The Cardiopulmonary System

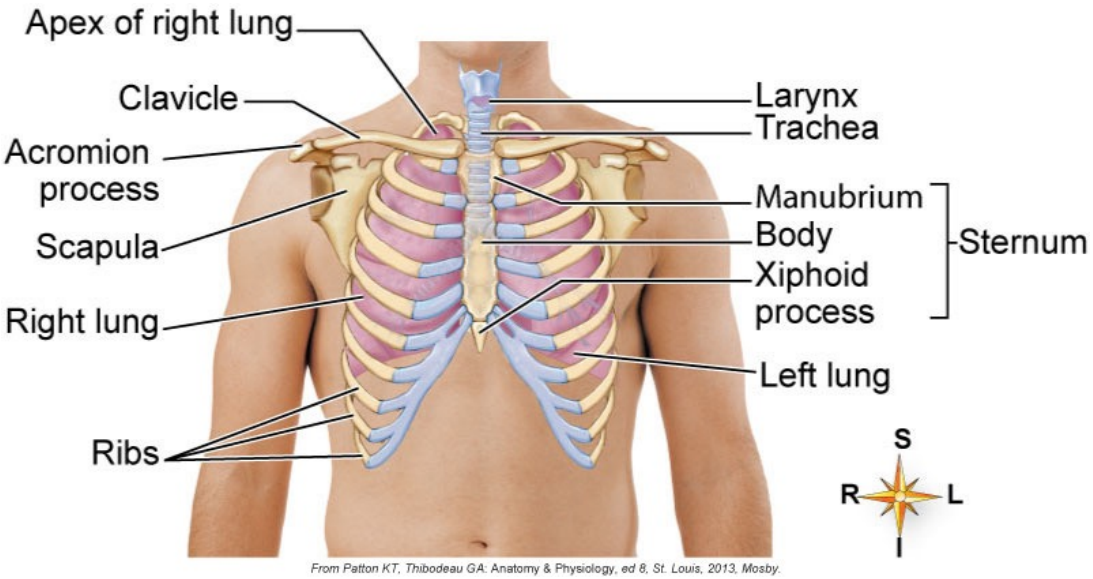
Robertson Chapter 11

The Thorax

- Region of chest formed by sternum, thoracic vertebrae, and ribs
- Thoracic cavity contains heart, greater and lesser blood vessels, lungs, trachea, esophagus, thymus gland, lymphatic structures, and many nerves
- Thoracic cage consists of sternum, ribs, intercostal muscles and thoracic vertebrae

The Thorax

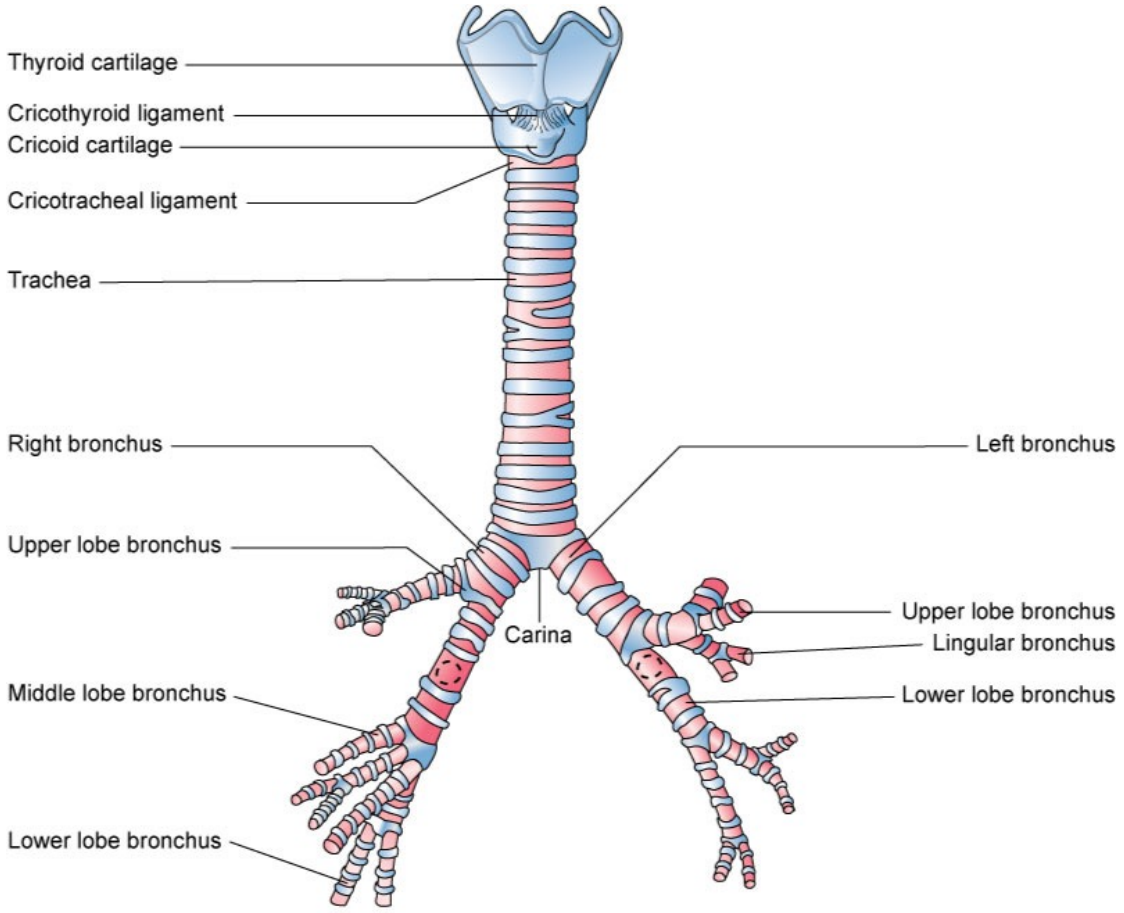
- Thoracic cage
  - 12 vertebrae connected to 12 ribs
    - Top 7 attached to sternum via cartilage
    - Lower 5 not attached to sternum
      - “Floating” ribs



Lungs

- Provide more than 70 sq. meters of epithelial tissue surface area for gas exchange
- Right lung
  - 3 lobes: Upper, middle, and lower
- Left lung
  - 2 lobes: Upper and lower
- Lungs are very elastic/dynamic
- For ventilation, need open (patent) airway
- 2 basic areas of airway:
  - Extrathoracic = Upper airway
  - Intrathoracic = Lower airways

Major Airways of Tracheobronchial Tree



From Hicks GH: Cardiopulmonary anatomy and physiology, *Philadelphia, 2000, Saunders.*

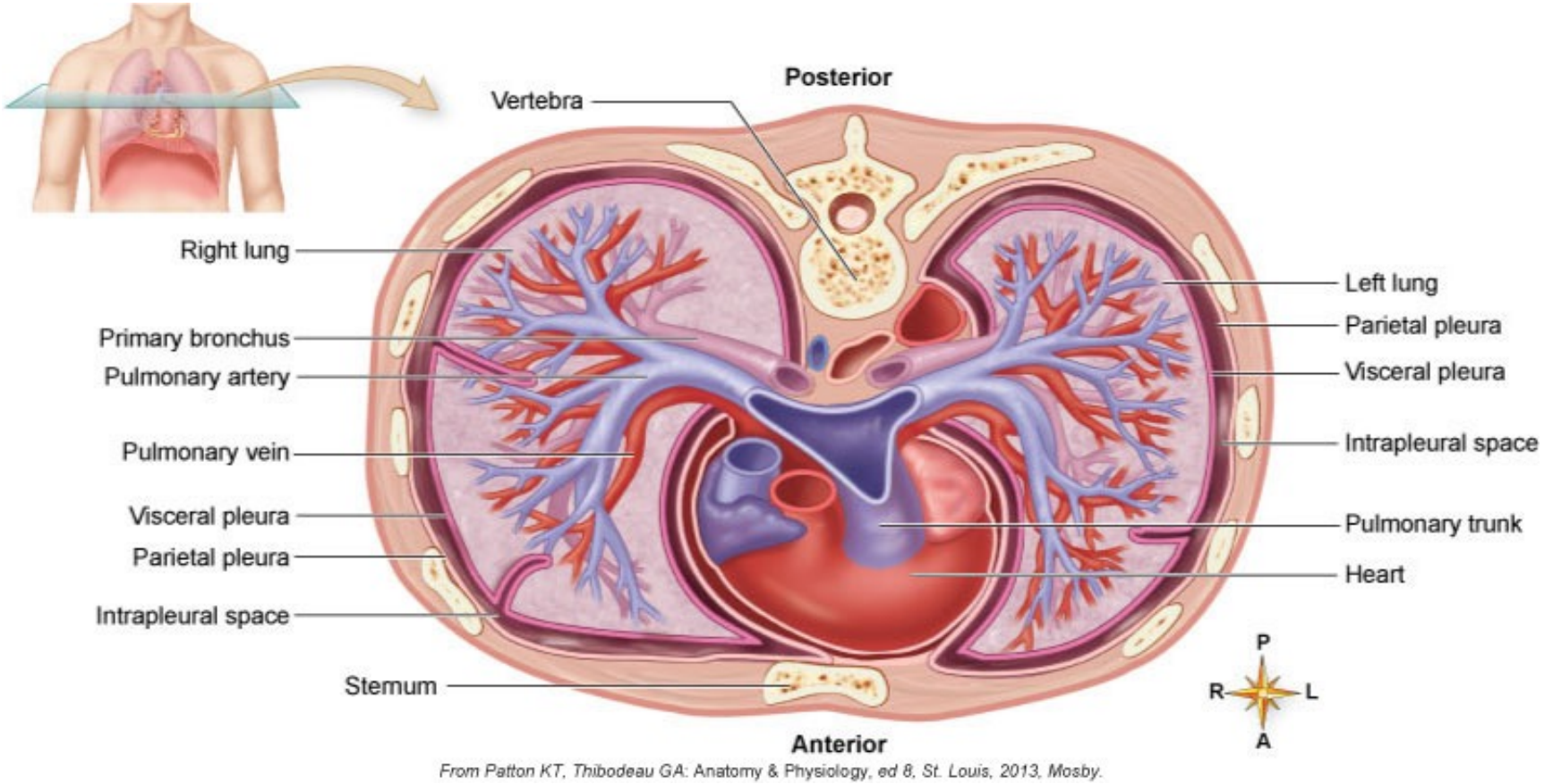
Lungs

- Air moves in and out of lung via upper airway through nose and nasopharynx and mouth and oropharynx
  - Nasopharynx = Uppermost area of back of throat
    - Begins with superior level of soft palate and extends into posterior portion of nasal cavity
  - Oropharynx = Oral portion of pharynx
    - Back of throat from soft palate down to upper edge of epiglottis
- Air moves down into trachea
- Trachea splits into 2 bronchi: Left mainstem and right mainstrem, which further divide into lobar bronchi and then into segmental bronchi

Lungs

- Airways continue to divide into bronchioles, which terminate into alveoli
- Alveolar sacs and abutting capillaries are where gas transfer occurs
  - Approximately 300,000,000 of these are present in normal adult human lung
- Diffusion = Exchange of O2 and CO2
- Lung is encased by pleura
  - Visceral pleura attached to surface of lung
  - Parietal pleura line wall of thorax
  - Pleural cavity/space = Slim space between visceral and parietal pleurae

Lungs and Pleura



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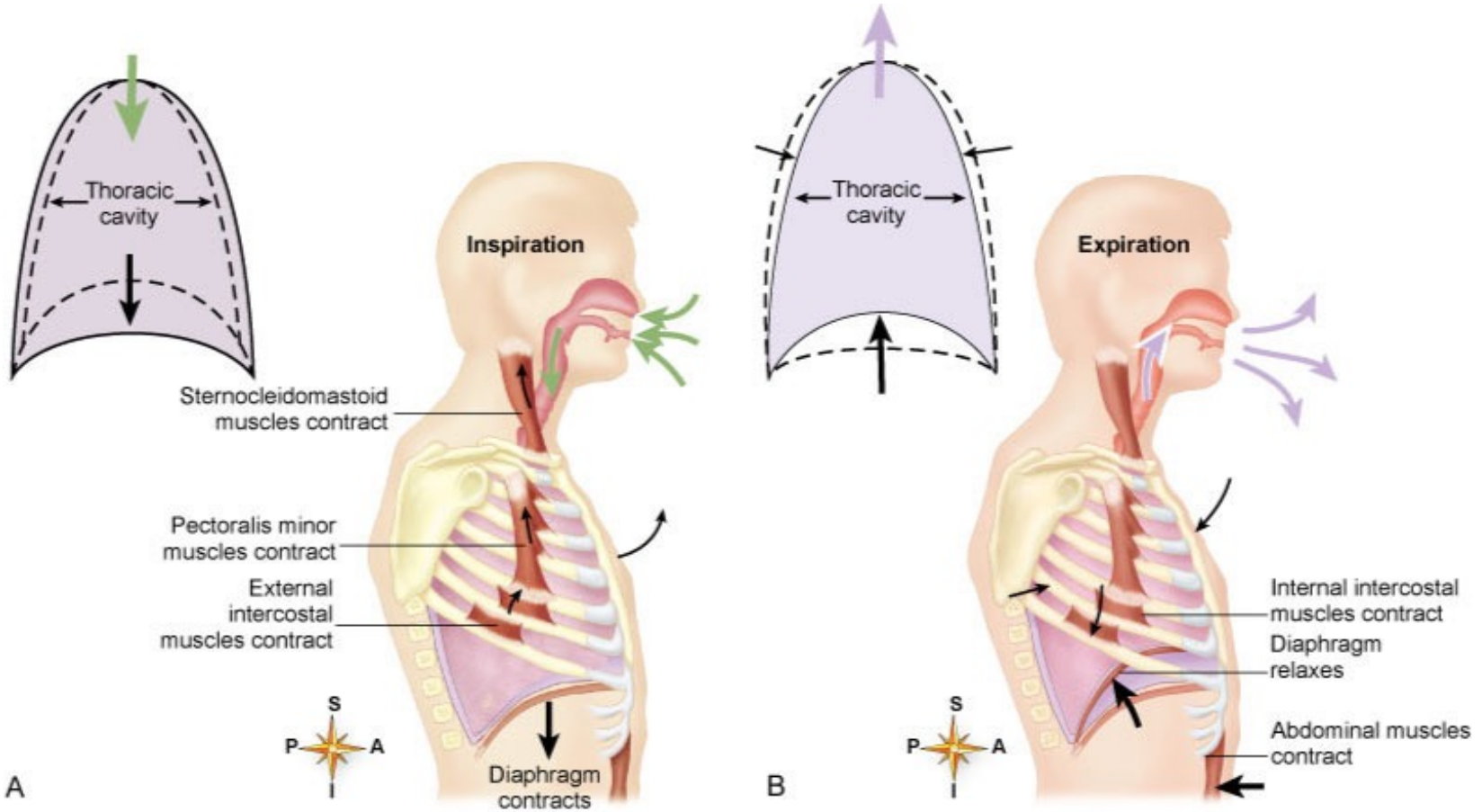
Muscles of Ventilation

- Diaphragm located below lungs
  - Primary muscle of ventilation
- Inhalation is active muscular event
  - Diaphragm moves downward, increasing size of lungs
  - This causes pressure inside thorax to become negative as compared to ambient pressure outside the body
  - Ambient air contains 21% O2
- Exhalation is passive muscular event
  - Occurs when diaphragm relaxes and lungs recoil back and push gases out into atmosphere

Inspiration and Expiration



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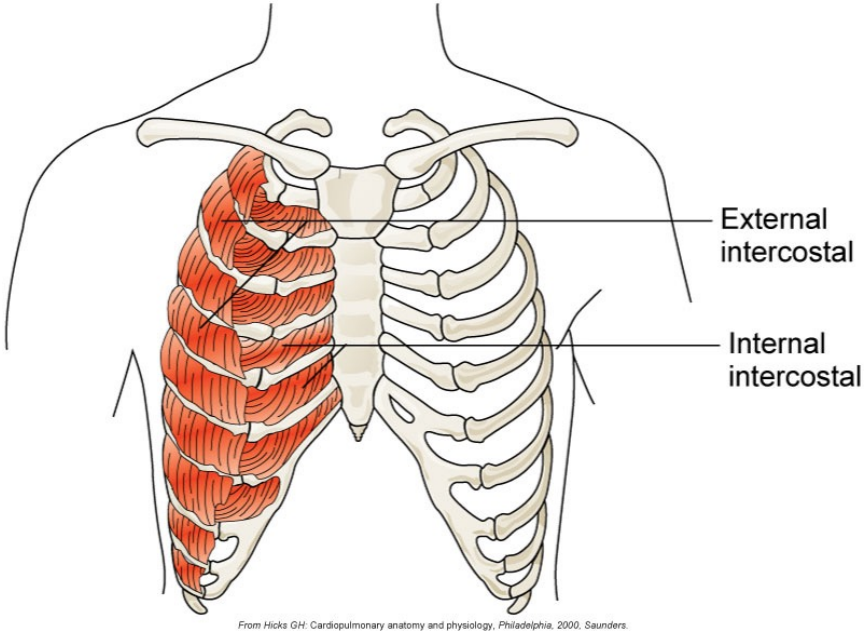
Accessory Muscles

- Accessory muscles recruited when body needs additional ventilation for gas exchange over baseline respiration
  - Example: Exercise, asthma attacks



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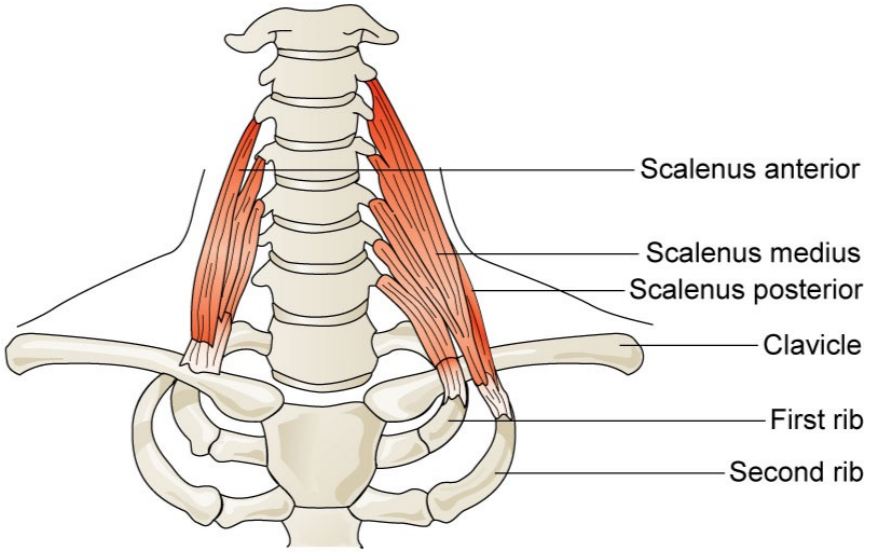

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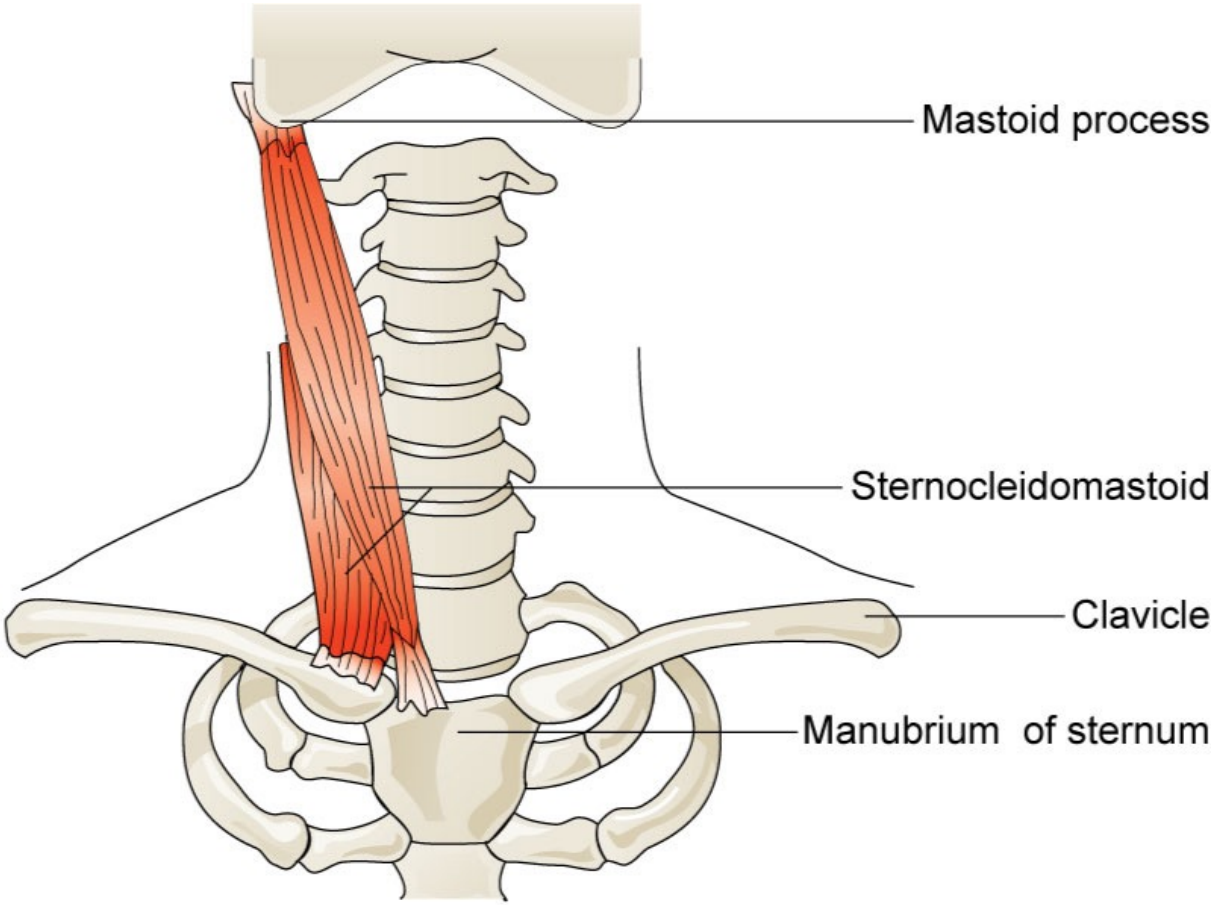
Accessory Muscles



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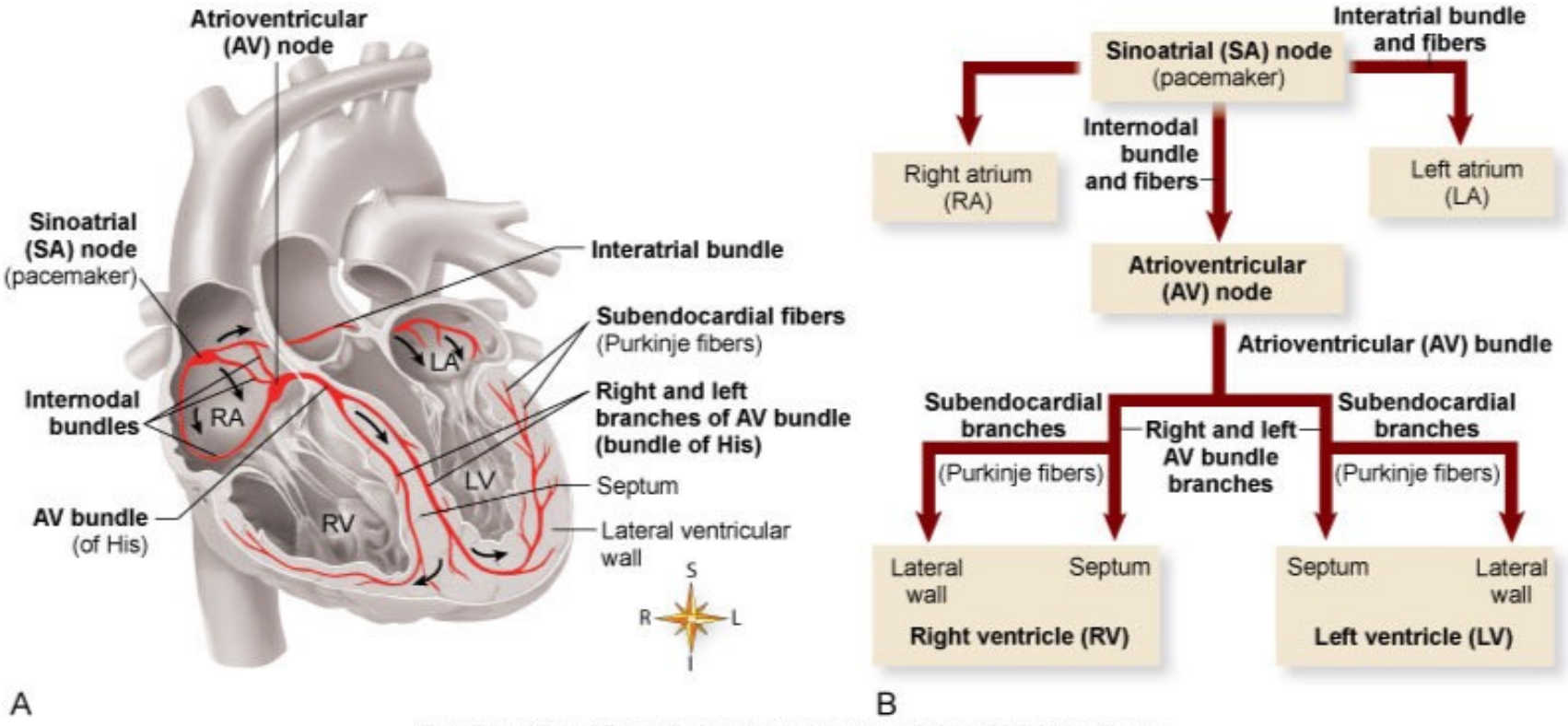
Heart

- Adult heart is about size of a closed fist
  - Sits in thorax on left side of chest in front of lungs
- Has four chambers
  - Right atrium
  - Left atrium
  - Right ventricle
  - Left ventricle
- Atria = Upper chambers
  - Smaller, less muscular
- Ventricles = Lower chambers
  - Larger, more muscular
  - Wall of LV is about twice as thick as RV
  - LV pushes blood through body
  - RV pushes blood through lungs

Heart

- Has four valves
  - Tricuspid is between RA and RV
  - Pulmonary is between RV and pulmonary artery
  - Bicuspid/Mitral is between LA and LV
  - Aortic is between LV and aorta

Conduction System of the Heart



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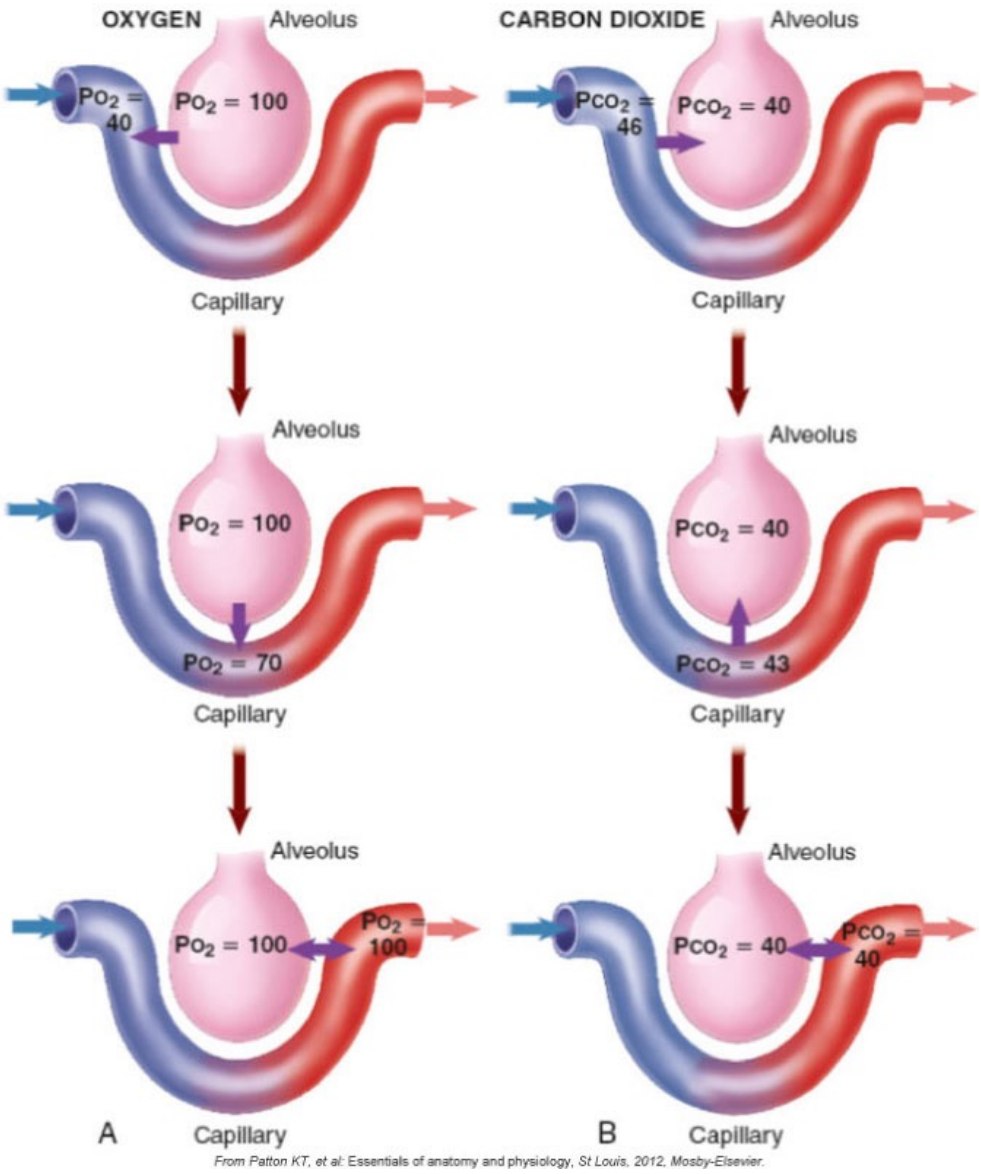
Oxygenation of the Blood

- RBCs carry O2 throughout body via Hb
- Venous system carries deoxygenated blood back to right side of heart
- RV pumps blood into pulmonary artery to both lungs
- Freshly oxygenated blood is returned to left side of heart to be pumped throughout body
- Veins carry deoxygenated blood to heart
- Arteries carry oxygenated blood away from heart
  - Pulmonary artery is only artery that carries deoxygenated blood
  - Pulmonary veins are only veins that carry oxygenated blood

Ventilation and Diffusion

- Internal respiration happens at cellular level
- O2 is inspired from atmosphere, diffused across alveolar-capillary bed into bloodstream, and delivered to cells
- CO2 is transported at cellular level and returns to alveolar-capillary bed where it off-loads and is exhaled
- Gas diffusion occurs when an area of high concentration is exposed to an area of low concentration

Pulmonary Gas Exchange



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Oxygen Transport

- RBCs are responsible for O2 transport in blood
  - Normal RBC has four Hb binding sites
    - If all four saturated with O2, it is 100% saturated
    - Normal O2 saturation is 95-100%
- O2 levels can be assessed by ABGs or pulse oximetry
- % of Hb with O2 is affected by:
  - Amount of O2 being delivered to blood
  - Acid-base balance of blood
  - Temperature of blood
  - Amount of Hb in blood
  - Amount of O2 required by tissues

Carbon Dioxide Transport

- Process of transporting CO_2 dissolved in blood plasma, RBC, or plasma protein to be released by lungs
  - About 5% of produced CO_2 is dissolved in blood plasma
  - 25% of CO_2 is transported bound to RBC Hb or plasma protein
  - Remaining 70% is transported via bicarbonate buffering system

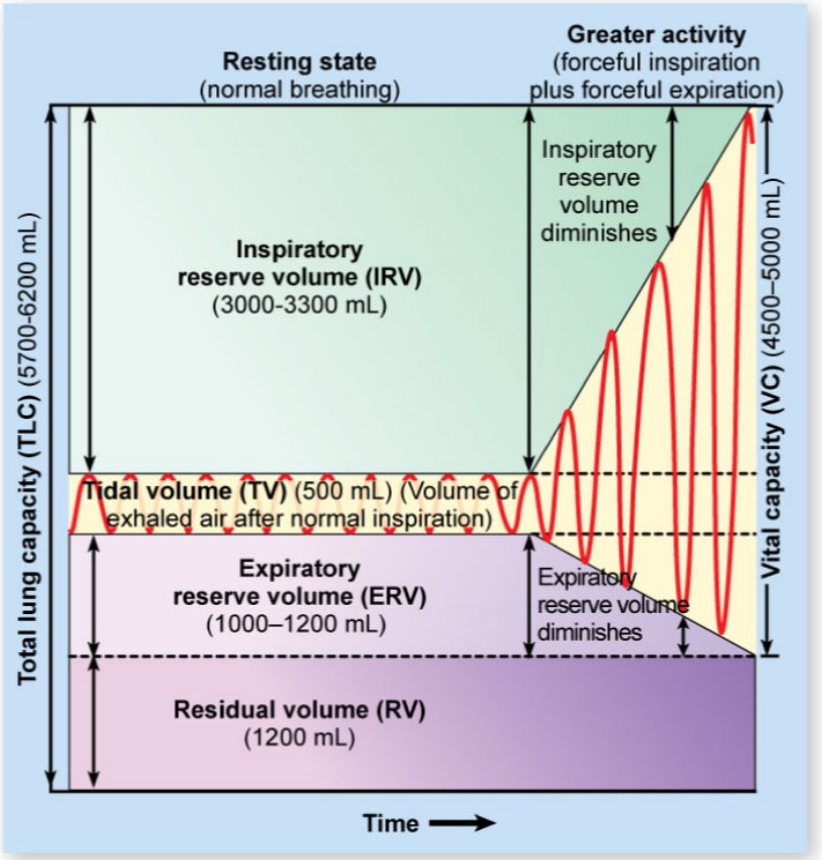
Lung Volumes and Capacities

- Measured in liters
- Capacity = 2 or more volumes added together
- Tidal volume (VT) = Volume of gas inspired and expired with each normal breath
- Inspiratory reserve volume (IRV) = Max volume that can be inspired above VT
- Expiratory reserve volume (ERV) = Max volume that can be expired after tidal breath exhalation

Lung Volumes and Capacities

- Residual volume (RV) = Volume that remains in lungs after maximal expiration
- Inspiratory capacity (IC) = VT + IRV
- Vital capacity (VC) = VT + IRV + ERV
- Functional residual capacity (FRC) = Amount of gas remaining in lungs following a normal tidal breath exhalation
  - ERV + RV
- Total lung capacity (TLC) = IRV + VT + ERV + RV

Pulmonary Ventilation Volumes and Capacities



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ABG Interpretation and Manipulation

- ABG = Gold standard for monitoring adequacy of ventilation and gas exchange
- Report ABGs in following order:
  - pH
  - $PaCO_2$
  - PaO_2
  - HCO_3^-
  - Base excess
  - SaO_2

ABG Values

Data	Definition	Normal Value
pH	Acidity or alkalinity of blood	7.35-7.45
PaCO2	Partial pressure of dissolved CO2 in blood	35-45 mm Hg
PaO2	Partial pressure of dissolved O2 in blood	80-100 mm Hg
HCO3	Plasma bicarbonate calculated from pH and PCO2	22-26 mmol/L
BE	Base excess (deficit) buffer produced by body	+3 to -3

ABG Interpretation and Manipulation

- Acidotic
  - pH < 7.35
  - PaCO2 > 45
  - HCO3- < 22
- Alkalotic
  - pH > 7.45
  - PaCO2 < 35
  - HCO3- > 26
- If pH normal but all other measures are not normal, this is compensated or chronic acid-base balance
- If pH abnormal, it is acute or uncompensated

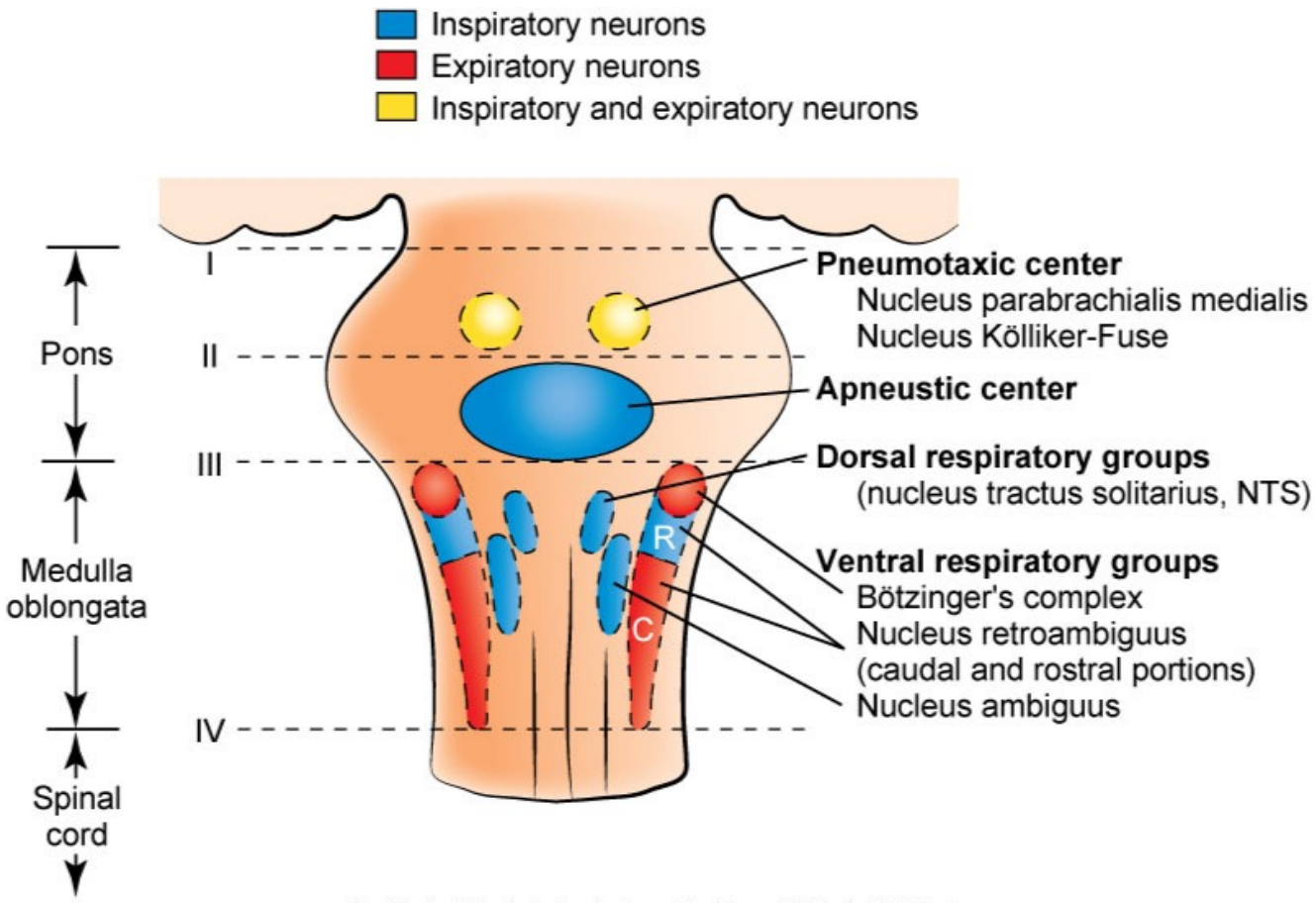
Regulation of Breathing

- ANS regulates respiratory function via:
  - Central chemoreceptors
  - Peripheral chemoreceptors
- Midbrain region houses respiratory center
  - Contains at least 3 sites sensitive to CO_2 in the CSF
  - Central chemoreceptors in medulla detect levels of CO_2 in blood
  - Increased CO_2 in CSF = Increase in concentration of hydrogen ions
    - Leads to decrease in CSF and blood pH
      - Decreased pH result of increased CO_2 concentration
    - Central chemoreceptors stimulate inspiratory center in response, sending signals to respiratory muscles
      - Increase in VT depth and RR, triggering heart to move blood

Regulation of Breathing

- Peripheral chemoreceptors = Aortic or carotid bodies
  - Extremely sensitive to decreases in blood O2 level
  - Stimulates respiratory center to take in breath
- Microscopic stretch receptors
  - Activated when lungs become distended
  - As lungs stretch, these receptors fire more frequently
    - These signals inhibit medullary cells, decreasing inspiratory stimulus
  - Breathing during wake controlled by voluntary and behavioral elements, chemical factors, and stretch receptors

Dorsal View of Brainstem



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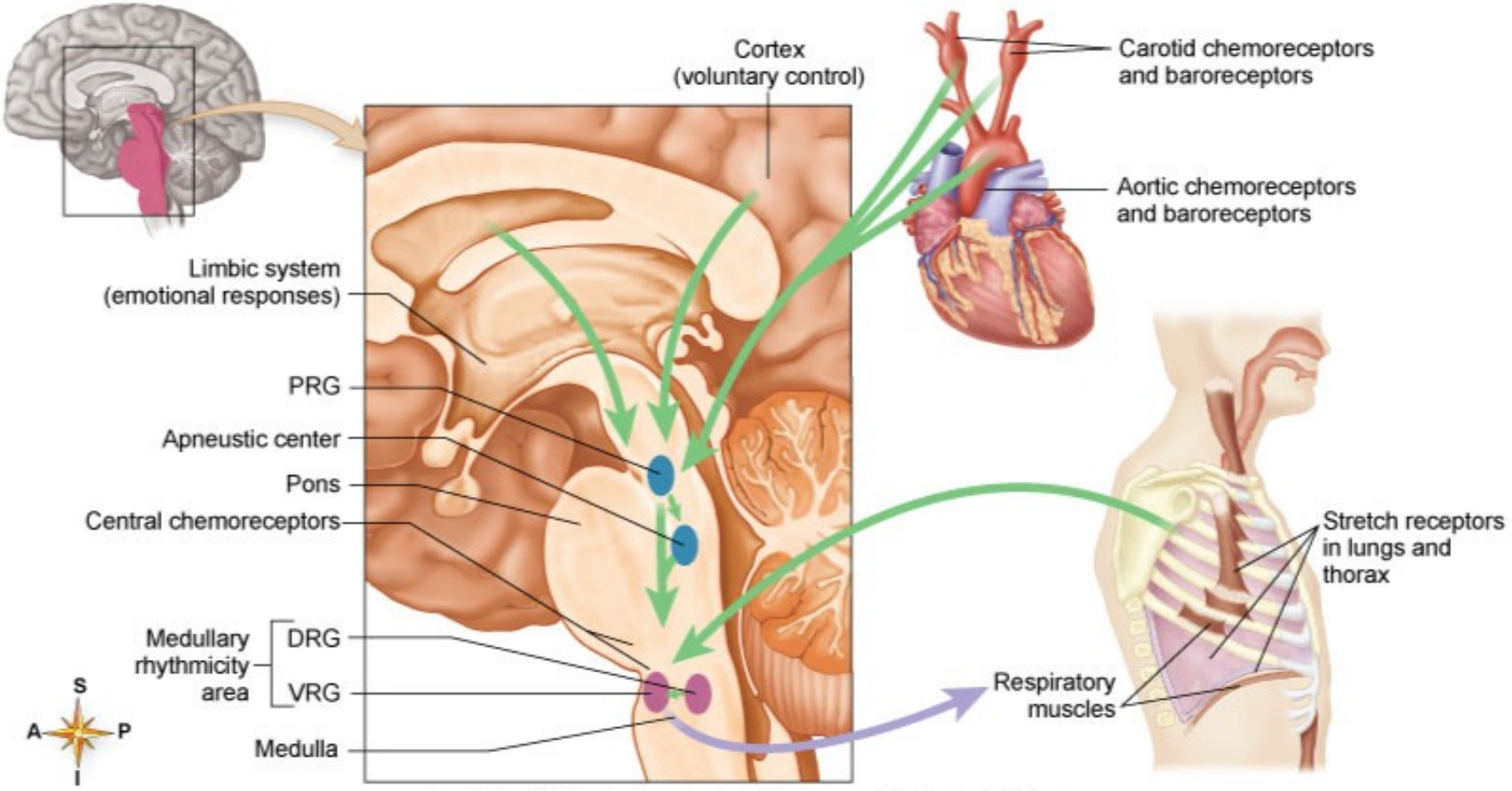
Regulation of Breathing



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Changes During Sleep

- Loss of voluntary control
- Decrease in usual ventilatory response to low O2 and high CO2 levels
- Hypoxemic and hypercapnic responses most depressed during REM
- Breathing during sleep depends on autonomic respiratory control system
  - Clear alterations in VT, alveolar ventilation, ABGs, and RR and rhythm

Changes in VT and Alveolar Ventilation

- During NREM
  - Breathing very regular in frequency and amplitude
  - Minute ventilation falls by 0.4-1.5 lpm
  - Reduction in VT, frequency, or both
- During REM
  - Irregular breathing
  - Sudden changes in frequency and amplitude of breaths
  - Similar minute ventilation reduction to NREM
- Changes in ABG values
  - $PaCO_2$ rises by 3-7 mm Hg
  - PaO_2 decreases by 3.5-9.4 mm Hg
  - SaO_2 decreases by < 2%

What Causes Hypoventilation in Sleep?

- Absence of tonic influence on brain (wake stimuli)
- Reduced sensitivity to hypoxemia and hypercapnia
- Increased upper airway resistance caused by reduced activity of pharyngeal dilator muscles in sleep
- Decreases in O2 consumption and decreases in CO2 production

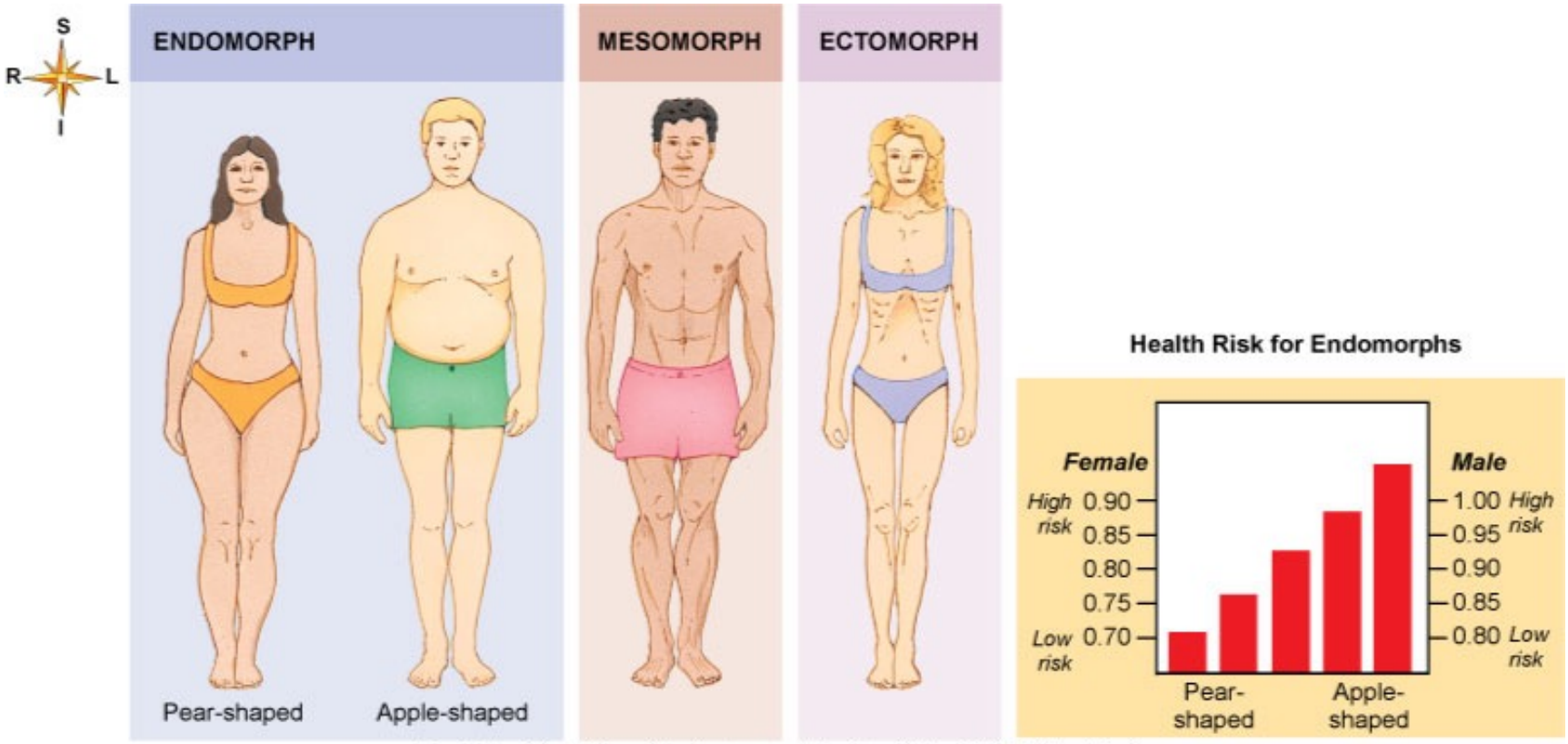
Respiratory Status

- Normal RR = 12-20 breaths/minute
- Normal HR = 60-100 bpm
  - Normal HR during sleep = 40-90 bpm
- Normal SpO2 = > 90%
- I:E ratio = 1:2 or 1:3
  - 1 second inhaling
  - 2-3 seconds exhaling
  - Example: 1:2 ratio
    - If RR is 10 breaths/minute:
      - 2 seconds inhaling
      - 4 seconds exhaling

Body Habitus

- “Physique” or “build”
- Adult body habitus types:
  - Overweight = Endomorphic
  - Normal = Mesomorphic
  - Underweight = Ectomorphic
- Excess body weight can affect breathing

Body Habitus



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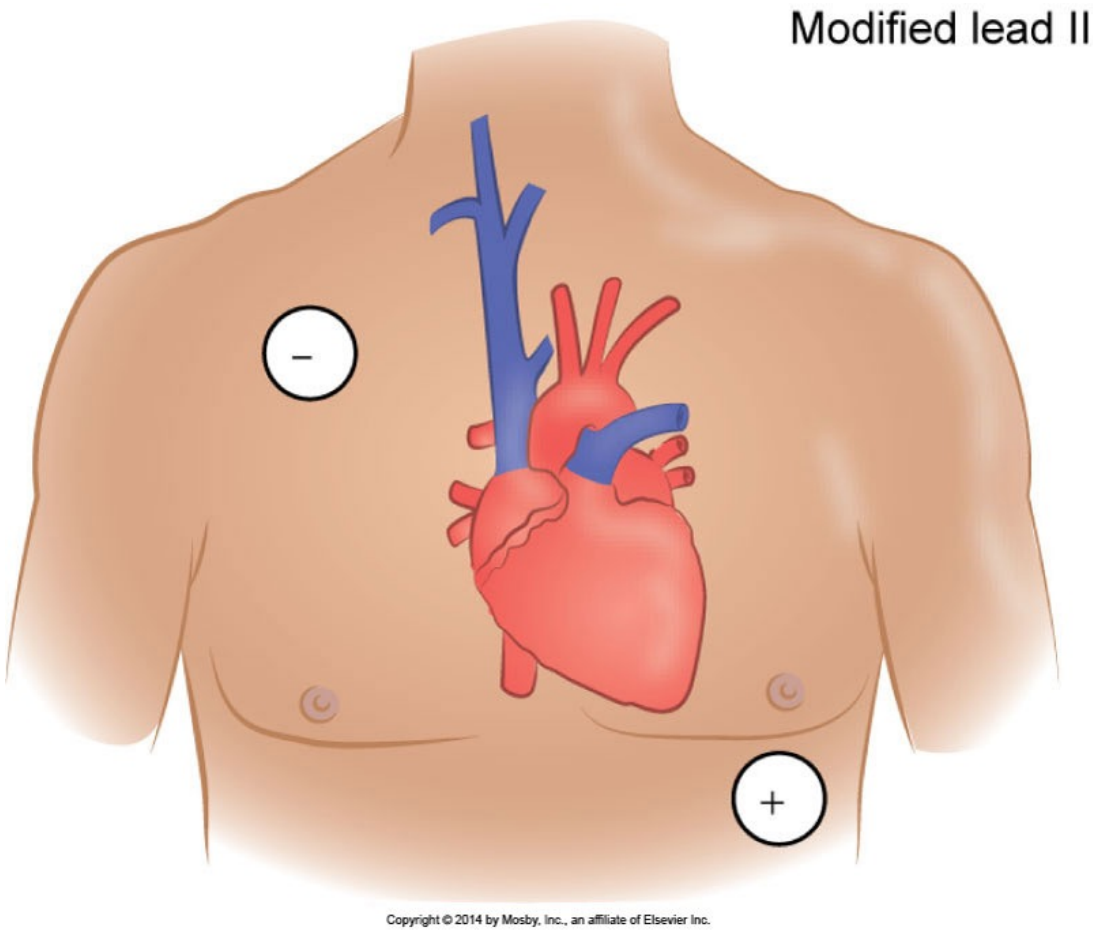
Skin Tone

- 1st: Look at whites of eyes
  - Yellow tinged could indicate liver problem
- Is skin pink or blue?
  - Bluish skin is sign of cyanosis, an indication of tissue hypoxia
    - Assessment includes inspection of oral mucosa, lips, nail beds, gums, palpebral conjunctivae, base of tongue, and palms
    - Typically seen when oxyhemoglobin is ≤ 60%

Modified Lead II Derivation

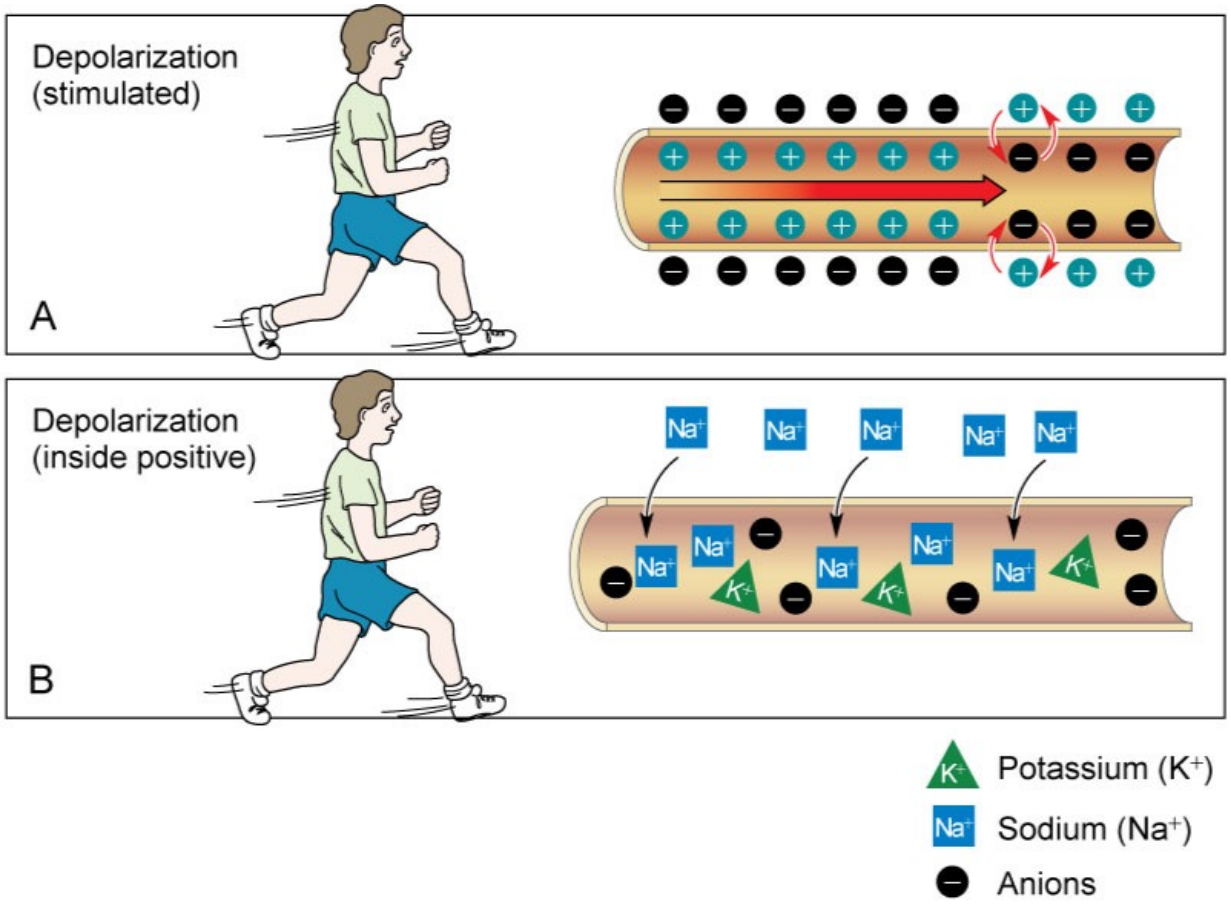
- Place ECG electrodes on right upper chest region and left lower thorax region
- Leads are polarized and improper placement will lead to inverted tracings



Electrical Pathway of Heart

- Depolarization = Fundamental electrical event of heart
  - Outside of cardiac cell more positively charged than inside in resting state
  - Contraction stimulated when sodium potassium pump allows more sodium and potassium to enter cell, creating a change in electrical balance
- Repolarization happens when sodium potassium pump expels sodium and potassium back out of cell to return to resting

Depolarization



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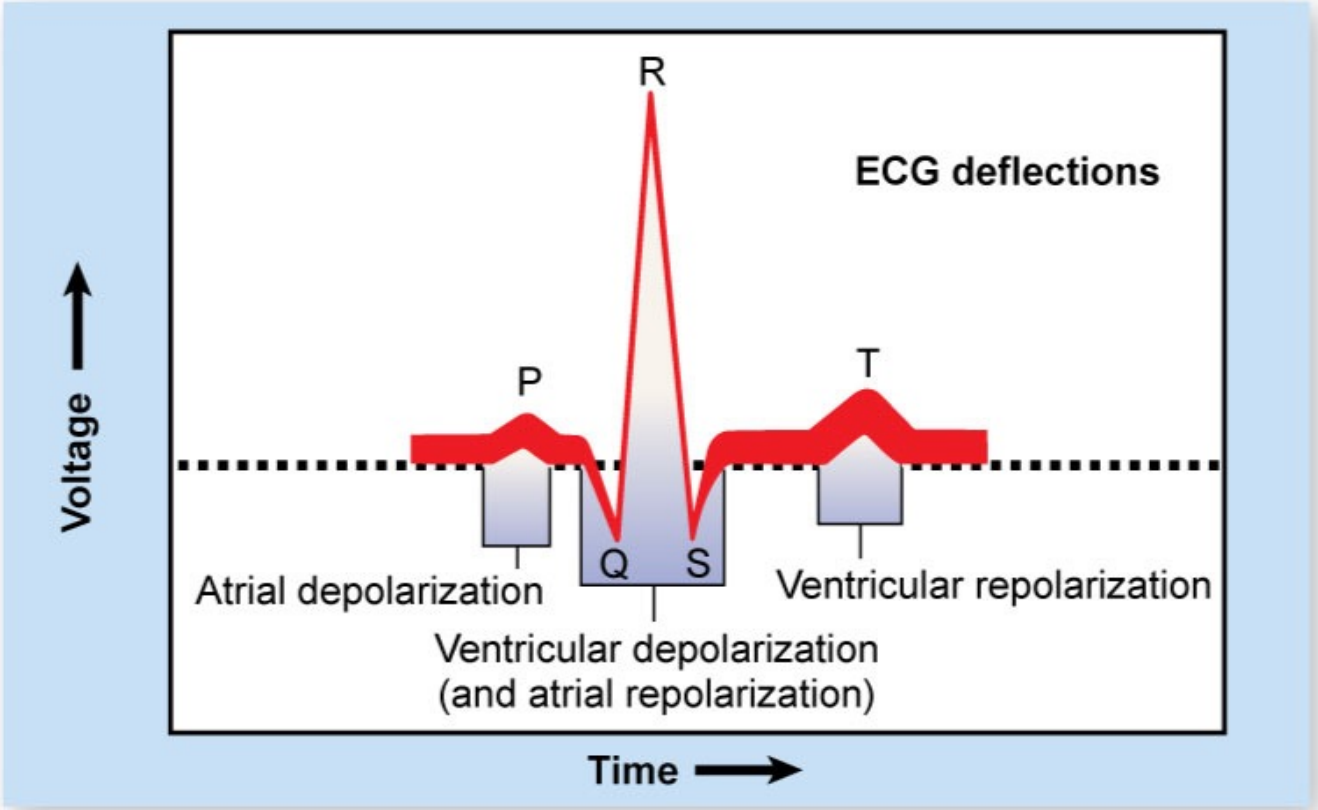
Electrical Pathway of Heart

- SA node = Pacemaker of heart
  - Rate is between 60-100 bpm
- Electrical heartbeat starts in SA node
  - Travels across Bachmann bundle to LA and through intranodal pathways to AV node
    - This causes P wave
  - Charge briefly “rests” in AV node area (return to baseline)
    - AV node has rate of 40-60 bpm
      - Backup pacemaker for SA node
  - Continues from AV node down through bundle of His and splits into left and right bundle branches
  - Continues down these paths until discharged at end of Purkinje fibers, causing ventricles to contract
    - Ventricular rate = 20-40 bpm

PQRST Complex

- P wave = Depolarization of atria
  - Originates from SA node typically
- QRS complex = Depolarization of ventricles
- T wave = Ventricular repolarization
- Atrial repolarization not seen because it happens at same time as ventricles depolarizing
  - Hidden in QRS

ECG



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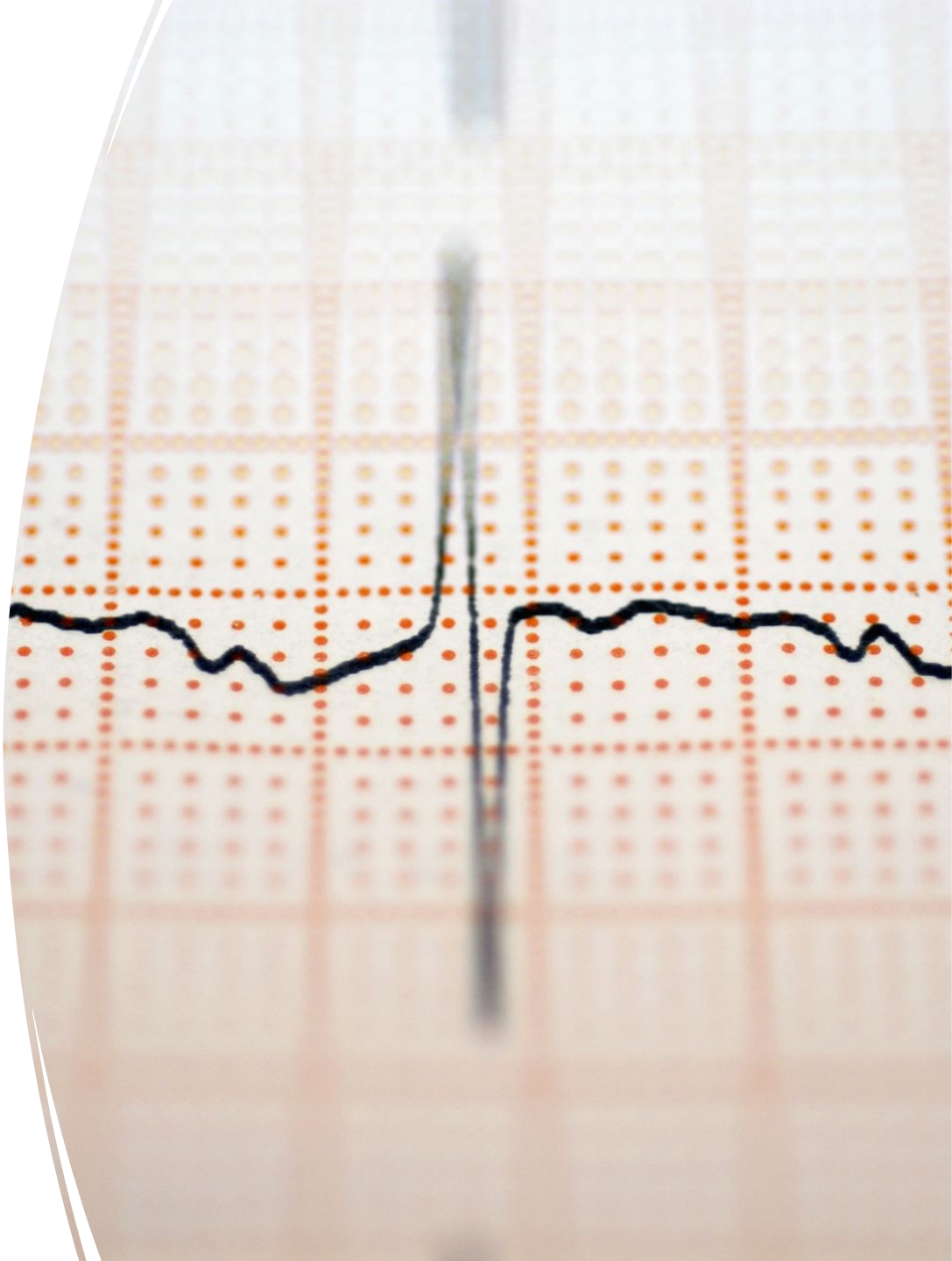
Dysrhythmia Interpretation

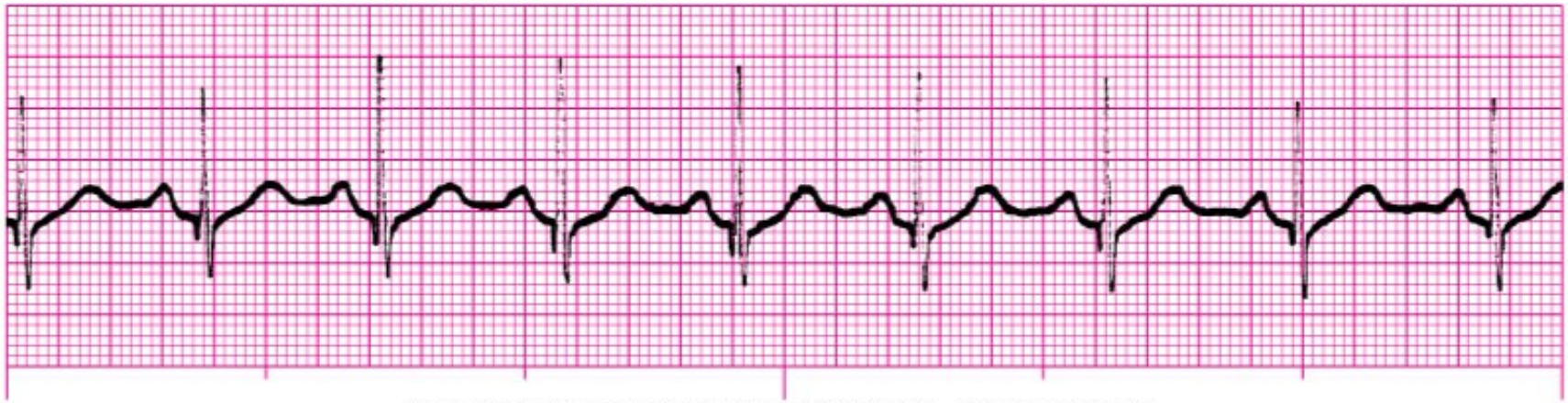
- Can be broken down into 5 sections:
  - Sinus mechanisms
  - Atrial rhythms
  - Junctional rhythms
  - Ventricular rhythms
  - AV conduction delays
- Contact physician if patient becomes symptomatic even if ECG looks normal

Dysrhythmia Interpretation

- Look at:
  - Rate
  - Is rhythm regular?
  - What are atria doing?
  - What are ventricles doing?
  - Relationship between atria and ventricles



Normal Sinus Rhythm



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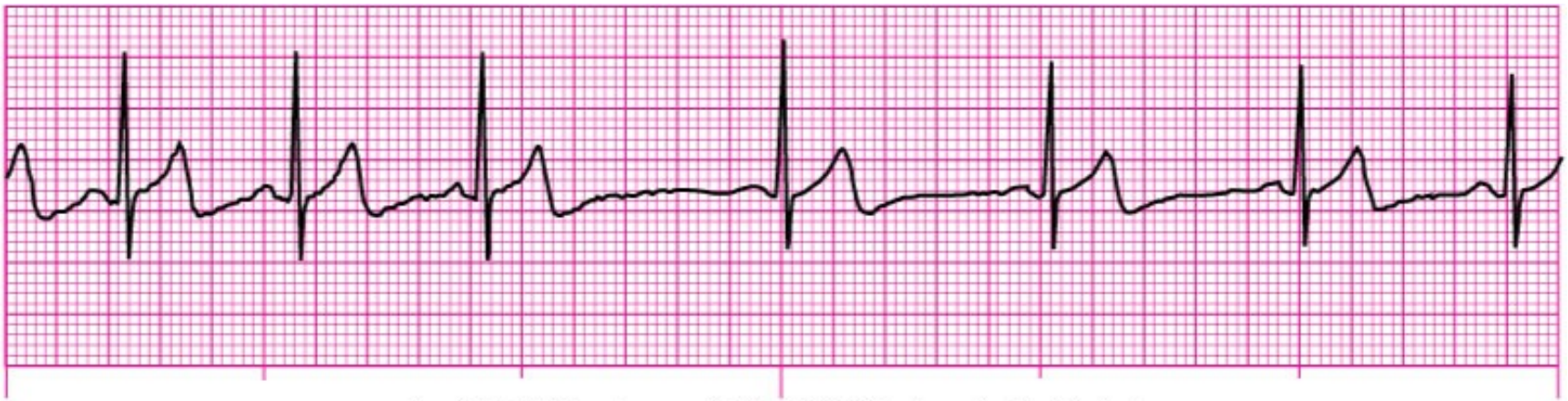
Sinus Arrhythmia

- Initiated at SA node but rate increases with inspiration and decreases with expiration
- Normal finding in kids and young adults



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Sinus Bradycardia

- Rate < 60 bpm
- Common in athletes
- Observed but not treated

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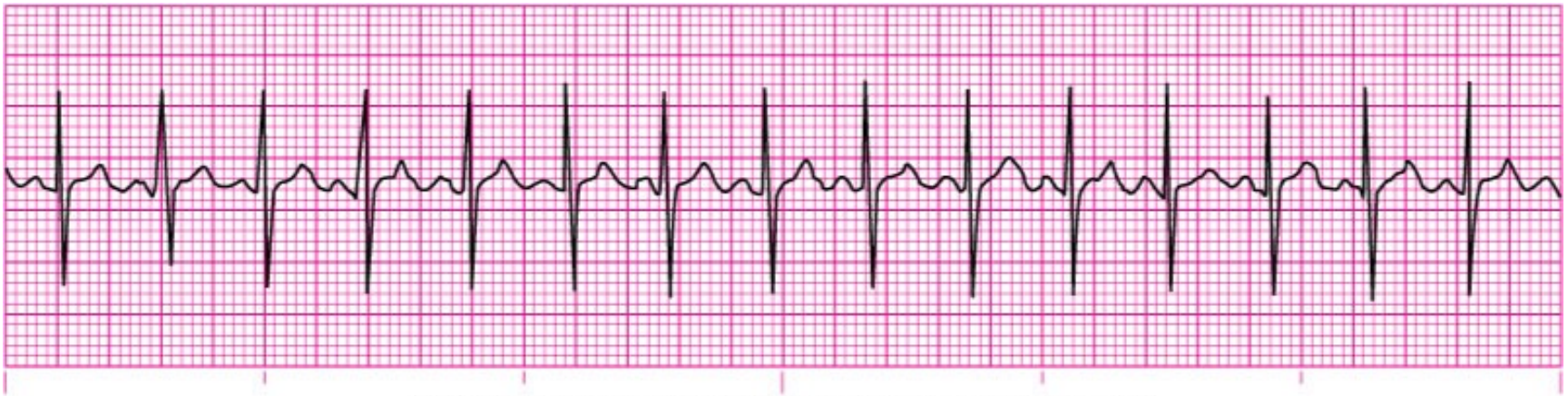
Sinus Tachycardia

- Rate > 100 bpm
- Normal response to exercise and other stressors
- May accompany hypoxia, infection, fever, heart condition, caffeine use, nicotine use, or decongestants
- If patient blue, labored breathing, or chest pain, call 911
  - But usually just document and monitor.



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Sinoatrial Block / Sinoatrial Pause

- When SA block/pause happens, one PQRST will be missing

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Sinus Arrest

- Not the exact distance of other P-P intervals
- More than one PQRST missing
- Periods of sinus arrest as long as 9 seconds have been reported in young people with normal cardiac function
    - Can be sign for later life-threatening ventricular arrhythmias

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PAC

- Impulse arises as an ectopic atrial focus (outside SA node)
- Results in abnormal P waves
- May be perceived as “skipped” beat

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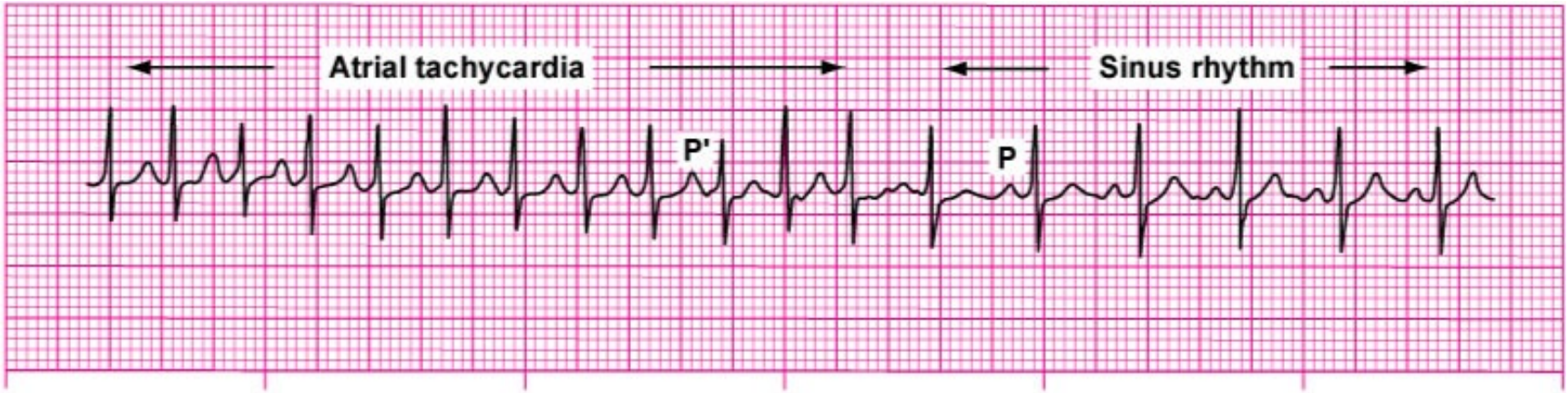
Atrial Tachycardia

- One of the supraventricular tachycardias
- Occurs when one or more cells or areas of tissue within atria become irritable and ectopic focus or foci are responsible for rhythm
- Paroxysmal A Tach appears and ends suddenly



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Multiformed Atrial Rhythm

- Previously known as wandering atrial pacemaker
- Occurs when dominant pacemaker shifts from SA node, atria, and AV junction
- Requires at least 3 variations of P wave size, shape, or direction

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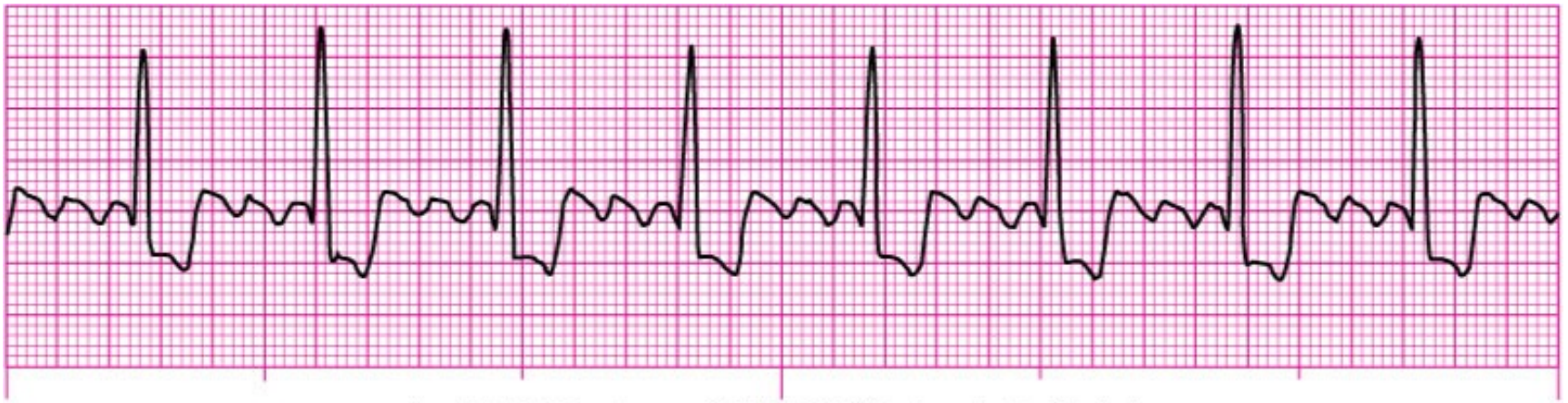
Atrial Flutter

- 2 or more sawtooth-shaped flutter waves occur before QRS
- Not considered life threatening



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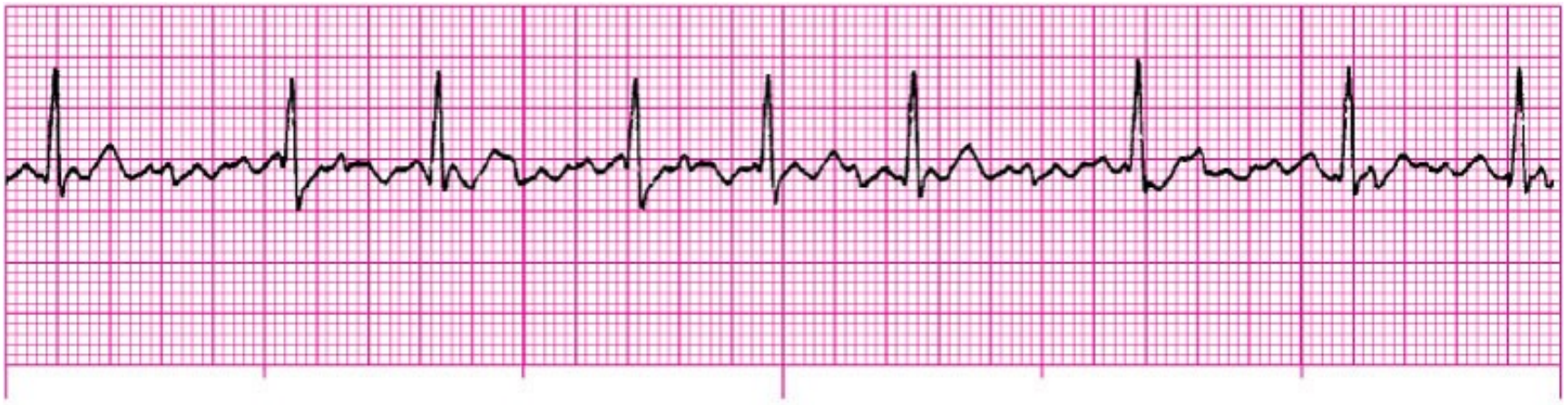
Atrial Fibrillation

- No coordinated activation process in atria
- Atria are quivering
- Clear relationship between A fib and OSA
  - 49% of A fib patients have OSA

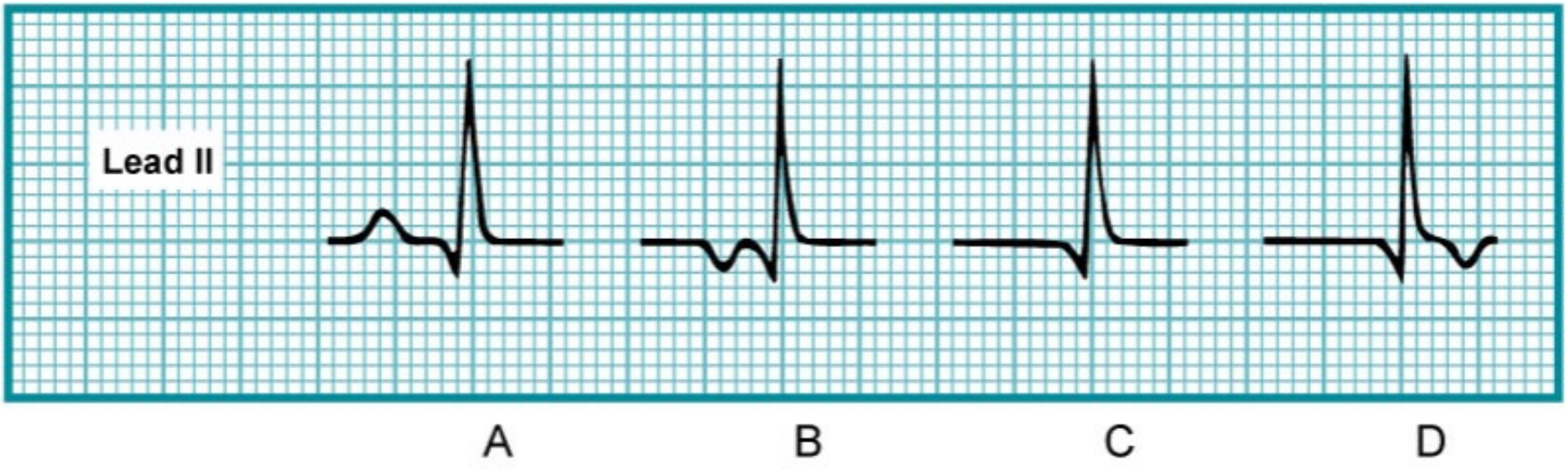


Junctional Rhythms

- Originate in AV node
- Occur because of failure or disruption in conduction that normally arises from SA node
- Impulse initiated in AV node, going backward through atria and forward to ventricles
  - Retrograde impulses cause inverted P wave
    - Inverted P wave can appear before or after QRS or gets buried in QRS
- Not considered life threatening

Junctional Rhythms

- A = Normal P wave
- B = Inverted P wave
- C = P wave hidden by QRS
- D = P wave after QRS



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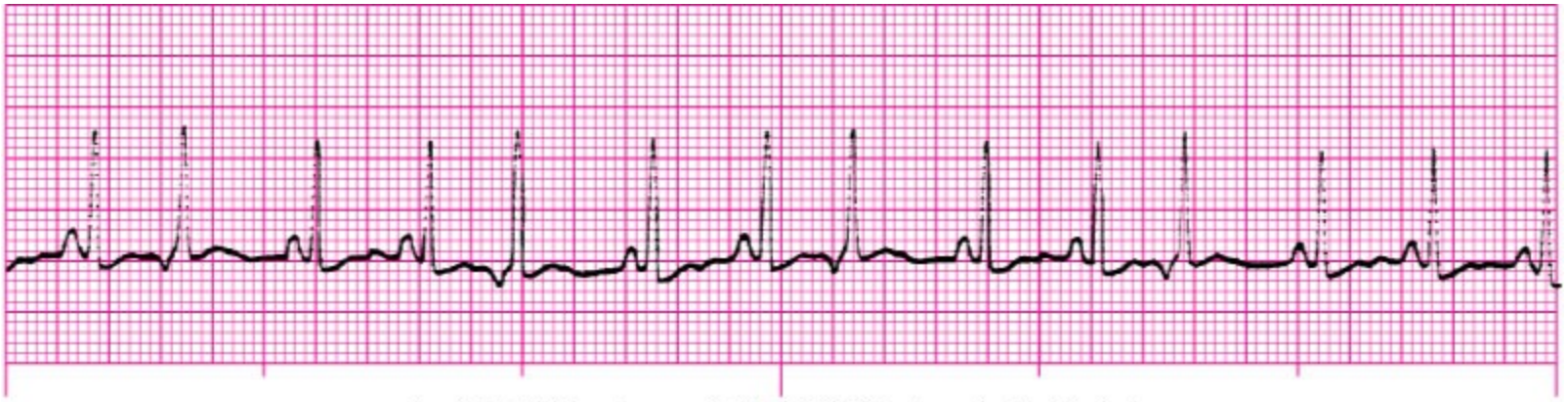
PJC



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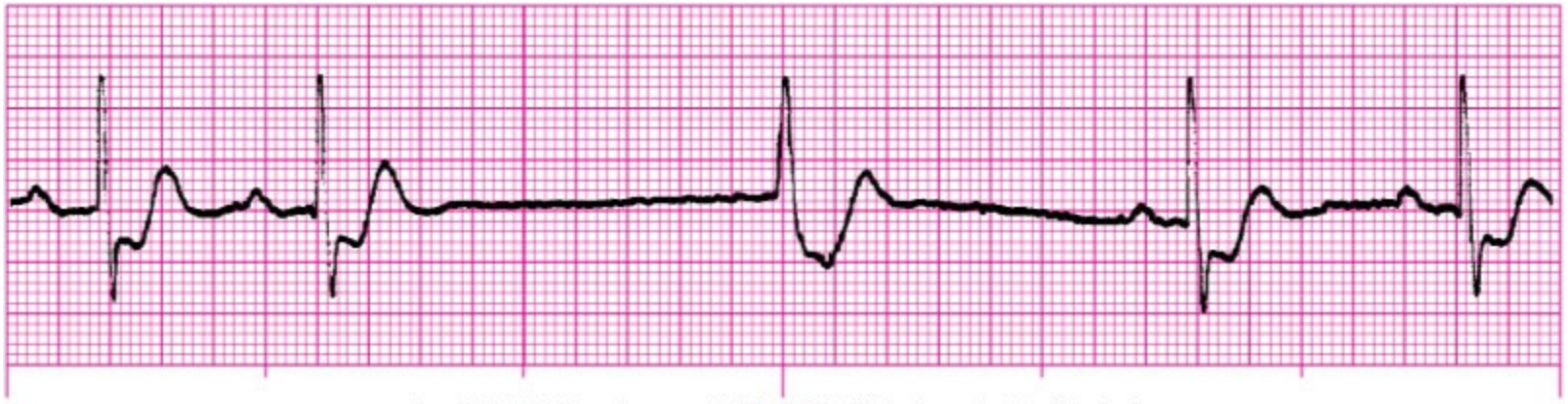
- Occurs when irritable focus within AV junction fires before next normal impulse is conducted at SA node
- Causes inverted P wave before, during, or after QRS
- If early junctional complex, PJC
- If late, it is junctional escape beat, which represents initiation of cardiac cycle by AV junction caused by failure of SA node

Junctional Escape Beats



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- Generated when an abnormally long amount of time has elapsed since previous beat
- Cardiac impulse not originated above the AV junction, so it takes over as the cardiac pacemaker
- Always late compared with underlying rhythm
- In picture, sinus arrest lasts about 1.4 seconds before impulse is generated by AV junction

Junctional Escape Rhythm

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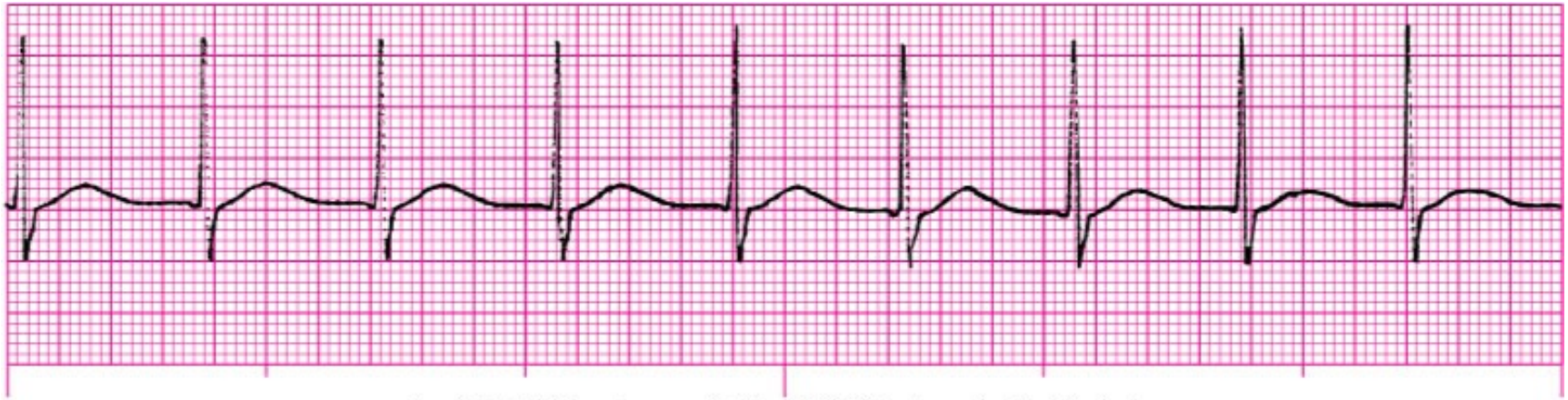
- Several junctional escape beats in a row
- AV junction takes over as heart's pacemaker at intrinsic rate of 40-60 bpm
- Tech should contact attending physician and document
- Pause is normal before all escape beats
- If rate is less than 40 bpm, it is junctional bradycardia.

Accelerated Junctional Rhythm

- Rate is faster than inherent rate for this area
  - Can be attributed to increased sympathetic nervous system activation, hypoxia, or an active MI
- Likely due to enhanced automaticity of bundle of His
- Needs to be brought to attention of attending physician



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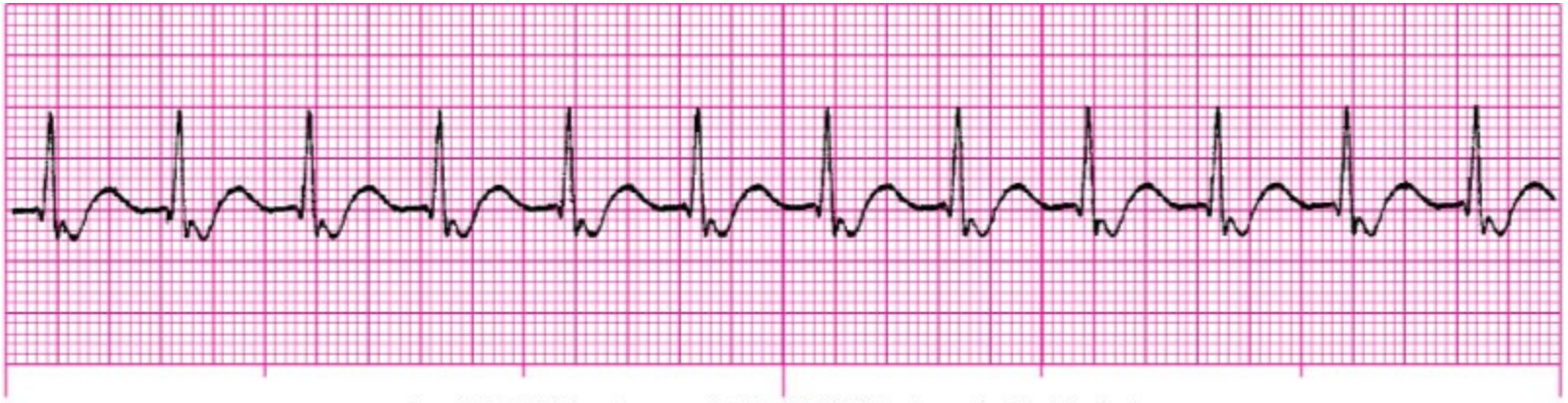
Junctional Tachycardia



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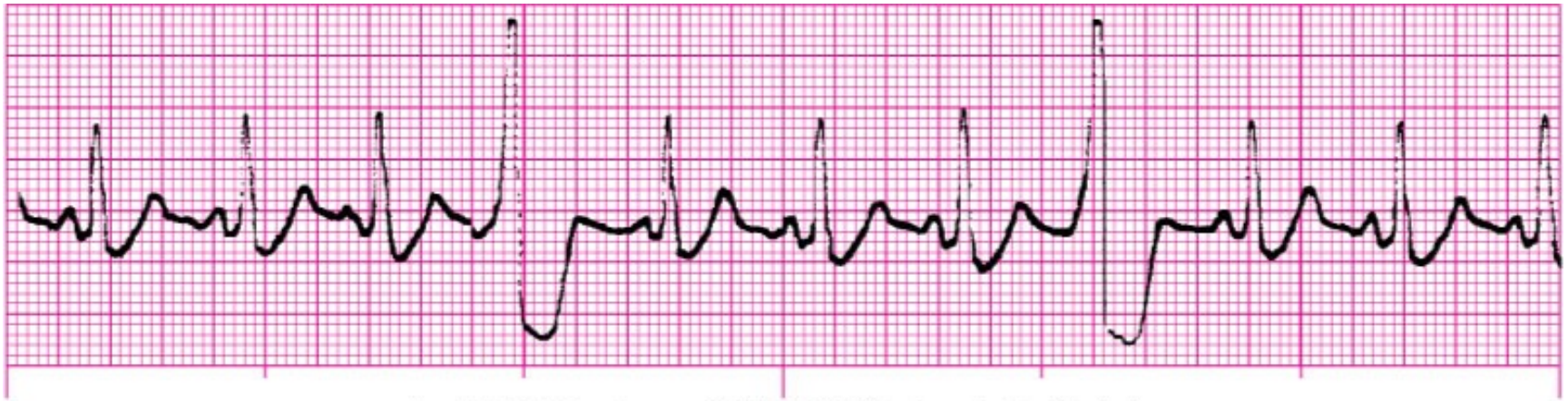
- Rate in picture is 120 bpm
- Ectopic rhythm that originates in pacemaker cells found in bundle of His
- Exists when 3 or more PJCs occur in a row at rate > 100 bpm
- Paroxysmal junctional tachycardia starts suddenly with PJC and ends abruptly
- Nonparoxysmal junctional tachycardia starts with accelerated junctional rhythm from which rate gradually increases

Ventricular Rhythms

- Originates below bifurcation of the bundle of His
- Frequently lack a P wave
- Have wide and bizarre QRS
- Ventricles are heart's least efficient pacemaker
  - Intrinsic rate of 20-40 bpm
- Ventricular dysrhythmias associated with SDB

Unifocal PVC

- Represents premature depolarization caused by irritable focus arising from ventricles
- The vast majority of these are benign
- 2 or more PVCs with same appearance arising from single irritable focus in ventricle



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Contact Physician If You See:

- Multifocal PVCs (arise from more than 1 area in ventricles and look different from each other)

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- R-on-T PVCs (PVC falls on T wave)

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Other Types Of PVCs

- Couplet (two PVCs in a row)

- Bigeminy (every other beat is a PVC)

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Other Types Of PVCs

- Trigeminy (every third beat is a PVC)

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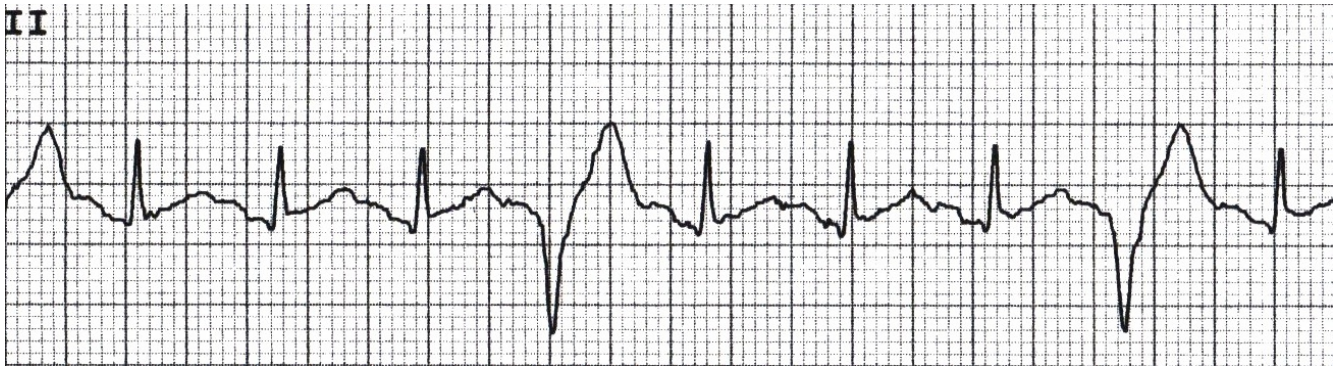
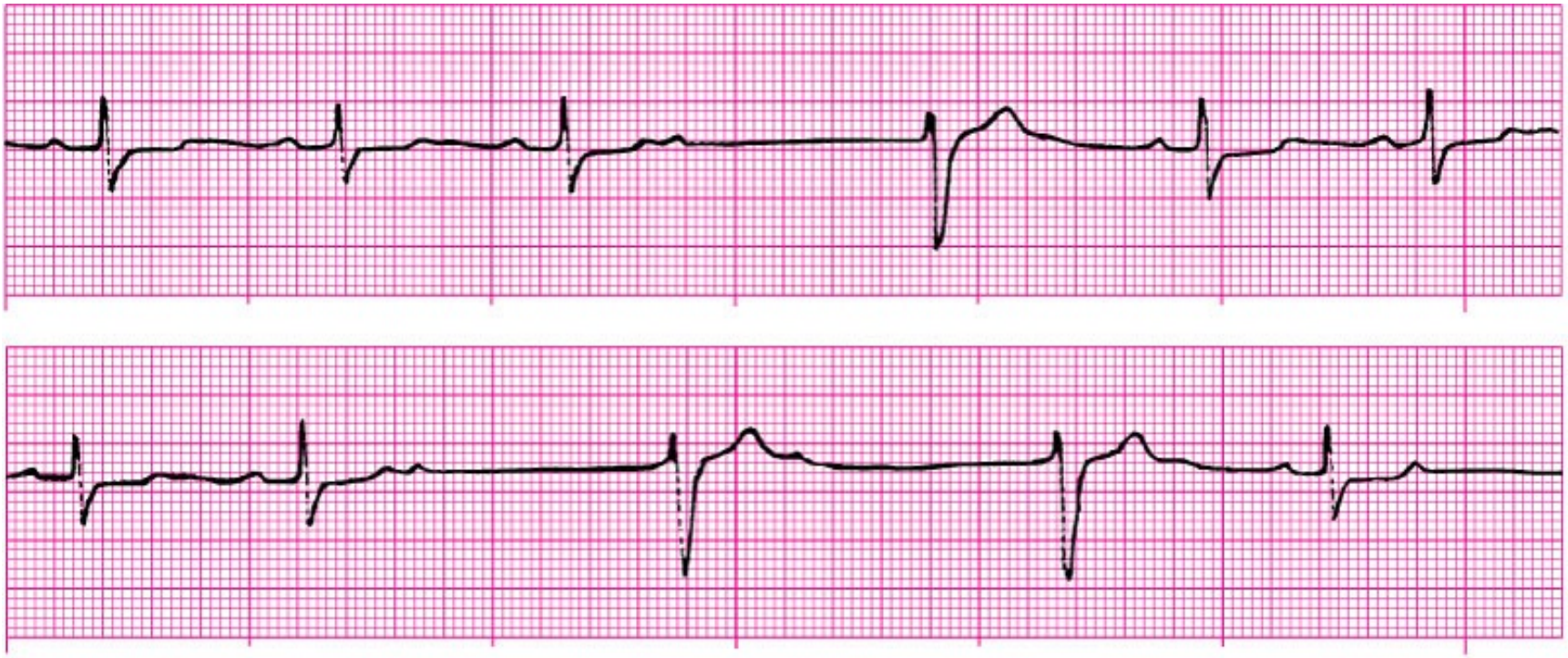
- Quadrigeminy (every fourth beat is a PVC)



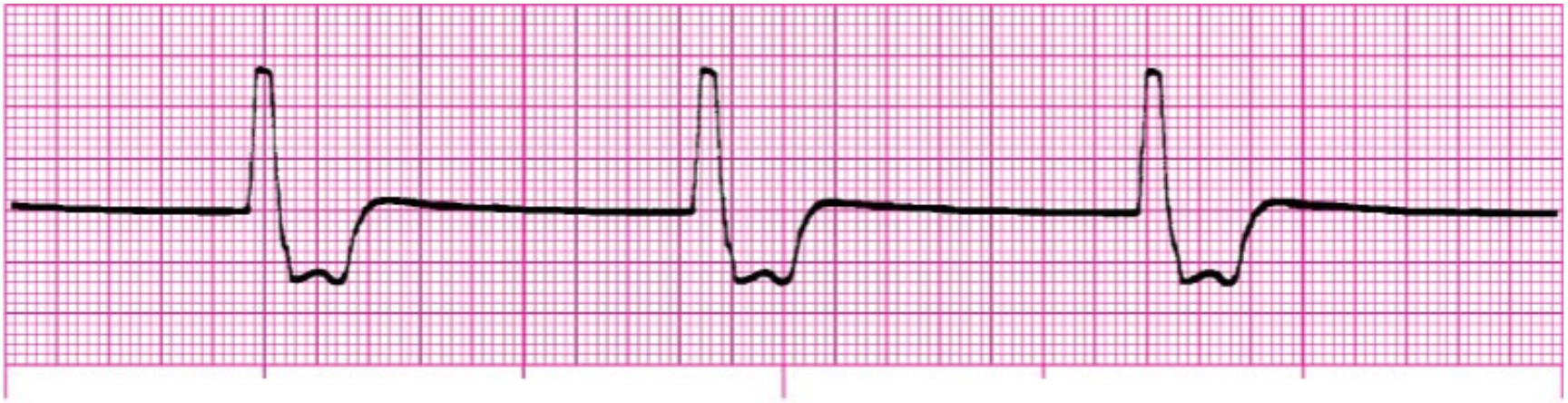
Ventricular Escape Beats

- Occurs following pause while waiting for supraventricular pacer to fire
- Escape beat is late compared to underlying rhythm
- Prevents more extreme slowing of heart and asystole
- Contact physician



Idioventricular Rhythm

- Rate = 20-40 bpm
- 3 or more ventricular escape beats in row
- Call EMS or attending physician immediately



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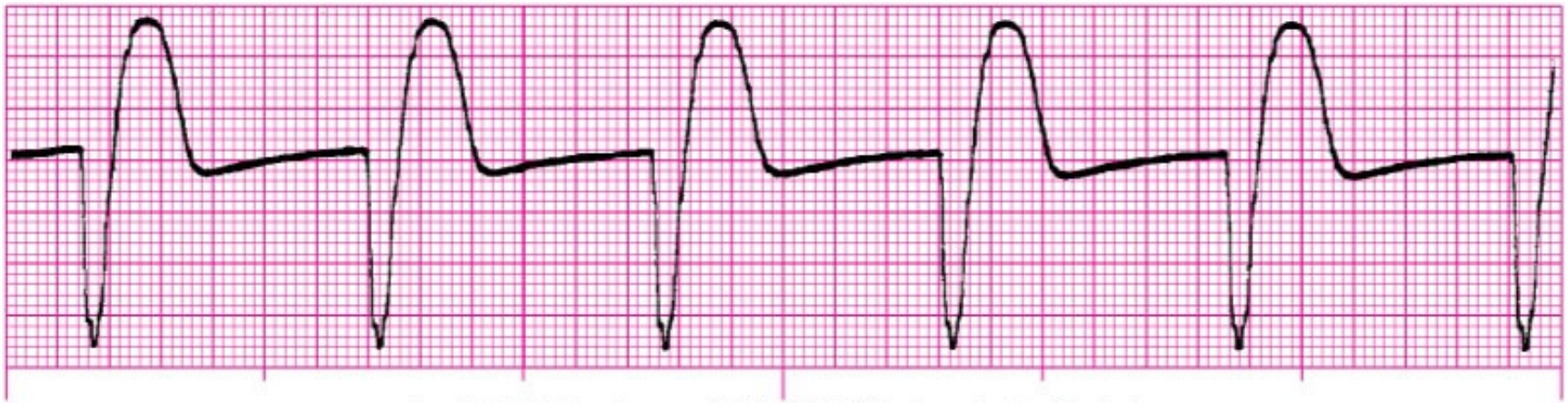
Accelerated Idioventricular Rhythm

- 3 or more ventricular escape beats in row at rate of 41-100 bpm
- Call EMS and attending physician immediately



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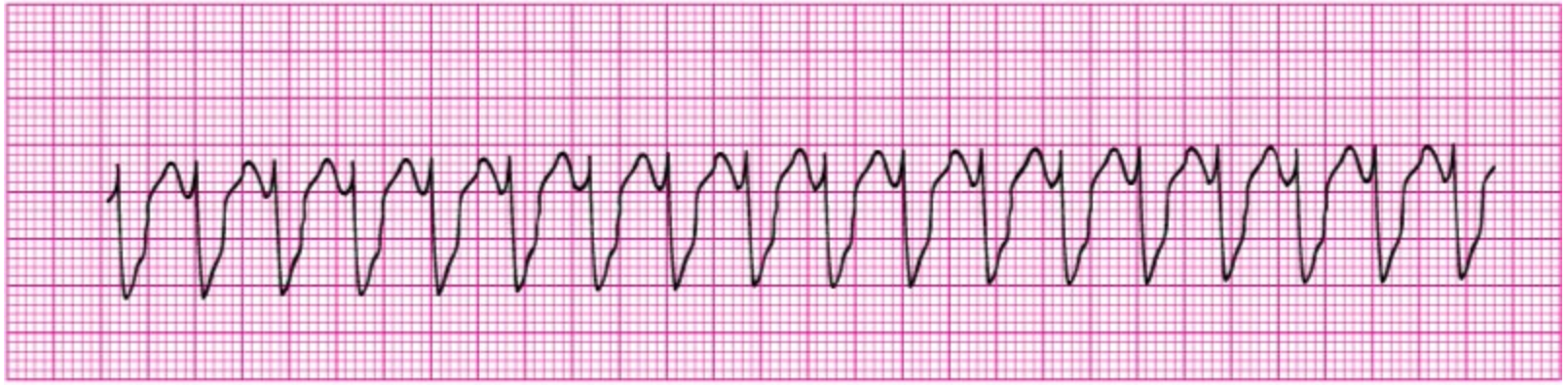
Ventricular Tachycardia

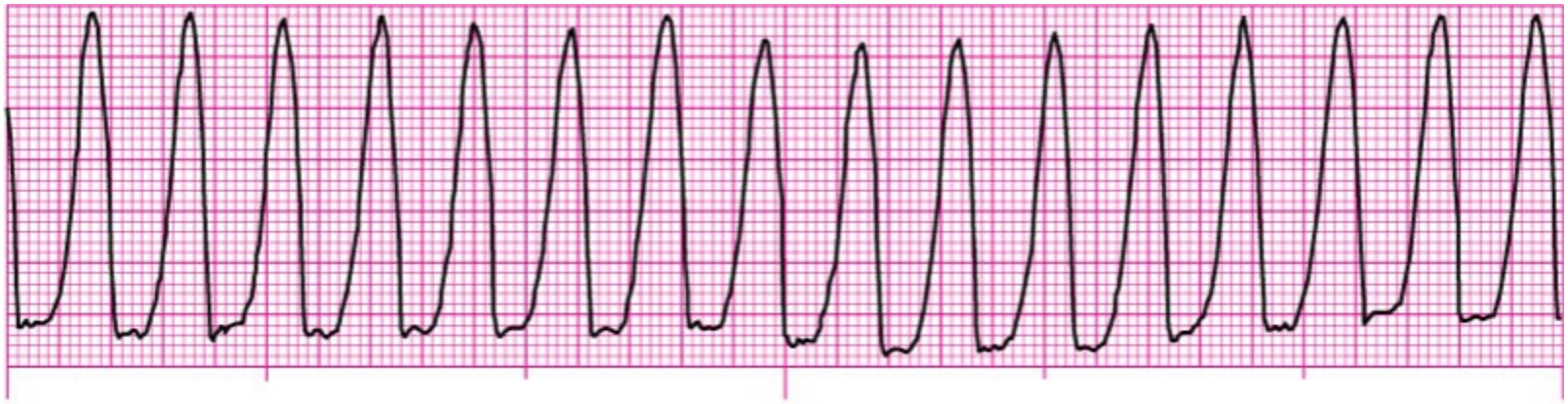
- 3 or more PVCS in row = Run of V Tach
  - Frequently seen in OSA patients
- Sustained V Tach = V Tach lasting > 30 seconds
  - Call EMS and begin CPR as warranted
  - Notify attending physician ASAP



From Wilkins RL, et al: Clinical assessment in respiratory care, ed 6, St Louis, 2010, Mosby.

Ventricular Tachycardia

- Monomorphic V Tach (rate = 100-250 bpm, regular)



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- Polymorphic V Tach (rate = 150-300 bpm, irregular)

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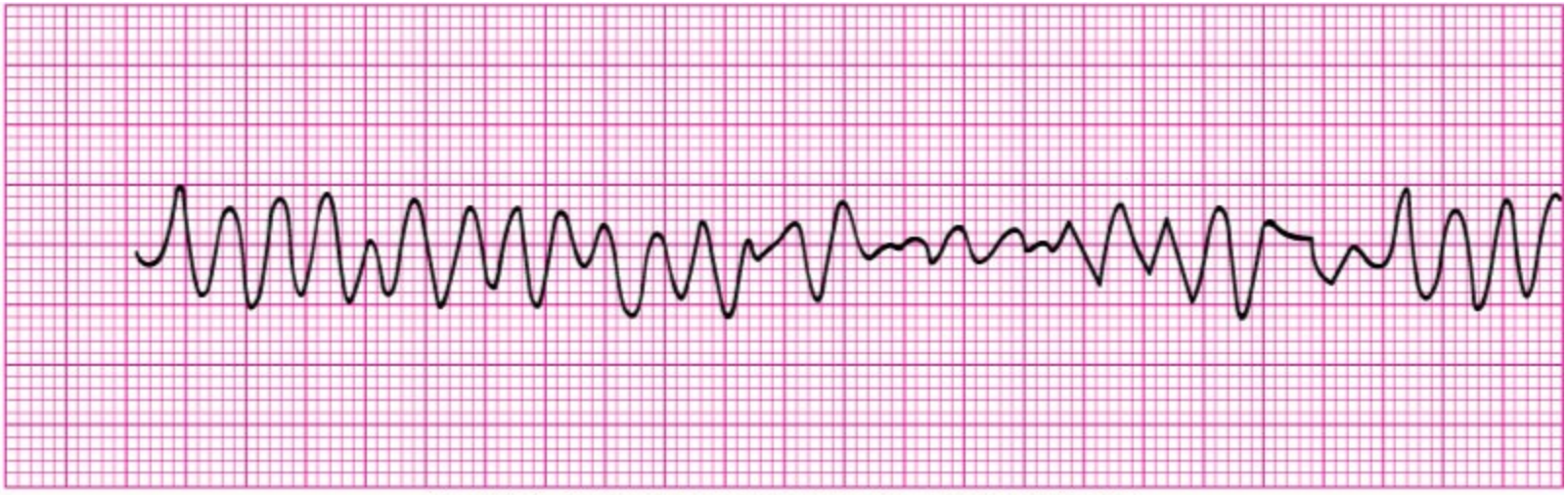
Ventricular Fibrillation

- Very little if any blood is being pumped
- Life threatening
  - Call EMS and begin CPR
    - Use AED if available
  - Always assess patient before beginning emergency procedures and don't just rely on monitor



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Ventricular Fibrillation

- Fine V Fib (can look like disconnected electrode)

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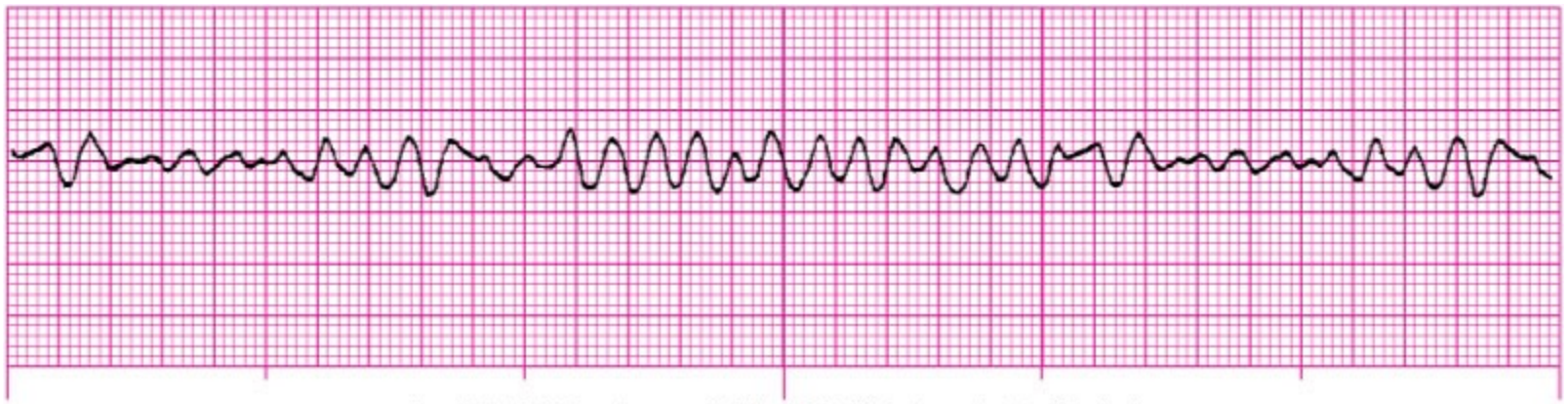
- Coarse V Fib (typical V Fib)



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Asystole (Cardiac Standstill)

- Absence of electrical and mechanical activity of heart
- Physically assess patient
  - If confirmed, contact EMS, start CPR, and notify attending physician

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Ventricular Standstill

- Pulseless electrical activity
- Occurs when SA node continues to fire impulse but heart muscle doesn't capture it

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First Degree AV Block

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- Usually occurs above bundle of His
- Identified by prolonged PR interval
  - Is constant, predictable, and benign
  - Represents slowing of atrial impulse through intranodal pathways
- Document and monitor

Second Degree AV Block Type I

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- Also known as Wenckebach, Mobitz type I
- Identified by a progressive lengthening of PR interval until dropped QRS
- Dysfunction of AV node
- Document and monitor

Second Degree AV Block Type II

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- Also known as Mobitz Type II
- Originates below bundle of His
- Identified by dropped QRS without warning
- Must be closely monitored as it can quickly progress to complete heart block
- Document and report to attending physician

Third Degree AV Block

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- Also known as complete heart block
- Most severe of heart blocks
- No relationship between atria and ventricles
  - P waves have no relationship with QRS
  - Signal from atria doesn't travel to ventricles due to completely blocked pathway
  - Atrial rate is that of atrial pacemaker
  - Ventricular rate is much slower and based on origin of escape rhythm
- Medical emergency so contact EMS if QRS is wide