



CVS PHYSIOLOGY



Modified NO: 7



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تدقيق ليث الخزاعلة

الدكتور: فاطمة ريلات

Cardiovascular Physiology

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In external sources section we added
arrhythmia chapter from Guyton and hall
textbook.

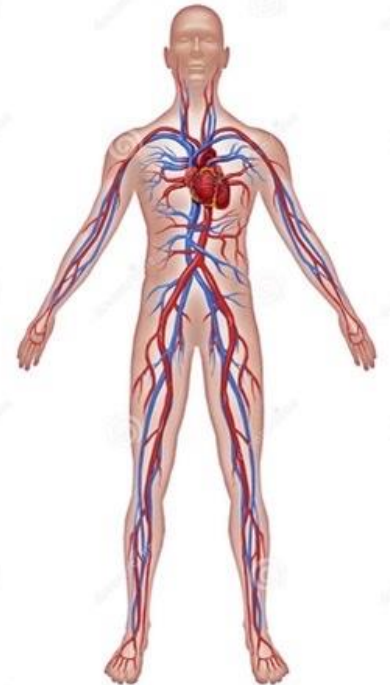
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Slides

Doctor

Additional info

Important



References

9TH Edition

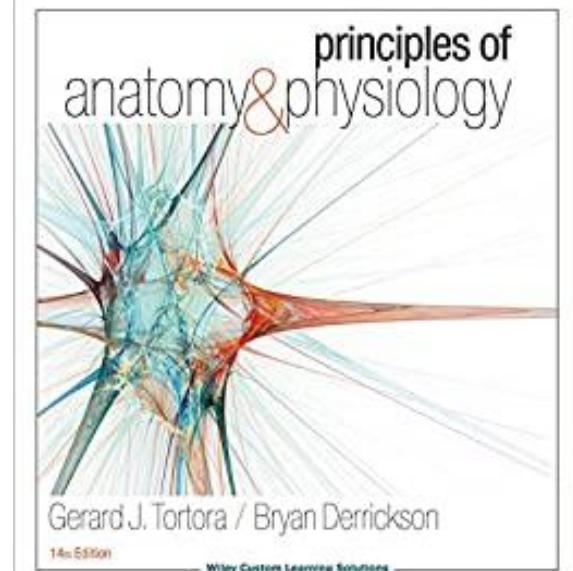
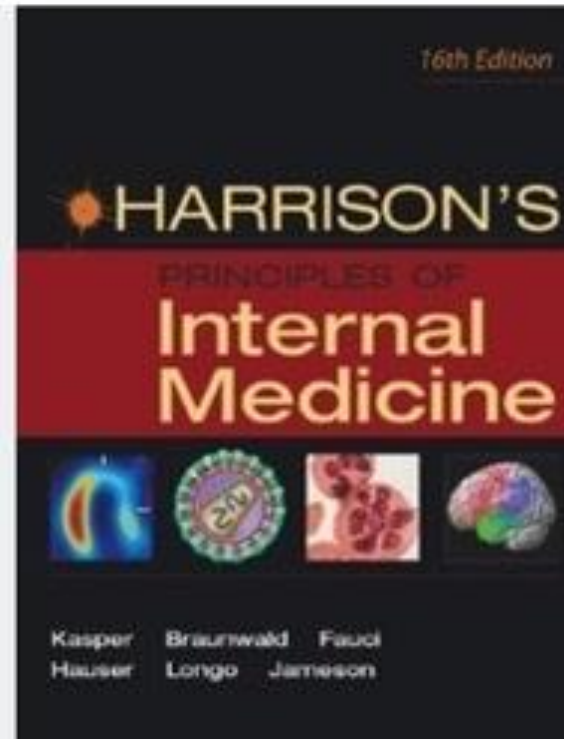
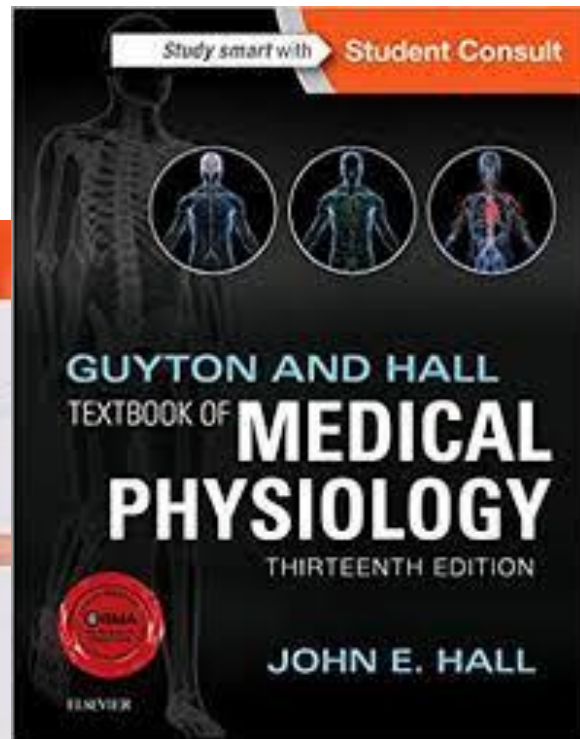
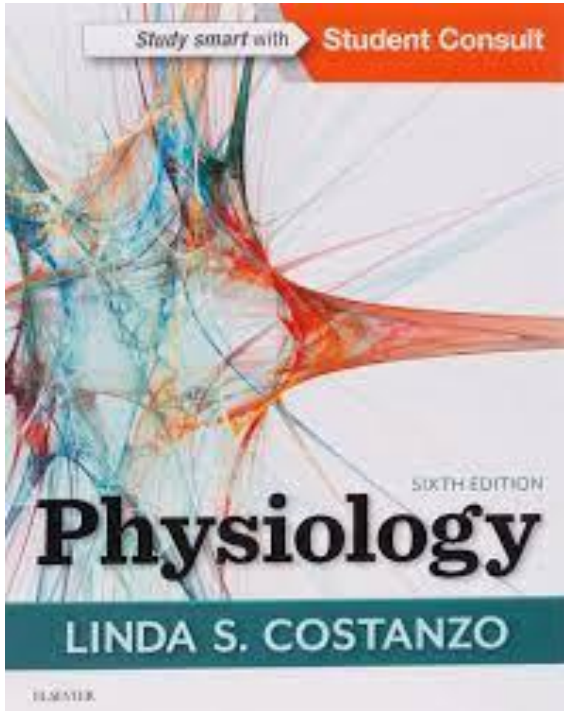
Human Physiology From Cells to Systems

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Cardiac Arrhythmias-1

Cardiac Arrhythmias (abnormal rhythms)

Normal rhythm is when cardiac impulse:

- originates in SA node
- rate: 60-100 bpm (beats per minute)
- follows the normal pathway in the conductive system.
Remember: SA node —> AV node (and AV delay) —> AV bundle (left and right) —> Purkinje fibers
- follows the normal velocity of conduction in different parts of the conduction system.

Cardiac Arrhythmias

Mechanisms by which cardiac arrhythmias can develop:

- enhanced autorhythmicity (in the pacemaker cells).
- triggered autorhythmicity (in the other cells).
- Reentry (in any part of the conduction or the cardiac system).

1. **Enhanced Autorhythmicity:** This occurs when pacemaker cells increase their firing rate due to factors like increased sympathetic activity, fever, or ischemia. This leads to abnormal, premature beats and arrhythmias, such as sinus tachycardia.

2. **Triggered Autorhythmicity:** Abnormal depolarizations (afterdepolarizations) follow an action potential and trigger arrhythmias.

- **Early Afterdepolarizations (EADs):** Occur during repolarization, often due to prolonged QT intervals, leading to torsades de pointes.

- **Delayed Afterdepolarizations (DADs):** Happen after full repolarization, commonly due to calcium overload, causing arrhythmias like ventricular tachycardia.

3. **Reentry:** A circular pathway of electrical activity that repeatedly excites the myocardium, causing sustained arrhythmias. This is common in atrial flutter and ventricular tachycardia and results from slowed or blocked conduction paths.

Cardiac Arrhythmias

The causes of the cardiac arrhythmias are usually one or a combination of the following abnormalities in the rhythmicity-conduction system of the heart:

- Abnormal rhythmicity of the pacemaker.
- Shift of the pacemaker from the sinus node to another place in the heart.
- Blocks at different points in the spread of the impulse through the heart.
- Abnormal pathways of impulse transmission through the heart.
- Spontaneous generation of spurious impulses in almost any part of the heart.

- Cardiac arrhythmia results from abnormalities in the rhythmicity or the conduction system.
- It may be caused by abnormality in the rhythmicity of the pacemaker (either increase or decrease), it also may be caused by shift of the pacemaker to be another structure in the heart, or it may be caused by a block in the conduction at different areas, or it may be caused by spontaneous generation of impulses at any part of the heart.
- There are many classifications for the types of arrhythmias, they can be divided according to the rate (either tachyarrhythmias – fast rhythms- ,or bradyarrhythmias –slow rhythms-).
- The other classifications is anatomical classification in which it can be divided according to the structure there is an abnormality in it , for example:
 - SA node arrhythmias.
 - Atrial arrhythmias.
 - AV node arrhythmias (junctional arrhythmias).
 - Ventricular arrhythmias (also divided into ventricular and supraventricular arrhythmias).

Cardiac Arrhythmias

- Rhythm disturbances result from abnormalities of impulse formation, impulse conduction, or both.
- Types:
- Rate: tachyarrhythmia vs bradyarrhythmia.
- Anatomical: supraventricular vs ventricular.

Cardiac Arrhythmias

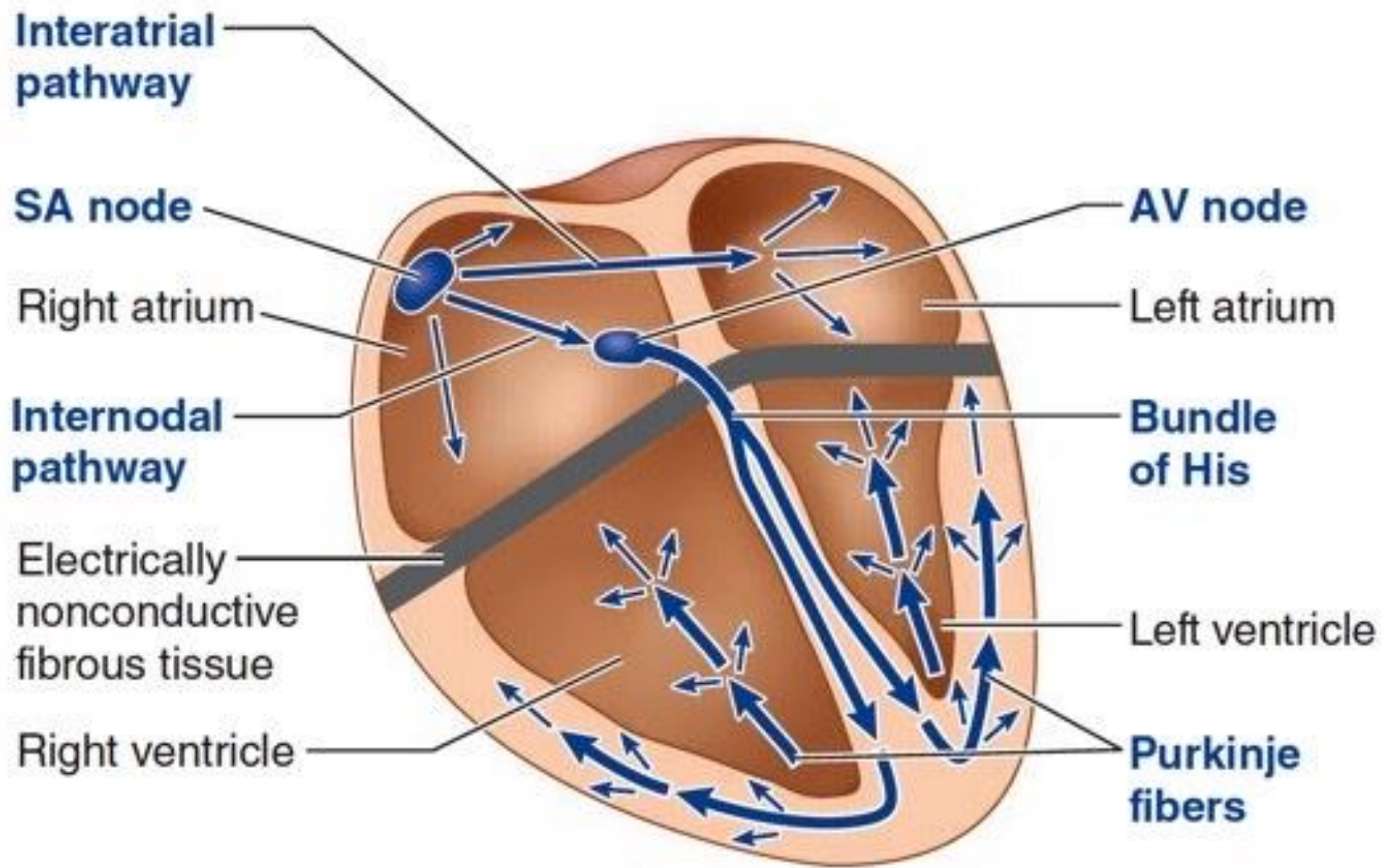
- Symptoms:
- Palpitations, dizziness or fainting, chest pain or shortness of breath, fatigue, or asymptomatic!

- Causes:
- Inherited, congenital, ischemia after myocardial infarction , aging (results in changing in the cells leading to arrhythmias), infiltrative diseases (such as rheumatic fever) , infections, electrolyte disturbances (changes mainly in the K and Ca) , medications(digoxine), iatrogenic (caused by doctors themselves, for example: during catheterisation there might be induction of certain foci for the arrhythmias), alcohol, nicotine and caffeine.

Cardiac Arrhythmias

- Diagnosis:
- ECG (the main diagnostic tool that leads us to many information about different types and locations of arrhythmias), monitors, and other diagnostic tools (such as echocardiogram, electrophysiology studies or MRI (Magnetic Resonance Imaging)).
- Treatment (depending on the cause of arrhythmias):
- Pharmacological (increase or decrease the heart rate), catheter ablation (to get rid of these foci), implantable devices such as: pacemaker or defibrillator, life style modification (such as alcohol cessation and nicotine smoking and limiting caffeine intake).

The term “foci” refers to specific points or locations where something originates or is concentrated. In the context of arrhythmias, **foci** refers to abnormal sites in the heart’s electrical conduction system where irregular electrical impulses originate. These ectopic foci are responsible for initiating abnormal heart rhythms, such as atrial fibrillation, ventricular tachycardia, or premature beats.

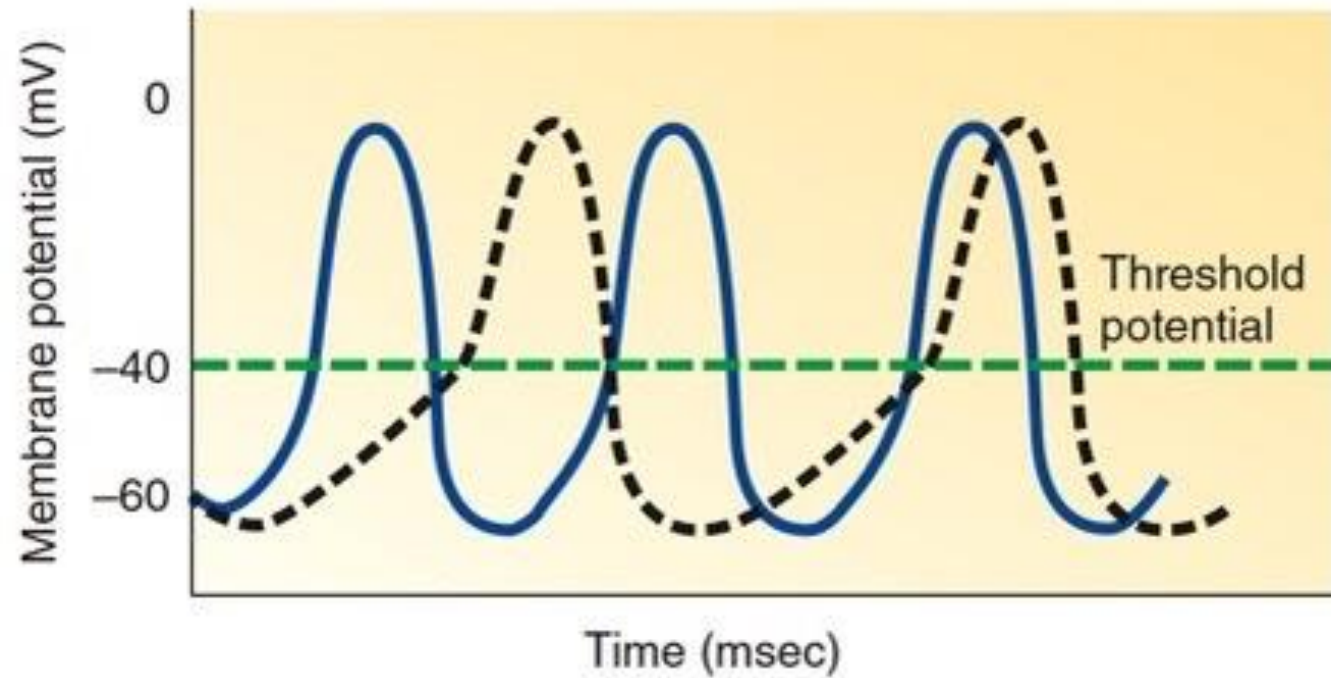


Sinus Tachycardia

- Fast heart rate: >100 beats per minute in adult.
- ECG: normal except heart rate (R-R interval) increased. In tachycardia, the number of R waves (or R-R intervals) per minute increases, but the length of each R-R interval decreases.
- Tachycardia can be physiological or pathological.
- Causes: hyperthermia (if you have fever), blood loss anemia, dehydration, strong emotions like anger or anxiety, weak heart muscle, and toxic conditions of the heart, also if you take certain medications or even drinking caffeine.
- So basically, any thing that stimulates the sympathetic system (sympathomimetic factors) can increase your HR.



Remember that the normal HR (heart rate) is 60-100 bpm. If the HR is > 100 bpm this indicates tachycardia. The ECG is normal except for the HR



- The dashed curve represents the normal autorhythmicity in the SA node.
- The blue curve represents the autorhythmicity in the SA node during sympathetic stimulation.

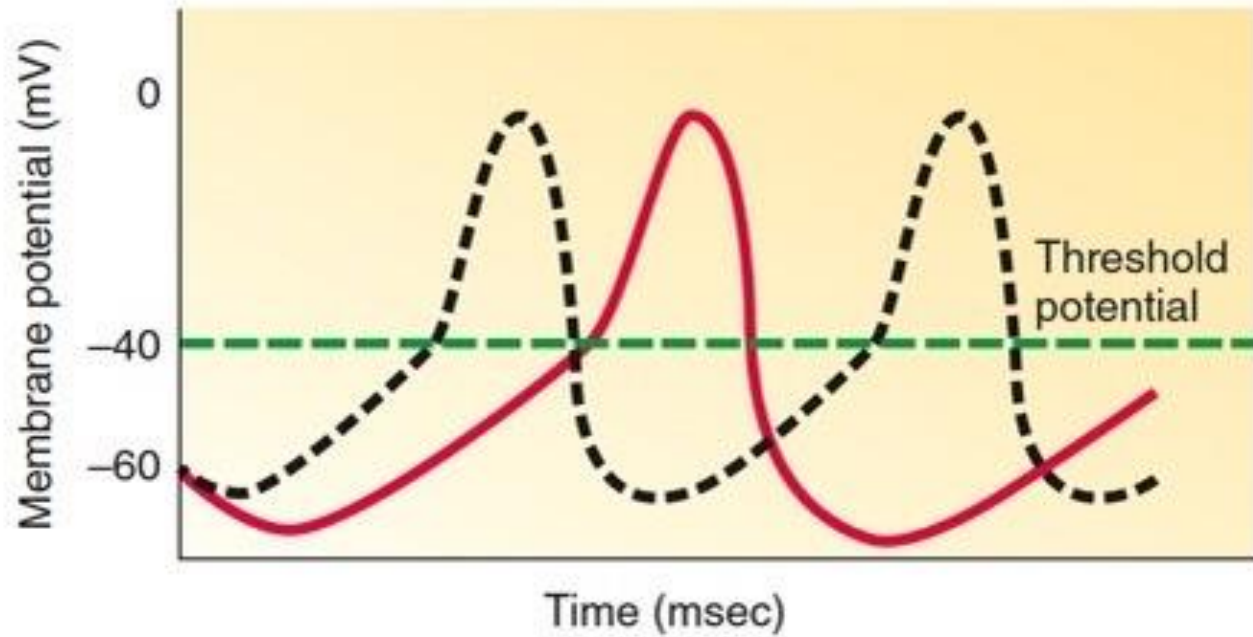
- Sympathetic stimulation can enhance autorhythmicity in the SA node. It can enhance the depolarisation by increasing the influx of cation such as Na and Ca, increasing the slope of depolarisation in the cells, so you can increase the rate of discharge or the impulse generation in the SA node which results in tachyarrhythmias. It also may cause other effects in other structures in the conduction system, such as in the AV node causing decrease in the AV delay, or within the atria and ventricle increasing the strength and the speed of contractions.

Sympathetic stimulation

- The main effect on SA node is to speed up depolarization so that threshold is reached more rapidly.
- In pacemaker cells, the rate of depolarization increases as a result of greater inward movement of Na and Ca.
- It decreases AV delay.
- In atria and ventricles, it increases strength and speed of contraction.

Parasympathetic stimulation

- It decreases HR.
- ACh ([acetylcholine](#)) slows heart rate primarily by increasing K permeability of the pacemaker cells by binding with muscarinic cholinergic receptors that are coupled directly to ACh-regulated K channels by a G protein.



- The dashed curve represents the normal autorhythmicity in the SA node.
- The red curve represents the autorhythmicity in the SA node during parasympathetic stimulation.

- Parasympathetic stimulations have the opposite effect, parasympathetic stimulation delays the SA node autorhythmicity by increasing the conduction of K leading to hyperpolarisation. This hyperpolarisation make it difficult for the SA node to fire, and therefore decrease the rate of discharge causing bradyarrhythmias.

Sinus bradycardia

- Slow heart rate: < 60 beats per minute.
- ECG: normal except heart rate.
- Bradycardia can be physiological or pathological.
- Causes: athletes, elderly, sleep, hypothermia, hypothyroidism, carotid sinus syndrome (when you touch your neck on the carotid sinus area will induce bradycardia, it can be used in different arrhythmias to help in the treatment of that arrhythmia).
- **Carotid Sinus Syndrome (CSS)**, also known as **Carotid Sinus Hypersensitivity**, is a condition characterized by episodes of bradycardia (slow heart rate), hypotension (low blood pressure), and syncope (fainting), often triggered by external pressure on the carotid sinus. The carotid sinus is a region located at the bifurcation of the common carotid artery, which contains baroreceptors that help regulate blood pressure.



Remember that the normal HR (heart rate) is 60-100 bpm. If the HR is <60 bpm this indicates bradycardia .
The ECG is normal except for the HR

Sinus arrhythmia

- Sinus arrhythmias can be either tachyarrhythmias, bradyarrhythmias, SA node dysfunction or SA node block.
- Sinus arrhythmia can result from any condition that alters the sympathetic and parasympathetic impulses to SA node.
- The respiratory type: during inspiration HR increases and decreases during expiration.
- Heart rate variability (HRV)

The time interval between two consecutive RS shouldn't be exactly the same thing in all beats, there should be some kind of variability because this heart rate variability reflects the balance in the ANS.

SA Nodes are under regulation between sympathetic and parasympathetic impulses; that healthy balance reflects some fluctuations in the heart rate; those fluctuations are called HRV, so if HRV is absent, that is a reflection of ANS dysfunction.

Sick sinus syndrome or SA dysfunction

- is a disorder of the SA node caused by impaired pacemaker function and impulse transmission producing a constellation of abnormal rhythms.
- These include atrial bradyarrhythmias, atrial tachyarrhythmias and, sometimes, bradycardia alternating with tachycardia often referred to as “tachy-brady syndrome.”

SA block

- the impulse from the sinus node is blocked before it enters the atrial muscle, may lead to atrial asystole.
- ECG: normal QRS, slow HR.



Atrial arrhythmia

Paroxysmal tachycardia

- Some abnormalities in different portions of the heart, including the atria, Purkinje system, or ventricles, can occasionally cause rapid rhythmical discharge of impulses that spread in all directions throughout the heart.
- This phenomenon is believed to be caused most frequently by re-entry.
- Because of the rapid rhythm in the irritable focus, this focus becomes the pacemaker of the heart.

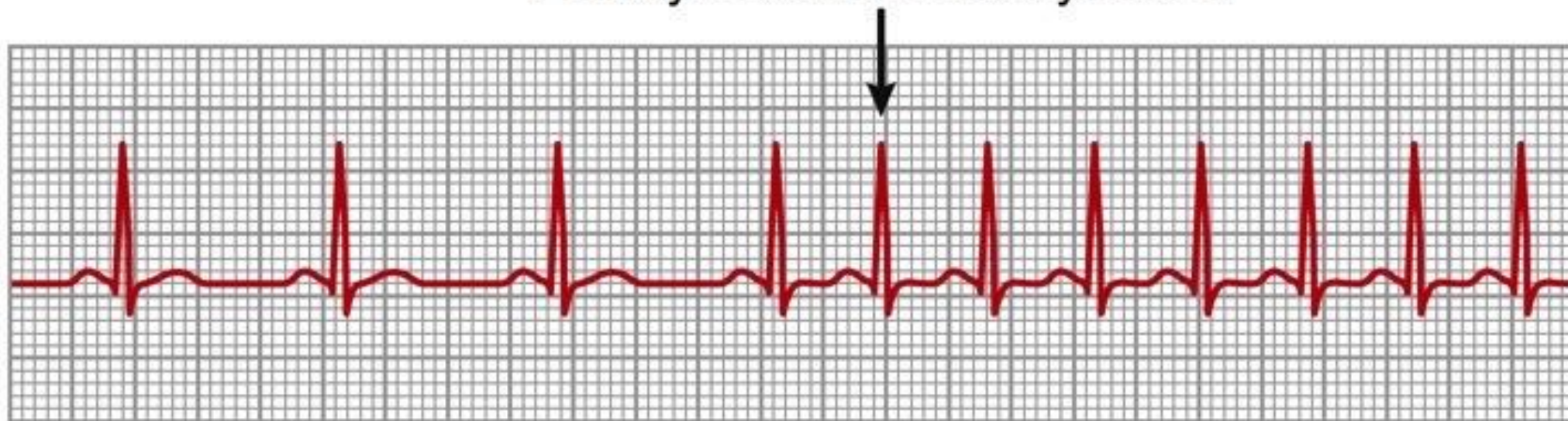
Paroxysmal tachycardia

- The term paroxysmal means that the heart rate becomes rapid in paroxysms, with the paroxysm beginning suddenly and lasting for a few seconds, a few minutes, a few hours, or much longer.
- The paroxysm usually ends as suddenly as it began, with the pacemaker of the heart instantly shifting back to the sinus node.

Paroxysmal tachycardia

- Paroxysmal tachycardia often can be stopped by eliciting a vagal reflex. A type of vagal reflex sometimes elicited for this purpose is to press on the neck in the regions of the carotid sinuses, which may cause enough of a vagal reflex to stop the paroxysm.
- Antiarrhythmic drugs may also be used to slow conduction or prolong the refractory period in cardiac tissues.

Paroxysmal atrial tachycardia



Premature contraction

- A premature contraction is a contraction of the heart before the time that normal contraction would have been expected.
- This condition is also called extrasystole, premature beat, or ectopic beat.
- Most premature contractions result from ectopic foci in the heart, which emit abnormal impulses at odd times during the cardiac rhythm.

Premature atrial contraction (PAC)

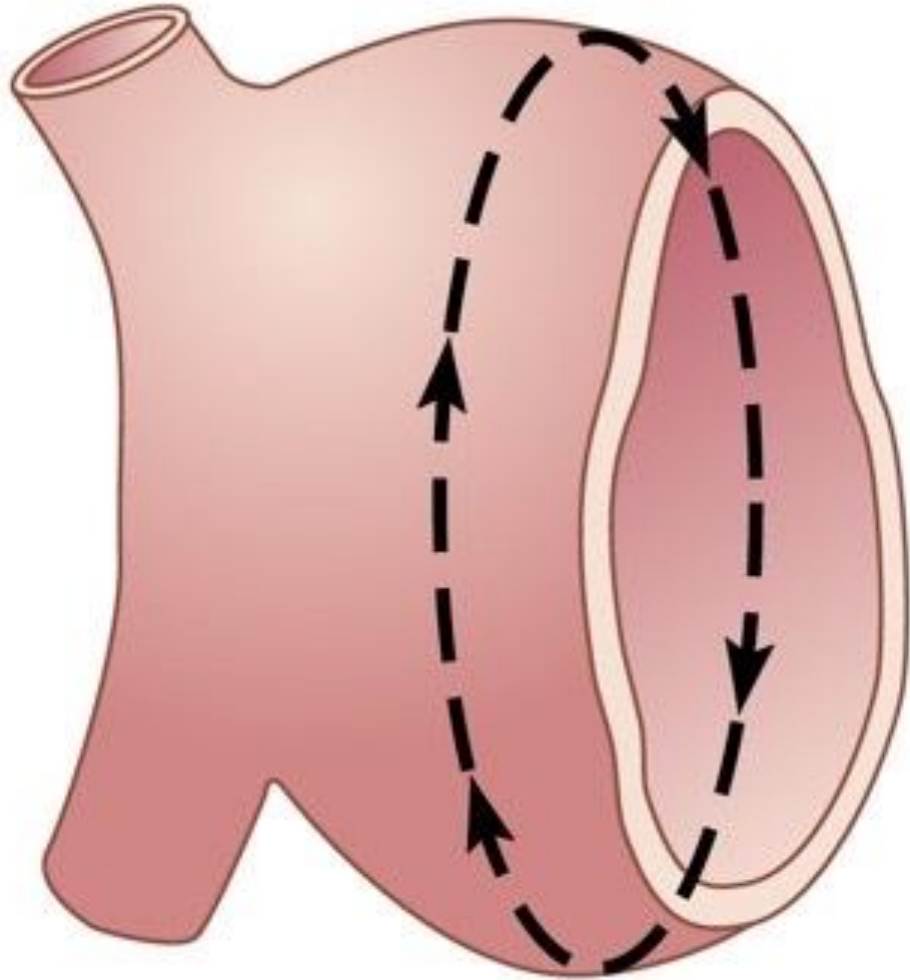
- PACs occur frequently in otherwise healthy people.
- They often occur in athletes, or in mild toxic conditions resulting from such factors as smoking, lack of sleep, ingestion of too much coffee, alcoholism, and use of various drugs.

Pulse deficit

- When the heart contracts ahead of schedule, the ventricles will not have filled with blood normally, and the stroke volume output during that contraction is depressed or is almost absent.
- Therefore, the pulse wave passing to the peripheral arteries after a premature contraction may be so weak that it cannot be felt in the radial artery.
- Thus, a deficit in the number of radial pulses occurs when compared with the actual number of contractions of the heart.

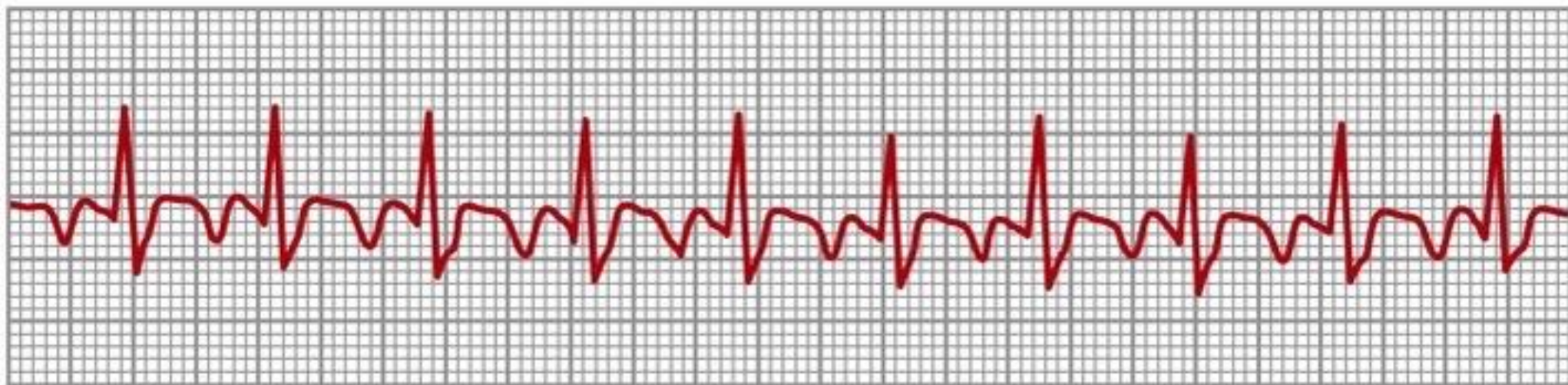
Atrial flutter

- **Very high** heart rate 200-350 bpm.
- Caused by reentry in the atria.
- signals reach the AV node too rapidly for all of them to be passed into the ventricles because the refractory periods of the AVnode and AV bundle are too long to pass more than a fraction of the atrial signals.
- Therefore, there are usually two to three beats of the atria for every single beat of the ventricles.



Atrial flutter

This graph illustrates the re-entry mechanism within the atria, which leads to atrial flutter. In this condition, electrical impulses discharge frequently and rapidly, causing the atria to contract at an abnormally high rate.



Toth appearance

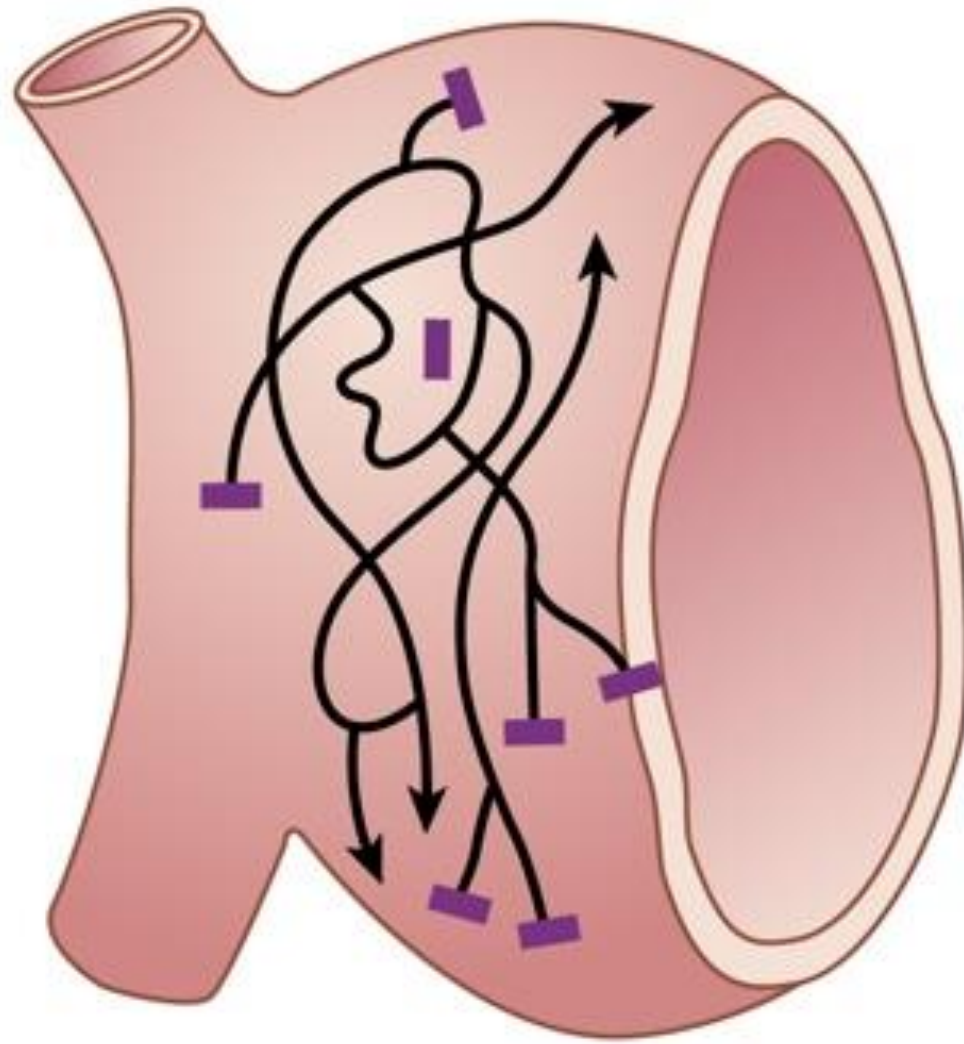
Atrial flutter

- P waves are strong and very frequent because of the contraction of semicoordinated masses of muscle.
- However, note that a QRS-T complex follows an atrial P wave only once for every two beats of the atria, giving a 2:1 rhythm and sometimes 3:1 .

Atrial fibrillation (Afib)

Atrial fibrillation (Afib) is not life-threatening condition, but it decrease the efficiency of heart's blood pumping by about 20%.

- Usually in dilated atrium.
- Not life threatening. However, due to the reduced atrial contractile function, blood can stagnate, allowing blood clots to form in the atrial appendage.
- These blood clots can dislodge and travel to the brain, causing stroke, or to other parts of the body.
- Therefore, patients with atrial fibrillation are often placed on anticoagulants to reduce the risk of embolism.



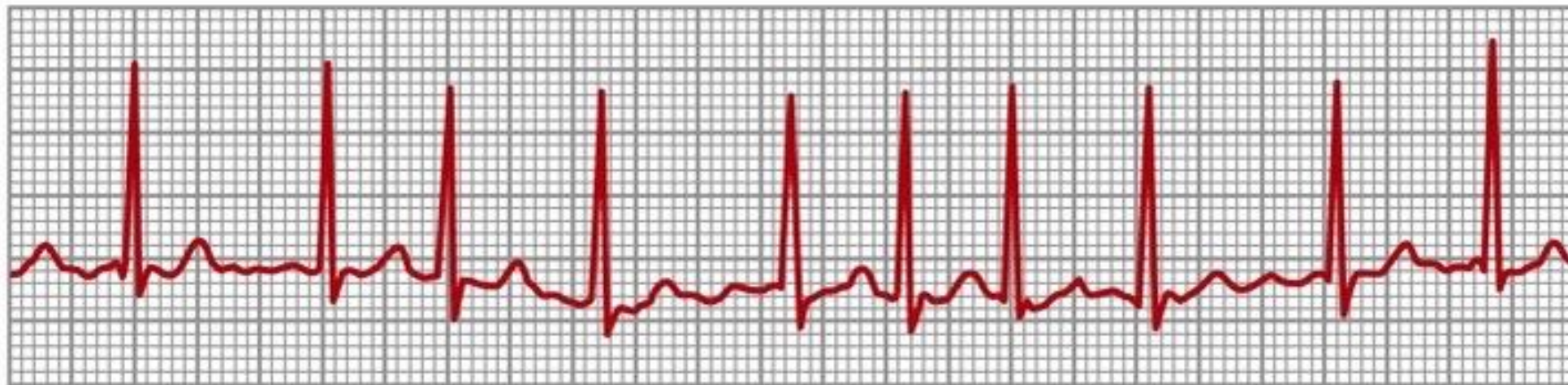
Atrial fibrillation

Atrial fibrillation (Afib)

- On ECG:
- Heart rate increased.
- Numerous small depolarization waves spread in all directions through the atria during atrial fibrillation causing asynchronous contractions. Because the waves are weak, and many of them are of opposite polarity at any given time, they usually almost completely electrically neutralize one another.
- Therefore, in the ECG, one can see either no P waves from the atria or only a fine, high-frequency, Very low voltage.
- Due to the disorganized electrical discharges from multiple foci, the signals cancel each other out, resulting in the absence or minimal appearance of the P wave on an ECG.

Atrial fibrillation (Afib)

- On ECG:
- QRS-T complexes are normal unless there is some pathology of the ventricles, but their timing is irregular.
- Treatment:
- Synchronized cardioversion: single electric shock is synchronized to fire only during the QRS complex when the ventricles are refractory to stimulation. A normal rhythm often follows if the heart is capable of generating a normal rhythm.



Thank you

أَلْعُمِرِ فِي الدُّنْيَا تُجِدُّ وَتَعْمُرُ

وَأَنْتَ غَدًا فِيهَا تَمُوتُ وَتُقْبَرُ

تُلَقِّحُ آمَالاً وَتَرْجُو نَتَاجَهَا

وَعُمُرِكَ مِمَّا قَدْ تُرَجِّيهِ أَقْصَرُ

Additional sources

[Arrhythmia, Physiology, Guyton and Hall](#)

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→V2			
V2→V3			



امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!