Heart Failure

Heart Failure (HF) -> Inability to provide adequate CO to supply demand of tissues. (or can do so at the expense of a raised filling P). Acute HF (MI) = Cardiogenic Shock. Chronic HF = CHF

Three 1° Causes of Heart Failure ->

① Pressure Overload -> Asking LV to pump at higher P than normal.
e.g. HT (Huge risk factor) or aortic stenosis (both have ↑ after load)

② Volume Overload -> e.g. aortic or mitral valve regurgitation
Blood in wrong chamber at wrong time, also ↑ stroke of V.

③ Contractile Dysfunction -> Muscle fibres can't prod. enough force.
e.g. ischaemia, myocardial disease, pregnancy, congenital cardiomyopathies.

Law of LaPlace -> Demonstration of balloons!

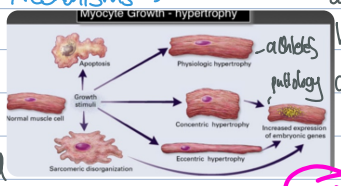
A small radius can hold back a lot of P.

The smaller the radius, the more P can be built up in a balloon, for a given wall thickness. So as V gets bigger, w/ the same P, the heart has to work harder. due to Heart Failure

Cardiac hypertrophy causes heart enlargement

Cardiac Compensatory Mechanisms ->

Physiologic -> normal ↑ stroke cause, ↑ muscle = normal. But if it is due to P overload + stress the myocytes are abnormal and you get ↑ exp. of embryonic genes.



3 Phases of Heart Failure ->

① Short Term Acute Failure -> Very fast. MI w/in seconds. w/in 30s
contractile fxn can't sustain CO. * Fxn reserves overwhelmed by overloads

② Compensated Hypertrophy -> Heart enlarges + adapts. Wall stress is normalized. Can be stable for years.

③ Chronic Failure -> If you don't remove the stim., you get exhaustion, kick in very quickly. cell death, and necrosis. Irreversible Disease.

Consequences of Cardiac Failure ->

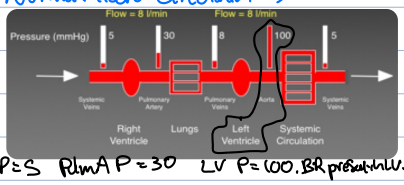
① Cardiac Changes -> Cardiac hypertrophy helps compensate but the new muscle is abnormal w/ ↑ risk of ischaemia, arrhythmia, sudden death.

-> ↑ wall stress -> ↑ O2 demand, capillary inadequacy -> O2 imbalance -> ischaemia + collagen deposition -> ↓ contractility, ↑ stiffness -> LV dilation -> LV failure.

② Capillary Filtration -> Normal: @ arterial end Hydrostatic P > colloid P so we extract fluid to lymph. This is reversed at venous end. So NO NET movement of fluid. Normal lungs: colloid P > Hydrostatic P so reverse fluid from lung. normal as we breathe humidified air. Heart Failure: Fluid movement in lungs is reversed hence in arterial end of capillaries HP > colloid P
So we get a NET gain of fluid in the lung. This is THE Major problem in HF

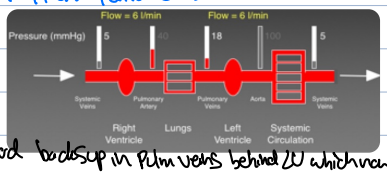
③ Pulmonary Congestion -> Fluid in the lungs makes it harder to breath. As HF progresses, the fluid not only accumulates in interstitium but also in alveoli which ↓ SA for gas exchange.

Normal Heart Circulation ->



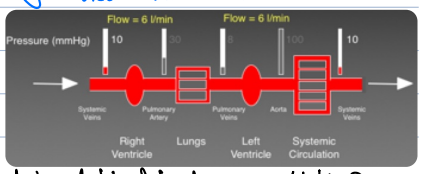
CVP = 5 PulmA P = 30 LV P = 100. BP presents in LV.
Body wants to keep LV P @ 100.

Left Heart Failure ->



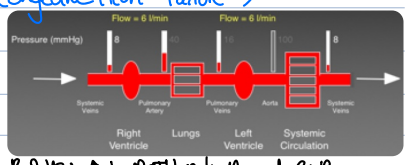
Blood backup in Pulm veins behind LV which now pushes back through lungs to raise P in Pulm A too. So Pulm P ↑. So now RV has to pump against this too ↑ P. So very quickly after left heart fails, right heart fails too which leads to

Right Heart Failure ->



We get blood backing up in the big systemic veins so CVP ↑ to 10.

Congestive Heart Failure ->



Results in ↑ in BOTH pulm A and CVP.
Congestive as P are high in Pulm circulation.
This is most common.

Pressure Overload ->

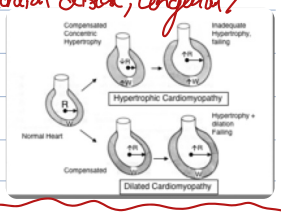
e.g. in HF or aortic stenosis
↑ P -> ↑ wall tension so heart responds in ↑ wall thickness (concentric hypertrophy). So now wall stress is normalized. This now thicker V is abnormal + unstable. So now over years we get hypertrophy + dilation. So radius ↑ so amount of wall stress ↑ and heart has to work harder.

Volume Overload ->

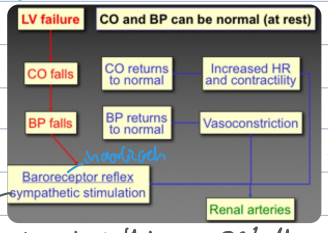
causes r of vent. but w/ ↑ vol. So again muscle has to work harder. This causes dilated hypertrophic myocardium, again ↑ radius + wall thickness -> wall stress normalized. So now when r ↑, we get myocardial failure w/ the vent big + dilated.

Contractile Dysfunction ->

Can go through either route of A) concentric hypertrophy or B) dilated cardiomyopathy
e.g. ischemic HD, myocardial disease, congenital

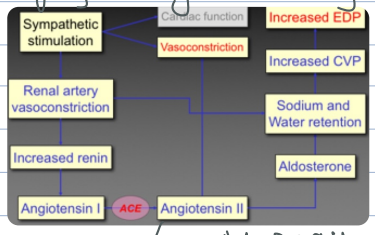


Left Ventricle is BAD.



Neurohumoral Compensation ->

• Signal to Symp to TADR. This happens in 30s. But now you have a failing heart that you are just throwing ADR + NA at. Bad is:



-> very potent vasoconstriction.

What keeps you alive in the short term:
↑ Symp, ↑ RAAS, ↑ AldO, ↑ myocardial hypertrophy
Also kill you in the long term!!!
β-blockers -| ↑ Symp
ACE-I -| ↑ RAAS
Spironolactone -| ↑ AldO

Renal Compensation ->

Overload due to ↑ afterload -> concentric hypertrophy (↑ V thickness)
Overload due to valve regurgitation -> dilated hypertrophic myocardium.
Contractile dysfxn due to not enough force -> concentric or dilated hypertrophy.

HF = inability to provide adequate CO to tissues.

HF Causes
- → Pressure overload (↑ afterload via HT or aortic stenosis) - leads to ↑ V thickness over time (concentric hypertrophy) so heart has to work harder.
- → V overload (aortic or mitral valve regurgitation, V infarct, ↑ P and stress V). ↑ dV ↑ w/ Vol so again. dilated hypertrophic myocardium.
- → Contractile dysfunction (can't prod. enough force - MI, ischemia, cardiomyopathy) can do either of concentric hypertrophy or dilated myopathy.

Law of LaPlace → a small radius can hold back a higher P. So w/ ↑ V at same P, heart has to work harder to pump.

Phases of HF → ① Acute failure → Final reserve overtaken by overload contractile fxn can't sustain CO. very fast
② Compensated hypertrophy → heart adapts, walls thicken, stress is normalized - can be stable for years.
③ Chronic failure → irreversible damage of structures not removed.

LHF → blood backs up into pulm veins and ↑ P in lungs + pulm A. Now RV has to pump against back 2 TP so you also get RHF which leads to congestive HF. which also ↑ P in CVP.

RHF → blood backs up into veins so ↑ CVP.

HF compensation → ① Neuro: when CO ↓ → huge sympathetic ↑ w/ ADR + NA to get ↑ contractility + HR.
② Renal → ↑ renin → ↑ BP → ↑ CO
↳ ↑ vasoconstriction.

- what keeps you alive short term is ↑ Symp, ↑ renin, ↑ Ald but :(also kills you long term.

HF consequences
- ① Cardiac hypertrophy: new muscle is abnormal w/ ↑ risk of ischemia.
- ② Capillary Filtration: we get edema into lungs as hydrostatic P > colloid P.
- ③ Pulm Congestion: ↑ fluid → ↓ SA for gas exchange so harder to breathe.

β-blocker ⊣ Symp
ACEi ⊣ RAAS
Spironolactone ⊣ Ald
↳ K+ sparing diuretic

All give the heart an easier work load to contract.