THE HEART AS A PUMP

MECHANICAL EVENTS OF THE CARDIAC CYCLE:

EVENTS IN LATE DIASTOLE:

- Late in diastole, the mitral and tricuspid valves between the atria and ventricles are open and the aortic and pulmonary valves are closed
- Blood flows into the heart throughout diastole, filling the atria and ventricles

ATRIAL SYSTOLE:

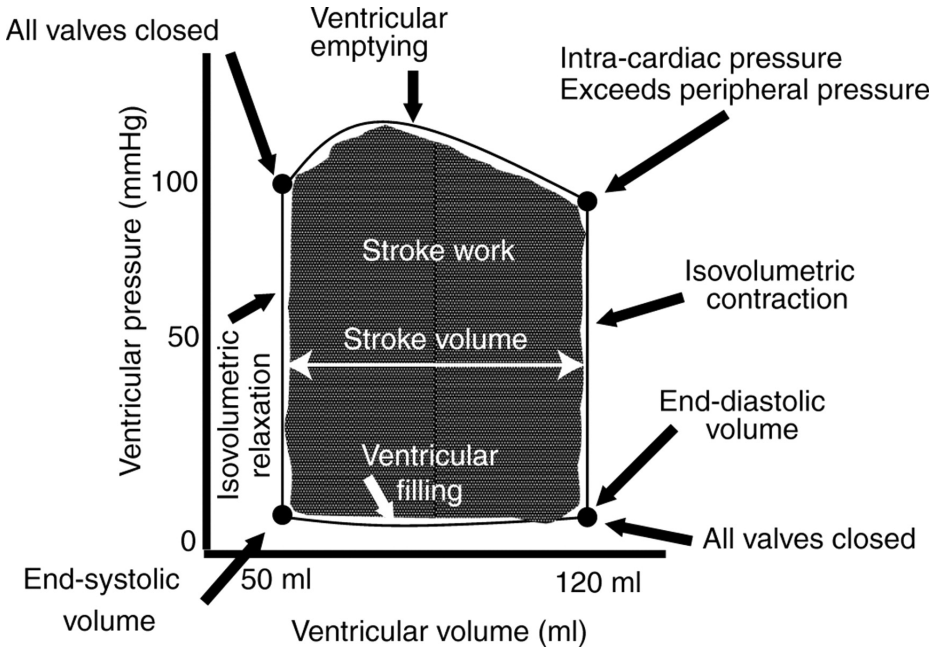
- Contraction of the atria propels some additional blood into the ventricles,
- **About 70% of ventricular filling occurs PASSIVELY DURING DIASTOLE**
- Contraction of the atrial muscle that surrounds the orifices of the SVC and IVC and pulmonary veins narrow their orifices and the inertia of the blood tends to keep blood in the heart
- There is some regurgitation of blood into the veins during atrial systole

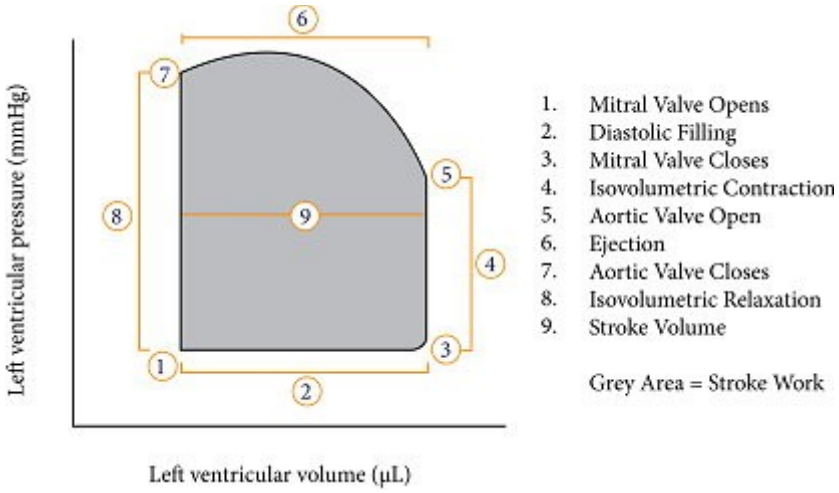
VENTRICULAR SYSTOLE:

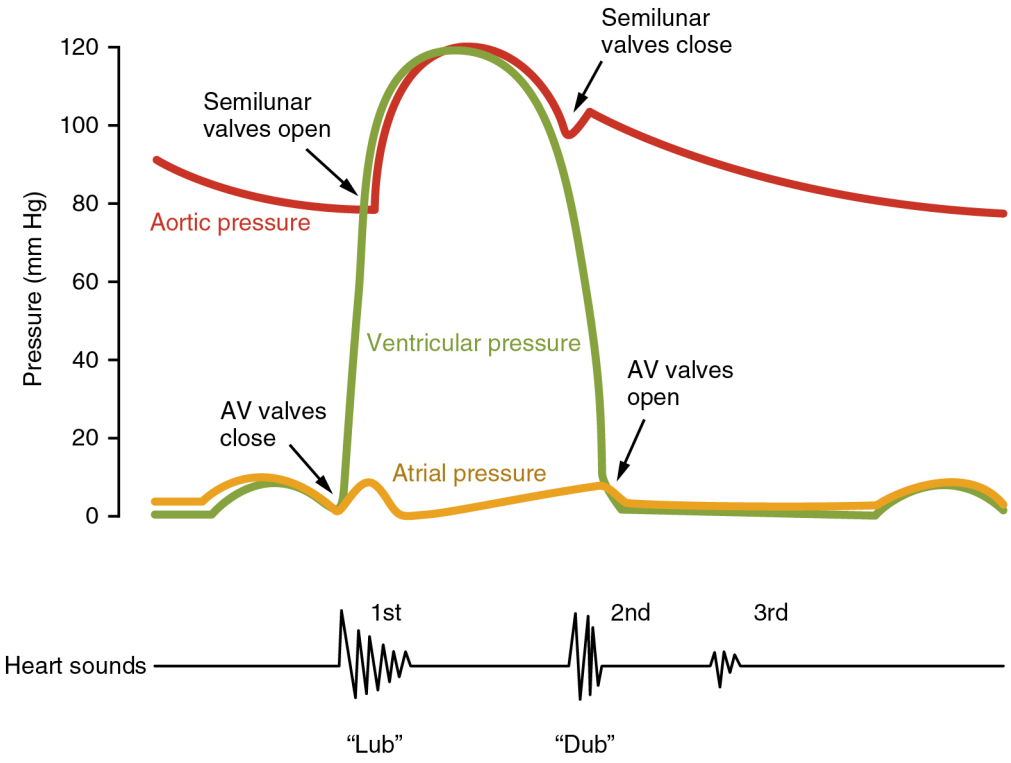
- At the start of ventricular systole, the mitral and tricuspid valves close
- Ventricular muscle initially shortens relatively little, but intraventricular pressure rises SHARPLY as the myocardium presses on the blood in the ventricle
    - ISOVOLUMETRIC VENTRICULAR CONTRACTION
        - Lasts until the pressures in the left and right ventricles exceed the pressures in the aorta and pulmonary arteries and the respective valves open
        - During isovolumetric contraction, AV valves bulge into the atria, causing a small but sharp rise in atrial pressure
- When the aortic and pulmonary valves open, the phase of VENTRICULAR EJECTION BEGINS
    - Ejection is rapid at firsts, slowing down as systole progresses
    - Peak LV pressure is about 120mmHg and RV pressure about 25mmHg
    - Stroke volume 70-90mL
    - THE END-DIASTOLIC VENTRICULAR VOLUME is about 130mL and 50mL of blood remains in each ventricle remains in each ventricle at the end of systole (END-SYSTOLIC VENTRICULAR VOLUME)
        - The EJECTION FRACTION is the percent of the end diastolic ventricular volume that is ejected with each stroke (normally about 65%)
            - Valuable index of ventricular function
    - LVEDV = 130ml
    - SV = 70ml
    - LVESV = 50ml
    - EF = 70/130 = approx. 55%

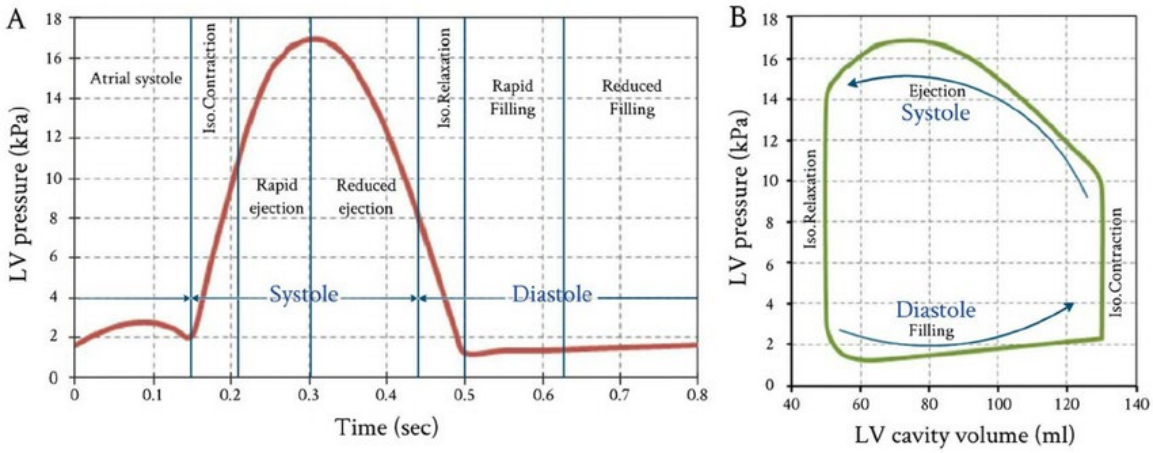
EARLY DIASTOLE:

- Once the ventricular muscle is fully contracted, the already falling ventricular pressure drop more rapidly
    - This is the period of **PROTODIASTOLE**
        - This ends when the momentum of the ejected blood is overcome and the aortic and pulmonary valves close, setting up transient vibrations in the blood and blood vessel walls
- After the valves are closed, pressure continues to drop rapidly during the period of **ISOVOLUMETRIC VENTRICULAR RELAXATION**
    - Ends when the ventricular pressure falls below the atrial pressure and the AV valves open, permitting the ventricles to fill
        - Filling is rapid at first, then slows as the next cardiac contraction approaches




Left ventricular pressure (mmHg)
Left ventricular volume (μL)
1. Mitral Valve Opens
2. Diastolic Filling
3. Mitral Valve Closes
4. Isovolumetric Contraction
5. Aortic Valve Open
6. Ejection
7. Aortic Valve Closes
8. Isovolumetric Relaxation
9. Stroke Volume
Grey Area = Stroke Work


Pressure (mm Hg)
120
100
80
60
40
20
0
Semilunar valves close
Semilunar valves open
Aortic pressure
Ventricular pressure
AV valves open
AV valves close
Atrial pressure
Heart sounds
1st
2nd
3rd
"Lub"
"Dub"



PERICARDIUM:

- The heart is separated from the rest of the thoracic viscera by the pericardium
- The pericardial sac normally contains 5-30mL of clear fluid, which lubricates the heart and permits it to contract with MINIMAL FRICTION

TIMING:

- Right atrial systole precedes left atrial systole
- Contraction of the RV starts after that of the left
    - Since the pulmonary arterial pressure is lower than aortic pressure, RV ejection begins before LV ejection
- During expiration, the pulmonary and aortic valves close at the same time
    - During inspiration, the aortic valve closes slightly before the pulmonary

- RA contracts before LA
- LV contracts before RV
- *But* RV ejects before LV

LENGTH OF SYSTOLE AND DIASTOLE:

- Cardiac muscle has the unique property of contracting and repolarising faster when the heart rate is high and the **duration of systole decreases**
    - The shortening is due mainly to a decrease in the duration of SYSTOLIC EJECTION
- **The duration of systole is much more fixed than that of diastole**
    - **When heart rate is increased, diastole is shorted to A MUCH GREATER DEGREE**
- It is during diastole that the heart muscle rests and coronary blood flow to the subendocardial portions of the LV occurs ONLY DURING DIASTOLE
    - Furthermore, most of the ventricular filling occurs during diastole
    - **At heart rates up to about 180, filling is adequate as long as there is AMPLE VENOUS RETURN**

        - At very high heart rates, filling may be compromised to such a degree that cardiac output per minute falls and symptoms of heart failure develop
- Because it has a prolong action potential, cardiac muscle is in its refractory period and will not contract in response to a second stimulus until near the end of the initial contraction
    - **THUS CARDIAC MUSCLE CANNOT BE TETANISED LIKE SKELETAL MUSCLE**

- **⇑⇑HR → ⇓Diastolic time →**
    - **⇓Coronary perfusion time**
    - **⇓Diastolic filling time → ⇓LVEDV**

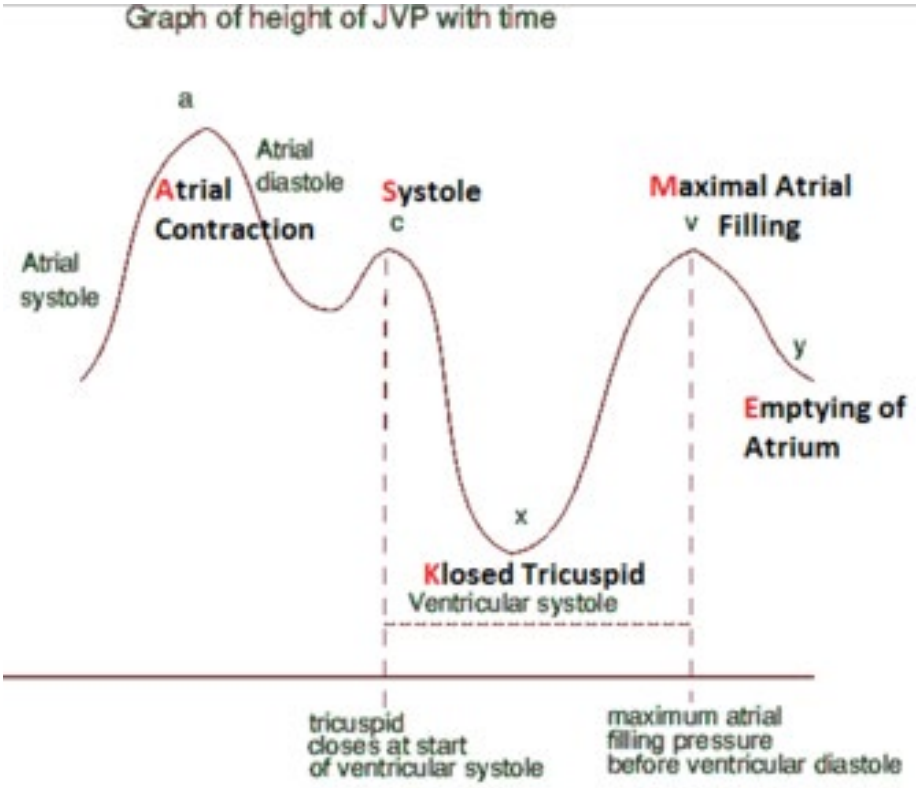
ARTERIAL PULSE:

- Blood forced into the aorta during systole not only moves the blood forward, but also sets up a pressure wave that travels along the arteries
    - The pressure wave expands the arterial walls and is palpable as the PULSE
    - **With advancing age, the arteries become more rigid, and the pulse wave moves faster**
- The strength of the pulse is determined by the PULSE PRESSURE and bears little relation to the MAP
    - It is strong when stroke volume is large
- When the aortic valve is incompetent, the pulse is particularly strong (large pulse pressure)
    - The force of systolic ejection may be sufficient to make the head nod with each heart beat
    - The pulse in aortic insufficiency is called COLLAPSING, CORRIGAN OR WATER-HAMMER
- THE DICROTIC NOTCH:
    - This is a small oscillation on the falling phase of the pulse wave caused by **vibrations set up when the aortic valve snaps shut**

ATRIAL PRESSURE CHANGES AND THE JUGULAR PULSE:

- Atrial pressure rises during atrial systole and continues to rise during isovolumetric contraction when the AV valves bulge into the atria
- The return of the AV valves to their relaxed position also contributes to this pressure rise by reducing atrial capacity
- The atrial pressure changes are transmitted to the great veins, producing THREE CHARACTERISTIC WAVES IN THE RECORD OF THE JUGULAR PRESSURE:
    - A-wave:
        - Due to ATRIAL SYSTOLE

        - Some blood regurgitates into the great veins when the atria contract, even though the orifices of the great veins are constricted
    - C-wave:
        - Transmitted manifestation of the rise in atrial pressure produced by the bulging of the tricuspid valve into the atria during isovolumetric ventricular contraction
    - V-wave:
        - Mirrors the rise in atrial pressure before the tricuspid valve opens during diastole
- In tricuspid insufficiency, there is a GIANT C-WAVE with each ventricular systole
- In complete heart block:
    - CANNON A WAVES
        - Occur whenever the atria contract while the tricuspid valve is closed (A-V dissociation)



HEART SOUNDS:

- First sound is caused by vibrations set up by the sudden closure of the mitral and tricuspid valves
- Second sound is associated with the closure of the aortic and pulmonary valves just after the end of ventricular systole

- **S2 is split during <u>inspiration</u>: A2 then P2**
- A soft, low pitched THIRD SOUND is heard about one third of the ways through diastole in many normal young individuals
    - **S3 = RAPID VENTRICULAR FILLING**
- S4
    - Sometimes heard immediately before the first sound when atrial pressure is high or the ventricle is stiff in conditions such as ventricular hypertrophy
    - **Atrial contraction against a stiff ventricle (HTN / AS)**
    - RARELY HEARD IN NORMAL INDIVIDUALS

MURMURS:

- ABNORMAL SOUNDS
- Blood flow is laminar and nonturbulent up to a critical velocity
    - Above this velocity and beyond an obstruction, blood flow is turbulent
        - Laminar flow is silent but turbulent flow creates sounds
- Murmurs due to disease of valves are relatively common:
    - AORTIC OR PULMONARY:
        - STENOSIS =>SYSTOLIC
        - REGURGITATION => INSUFFICIENCY
    - MITRAL OR TRICUSPID:
        - STENOSIS =>DIASTOLIC
        - REGURGITATION => SYSTOLIC
- In patients with congenital interventricular septal defects, flow from the left to the right ventricle causes a systolic murmur

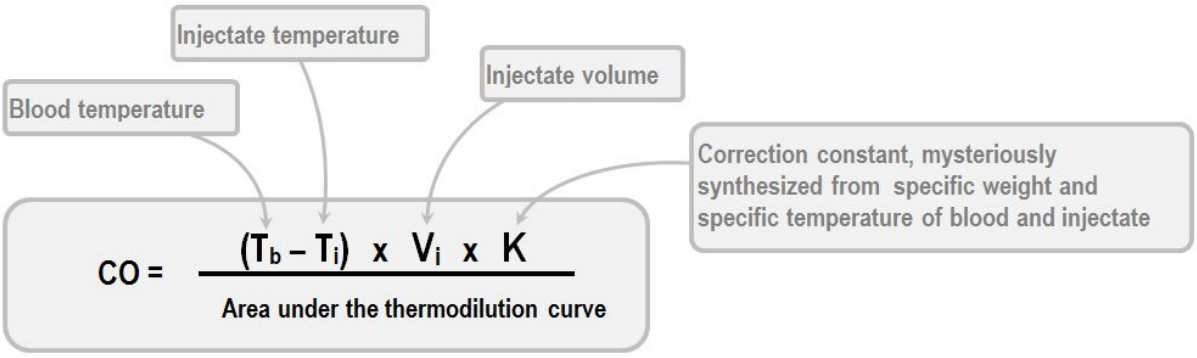
<u>CARDIAC OUTPUT:</u>

METHODS OF MEASUREMENT:

- Cardiac output can be measured with an electromagnetic flow meter placed on the ascending aorta (experimental animals)
- Two methods of measuring output are applicable to humans
    - **DIRECT FICK PRINCIPLE:**
        - States that the amount of a substance taken up by an organ per unit time is equal to the arterial level of the substance minus the venous level (A-V DIFFERENCE) times the blood flow
        - **Uptake = (C_A-C_V) x CO**
        - **CO = VO2 / (C_A-C_V)**
        - O2 consumed by the body in a given period and dividing this value by the A-V difference across the lungs
        - The arterial content can be measured in a sample obtained from any convenient artery

        - A sample of venous blood in the pulmonary artery is obtained by means of a cardiac catheter

    - **INDICATOR DILUTION METHOD:**
        - A known amount of a substance is injected into an arm vein and the concentration of the indicator in serial samples of arterial blood is determined
        - The output of the heart is equal to the amount of indicator injected divided by its average concentration in arterial blood after a single circulation through the heart
        - **CO = amount injected / average conc in arterial blood**
        - Indicator used can be:
            - Dye
            - Radioactive isotope
            - Cold saline (**THERMODILUTION METHOD**)



CARDIAC OUTPUT IN VARIOUS CONDITIONS:

- Amount of blood pumped out of each ventricle per beat = STROKE VOLUME
    - **About 70mL in a man of average size in the supine position**
- The output of the heart per unit time is the CARDIAC OUTPUT:
    - **Averages about 5L/min**
- There is a correlation between resting cardiac output and BODY SURFACE AREA:
    - The output per minute per square metre of BSA = **CARDIAC INDEX**
    - **Cardiac index > 3**

FACTORS CONTROLLING CARDIAC OUTPUT:

- Variations in cardiac output can be produced by changes in:
    - CARDIAC RATE
    - STROKE VOLUME
- The cardiac rate is controlled primarily by the CARDIAC INNERVATION:
    - Sympathetic stimulation increasing the rate

    - Parasympathetic stimulation decreasing it
- The stroke volume is also determined in part by NEURAL INPUT
    - Sympathetic stimuli make the myocardial muscle fibres contract with greater strength at any given length
    - Parasympathetic stimuli having the opposite effect
    - When the strength of contraction increases without an increase in fibre length, more of the blood that normally remains in the ventricles is expelled
        - I.e. THE EJECTION FRACTION INCREASES and the end-systolic ventricular blood volume falls
- The cardiac accelerator action of catecholamines is referred to as their CHRONOTROPIC ACTION
    - Effect on strength of contraction is called INOTROPIC ACTION
- The force of contraction of cardiac muscle depends on:
    - PRELOADING
        - Degree of stretch
    - AFTERLOADING

- **Preload** - the degree to which the myocardium is stretched before it contracts
- **Afterload** - the resistance against which blood is expelled: i.e. the load against which the heart must work to expel blood from the ventricle.

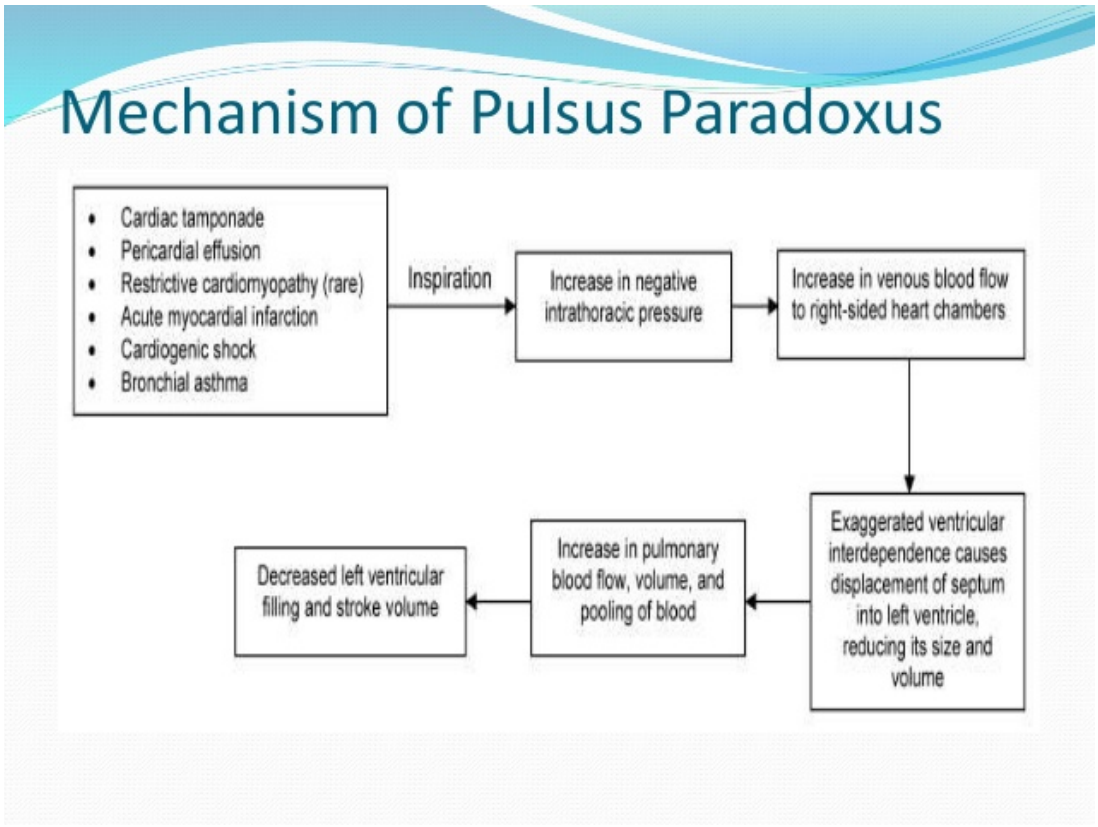
RELATION OF TENSION TO LENGTH IN CARDIAC MUSCLE:

- As the muscle is stretched, the developed tension increases to a maximum and then declines as stretch becomes more extreme
- STARLING:
    - **The energy of contraction is proportional to the initial length of the cardiac muscle fibre**
    - This became known as STARLING'S LAW OF THE HEART or the FRANK-STARLING LAW
- For the heart, the length of the muscle fibres (I.E. THE EXTENT OF THE PRELOAD) is proportionate to the END-DIASTOLIC VOLUME
    - The relation between ventricular stroke volume and end-diastolic volume is called the FRANK-STARLING CURVE

FACTORS AFFECTING END-DIASTOLIC VOLUME:

- An increased in intrapericardial pressure limits the extent to which the ventricle can fill
    - So does a DECREASE IN VENTRICULAR COMPLIANCE:
        - Increase in ventricular stiffness produced by MI, infiltrative disease and other abnormalities

- An increase in total blood volume increases venous return (MSFP)
- Constriction of the veins reduces the size of the venous reservoirs, decreasing venous pooling and thus increasing venous return
- An increase in the normal negative intrathoracic pressure increases the pressure gradient along which blood flows to the heart, whereas a decrease in pressure gradient IMPEDES VENOUS RETURN



Pulsus paradoxus = >10mmHg fall in SBP during inspiration

MYOCARDIAL CONTRACTILITY:

- The contractility of the myocardium exerts a major influence on stroke volume
- When the sympathetic nerves to the heart are stimulated (β1 Gs), the whole length-tension curve shifts upward and to the left
- Changes in cardiac rate and rhythm also affect myocardial contractility
    - Ventricular extrasystoles condition the myocardium in such a way that the next succeeding contraction is stronger than the preceding normal contraction
    - This **POSTEXTRASYSTOLIC POTENTIATION** is INDEPENDENT OF VENTRICULAR FILLING
        - Due to increased **availability of extracellular calcium**

- The catecholamines exert their inotropic effect via an action on CARDIAC BETA-1 ADRENERGIC RECEPTORS, with resultant activation of **adenylyl cyclase** and increased intracellular **CAMP**
- **HYPERCAPNIA, HYPOXIA AND ACIDOSIS ALL DECREASE CONTRACTILITY**
    - **I.e. situations of SHOCK reduced cardiac contractility**
- The contractility of the myocardium is also reduced in heart failure (INTRINSIC DEPRESSION)
    - Mechanism unknown

INTEGRATED CONTROL OF CARDIAC OUTPUT:

- During muscular exercise, there is increased sympathetic discharge, so that myocardial contractility is increased and the heart rate rises
    - The INCREASE IN HEART RATE IS PARTICULARLY PROMINENT in normal individuals
        - There is only a modest increase in stroke volume
        - HOWEVER:
            - Patients with TRANSPLANTED hearts are able to increase their cardiac output during exercise in the absence of cardiac innervation through the operation of the Frank-Starling mechanism
            - If venous return increases and there is no change in sympathetic tone, venous pressure rises, diastolic inflow is greater, ventricular end-diastolic pressure increases and the heart muscle contracts more forcefully (and by Bainbridge reflex, with ⇑HR)
        - Normal hearts probably never dilate to the point that they are on the "DESCENDING LIMB" of the Frank-Starling curve
            - I.e. a point where further stretch DECREASES rather than increases stroke volume

OXYGEN CONSUMPTION BY THE HEART:

- The basal O2 consumption by the myocardium is about 2mL/100g/min
    - This value is considerably higher than resting skeletal muscle
- O2 consumption by the beating hart is about 9mL/100g/min
- **Cardiac venous O2 tension is LOW and little additional O2 can be extracted from the blood in the coronaries, so increases in O2 consumption require increases in coronary blood flow**
- **I.e. at baseline, myocardium extracts most of the O2 from coronary blood, so increased oxygen provision must come in the form of increased flow.**

- The O2 consumption by the heart is determined primary by the:
  - **INTRAMYOCARDIAL TENSION (Leplace)**
  - THE **CONTRACTILE STATE** OF THE MYOCARDIUM
  - THE **HEART RATE**
- The work of the heart is the PRODUCT OF STROKE VOLUME AND MAP (in the pulmonary artery for RV and aorta for LV)
  - **CARDIAC WORK = SV x MAP**
  - This is the area within the volume-pressure loop
  - Stroke work of the LV is approximately seven times the stroke work of the RV (same SV, but systemic MAP = 100 vs. pulmonary MAP = 15
- For reasons not fully understood
  - **PRESSURE WORK PRODUCES A GREATER INCREASE IN O2 CONSUMPTION** THAN VOLUME WORK
    - In other words, **an increase in afterload causes a greater increase in cardiac O2 consumption than an increase in preload does**