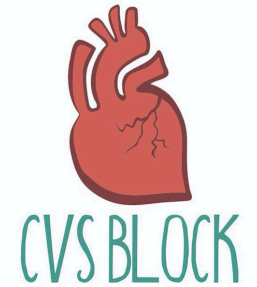




Cardiac Science: Cardiac Arrhythmias



Red: very important.

Green: Doctor's notes.

Pink: formulas.

Yellow: numbers.

Gray: notes and explanation.

Physiology Team 436 – Cardiovascular Block Lecture 6

Lecture: If work is intended for initial studying.

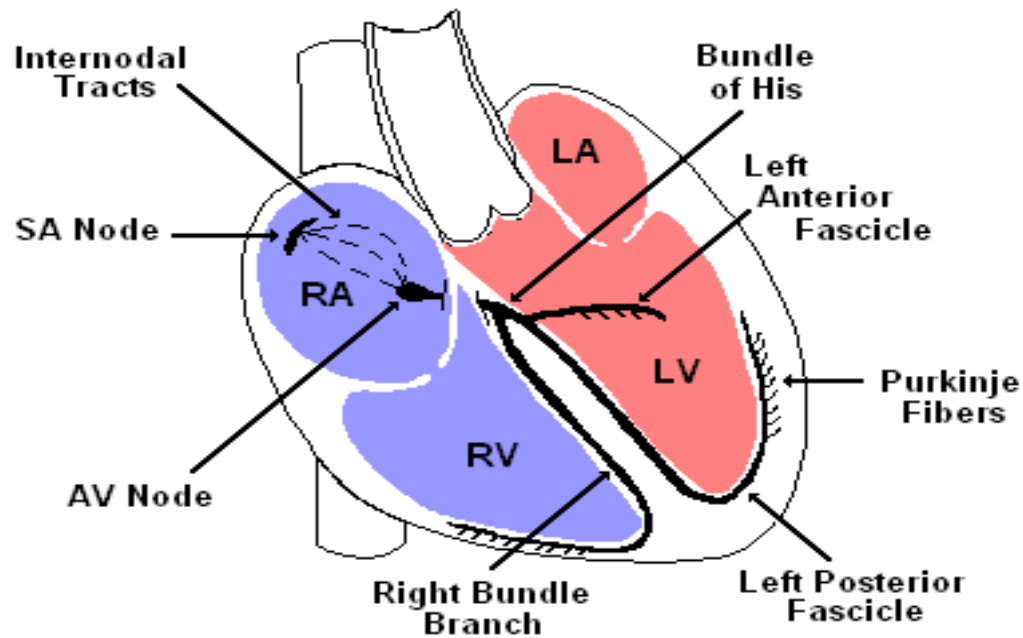
Review: If work is intended for revision.

Objectives

Study Smart: focus on mutual topics.

- ▶ From the students' guide:
- ▶ Identify normal rhythm
- ▶ Identify the main pathophysiological causes of cardiac arrhythmias (Arrhythmogenesis) } Identify sinus arrhythmias
- ▶ Define different ectopic foci of excitation and the mechanism of re-entry phenomena } Identify conduction block.
- ▶ Identify electrolyte abnormalities (K^+ , Ca^{++})
- ▶ Enumerate the common arrhythmias and describe the basic ECG changes

The Normal Conduction System



SA node:

cardiac pacemaker

Paces at rate of 60-100 bpm (average of 70 bpm)

AV node: 45-60 bpm

Purkinje: 15-45 bpm

ONLY IN MALES' SLIDES

ONLY IN FEMALES' SLIDES

Interpretation

- ▶ Develop a systematic approach to reading EKGs and use it every time.
- ▶ The system we will practice is:
 - ▶ Heart Rate
 - ▶ Rhythm
(including intervals and blocks)
 - ▶ Axis
 - ▶ Hypertrophy
 - ▶ Ischemia

Interpretation: Heart Rate

Rule of 300: Divide 300 by the number of boxes between each QRS = rate

- HR of 60-100 per minute is normal
- HR > 100 = tachycardia
- HR < 60 = bradycardia

BOTH MALES' & FEMALES'
SLIDES

Number of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

Heart rate in picture below: $(300 / 6) = 50$ bpm



Heart rate may vary depending on:

- Age
- Activity
- Time of day

Interpretation-EXTRA NOTES

when you look at ECG it is like when you look at an elephant, there's a lot of things to see.

- if the SA node stops working for some reason the other part of the heart will take over,
- *also you have to know the approach, what's approach? Approach is systematic way of how to read ECG, if you have this approach you'll not miss anything,*

The approach we follow :

- **Rate** (rapid-normal-slow)
- **Rhythm** (coming from SA node or not **"this is very important"**)
- **Axis** (to tell me how is the heart sitting in the chest? *"it is sitting in the left side towards the middle"*, so the force of the activity should go to the left side *"downward and a little bit towards the left"*, otherwise it will be abnormal)
- **Hypertrophy** (from axis you can tell me if there's hypertrophy or not, means there's enlargement of the heart or hypertrophy of the wall)
- **Ischemia** (means one part of the muscles won't get enough blood, what's going to happen? I'll have problems, scarring, damaged part of the muscles, and electrical system around that part won't work anymore, it'll be irritable, this irritation "زي الالتماس الكهربائي بالضبط" will cause what we call arrhythmia either ventricular arrhythmia or atrial arrhythmia)

Normal Sinus Rhythm; NSR

- ▶ Regular
- ▶ Each P followed by QRS with resulting
- ▶ PR interval is constant and within normal range
- ▶ PP interval is constant.

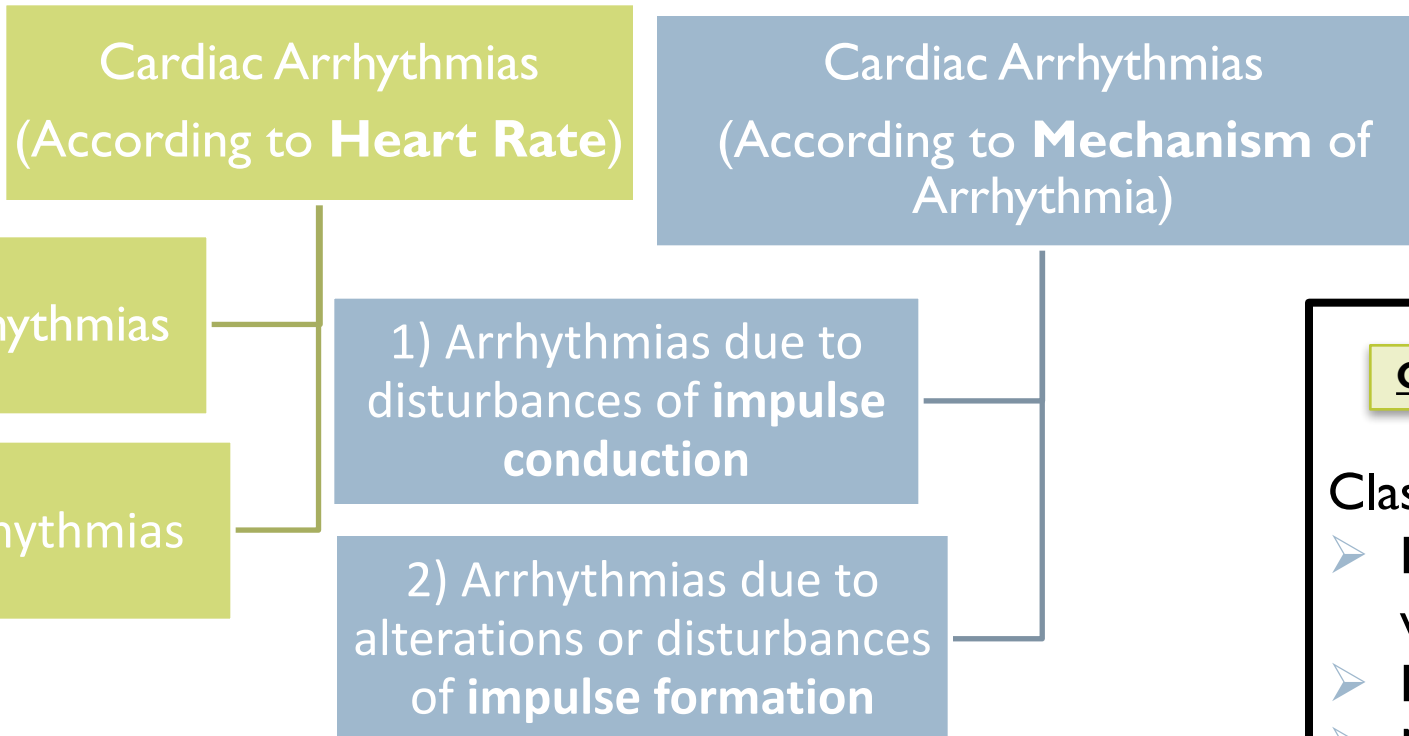


	Bradyarrhythmia	Tachyarrhythmia
SA node	Sinus Bradycardia	Sinus tachycardia
	Sick Sinus Syndrome	
Atria		Atrial Premature Beats
		Atrial Flutter
		Atrial Fibrillation
		Paroxysmal SVT
		Multifocal Atrial Tachycardia
AV node	Conduction Blocks (1,2 and 3)	
	Jxal escape rhythm	
Ventricles	Ventricular escape rhythm	Ventricular premature Beats
		VT
		Torsades de pointes
		Ventricular Fibrillation

ONLY IN FEMALES' SLIDES

Classification of Cardiac Arrhythmias

ONLY IN FEMALES' SLIDES



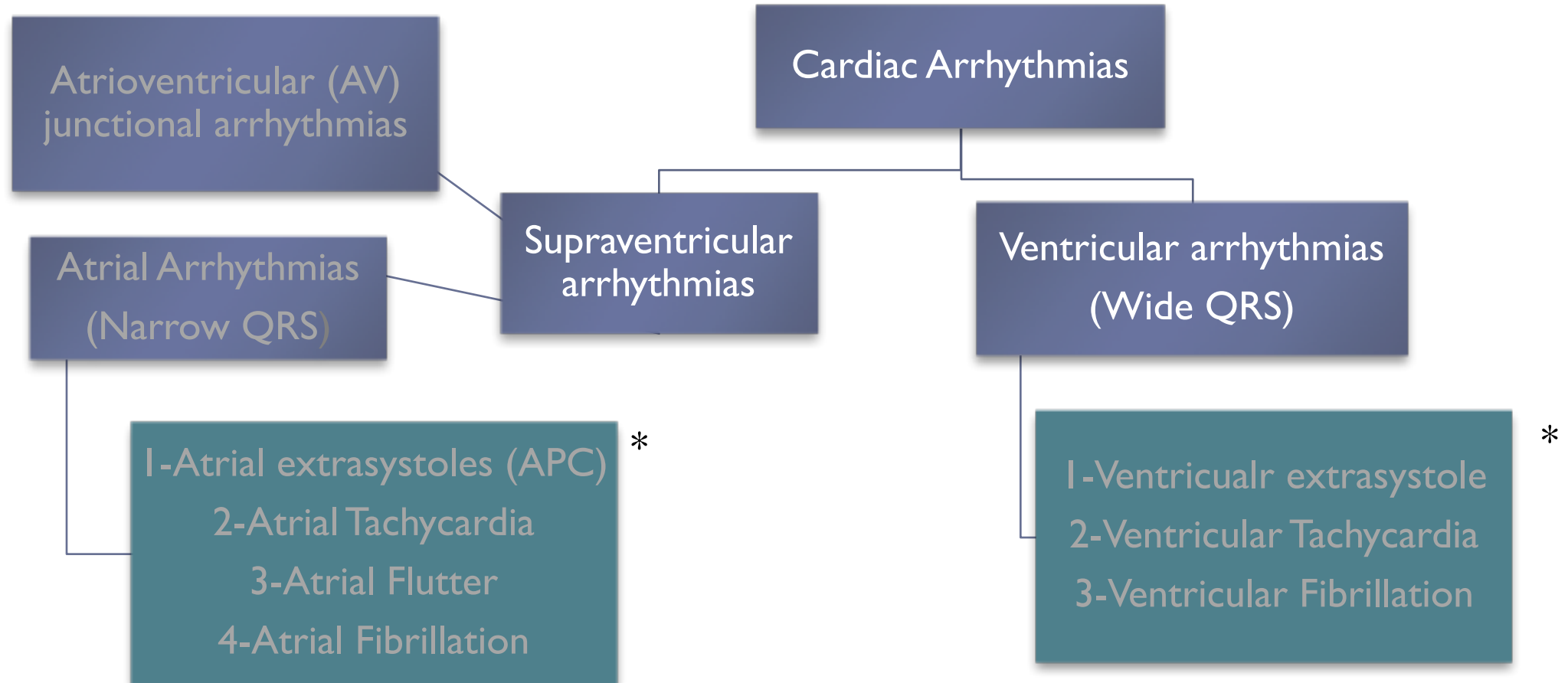
Blocks at different points in the spread of impulse through the heart

ONLY IN MALES' SLIDES

- Classifications of Cardiac Arrhythmias:
- Rate above or below normal (tachy vs. bradycardia)
 - Regular or irregular rhythm
 - Narrow or broad QRS complex
 - Relation to P wave
 - Supraventricular Vs. ventricular

Anatomical Classification of Cardiac Arrhythmias (origin of the arrhythmia)

ONLY IN FEMALES' SLIDES



Extrasystole = premature beats = premature complexes = premature contractions

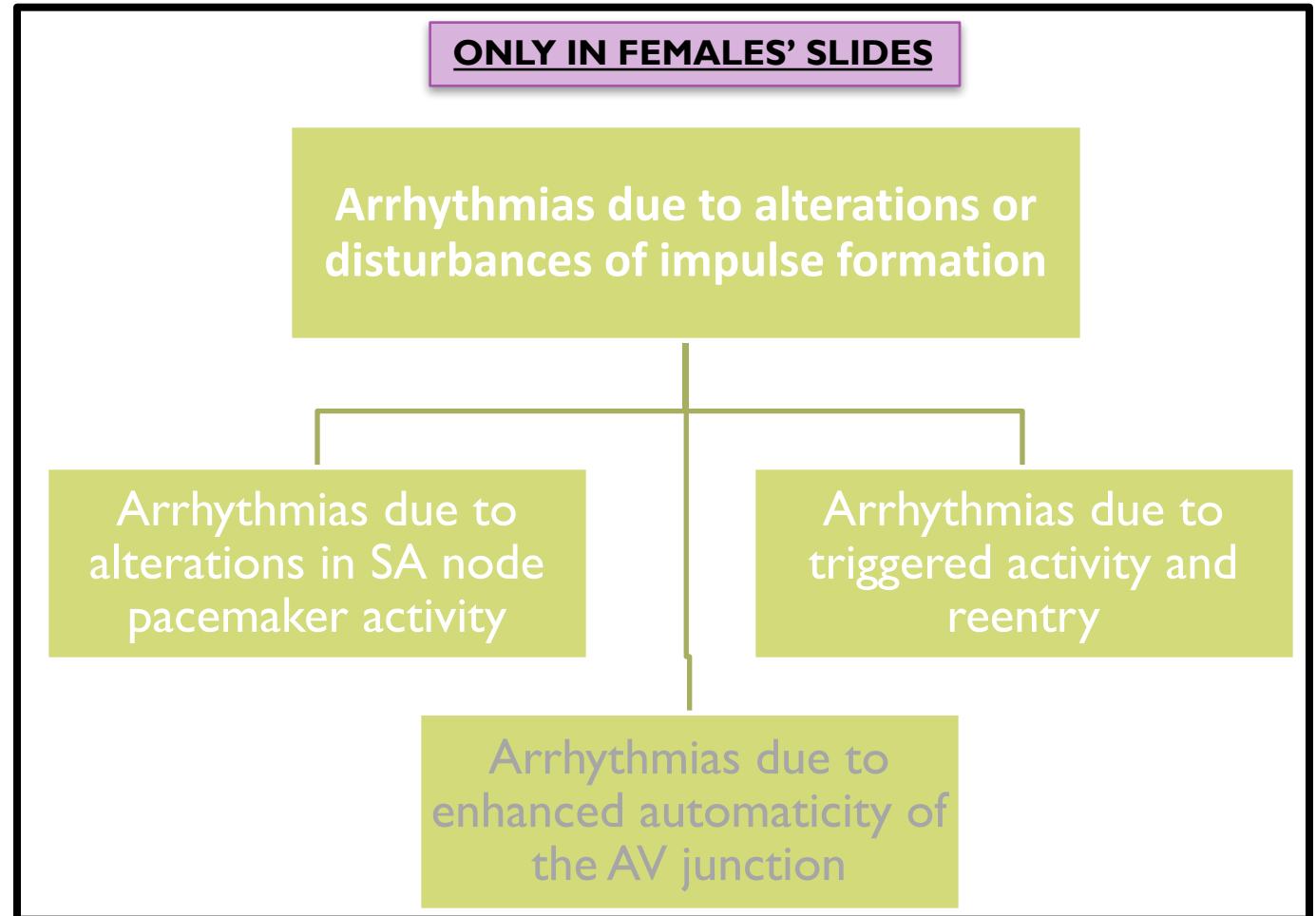
Arrhythmias Due to Alterations or Disturbances of Impulse Formation

▶ Mechanisms:

1. Enhanced automaticity.
2. Triggered activity.
3. Reentrant circuits (circus movement).

Other mechanism

- Abnormal rhythmicity of the pacemaker
- Blocks at different points in the spread of impulse through the heart



Mechanisms of Arrhythmias Due to Alterations or Disturbances of Impulse Formation EXTRA

I. Enhanced automaticity	2. Triggered activity	3. Re-entry (Lewis circus movement)
<p>Enhanced automaticity is defined as an inappropriate increase in the rate of discharge of a tissue having physiological pacemaker properties (i.e., sinus node, atrioventricular node or Purkinje fibers).</p>	<p>The term triggered activity is used to define the appearance of automaticity as a result of external stimulation, and may arise in tissues that do not demonstrate physiological automaticity.</p>	<p>Re-entry (reentry): occurs when a cardiac impulse re-excites some region of the heart through which the impulse has already passed in that heart beat.</p> <p>Unidirectional or partial conduction block is required to initiate re-entry.</p>

Causes of Cardiac Arrhythmias

1. Abnormal rhythmicity of the pacemaker
 2. Shift of the pacemaker from the sinus node to another place in the heart
 3. Blocks at different points during the spread of the impulse through the heart
 4. Abnormal pathways of impulse transmission through the heart
 5. Spontaneous generation of spurious impulses in almost any part of the heart
- Rate above or below normal
 - Regular or irregular rhythm
 - Narrow or broad QRS complex
 - Relation to P waves

Arrhythmias Due to Alterations in SA Node Pacemaker Activity

1) Sinus Bradycardia

- ▶ Heart rate slower than 60 per minute

1- Physiological Causes:

--Expiration.

2- Pharmacological and pathological Causes:

- Parasympathetic stimulation



2) Sinus Tachycardia

- ▶ Heart rate faster than 100 per minute

Causes:

1- Physiological:

-During exercise, sympathetic stimulation, increased body temperature

-Inspiration

2- Pharmacological and pathological:

-Sympathetic stimulation

- Digoxin drug.



Differential Diagnosis of Tachycardia

Tachycardia	Narrow Complex	Wide Complex
Regular	Sinus Tachy SVT Atrial flutter	ST w/ aberrancy SVT w/ aberrancy VT
Irregular	A-fib A-flutter w/ variable conduction MAT	A-fib w/ aberrancy A-fib w/ WPW VT

Arrhythmias Due to Alterations in SA Node Pacemaker Activity

- ▶ **3) Respiratory sinus arrhythmia**
- ▶ Sinus arrhythmia is an increase in heart rate during inspiration and a decrease during expiration
- ▶ It is a normal finding in children and young adults and tends to diminish or disappear with age.

Causes:

I- Sinus arrhythmia is due to variations in the vagal tone that affects the SA node.



ONLY IN MALES' SLIDES

Sinus Arrhythmia: Result from spillover of signals from the medullary respiratory center into the adjacent vasomotor center during inspiration and expiratory cycles of respiration. The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart

Atrial and Ventricular Extrasystoles

ONLY IN FEMALES' SLIDES

- ▶ Extrasystoles = premature beats = premature contractions = premature complexes
- If the focus is in the ventricles, the premature beat is called **ventricular premature beat**.
- If the focus is in the atrial wall, the premature beat is called **atrial premature beat**.
- Ectopic foci can also cause a premature beat in **AV junction**.
- ▶ **Causes:**
 - Ischemia
 - Irritation of cardiac muscle by calcified foci
 - Caffeine

Abnormal Cardiac Rhythms that Result from Impulse Conduction Block

Sinoatrial Block

The impulse from the S-A node is blocked before it enters the atrial muscle → Cessation (no) of P waves

A-V Block

When impulse from the S-A node is blocked

Causes:

Ischemia of the A-V node

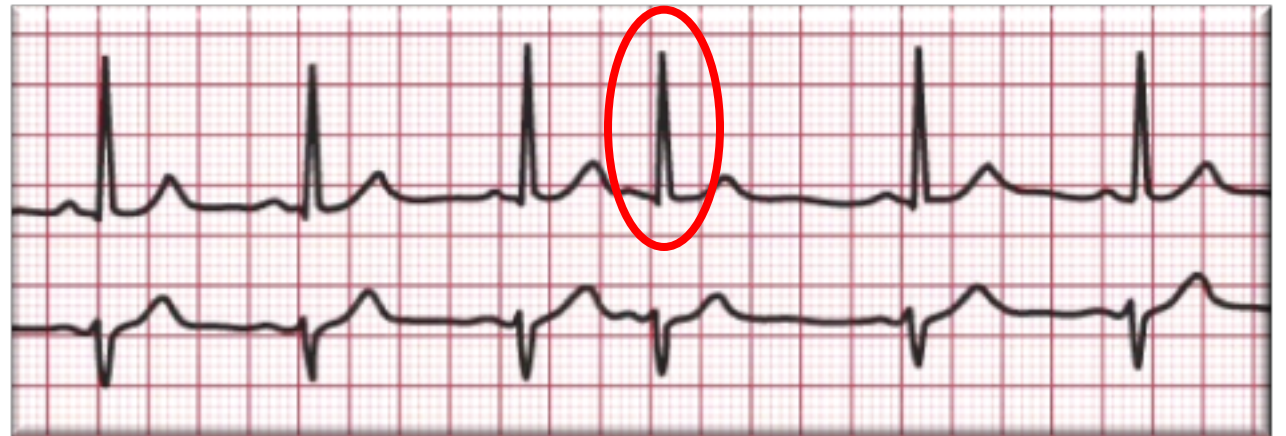
Compression of the A-V node by scar formation

Inflammation of the A-V node

Strong vagal stimulation

Atrial Extrasystoles

- Short P-R interval depending on how far the ectopic foci from the AV node.
- Pulse deficit if there is no time for the ventricles to fill with blood.
- The time between the premature contraction and the succeeding beat is increased (Compensatory pause).

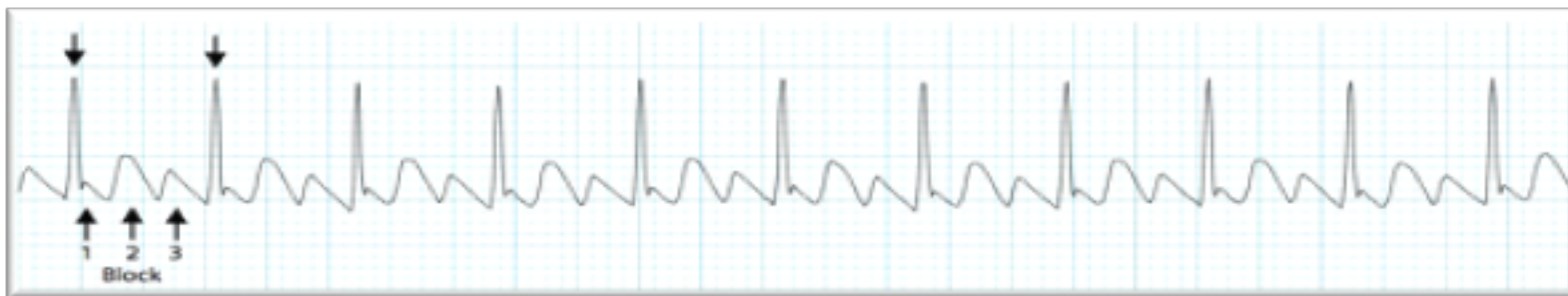
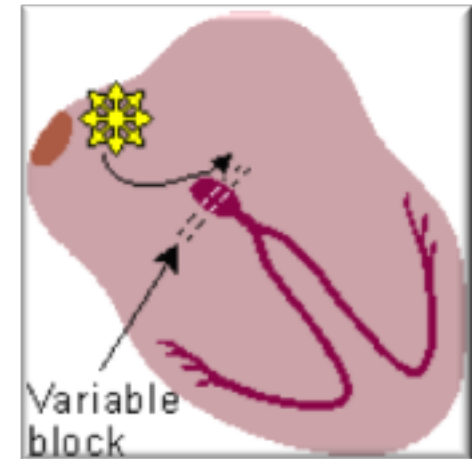


Atrial Flutter

- ▶ Atrial flutter occurs when the atria are stimulated to beat at a rate **250** beats per minute.
- ▶ Atrial flutter results in poor atrial pumping since some parts of the atria are relaxing while other parts are contracting.

ONLY IN MALES' SLIDES

- ▶ A single large wave travels around and around in the atria
- ▶ The refractory period of the AV node causes **2-3** beats of atria for one single ventricular beat **2:1** or **2:3** rhythm.



Atrial Flutter				
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12
V: <300 bpm				

Atrial Fibrillation (AF)

- Same mechanism as ventricular fibrillation.
- It can occur only in atria without affecting the ventricles.
- It occurs more frequently in patients with enlarged heart.
- The atria do not pump if they are fibrillating.
- The efficiency of ventricular filling is decreased 20 to 30%
- No P wave, or high frequency of low voltage P wave
- A person can live for years with atrial fibrillation
- Treatment: DC shock



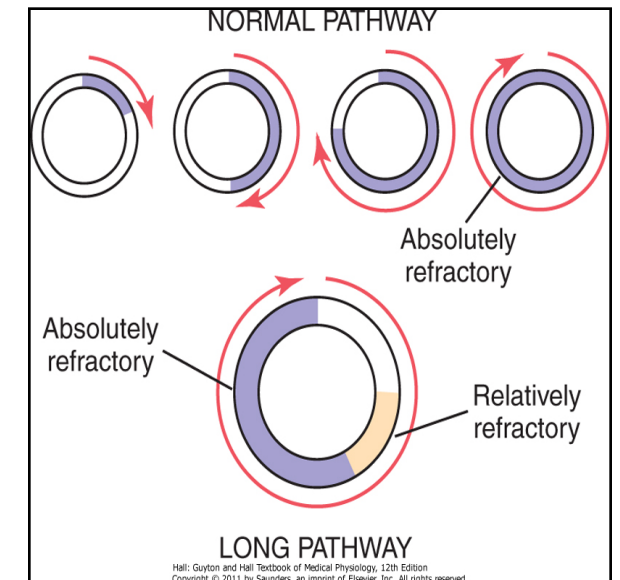
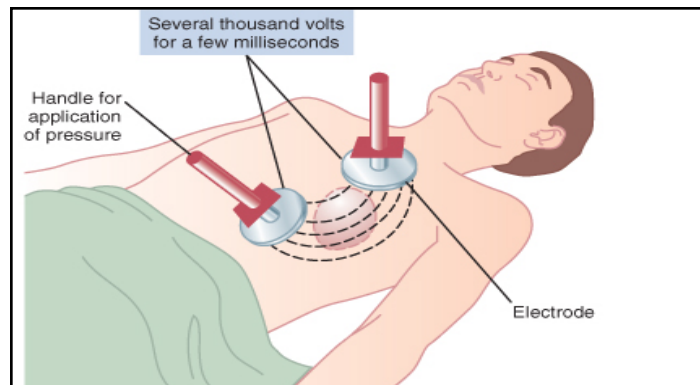
Ventricular extrasystoles

- ▶ Prolong QRS complex because the impulses are carried out with myocardial fibers with slower conduction rate than Purkinje fibers
- ▶ Increase QRS complexes voltage because QRS wave from one ventricle can not neutralize the one from the other ventricle
- ▶ After PVCs, the T wave has an electrical potential of opposite polarity of that of the QRS because of the slow conduction in the myocardial fibers, the fibers that depolarizes first will repolarize first
- ▶ Causes: drugs, caffeine, smoking, lack of sleep, emotional irritations



Ventricular Fibrillation

- **The most serious of all arrhythmias**
- **Cause:** impulses stimulate one part of the ventricles, then another, then itself.
- Many part contracts at the same time while other parts relax. (Circus movement)
- **Causes:** sudden electrical shock, ischemia
 - ▶ Tachycardia
 - ▶ Irregular rhythm
 - ▶ Broad QRS complex
 - ▶ No P wave
- ▶ Treatment : DC shock



Abnormal Cardiac Rhythms that Result from Impulse Conduction Block

- **Sinoatrial Block**

- Blockade of the S-A node impulse before entering atrial muscle.
- Cessation of P wave (difficulty of seeing p wave)



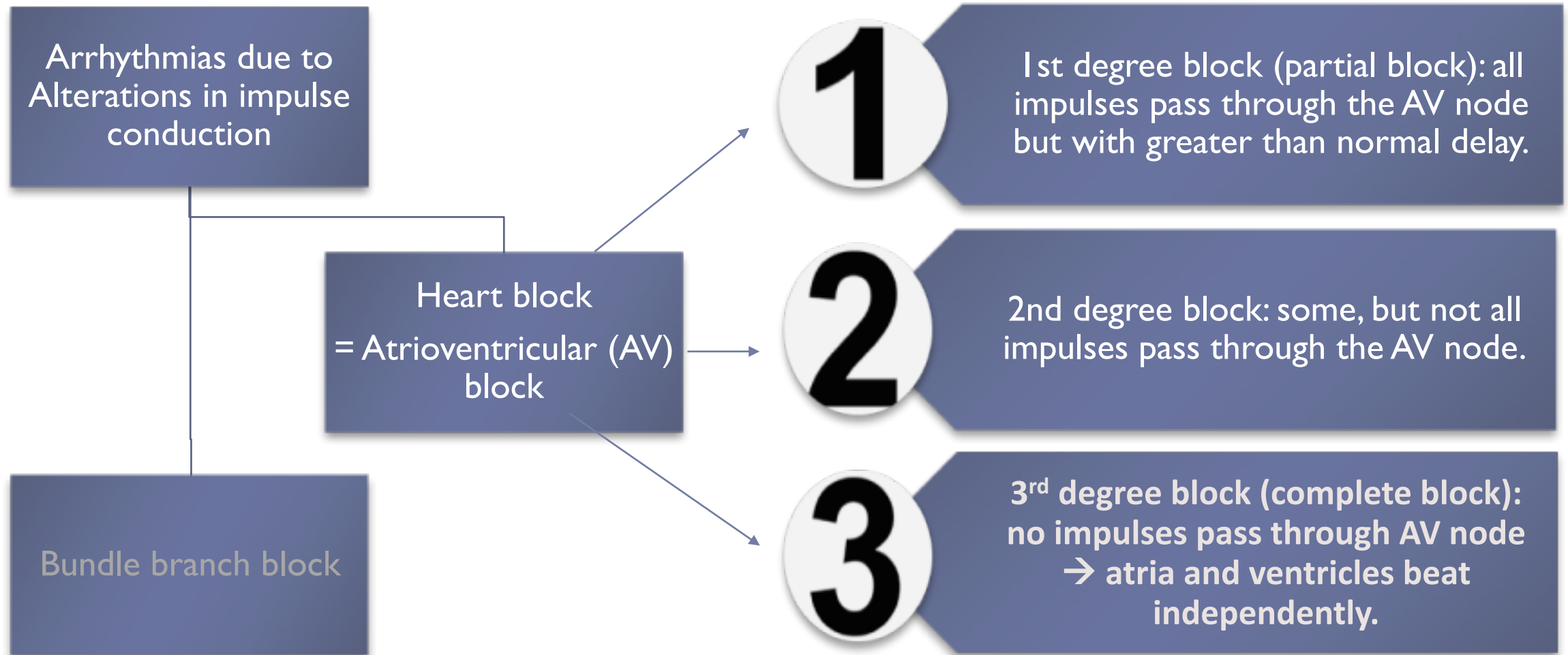
- **A-V Block**

When impulse from the S-A node is blocked

- First degree block
- Second degree block
- Third degree block

- Causes of SA and AV Block:
 - Ischemia of the A-V node
 - Compression of the A-V node by scar formation
 - Inflammation of the A-V node
 - Strong vagal stimulation

Other Arrhythmias Due to Disturbances of Impulse Conduction (impulse conduction block)



Heart Block

Heart Block

1st°

- Constant PR prolongation without drop beat.

2nd°

- **Mobitz1:** Progressive PR prolongation + drop beat.
- **Mobitz2:** Constant PR prolongation + drop beat.

3rd°

- Complete dissociation between P and QRS.

Heart Block = Atrioventricular (AV) Block

- ▶ 1st degree block: all impulses pass through the AV node but with greater than normal delay. Every P wave is followed by R wave.

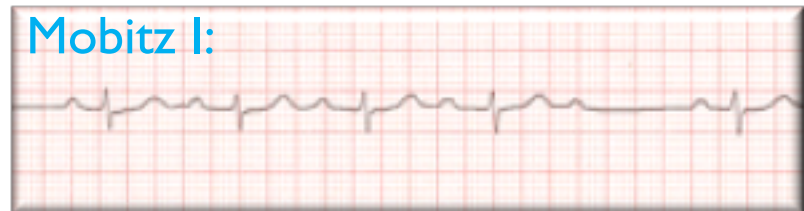
P-R interval is prolonged = 0.2 second



- ▶ 3rd degree AV block: (complete heart block).
- Complete dissociation of P wave and QRS waves
- Ventricle escape from the influence of S-A node
- ▶ Atrial rate is 100 beats/min
- ▶ Ventricular rate is 57 beats/min. (40 in females)
- ▶ Stroke-Adams Syndrome::AV block comes and goes

- ▶ 2nd degree AV block:

- P-R interval > 0.25 second
- Only few impulses pass to the ventricles
→ atria beat faster than ventricles
→ “dropped beat” of the ventricles
- 1. **Mobitz I:** Progressive increase in P-R interval until one QRS is dropped.
- 2. **Mobitz II:** Constant PR prolongation + dropped beat



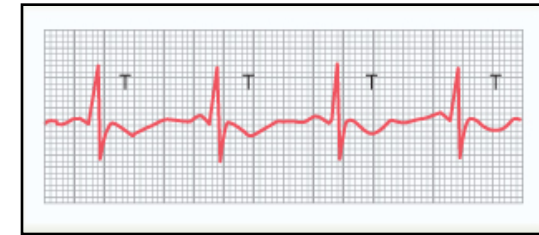
Diseases in ECG:

1. Ischemia: common use of ECG is acute chest pain.

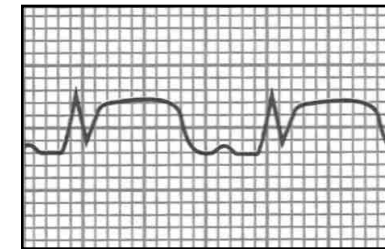
Cause: restriction of blood flow to the myocardium due to

- reversible: angina pectoris
- Irreversible: myocardial infarction.
- Ischemia → injury → infarction

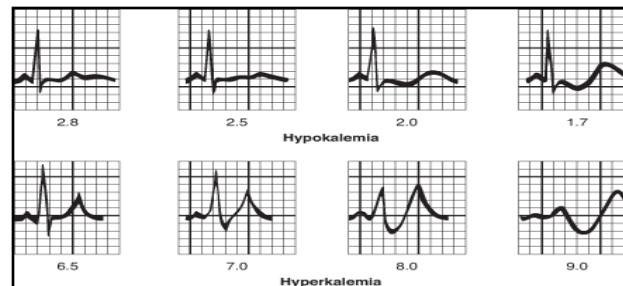
2. Reversible Ischemia: Inverted T wave - ST segment depression.



3. Myocardial infarction: ST segment elevation – Deep Q wave. Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue.



4. Hypokalemia: Flat T wave



5. Hyperkalemia: Tall peak T wave.

Quiz

- ▶ <https://www.onlineexambuilder.com/arrhythmias/exam-139189>

[Link to Editing File](#)

(Please be sure to check this file frequently for any edits or updates on all of our lectures.)

References:

- Girls' and boys' slides.
- Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.)

Thank you!

اعمل لترسم بسمة، اعمل لتمسح دموعه، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

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