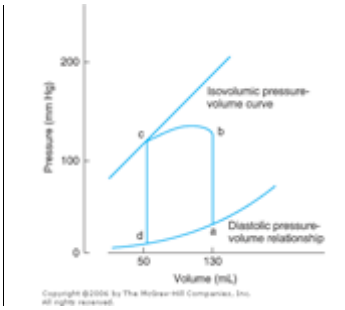
The heart as a pump

How much ventricular filling is provided by atrial contraction?
30%

Describe the pressure Vs volume loop in the LV throughout the cardiac cycle



Pressure–volume loop of the left ventricle. During diastole, the ventricle fills and pressure increases from d to a. Pressure then rises sharply from a to b during isovolumetric contraction and from b to c during ventricular ejection. At c, the aortic valves close and pressure falls during isovolumetric relaxation from c back to d.

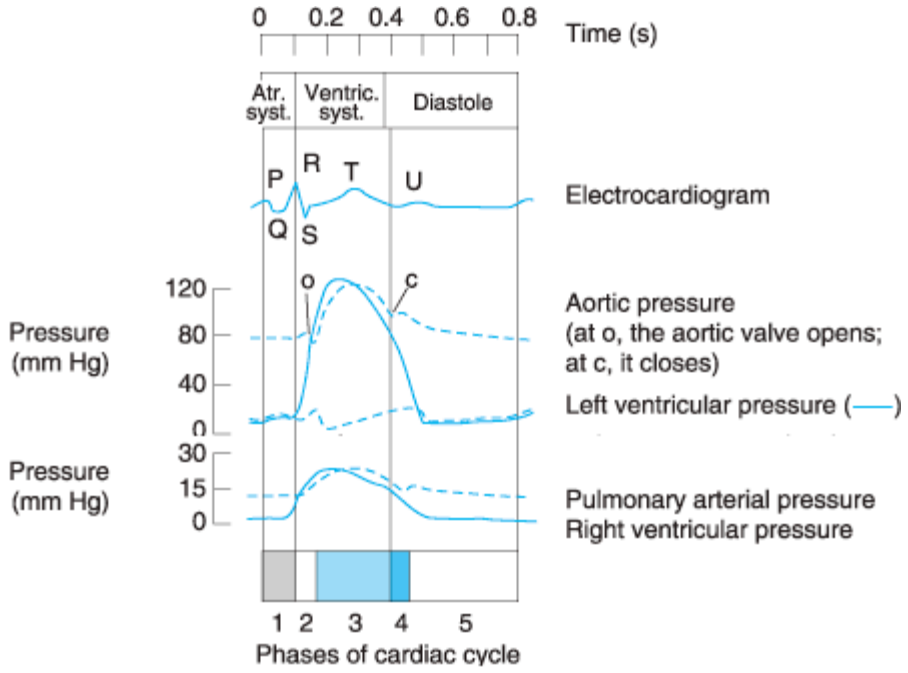
What is end diastolic volume, stroke volume and ejection fraction?

End diastolic volume is 130mL. Each ventricular contraction ejects about 70 – 90 mL of blood. The fraction of blood ejected is about 65%. This is a valuable index of ventricular function.

How does the duration of diastole change with changes in heart rate and why is this important.

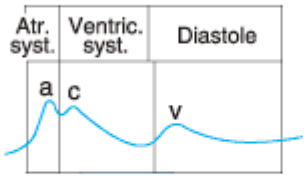
Cardiac muscle contracts and repolarizes faster when the heart rate is high. At high heart rates, the duration of diastole is shortened to a much greater degree than systole. It is during diastole that the heart muscle rests and coronary blood flow to the subendocardial portions of the left ventricle occurs only during diastole. It is also during diastole that most ventricular filling occurs. At rates up to about 180, filling is adequate as long as there is ample venous return and cardiac output per minute is increased by an increase in heart rate. Filling can become compromised to such a degree that cardiac output falls and heart failure develops.

Describe the pressures in the right ventricle and pulmonary artery during the cardiac cycle.



Describe jugular venous pressures during cardiac cycle and with respiration.

Atrial pressure changes are transmitted to the great veins. Atrial systole causes the a wave. The tricuspid valve bulges into the atria during isovolumetric ventricular contraction. The v wave mirrors the rise in atrial pressure before the tricuspid valve opens again. Venous pressure falls during inspiration as a result of the increased negative intrathoracic pressure and rises again during expiration.



Jugular venous pressure

What causes the arterial pulse and dicrotic notch?

Blood forced into the aorta during systole sets up a pressure wave that travels along the arteries. The pressure wave expands the arterial walls as it travels and this expansion is palpable as the pulse. The rate at which it travels is independent of and much higher than the velocity of blood flow. With age, the arterial walls become more rigid and the pulse wave move faster. The strength of the pulse is determined by the pulse pressure and bears little relationship to the mean pressure.
The dicrotic notch is a small oscillation on the falling phase of the pulse wave caused by vibrations set up when the aortic valve snaps shut. It is visible on the pressure wave recording, but not palpable. It also occurs on the pulmonary artery pressure curve produced by closure of the pulmonary valve.

What are the third and fourth heart sounds?

The third heart sound is soft and low pitched and occurs in young individuals about a third of the way through diastole. It coincides with the period of rapid ventricular filling and is probably due to vibrations set up by the inrush of blood.

The fourth heart sound can sometimes be heard immediately before the first heart sound when atrial pressure is high or the ventricle is stiff in conditions such as ventricular hypertrophy. It is due to ventricle filling and is rarely heard in normal adults.

What is the cardiac index?

Output of the heart (stroke volume) per unit time (heart rate) is the cardiac output. In a resting adult, it's about 5L/min. There is a correlation between resting cardiac output an body surface area. The output per minute per square metre of body surface area is called the cardiac index, and it averages about 3.2L/min/m2.

What is preload and afterload?

It is the degree to which the myocardiam is stretched before it contracts and the afterload is the resistance against which blood is expelled.

What is Starling's Law of the Heart?

The energy of contraction is proportional to the initial length of the cardiac muscle fibre. The length of the cardiac muscle fibre, the preload, is proportional to the end diastolic volume.

What factors affect end diastolic volume?

Increased by:

- Increased atrial contraction.
- Increased amount of blood returning to the heart:
  - Increased total blood volume
  - Increased venous tone
  - An increase in the normal negative intrathoracic pressure increases the pressure gradient along which blood flows to the heart.
  - Muscular activity as a result of the pumping action of skeletal muscle

Decreased by:

- Raised intrapericardial pressure limits the extent to which the ventricle can fill.
- Decrease in ventricular compliance does the same.
- Decreased amount of blood returning to the heart:
  - Decreased of the negative intrathoracic pressure (eg expiration, positive pressure ventilation) decreases the pressure gradient along which blood flows to the heart and hence impedes venous return.
  - Standing decreases venous return

What factors affect myocardial contractility?

Increased

- Sympathetic stimulation
  - Noradrenaline released from nerve endings
  - Circulating noradrenaline and adrenaline
    - Via cardiac β1 adrenaergic receptors
    - Increases intracellular cAMP
  - Xanthines (caffeine and theophylline) inhibit cAMP breakdown
  - Glucagon increases cAMP formation
- Digoxin increases intracellular Ca++ increasing contraction

Decreased

- Vagal stimulation
- Hypercapnia
- Hypoxia
- Acidosis
- Drugs
- Intrinsic depression in heart failure
- Loss of myocardium - Infarction

What is the Frank Starling Curve and how is it affected by myocardial contractility?

The relation between ventricular stroke volume and end diastolic volume is called the Frank Starling curve.