Cardiovascular System

Functions

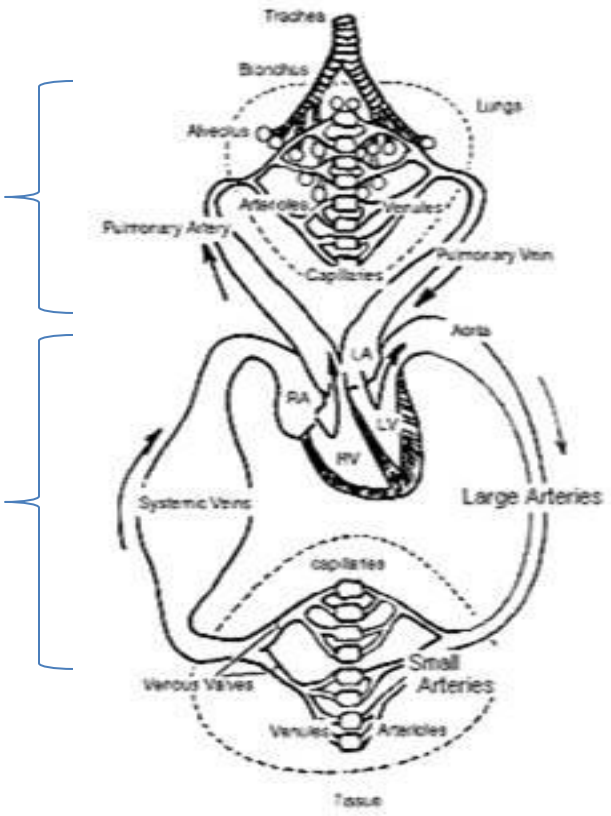
1. Transports oxygen and nutrients to the tissues of the body
2. Removes carbon dioxide and waste products from the tissues
3. Regulates body temperature and pH
4. Transports and distributes hormones and other substances throughout the body

Components

1. Heart – the central pump
2. Closed system of blood vessels
3. Blood – fluid medium – through which various substances are transported

Types of Circulatory Systems

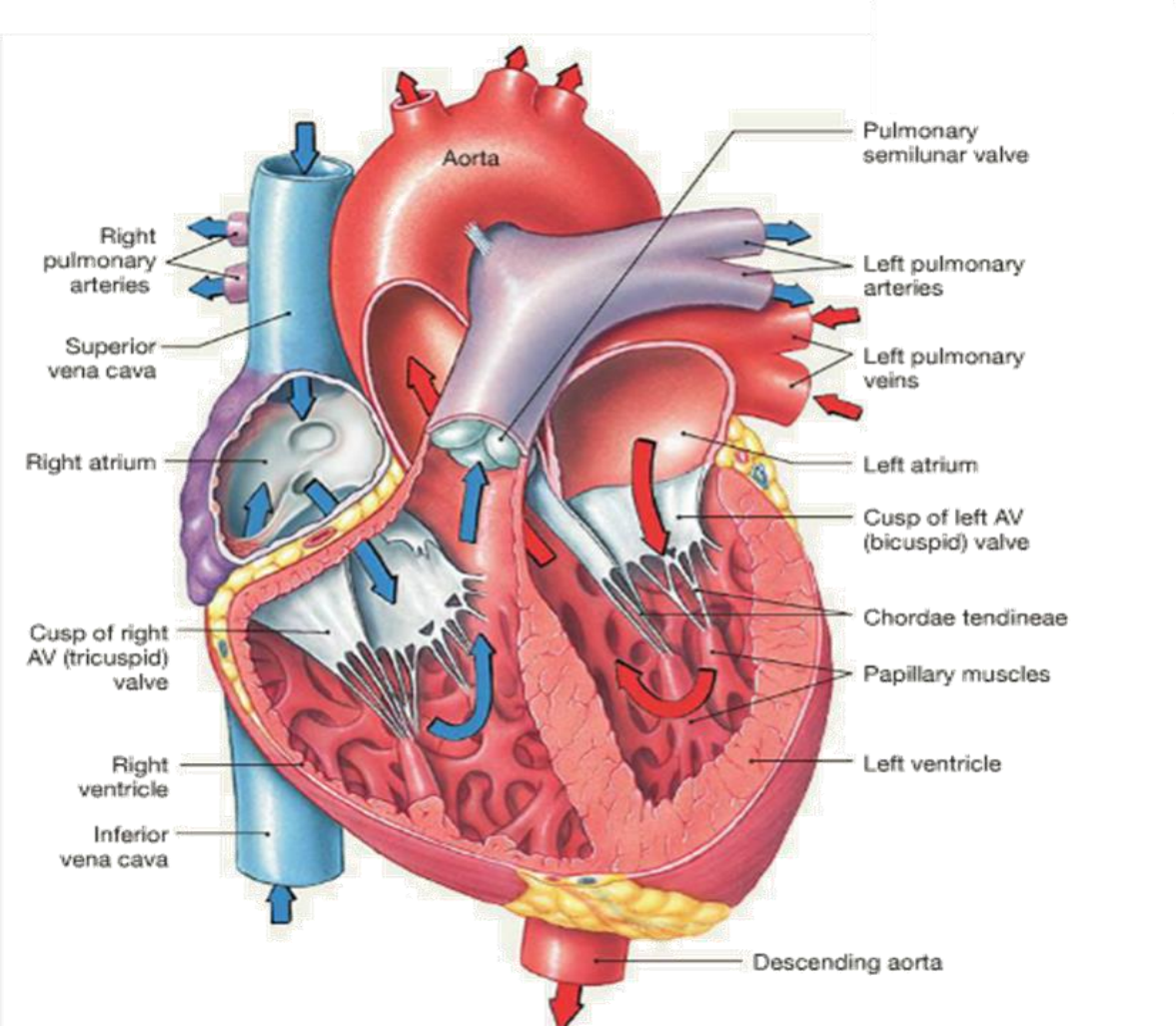
1. Pulmonary circulation
    - Includes the blood vessels that carry blood to and from the lungs
    - Provides exchange of gases (oxygen and carbon dioxide) between the atmosphere and the blood
2. Systemic circulation
    - Includes the blood vessels that carry blood to and from the rest of the body
    - Capillaries are the exchange vessels where oxygen and nutrients pass into the tissues and carbon dioxide and waste products are taken up into the blood



Blood Volume Distribution

- Total blood volume in normal humans is 5-6 L
- 70% is contained in the veins
- 10% is contained in the atrial system
- 15% in heart and lungs
- 5% in capillaries

Anatomy of the Heart



Myocardial Cells

- Atrial and ventricular *contractile* muscle fibres
    - Have many similar features and contract the same way as many skeletal muscle fibres, although they are structurally different
- Specialized excitatory and *conductive* cardiac cells
    - Contract very weakly because they contain very few contractile elements (myofibrils)
    - Instead, they have special properties of self-excitability (able to spontaneously generate an action potential) and are able to rapidly conduct these impulses to atrial and ventricular muscles
    - Thus, these specialized cells provide a self-excitatory system for the heart to generate impulses and a transmission system for rapid conduction of impulses through the heart

1. Contractile Cells
    - Have similarities to muscle cells
    - Contract
2. Conducting/Nodal Cells
    - Similarities to nerve cells
    - Generate/transmit AP

Similarities between cardiac and skeletal muscle cells:

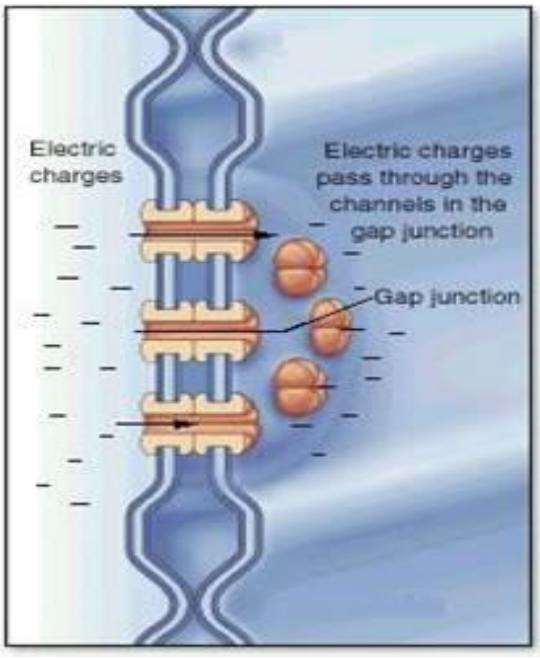
- Striated
- Contain similar contractile proteins (actin and myosin)
- Ca^{++} release from SR triggers contraction

Differences from skeletal muscle:

- Short and branched
- One third of volume is occupied by mitochondria – produce ATP
- Extract 80% of the oxygen from the blood
- Joined by intercalated discs that contain gap junctions – allow movement of ionic currents (APs) between cells

Intercalated Discs

- Myocardial cells are joined by intercalated discs that contain gap junctions
- The gap junctions allow movement of ionic currents (APs) between cells
- This way, one AP in one contractile cell will spread to all the others, so when one cell contracts, they all contract
- This is different from skeletal muscle



Nodal and Conducting Cells of Heart

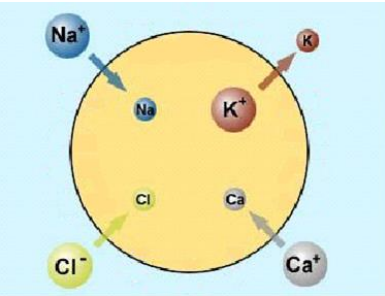
- Contain few contractile proteins
- *self-excitable: spontaneously generate APs
- Rapidly conduct AP through the heart
    - More like nerve cells

1. Nodal Cells
    a. Sinoatrial (SA) node
    b. Atrioventricular (AV) node
2. Conducting Cells
    a. Bundle of His
    b. Purkinje Fibers

The SA Node

- The pacemaker
- The adult heart normally contracts at a rhythmic rate of 72 beats per minute
- The self-excitory impulse that gives rise to each heart beat originates in the SA node
- Capability of self-excitation is greatest in the SA node
    - It has the fastest spontaneous generation o the action potential compared to all other areas of the heart
    - For this reason, the SA node ordinarily controls the rate of beats of the entire heart
- Located in the upper posterior wall of the right atrium

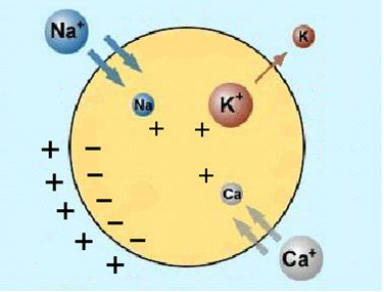
Concentration gradients for ions in and around a typical cell:



Excitable cells

Self-excitability caused by:

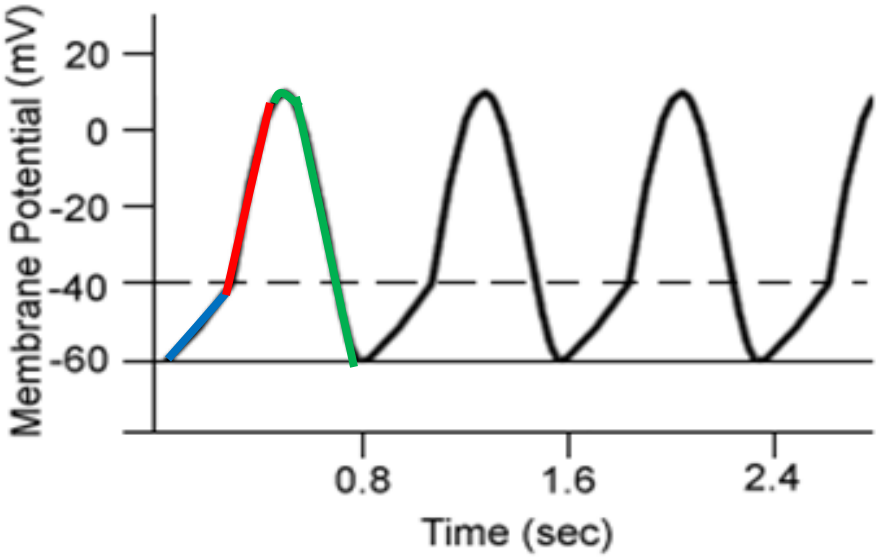
1. The cells of the SA node have relatively greater sodium and calcium permeability, compared to other cells of the heart
2. The potassium permeability of SA node cells decreases during diastole
    - Less potassium leaves these cells during relaxation of the heart



SA node cells

- Because of these properties, the SA nodal cells do not have a stable resting membrane potential
- This rhythmic generation of action potentials determines the heart rate
- The rate of discharge of APs by the SA node (and therefore heart rate) can be altered by changing the slope of what is called the pacemaker potential

SA Nodal Action Potential:

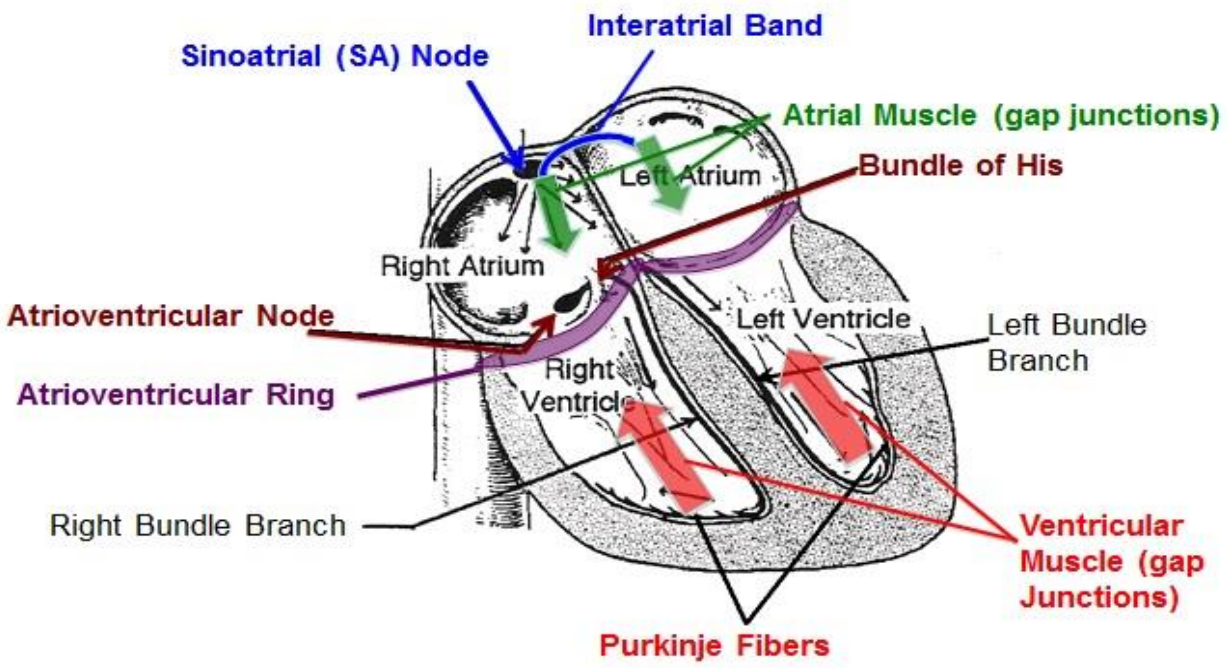


-Membrane potential varies between -60mV and +15mV

-Has a threshold of -40mV

1. Pre-potential (pacemaker potential)
    - Slow depolarization
    - Increased permeability of cells to Na^+ and Ca^{++}
    - Decreased permeability to K^+
    - Membrane potential reaches threshold
2. Depolarizing Phase
    - At threshold (-40 mV), Ca^{++} VG channels open
    - Ca^{++} flows into SA nodal cell
    - Membrane potential reaches +15mV
3. Repolarization
    - Ca^{++} VG channels begin to close
    - K^+ VG channels begin to open
        - Rounded peak
    - K^+ leaves the cell
    - Membrane potential returns to -60mV
    - K^+ channels begin to close and cycle repeats

Conducting System of the Heart



The speed of the action potential throughout the heart varies depending on its location in the conducting system

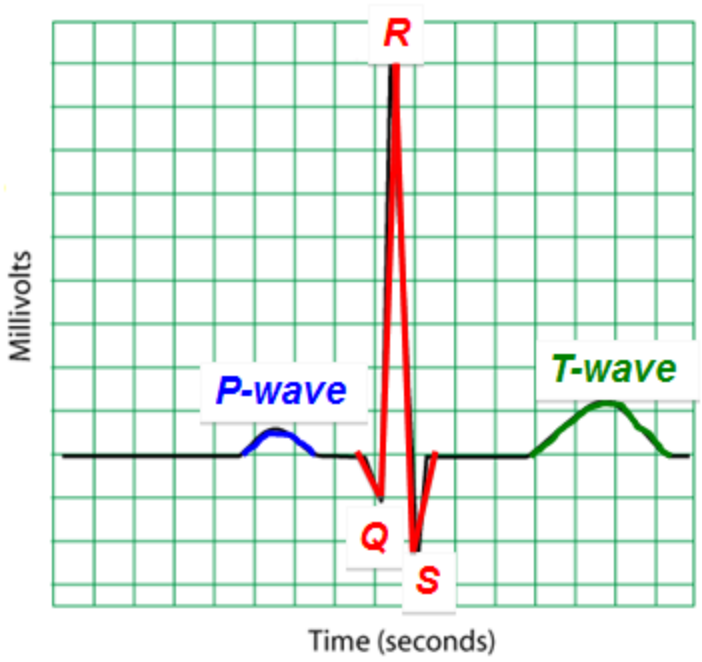
Location	Speed (m per sec)
SA node	0.05
Atrial muscle	1
AV node	0.03-0.05
Bundle of His	1
Purkinje fibres	5
Ventricular muscle	1

Slows down

Speeds up

Electrocardiogram

- Body fluids conduct electricity
- Cardio impulses pass through to heart to surrounding tissue and to surface of the body
- Electrodes can pick up these impulses
- ECG is the *sum* of all the electrical events in the heart, both depolarizing and repolarizing



P-wave

-depolarization of atrial muscle

QRS complex

-depolarization of ventricular muscle

T-wave

-repolarization of ventricular muscle

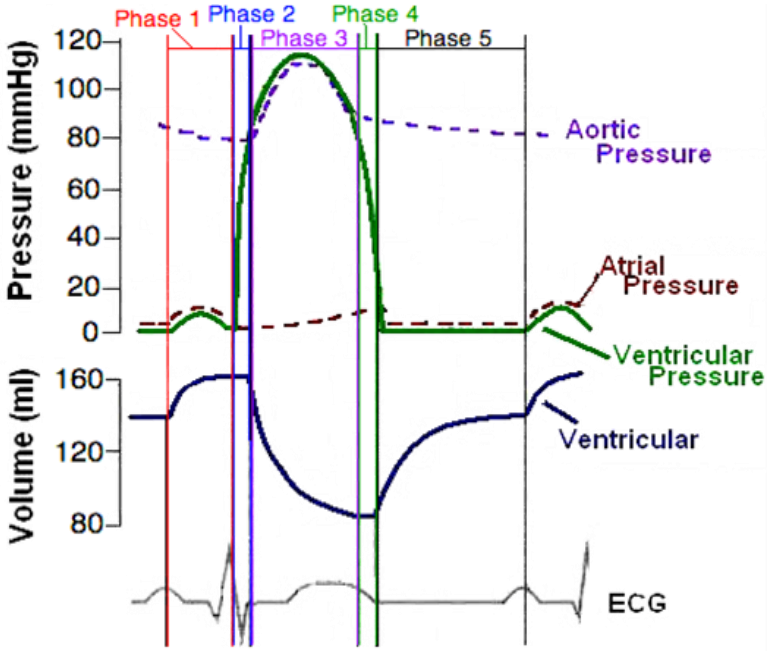
What can the ECG tell you about the health of the heart?]

- The extent and type of disturbances in rhythm and conduction
- The extent and location of myocardial damage
- Effects of drugs
- Heart rate

The Cardiac Cycle

- Shows all the mechanical and electrical events during a single contraction on the left side of the heart while the person is at rest
- Includes:
    - Pressure changes – in aorta, left atrium, left ventricle
    - Volume changes – in left ventricle
    - Valves opening and closing
    - ECG
- Consists of a period of systole (ventricle contraction [squeezing]) and a diastole (ventricular relaxation [dilation])
- Each cycle is initiated by the SA node
- Impulse spreads quickly throughout atria, is conducted into the AV node and then into ventricles
- Pause at the AV node, allowing atrium to contract before ventricles
    - Allows atrium to empty blood into ventricles prior to the ventricular contraction that empties blood into vascular system
- Blood always moves from high pressure to low pressure

Five Phases of Cardiac Cycle



1. Atrial systole
2. Early ventricular systole
   - Isovolumetric ventricular contraction
3. Ventricular systole
   - Rapid ejection period
4. Early ventricular diastole
   - Isovolumetric ventricular relaxation
5. Late ventricular diastole
   - Ventricular filling

1. Atrial Systole
   - P wave represents the depolarization of the atria → atria contract
   - Atrial pressure is greater than ventricular pressure (AV valves are already open)
   - Ventricles (continue to) fill with blood (last 30%) → end diastolic volume (EDV)

2. Early Ventricular Systole
   - QRS complex represents depolarization of ventricles
     - Ventricles begin contracting
     - Pressure builds in ventricles
   - Ventricular pressure exceeds atrial pressure → AV valve closes
   - NO change in volume (ventricular pressure < aortic pressure)

3. Ventricular Systole (rapid ejection period)
   - Ventricles continue to contract
   - Ventricular pressure exceeds aortic pressure
     - Aortic valve opens
     - Blood leaves ventricle
   - Ventricular volume decreases, however not all blood leaves ventricle
     - Left with end systolic volume (ESV)
   - T-wave

4. Early Ventricular Diastole (Isovolumetric ventricular relaxation)
    - Ventricles relax causing ventricular pressure to drop
    - Blood in aorta tries to flow back into ventricles → aortic valve closes
    - Ventricular pressure still greater than atrial pressure → no change in volume

5. Late Ventricular Diastole (ventricular filling)
    - Ventricles continue to relax
    - Pressure in ventricles drops below pressure in the atria → AV valve opens
    - Blood enters ventricles (70% of filling takes place)

CYCLE REPEATS

Contribution of Atrial Contraction to Ventricular Filling

- 70% of ventricular filling occurs when the ventricles relax (phase 5)
- Last 30% of filling occurs when atria contract (phase 1)
- Blood flows passively into the ventricles when the pressure in the atria exceeds that in the ventricles
- This pressure gradient begins during late ventricular diastole and when the ventricles are relaxing
- Continues until the atria have finished contracting (end of atrial systole)

Cardiac Output (CO)

= the amount of blood pumped by each ventricle in one minute

CO = heart rate (in beats per minute) X stroke volume (mL)

Stroke Volume (SV) = the amount of blood pumped by each ventricle during one contraction

- Normal resting CO value = 5L/min
    - Resting HR=70 beats/min
    - Resting stroke volume= 70-80 mL
- CO output can increase during exercise up to 20-40L/min
- Can be varied by changing the heart rate or stroke volume

The Control of Heart Rate

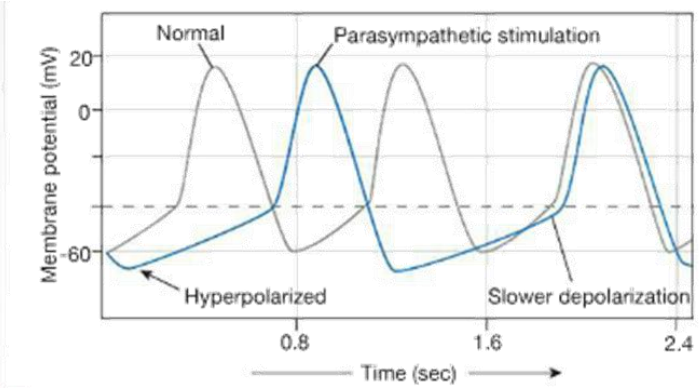
- Resting heart rate = 70 beats/min

max HR = 220 – age of person

- ANS exerts powerful control over heart rate as well as force of cardiac contraction
- The heart is supplied with both parasympathetic and sympathetic nerves
- Both the PSN and SNS change the slope of the pacemaker potential – but in different ways

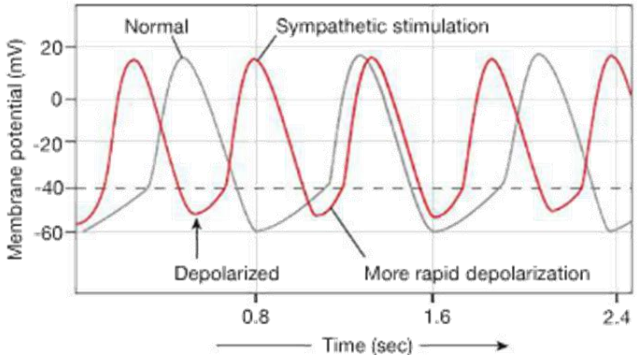
Parasympathetic System

- "Rest and Digest" system
- Decreases HR
- PNS innervates the SA and AV nodes via the *vagus nerve* (and to a lesser extent atrial/ventricular muscle)
- PNS releases the neurotransmitter Ach → increases K^+ efflux and decreases Ca^{2+} influx
    - Makes cell walls more permeable to potassium
- = Decreases slope of pacemaker potential



Sympathetic System

- "Fight or Flight System"
- Increases HR
- SNS innervates SA & AV nodes *and* ventricular muscle
- Releases norepinephrine (and hormone epinephrine from adrenal gland) → increase Na^+ and Ca^{2+} influx
    - Makes cells walls more permeable to sodium
- = increases slope of pacemaker potential



Overall Control of Heart Rate

When a heart rate less than 100bpm is required by the body (at rest), the PNS is activated. The more it is activated the slower the heart rate

At rest, the average heart rate is roughly 70 bpm; therefore there is always PNS activity at rest

This is called "Vagal Tone"

HR = 100 bpm → no PNS or SNS
HR < 100 bpm → PNS is active
HR > 100 bpm → SNS is active

	PNS	SNS
Neurotransmitter released	Acetylcholine	Norepinephrine
Active at?	<100 bpm	>100 bpm

Control of Stroke Volume

Stoke volume = the amount of blood pumped by each ventricle in one contraction

$$CO = HR \times SV$$

- At rest, SV is approximately 70 mL but can reach 110 to 200 mL during max. exercise

Major factors that control SV:

- Input from the autonomic nervous system
- Preload (end diastolic volume)

Parasympathetic Effects

- PSN releases NT acetylcholine
- Ach closes Ca^{++} channels
- Less Ca^{++} flowing into cardiac contractile cells
- Decreases force of contraction
- Decreased SV (and CO)

Sympathetic Effects

- SNS releases NT norepinephrine
- NE opens Ca^{++} channels
- More Ca^{++} flows into cardiac contractile cells
- Increases force of contraction
- Increases SV (and CO)

	Parasympathetic Nervous System	Sympathetic Nervous System
Neurotransmitter Release	Acetylcholine	Norepinephrine (epinephrine released by adrenal glands)
Ca++ Channels	Closes Ca++ channels in the contractile myocardial cells	Opens more Ca++ channels in the contractile myocardial cells
Ca++ into Cardiac Contractile Cells	Less Ca++ enters the contractile myocardial cells	More Ca++ enters the contractile myocardial cells
Force of Contraction	Decreases the force of contraction	Increases the force of contraction
Stroke Volume	The SV is decreased	The SV is increased.
Cardiac Output	The Cardiac Output decreases.	The Cardiac Output increases
Examples	CO = SV (60 mL) **x** 72 bpm = 4.32L	CO = SV (90mL) **x** 72 bpm = 6.48L
INNERVATION	SA and AV node LESS on atrial & ventricular muscles	SA and AV node MORE on ventricular muscle

- During diastole, volume of each ventricle increases to about 160 mL
  - =end-diastolic volume (EDV)
- During systole, the ventricles empty about 70 mL of blood into aorta
  - =stroke volume
- What remains in the ventricle after it has contracted is about 90 mL
  - =end-systolic volume (ESV)

Stoke volume = EDV – ESV

Where EDV = volume of blood in ventricles at end of diastole (just before contracting)

And ESV = volume of blood in ventricles at the end of systole (just after contracting)

- Activation of the SNS (exercise/stress) will cause the heart to contract more forcefully and more blood will be ejected during systole
    - ESV will decrease to as low as 10-30 mL
- When large amounts of blood flow into ventricles
    - EDV will increase to as high as 200-250 mL
- By increasing EDV and decreasing ESV, the stroke volume can be increased

Preload Effects

- Preload = the "load" on the cardiac muscle (ventricles) just before contraction
- Comes from the blood in the ventricles that stretches the ventricular muscle
- Therefore, increased EDV → increased preload → increased SV

Increased EDV = Increased preload

↓

Increases in stretch of myocardial cells

↓

Increases the force of contraction of these cells when the heart contracts

↓

Increases amount of blood ejected

↓

Increases stroke volume

Frank-Starling Law of the Heart

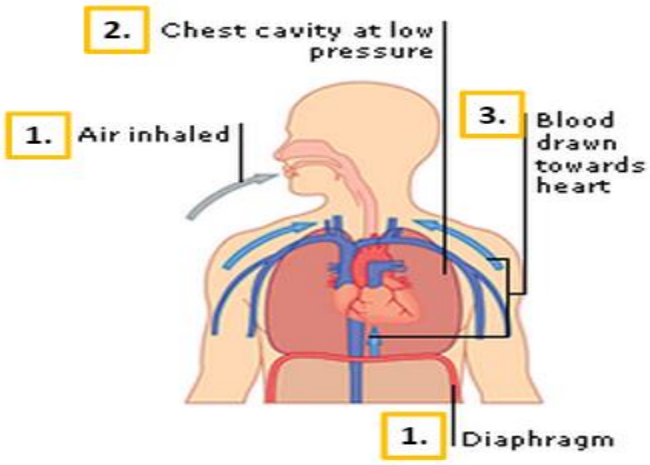
= An increase in end diastolic volume (EDV) will cause an increase in stroke volume (SV)

Venous Return to the Heart

Mechanisms contributing to increase venous return & increase in EDV (preload)

During dynamic exercise:

1. Muscle Pump
2. Respiratory Pump
    - Pressure differences help increase venous return during inhalation



3. Sympathetic Nervous System
    - Increased HR and SV → small constriction (squeeze) of veins

Venous Return & Cardiac Output

Increased venous return = fuller ventricles

↓

Increased EDV

↓

Increased preload (force on myocardial cells)

↓

Increased force of contraction (because of stretch)

↓

Increased stroke volume

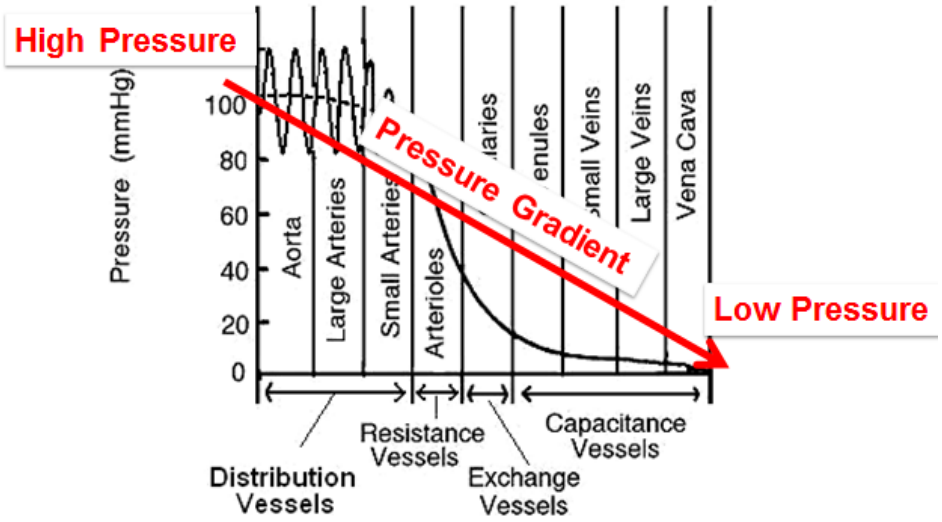
↓

Increased cardiac output

Pressure, Flow and Resistance (throughout circulation)

- The pressure change (or pressure gradient) in the circulatory system drives blood through vessels
- Blood flows from *high pressure (P1)* to *low pressure (P2)* down a pressure gradient
- Blood flow is decreased by resistance encountered by the blood

$$\text{Blood flow} = \frac{\text{pressure gradient}}{\text{resistance}} \quad \text{OR} \quad \text{blood flow} = \frac{P1 - P2}{\text{resistance}}$$



$$\text{Resistance} = \frac{L\eta}{r^4} = \frac{1}{r^4}$$

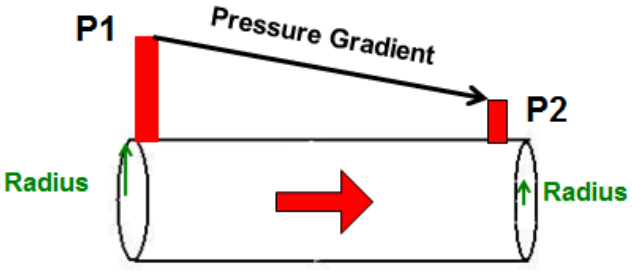
Where,

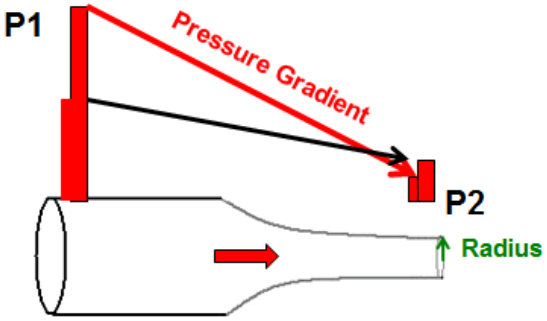
L = length of vessel
η = viscosity of the fluid
r = radius of vessel

- The drop in pressure from high (in aorta) to low (in veins) is what causes the blood to flow through the circulation
- Therefore, **blood flow** is affected by the **pressure change** between two points (P1 and P2) and the **resistance** the blood encounters as it moves through the circulatory system

Blood flow = (P1 – P2) X r^4

Constriction in a Blood Vessel





- Even though the pressure gradient has increased, the constriction causes a decrease in blood flow because the smaller radius has a larger effect on blood flow