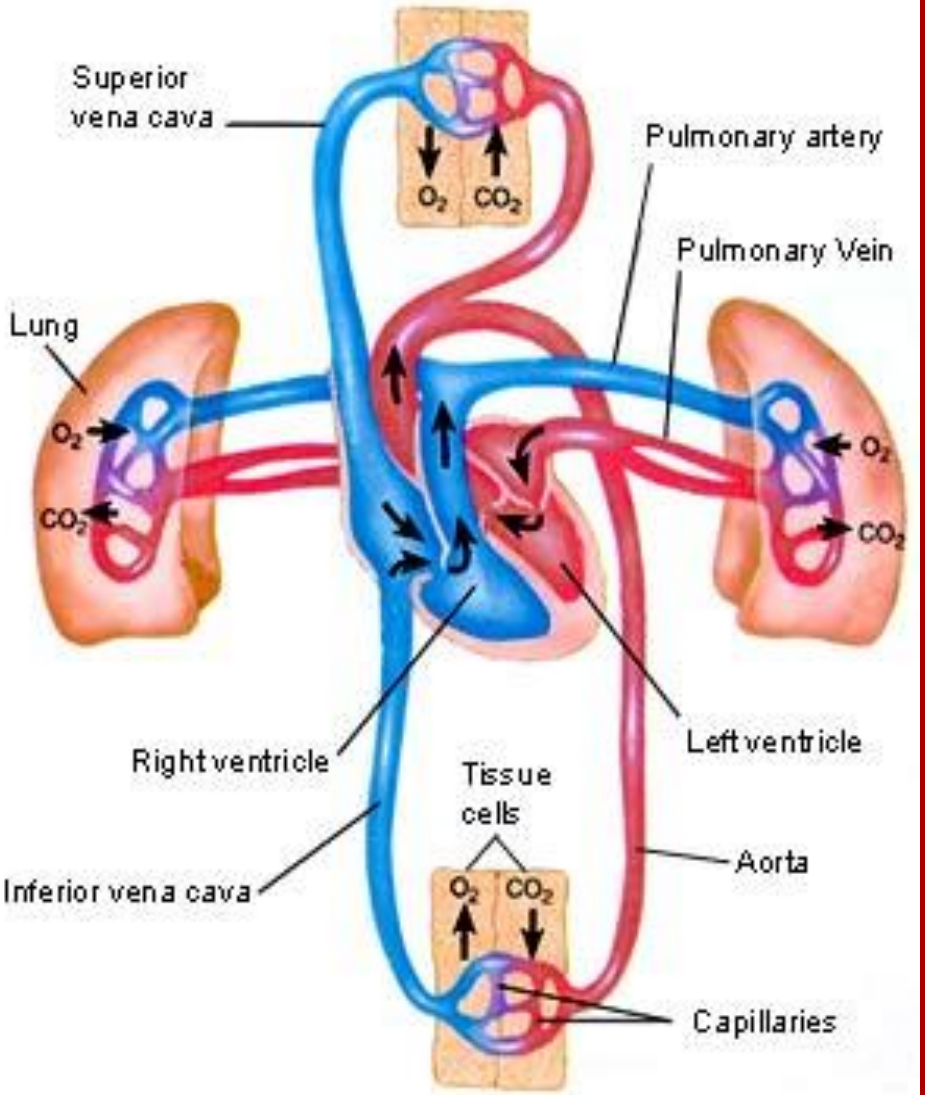
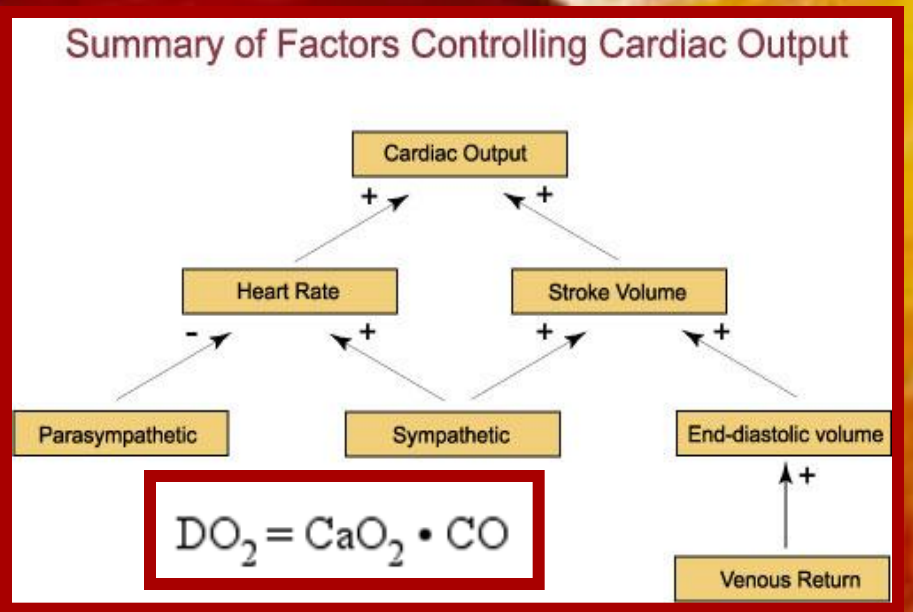
Congestive Heart Failure

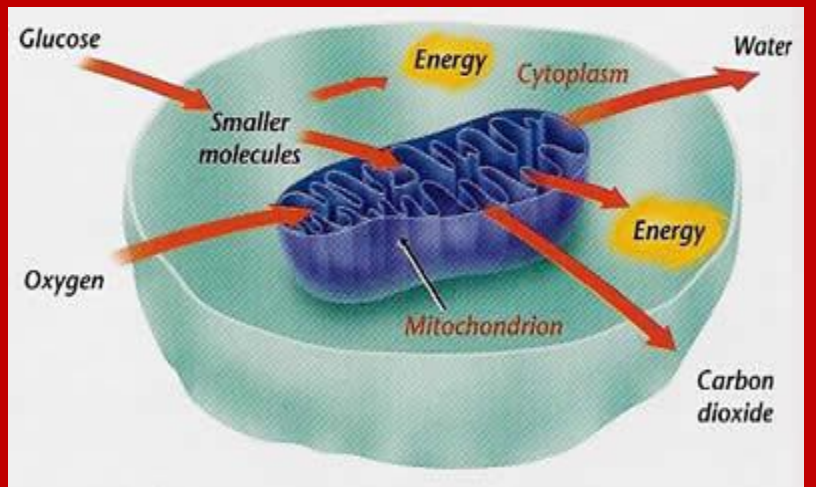
By:

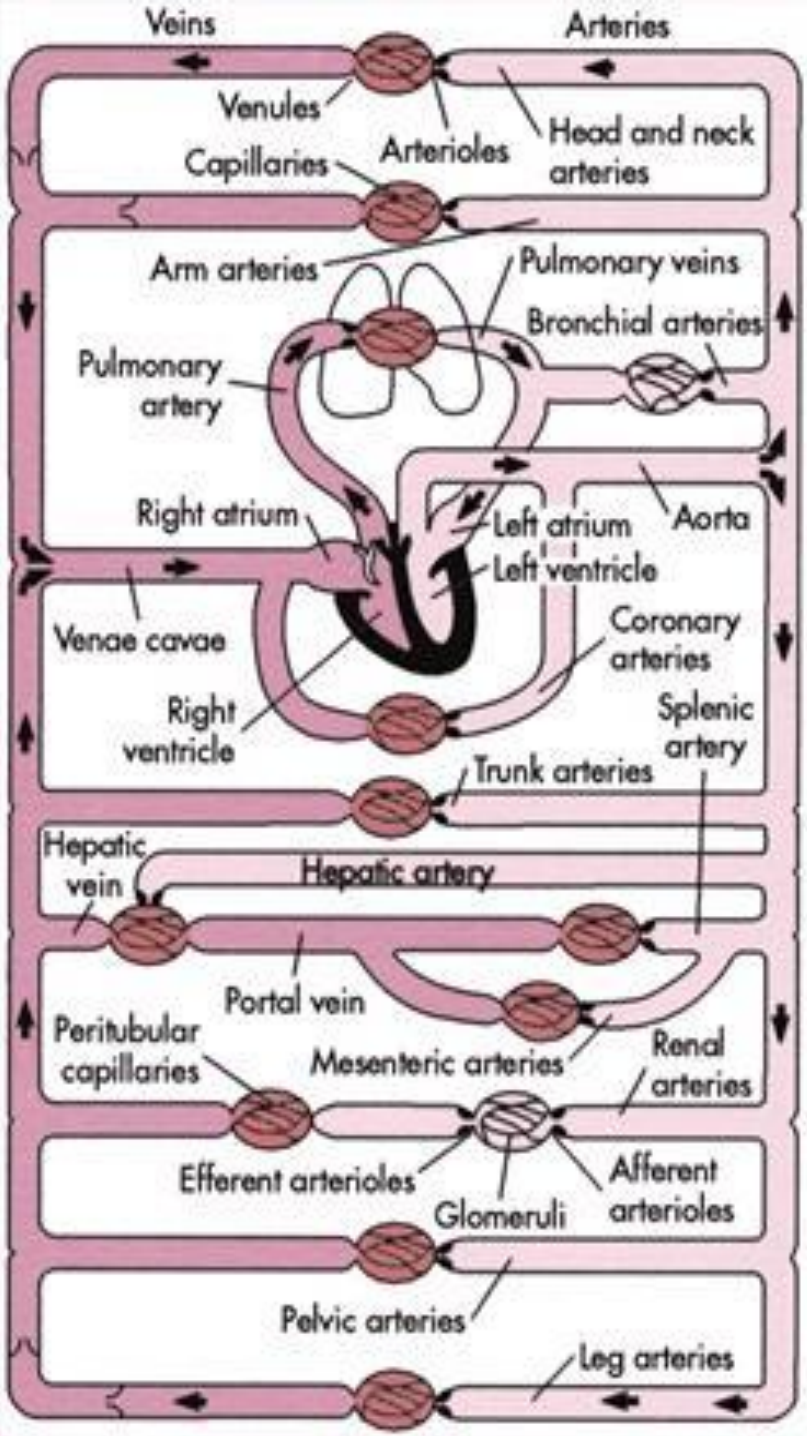
Maureen Luschini

Cardiac Physiology








Veins
Arteries
Venules
Capillaries
Arterioles
Head and neck arteries
Arm arteries
Pulmonary veins
Bronchial arteries
Pulmonary artery
Right atrium
Left atrium
Aorta
Left ventricle
Venae cavae
Coronary arteries
Right ventricle
Splenic artery
Trunk arteries
Hepatic vein
Hepatic artery
Portal vein
Peritubular capillaries
Mesenteric arteries
Renal arteries
Efferent arterioles
Glomeruli
Afferent arterioles
Pelvic arteries
Leg arteries

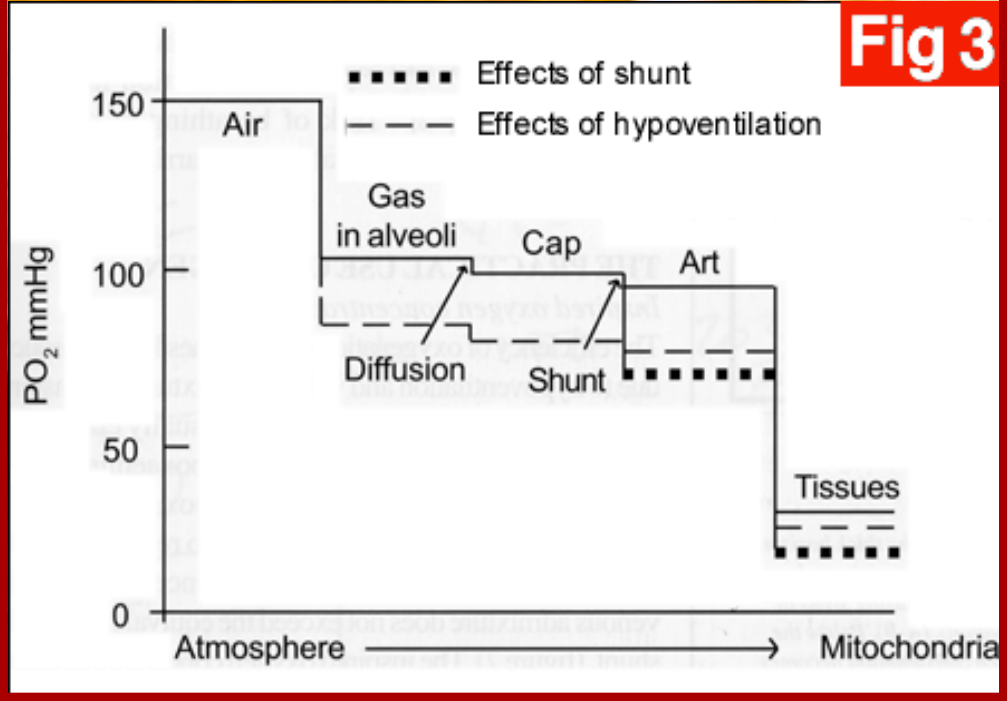


Fig 3
Effects of shunt
Effects of hypoventilation
150
100
50
0
PO_2 mmHg
Air
Gas in alveoli
Cap
Art
Diffusion
Shunt
Tissues
Atmosphere
Mitochondria

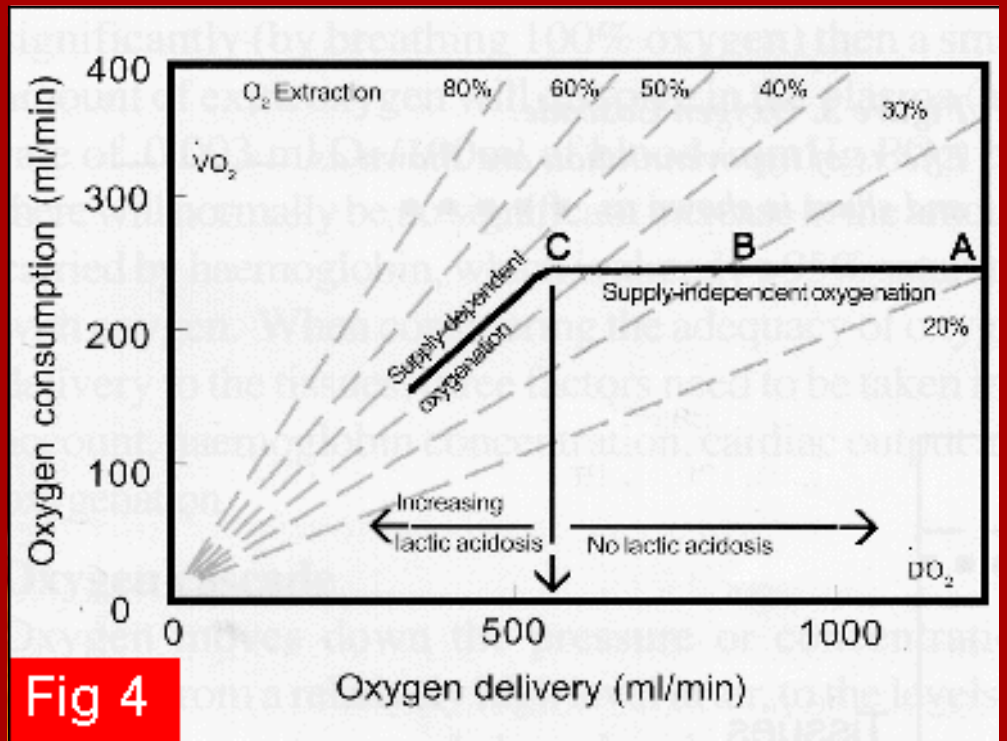
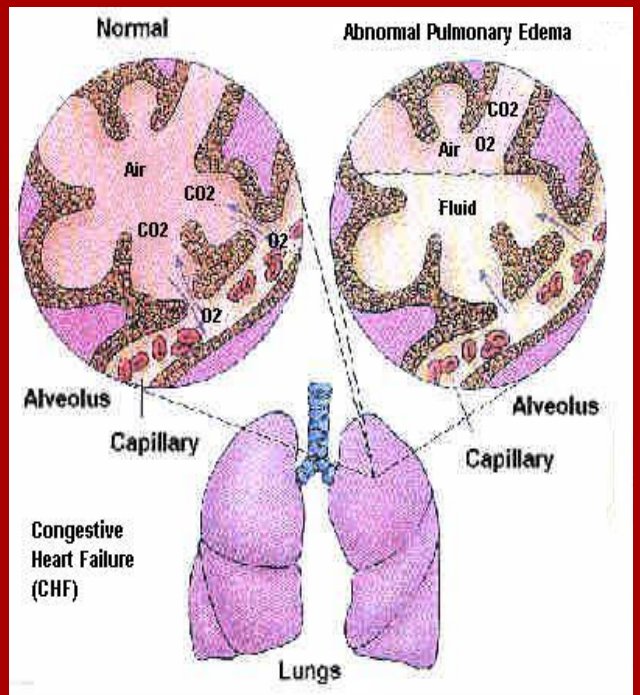
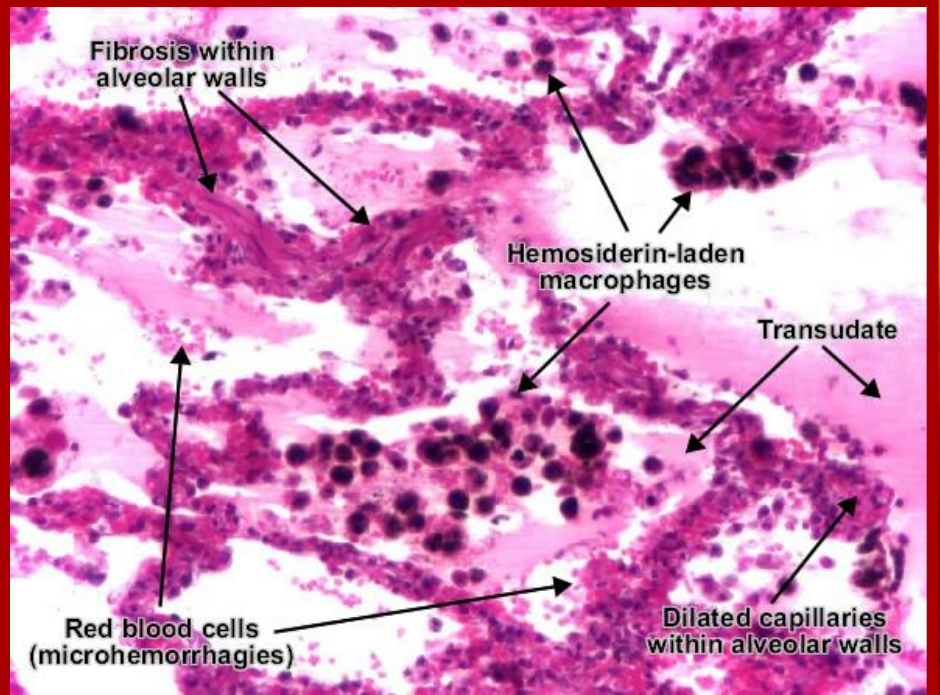

400
300
200
100
0
Oxygen consumption (ml/min)
O_2 Extraction
80%
60%
50%
40%
30%
20%
$\dot{V}O_2$
C
B
A
Supply-dependent oxygenation
Supply-independent oxygenation
Increasing lactic acidosis
No lactic acidosis
$\dot{D}O_2$
0
500
1000
Oxygen delivery (ml/min)

Fig 4

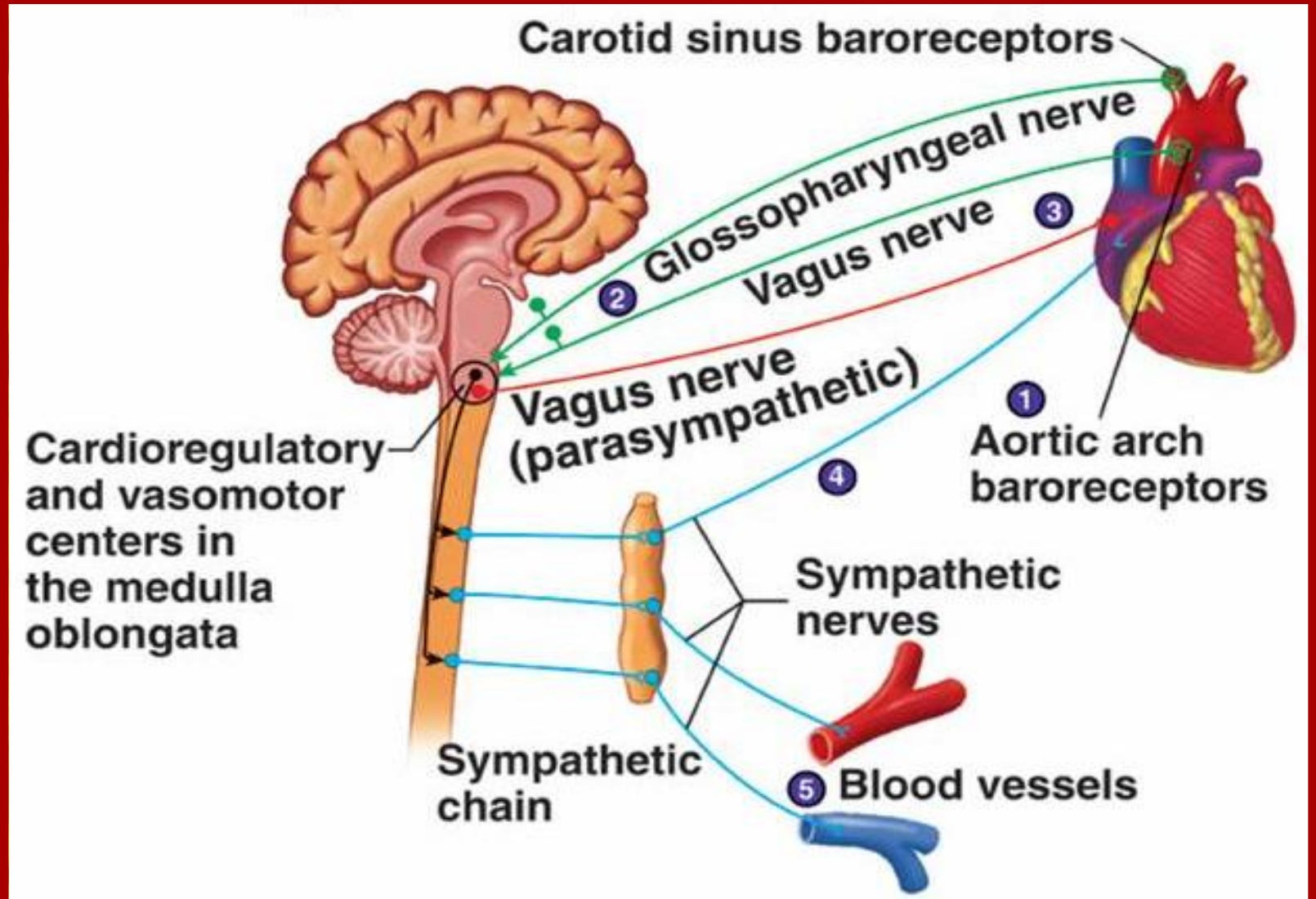
Congestive Heart Failure

- Clinical syndrome
- Abnormal cardiac function results in retention and accumulation of Na and H_2O resulting in congestion and edema





Immediate Response to CHF: BR's

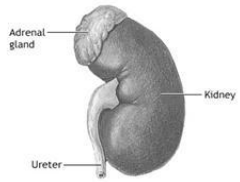


Prolonged CHF: RAAS

Cardiovascular Pharmacology

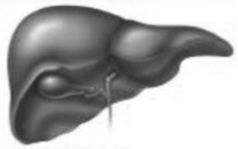
Renin-Angiotensin-Aldosterone-System (RAAS)

- In response to low pressures, the JG cells also release renin, initiating the RAAS system in an attempt to increase systemic BP and subsequent renal blood flow and GFR



Renin: released by JG cells into blood

Angiotensinogen: released by liver cells

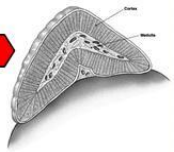


Renin cleaves Angiotensinogen into Angiotensin I

Angiotensin Converting Enzyme converts Angio I to Angio II, mainly in lungs; Also breaks down Bradykinin, a potent vasodilator

Arteries: Angio II is a potent vasoconstrictor (40x > NE); Also numbs baroreceptors to reduce reflex bradycardia

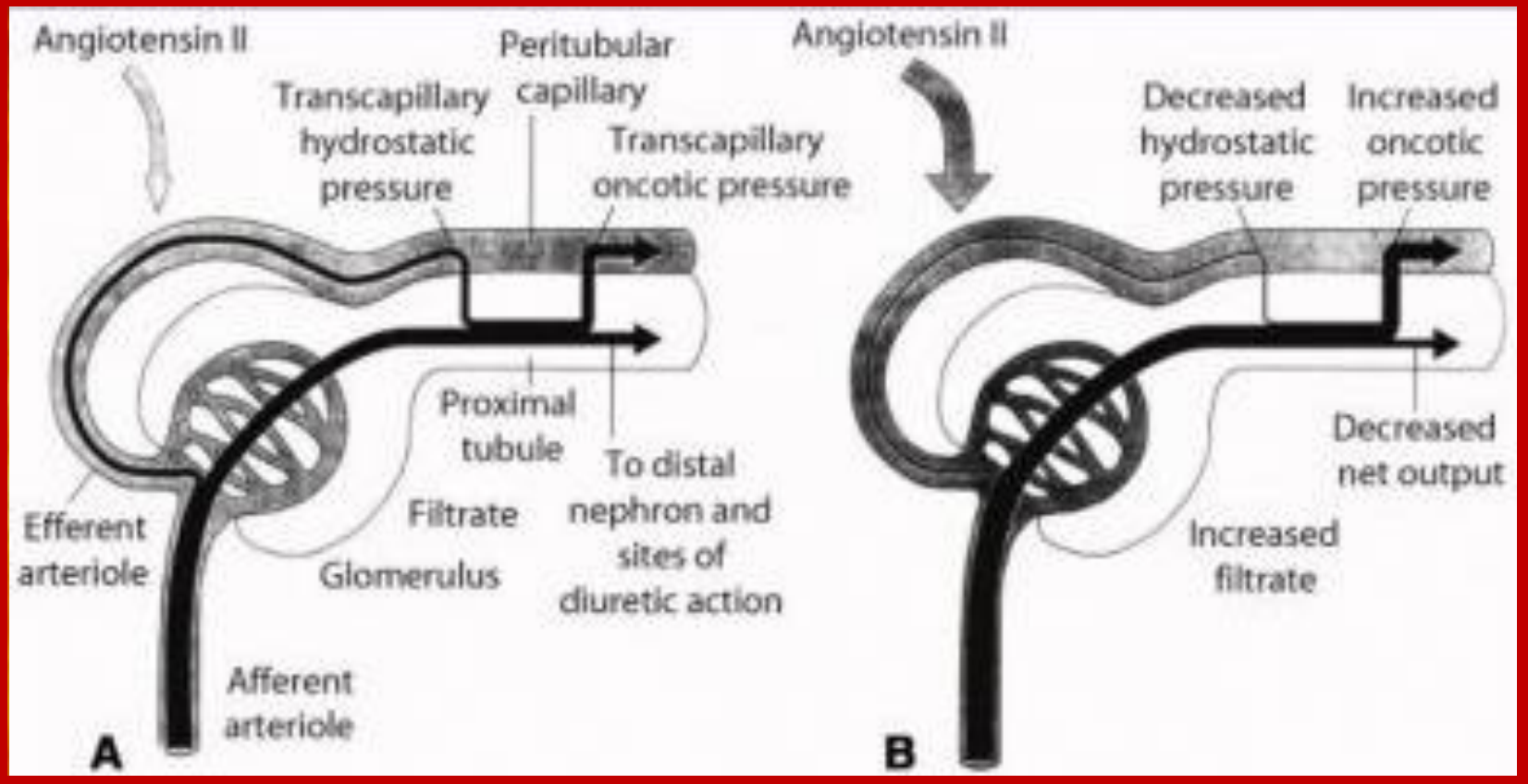
Pituitary: Angio II stimulates release of ADH which increases water resorption in CD; Ups thirst



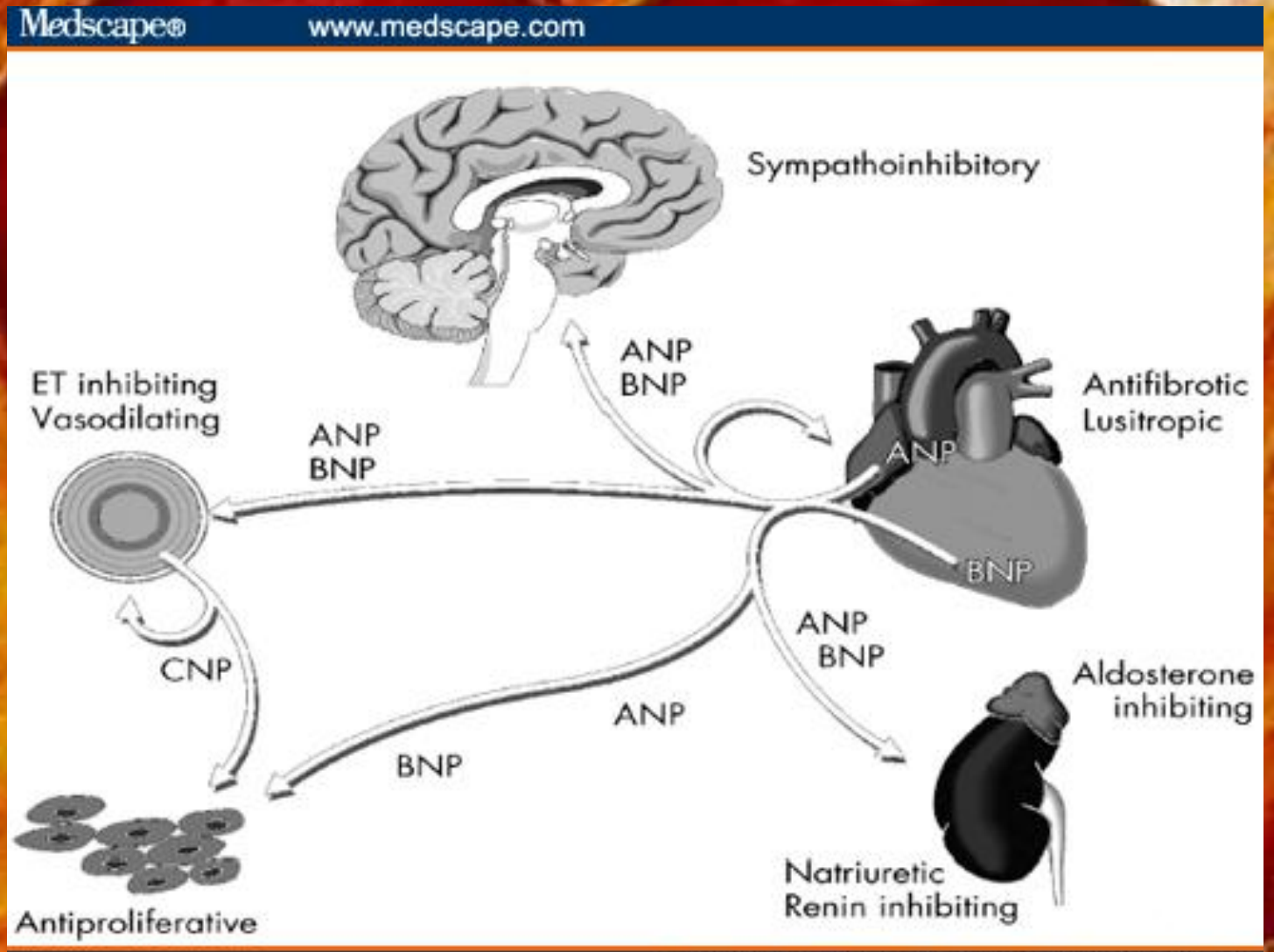
Adrenal: Angio II stimulates Aldosterone, which saves Na and water, excretes K in CD

Ca channels: Angio II opens Ca channels leading to vasoconstriction and + Inotropy

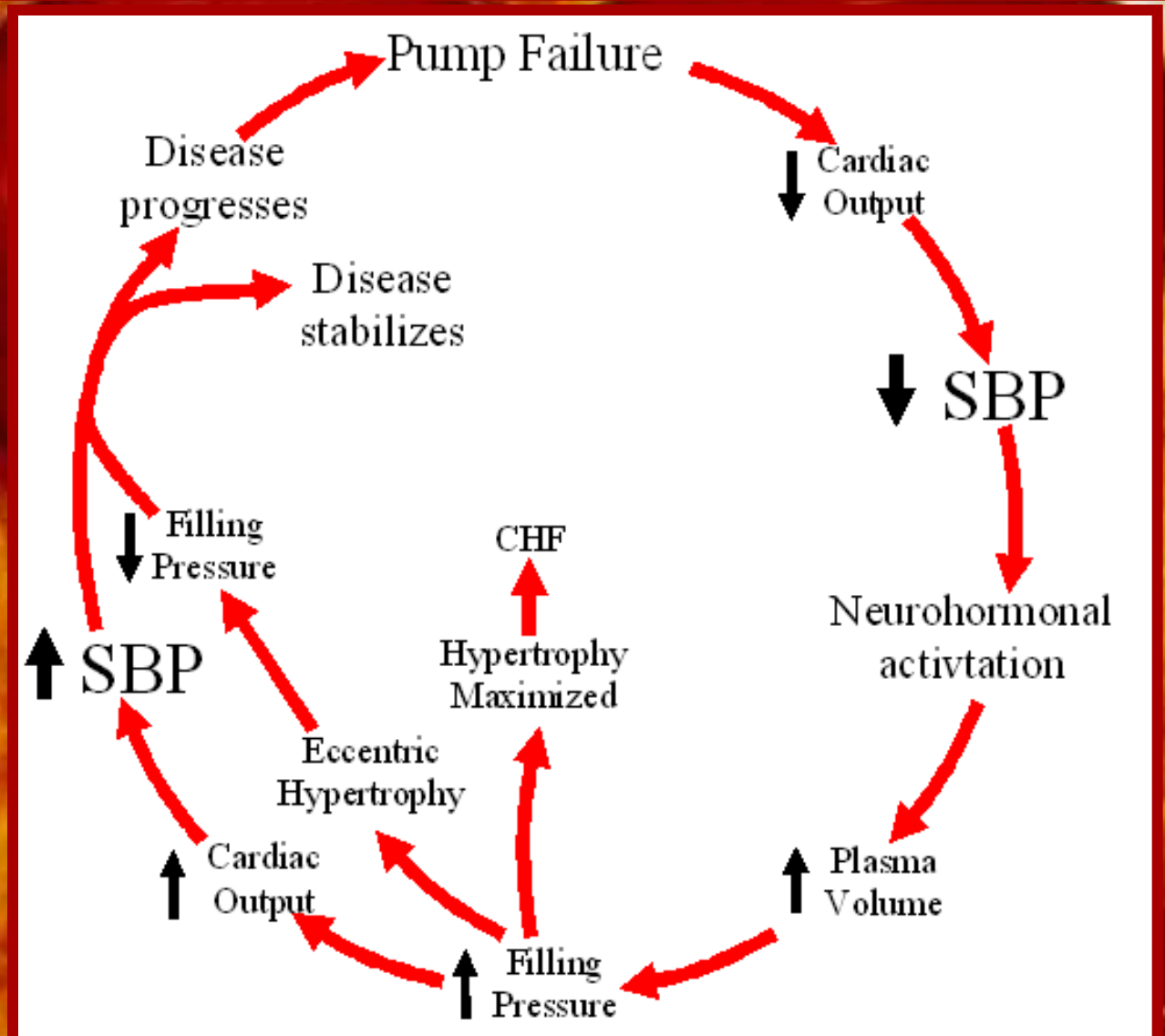
CHF & RAAS

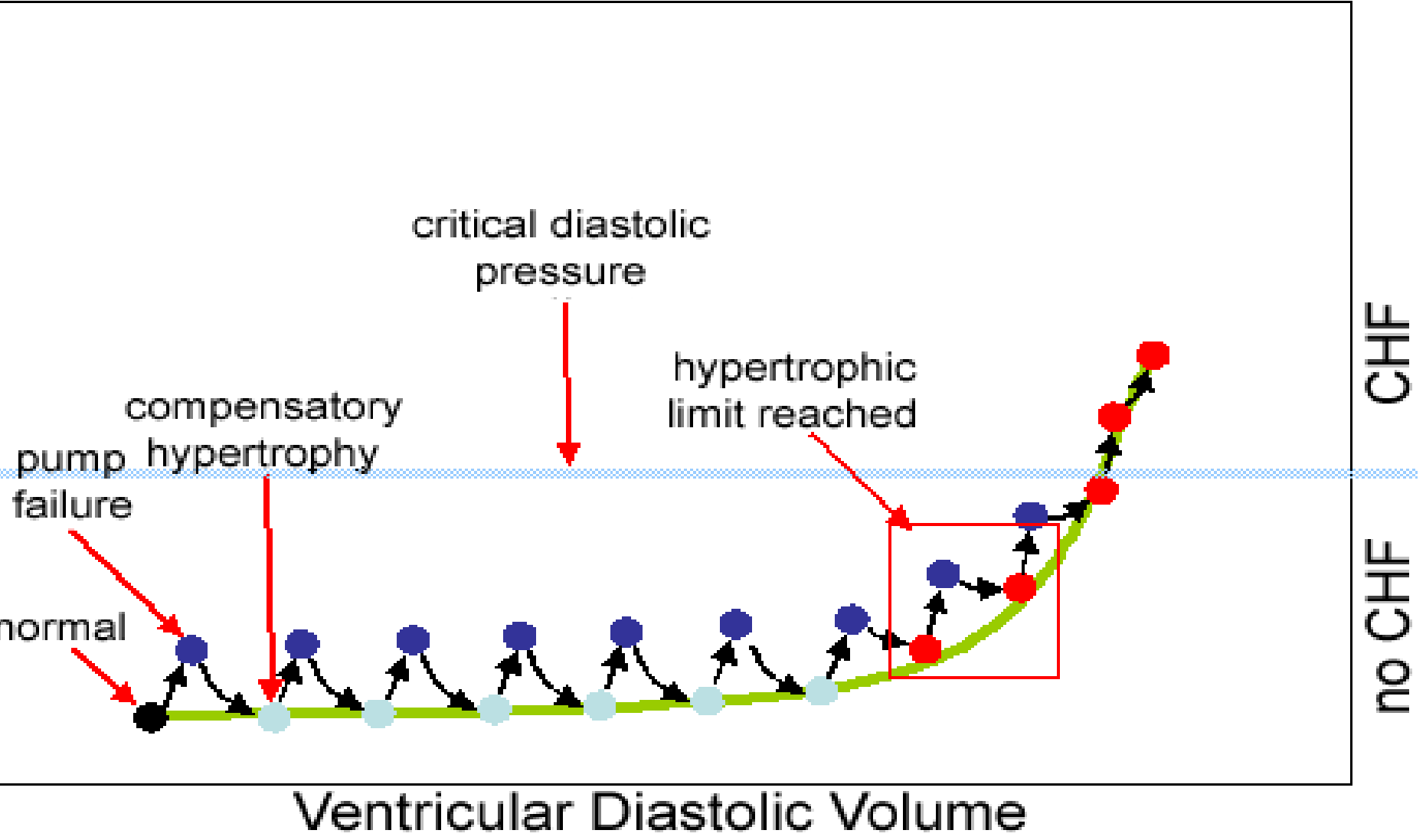

Angiotensin II
Peritubular capillary
Transcapillary hydrostatic pressure
Transcapillary oncotic pressure
Proximal tubule
To distal nephron and sites of diuretic action
Filtrate
Efferent arteriole
Glomerulus
Afferent arteriole
A
Angiotensin II
Decreased hydrostatic pressure
Increased oncotic pressure
Decreased net output
Increased filtrate
B

Prolonged CHF: NP's



CHF: A Viscous Cycle



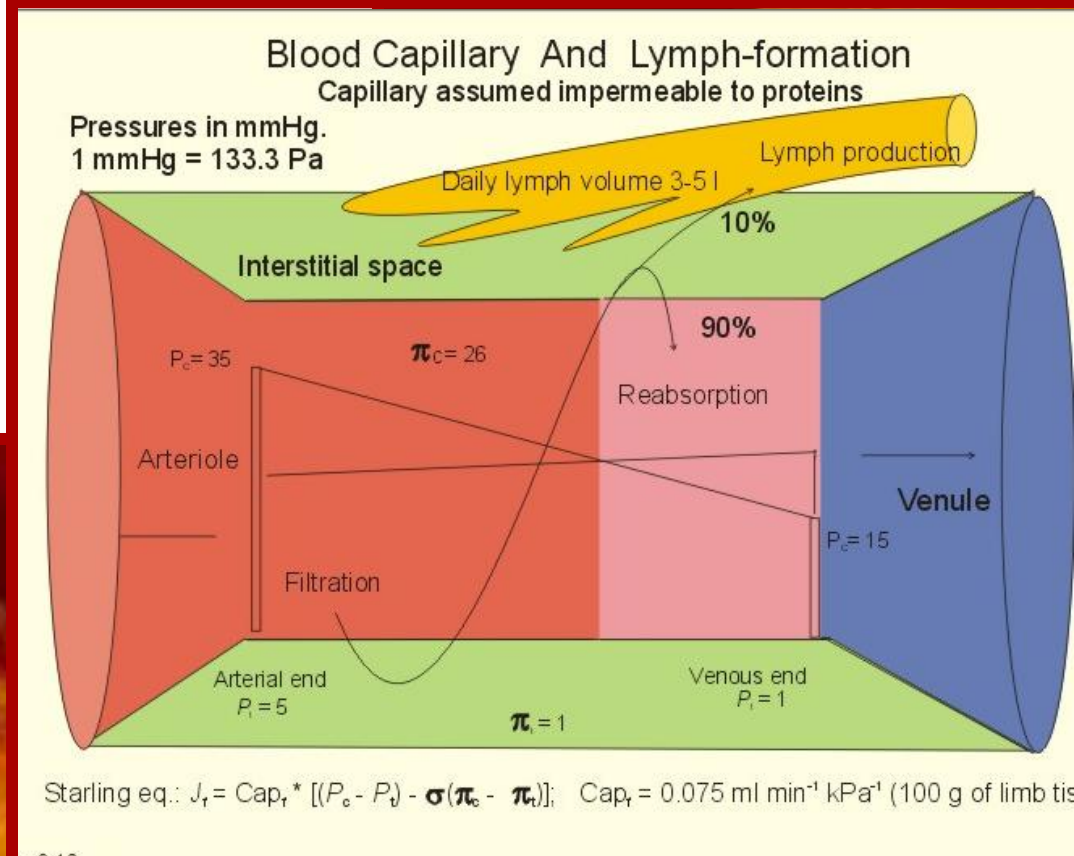


failure, fluid-retaining mechanisms increase diastolic pressure (dark blue circles).
tory eccentric hypertrophy (light blue circles) reduces filling pressure to near-normal
ng ventricular volume. If pump failure progresses, a hypertrophic limit is reached (red
which point ventricular diastolic pressure rises and stretches the ventricles. With
ump failure, the threshold diastolic pressure is reached (blue line) and CHF ensues.

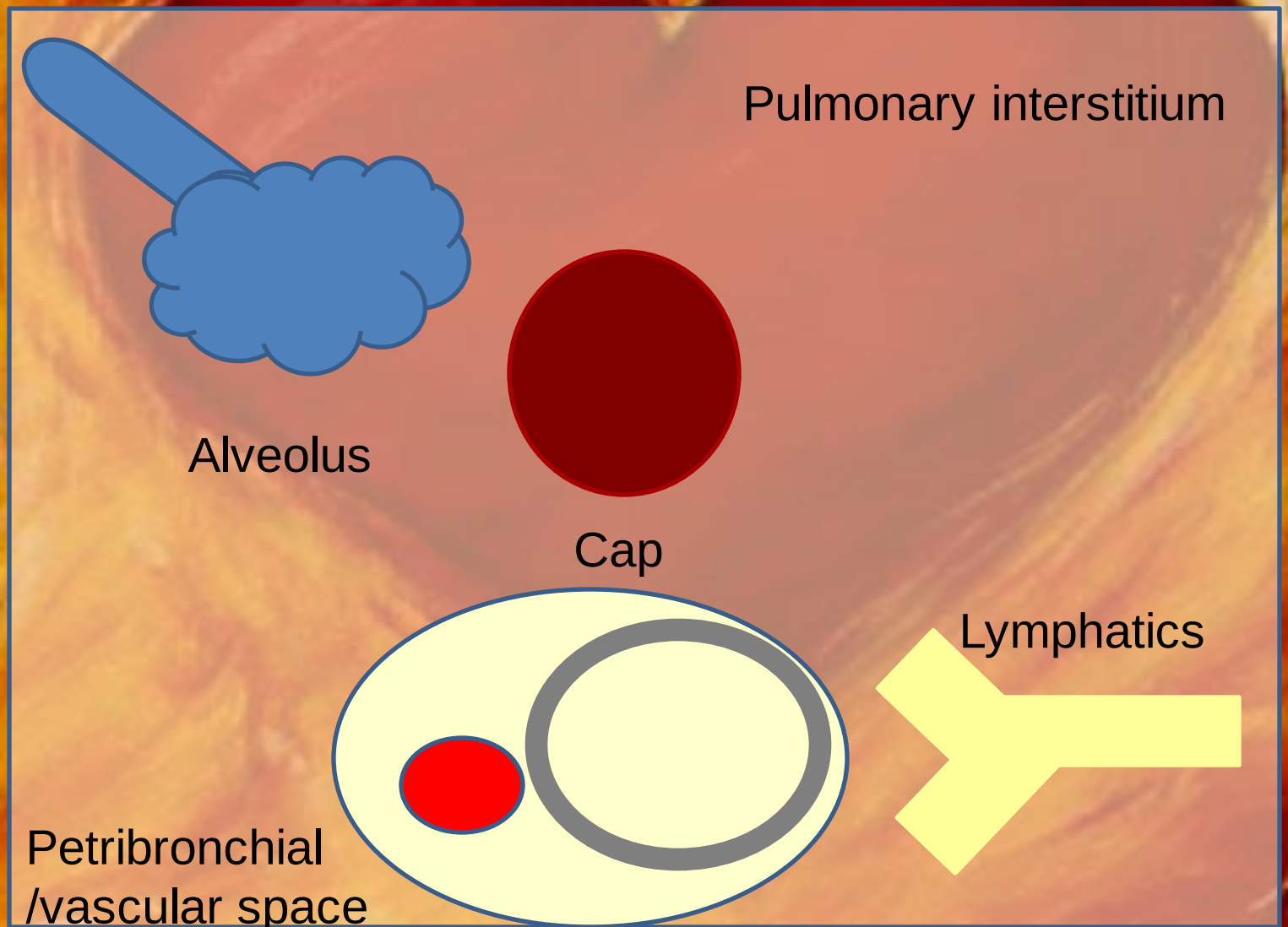
Fluid Accumulation in Lungs

$$\text{Flux} = K_{fc}\left[\left(P_{iv} - P_{is}\right) - \sigma_d\left(\pi_{iv} - \pi_{is}\right)\right]$$

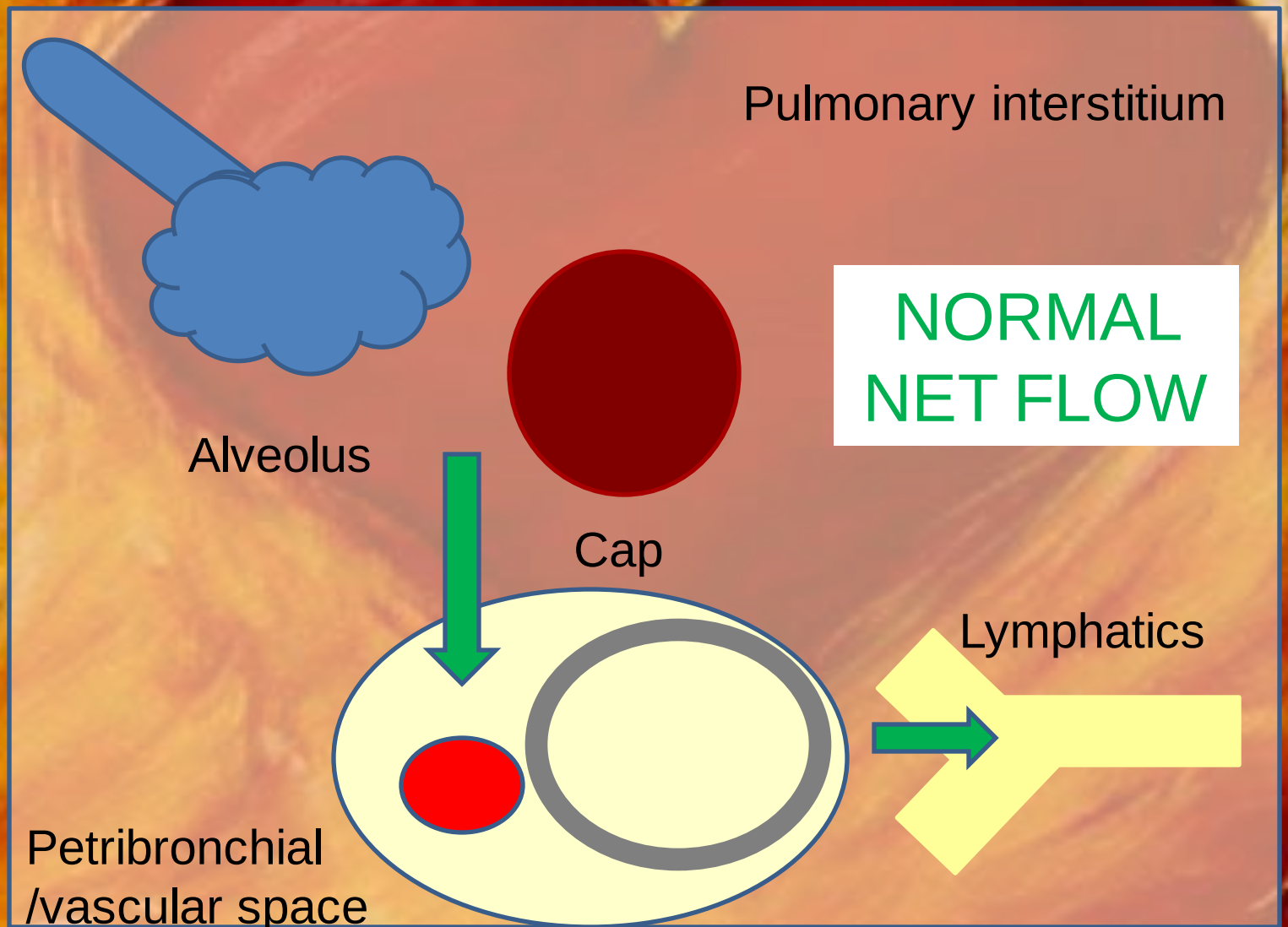
K_{fc}	capillary filtration coefficient
P_{iv}	intravascular pressure
P_{is}	interstitial pressure
π_{iv}	intravascular oncotic pressure
π_{is}	interstitial oncotic pressure
σ_d	reflection coefficient



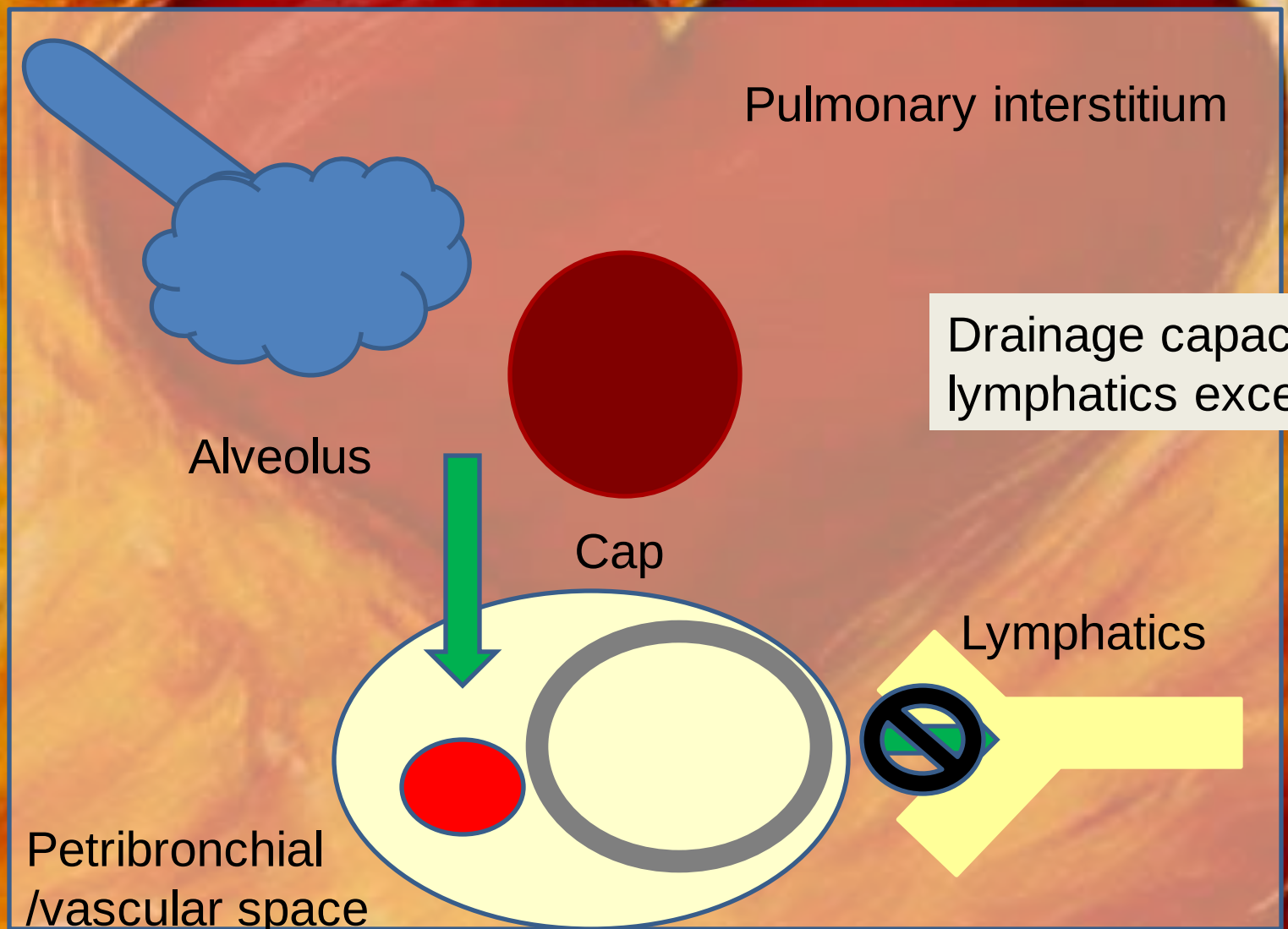
Fluid Accumulation in Lungs



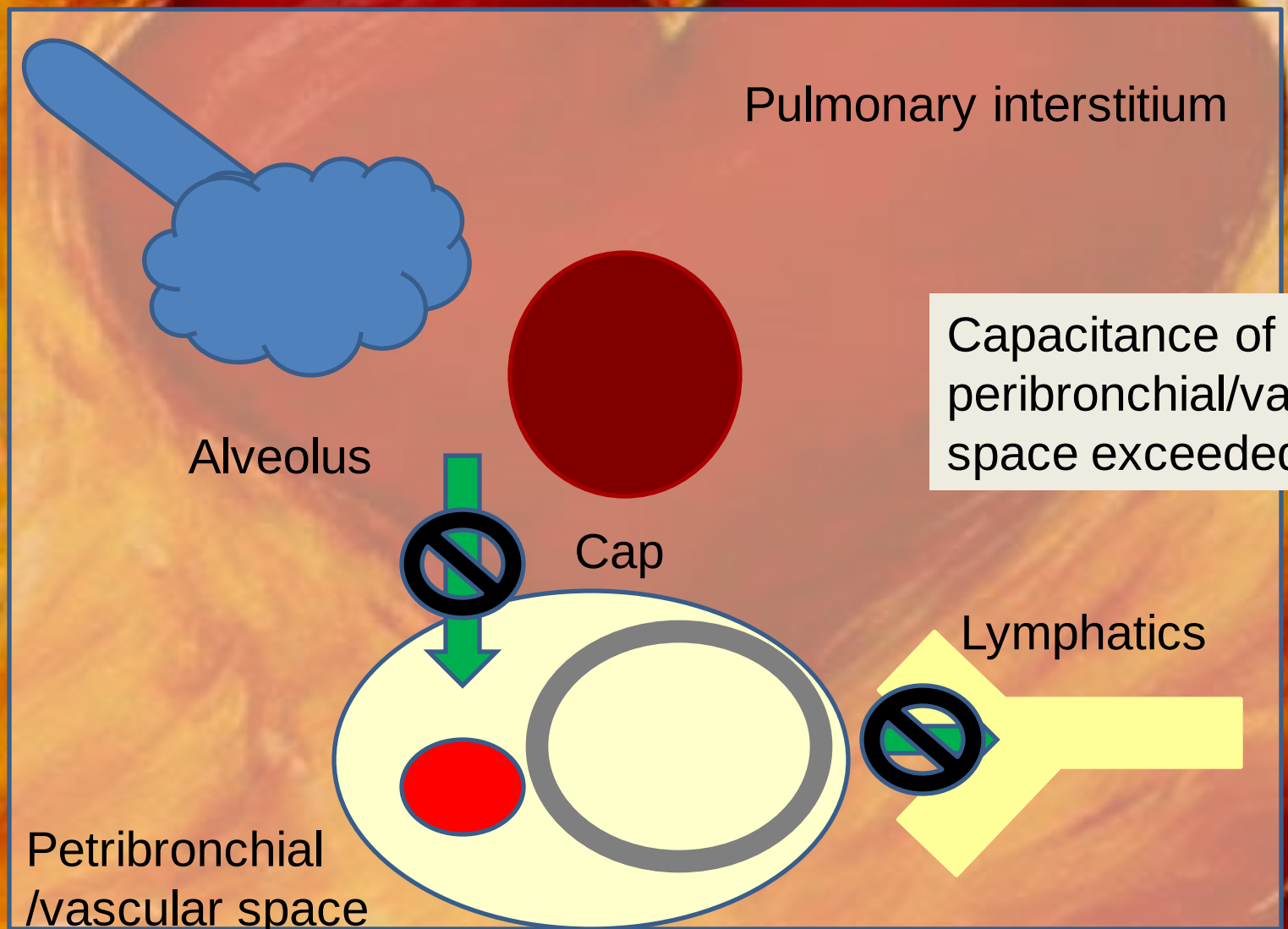
Fluid Accumulation in Lungs



Fluid Accumulation in Lungs

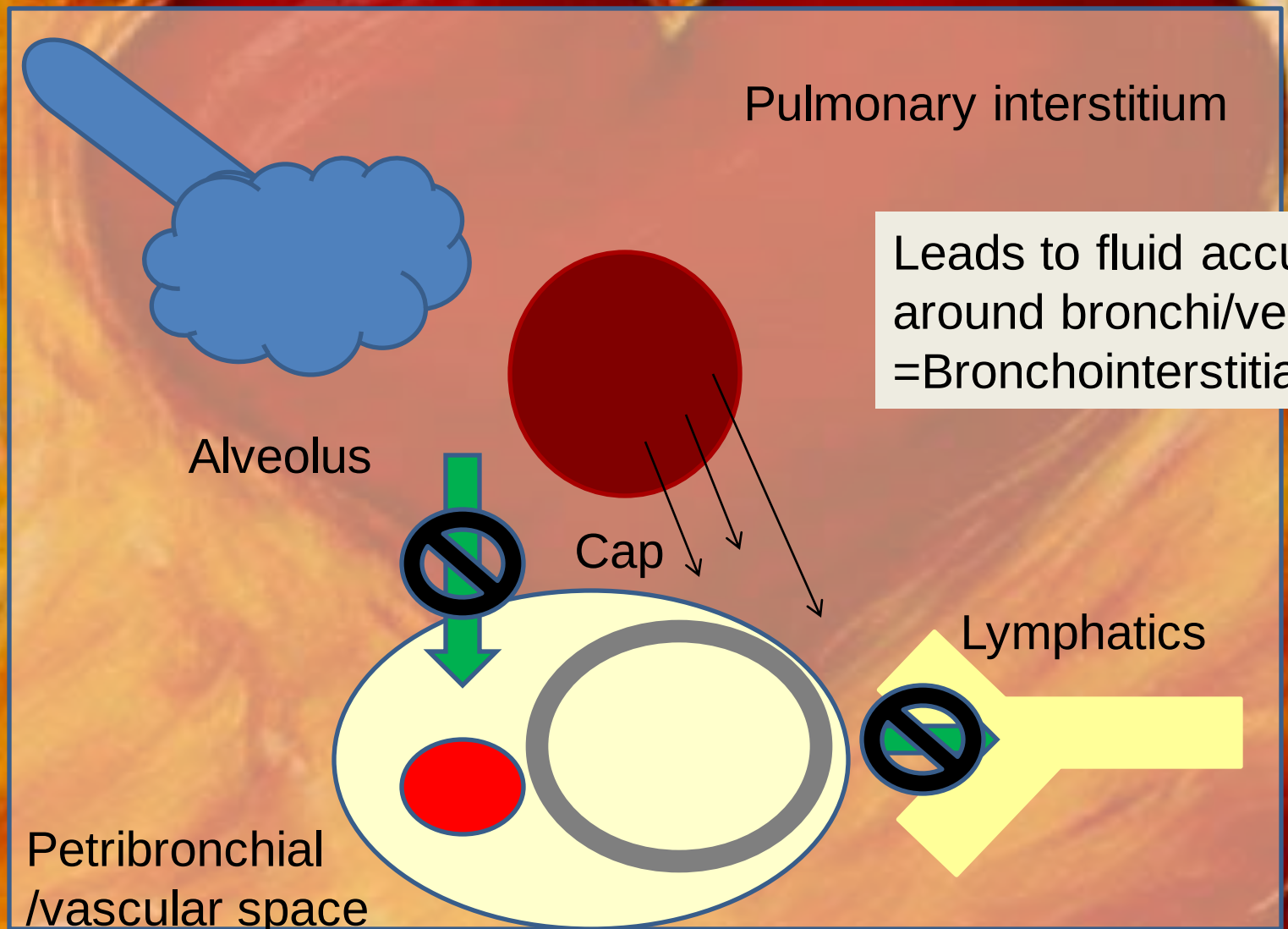


Fluid Accumulation in Lungs



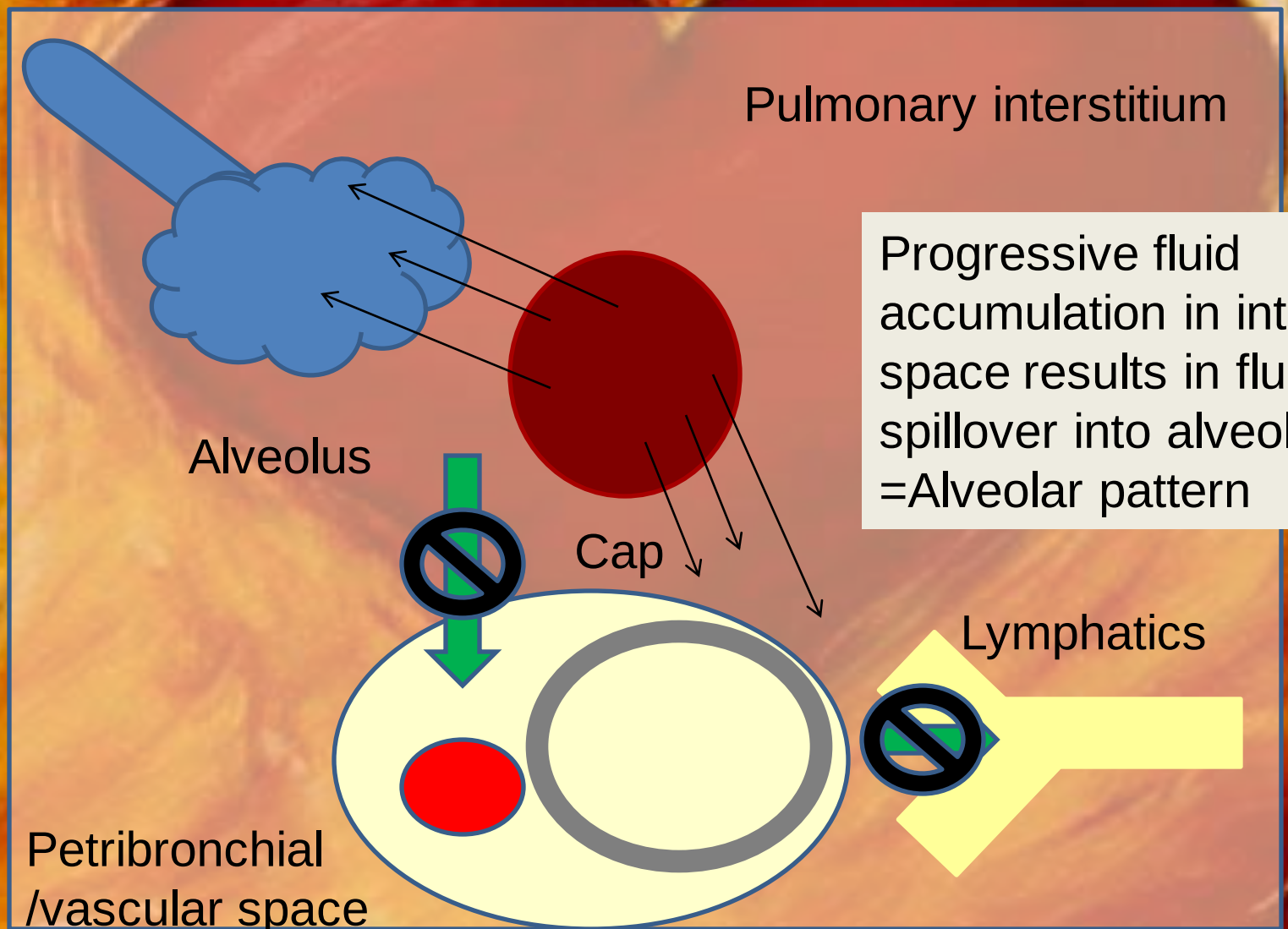
Capacitance of peribronchial/vascular space exceeded

Fluid Accumulation in Lungs



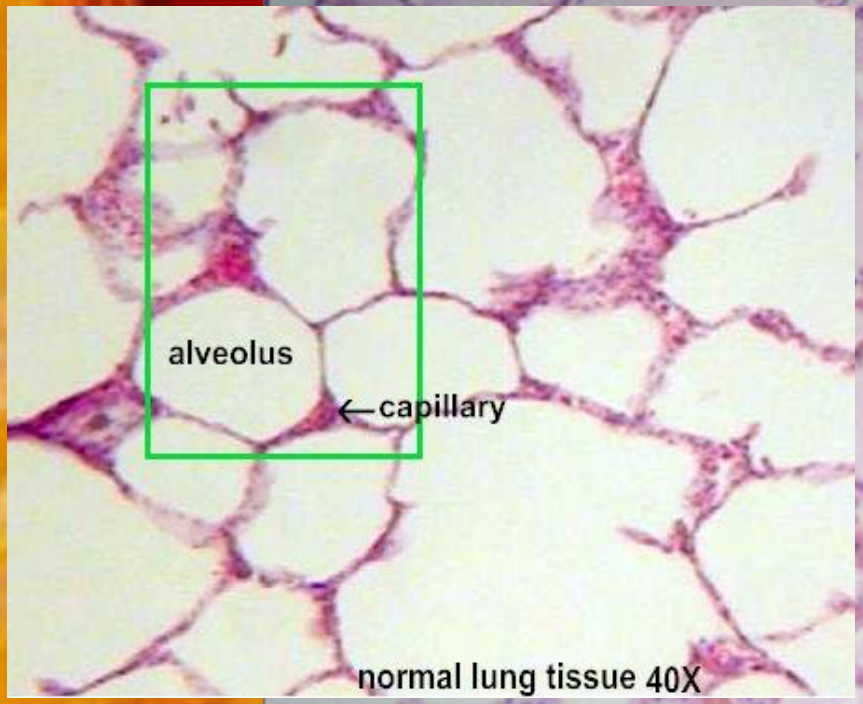
Leads to fluid accumulation around bronchi/vessels =Bronchointerstitial pattern

Fluid Accumulation in Lungs



Progressive fluid accumulation in interstitial space results in fluid spillover into alveoli =Alveolar pattern

Fluid Accumulation in Lungs



Causes of CHF

Table 8-1. Common cardiovascular disorders classified by functional alteration

Functional classification	Morphologic classification	Examples
Primary systolic myocardial failure		Dilated cardiomyopathy
Secondary systolic myocardial failure	Infectious myocarditis Drugs, chemicals, toxins Physical damage Nutritional Ischemic Infiltrative Other cardiac disorders	Bacterial, fungal, viral, protozoal Doxorubicin (Adriamycin), alcohol Heat stroke, electric shock, trauma Taurine deficiency Atherosclerosis Neoplasia, metabolic (amyloid) Valvular insufficiency, shunts
Pressure overload	Hypertension Anatomic outflow obstruction Dynamic outflow obstruction	Systemic or pulmonary hypertension Aortic stenosis, pulmonic stenosis Hypertrophic obstructive cardiomyopathy
Primary volume outflow	Valvular insufficiency Left-to-right shunt High-output states	Mitral insufficiency Patent ductus arteriosus, ventricular septal defect Hyperthyroidism, atrioventricular fistula, anemia
Impedance to cardiac inflow	Pericardial disease Diastolic dysfunction Atrioventricular valvular obstruction Space-occupying lesions	Pericardial effusion, constrictive pericarditis Hypertrophic and restrictive cardiomyopathy Mitral and tricuspid stenosis Atrial mass lesions, right ventricular outflow tract tumors, Budd-Chiari syndrome

CHF: Clinical Signs

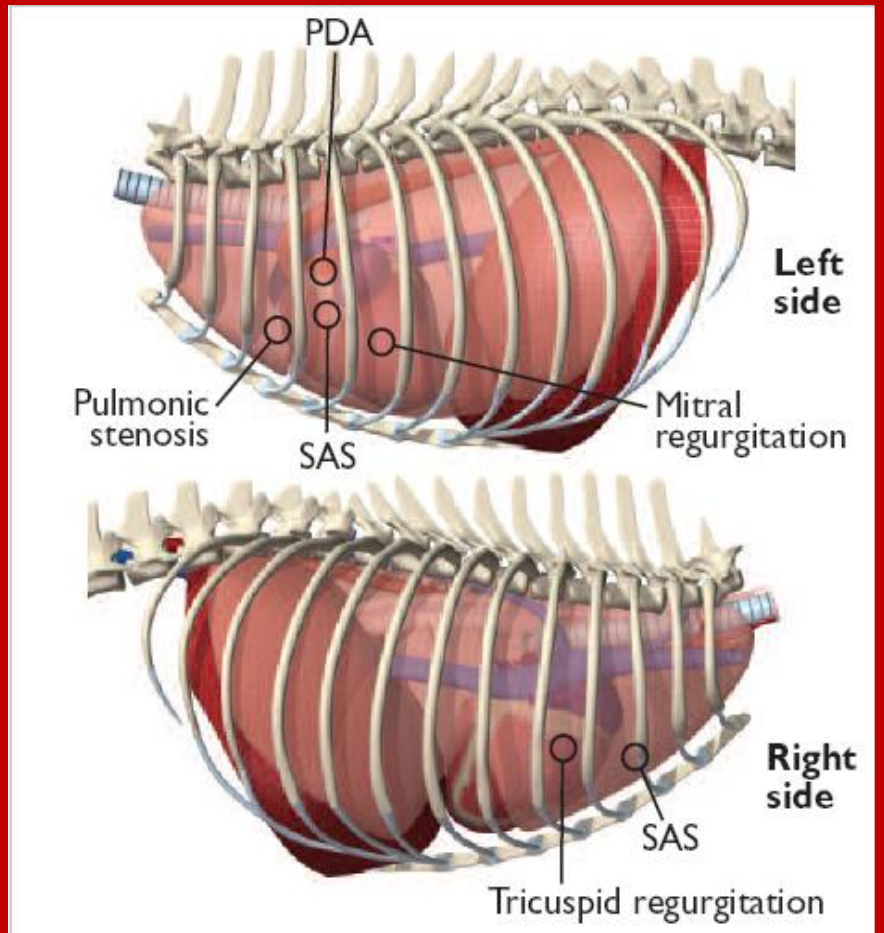
- Exercise intolerance
- Lethargy
- Anorexia
- Tachypnea
- Cough
- Orthopnea
- Respiratory distress

CHF: Clinical Signs

Table 1. Clinical and Physical Examination Signs Associated with Cardiac and Pulmonary Diseases

	Sign						
Disease/Condition	Coughing	Exercise intolerance	Syncope	Cyanosis	Pulmonary crackles	Poor pulse quality	Palpable dysrhythmia
CHF	Yes	Yes	±	±	Yes	±	±
Pulmonary parenchymal disease	±	±	±	±	±	No	±
Heartworm disease	Yes	±	±	±	±	±	±
Tracheal collapse	Yes	±	No	±	No	No	No
Fungal disease	Yes	±	No	±	±	No	No
Neoplasia	Yes	±	±[a]	±	±	No	±[a]

CHF: Physical Exam and POC Tests

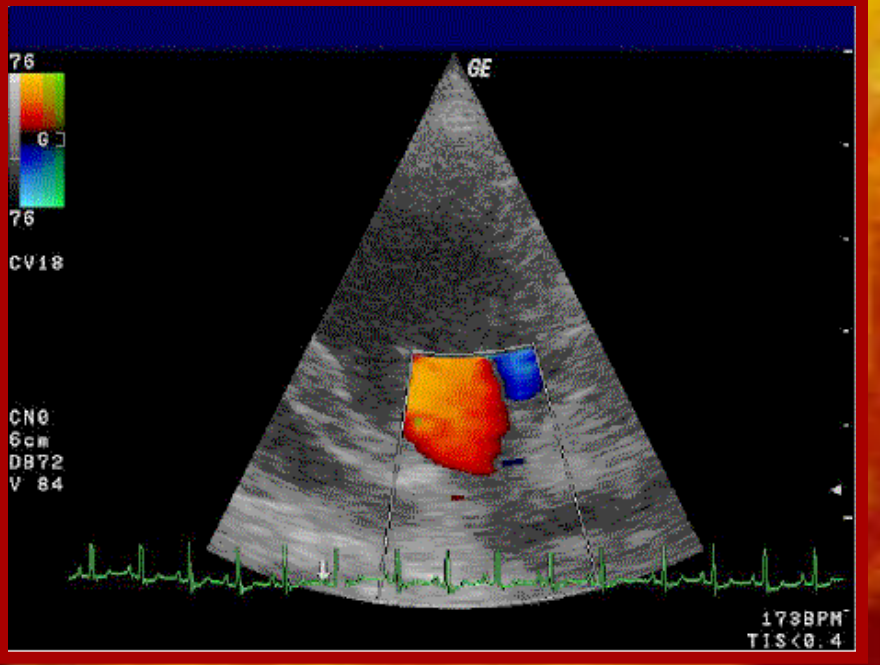


+/- murmur, arrhythmia

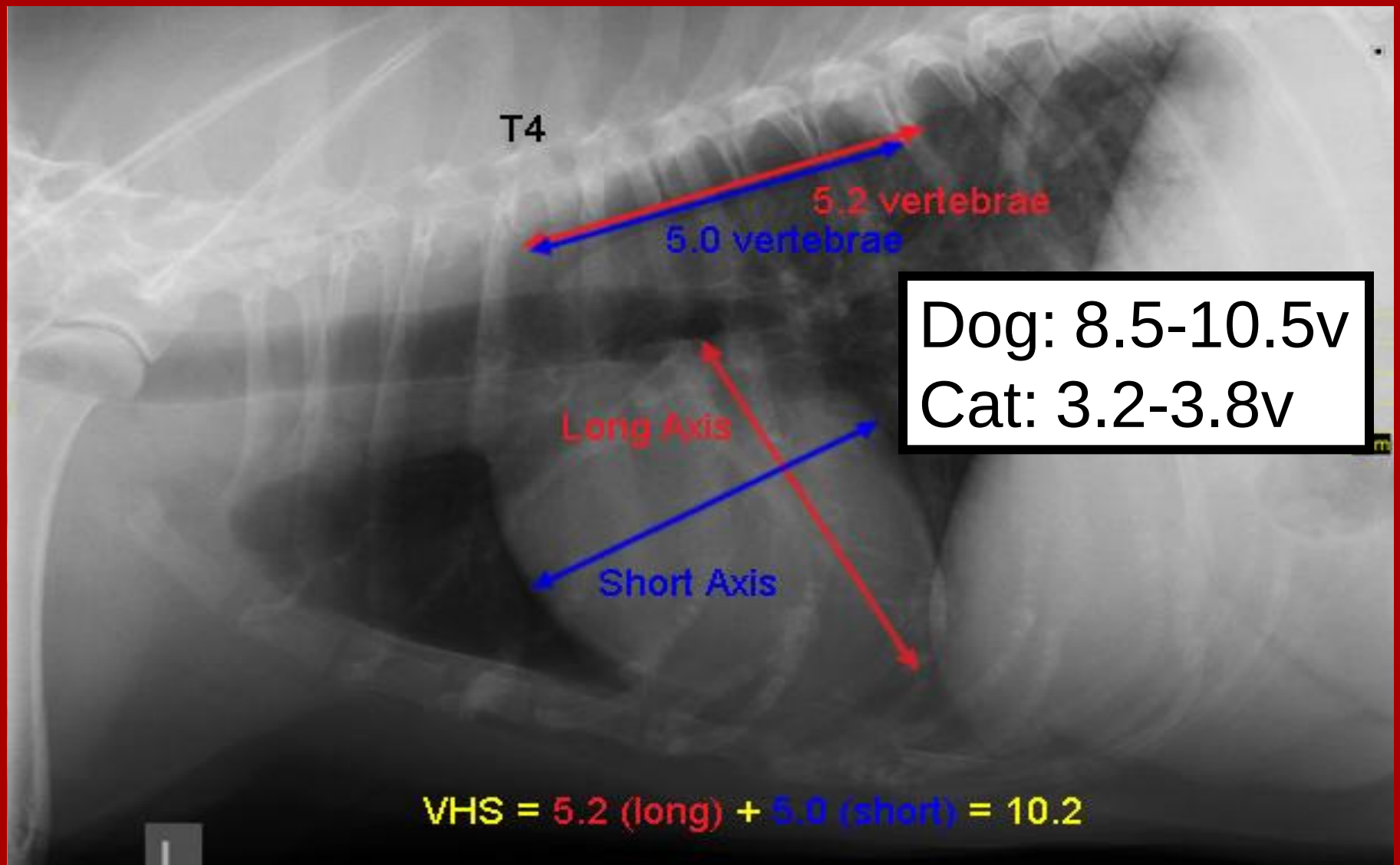
- Pale mm
- CRT > 2sec
- Cyanosis
- Tachycardia
- Cool limbs
- Hypothermia
- Increased lactate
- Low venous PaO_2
- Azotemia

CHF: Diagnosis

- ECG (minor cotribution)
- Thoracic radiographs
- Echocardiography
- Pulmonary fluid analysis

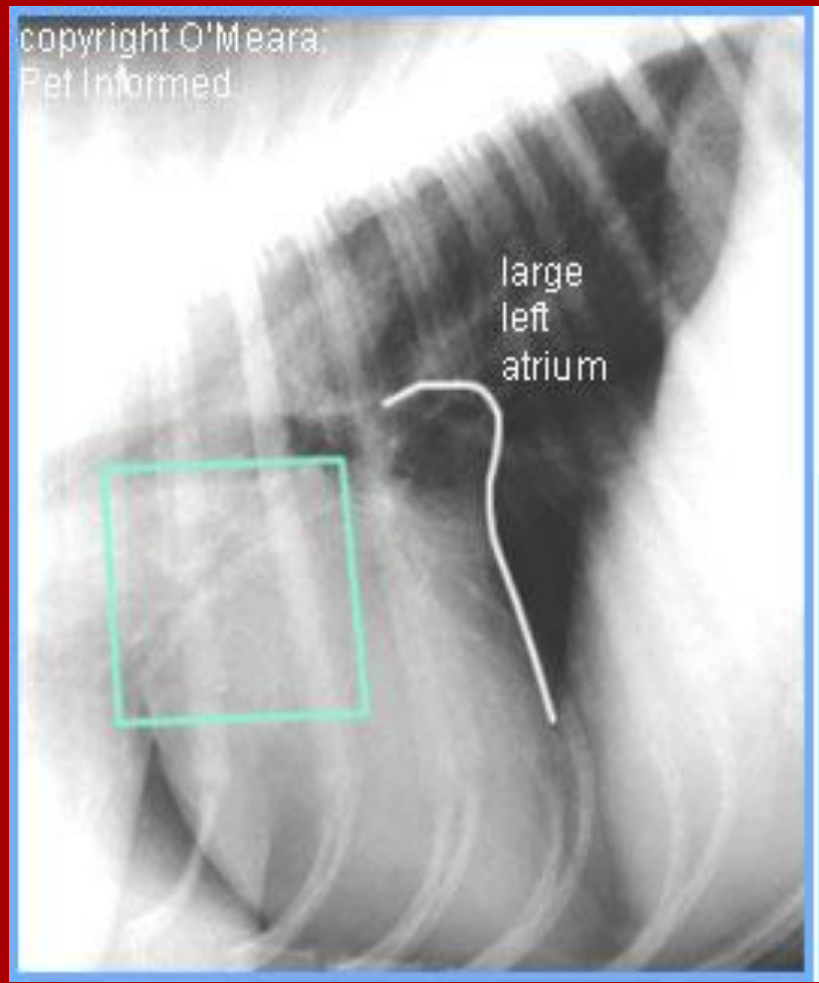


CHF: Thoracic Radiographs

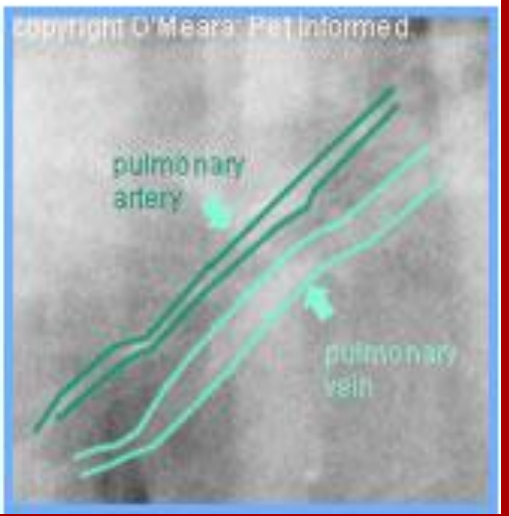


Dog: 8.5-10.5v
Cat: 3.2-3.8v

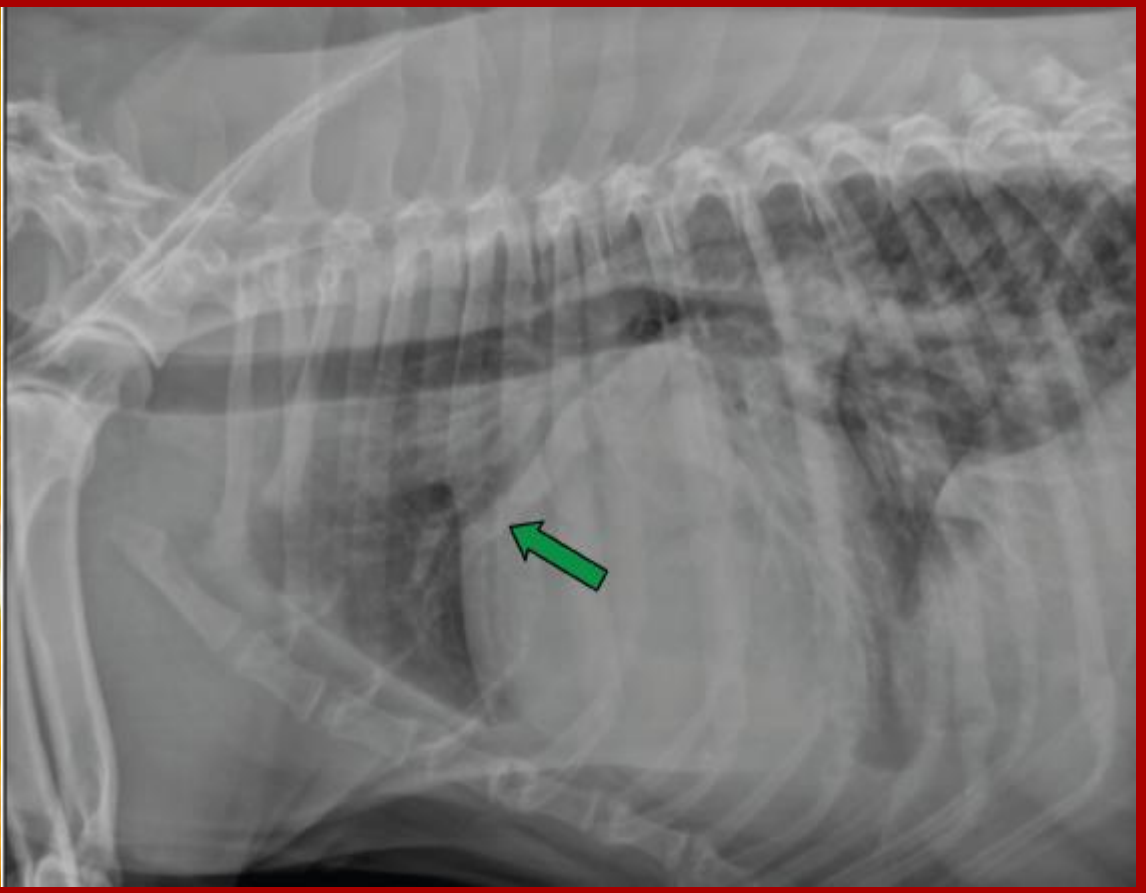
CHF: Thoracic Radiographs

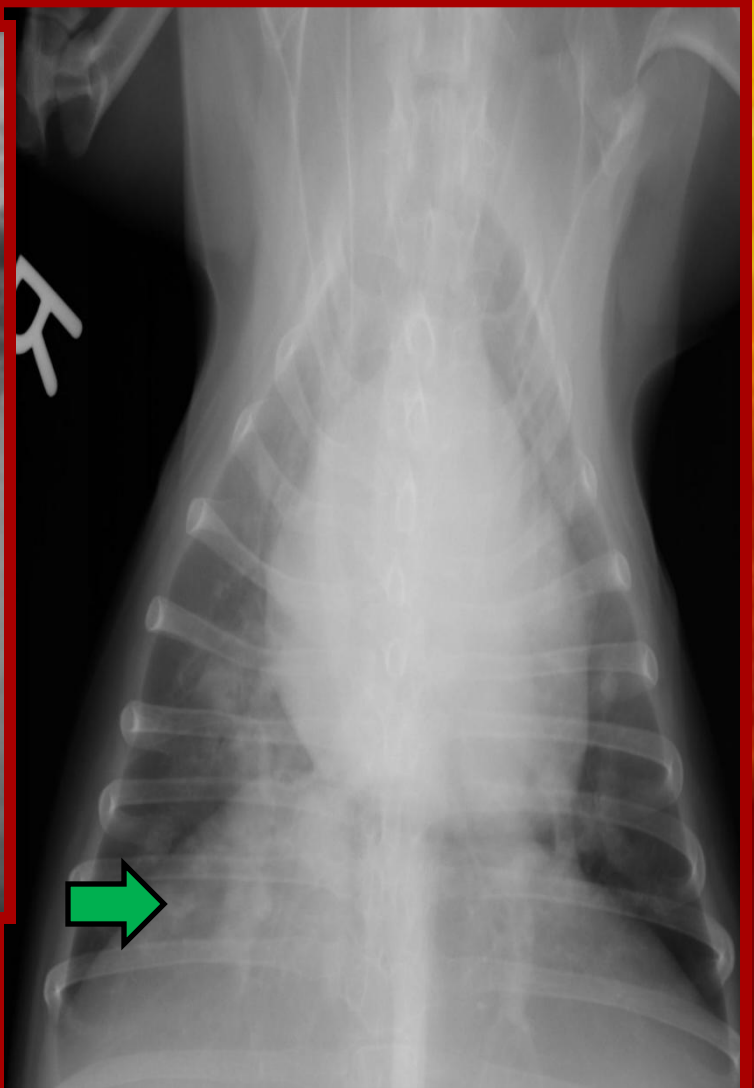


Thoracic Radiographs



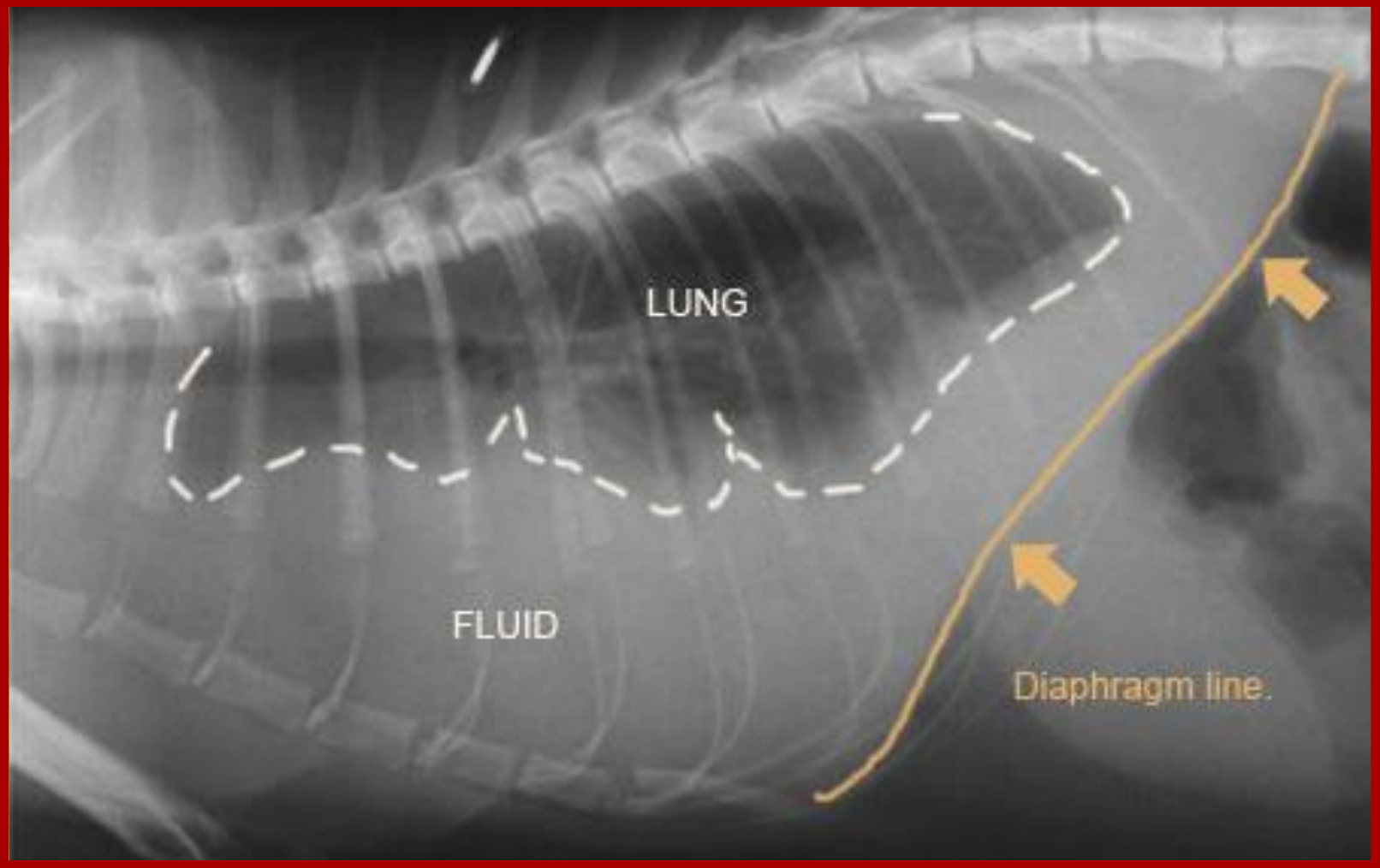
CHF: Thoracic Radiographs





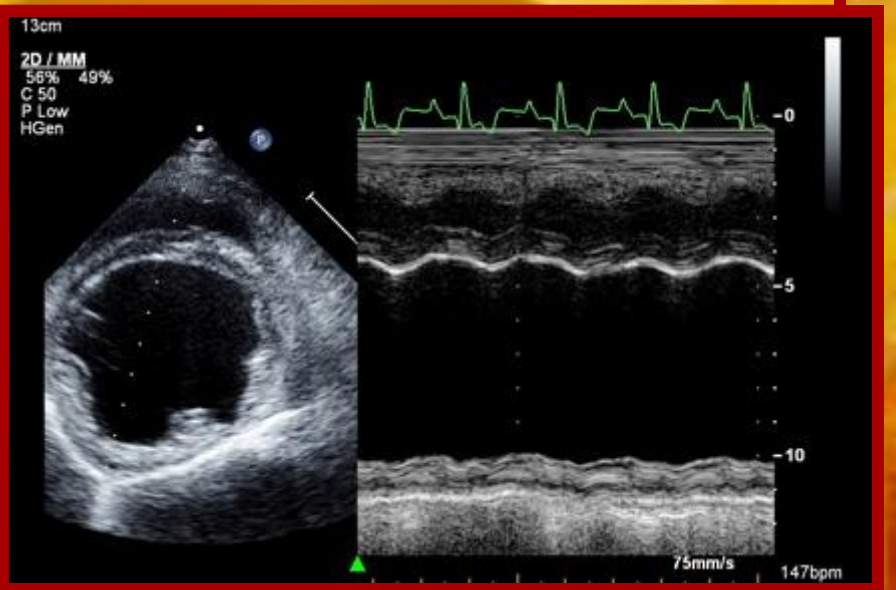
Pulmonary veins>pulmonary arteries

Cats…Always the Exception

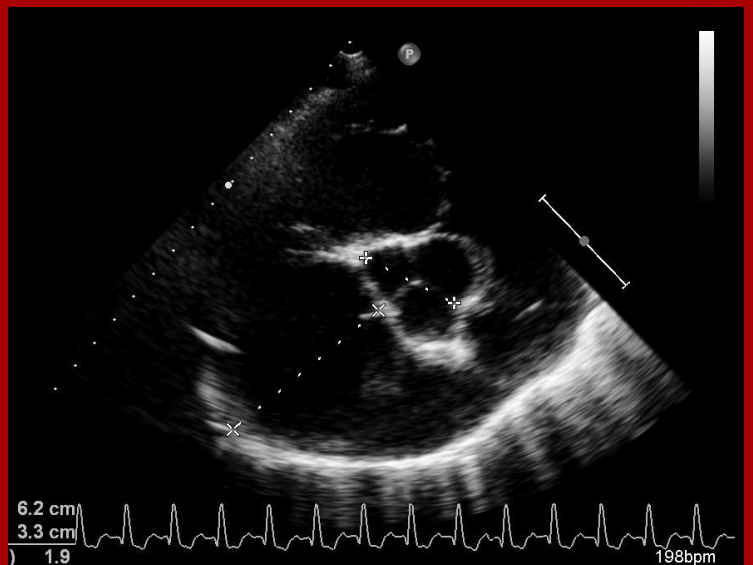


CHF: Echocardiography

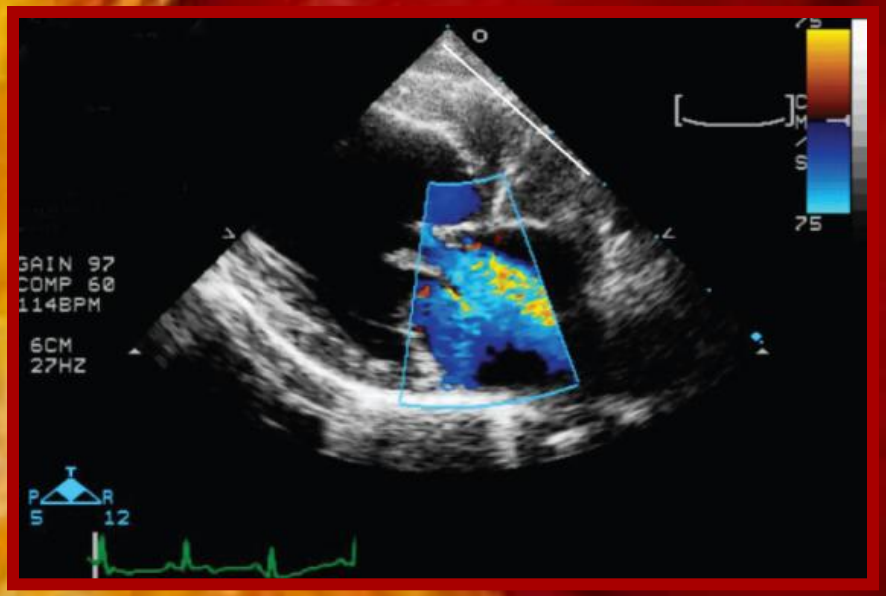
- Evaluate heart function
- Diagnose cardiac disease as cause of CHF
- Left atrial enlargement

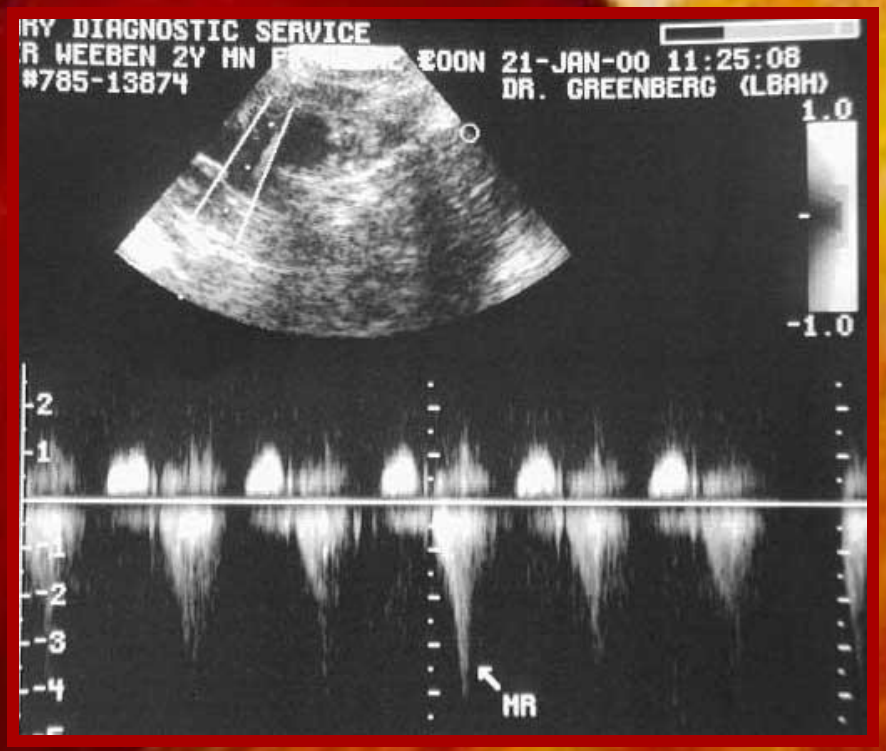


Normal LA:AO for dogs and cats: <1.5-1.5



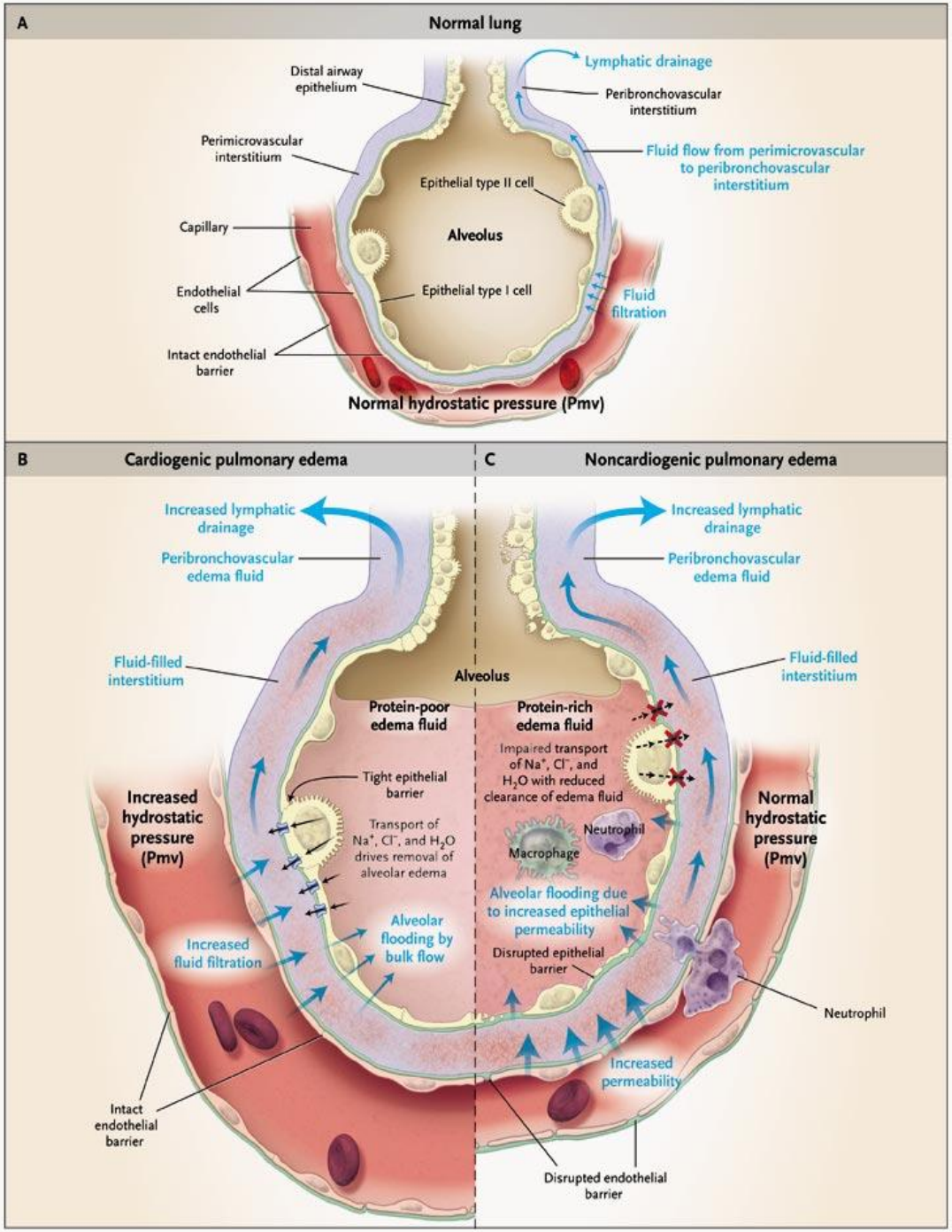
CHF: Echocardiography





Normal velocity across AV valves:
<2m/s
Velocities associated with regurgitation:
5-6m/s

Modified Bernouille Equation
$P=4V^2$



Pulmonary Fluid Analysis

Protein content of pulmonary fluid is < 0.5 that of peripheral blood

CHF: Emergent Treatment

- Improve oxygenation
  - Oxygen supplementation
  - Resolve pulmonary edema
- Improve CO

CHF: Emergent Treatment

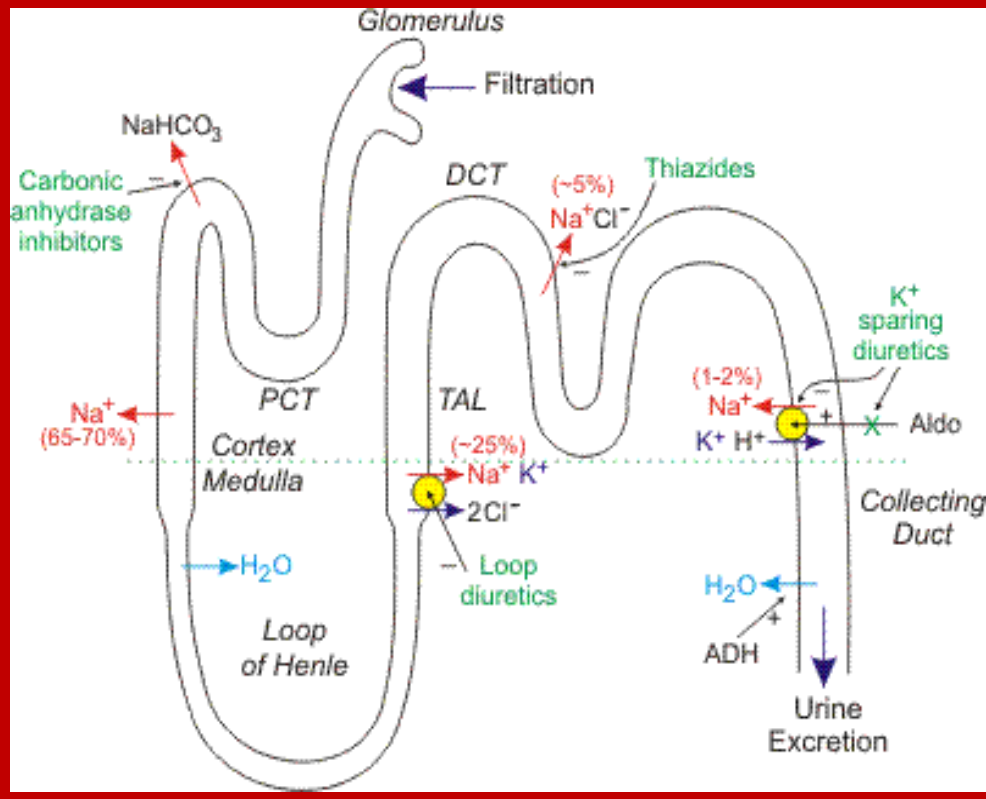
- Minimize stress
- Oxygen therapy
- Diuretics
- Vasodilators

Table 2
Approximate FiO_2 levels for different methods of oxygen administration

Method of oxygen administration	Approximate inspired oxygen concentration (FiO_2)
O_2 cage	40%
Nasal cannula (unilateral)	40–50%
Nasal cannula (bilateral)	50–60%
Tight-fitting face mask	70–90%
Oxygen tent	60–70%
Intubation	100%

CHF: Emergent Treatment

- Diuretics
  - Furosemide
    - 2-4mg/Kg IM, IV, SQ
    - CRI 0.7-1mg/kg/hr
- Vasodilator
  - Nitroglycerin paste
    - ¼-1in q8hr
    - V > A

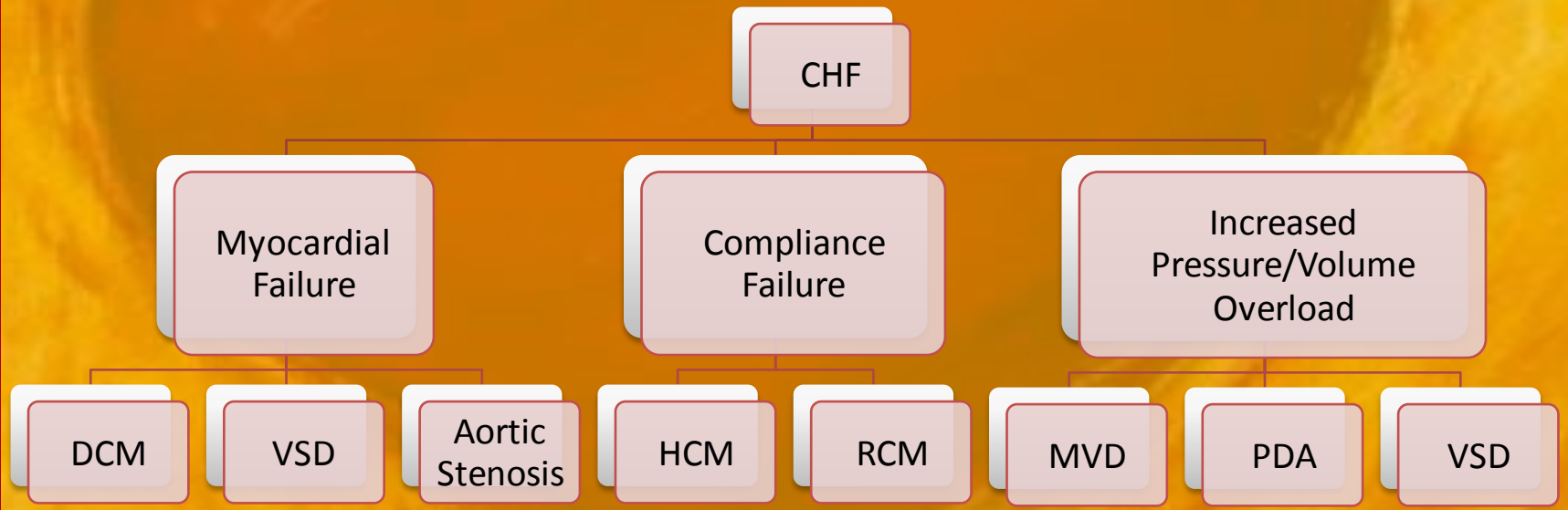


Congestive Heart Failure
Take 2

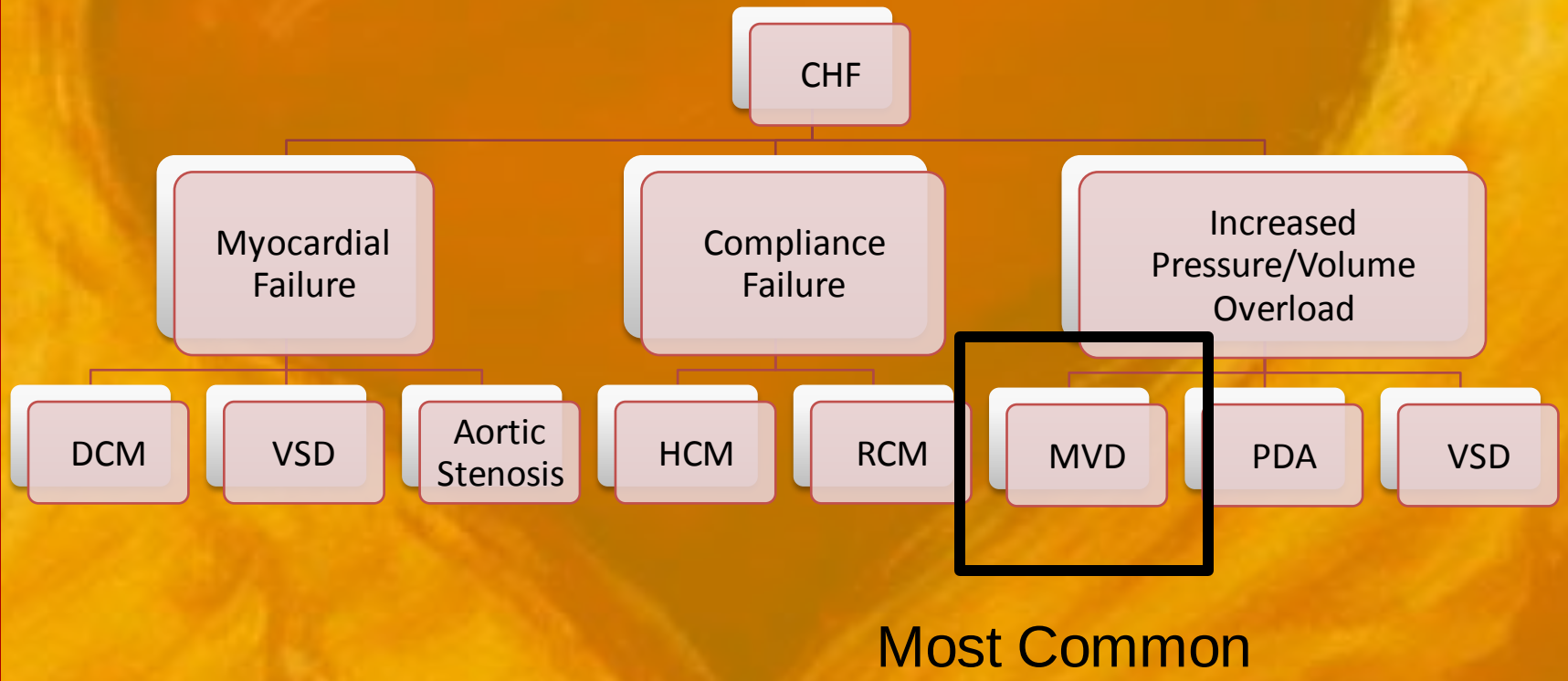
By:

Maureen Luschini

Congestive Heart Failure: Etiology



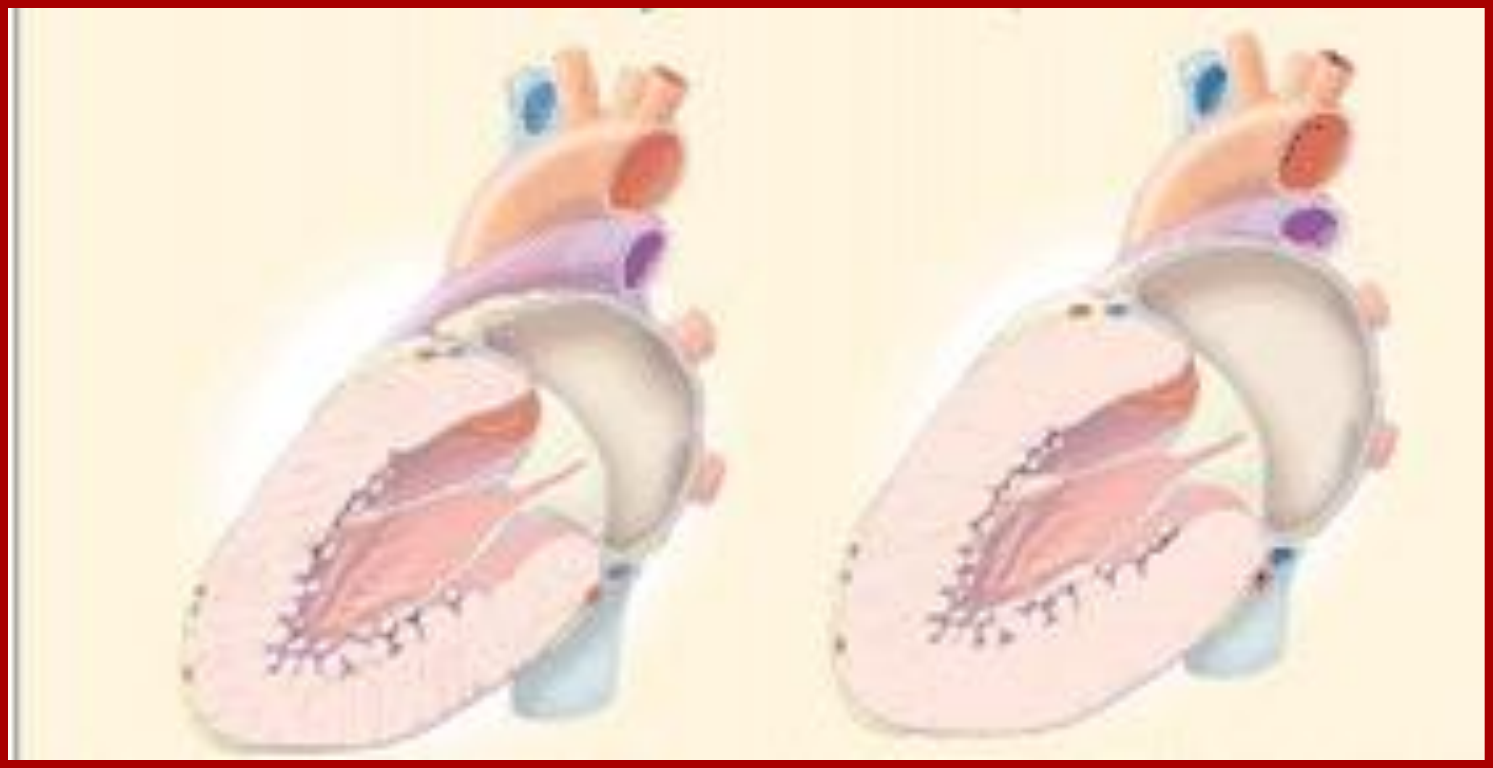
Congestive Heart Failure: Etiology



Mitral Valve Insufficiency

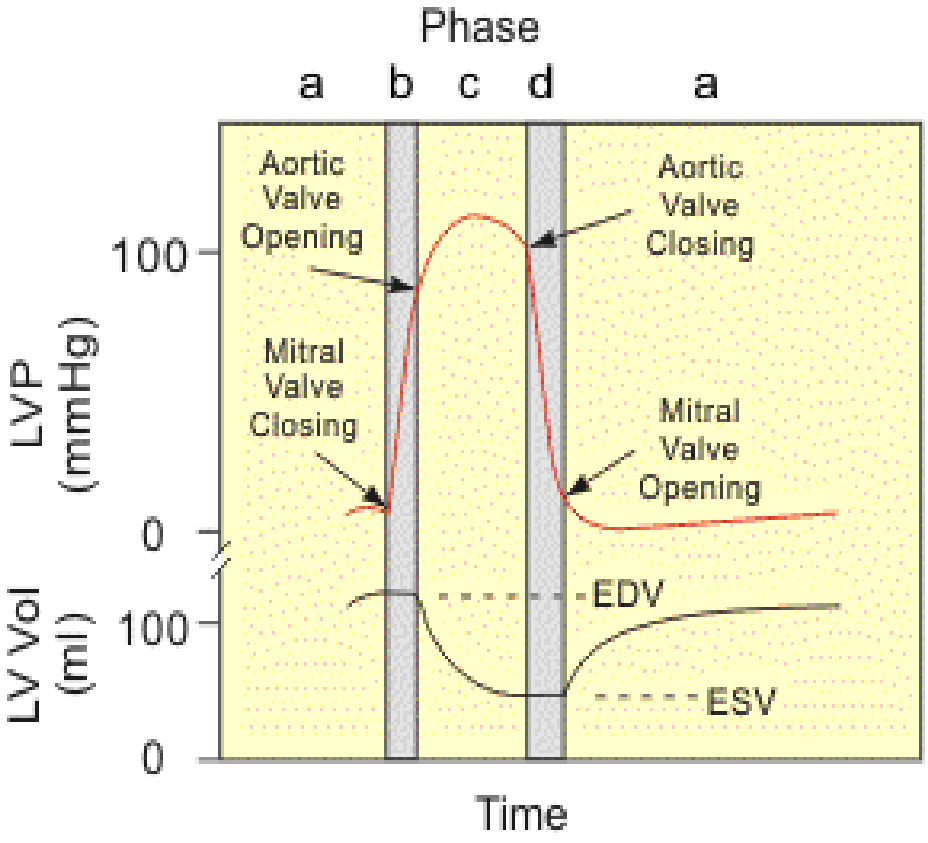
- Small toy breed dogs
- Males>females
- Cause: endocardiosis
  - Degeneration with MPS deposition
- Progesses with age
- Hx: cough, exercise intolerance, syncope, anorexia/weight loss
- CS: left systolic apical murmur, +/- arrhythmia

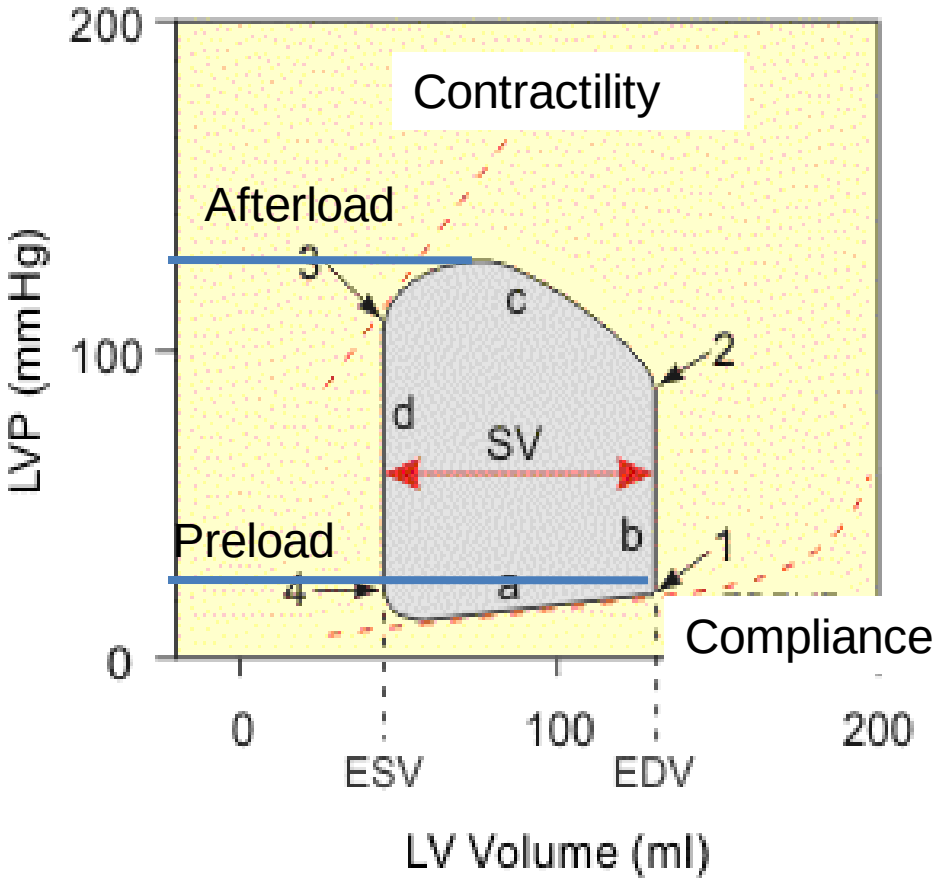
Cardiac Remodeling in MVI



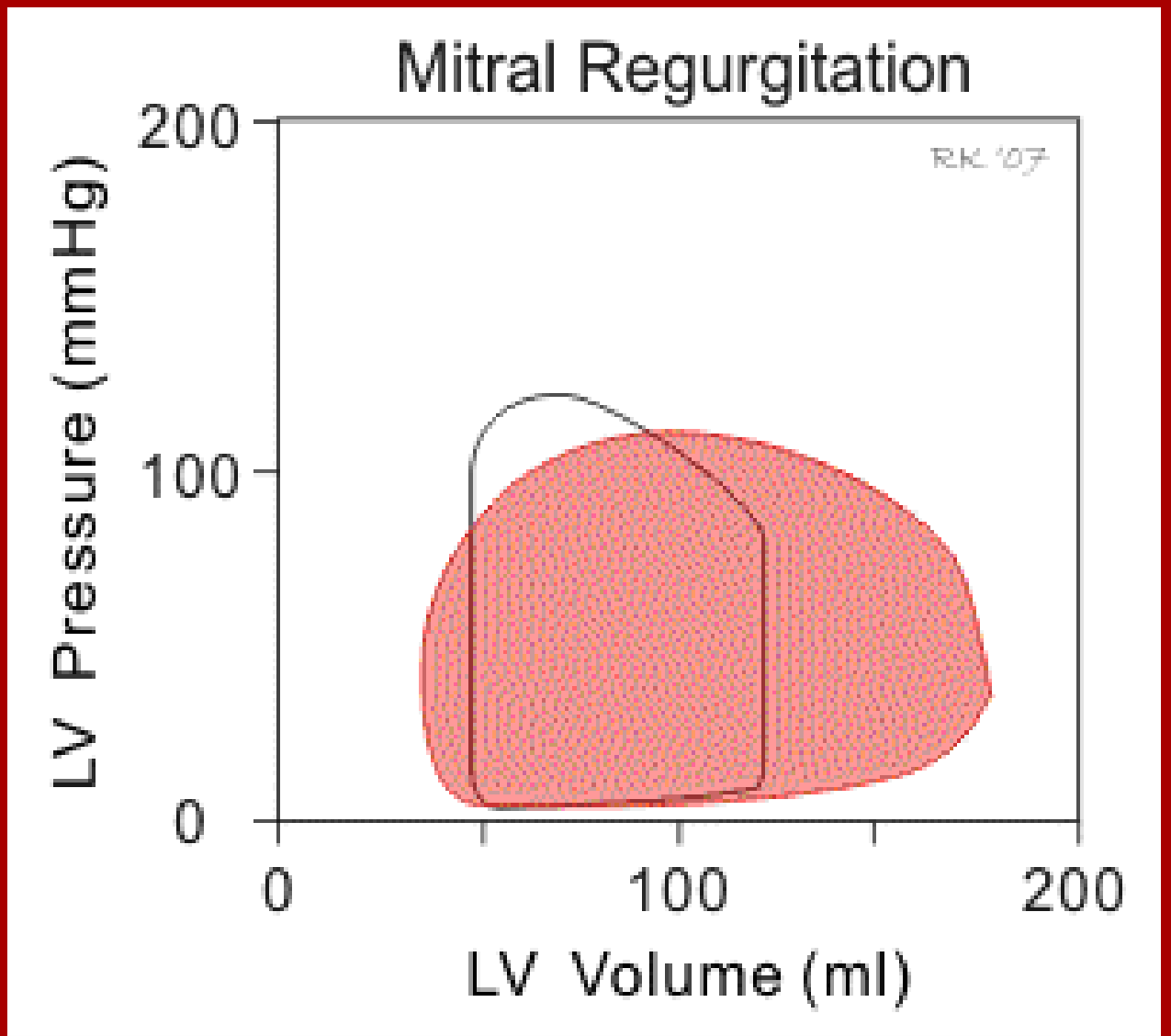
Compensatory volume overload (concentric) hypertrophy in LV and pressure overload (eccentric) hypertrophy in LA

Normal Cardiac Physiology

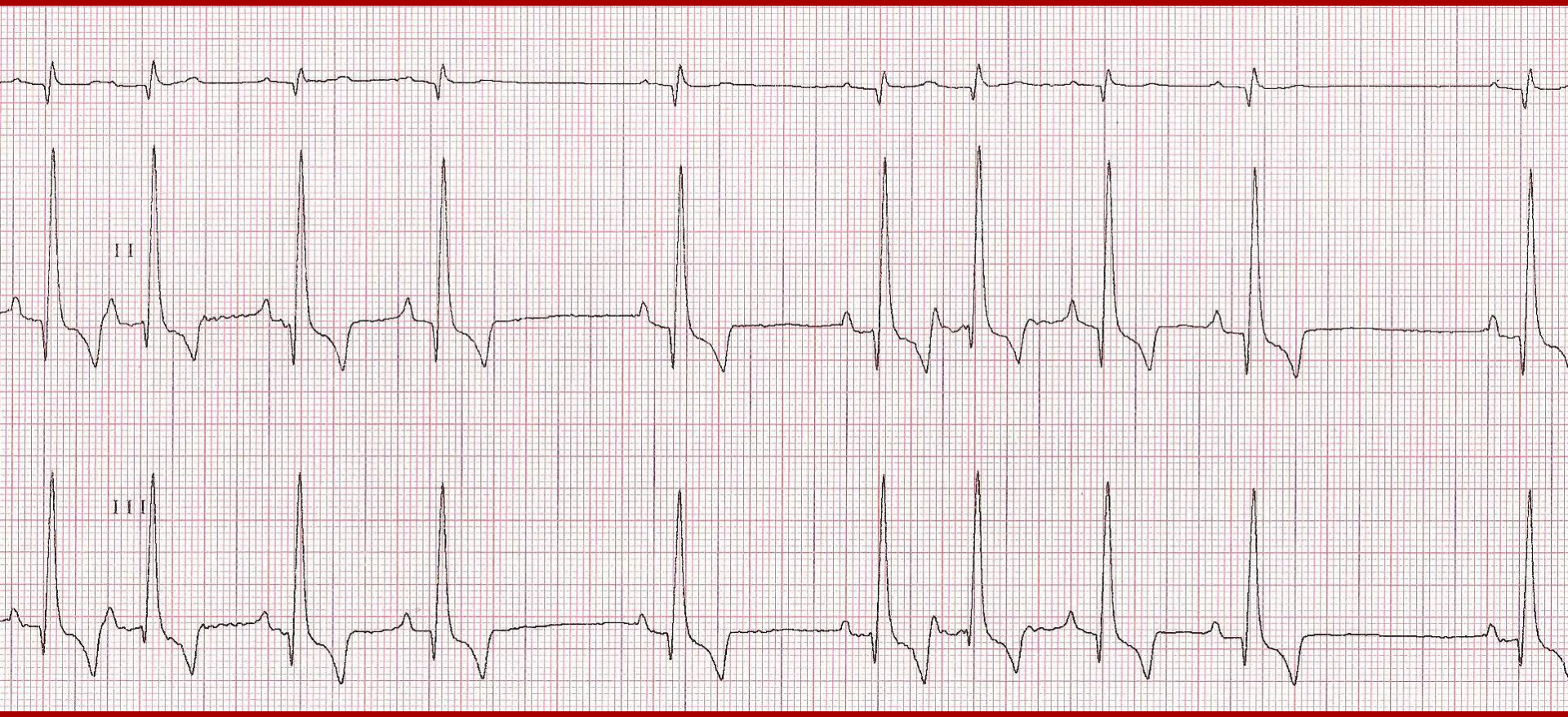




Uncompensated MVI



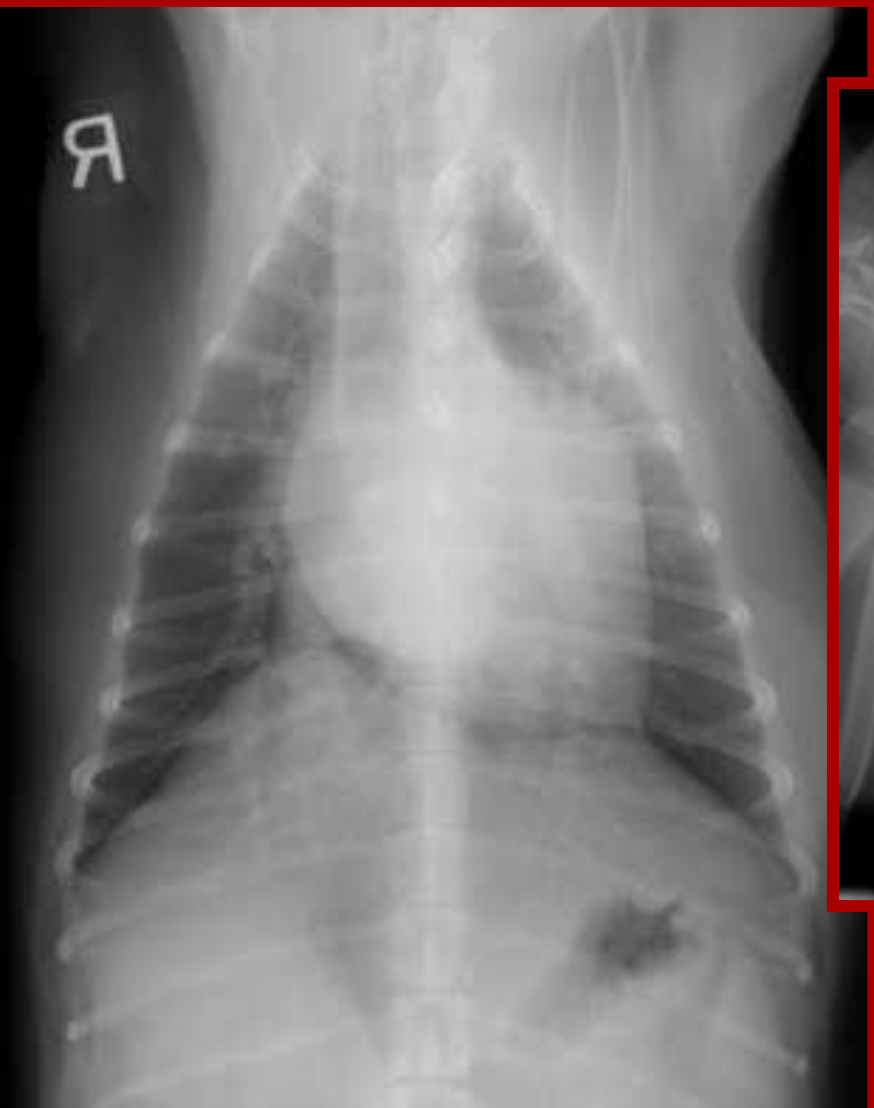
ECG Findings in MVI

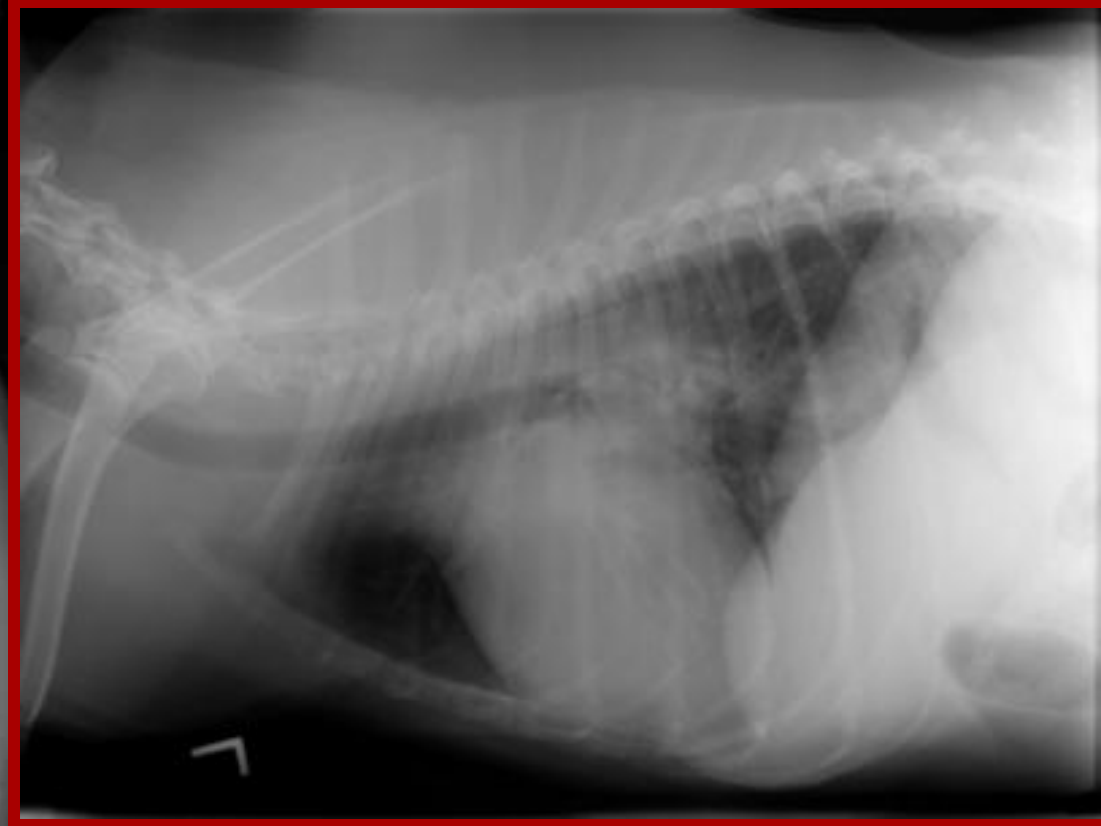


LA enlargement: wide p wave

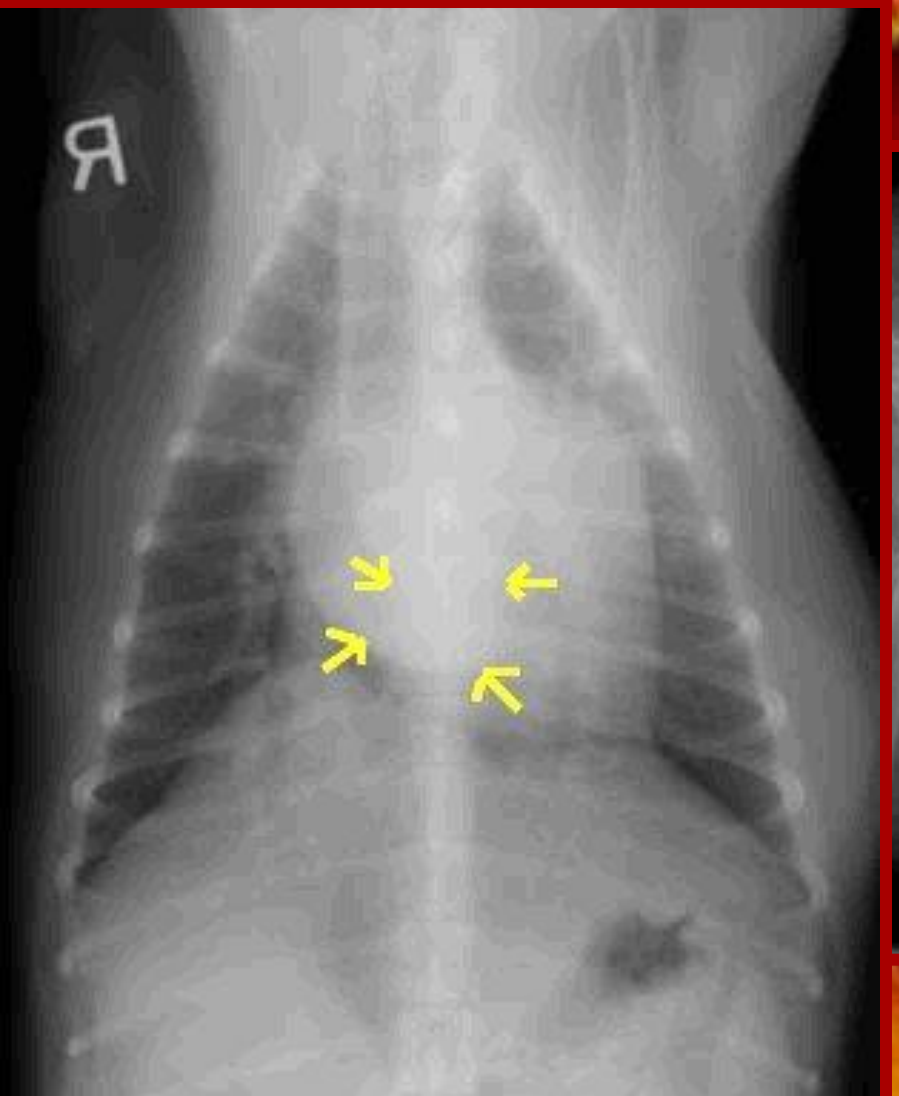
LV enlargement: tall R, wide QRS, left axis shift

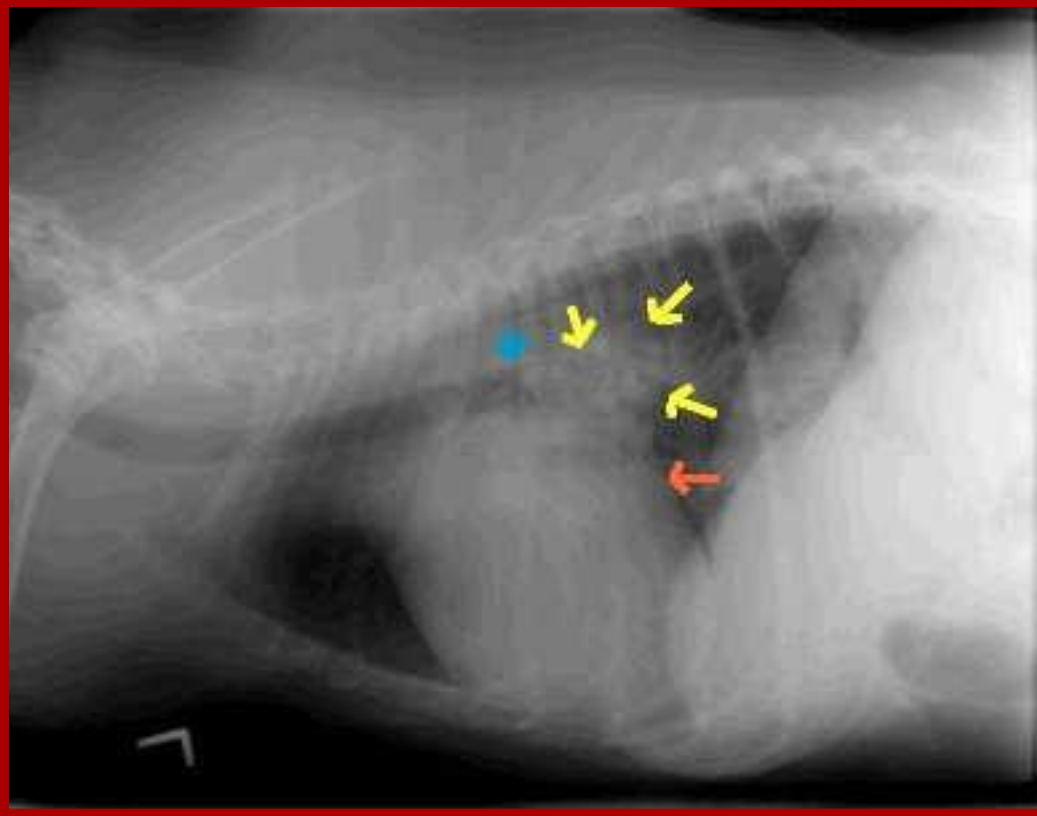
Radiographic Findings



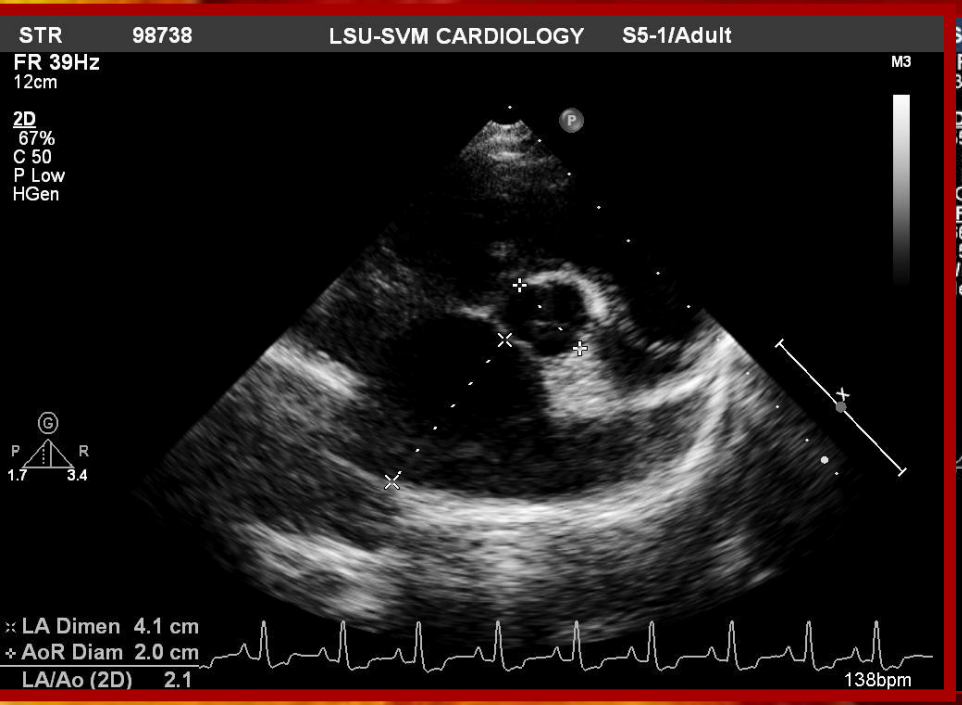


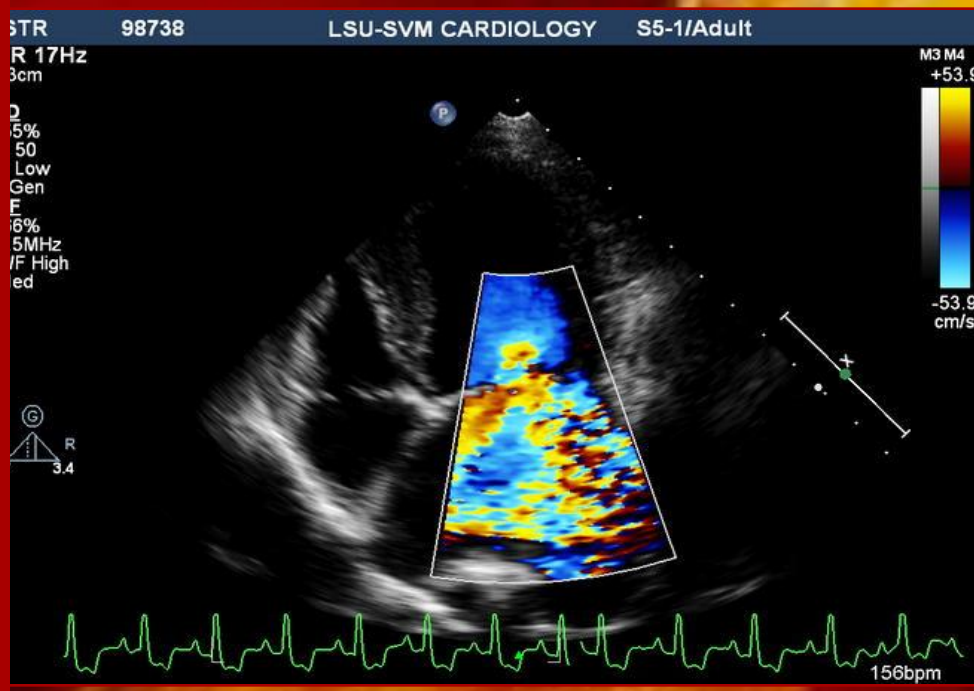
Radiographic Findings





Echocardiographic Findings of MVI

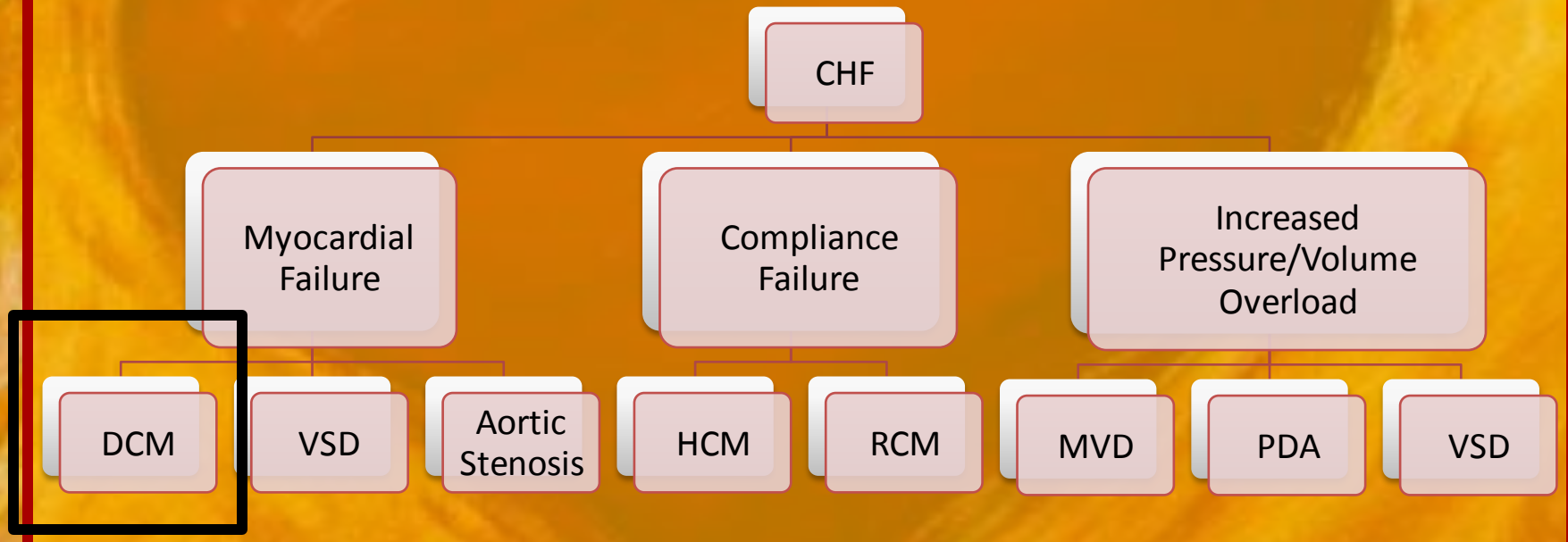




MVI CHF Treatment

- Furosemide, O_2 therapy
- ACE inhibitor
- +/- Pimobendan
- Manage arrhythmias

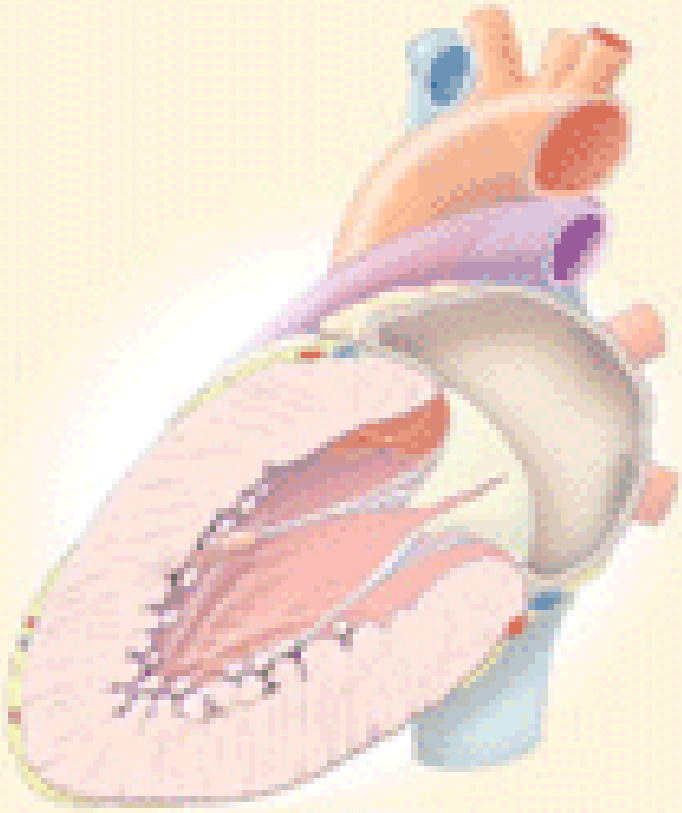
Congestive Heart Failure



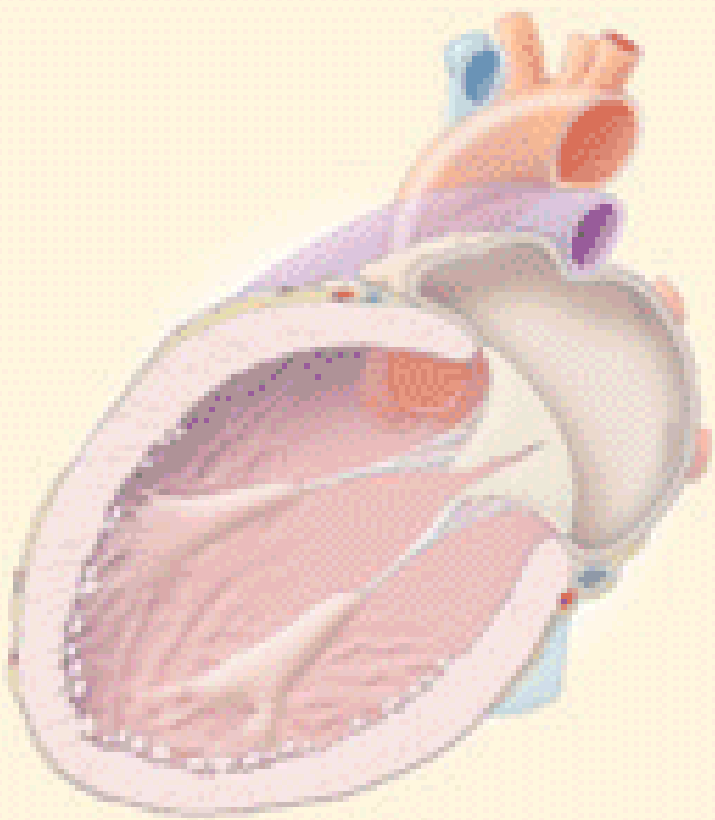
Dilated Cardiomyopathy

- Primary myocardial disorder
- Reduced contractility and ventricular dilation
- Genetic factors, tachycardia, taurine/carnitine deficiency, toxins, immunologic, viral
- Purebreeds>Mixed
  - Dobies, Boxers, PWD
  - ACSp, GRet, Labs, IWH, SB
- Males>females
- Adults
  - PWD: young

Cardiac Remodeling

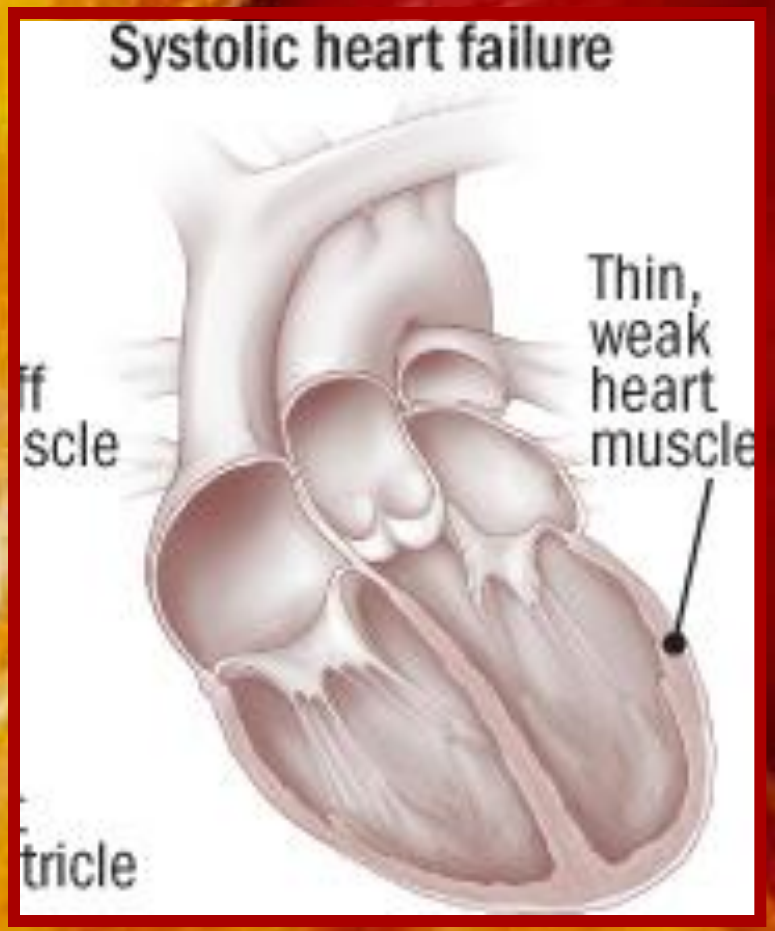


Normal heart



Dilated heart
(systolic heart failure)

Dilated Cardiomyopathy



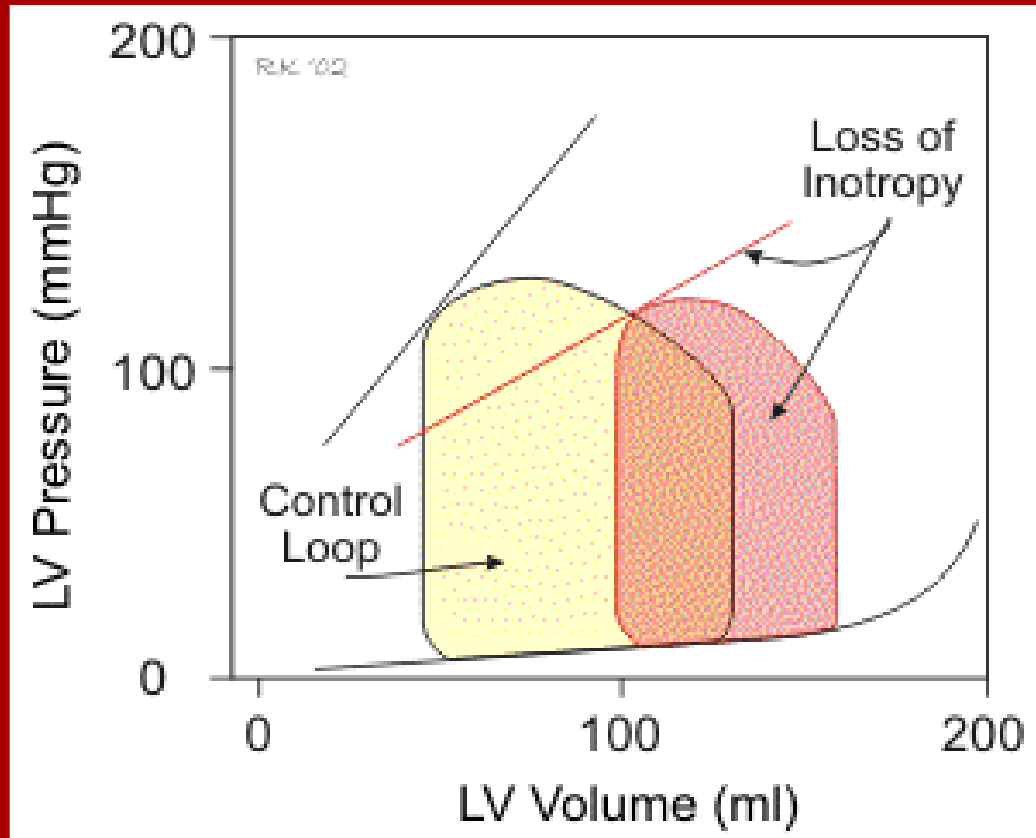


Figure 2. Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

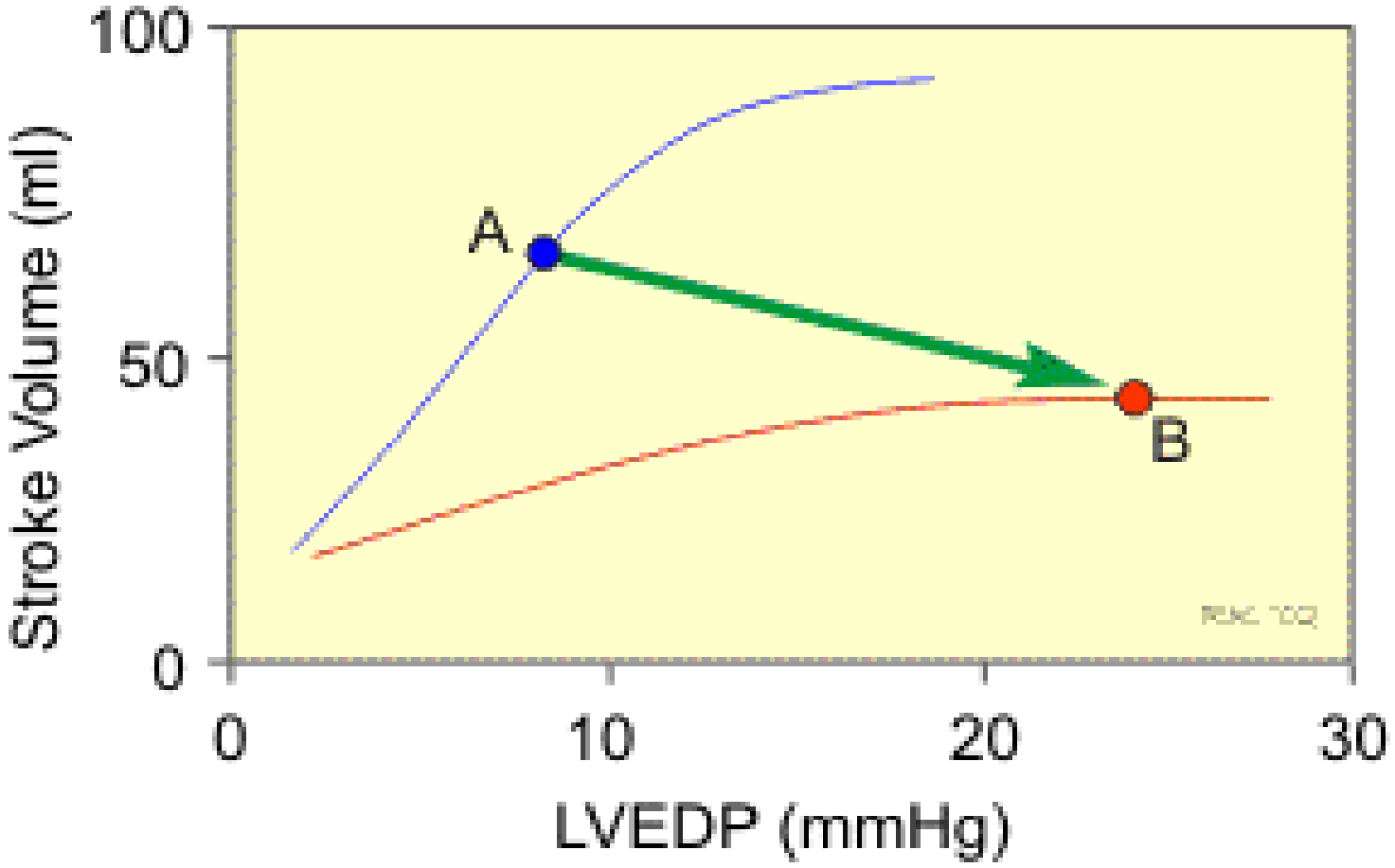


Figure 1. The Frank-Starling relationship showing the effects of heart failure (ventricular systolic dysfunction, loss of inotropy) on stroke volume and ventricular preload (left ventricular end-diastolic pressure, LVEDP). Point A, control point; point B, ventricular failure.

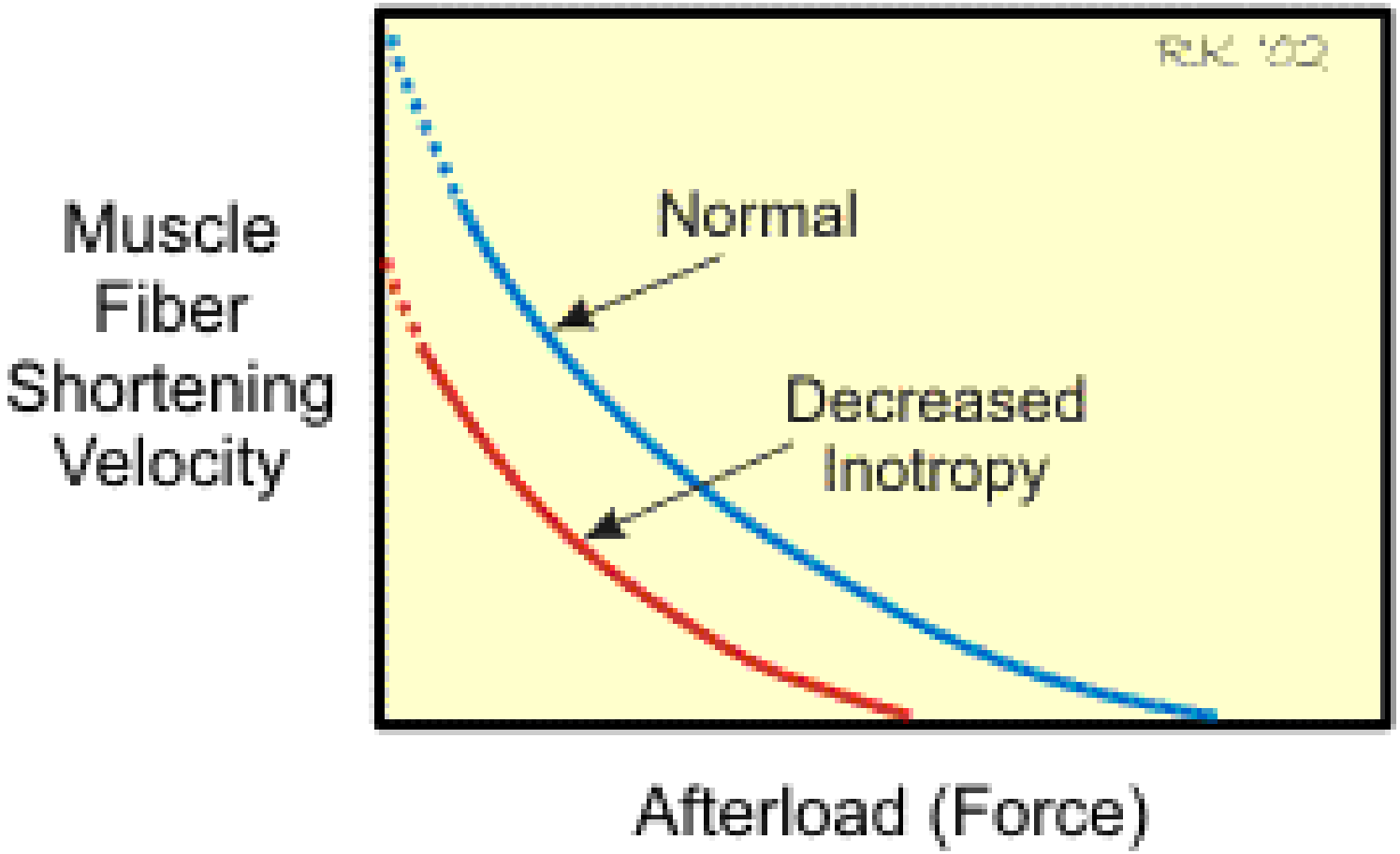
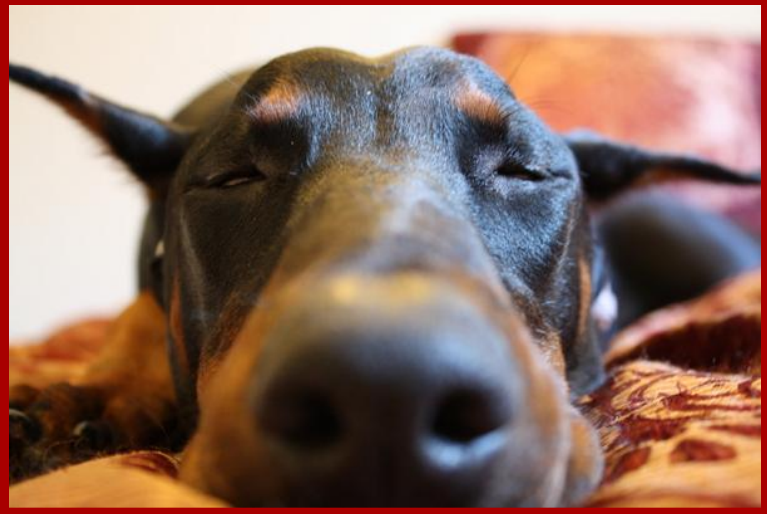


Figure 3. Effects of ventricular failure (decreased inotropy) on the force-velocity relationship. Decreased inotropy decreases velocity of fiber shortening at any given afterload.

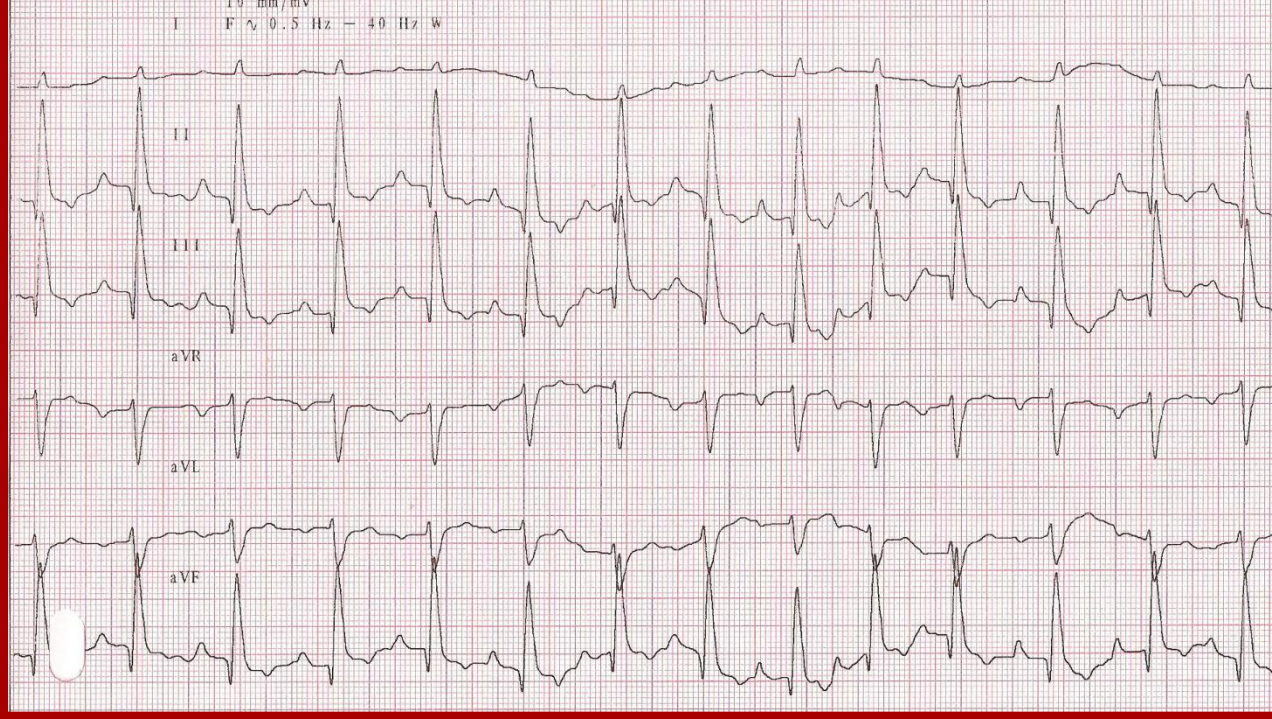
Clinical Signs

- Cachexia, weakness, lethargy, dyspnea
- Sinus tach, A-fib, VPC's
- +/- gallop rhythm
- Murmurs (MVI, TVI)
- +/- jugular distension
- +/- abdominal fluid wave

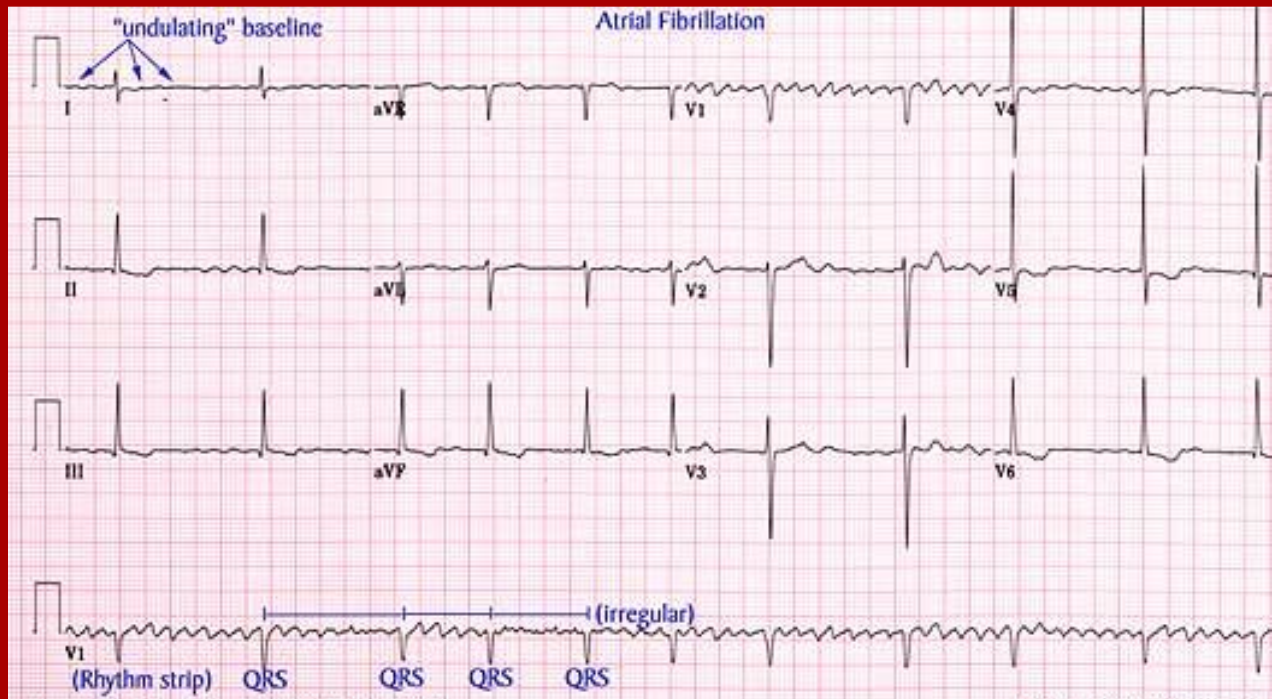


ECG Findings

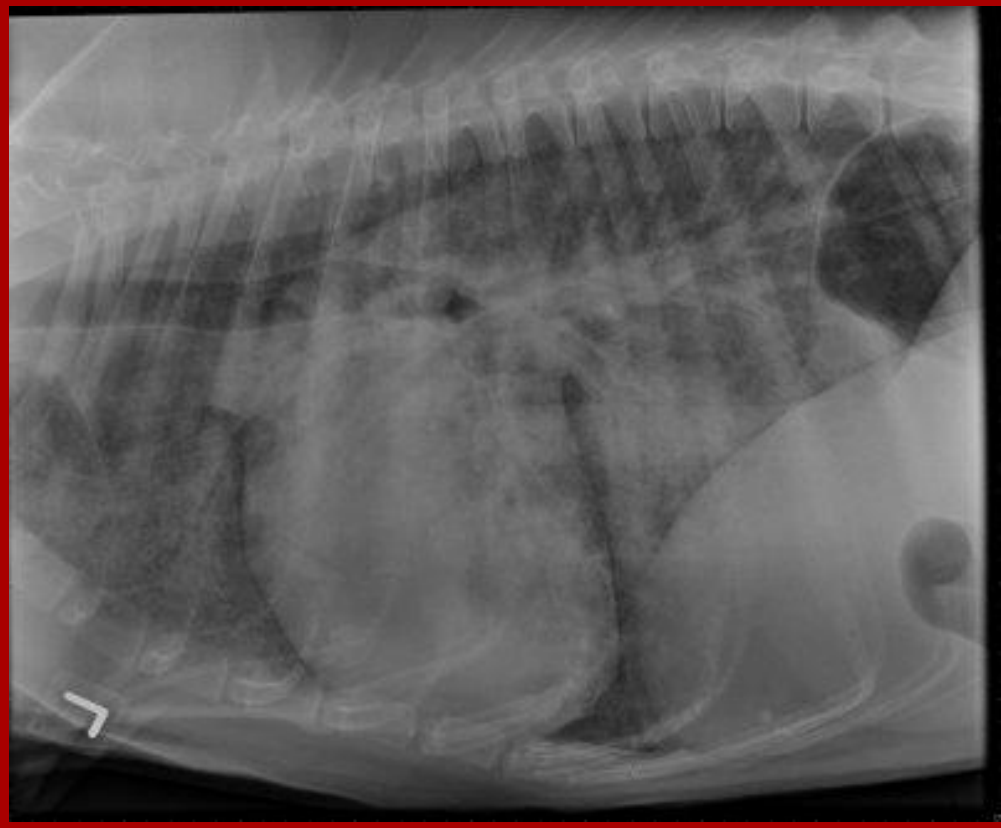
SVT with LBBB



A-fib

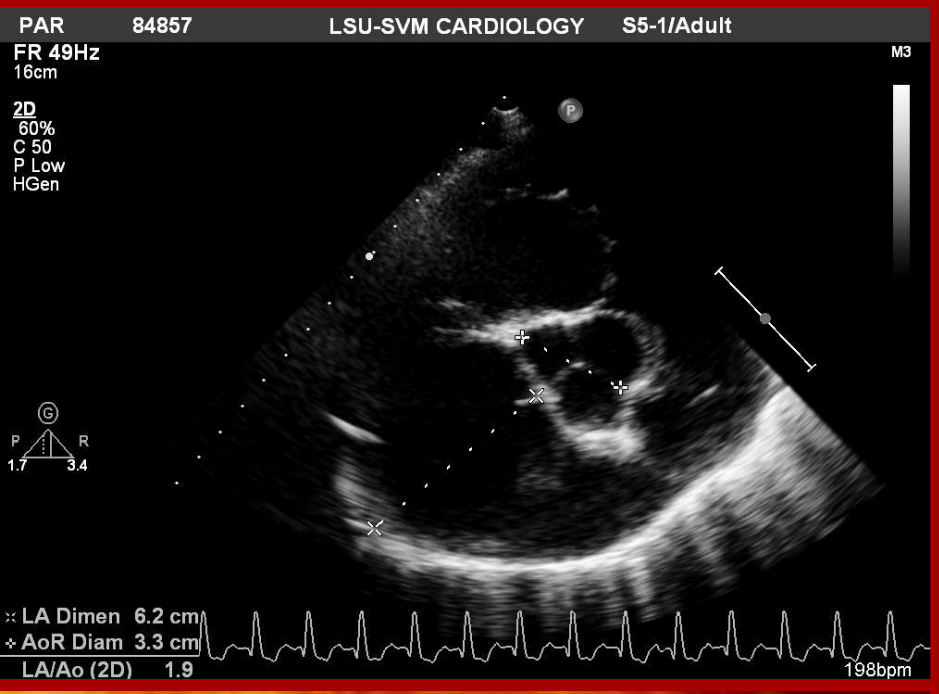


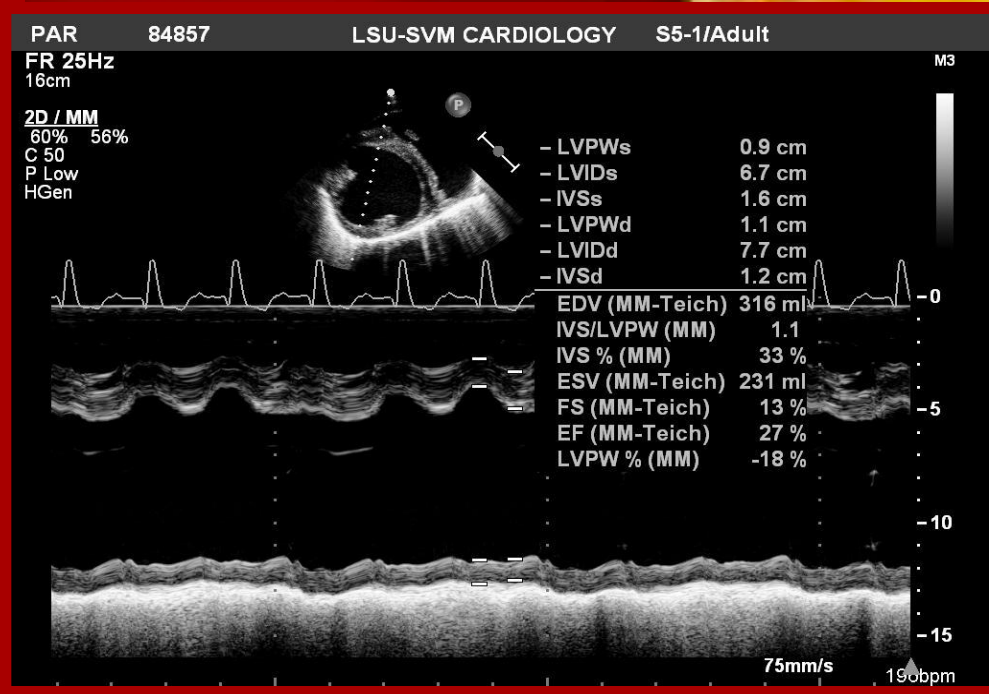
Radiographic Findings



+/- pleural effusion

Echocardiographic Findings





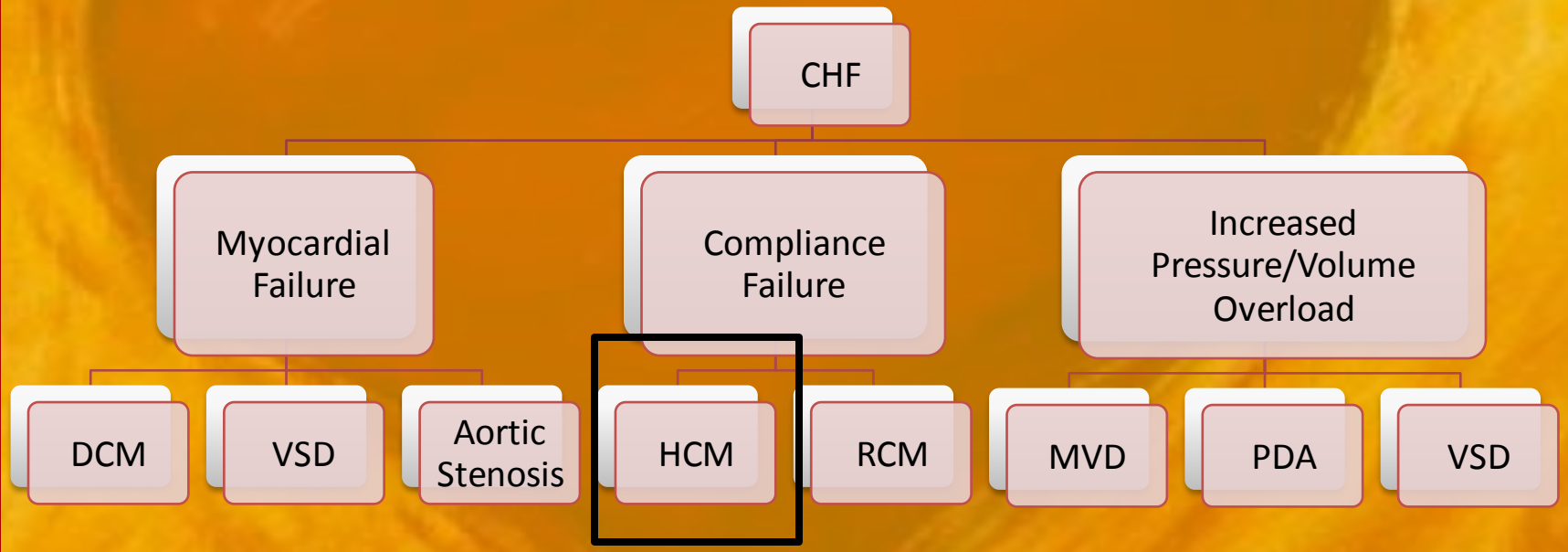
Echo: gold standard for dx (decreased FS)

Normal FS: 15-25%

DCM CHF Treatment

- Furosemide, O_2 therapy
- ACE inhibitor
- Positive inotrope
  - Dobutamine Vs Pimo
- Preload reduction
  - NG ointment, SNP
- Manage arrhythmias
  - Atrial: diltiazem, β blockade, digoxin
  - Ventricular: lidocaine, procainamide, mexilitine

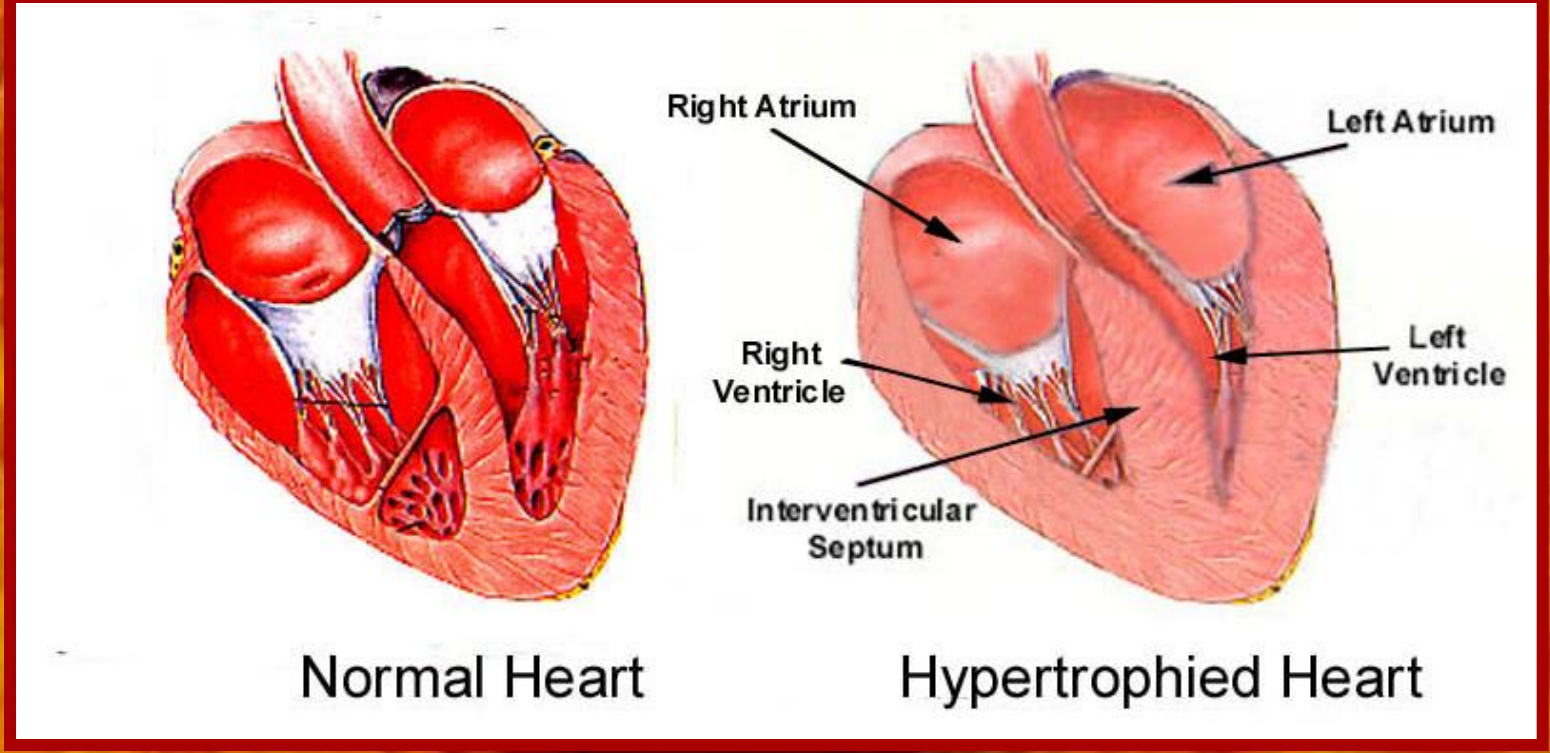
Congestive Heart Failure: Etiology



Hypertrophic Cardiomyopathy

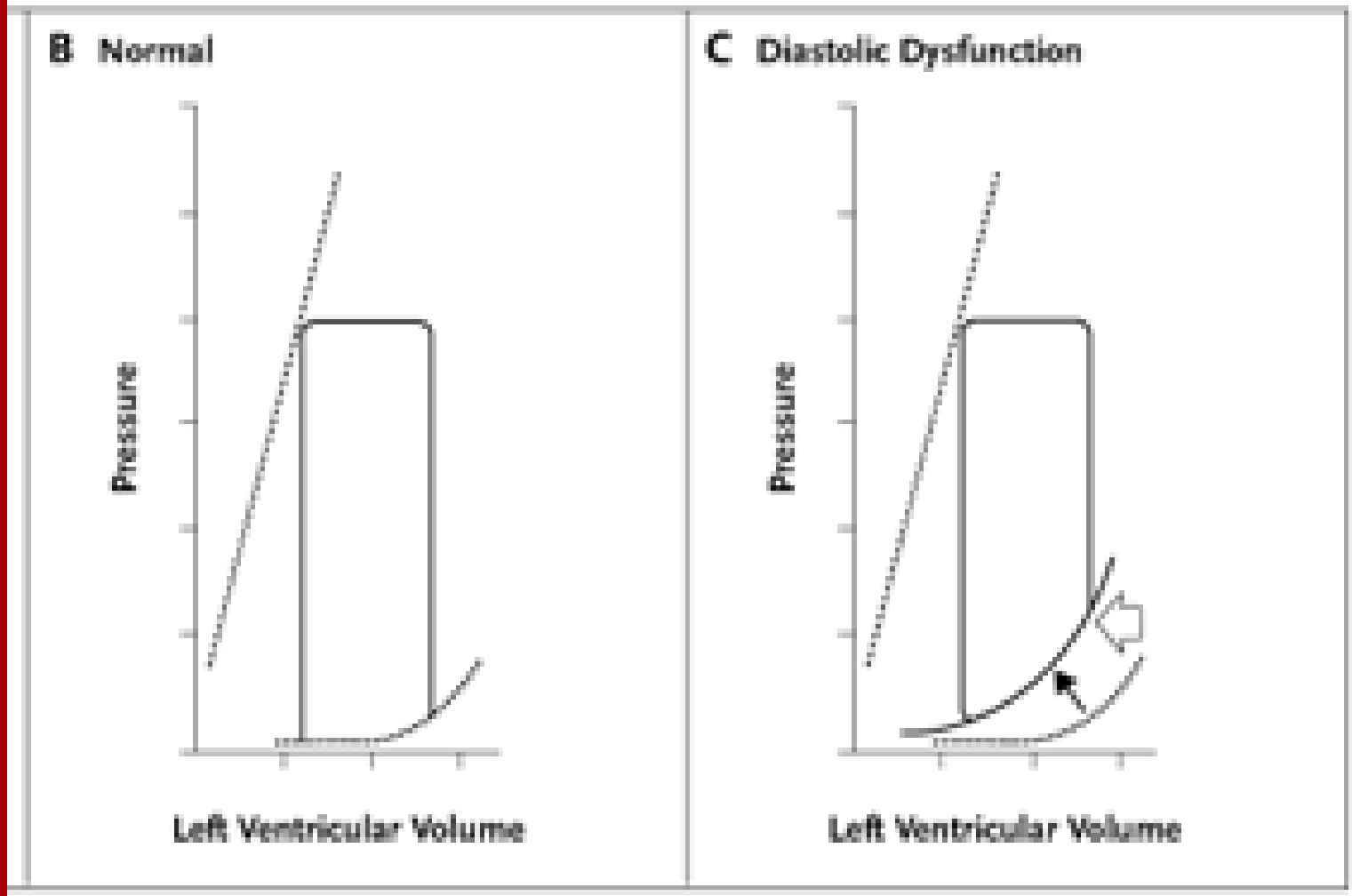
- Most common feline CV disease
- Idiopathic
  - Familial in Maine Coon cats
- Hypertension and hyperthyroidism need to be ruled out
- Middle aged males
- Hx: lethargy, inappetence, weight loss, hiding, syncope, respiratory distress, +/- posterior paralysis
  - Cough is rare
- CS: +/- murmur, gallop or arrhythmia,
- Sudden death!

Hypertrophic Cardiomyopathy

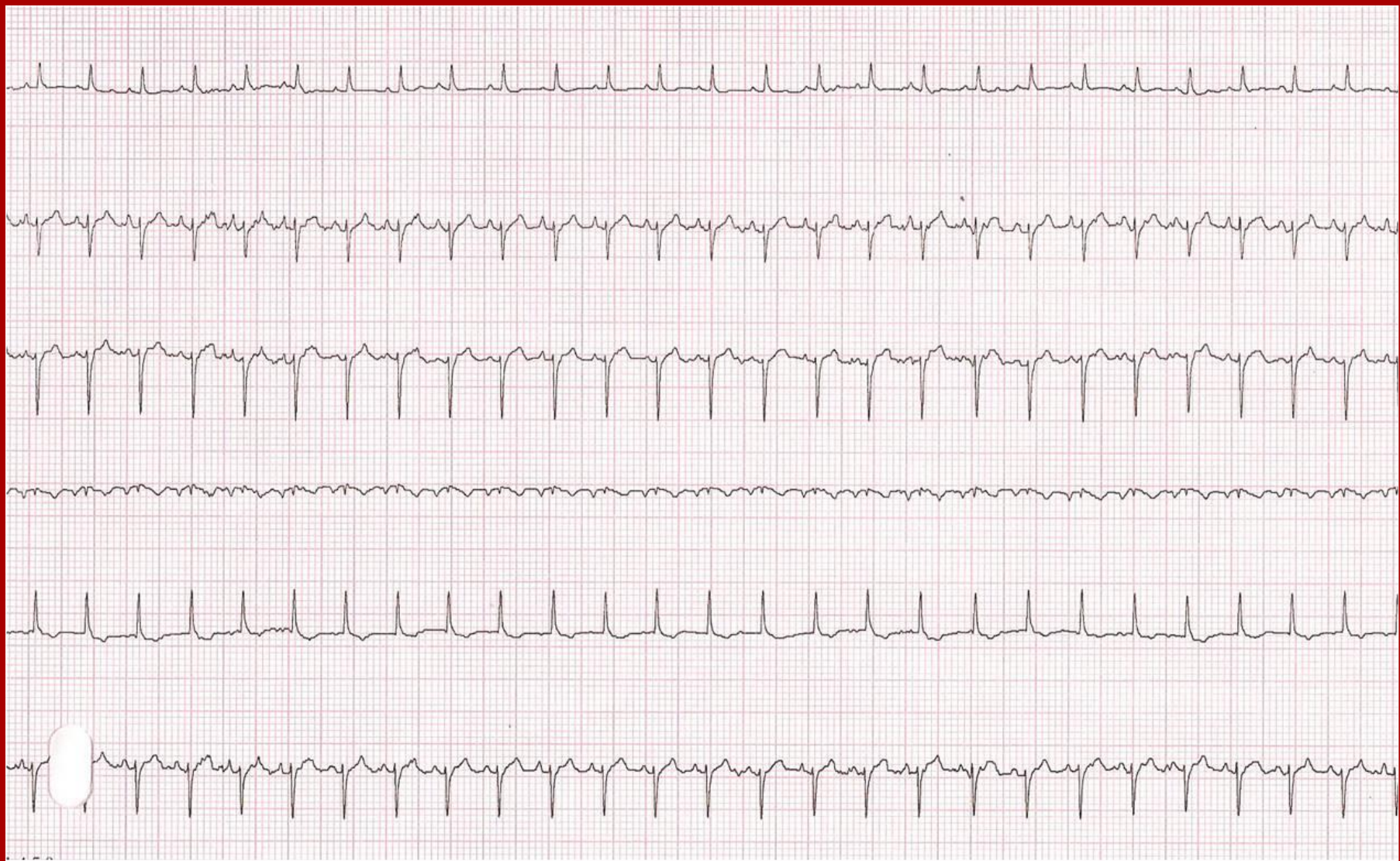


- Increased cardiac mass with hypertrophied, non-dilated LV
- +/- dynamic obstruction of LVOFT
- SAM may be present

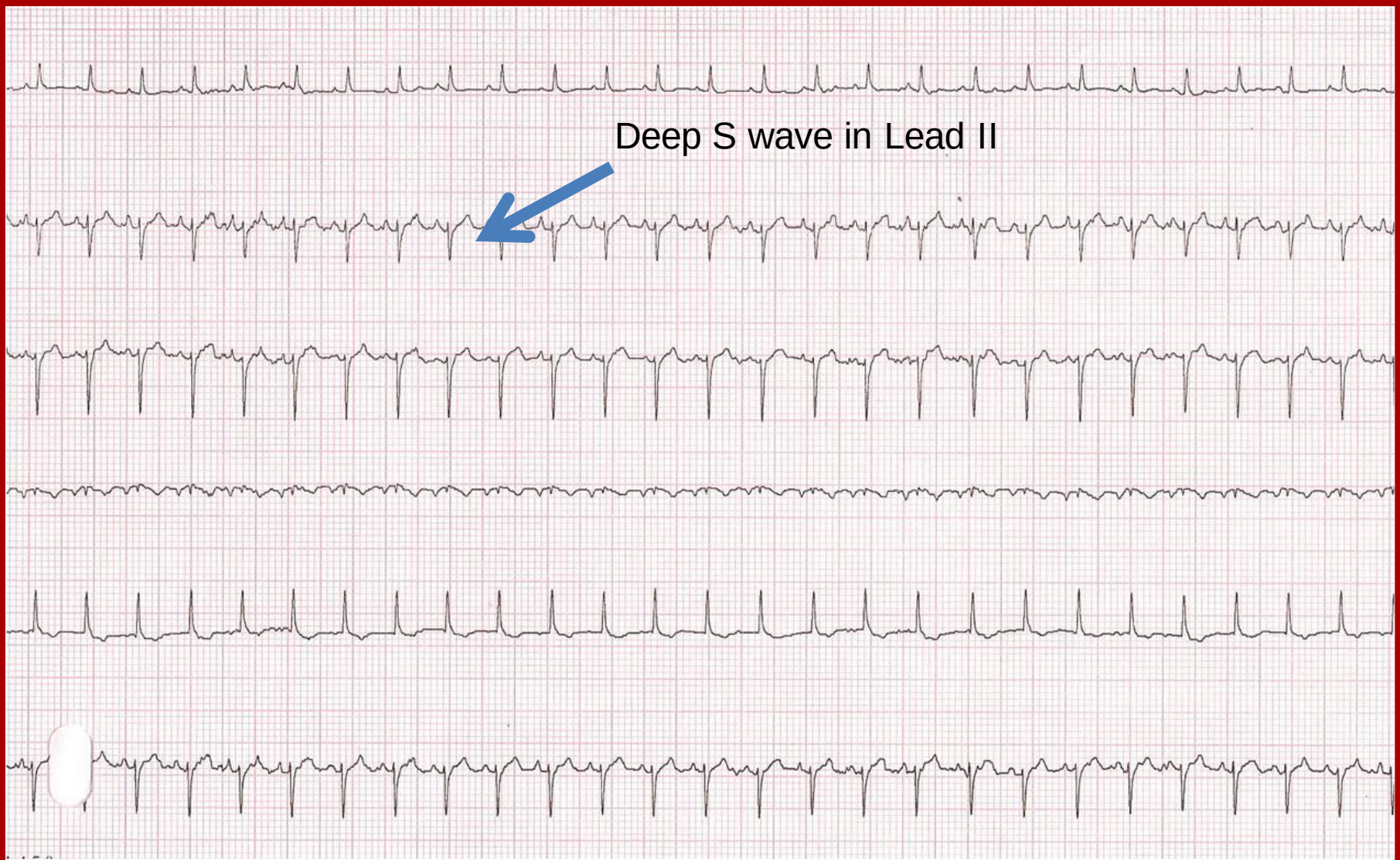
Hypertrophic Cardiomyopathy



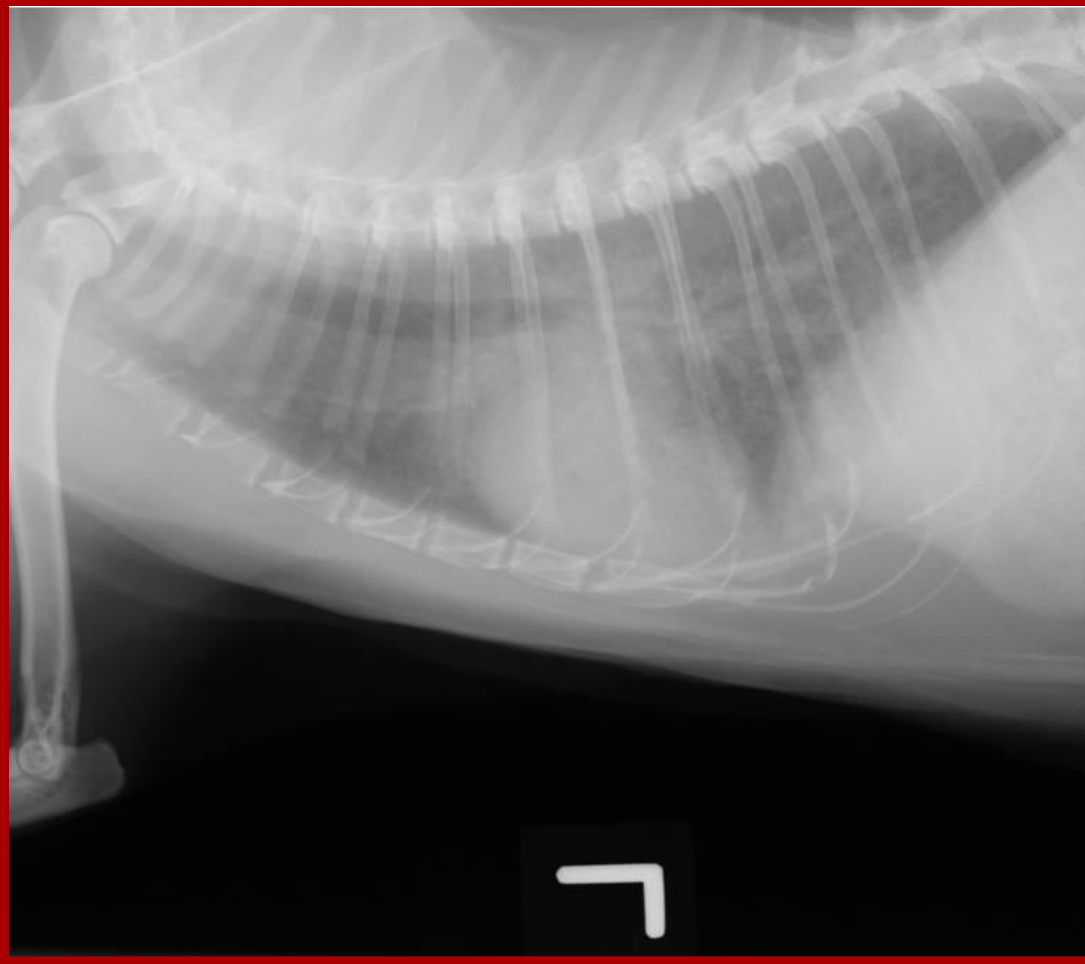
ECG Findings in HCM



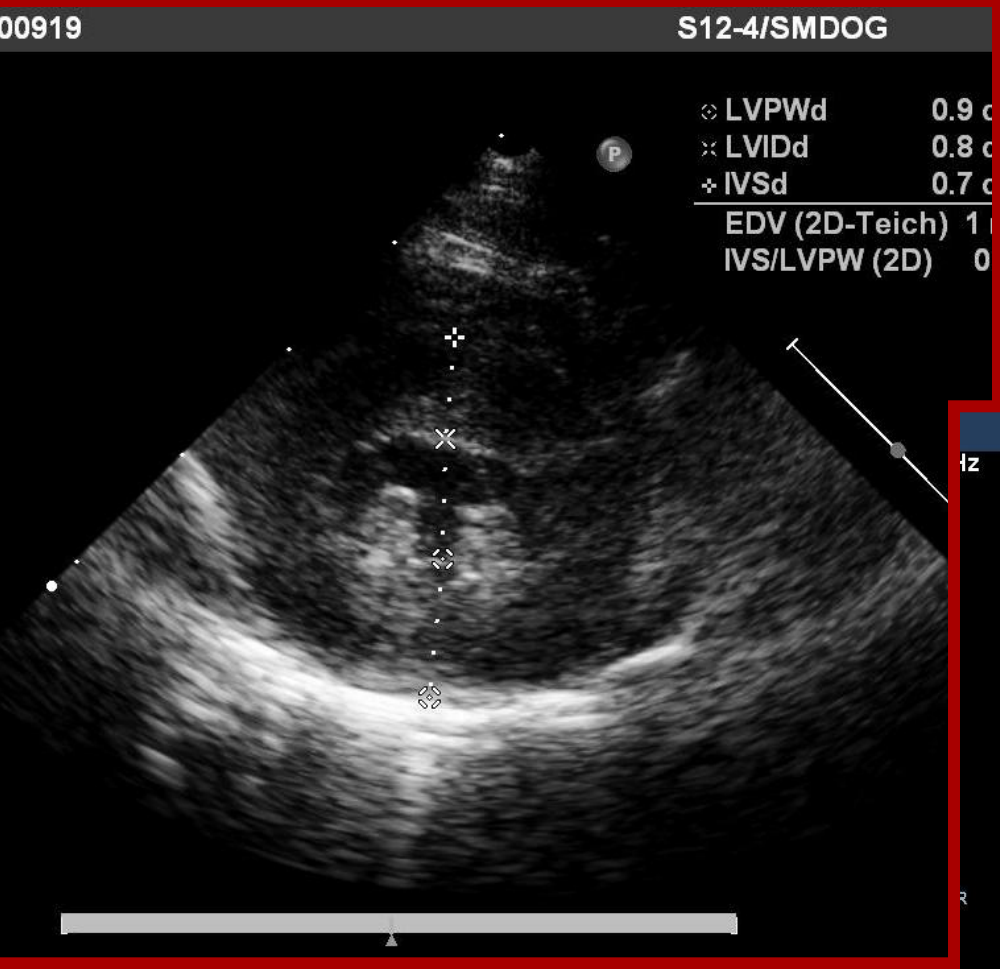
ECG Findings in HCM

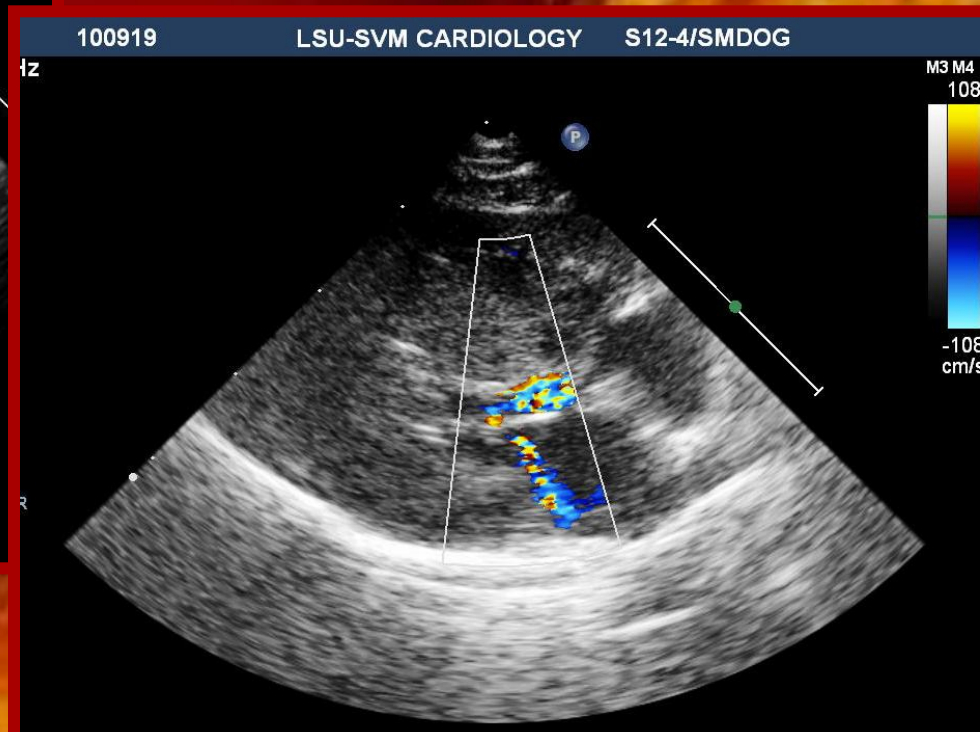


Radiographic Findings of HCM

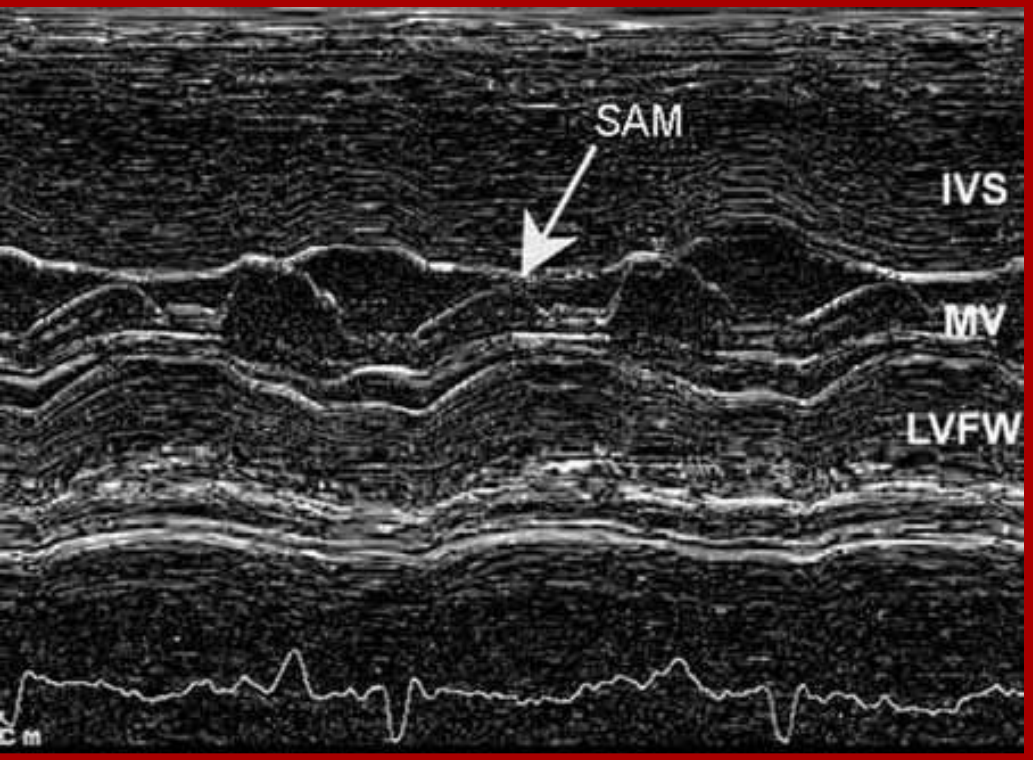


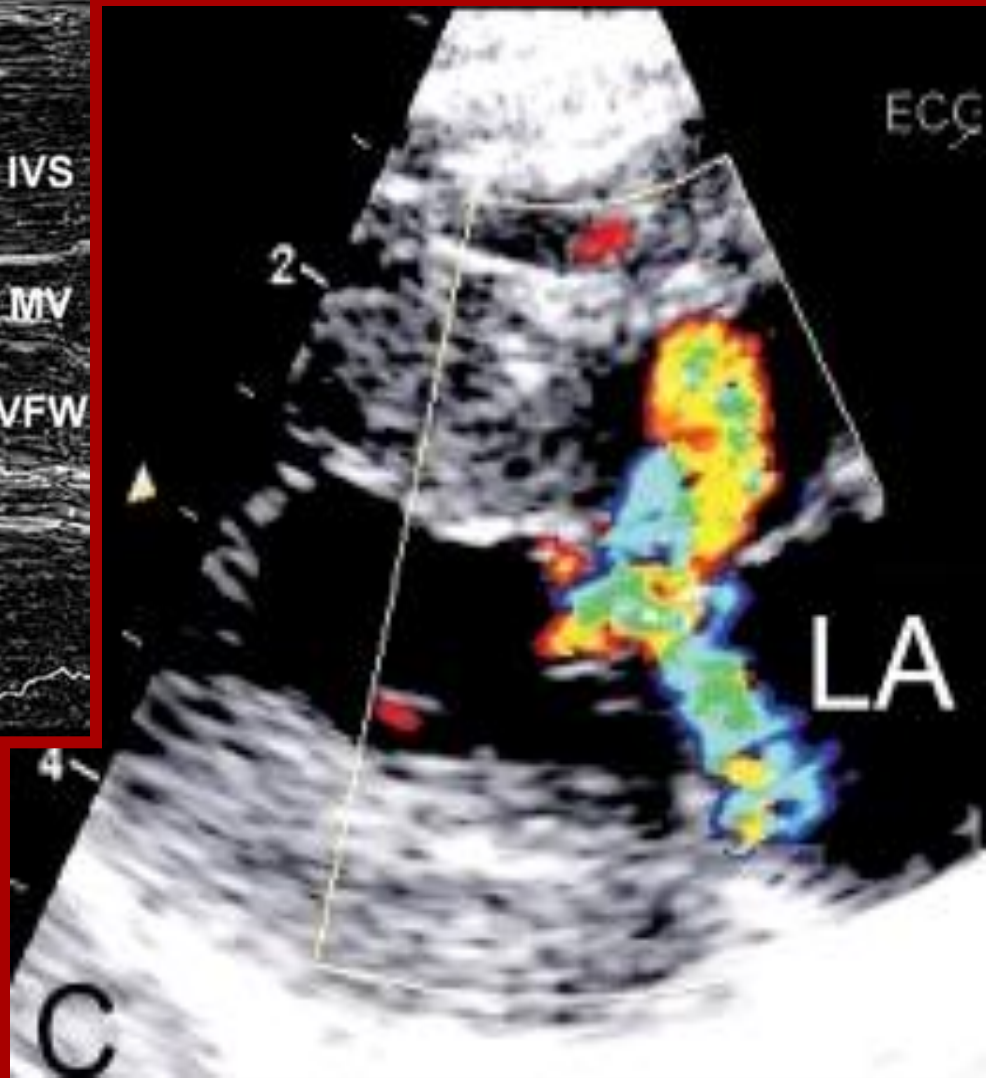
Echocardiographic Findings of HCM



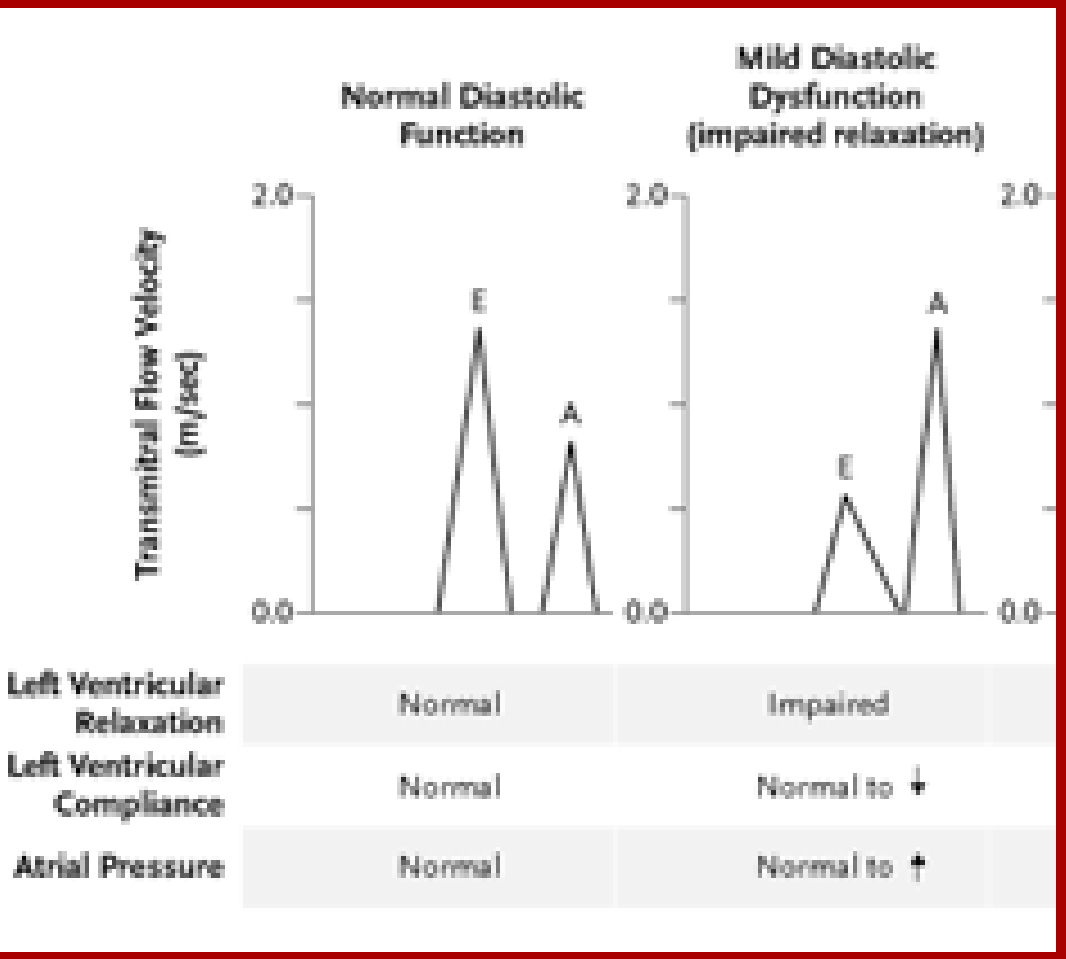


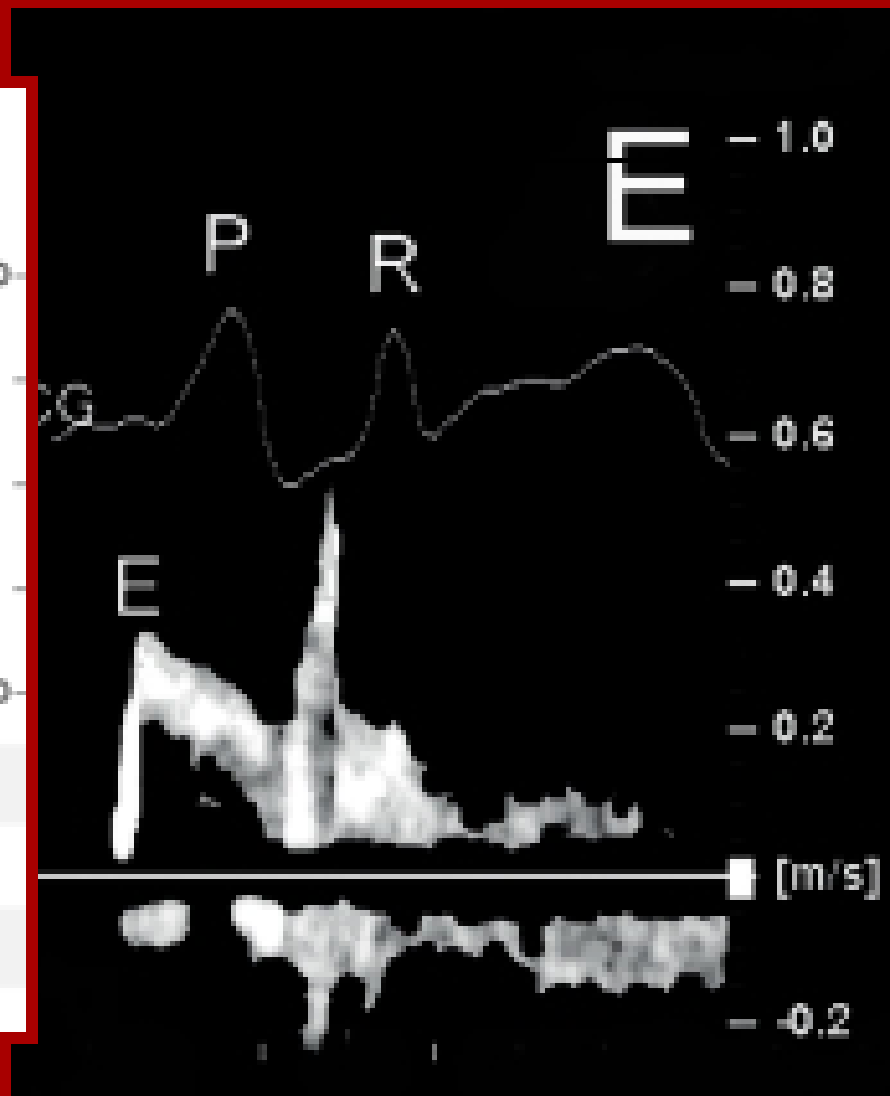
Echocardiographic Findings of HCM





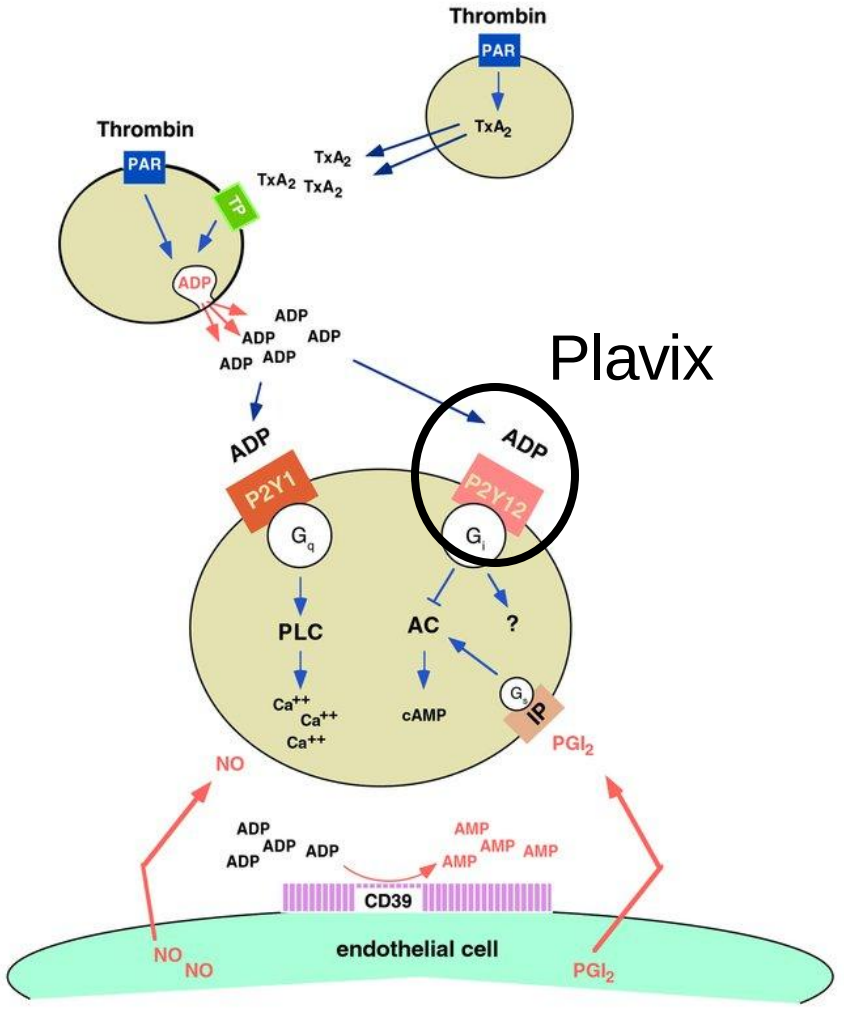
E to A Reversal with HCM



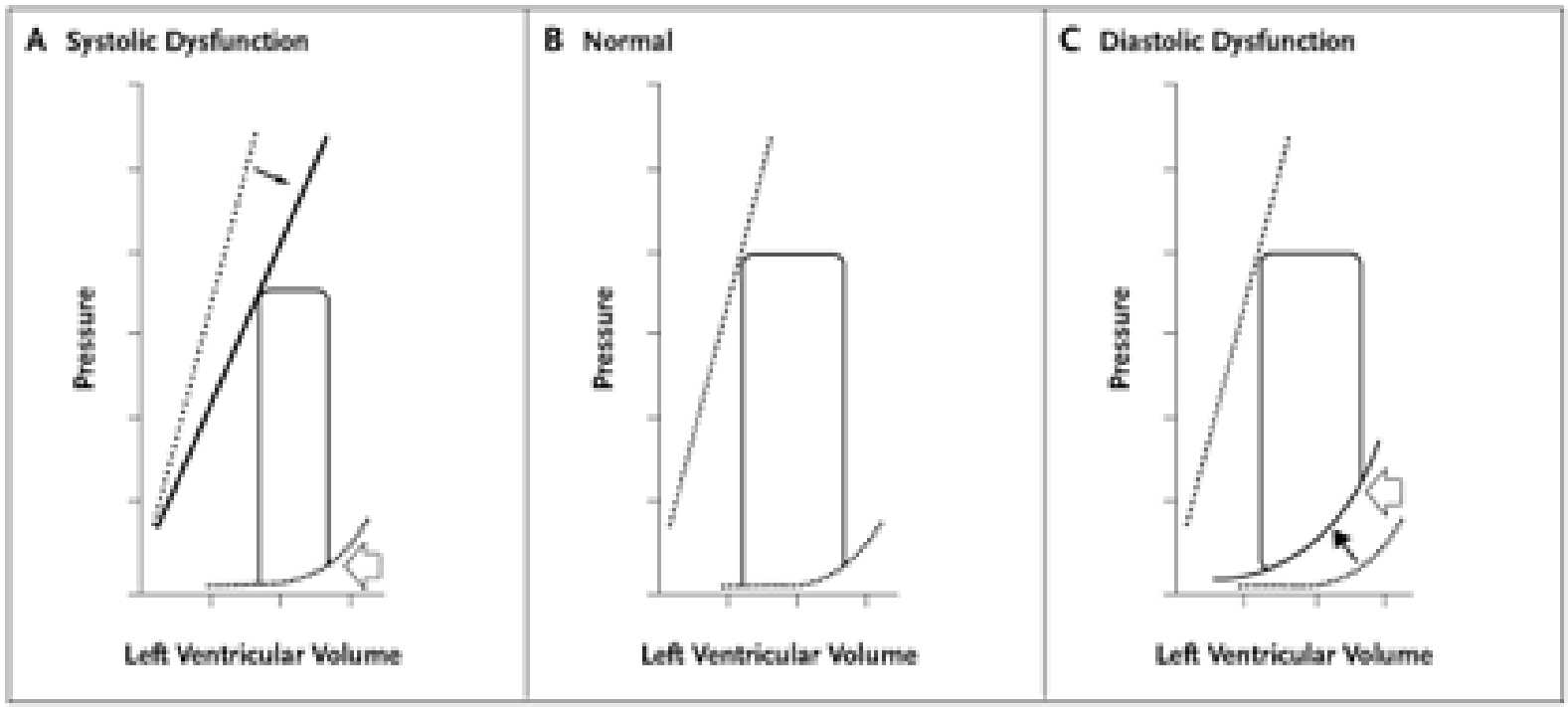


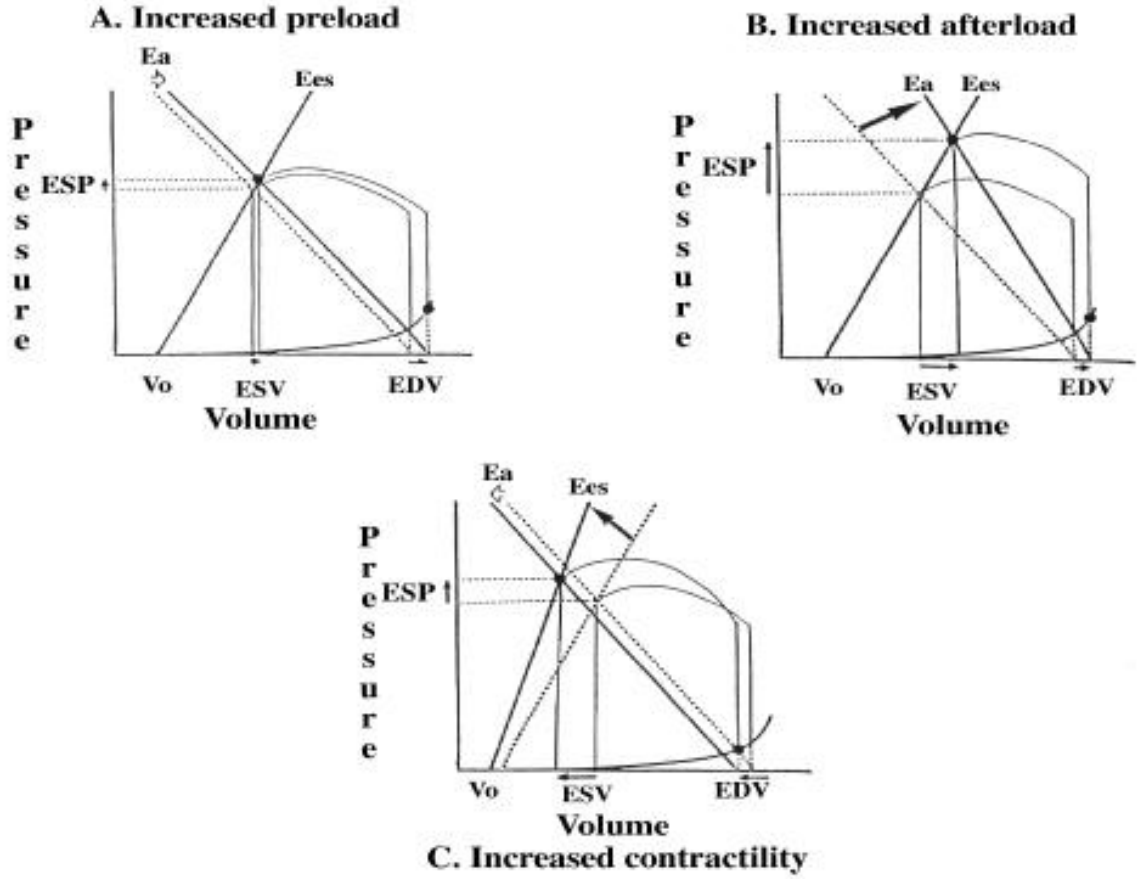
HCM CHF Treatment

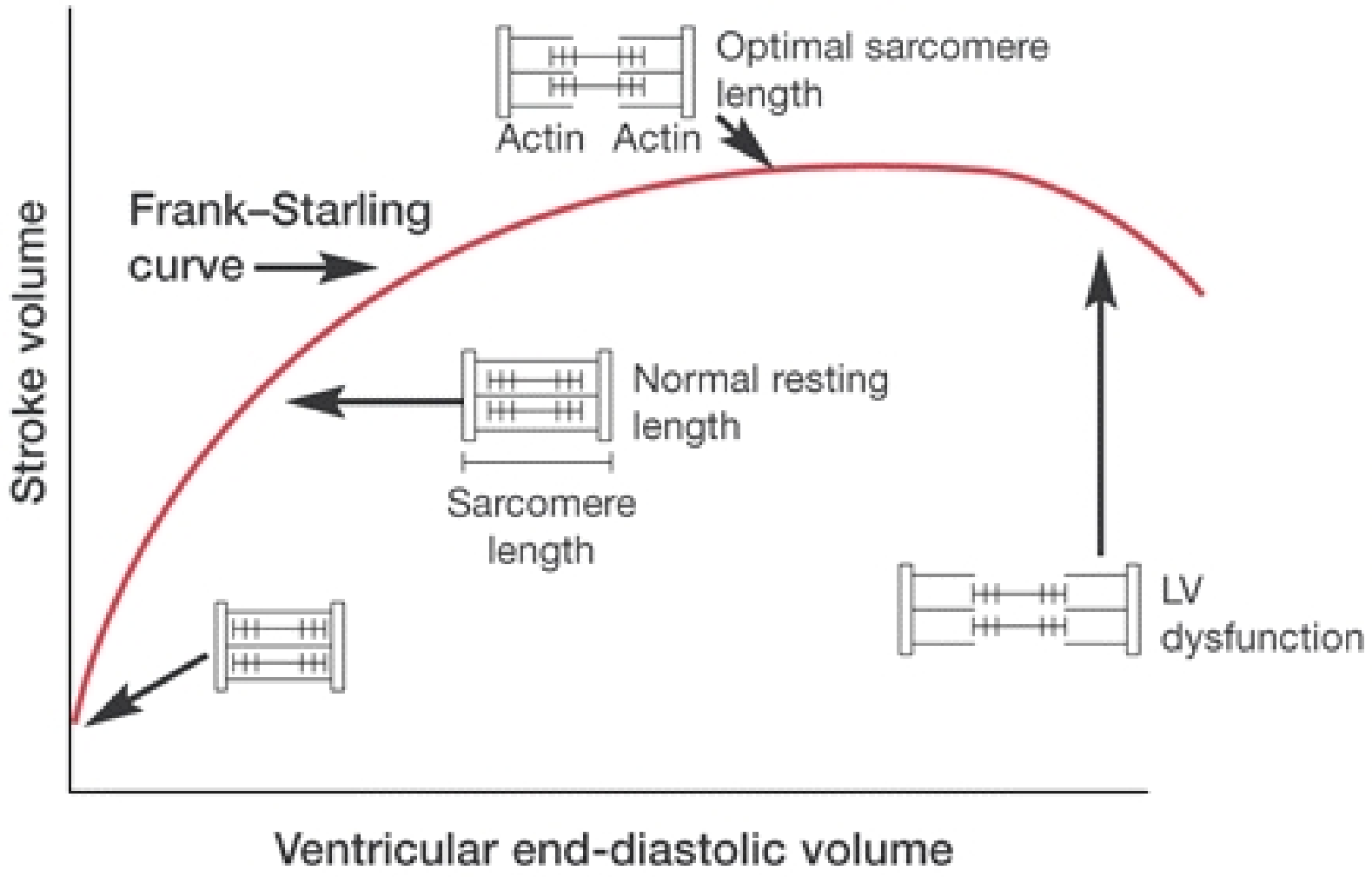
- Furosemide
- O_2 therapy
- +/- ACE inhibitor
- +/- Anti-thrombotics
  - Aspirin
  - Plavix
- Rate control

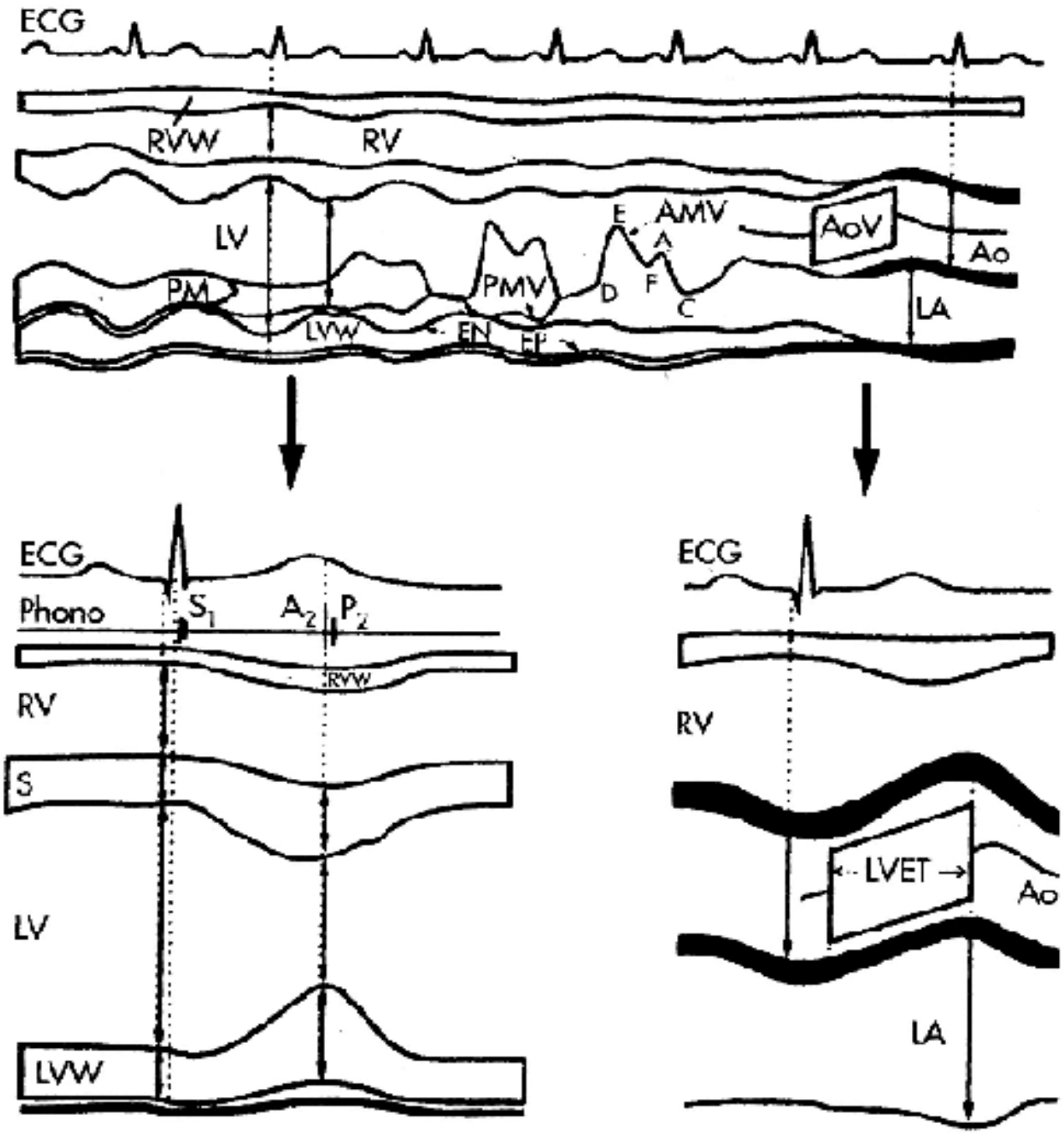


Review

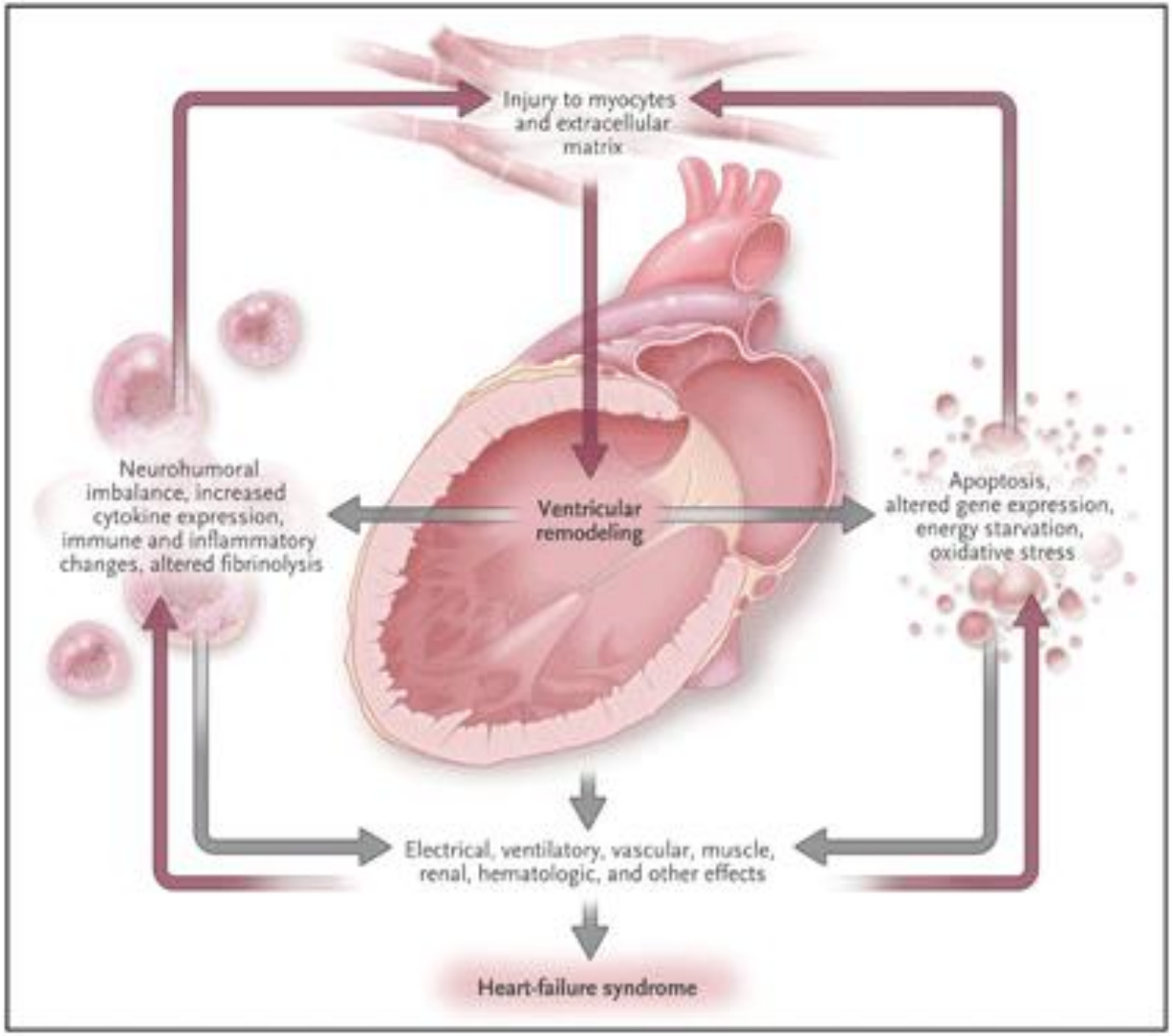

A Systolic Dysfunction
Pressure
Left Ventricular Volume
B Normal
Pressure
Left Ventricular Volume
C Diastolic Dysfunction
Pressure
Left Ventricular Volume


A. Increased preload
Ea
Ees
Pressure
ESP
Vo
ESV
EDV
Volume
B. Increased afterload
Ea
Ees
Pressure
ESP
Vo
ESV
EDV
Volume
Ea
Ees
Pressure
ESP
Vo
ESV
EDV
Volume
C. Increased contractility
Ea
Ees
Pressure
ESP
Vo
ESV
EDV
Volume
D. Increased stiffness
(or decreased lusitropy)
Ea
Ees
Pressure
ESP
Vo
ESV
EDV
Volume
E. Increased lusitropy
(or decreased stiffness)

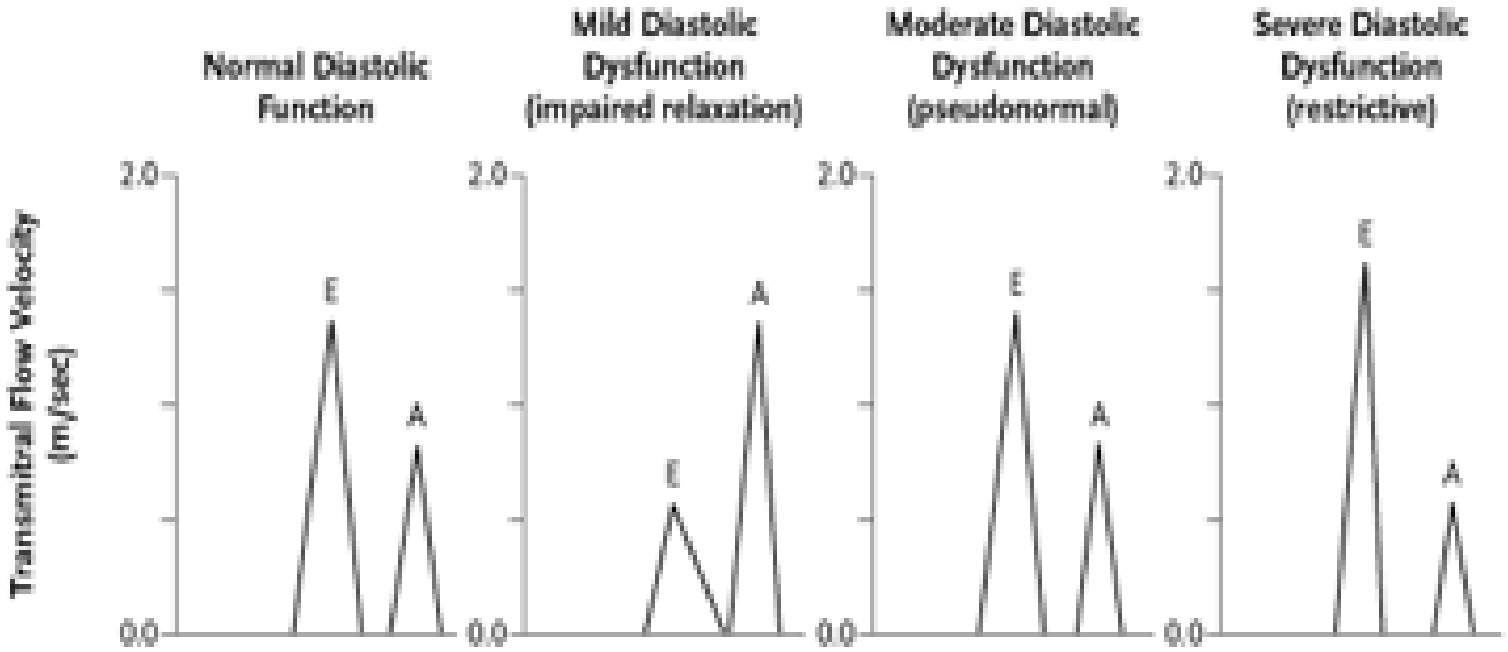

Optimal sarcomere length
Actin
Actin
Frank–Starling curve
Normal resting length
Sarcomere length
LV dysfunction
Stroke volume
Ventricular end-diastolic volume


ECG
RVW
RV
LV
PM
LVW
PMV
E
A
D
F
C
AMV
AoV
Ao
LA
EN
ECG
Phono
S1
A2
P2
RVW
RV
S
LV
LVW
ECG
RV
LVET
Ao
LA

Cardiac Remodeling



Review



Left Ventricular Relaxation	Normal	Impaired	Impaired	Impaired
Left Ventricular Compliance	Normal	Normal to ↓	↓↓	↓↓↓
Atrial Pressure	Normal	Normal to ↑	↑↑	↑↑↑