Atrial Arrhythmias

Objectives

- By the end of the lesson the learner should be able to:
- Review mechanism of dysrhythmias
- Describe ECG characteristics, causes, signs and symptoms and management of atrial rhythms

Arrhythmias/ dysrhythmias

- Any disturbance in the normal sequence of impulse generation & conduction in the heart
- Arrhythmias may occur with or without underlying heart disease e.g. palpitations
- The 3 main mechanisms of an arrhythmia are:
- Altered/Enhanced automaticity
- Triggered activity
- Re-entry

Arrhythmias/ dysrhythmias

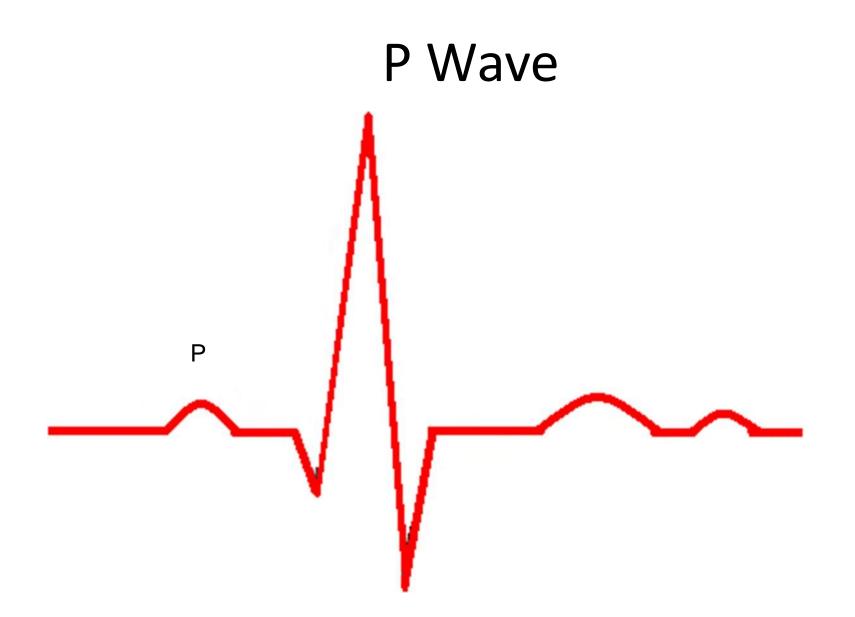
- The 3 main mechanisms of an arrhythmia are:
- Altered/Enhanced automaticity- Other parts of the heart may act as pacemaker cells and "fire" just before the normal pacemaker (SAN) sends out an impulse
- **Triggered activity-** An abnormal electrical impulse during repolarization
- Re-entry- Spread of an impulse through tissue already stimulated by that same impulse through an accessory pathway

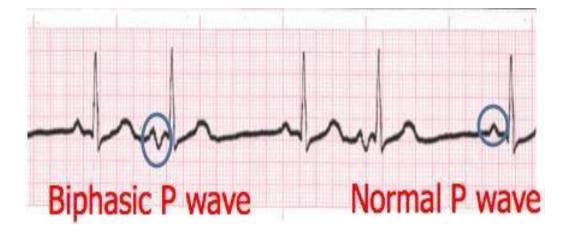
Introduction

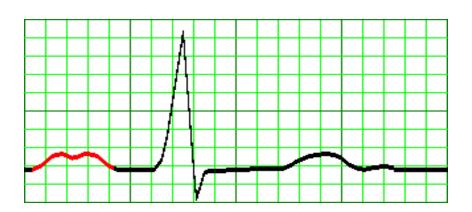
 Atrial arrhythmias reflect abnormal electrical impulses formation and conduction in the atria and the P wave differs in configuration to the P wave caused by an impulse from the SAN

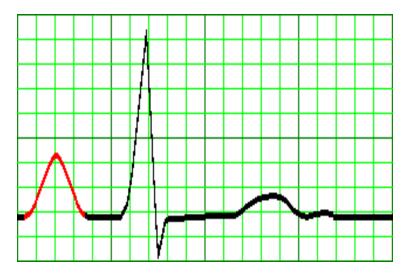
Remember Characteristics of a Normal P Wave

- Normal Characteristics of the P wave:
 - Represents atrial depolarization
 - Smooth & round
 - No more than 2.5mm in height (2 small squares)
 - No more than 0.11 seconds in duration (2.5 small squares)
 - Abnormally shaped P wave can be notched, pointed, flattened or biphasic

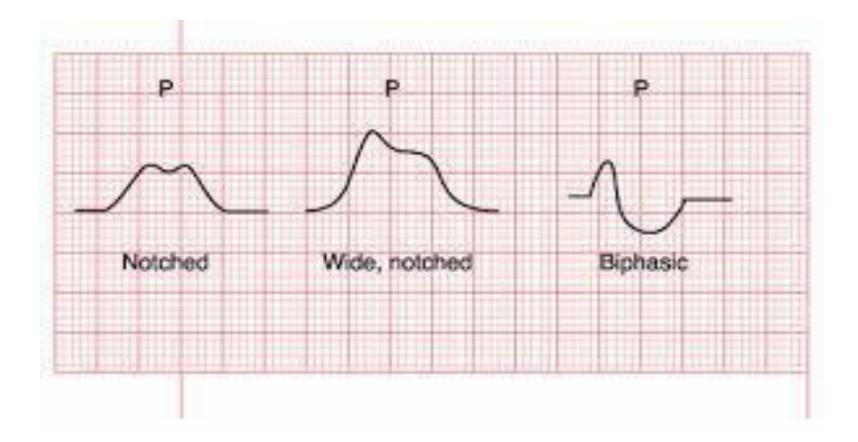








Abnormal P wave morphology



Introduction

- In atrial arrhythmias, cells in the atria may fire abnormally, which interferes with electrical signals coming from the Sino atrial (SA) node.
- A series of early beats in the atria speeds up the heart rate.
- This rapid heartbeat does not allow enough time for the heart to fill before it contracts so blood flow to the rest of the body is compromised.

Types of atrial arrhythmias

- Premature atrial complexes (PACs)
- Wandering atrial pacemaker
- Multifocal atrial tachycardia
- Atrial tachycardia
- Atrial flutter
- Atrial fibrillation
- Wolf Parkinson-white syndrome (WPW)
- Atrio-ventricular nodal reentrant tachycardia (AVNRT)
- Atrio-Ventricular Reentrant tachycardia (AVRT)

Supraventricular tachycardia

- Arrhythmias that start above the AV node are called supraventricular (above the ventricles) arrhythmias.
- "Supra" means above and "ventricular" refers to the lower chambers of the heart (ventricles).
- Supraventricular tachycardias (SVT) can cause the heart to beat very rapidly and inefficiently causing the body to receive an inadequate blood supply.

Supraventricular tachycardia

- During an episode of Supraventricular Tachycardia (SVT), another part of the heart overrides the SAN with faster impulses.
- The source or 'trigger' of the impulse in an SVT is somewhere above the ventricles, but the impulse then spreads to the ventricles.

• The heart then contracts faster than normal.

Supra-ventricular tachycardia

- Forms of SVT include:
- Paroxysmal Supraventricular Tachycardia (PSVT)
- Atrial Tachycardia
- Reentrant Tachycardias (AVNRT and AVRT)
 - Atrio-ventricular nodal re-entrant tachycardia
 - Atrio-ventricular reentry tachycardia
- Atrial Fibrillation
- Atrial Flutter

N.B The first 4 are the most common SVT forms

Treatment of supraventricular tachycardia

- Vagal maneuvers
 - Carotid massage
 - Valsava maneuver
 - Holding breath
- Adenosine- administered within 10 to 25 seconds via a peripheral vein followed by 20 mls of a flash because it has a short half-life (less than 10 seconds)
- Verapamil

Types of atrial arrhythmias

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- Atrio-ventricular nodal reentrant tachycardia (AVNRT)
- Atrio-Ventricular Reentrant tachycardia (AVRT)

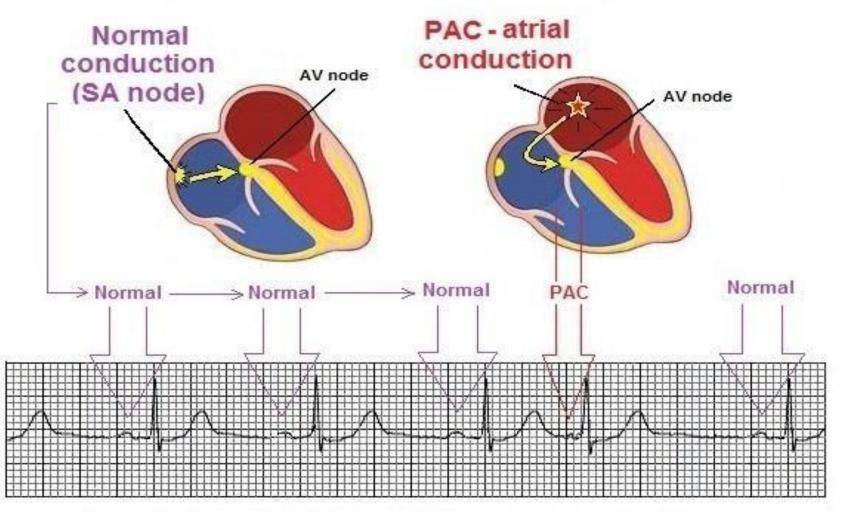
PREMATURE ATRIAL CONTRACTION (PACS)

Atrial Arrhythmias

Premature atrial complexes (PACs)

- Occurs when an impulse is generated by an irritable area of tissue in the atria (altered automaticity)
- Occurs before next SAN impulse is fired
- May occur in a normal healthy heart
- Described by patient as being "aware" of heart beat or "skipping" a beat (palpitations)

Premature Atrial Contraction (PAC)

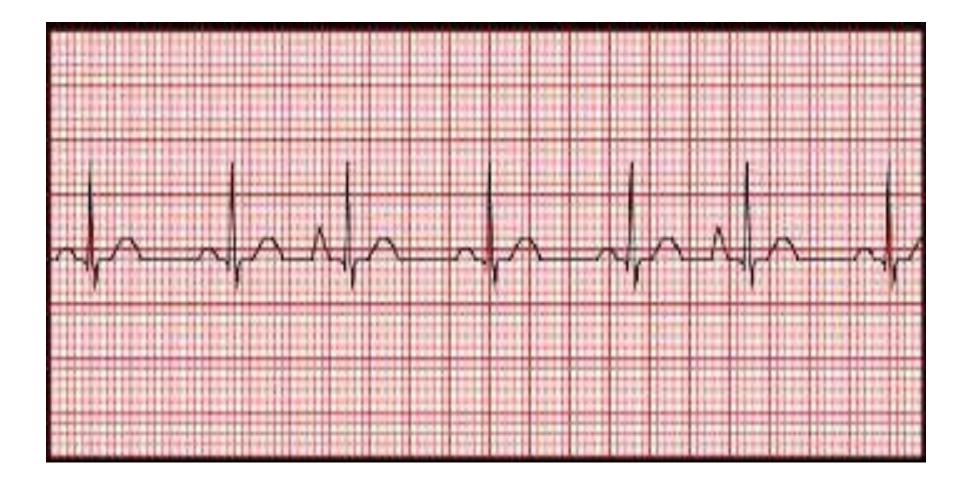


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Characteristics of PACs

- Rate: depends on underlying rhythm
- Rhythm: regular with premature beats
- P wave: abnormally shaped from a P wave from the SAN and may not be followed by a QRS complex
- At times the P wave maybe buried within the T wave
- P-R interval: may be prolonged, shorter
- R-R Interval: unequal since PACs present
- QRS Complex: usually narrow or maybe absent

Premature Atrial Complex (PACs)



Premature Atrial Contraction (PAC)



Premature Atrial Contraction (PAC)



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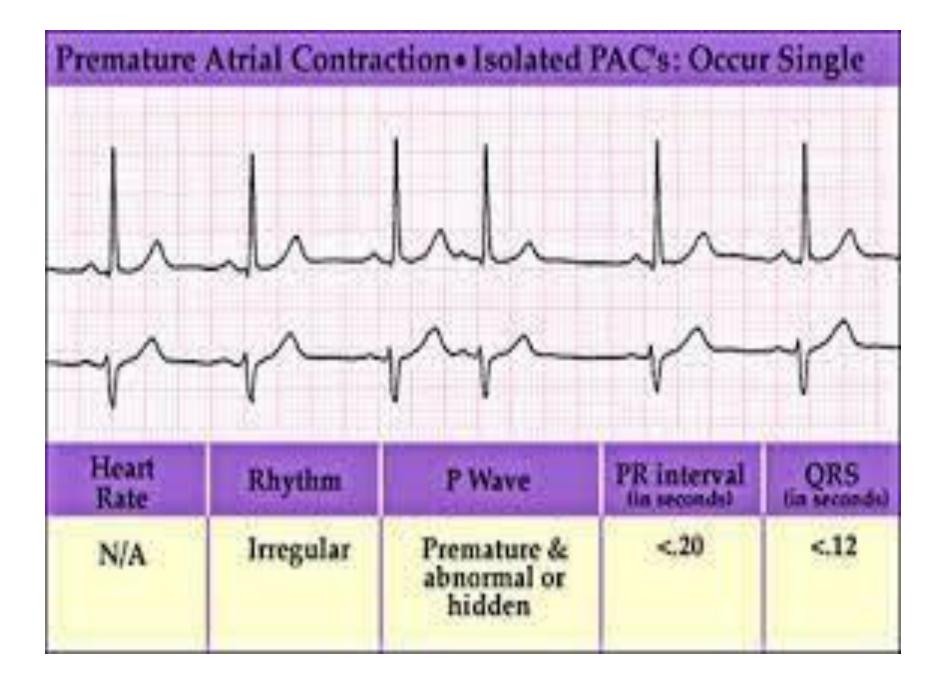
25mm/sec 10mm/mV

Causes of PACs

- Can be triggered by:
- Idiopathic
- Anxiety and stress
- Fever
- Increased sympathetic input e.g. caffeine, drug interactions
- Cardiac ischemia
- Heart disease.

Treatment of PACs

- Usually not clinically significant
- Treat underlying cause
- Decrease caffeine consumption
- Decrease stress
- Medications:
 - anxiolytics
 - beta blockers
 - calcium channel blockers



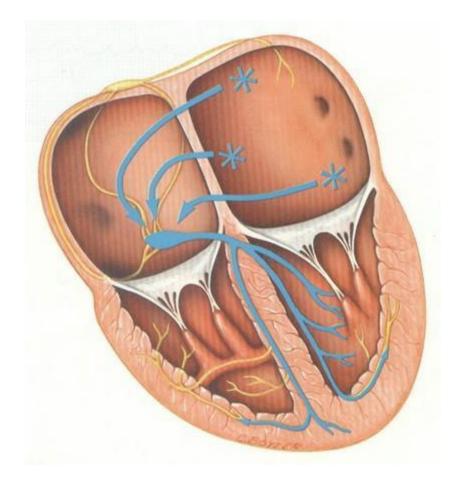
Atrial Arrhythmias

WANDERING ATRIAL PACEMAKER

Wandering atrial pacemaker

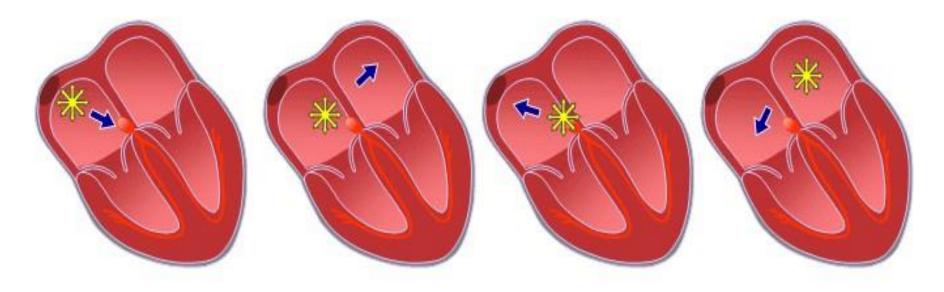
- Occurs when **multiple areas (ectopic foci)** within the atria generate consecutive action potentials that are all conducted to the ventricles.
- Thus, each QRS complex will be preceded by a P wave, however each P wave will have a different morphology since it is originating from a different area (SAN, atrial cells)

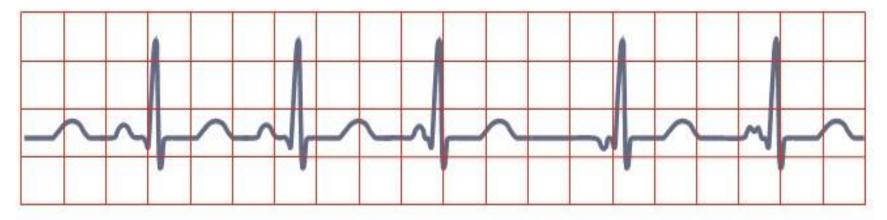
Wandering atrial pacemaker



WANDERING PACEMAKER

Impulses originate from varying points in atria

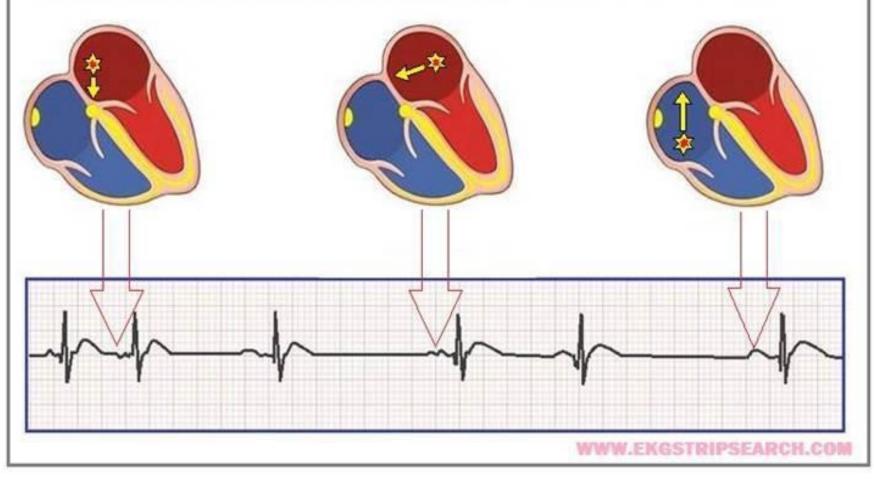




Variation in P-wave contour, P-R and P-P interval and therefore in R-R intervals

What's up with Wondering Atrial Pacemaker?

- 1. The heart rate is below 100 bpm
- 2. It's a Multifocal Atrial rhythm (originating from the atria)
- 3. It will have at least 3 different P-wave morphologies
- 4. The pacemaker site shifts between the SA node, Atria and AV node



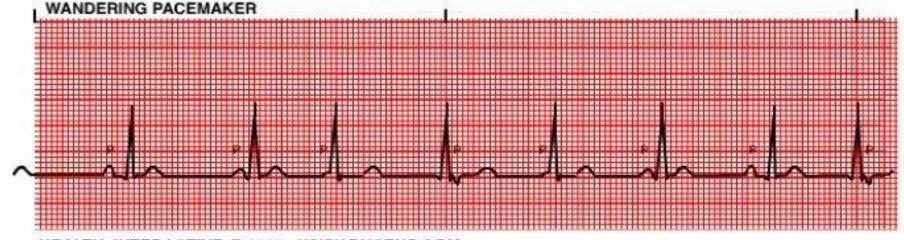
Wandering atrial pacemaker

- It is characterized by three or more P wave morphologies and a rate less than 100 beats per minute.
- It may reflect electrical abnormalities in one or both atria that increase the likelihood of multifocal atrial tachycardia or other atrial arrhythmias.
- Wandering atrial pacemaker is also called multiformed atrial rhythm

Characteristics of wandering atrial pacemaker

- HR: usually 60-100 bpm
- Rhythm: irregularly irregular
- P Waves: variable including normal appearing P waves, there are ≥ 3 distinct P-wave morphologies
- P-R Interval: unequal, varies
- R-R Interval: unequal, varies
- QRS Complex: Normal (0.06-0.10 sec)
- T wave normal. If heart rate exceeds 100 bpm, then rhythm may be multifocal atrial tachycardia (MAT)

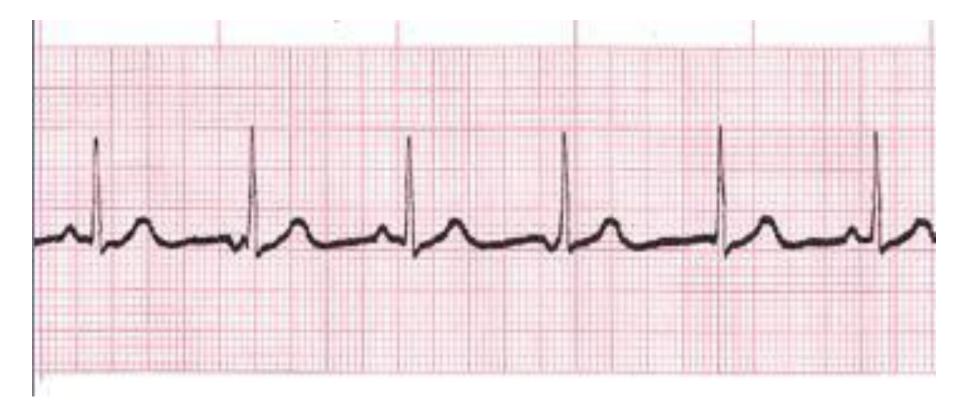
Wandering Atrial Pacemaker (WAP)



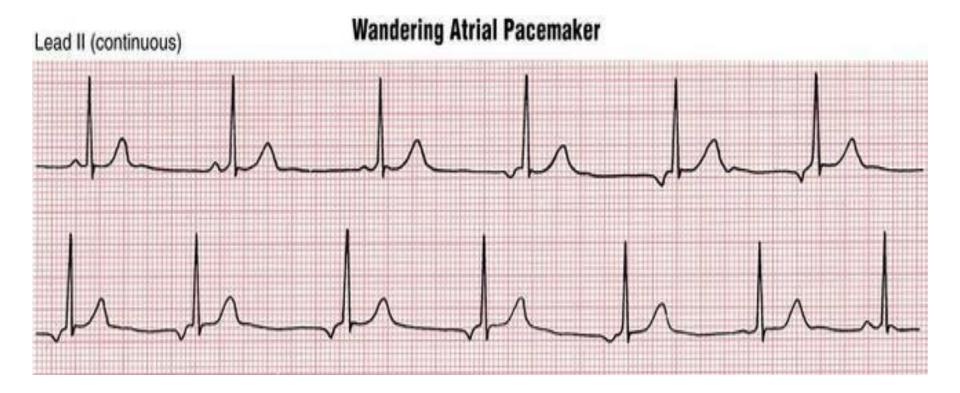
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Rhythm: Slightly Irregular Rate: Usually 60 – 100 bpm; sometimes slower Pwaves: Morphology of each P wave differs PRI: 0.12 – 0.20 sec; inconsistent QRS: Narrow (< 0.12 sec); sometimes wide

Wandering atrial pacemaker



Wandering atrial pacemaker



Wandering atrial pacemaker

- Causes:
- May occur normally in healthy individuals- due to varying vagal tone.
- With increased vagal tone the SA Node slows (bradycardia), allowing a pacemaker in the atria or AV Nodal area, which may briefly become slightly faster.
- Digitalis toxicity

Treatment of wandering atrial pacemaker

- Patients with wandering atrial pacemaker are usually not symptomatic.
- Patients have a normal heart rate
- Treat the underlying cause

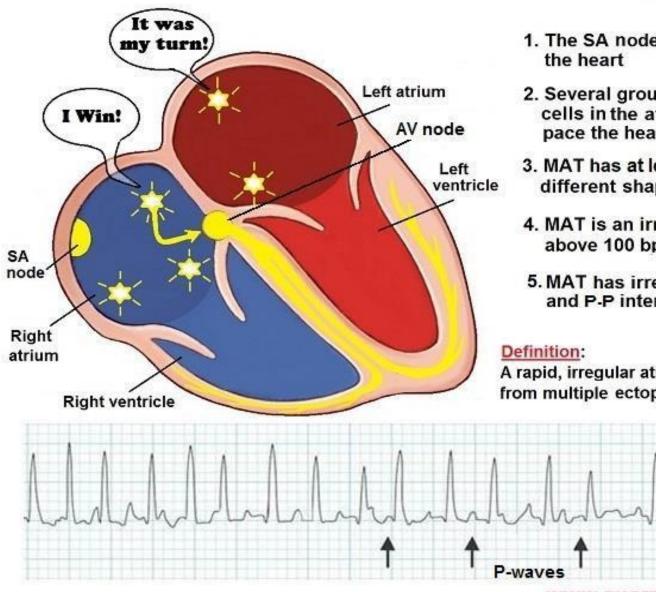
Atrial Arrhythmias

MULTIFOCAL ATRIAL TACHYCARDIA

Multifocal Atrial tachycardia

- Involves multiple ectopic sites stimulating the atria (like in wandering atrial pacemaker)
- Also referred to as chaotic atrial tachycardia
- The P wave vary and atrial and ventricular contractions are irregular
- Its similar to WAP (wandering atrial pacemaker) the only difference is that the rate is > 100b/min
- An uncommon ECG rhythm
- Usually seen in persons with COPD or severe systemic disease (e.g. sepsis, shock)

What's up with Multifocal Atrial Tachycardia ?



- 1. The SA node is not pacing
- 2. Several groups of excitable cells in the atria compete to pace the heart
- 3. MAT has at least three or more different shaped P-waves.
- 4. MAT is an irregular rhythm above 100 bpm
- 5. MAT has irregular P-R, R-R and P.P intervals

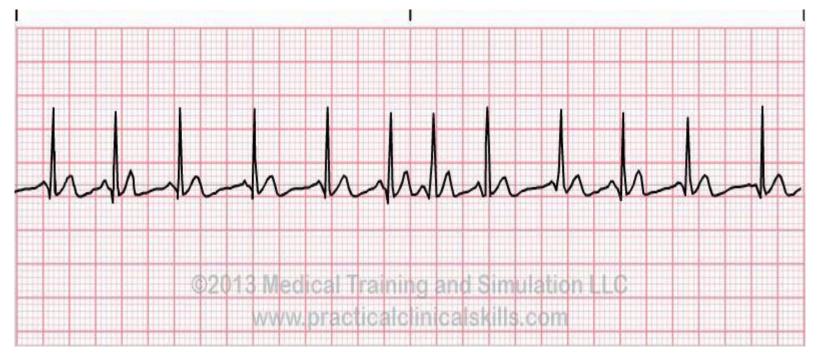
A rapid, irregular atrial rhythm arising from multiple ectopic foci within the atria.

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Characteristics of multifocal atrial tachycardia

- HR: usually >100 bpm
- Rhythm: irregularly irregular
- P Waves: variable including normal appearing P waves, there are ≥ 3 distinct P-wave morphologies
- P-R Interval: unequal, varies
- R-R Interval: unequal, varies
- QRS Complex: usually narrow

Multifocal Atrial tachycardia (MAT)



Multifocal Atrial Tachycardia (MAT)

At least 3 different P wave morphologies

- · Rate 100 to 180
- Irregular

Etiology

- Secondary to chronic lung disease (COPD)
- Hypoxia
- Pulmonary hypertension

Management

- Oxygen
- Treatment of underlying condition
- Rate control

Muhhhhhhhhhhhhhhhh

WHAT IS MULTIFOCAL ATRIAL TACHYCARDIA?

Multifocal Atrial Tachycardia is a pathological cardiac condition in which the heart beats much faster than it normally should.

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Multifocal Atrial tachycardia (MAT) • Treatments:

- Treat underlying cause
- Vagal maneuvers
- Ca⁺⁺ channel blockers (verapamil)

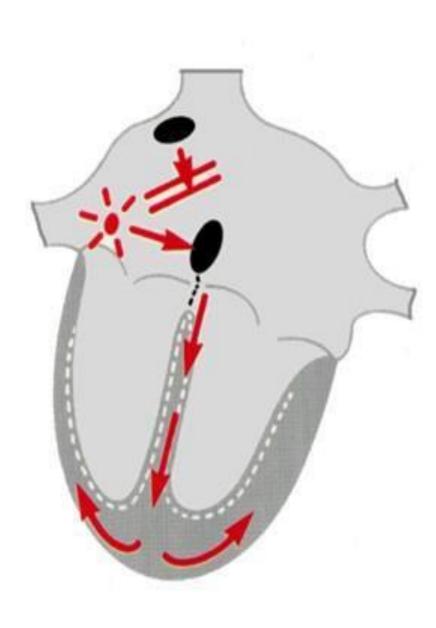
Multifocal Atrial tachycardia (MAT)

- Vagal maneuvers- methods used to stimulate baroreceptors in the aortic arch to cause bradycardia by slowing down the conduction through the AV node
 - Carotid massage
 - Coughing
 - Valsava maneuver
 - Abdominal pressure

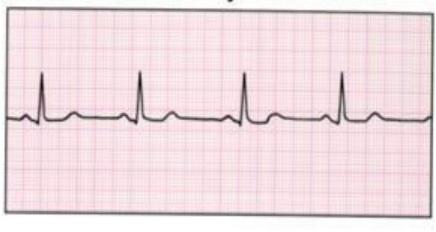
Atrial Arrhythmias

ATRIAL TACHYCARDIA

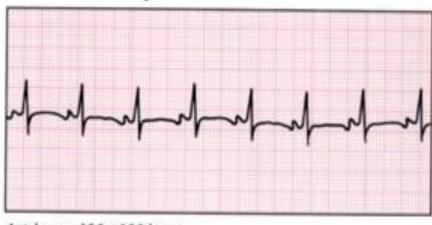
- Atrial tachycardia is caused by the consistent, rapid atrial activation from a **single atrial focus**. (altered automaticity)
- Heart rate is usually 150 to 200 beats/min therefore the rapid atria rate overrides the SAN and becomes the pacemaker
- The rapid rate prevents adequate ventricular filling
- At very rapid rates only every second P may be followed by QRS



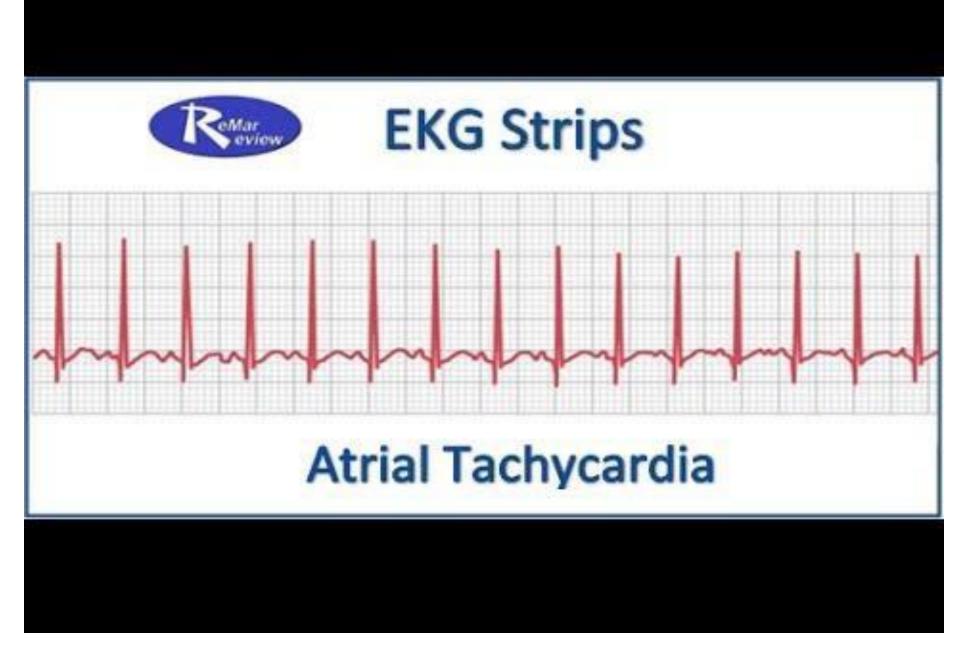
Normal Sinus Rhythm



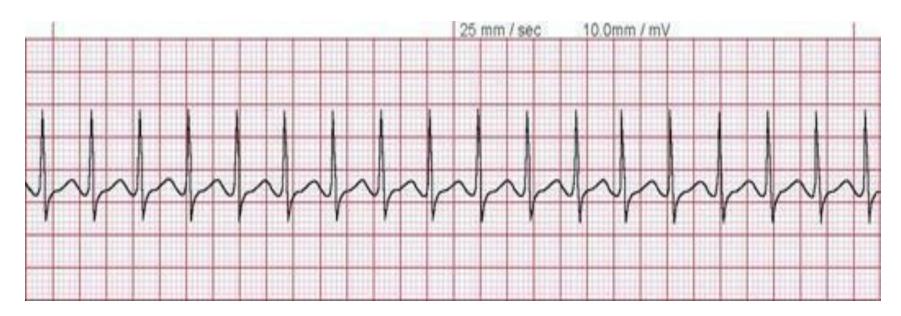
Atrial Tachycardia



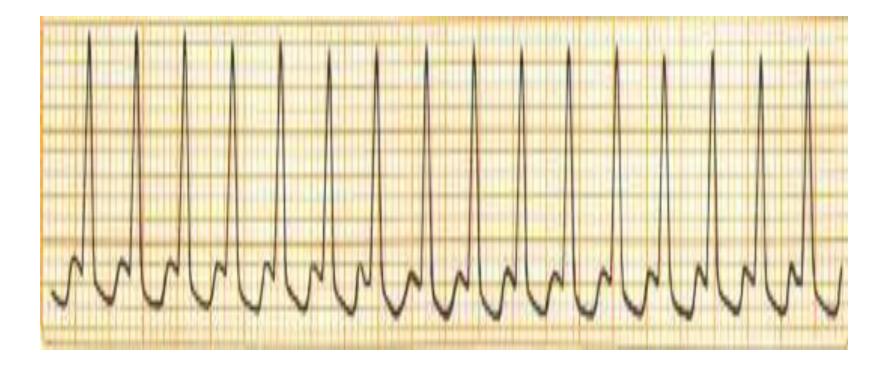
Atrial rate = 120-180 bpm



 It maybe difficult to identify an atrial tachycardia on an ECG hence if the QRS complex is narrow and it's a fast rhythm it's referred to as SVT- supraventricular tachycardia



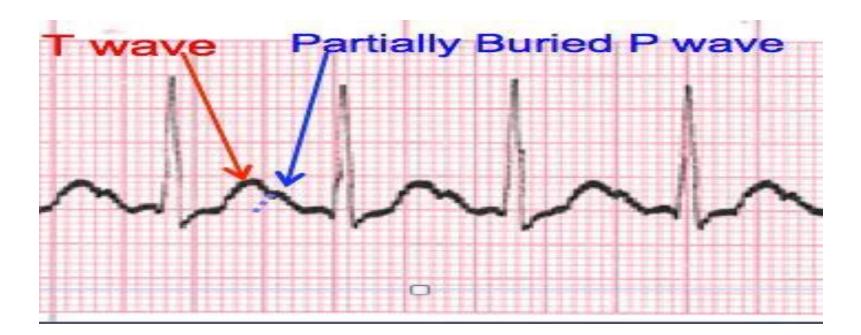
ATRIAL TACHYCARDIA



- Causes:
- Structural heart disorders e.g. pericarditis
- Drugs e.g. digoxin
- Alcohol

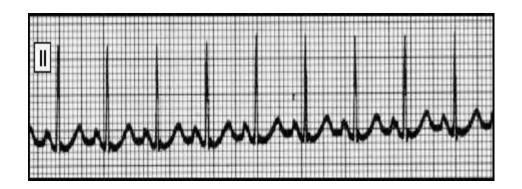
Characteristics of atrial

tachycardia
P waves, which differ in morphology from normal sinus P waves, precede QRS complexes but may be hidden within the preceding T wave

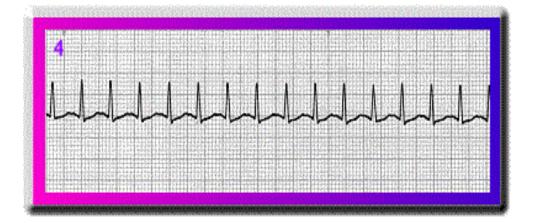


Characteristics of atrial

tachycardia
Atrial tachycardia may appear similar to a sinus tachycardia only that the P wave morphology is different



Sinus Tachycardia

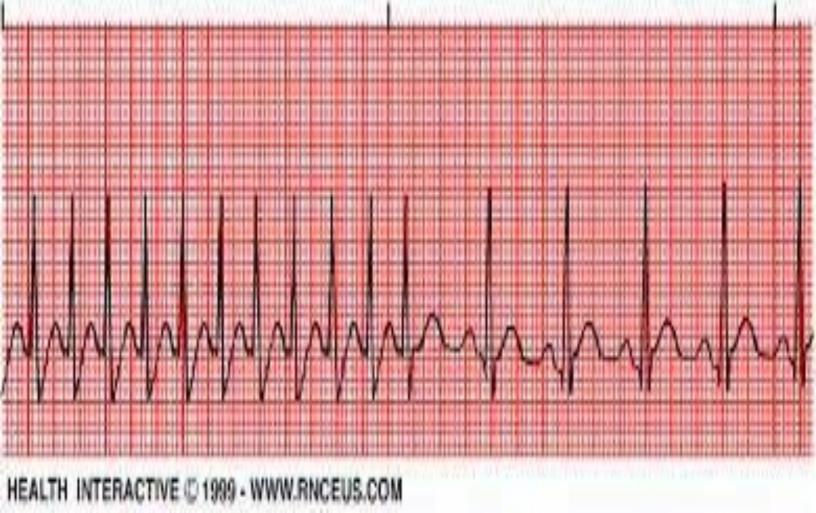


Atrial Tachycardia

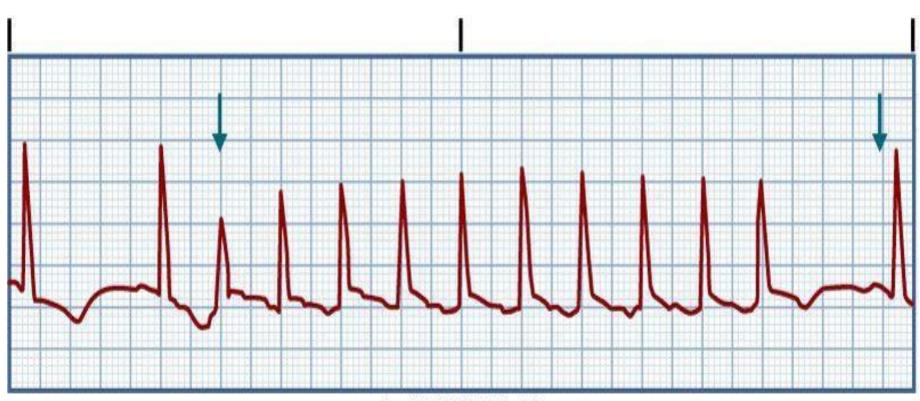
Characteristics of atrial

tachycardia
Atrial tachycardia may also begin suddenly and end suddenly and is called paroxysmal atrial tachycardia

PAROXYSMAL ATRIAL TACHYCARDIA



Paroxysmal SVT



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- Causes:
- Electrolyte imbalance
- Catecholamine release
- MI
- Infection
- Stimulants e.g. caffeine

- Signs and symptoms:
- Dyspnea, chest pain
- Palpitations, weak rapid pulse
- Syncope
- Dizziness
- Hypotension

• Treatment:

- Aim- control the HR
 - Vagal maneuvers
 - Oxygen
 - Adenosine 6mg iv rapid push- slows rate of SAN and slows conduction time through the AV node
 - Calcium channel blockers (verapamil, diltiazem)
 - Beta blockers (propranolol)
 - Synchronized cardioversion (if severe)

- Cardioversion is defined as a brief electric shock through the chest to the heart.
- Usually performed under sedation
- Synchronized cardioversion is timed to avoid the vulnerable period of the ECG wave
- The shock is delivered at the R wave

Atrial Arrhythmias

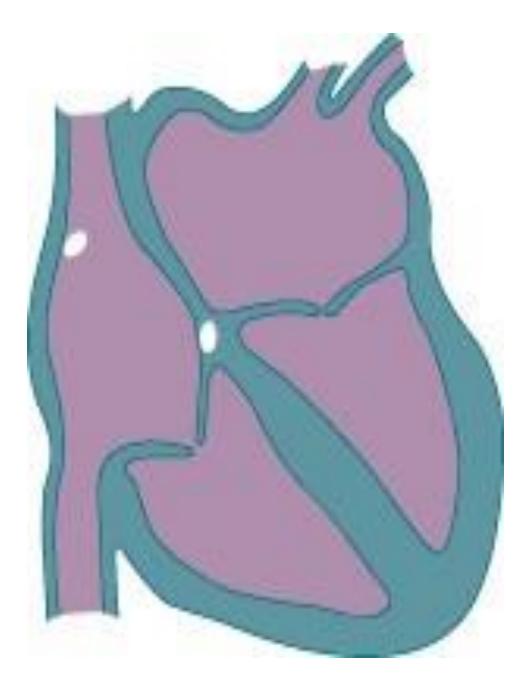
ATRIAL FLUTTER

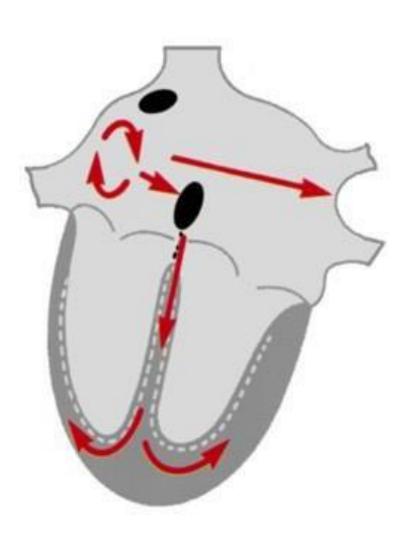
Atrial Flutter

- Electrical signals come from the atria at a fast but even rate, causing the ventricles to contract faster and increase the heart rate
- The atria are stimulated so quickly that they can not contract or squeeze; compromising cardiac output
- Atrial rate ranges from 250-350bpm

Atrial Flutter

- Atrial flutter is due to a re-entrant circuit in the atria commonly in the right atrium.
- This re-entrant-circuit causes the **atria** to beat at about 300 beats per minute while the ventricles beat at a slower rate (often 75 to 150 beats per minute).
- Atrial flutter is less common than atrial fibrillation.





Normal Sinus Rhythm

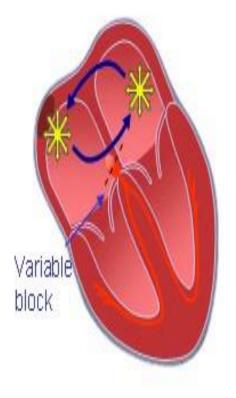


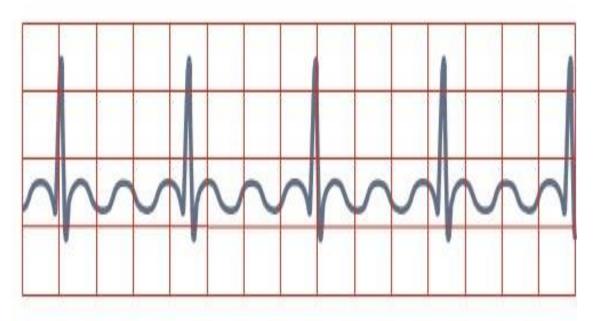
Atrial Flutter



Atrial rate = 260 - 320 bpm (ventricular rate 1:1, 2:1, 3:1; variable depending on AV

ATRIAL FLUTTER Impulses travel in circular course in atria

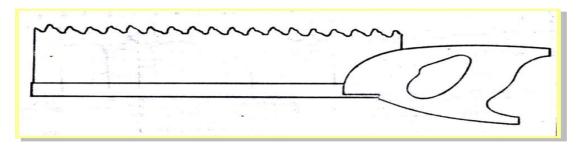




Rapid flutter waves, ventricular response irregular

Characteristics of Atrial Flutter

- ECG characteristics:
- P wave: Appears as saw toothed flutter waves

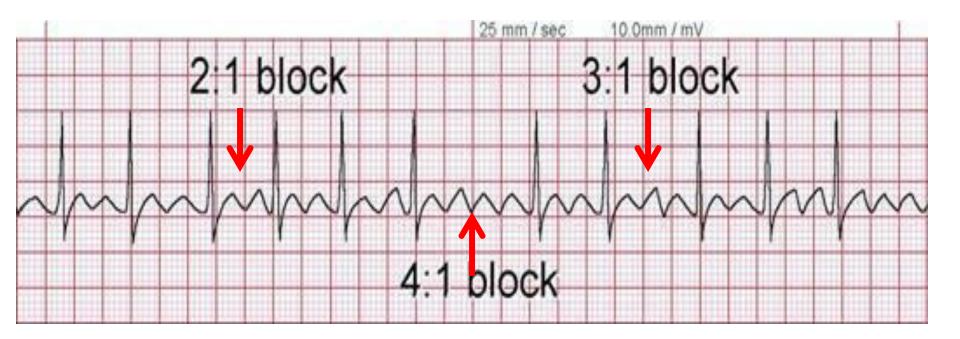


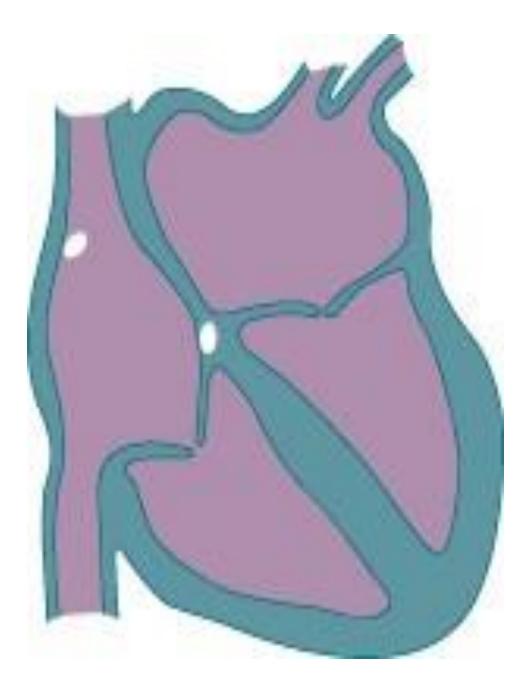
- Rate: 250-350 bpm for atrial, but ventricular rate is often slower
- P: QRS ratio: 2:1 or 3:1
- P-R Interval: unpredictable/ not measurable

Characteristics of Atrial Flutter

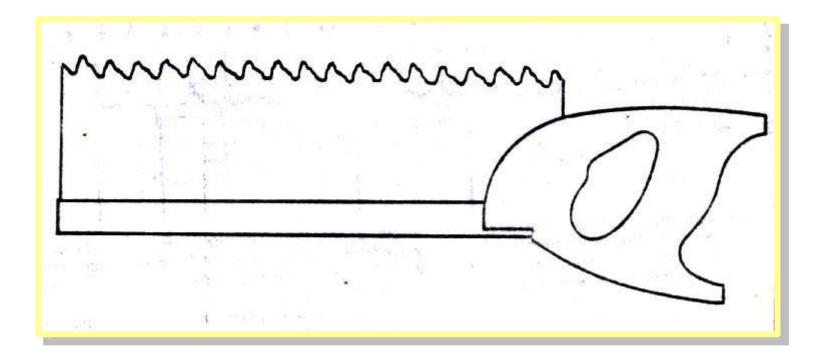
- ECG characteristics:
- R-R Interval: usually equal except with variable AV block
- QRS Complex: usually narrow

Atrial Flutter

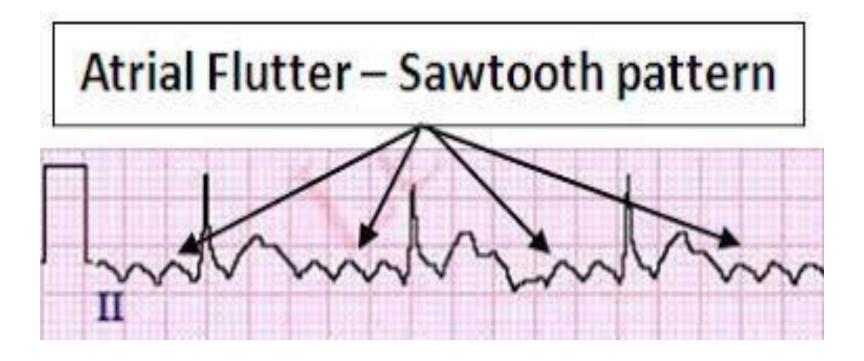


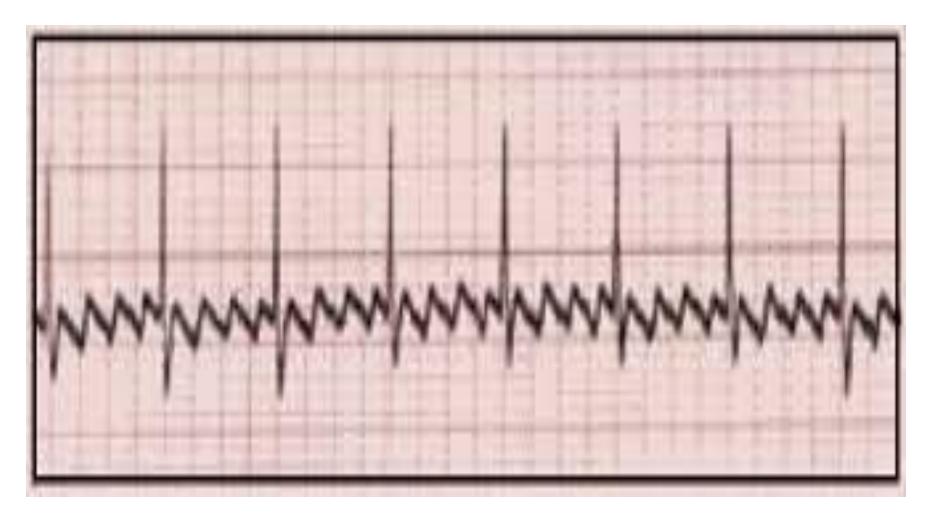


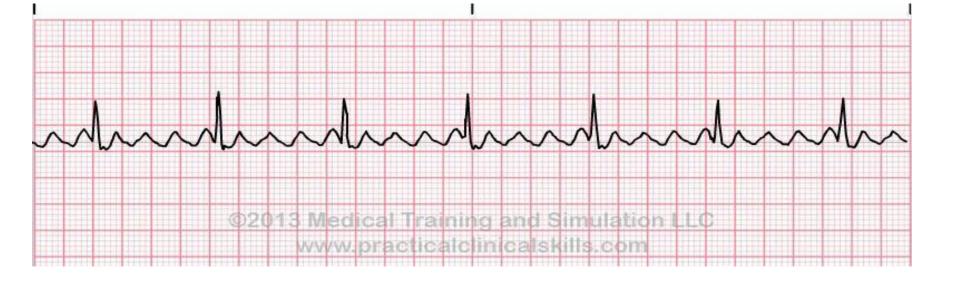
2:1 block on ecg

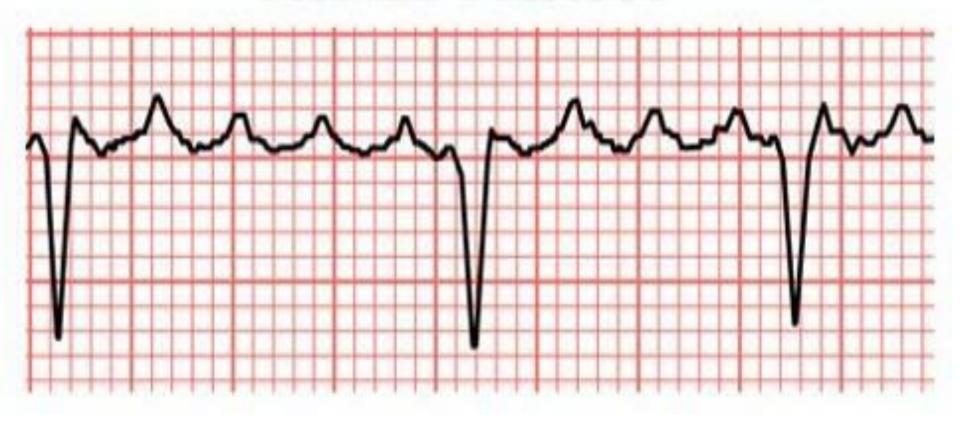


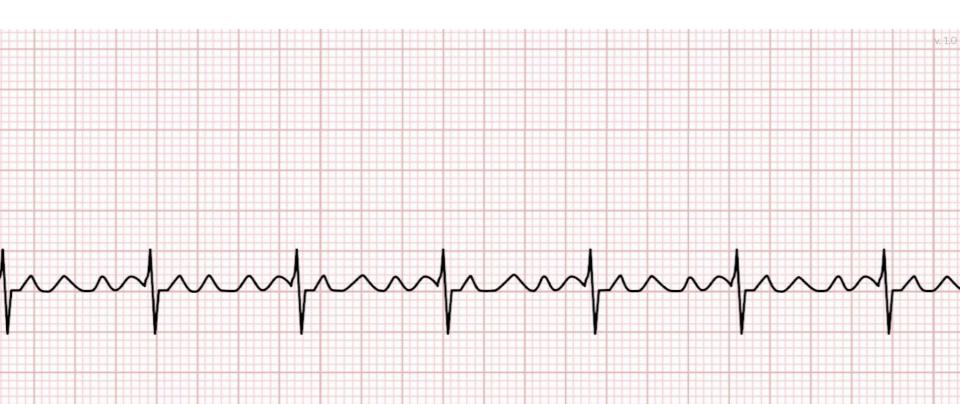
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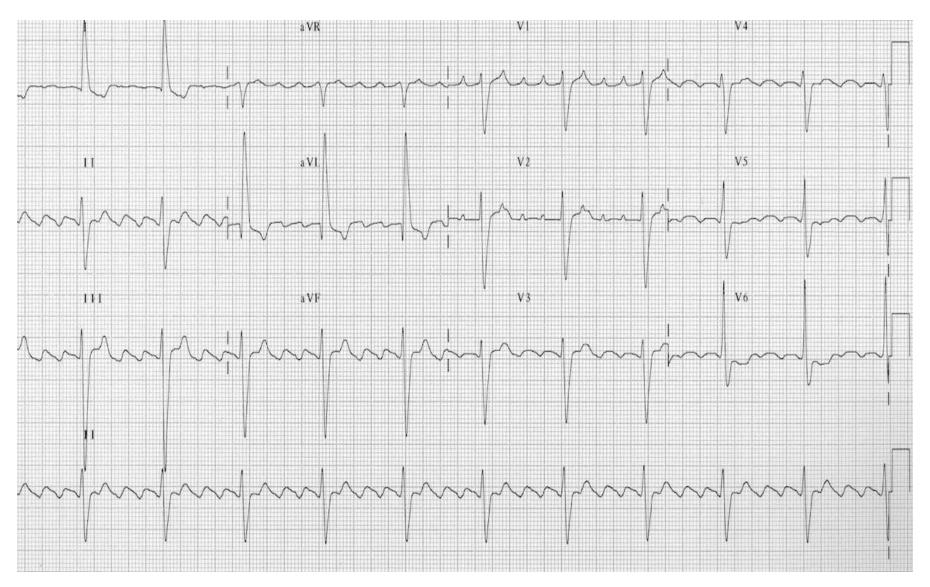


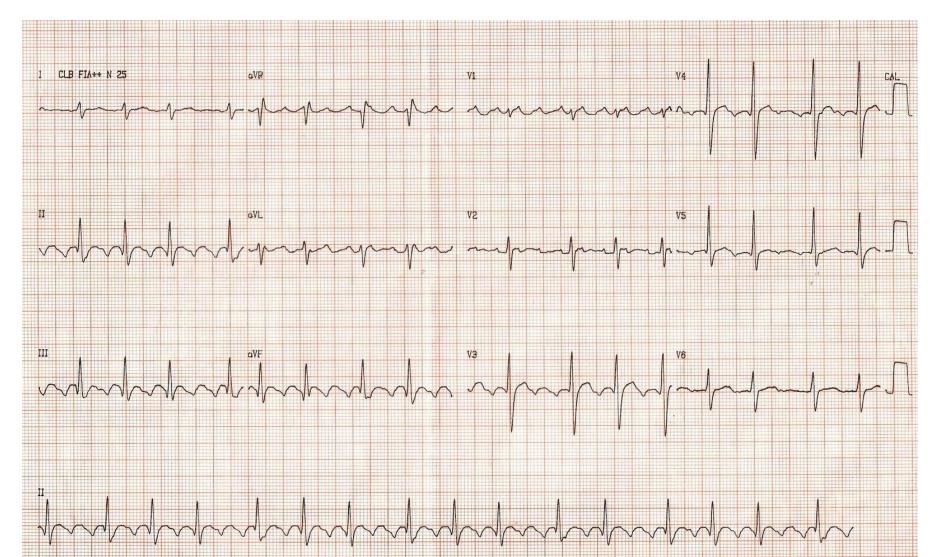










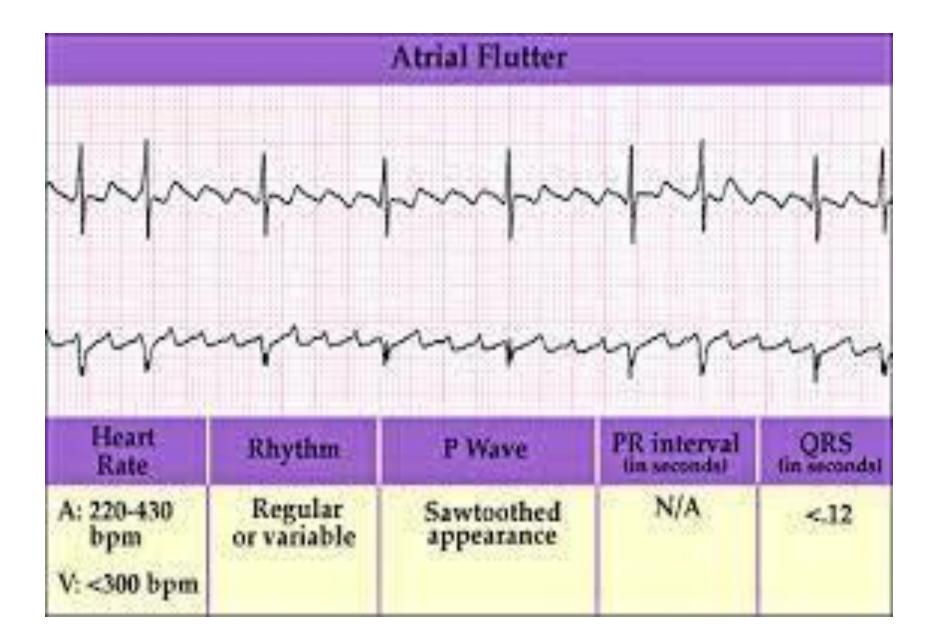


- Causes of atrial flutter:
- Heart abnormalities e.g. mitral valve stenosis
- Heart diseases e.g. CCF
- Conditions that affect heart function e.g. hyperthyroidism, pulmonary embolism, emphysema, COPD
- Consumption of substances that may affect the heart's electrical impulse e.g. alcohol, caffeine
- Open heart surgery

- Signs and symptoms:
- Palpitations
- Decreased cardiac output may cause symptoms of hemodynamic compromise (e.g, chest discomfort, dyspnea, weakness, syncope).

• Treatment:

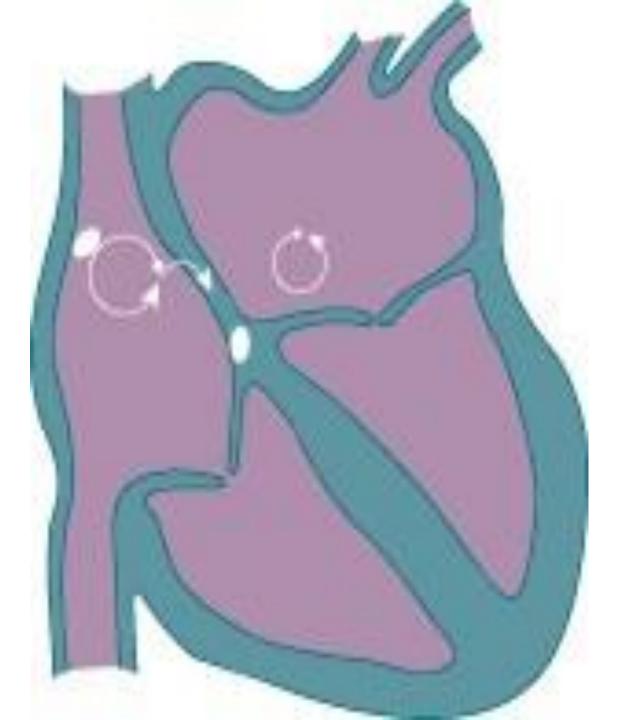
- Synchronized cardioversion
- Radiofrequency catheter ablation (technique that electronically burns and destroys some abnormal conduction pathways in the atria.)
- Drugs: (goal: rate control)
 - Ca⁺⁺ channel blockers to delay AV conduction
 - Amiodarone to delay AV conduction + prolong myocardial action potential

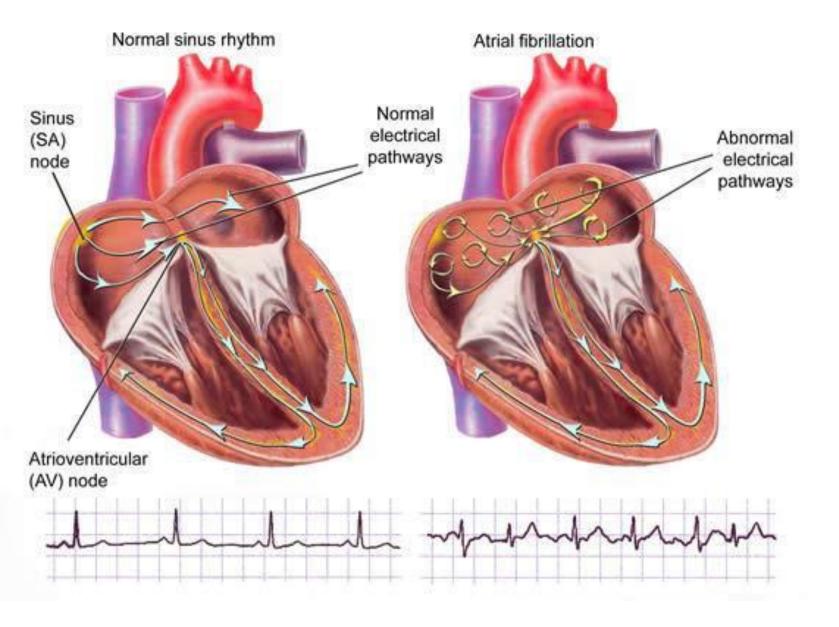


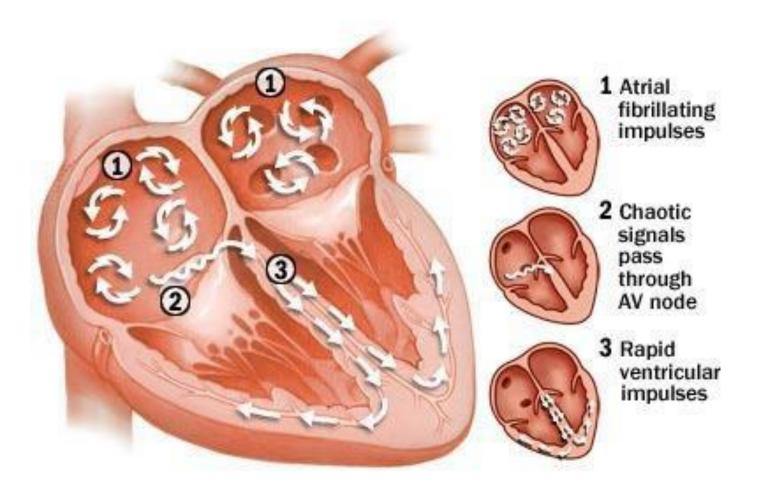
Atrial Arrhythmias

ATRIAL FIBRILLATION

- Occurs due to many foci points in the atria firing at the same time in a chaotic fashion leading to an absence of coordinated atrial contraction.
- The impulses begin and spread through the atria, competing for a chance to travel through the AV node.
- The resulting rhythm is disorganized, rapid, and irregular.
- Atria are said to quiver "like a bag of worms"







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Atrial fibrillation vs. SVT

- Atrial fibrillation is very similar to supraventricular tachycardia (SVT).
- Both conditions affect the atria of the heart.
- The difference is in the regularity of the heartbeat.
- SVT is a fast, regular heartbeat; a-fib is a fast, irregular heartbeat.
- In a-Fib, electrical signals fire in a very fast or uncontrolled fashion, causing the atrial chambers of the heart to twitch, quiver or contract.

- It is the most common atrial arrhythmia, and 85% percent of people who experience it are older than 65 years.
- Atrial fibrillation can cause blood to pool in the atria and this stasis may lead to formation of a blood clot

- Such clots can travel from the heart and lodge in an artery in another part of the body.
- If one of these clots blocks a vessel in the brain, it can cause a stroke.
- Atrial fibrillation causes about 15% of all strokes.
- Underlying heart disease or hypertension increases the risk of stroke from atrial fibrillation

1) Several electrical signals cross the atria at the same time 2) Atria (top chambers of the heart) quiver instead of contracting properly. This can cause blood to pool and form a clot.

Blood

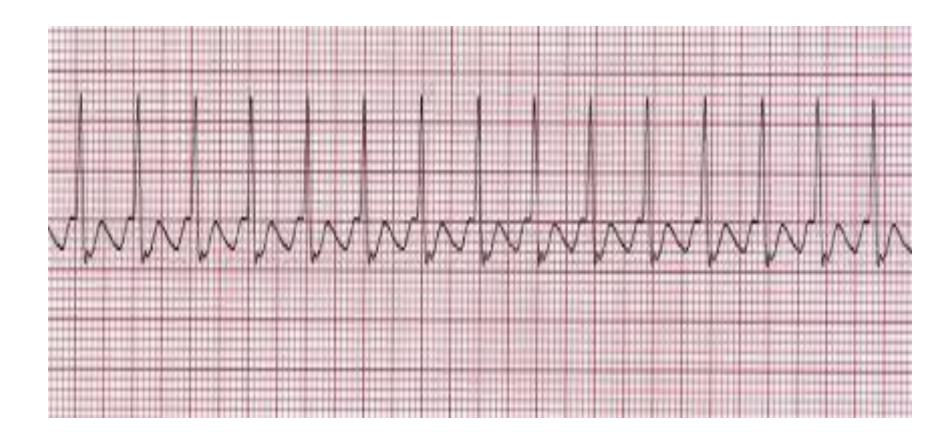
3) Some electrical signals reach the ventricles through the atriovetricular node

> Ventricles contract in an irregular and uncoordinated way.

When the rate of the electrical signals is very high, the atria quiver instead of contracting properly. They fail to pump all the blood out of the atria. The blood can pool and may form a clot. If a blood clot leaves the atria it can block an artery in the body, causing severe complications, such as stroke.

- ECG characteristics:
- HR: It can be variable and depends on how many atrial impulses are transmitted from the atria to the ventricles
- Atrial rate may be very fast (350-600), avg of 400
- Ventricular rate may be slow, normal or fast
- Rhythm: irregularly irregular (the AV node allows some of the impulses to pass through at variable intervals)
- Irregularly irregular ventricular response

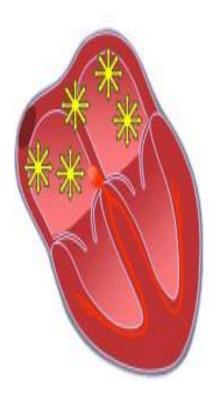
- ECG characteristics:
- P Waves: No P waves; fibrillation (f) waves
- P-R Interval: not applicable/ absent
- R-R Interval: usually unequal
- QRS Complex: usually narrow

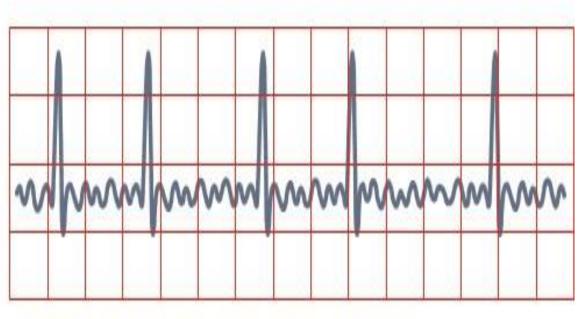




ATRIAL FIBRILLATION

Impulses have chaotic, random pathways in atria





Baseline irregular, ventricular response irregular

Risk factors of A-Fib

- Persons above 60 yrs of age
- Diabetes mellitus
- Hypertension
- Coronary artery disease
- Previous open heart surgery
- Valvular heart diseases

- Excessive alcohol or stimulants use
- Thyrotoxicosis
- Congestive heart failure

Signs & symptoms of A-Fibrillation

- Some patients may have no symptoms and it may be discovered during a routine hospital visit
- Shortness of breath
- Palpitations
- Chest pain
- Fatigue
- Dizziness
- Pre-syncope









THUNDER RUMBLING IN MY CHEST.



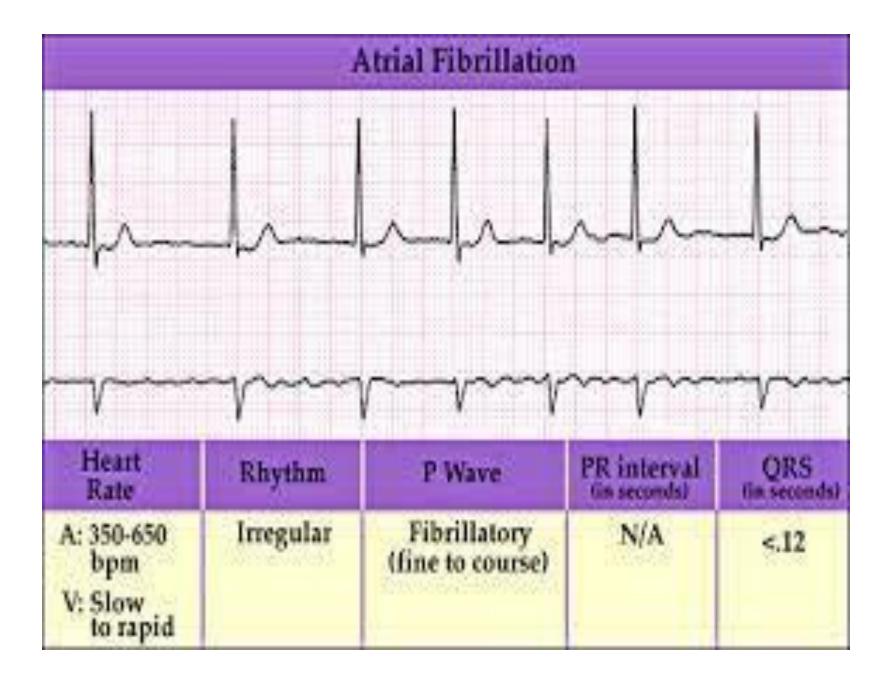
- Aims of treatment:
- Prevention of embolic stroke from thrombi that form in the fibrillating left atrium (coumadin, aspirin)
- Control the rate of the ventricular response (**beta-blockers, calcium channel blockers, digoxin**)
- Conversion and maintenance of the atrial rhythm to sinus rhythm (**drug therapy, catheter ablation**)

- Stroke prevention (48 hr rule):
- Rem: During atrial fibrillation the blood may pool and promote clot formation in the atrial
- If cardioversion is successful and a normal sinus rhythm (NSR) is achieved, the atria will contract forcibly and dislodge these clots to the pulmonary and systemic circulation which predisposes the patient to stroke

- Stroke prevention (48 hr rule):
- Patients with A-fib for more than 48 hrs or longer should be adequately anticoagulated to achieve an INR between 2-3 for at least 3 weeks prior to cardioversion
- Anticoagulants are used to minimize the risk of thrombo-embolism associated with cardioversion
- After cardioversion, the patient is put on anticoagulants for an additional 4 weeks

- Treatment:
- Rate control:
- These drugs slow the conduction at the AV node
 - Beta blockers- metoprolol
 - Calcium channel blockers -diltiazem
- Rhythm control: (conversion to sinus rhythm)
- These drugs convert atrial rhythms to sinus rhythm
 - Amiodarone 150 mg iv infusion
 - Choice of drug to use is dependent on the underlying illness that the patient has

- Other forms of treatment:
- Cardiac ablation and permanent pacing
- Relieve hypoxia via oxygen
- Cardioversion —if symptomatic (carry out cardioversion with a lot of caution)



WOLFF-PARKINSON-WHITE SYNDROME (WPW)

Atrial Arrhythmias (RE-ENTRY MECHANISM)

Atrio-Ventricular Reentrant tachycardia (AVRT)

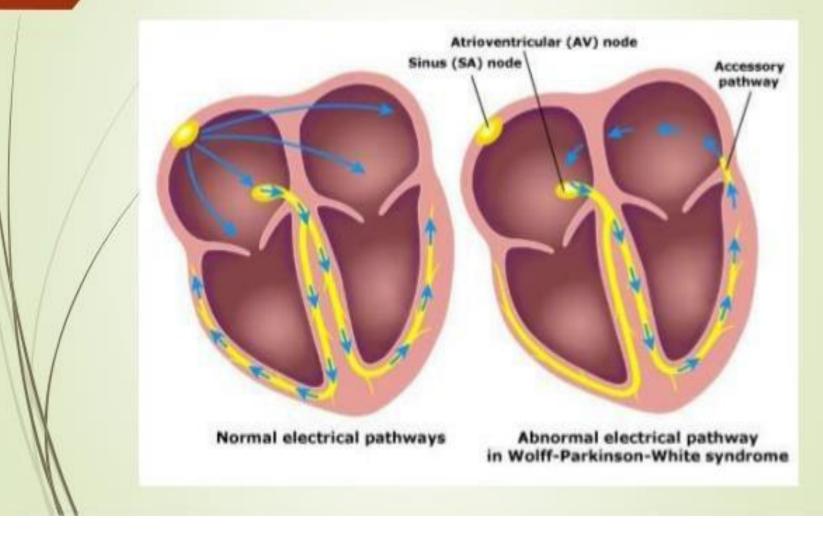
- In AVRT there is a large circuit comprising the AV node, the His bundle, the ventricle and an abnormal connection from the ventricle back to the atrium.
- This abnormal connection is called an accessory pathway

Atrio-Ventricular Reentrant tachycardia (AVRT)

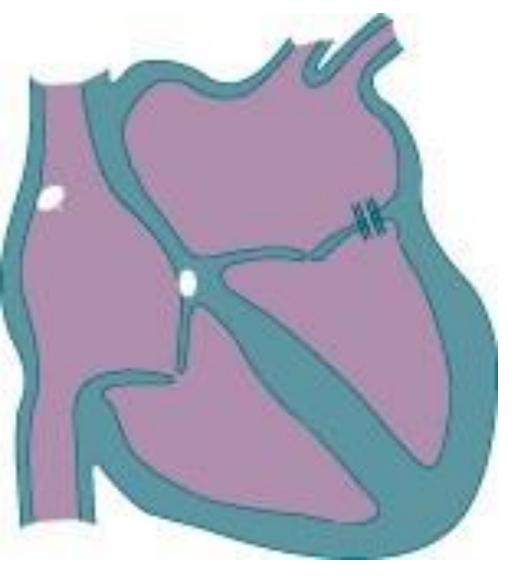
- This pathways cause the ventricles to depolarize earlier than expected
- Accessory pathways result from incomplete separation of the atria and the ventricles during fetal development.
- One example of these accessory pathways is **bundle** of kent which causes Wolff-Parkinson-White syndrome arrythmia

- Wolff-Parkinson-White syndrome is a condition in which there is an extra electrical pathway between the atria and ventricles
- This extra pathway is called **bundle of kent**
- It can lead to periods of rapid heart rate (tachycardia)

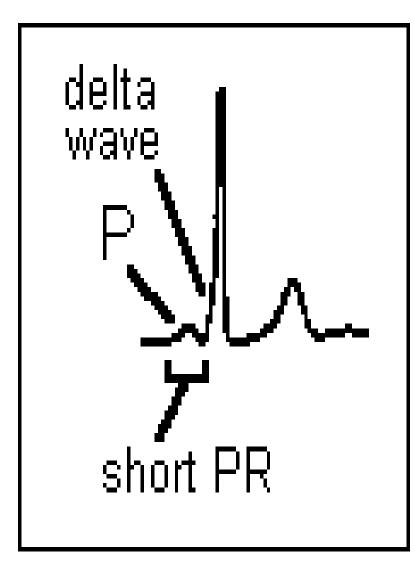
Wolf Parkinson White Syndrome (WPW)

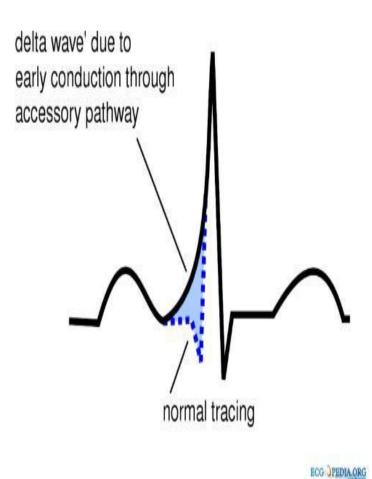


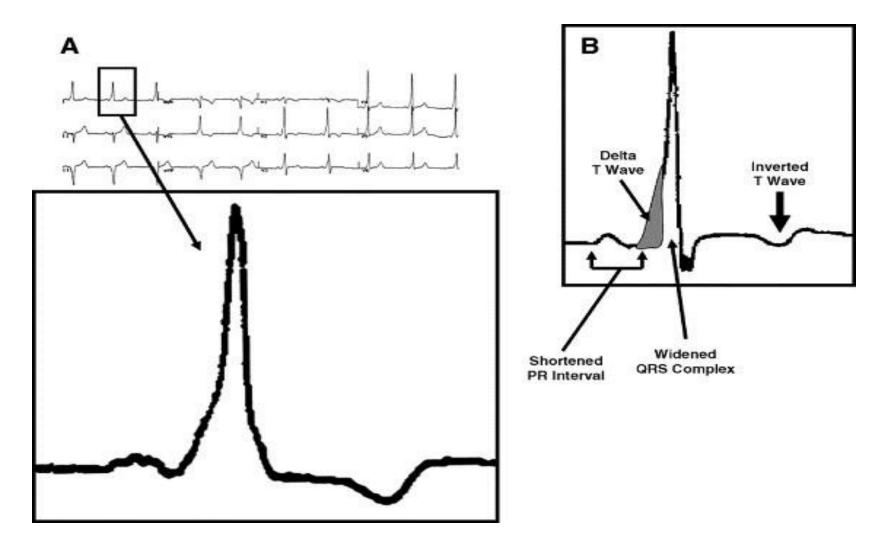
- Depolarization begins at the SA node
- The wave of depolarization spreads across the atria
- It reaches the AV node and the accessory bundle
- Conduction is delayed as usual by the in-built delay in the AV node
- However, the accessory bundle has no such delay and depolarization begins early in the part of the ventricle served by the bundle

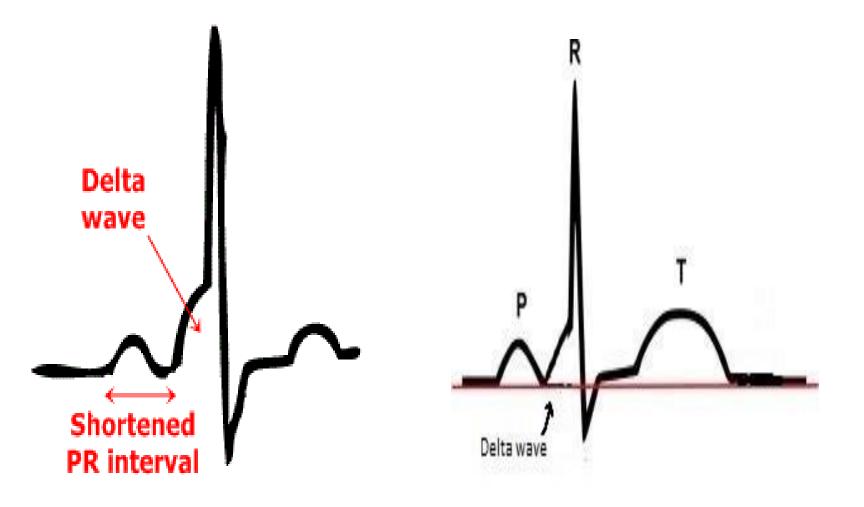


- This results in earlier onset of left ventricular depolarization.
- The premature depolarization is represented by the delta wave on ECG
- A delta wave is an initial slurring of the QRS complex





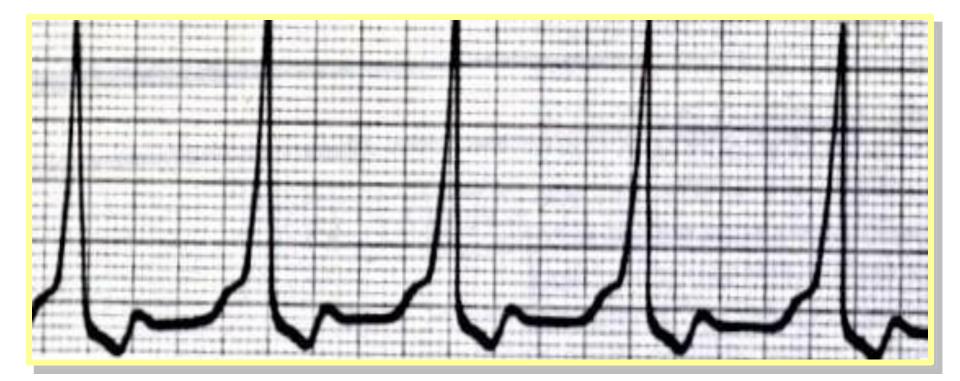


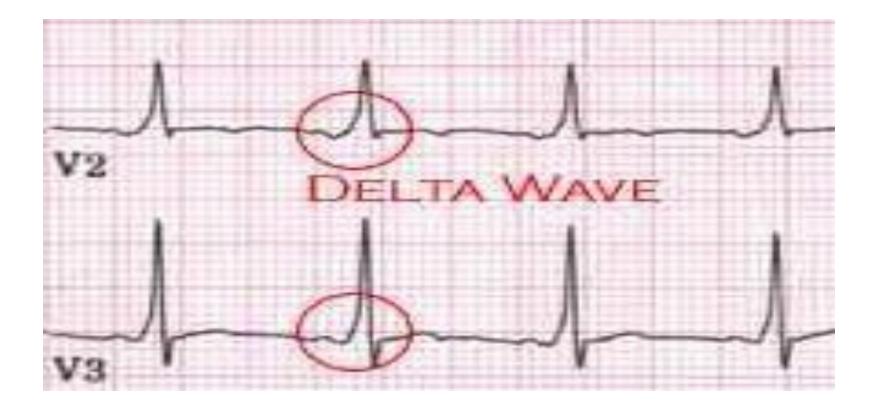


- ECG features:
 - Short PR interval
 - Presence of delta wave
 - Widening QRS Complex because of spread of the impulse in the ventricles slowly

WPW

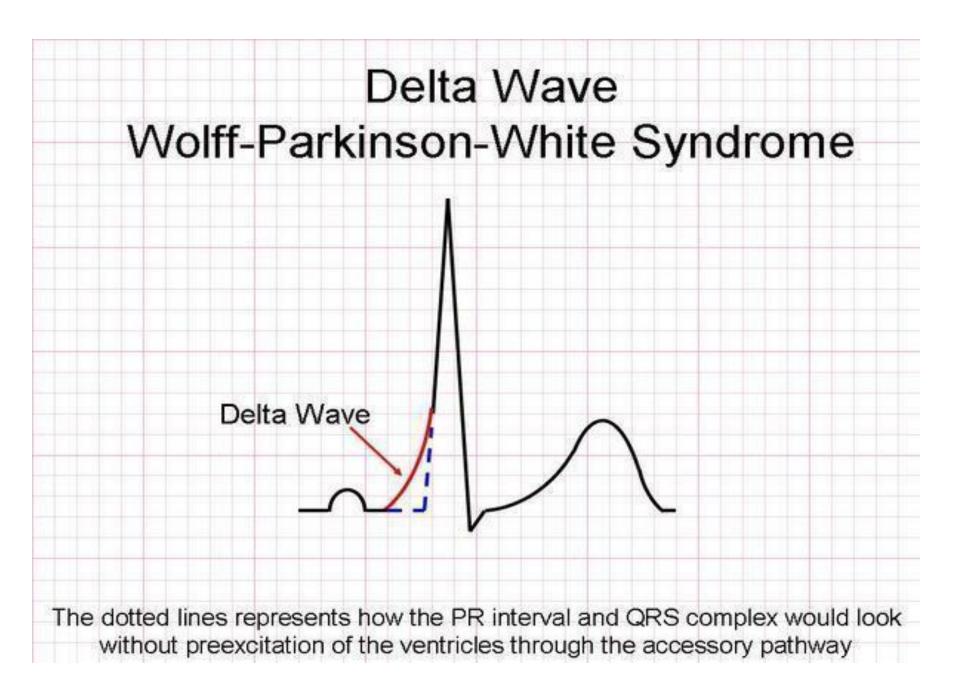
(Wolf Parkinson White Syndrome)





WPW:

think Amiodarone



WPW

(Wolf Parkinson White Syndrome)

- Signs and symptoms of WPW
- Chest pain or chest tightness
- Dizziness
- Light-headedness
- Fainting
- Palpitations
- Shortness of breath

WPW

(Wolf Parkinson White Syndrome)

• Treatment:

- Medicine- amiodarone 150 mg iv infusion
- Electrical cardioversion
- Catheter ablation- This procedure involves inserting a tube (catheter) into a vein through a small cut near the groin up to the heart area. When the tip reaches the heart, the small area that is causing the fast heart rate is destroyed using a special type of energy called radiofrequency or by freezing it (cryoablation).

AV- NODAL RE-ENTRANT TACHYCARDIA (AVNRT)

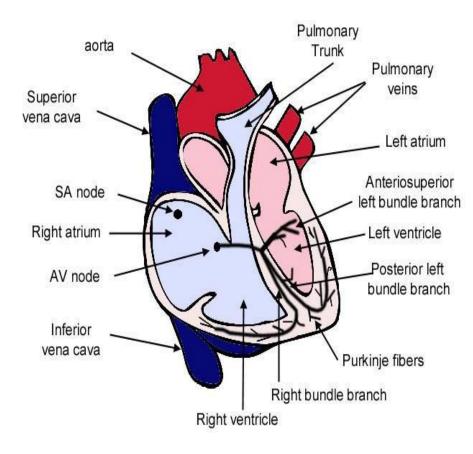
Atrial Arrhythmias (RE-ENTRY MECHANISMS)

Atrioventricular nodal reentrant tachycardia (AVNRT)

- Atrio-ventricular nodal reentrant tachycardia (AVNRT) is an arrhythmia that occurs because an extra pathway lies in or near the AV node
- This pathway causes the impulses to move in a circle manner and re-enter areas it already passed through.

The conducting system

- SA Node
- Inter-nodal pathways
- AV Node
- Bundle of HIS
- Bundle Branches
- Purkinje Fibers

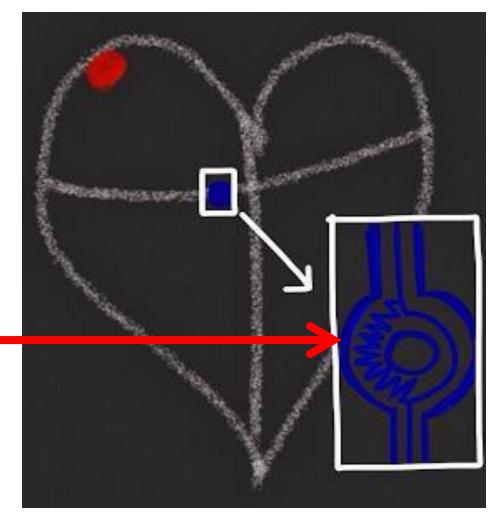


Conducting system

- The AV node is responsible for conduction of the impulse from the atria to the ventricles.
- The impulse is delayed slightly at this point to allow complete filling of the ventricles before they contract.
- The impulse continues through the Bundle of His and down the left and right bundle branches of the Purkinje fibers.
- The Purkinje fibers conduct the impulse to all parts of the ventricles, causing contraction

AV Node explained

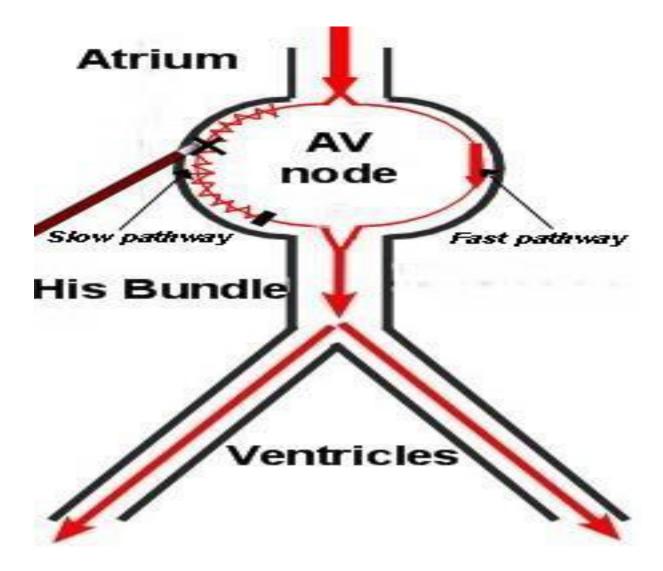
- The AV Node itself is a little more complex than a single pathway from top to bottom.
- Under a microscope it actually looks like this



AV Node explained

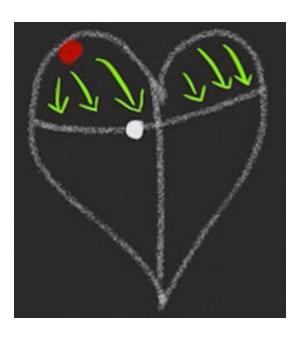
- Therefore the AV Node in a large percentage of people has TWO pathways that a signal can take from the Atria to the Ventricles
- One of the pathways is a relatively FAST Pathway (carries the message quickly) and the other pathway is a relatively SLOW pathway (carries the message slowly)
- The zig-zag line shows the slow pathway and the straight line the fast Pathway. The pathways are connected at the top and bottom of the AV Node.

Electrical pathway of an impulse at the AV Node



Conduction at the AV Node

- Step 1 & 2: The SA Node starts a heart beat that travels cell to cell through the atria and arrives at the AV Node
- Step 1





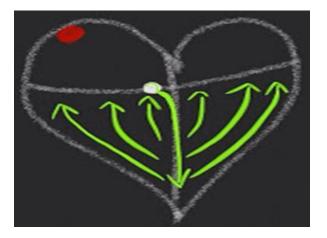


Conduction at the AV Node

- Step 3 & 4: The signal travels relatively quickly around the fast pathway (green line) and down into the ventricles starting a normal ventricular conduction; the signal travels slowly down the slow pathway.
- Step 3





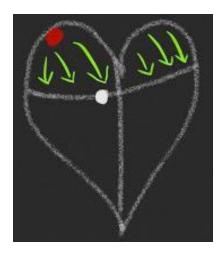


Conduction at the AV Node

- Step 5: When the signal travelling down the slow pathway eventually arrives at the bottom of the AV Node, all the tissue that has recently been active is in the refractory period (absolute refractory period).
- Therefore the signal hits a dead end and dissipates (is cancelled out).



Step 1:



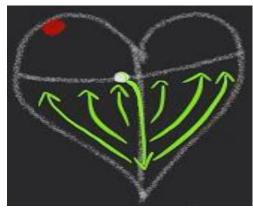
Step 2:



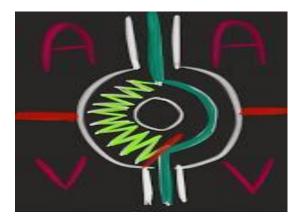
Step 3:



Step 4:



Step 5:



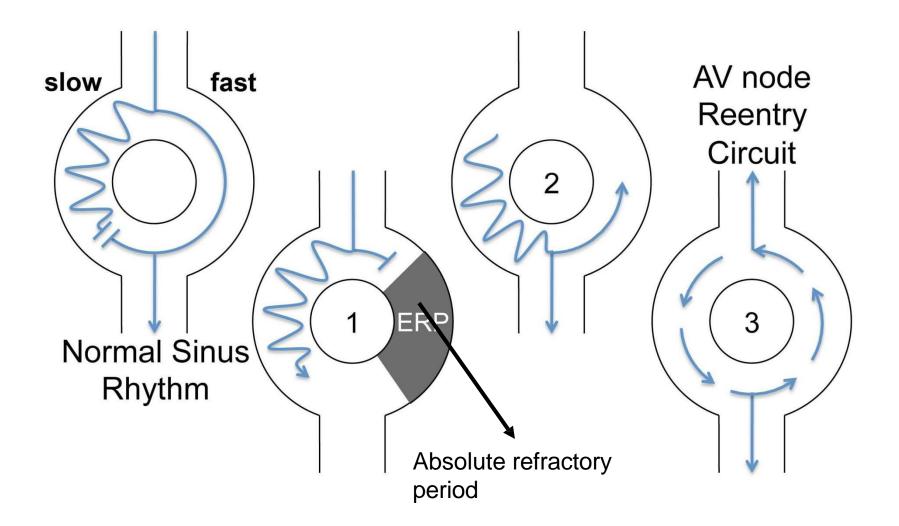
AVNRT Pathophysiology

- Pathophysiology:
- Normally, impulses from the atria travel to the ventricles only through the fast pathway
- When a **premature atrial impulse** reaches the AV node, it encounters a refractory fast pathway and must enter the slow pathway

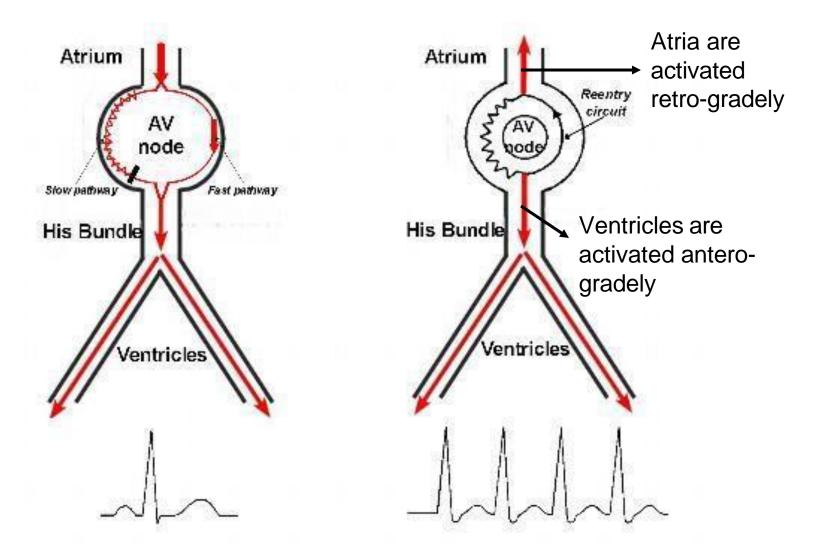
AVNRT Pathophysiology

- By the time the impulse on the slow pathway reaches the end of the AV node, the fast pathway has reset, allowing the impulse to travel backwards up the fast pathway
- This creates a reentry circuit which cycles round, activating the ventricles antero-gradely and the atria retro-gradely (backwards)
- The atria are unable to empty contents into the ventricles and so blood is backed up into the venous system.

AVNRT



AVNRT



AVNRT Pathophysiology

- The premature atrial impulse may occur spontaneously in patients or upon provocation with exertion, coffee, tea or alcohol
- It may occur in young and healthy patients as well as those suffering from chronic heart disease.

Atrioventricular nodal reentrant tachycardia (AVNRT)

- The cause of the extra pathway in the AV node, is unknown.
- The extra pathway is present since birth, but most often causes an arrhythmia after the heart has reached its full size when the patient is in their 20s or 30s.
- Women (75%) are more likely to develop the condition than men.
- Avnrt can be diagnosed via holter monitoring and ECG

Atrioventricular nodal reentrant tachycardia (AVNRT)

- On ECG:
- P waves are often hidden or immediately after QRS and are usually in an opposite direction to QRS
- May have a notched QRS appearance



AVNRT

- ECG Characteristics:
- P wave is often absent because it is usually hidden in the QRS complex
- When present, the P wave will be upside down because it is travelling in the opposite direction, towards the electrode it normally moves away from.
- Narrow complex QRS

Atrioventricular nodal reentrant tachycardia (AVNRT)

- Patients will typically complain of the sudden onset of rapid, regular palpitations
- The patient may experience a brief drop in blood pressure causing pre-syncope or occasionally syncope
- AVNRT is not a life threatening arrythmia

Atrioventricular nodal reentrant tachycardia (AVNRT)

- Treatment:
- If patient is stable:
- Adenosine, amiodarone, calcium channel blockers, beta blockers
- If unstable:
- Synchronized cardioversion

- Vagal stimulation
- Synchronized cardioversion
- Drugs:
 - Adenosine 6mgs, 12mgs iv rapid push with flush
 - Amiodarone 150mgs iv infusion
 - Calcium channel blockers
 - Beta blockers

• Vagal stimulation:

- Vagus nerve; 10th cranial nerve
- Has innervations to the viscera- heart, lungs, stomach
- Can be stimulated via coughing, squatting, carotid massage, valsava maneuver, abdominal pressure
- Stimulation causes decreased heart rate

- Synchronized cardioversion:
- Shock delivery that is timed with the R wave deflection
- Avoids shock delivery during the relative refractory phase of myocardial activity (can cause Vfib)
- Electrical doses are lower than for un-synchronized cardioversion
- Usually performed under sedation or anesthesia

- Synchronized cardioversion:
- Electrical doses:
- A fib 100-200J
- A flutter & SVT 50-100J (200, 300, 360)

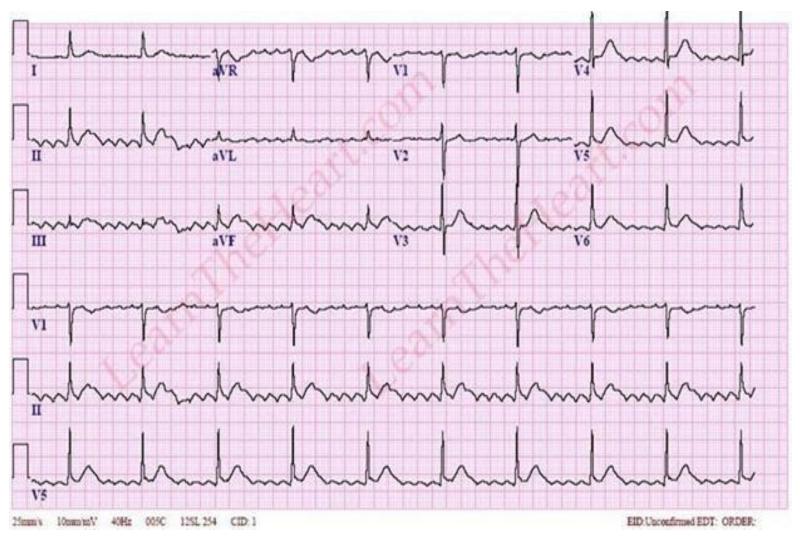
The End

(Whew!!!!!)

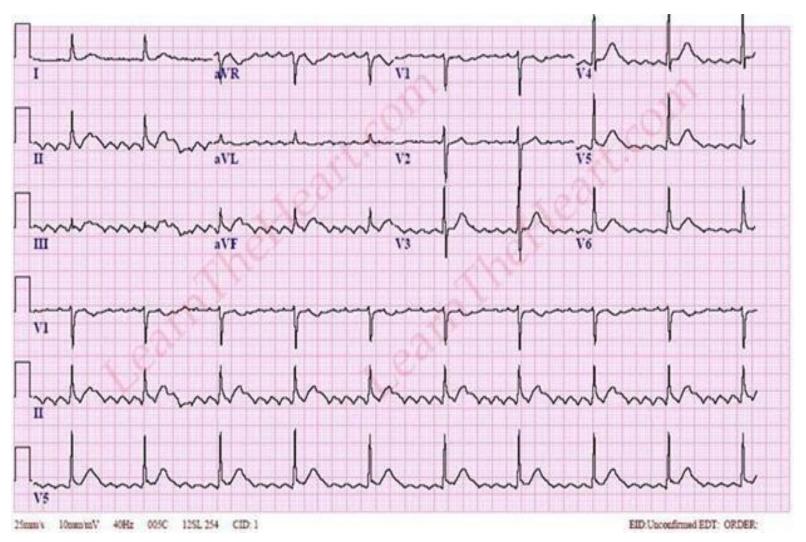


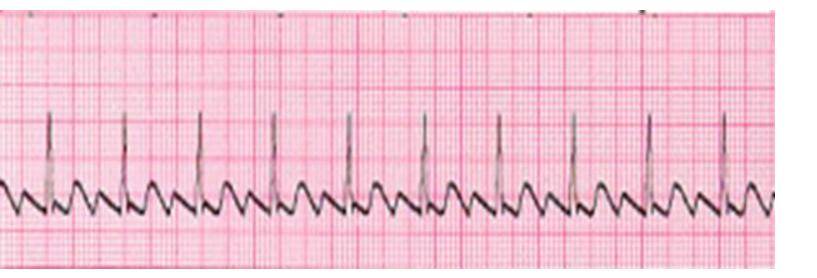
No.1- Normal sinus rhythm



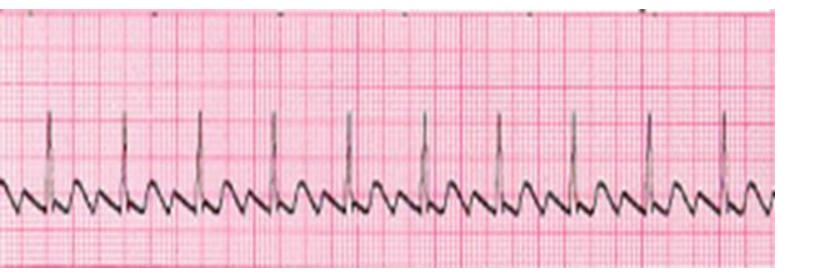


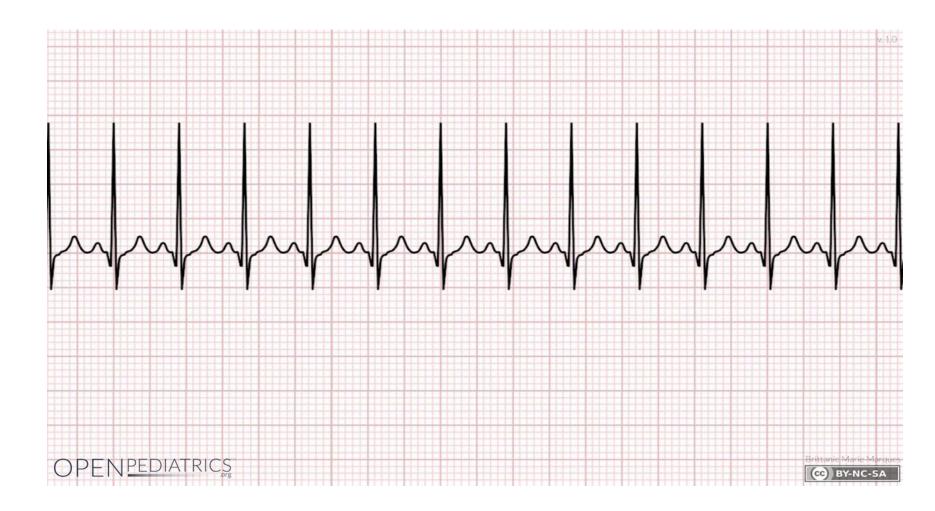
No. 2- Atrial flutter



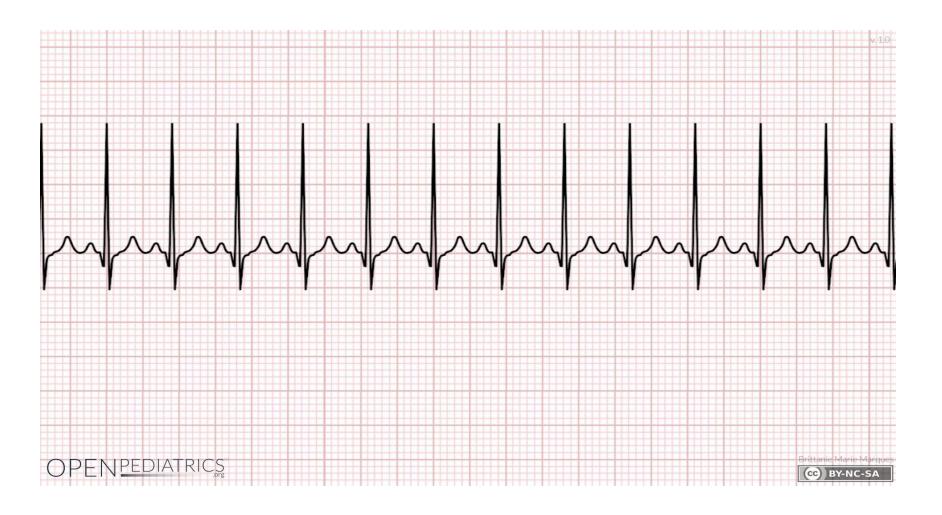


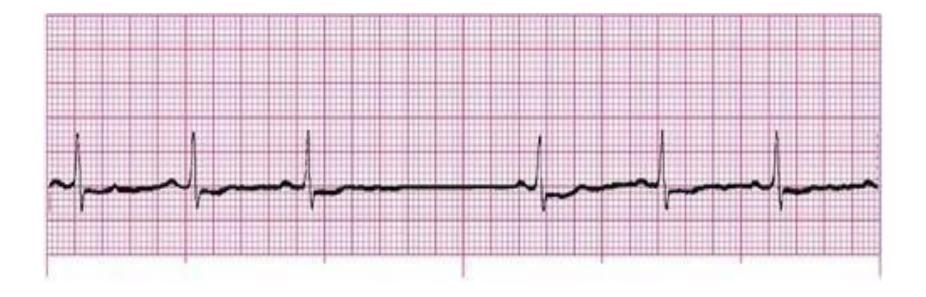
No.3- Atrial Flutter



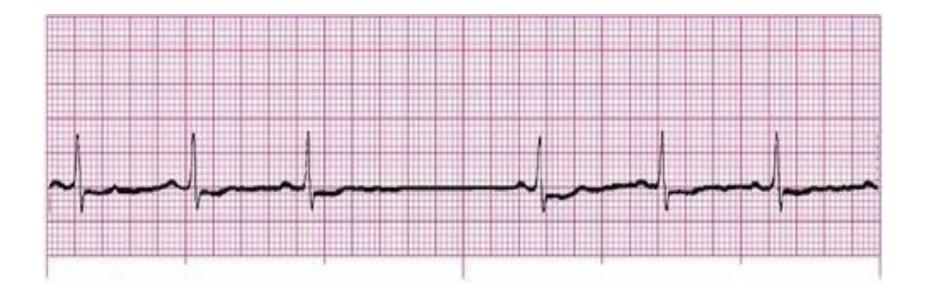


No. 4- Sinus tachycardia





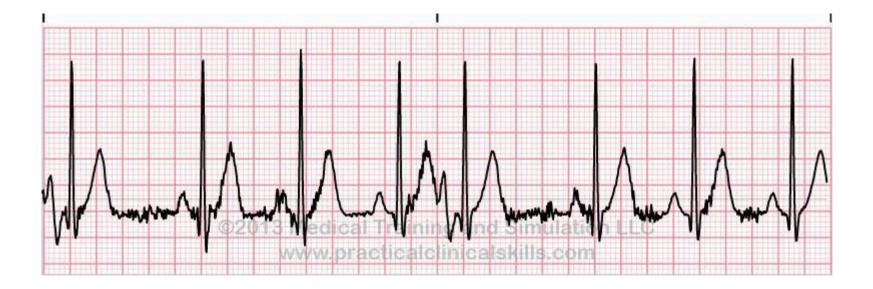
No. 5 - Sino-atrial block



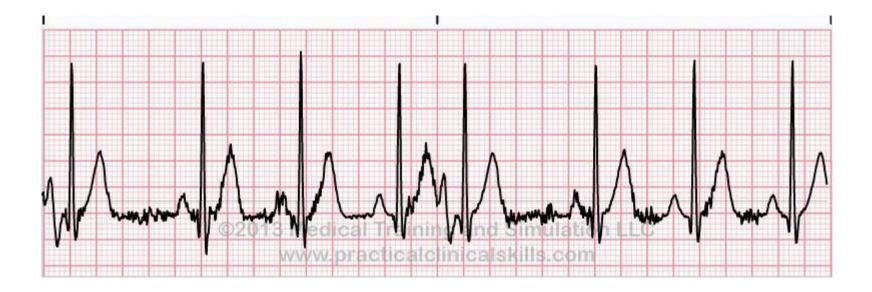


No. 6- Premature atrial complexes





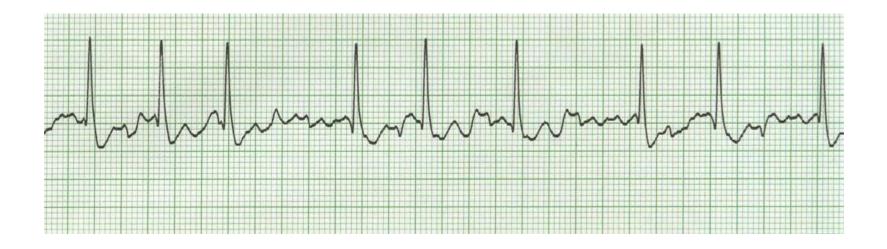
No. 7- Premature Atrial Complex with artifact



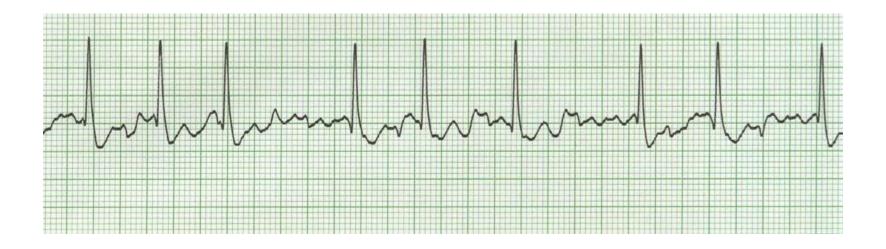


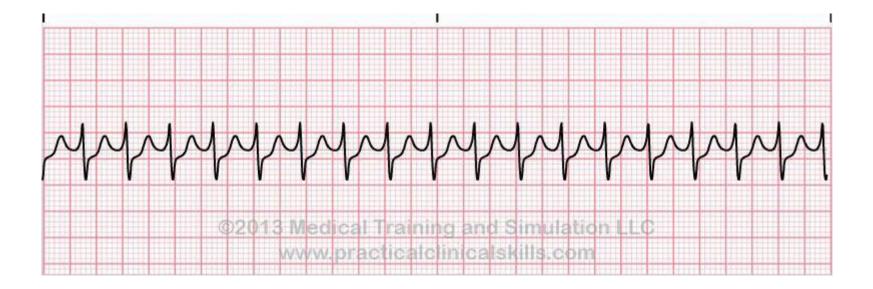
No. 8- Sinus Bradycardia





No.9- Atrial fibrillation





No.10- Supraventricular tachycardia

