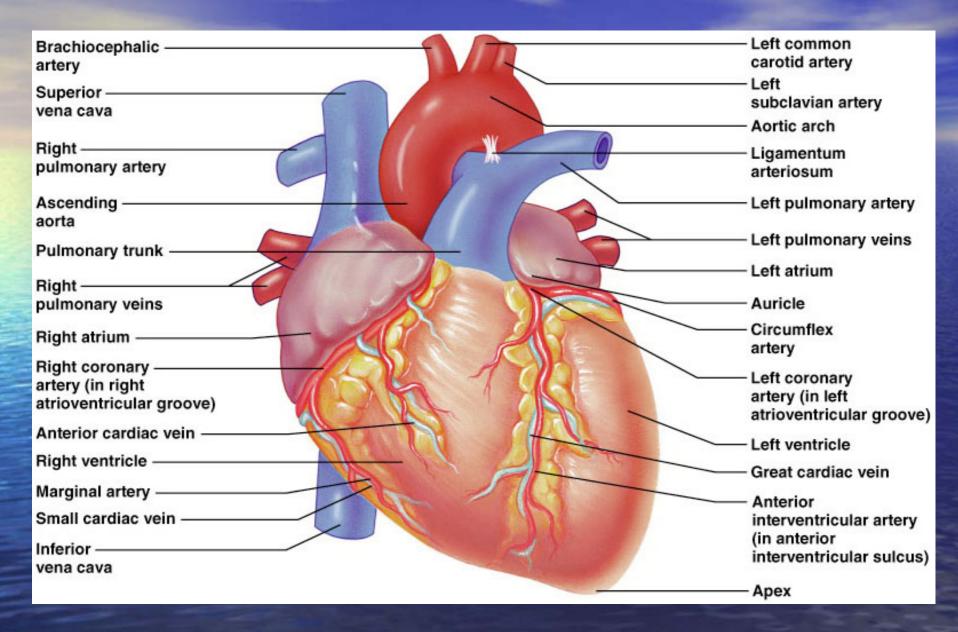
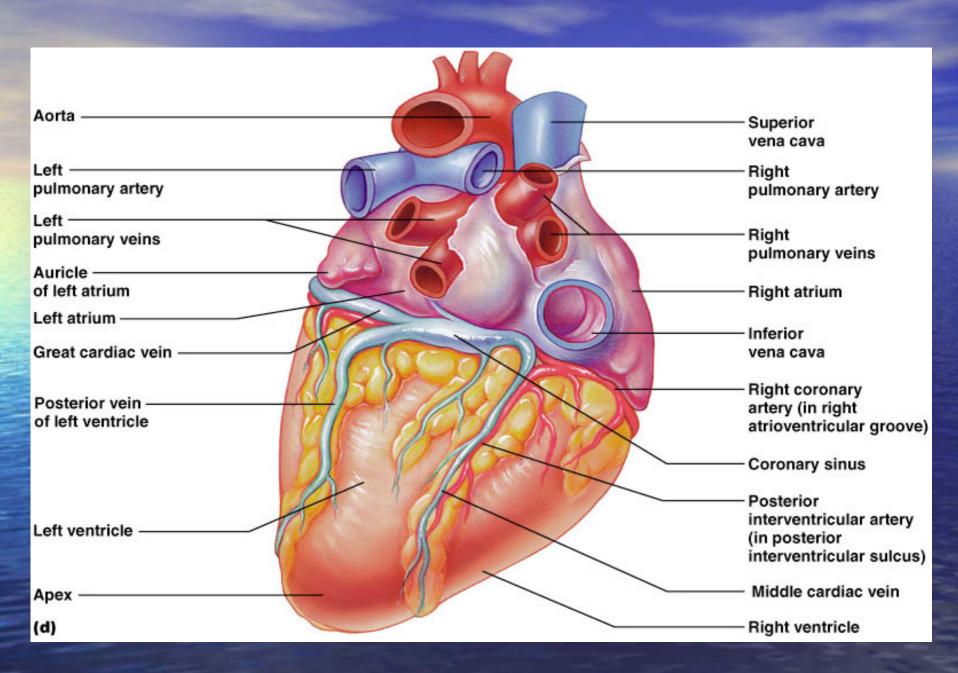
Special Circulation Dr. Mohammed Alzoghaibi

Provides blood flow to the heart

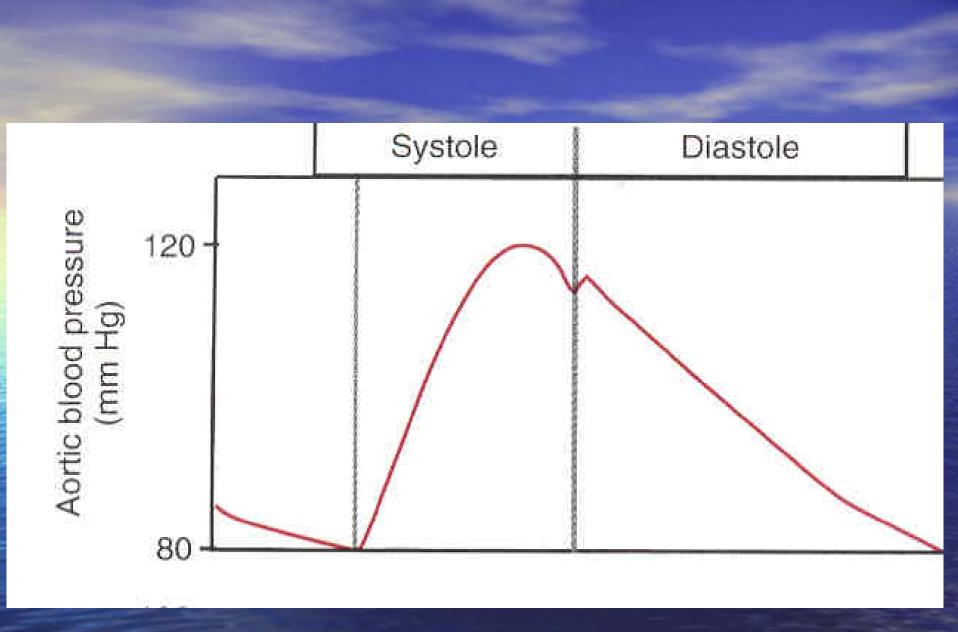
- Heart m. consumes as much O2 as does equal mass of SM during vigorous Ex
- Heart tissue extracts max amount of O2 during rest

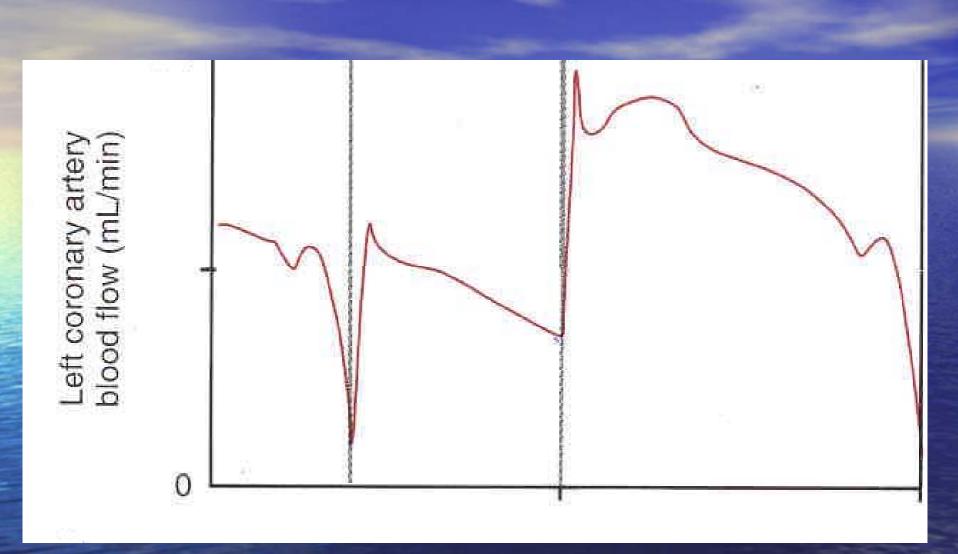
Limited anaerobic glycolysis in the heart, so the only way to < E is by BF</p>

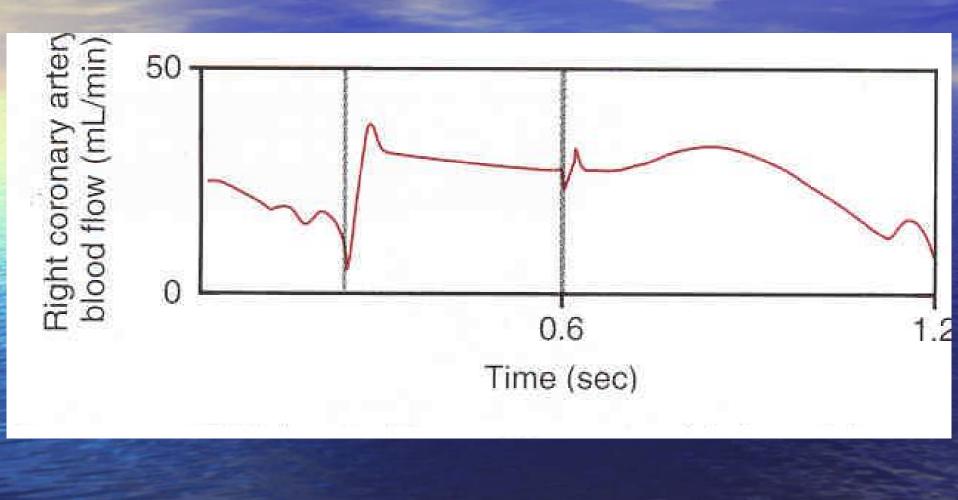




Perfusion of cardiac muscle from epicardium – endocardium Cardiac systole CBF is 10-30% of that during Cardiac diastole Systolic compression on RV is minimum Systolic compression has more effect on BF in the endocardial layer







Coronary Vascular Resistance

 75 % of CVR occurs in vessels with diameter ≤ 200 um (small arteries and arterioles)

Mechanisms for Controlling Blood Flow

- Local Control:
- Autoregulation
 - e.g., increase or decrease arterial pressure
- Active hyperemia
 - BF to an organ is proportional to its metabolic activities

Reactive hyperemia an increase in BF in response to a previous reduction in BF to certain organ

Hypothesis explains the local control of BF

Myogenic hypothesis
 explains autoregulation:
 vascular smooth
 m. stretching --> constriction --> resistance

 Metabolic hypothesis
 The O2 delivery to the tissue can be matched the O2 consumption by changing the Resistance

Factors affecting CBF: Chemical factors: 1. - Cardiac metabolism - Adenosine 2. NO 3. H **4**. CO2 5. **v** O2

Sympathetic innervation

- Constrictor mechanism is important in equalizing BF thru the layers than BF
- α1 (in CA and large arterioles) makes constriction
- β2 (in small arterioles) makes vasodilation
 β1 activation → heart rate

Sympathetic innervation

- Partial vasoconstriction of CA limits the retrograde BF during ventricular systole
 Prevents part of decreased flow in deep layers
- coronary vascular resistance (CVR)