
  - heartworm

Vasovagal syncope – stimulation of the vagus nerve results in increased parasympathetic tone and decreased in heart rate and BP → decreased blood supply to the brain → syncope
  • eg enlarged LV due to SAS

**Ascites**

Causes
- Decompensated R CHF fluid = modified transudate
- Hypoalbuminaemia fluid = pure transudate
- Ruptured vessels fluid = blood
- Inflammation fluid = exudates
- Ruptured GB fluid = bile
- Ruptured bladder fluid = urine

C/S – abdominal distension
- positive fluid wave

DDx for abdo distension – weak abdo muscles eg hyperA
- Gas eg GDV
- organ enlargement eg neoplasia
- faeces
- ascites

Diagnosis – abdominocentesis + fluid analysis
- CBC + biochemistry – detect hypoproteinaemia

Fluid Types and analysis
- Pure transudate – severe hypoproteinaemia
  - will see concurrent dependant oedema
  - portal hypertension
    Cause – prehepatic – portal vein
    - hepatic – liver parenchymal damage
  - gross fluid = clear
  - fluid analysis – high bile acids
- Modified transudate – portal hypertension
  Cause - posthepatic – caudal vena cava
Diagnosing Ascites secondary to RCHF

**Fluid analysis** – greater specific gravity than transudate (more cells and protein)
- greater protein content that transudate

**Physical Exam**

*Mucous Membranes*
- Pale = anaemia or peripheral vasoconstriction
- Injected (v. red) = polycythaemia, sepsis, excitement or other causes of vasodilation
- Cyanosis = respiratory disease, R to L shunt, metHb or cold exposure
- Jaundice = haemolysis, hepatic disease, post-hepatic disease (bile problem) or sepsis

**CRT**
- Normal = < 2 secs
- Increased = poor perfusion (eg hypovolaemia, vasoconstriction (↑SNS), dehydration or heart failure)

**Jugular veins**

- Normal - no distension (when head in normal position)
  - pulsations palpable < 1/3 neck

**Distension and pulsation**
- Indicates increased pressure or occlusion of the venous system
- Causes – R CHF (increase in right ventricular diastolic pressure)
  - cranial mediastinal mass
  - jugular vein/cranial vena cava thrombosis

**Often not visible w/out clipping/wetting fur or palpating**

During observation - Place animal on side and lift dorsal legs above head – if distension/pulsation remains then it is due to R CHF

**ECG** (1 of 3 patterns) – determining cause of jugular pulsation/distension
- A, C and V waves = pressure in jugular vein 2° to heart contractions
- A wave (atrial contraction) → C wave (ventricular contraction – AV valve bulges into atrium) → v wave (atrial filling)
- all 3 cause ↑ atrial pressure and therefore increased venous pressure

- exaggerated a-waves
  - decreased right ventricular compliance (stiffer and less elastic)
- cannon a-waves (v. loud a-wave)
  - atrioventricular dissociation (atria & ventricles contract together – AV valves closed)
- prominent v-waves
  - AV valve regurgitation during ventricular systole

**Hepatojugular reflex** - Apply abdo pressure, if jugular distension persists = most likely R CHF

**Precordial impulse palpation**
- Normal: strongest over L apex
Displacement: altered heart size or position eg lung atelectasis, mass, hernia, cardiomegaly
Intensity: fat, weak contractions, pericardial effusion, intrathoracic mass, air
Thrill: Grade V or VI cardiac murmurs

Cardiac Auscultation

- stethoscope diaphragm = high freq sounds (S1 and S2 in dogs, S1, S2, S3 ad S4 in cats)
- stethoscope bell = low freq sounds (S3 and S4 in dogs)

**Left Side**
- P= Pulmonary Valve, A= Aortic Valve, M=Mitral Valve
- P (Base)  
- M (Apex)

**Right Side**
- T= Tricuspid Valve

**Technique**

- auscultate BOTH sides of the chest - L and R apex and base
- sternal and parasternal regions (cat only).

**Heart Sounds**

S1 = AV valves closing (onset of ventricular systole)
   - ECG – at the end of the QRS complex
   - Sounds - Louder, longer and lower than S2 (‘Lub’)

S2 = Aortic and pulmonic valves close (onset of ventricular diastole)
   - ECG - at the end of the T wave
   - Split S2 - due to pathological changes (eg. pulmonary hypertension)
     - normal in large breed dogs – aortic valve closes slightly earlier than pulmonary valve normally. Far more audible in large breed dogs

S3 = rapid passive ventricular filling
   - Sound generated by ventricular wall vibration
   - Causes – dilated congestive heart failure
     - severe hypertension
     - myocardial infarcts
     - Severe mitral regurgitation (may be mistaken for S2)

S4 = atrial contraction
   - Sound generated by stiff/overdistended ventricle that is filled rapidly (blood splashes around)
   - Causes - feline cardiomyopathies
     - dogs with 3rd degree AV block

**Summation gallop:**
   - Cause - HR>180 → rapid ventricular filling & atrial systole occur v. close together
   - can’t decide whether sound is S3, S4 or both

**Altered heart sound intensity:**
- ↑ S1 = ↑ sympathetic tone (↑ FOC) or a thin chest wall (↑ sounds conduction)
- ↑ S2 = pulmonary hypertension
Cardiac Murmurs
Sounds generated by turbulent blood flow (laminar flow becomes turbulent)

Causes - flow velocity increases
- blood viscosity decreases
- heart chambers abruptly increase

Thrills - turbulent flow strong enough to detect vibrations via palpation (precordial)

Murmur classification
Timing: systolic, diastolic, continuous
Location: L, R, base, apex, sternal, parasternal (PMI – point of max intensity)
Intensity: grades 1-6
Pitch (frequency): ejection, regurgitation of blood

Grading:
1. Very soft - heard in quiet room, must listen carefully (often not even sure if murmur there)
2. Soft murmur, easily heard but only on one spot
3. Moderate intensity murmur heard in multiple spots
4. Loud murmur, no precordial thrill
5. Loud murmur with a precordial thrill
6. Heard with stethoscope off the chest wall, precordial thrill present

Cats - low grade, dynamic parasternal murmurs are present in many normal cats
Cause - dynamic right ventricular outflow obstruction (DRVO) idiopathic or occurs in diseases with high output states
NB: Old cats w/ heart murmurs = chk hypertension, hyperthyroidism & chronic renal failure.

Arterial pulses
Hyperkinetic: large difference between systolic and diastolic pressure
Hypokinetic: small pressure difference or slow rise to maximum systolic pressure (eg SAS)
Asymmetrical /absent = thromboembolism

<table>
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<th>Abnormality</th>
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<th>Location</th>
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<tr>
<td>L AV valve</td>
<td>Systolic</td>
<td>L apex</td>
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<td>R AV valve</td>
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<tr>
<td>Aortic Valve</td>
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<td><strong>Stenoses</strong></td>
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<td>Arteriovenous Shunts</td>
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<td>Atrial septal defects</td>
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<td>Ventricular septal defect</td>
<td>Systolic</td>
<td>R apex or base</td>
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<tr>
<td>Patent ductus arteriosus</td>
<td>Continuous</td>
<td>L precordium (cranial to heart)</td>
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**Valvular Disease**

**Endocardium**

**Misc. Lesions** - haemorrhage (eg due to hypoxia)
- subendocardial fibrosis (eg “jet lesions” in atria due to turbulent blood flow)
- subendocardial mineralisation (eg vit D rodenticide toxicity)

**Endocardiosis**

Myxomatous valvular degeneration (MVD) = Canine Chronic Valvular Disease
- non-inflammatory endocardial disease only significant in dogs

Endocardium affected – mitral and tricuspid valve leaflets and chordae tendinae
- mitral valve lesions are most common
- semilunar valves **rarely** affected (aortic and pulmonic valves)
- tricuspid valve usually affected with mitral valve, rarely alone

Gross – thickened, distorted valve leaflets
- free margins of the valves most affected

Risk factors – old dogs (rare in most dogs <5yo)
- males
- small breeds
- less common in large dogs but develop heart failure earlier and more severely

Exception – Cavaliers - mitral insufficiency murmurs in 10% of dogs < 1 yo

Necropsy - Common incidental finding

Role in heart Dz - 75% of heart disease in dogs due to mitral regurgitation from MVD

Aetiology – idiopathic (not understood)

Pathophysiology - proliferation of loose fibroblastic tissue + deposition of acid glycosaminoglycans + collagen degeneration

\[ \text{Increasing valvular insufficiency} \]

\[ \text{Regurgitation and volume overload} \]

Atrial dilation, left ventricular dilation & eccentric ventricular hypertrophy
Left atrial endocardium can become irregularly thickened by fibrosis (jet lesions)

Mild MVD - few small nodules on valve margins
Severe MVD – gross valve distortion by grey nodules
  - valves thickened
  - ruptured chordae tendineae.

**Diagnosing MVD**

**History**
Chronic progression – most commonly over several years
  - Heart murmur initially present, develops to heart failure (6-9 y; less in CKCS)
Acute progression if chordae tendineae rupture
Heart murmur Detection - One of two presentations most common:
  1. Heart murmur incidental finding on physical exam for other condition
  2. Cough – most common presenting complaint
    Cause – LCHF
      - left atrial pressure on main stem bronchus

Sudden death if L atrium tears

**Physical Examination**

**Auscultation**
S1 Intensity - increased in dogs with Mitral Regurgitation as valve opens v. wide
Murmur - PMI over mitral area
Intensity of murmur corresponds to disease severity
Systolic click = high-freq sound btw S1 & S2 due to mitral valve buckling into left atrium
Holosystolic murmur - immediately after S1 and lasts until S2
Pansystolic murmur - immediately after S1 and lasts through S2
  - v. common

NB: tricuspid regurgitation difficult to detect as mitral murmur masks it

**Arrhythmias** – diagnose w/ ECG
  Cause - Atrial Premature Contractions (APC)
  - Ventricular Premature Contractions (VPC)

Respiratory Arrhythmia – not present
  – PNS causes resp arrhythmia, heart failure = SNS dominates

Pulse – normal/tachycardia (↓ ejection time due to rapid left ventricular contraction)

NB: normal lung sounds doesn’t rule out pulmonary oedema

**Radiography** – 3 criteria
  Left atrium – enlarged, can judge disease progression by size
  Lungs - Pulmonary oedema caudodorsally and in hilar early
  Pulmonary vessels dilated

**Echocardiology**
  **Not essential for Dx and Rx in most cases**
  Valves - hyperechoic, distorted, nodular thickening
    – impossible to distinguish from infective endocarditis
Left Atrium – enlarged, size correlates with disease severity.
Left Ventricule - end-diastolic diameter increases with disease severity
- normal size in small dogs until end-stage disease

**Stages of Heart Failure**
A – dog w/ breed disposition for heart failure but no murmur audible
B1 – audible heart murmur, no eccentric hypertrophy. Not yet in heart failure
B2 – audible heart murmur with eccentric hypertrophy. Not yet in heart failure
C – heart failure, managed by medication
D – heart failure refractory to medication
NB: first audible heart murmur ➔ first clinical signs can take 5-6 years

**MVD Treatment**
No therapy slows the progression of mitral valve degeneration

**Asymptomatic dogs w/ murmurs - ACE inhibitors not beneficial!**

Emergency Therapy
- O2
- rest (↓ stress)

CHF
- low salt diet
- pimobendan (vetmedin) – positive inotrope - ↑ FOC
  - vasodilation - ↓ preload (veins) and ↓ afterload (arteries)
- frusemide- ↑ H2O loss ➔ ↓ volume ➔ ↓ preload ➔ ↓ BP
- spironolactone (diuretic) – counters frusemide to avoid hypokalaemia (need K for muscle)
- ACEinhibitor – inhibits RAAS to prevent hypertension

REMEMBER – cough + murmur does NOT mean L CHF, need 3 criteria on xray!

**Infective Endocarditis** (rare)
Cause - bacterial infection
- fungal infection (rare)

Bacterial endocarditis
Lesions - vegetative lesions on valves
↓
Unable to close properly
↓
regurgitation

Acute bacterial endocarditis:
Presentation - acute systemic illness
Organisms – v. virulent bacteria
More common in dogs (esp large breed old dogs)

Chronic bacterial endocarditis:
Presentation - heart failure
Organisms – less virulent
Disease time course – weeks ➔ months
More common in cats

Valves affected - aortic and mitral valves most common

**Pathogenesis**
Bacteraemia
↓
bacteria to colonise a heart valve
↓
endothelial damage
↓
platelets and fibrin attach
↓
Vegetative lesions develop
(large friable masses = clots containing bacteria)
↓
Valvular regurgitation

Sequelae – L CHF
  - Vegetative lesions commonly cause thromboemboli esp in kidney and spleen

Factors enabling bacterial colonisation
  - valve lesions
  - Dogs with SAS (sub-aortic stenosis) – increased ventricular turbulence
    NB: prophylactic A/B’s should always be given if bacteraemia is likely in these dogs

Common pathogens - *Staph, Strep, E. coli*

Killing bacteria in vegetative lesions very difficult as clot protects from phagocytosis and A/B’s

**Common Hx**
- Dogs - large purebred dogs
  - systemic illness - May see discospondylitis, polyarthritis (sterile or septic), embolisation
- Cats - present with CHF and no systemic illness (rare in dogs)

**Physical Examination**

**Cardiac Auscultation**
- Systolic Murmur due to mitral regurgitation or aortic stenosis
  OR
  - Diastolic murmur due to aortic regurgitation

Bounding Pulse w/ aortic regurgitation
  - ↑ systolic pressure + ↓ diastolic pressure
  - forceful pulse that quickly disappears

**Gait**
- lame w/ swollen joints  (concurrent immune mediated polyarthritis common)

**Diagnostic Tools**
- Echocardiology
- Blood culture - poor sensitivity - prior antibiotics?
  - fastidious organisms (nutrient requirements = difficult to culture)
  - poor laboratory technique?
  - lack of continuous shedding of some bacteria
  - poor specificity – many organisms can cause endocarditis
- Common clinical pathology - neutrophilia w/ left shift
  - azotaemia
- glomerulonephritis

**Treatment**
Endocarditis - parenteral A/B’s for 2 weeks then orally for 4-6 weeks
Empirical Therapy - ampicillin and fluoroquinolone

Treat concurrent dz – CHF, renal/splenic infarction etc

Prognosis = Poor (20% survival)
Common Sequelae can be fatal – CHF
- embolisation (esp renal infarcts)
- sepsis

**Heart Disease and Heart Failure**

**Heart Disease** = abnormalities of heart structure or function

Cause – congenital or acquired
Significance – incidental finding or clinically significant

Incidental finding example – L AV regurgitation w/out c/s = heart disease w/out heart failure
Clinically Significant – L AV regurgitation w/ c/s = heart disease w/ heart failure

**Heart Failure** = heart disease exceeds cardiac reserve and compensatory mechanisms are inadequate or detrimental. C/S and pathological indicators are present

Heart is unable to meet metabolic demands of the body

Forward failure → low output  
Backward failure → congestion

**Vicious Heart Failure Cycle**

\[ \downarrow \text{Co} \quad \quad \downarrow \]

**Compensatory mechanisms**
- ↑ SNS
- ↑ RAAS
- ↑ ADH

↓ **Excessive vasoconstriction, Na+ and H2O retention**

↓ ↑ **Preload and Afterload**

**Heart Failure**

**Main Underlying Causes of Heart Failure:**
- Sustained pressure overload
- Sustained volume overload
- Systolic dysfunction (altered contractility of myofibres or loss of myofibres – eg fibrosis)
- Diastolic Dysfunction (↓ ventricular compliance/elasticity)
- Abnormal heart rate or rhythm

**Compensatory Mechanisms**
Aim - increase cardiac output to meet metabolic demands.

Short-term: mechanisms are beneficial (eg massive blood loss)
Long-term: mechanisms result in the signs of heart failure

**Systemic Neurohumoral Compensatory Mechanisms**
inadequate cardiac function → SNS Activation & renin-angiotensin-aldosterone system activation

SNS + RAAS -
- maintain blood volume
- maintain central venous pressure
- maintain venous return to the heart
- maintain cardiac output

SNS + RAAS results - ↑ preload (expanded blood volume)
- ↑ afterload (peripheral vasoconstriction)
- ↑ capillary hydrostatic pressure → oedema and effusions

**Activation of SNS**

![Diagram of SNS activation]

NB: baroreceptors and chemoreceptors located in carotid body and aortic arch. Transmit tonic inhibitory signals to vasomotor centre via vagal nerve. ↓ signals = ↑ sympathetic tone

Heart failure – baroreceptors decrease in responsiveness → sustained ↑ sympathetic tone

**Renin-Angiotensin-Aldosterone System** (crucial to heart failure)

**Activation of RAAS**

![Diagram of RAAS activation]
Peripheral vasoconstriction (↑ afterload) → ADH release → ↓ Na+ reabsorption in DCT → ↓ H2O retention (↑ preload) → ↑ Blood Pressure

Aldosterone release from adrenal cortex → ↓ Na+ reabsorption in DCT → ↑ renal water absorption + ↑ thirst + vasoconstriction (↑ preload and afterload)

**Angiotensin 2**

Other effects - ↑ adrenaline release
- alters baroreceptor responsiveness (overrides normal physiological control)
- influences cardiac and vascular remodelling (eg hypertrophy)
- Alter other factors – eg endothelins, vasodilatory kinins and atrial natriuretic peptide

**Atrial Natriuretic Peptide (ANP)**

Location - granules in atrial cells, released in response to atrial stretch
Role - natriuretic and diuretic
- Antagonises angiotensin II, ADH and aldosterone
  i.e. prevents volume/pressure overload on atria by increasing fluid loss
Heart failure – ANP degraded in circulation by plasma neutral endopeptidase (released due to ↑ SNS)

**Intrinsic cardiac compensatory mechanisms**

2 mechanisms - dilation
- hypertrophy

External work load of the heart = SV x mean ejection pressure

Factors increasing work load of the heart
- volume overload (↑ preload = increased diastolic work load)
  - cause - ↑ blood volume entering a heart chamber during diastole
- pressure overload (↑afterload = increased systolic work load)
  - cause - ↑ resistance to chamber outflow during systole
- pressure-volume overload (↑ preload and ↑ afterload)

**Heart valve abnormalities (stenosis or insufficiency)** = most common causes of volume & pressure overload

Valvular stenosis = ejection murmur
  → pressure overload

Valvular insufficiency = regurgitant murmur
  → volume overload

**Cardiac Dilation** = enlargement of chamber/s w/ thinning of the wall and ↑ in chamber volume

Cause – compensatory response to volume overload

Development - rapid

Gross – globose (rounder)
- enlarged chambers
- thin chamber wall
- thin ventricular papillary muscles

Chronically dilated chambers → diffuse subendocardial fibrosis
Mechanism of chamber dilation
Volume overload $\rightarrow$ ↑ end-diastolic volume $\rightarrow$ myofibres stretching $\rightarrow$ ↑ FOC $\rightarrow$ ↑ SV

Advantages – enables beat-to-beat adjustments to balance the output of both ventricles
- allows increase in cardiac output in response to a volume overload (eg. arteriovenous shunts, AV and semilunar valve insufficiencies).

Disadvantages - myofibre stretching beyond a limit $\rightarrow$ ↓ tension development & ↓ contractility

**Law of Laplace**

$S = (P \times R)/T$

- $S$ = Stress on wall
- $P$ = Pressure in chamber
- $R$ = radius of lumen
- $T$ = 2x wall thickness

Dilation - If a chamber dilates (↑ $R$), wall stress must also increase (via contraction) in order to create the same pressure as a non-dilated heart (enough to open the valves)

Therefore - dilated heart at risk of failing as it must expend LOTS of energy to eject the blood volume

**Cardiac Hypertrophy** = reversible, compensatory increase in myocyte SIZE (not number)

Myofibres – increase in length and diameter and have more sarcomeres (contractile units)

Development - takes time to develop

Cause – compensatory response to chronic pressure overload or volume overload

Gross – Right sided hypertrophy – heart base broader
- Left sided hypertrophy – heart appears longer
- Bilateral hypertrophy – heart appears rounder

Chamber Radius – decreased in concentric hypertrophy (↓ intraventricular pressure needed)
- increased in eccentric hypertrophy (same intraventricular pressure needed)

Role in Heart Disease –
- Hypertrophied myofibres may have ↓ contractility (blood supply doesn’t ↑ during hypertrophy $\rightarrow$ not enough blood)
- Abnormalities in myofibre oxidative phosphorylation, ca++ movements & Ad/NorAd metabolism.

**Law of Laplace**

$S = (P \times R)/T$

- $S$ = Stress on wall
- $P$ = Pressure in chamber
- $R$ = radius of lumen
- $T$ = 2x wall thickness

Hypertrophy – If stress on wall increases (due to ↑ $P$ or $R$) the heart can compensate by increasing wall thickness

i.e. hypertrophy takes pressure off the heart

2 anatomic forms of cardiac hypertrophy:

**Concentric Hypertrophy**
- Increase in myocardial mass w/out increased in end-diastolic volume
- chamber volume (lumen size) may decrease

Cause - pressure overloads (↑ systolic pressure)
- Eg - pulmonic stenosis/subaortic stenosis
- systemic hypertension

Gross L ventricular concentric hypertrophy - ↑ wall thickness
- ↑ papillary muscles thickness
- ↑ trabeculae carnae thickness
Gross R ventricular concentric hypertrophy - ↑ thickness of moderator band (trabecula septomarginalis)

Result – thicker ventricle is harder to dilate (stiffer wall) → ↑ diastolic pressure → ↓ diastolic volume

Eccentric Hypertrophy
- Increase in myocardial mass w/ increase in end-diastolic volume

Cause - volume overload (↑ diastolic pressure)
- Eg - valvular insufficiencies
- septal defects (blood shunting)
- ↑ RAAS activation
Gross – globose heart
- attenuated papillary muscles (weak and thin)
- wall normal thickness (seems thinner and look dilated, actually illusion due to heart size)

Congestive Heart Failure

- Unilateral RCHF - uncommon – usually occurs w/ LCHF
  Cause - cor pulmonale = long term ↑ pulmonary a pressure
  Pathogenesis pulmonary pathology
  ↓ Pulmonary hypertension
  ↓ right ventricular failure

L CHF → pulmonary venous hypertension and oedema

C/S – tachypnoea (↑ blood CO₂ detected by medullary respiratory centre)
- dyspnoea ± orthopnoea (difficultly breathing unless standing w/ elbows abducted)
- coughing (common in dogs not cats)
- Exercise intolerance - Pulmonary congestion or oedema
  - ↓ responsiveness of the heart to SNS
  - ↓ CO
  - impaired vasodilation due to ↑ SNS and RAAS (compensatory changes)
  - ↑ vascular wall Na⁺ conc. → stiff vessels
- cyanosis (severe pulmonary oedema impairs gas exchange)

NB: cats ↓ exercise to compensate for ↓ resp reserve, often not detected until much later

**Must investigate cough to determine 1° respiratory vs 1° cardiac causes

R CHF → systemic venous hypertension
  → jugular distension
fluid in body cavities
→ hepatomegaly and spenomegaly
→ dependent peripheral oedema

Causes of R CHF – pericardial disease – fibrosis/effusion → can’t expand
- right sided endocardial or myocardial disease – dilated cardiomyopathy
  - tricuspid dysplasia
  - ARVC
- pulmonary hypertension – eg due to L CHF

Presenting C/S varies – ruminants and horses = dependent subcutaneous oedema
  – dogs = ascites
  – cats = pleural effusion (also develop pleural effusions in left heart failure)

Diagnosis of RCHF – observe/palpate jugular distension
  - measure central venous pressure (measure around neck) - ↑ BP

Low output (forwards) heart failure
Causes – arrhythmias
  – R → L shunts
  – excitement in animals with ↓ cardiac reserve (eg heart failure)
  – Circulatory Failure - overuse of diuretics or vasodilators cause
    - acute haemorrhage
    - dehydration

C/S– exercise intolerance
  – syncope
  – cyanosis
  – pre-renal azotaemia
  – pale mucous membranes
  – CRT > 2 secs

Circulatory Failure
- ↓ effective circulating blood volume that may/may not result in heart failure

Cardiomyopathies

Definitions -
Fibrillation = muscle twitching where individual muscle fibers acting without coordination
VPC = ventricular premature contraction
  - ventricular depolarisation originated below AV node
  - ECG – large wide QRS complex and large, inverted T-wave

Myocarditis – cause – usually 2° to systemic disease
  - bacteria - eg Bartonellosis, Lymes disease
  - viral – eg canine parvovirus
  - in 4-8 week old puppies w/ low maternal immunity
  - parasitic - Toxoplasma gondii
  - rarely primary
Primary Cardiomyopathies – problem w/ heart itself
  - Hypertrophic cardiomyopathy (HCM)
  - Restrictive cardiomyopathy (RCM)
  - Unclassified cardiomyopathy (UCM)
  - Dilated cardiomyopathy (DCM)
  - Arrhythmogenic right ventricular cardiomyopathy (ARVCM)
  - Excessive left ventricular moderator bands
  - Persistent Atrial Standstill

Secondary Cardiomyopathies – problem elsewhere affects heart
  – Metabolic - Nutritional (taurine deficiency)
    - Endocrine (eg thyrotoxicosis, acromegaly - ↑ GH, diabetes mellitus)
    - Toxic
    - Anthracyclines (doxorubicin)
  
  - Infiltrative - Neoplastic
    - Hypereosinophilic syndrome
    - Glycogen storage disorders
    - Mucopolysaccharidosis
  
  - Vascular - Systemic hypertension
    - Anaemia (LV dilatation)
    - Ischaemia
    - infarcts
  
  - Physical agents - Heat stroke
  
  - Genetic – hypertrophic cardiomyopathy
    - X-linked muscular dystrophy

Canine Cardiomyopathies

Dilated Cardiomyopathy – DCM

  systolic dysfunction

Aetiology – idiopathic
  - congenital – defective gene coding for mitochondrial function
    - large breeds
  
  - taurine deficiency – dietary deficiency
    - congenital defect in taurine metabolism

Epidemiology – really rare in cats
  - most common cardiomyopathy in dogs
  - breeds – mainly large breeds – Doberman, Irish Wolfhounds,
    Newfoundland & Cocker spaniels
  
  - Prevalence increases w/ age – usually adult onset (7-8 years)
  - more common in males

Heart function - ↓ contractility
  - ↑ end-systolic ventricular volume (dilation) = systolic dysfunction
  - 2° eccentric hypertrophy of LV (volume overload)
  - all chambers dilated, LA and LV most notable

DDx when investigating DCM – similar C/S to many myocardial diseases
  eg viral, nutritional (taurine deficiency), toxic (chemo) and genetic

Stages of DCM

Occult stage - Abnormal heart rhythm detected on ECG or Echo
  - usually incidental finding
  - No clinical signs
  - duration varies – weeks → months → years