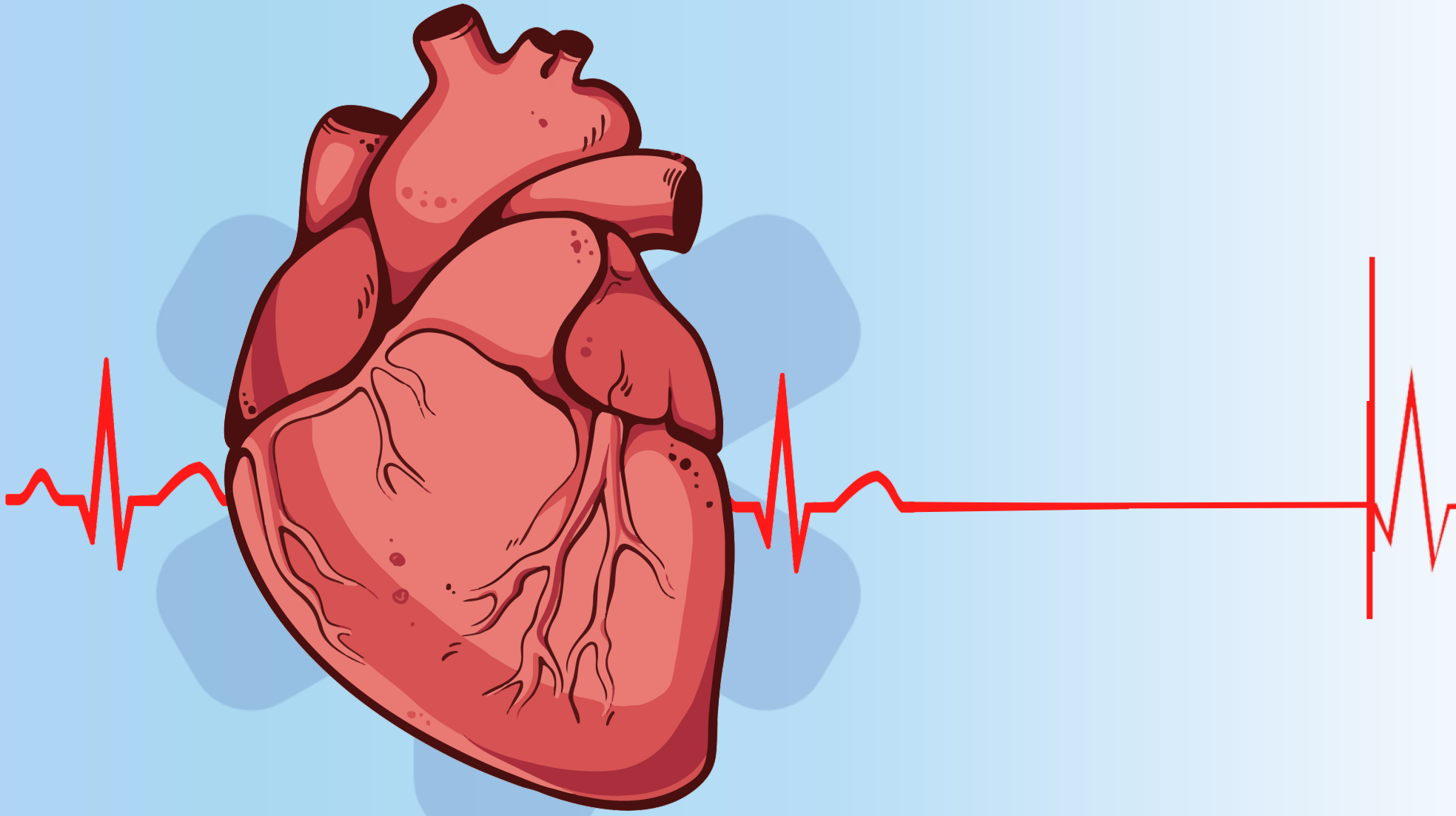

Arrhythmia

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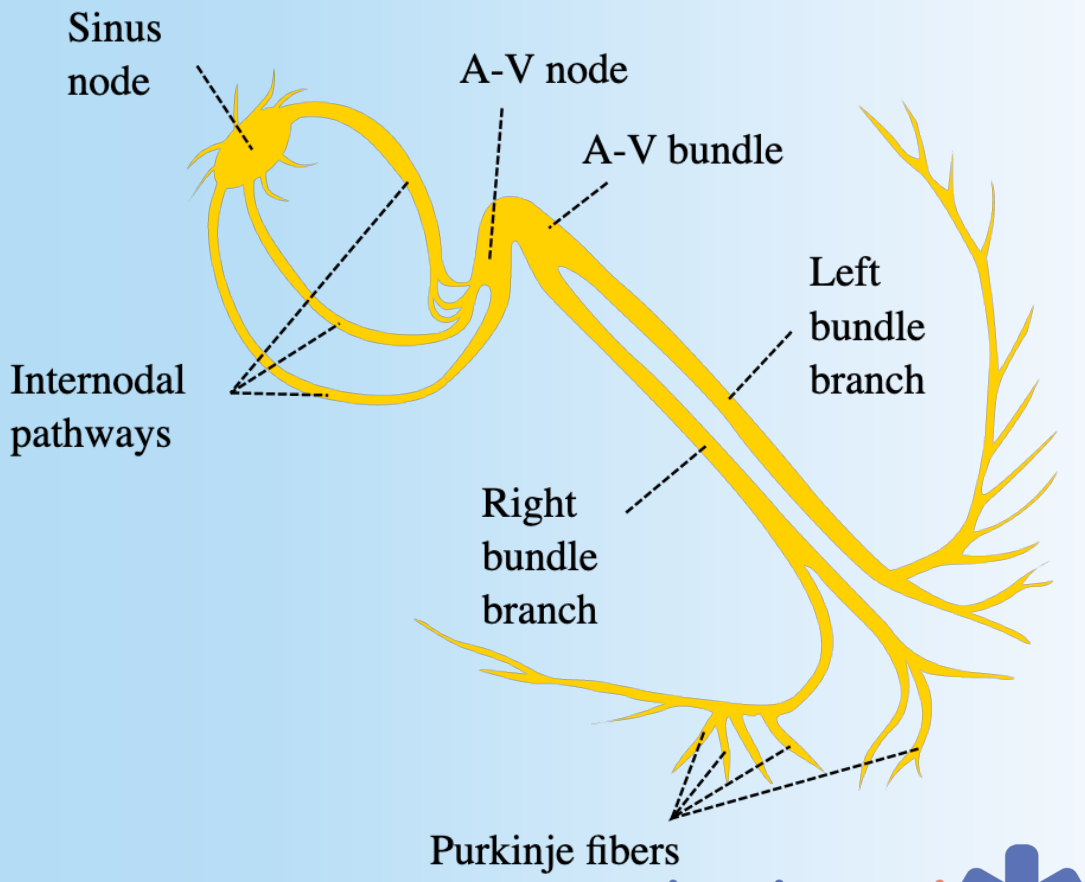
Cardiac conduction system

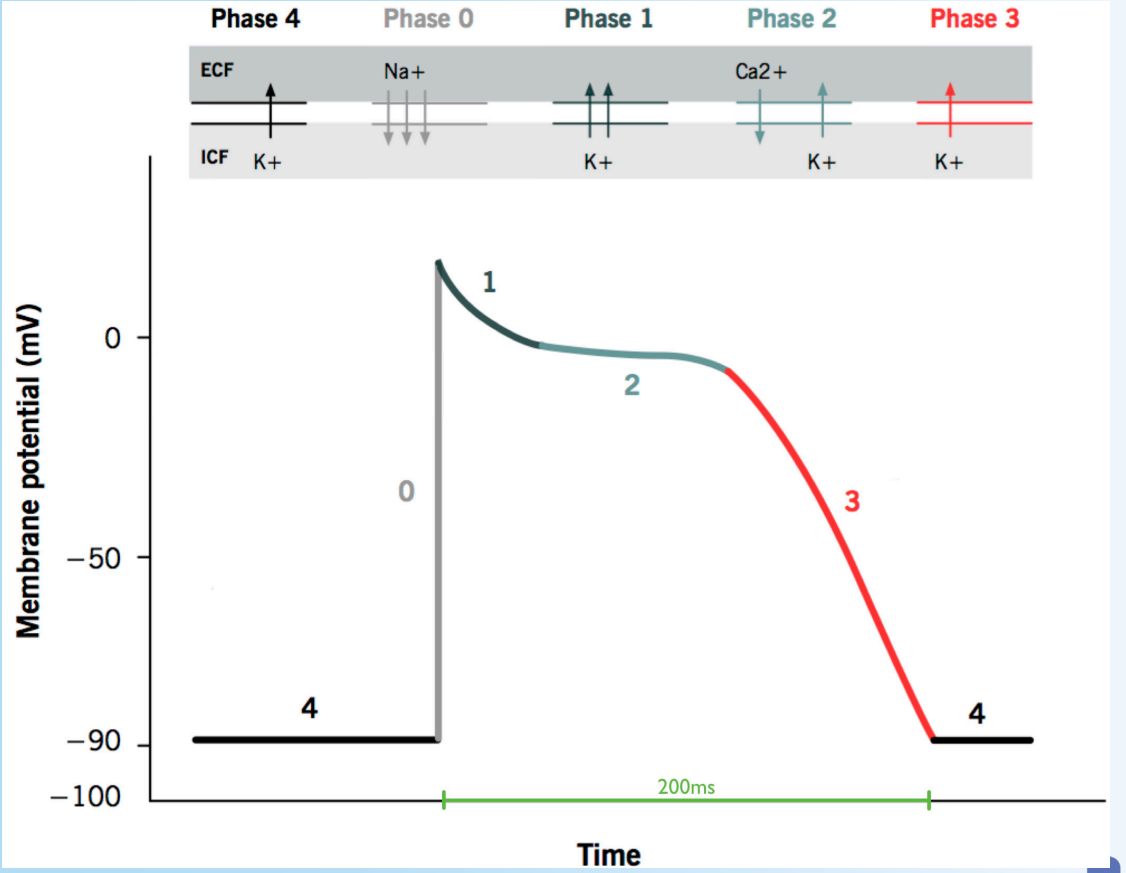
Spontaneous depolarization rate

- **Sinus node (SAN)** — 60-100 BPM
  - Right atrium
- **Atrioventricular node (AVN)** — 40-60 BPM
  - Between atrias & ventricles
  - Delayes signal
- **His bundle** — 40-45 BPM
  - Left & right bundle branch
- **Purkinje fibers** — 20-40 BPM
  - In ventricular sub-endocardium



Cardiac muscle cell action potential

- **Phase 4**
  - Resting phase: Na^+ & K^+ channels closed (-90mV)
  - Pos.current to threshold (-70mV) → phase 0
- **Phase 0 - *depolarization***
  - Fast Na^+ channels open and close → positive surge (+20mV)
- **Phase 1**
  - Quick repolarization by brief opening of K^+ channels & K^+ efflux
- **Phase 2 – *plateau phase***
  - Balance between Ca^{2+} influx & K^+ efflux (0mV)
  - Ca channels slowly close → phase 3
- **Phase 3 - *repolarization***
  - Efflux of K^+ & low membrane permeability for other ions → rapid repolarization



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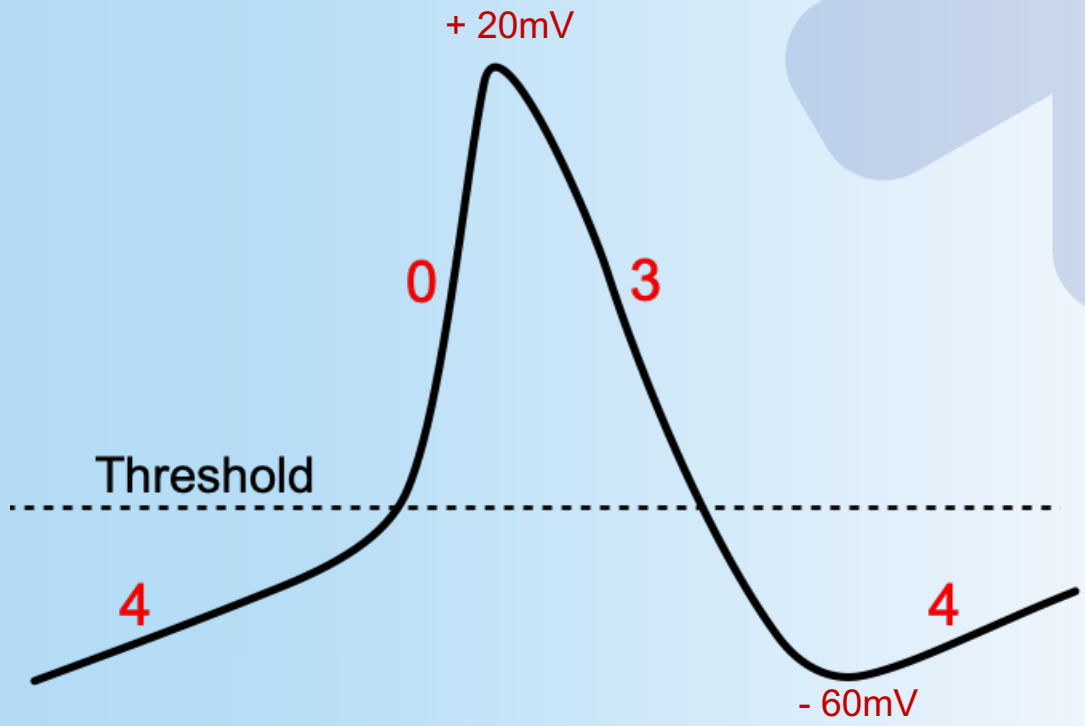
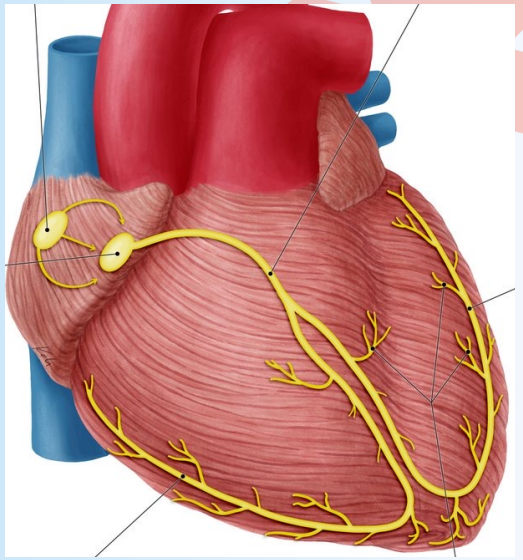
Pacemaker cells action potential

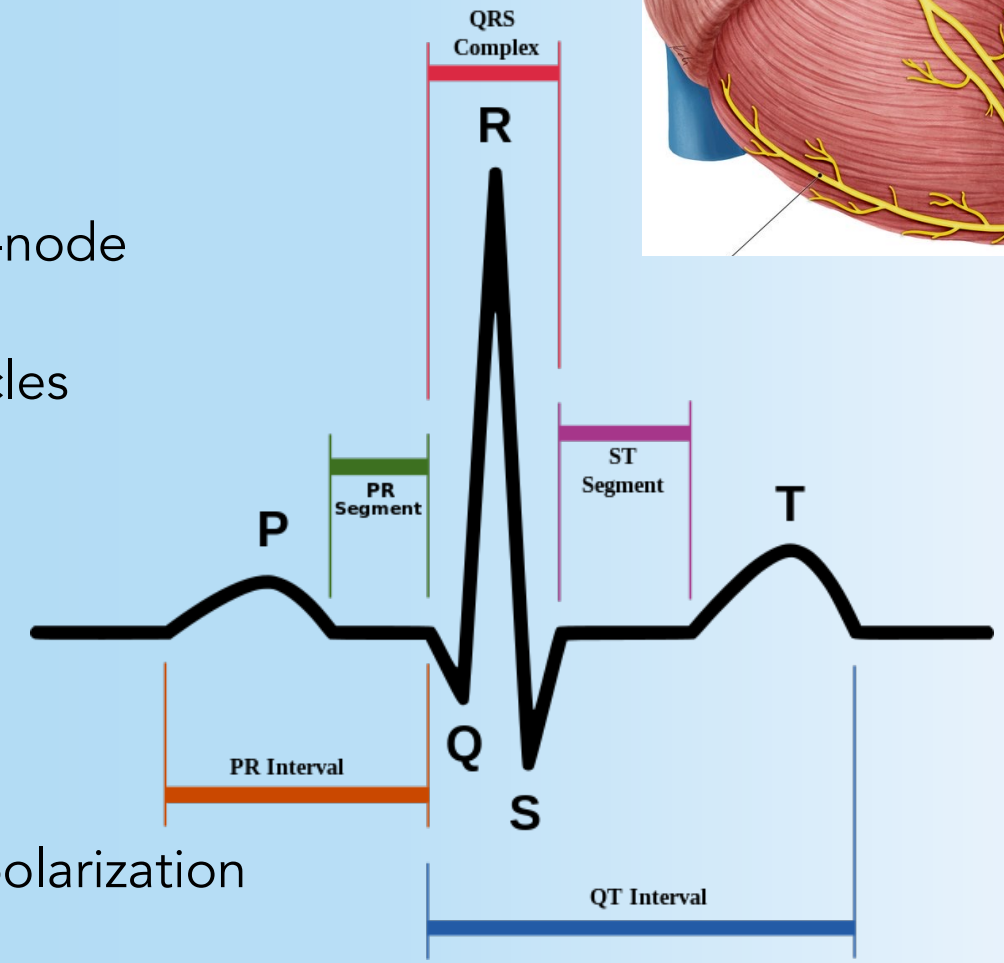
- **Phase 4**
  - Pacemaker/funny current (If)
    → spontaneous gradual depolarization:
    - Slow Na^+ influx
    - Gradually decreasing K^+ efflux
- **Phase 0 - *depolarization***
  - Fast Ca^{2+} channels allow Ca^{2+} influx & upstroke of action potential
  - Upstroke: less rapid & lower in amplitude
- **Phase 3 - *repolarization***
  - Repolarization through gradually decreased Ca^{2+} influx and increased K^+ efflux



ECG-tracing



- P-wave: Depolarization of the atria
- PR-interval: Slowed conduction through AV-node
- QRS-complex: Depolarization of the ventricles
- ST-segment: Ventricles are depolarized.
  Roughly coincides with plateau phase.
- T-wave: ventricular repolarization
- QT-interval: time from ventricular de- to repolarization



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Bradyarrhythmias

Brady = slow

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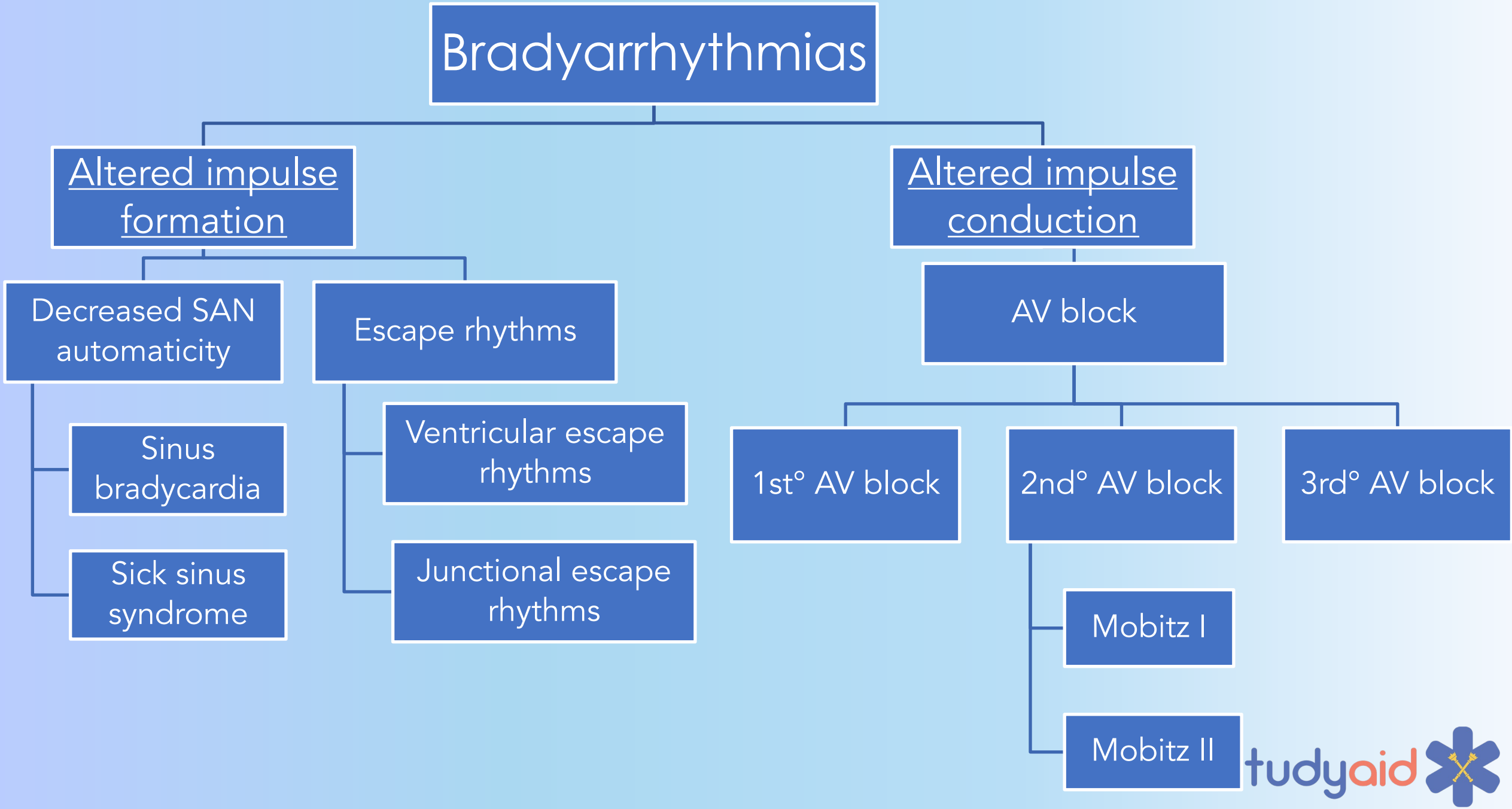
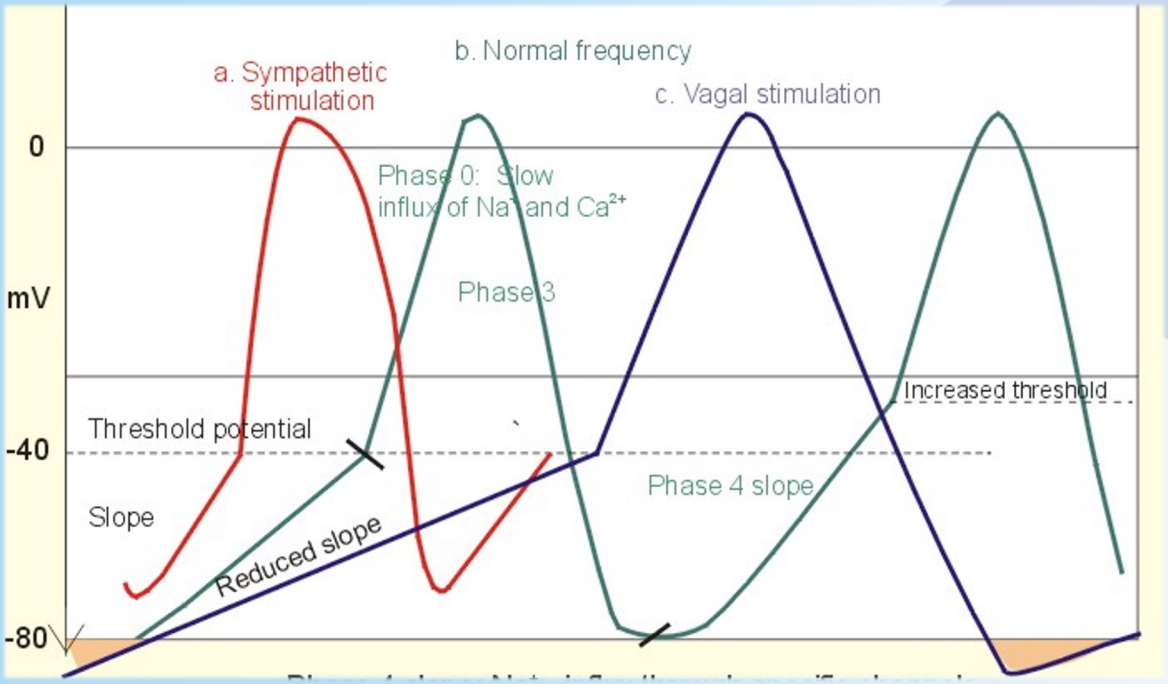
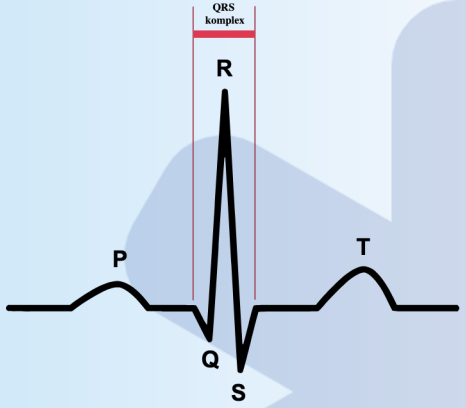

Bradyarrhythmias
Altered impulse formation
Altered impulse conduction
Decreased SAN automaticity
Escape rhythms
AV block
Sinus bradycardia
Sick sinus syndrome
Ventricular escape rhythms
Junctional escape rhythms
1st° AV block
2nd° AV block
3rd° AV block
Mobitz I
Mobitz II
tudyaid

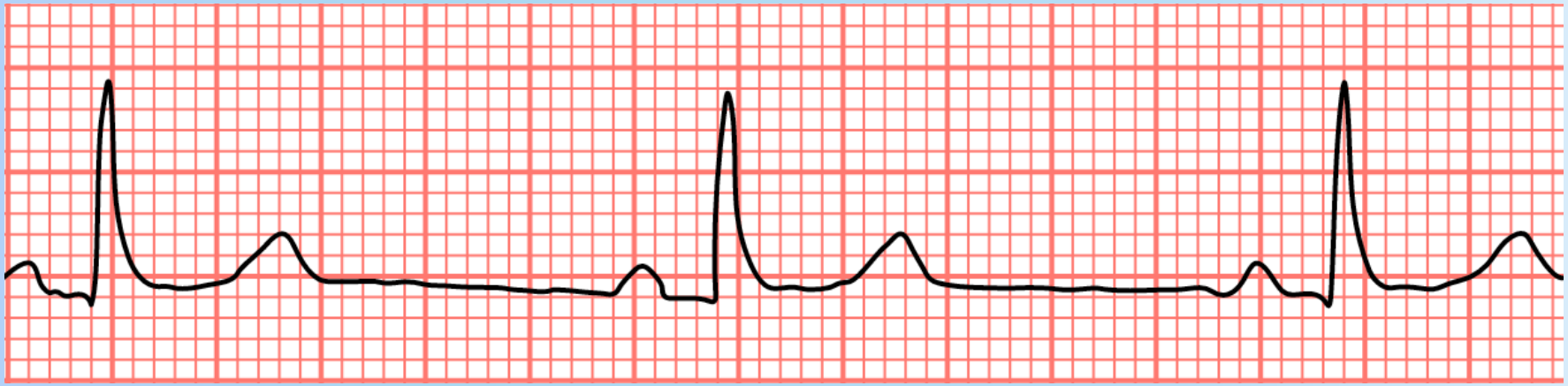
Sinus bradycardia

- Increased vagal tone → changes in pacemaker potential
  - Reduced funny current → decreased slope in phase 4
  - More neg. resting potential (-60 to -90mV)
  - More pos. threshold potential
- Physiological bradycardia: Resting, sleeping, young age, medications
- Pathological bradycardia: Failure of impulse initiation
  - Myocardial infarction of the inferior wall
  - Myocarditis, pericarditis, rheumatic fever...



Sinus bradycardia



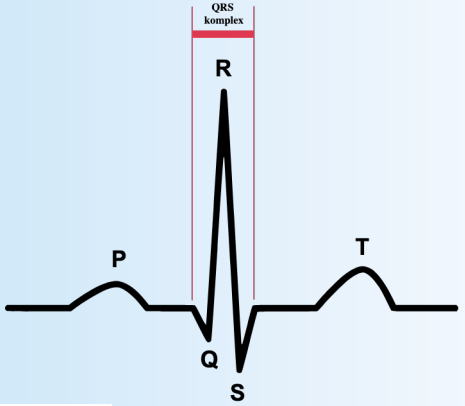


- HR = 300/number of large squares between R waves
- Bradycardia = HR <60 BPM
- Only treated when symptomatic:
  - Lightheadedness, fatigue, syncope

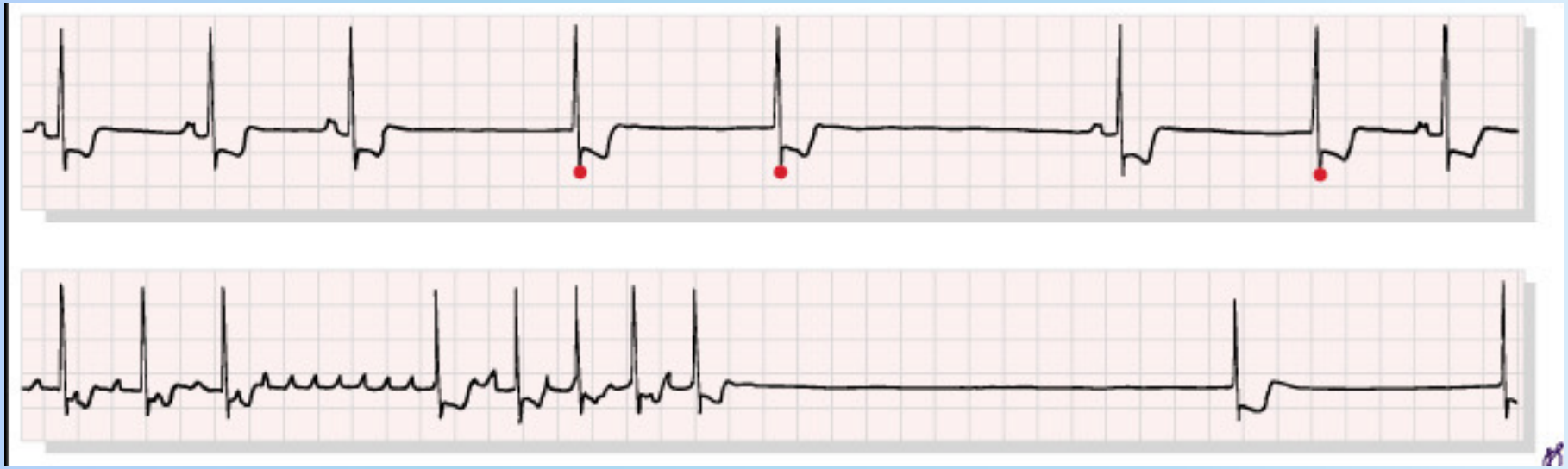
Sick Sinus Syndrome - SSS

- Sinus node is no longer able to spontaneously depolarize as normal
- Associated with various types of bradyarrhythmias
  → hypotension, dizziness and syncope
- Causes:
  - Idiopathic degenerative fibrosis (most common)
  - Ischemia
  - Atrial fibrillation
  - Heart failure, cardiomyopathy
  - Iatrogenic (beta-blockers, Ca^{2+}-channel blockers, antiarrhythmics)

Sick Sinus Syndrome - SSS



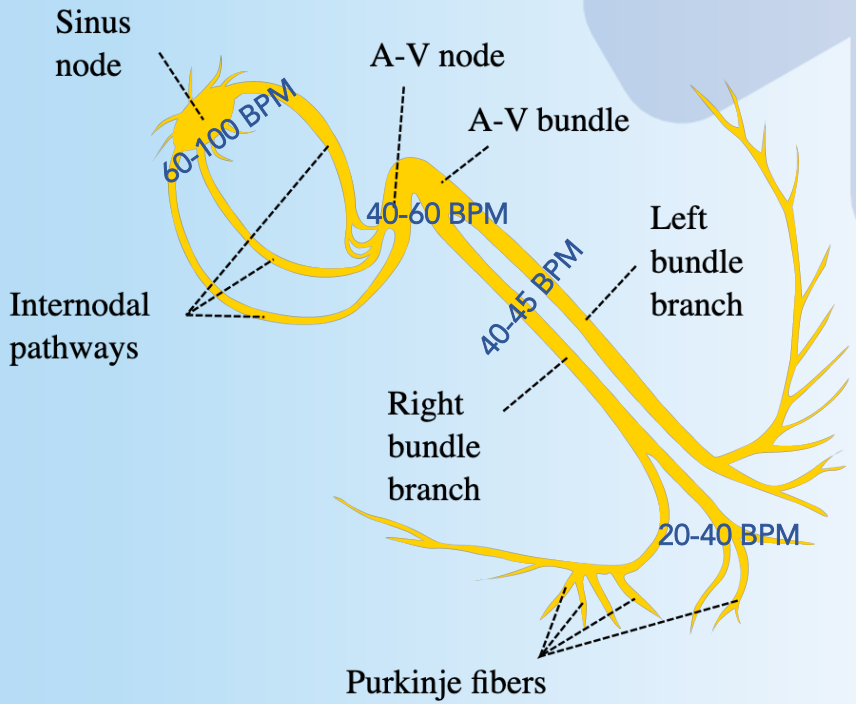
Tachycardia-bradycardia syndrome



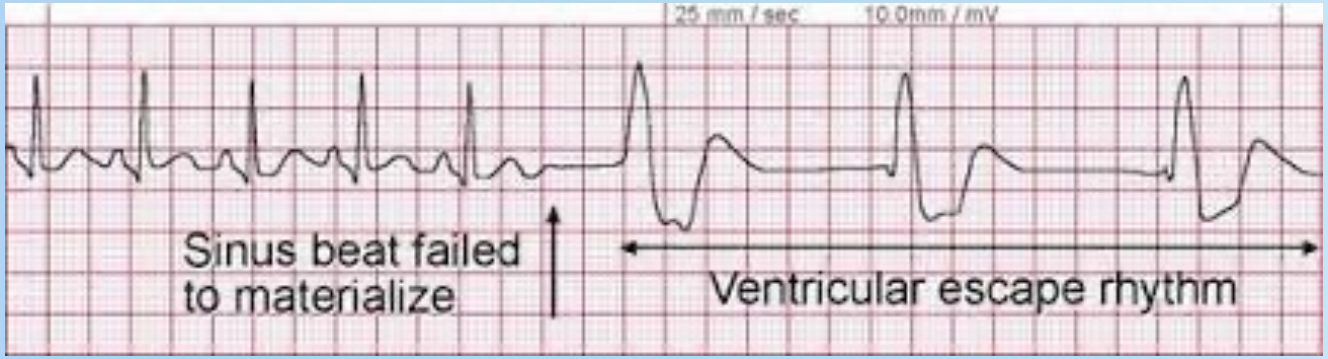
- Bradycardia, followed by tachycardia, followed by episodes of sinus arrest (no heart beat >3 sec.)
- Atrial fibrillation often present
- Tachy-brady syndrome complicates ≈50% of SSS cases

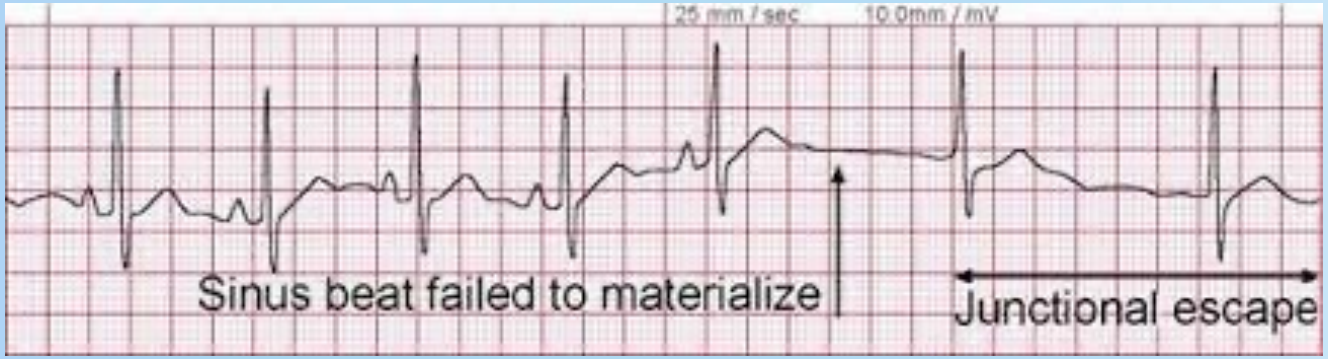
Escape rhythms

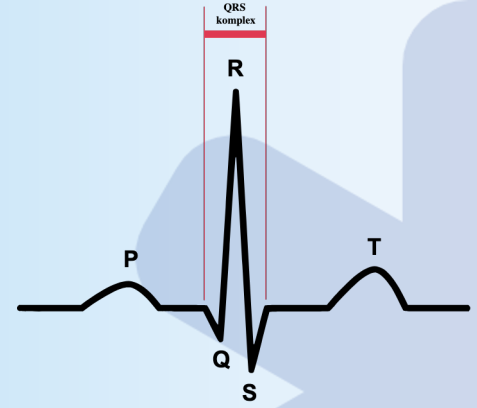
- Rhythm arising from further down the conduction system
  - Junctional escape rhythm
    - AV node or proximal His bundle
  - Ventricular escape rhythm
    - Left/right bundle branch, purkinje fibers or ventricular myocardium
- Etiology
  - Severe sinus bradycardia
  - Sinus arrest
  - High-grade AV-block
  - Iatrogenic (beta-blockers, Ca^{2+}-channel blockers)
- Treatment
  - Treat underlying cause
  - Treat as bradycardia if symptomatic

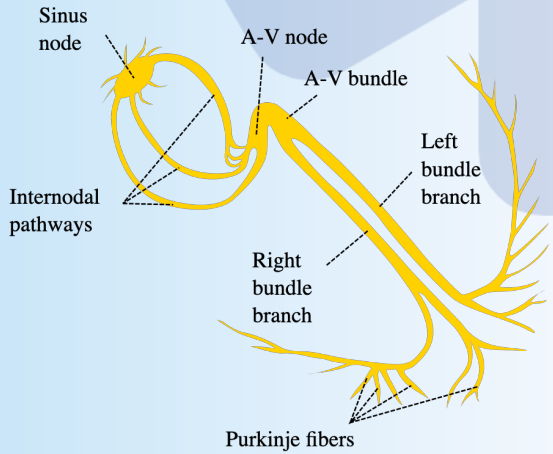


Escape rhythms









- Bradycardia, followed by escape rhythm
- Beats arising from outside SAN have different morphology on ECG

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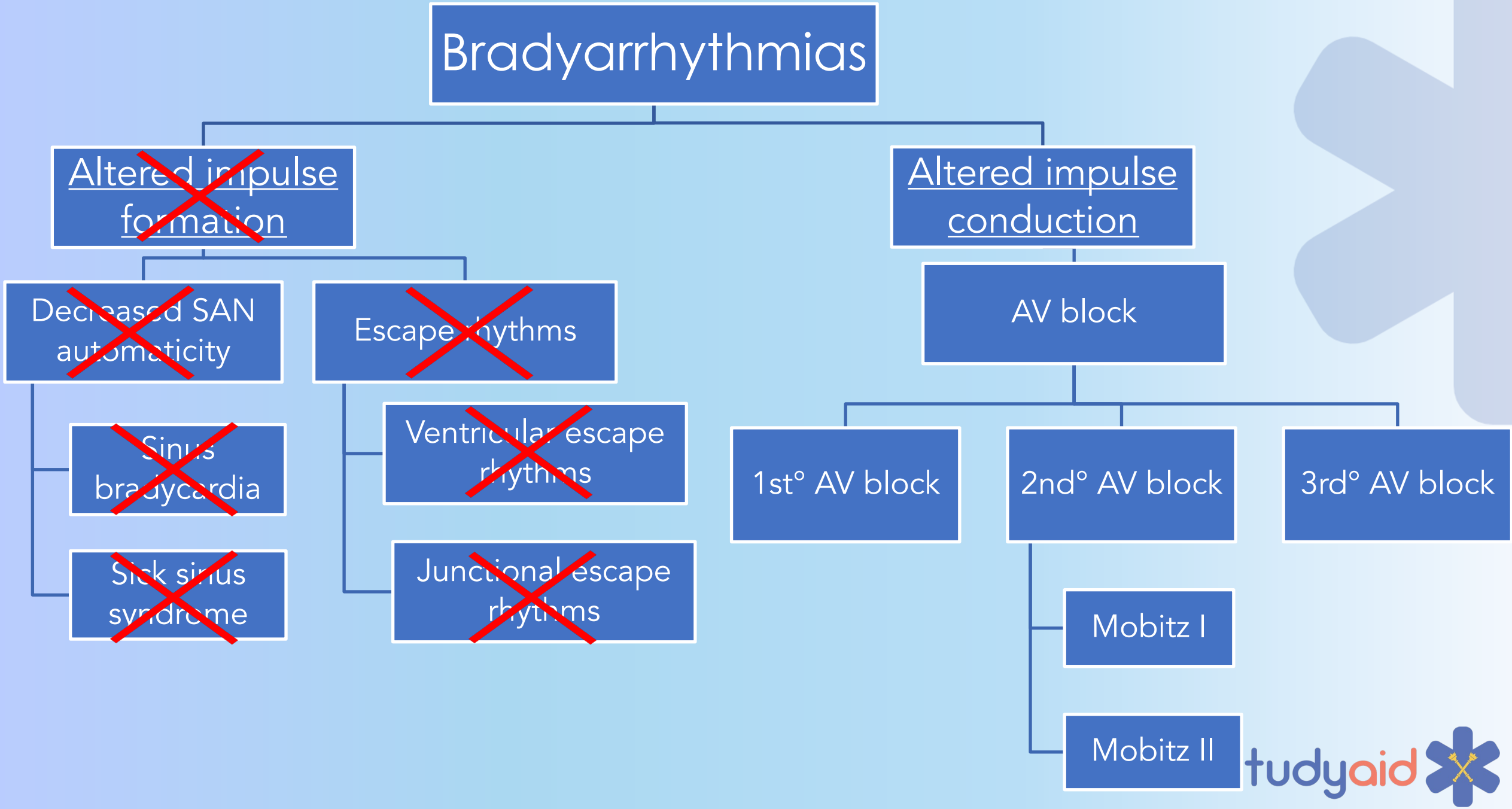
Bradyarrhythmias
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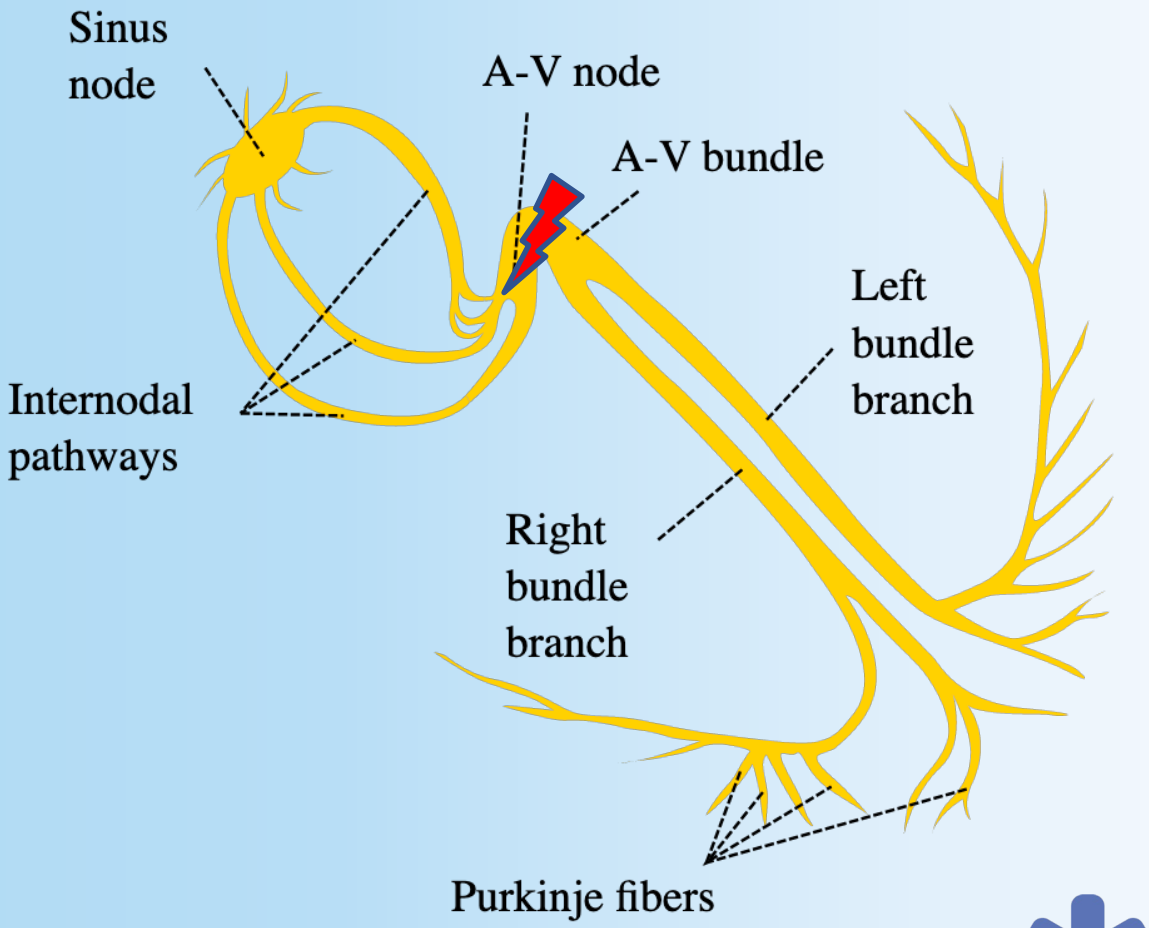
1st° AV block
2nd° AV block
3rd° AV block
Mobitz I
Mobitz II
tudyaid

Conduction block

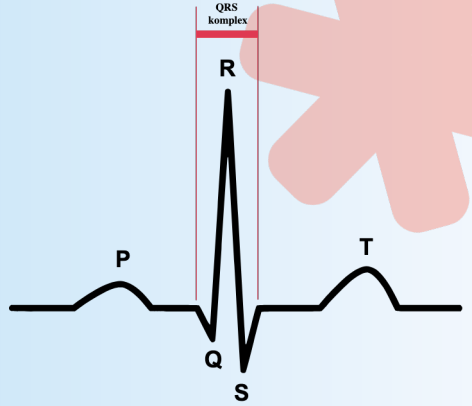
- Impulse is not able to travel normally through conduction system
  - Bidirectional block → AV-block
  - Unidirectional block → re-entry tachycardia

- AV-block
  - 1st degree AV-block
  - 2nd degree AV-block
    - Mobitz I/Wenckebach
    - Mobitz II
  - 3rd degree AV-block

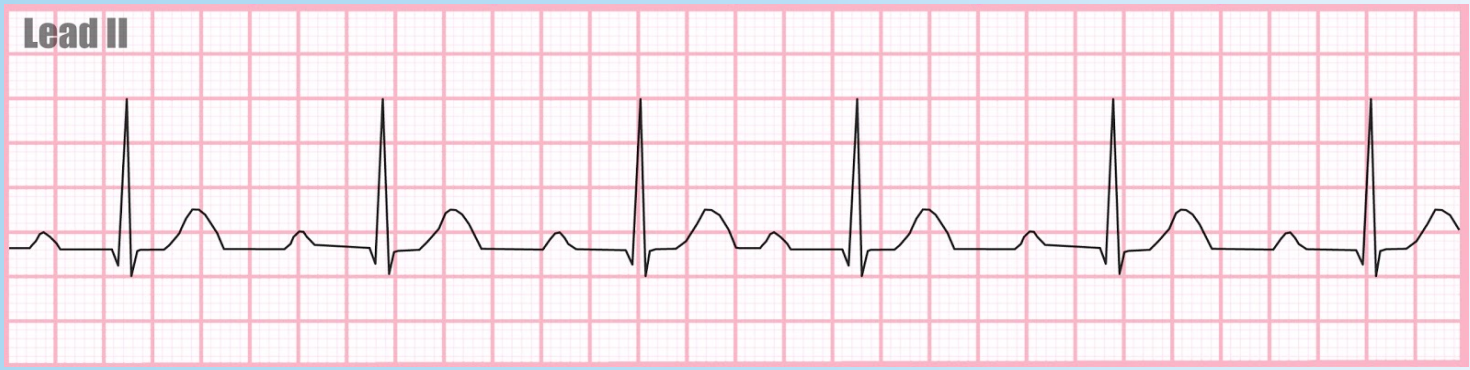


Conduction block

1st degree AV-block



- PR interval longer than normal (>200ms)
- Common in older patients. Clinically insignificant.
- Etiology:
  - AV nodal fibrosis
  - High vagal tone
  - B-blockers
  - Hypokalemia



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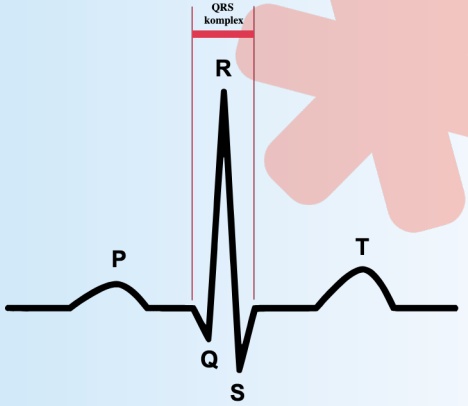
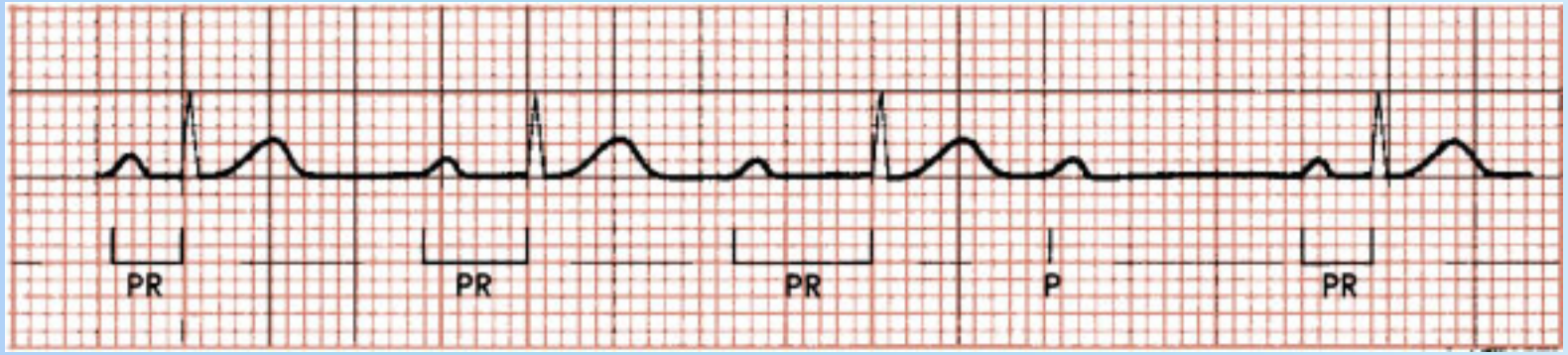
Conduction block

2nd degree AV-block, Mobitz I/Wenckebach



- Gradual prolongation of PR interval until a P wave is not conducted through AV-node → *missed beat*
- PP interval is constant, PR interval is variable
- Only treated when symptomatic



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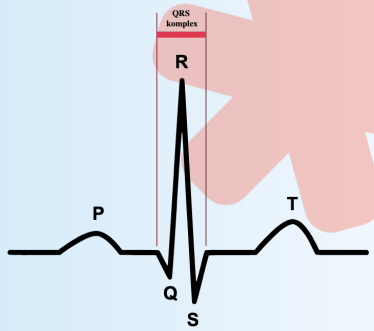
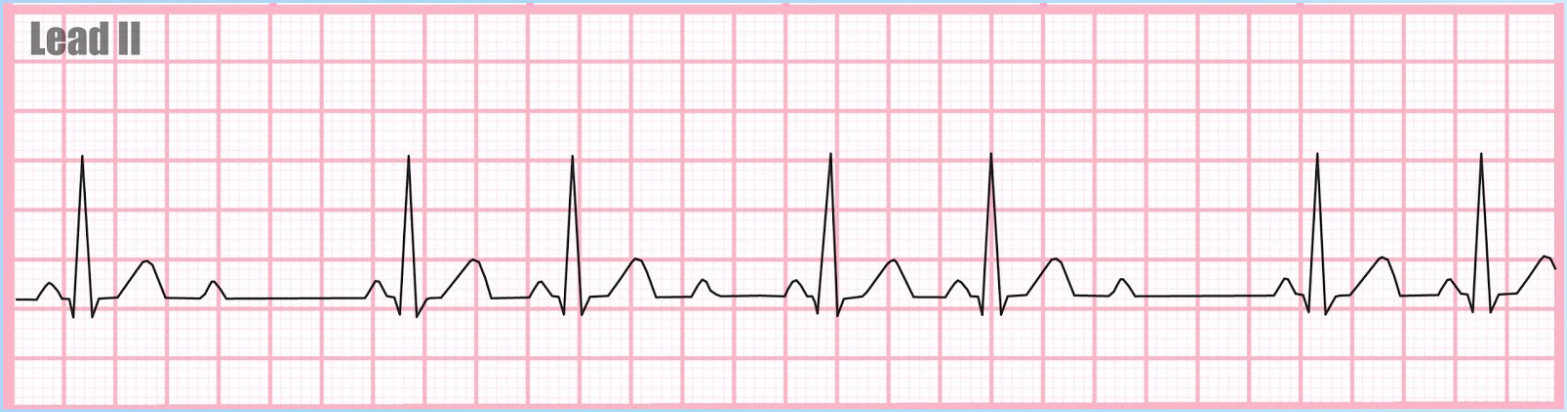
Conduction block

*2nd degree AV-block, **Mobitz II***



- Constant PR duration, but some P waves are not conducted through AV-node → *missed beat*
- PP interval is constant & PR interval is constant
- Associated with:
  - progression to 3rd degree AV-block, cardiogenic shock and sudden cardiac death
- Treated with implantation of permanent pacemaker



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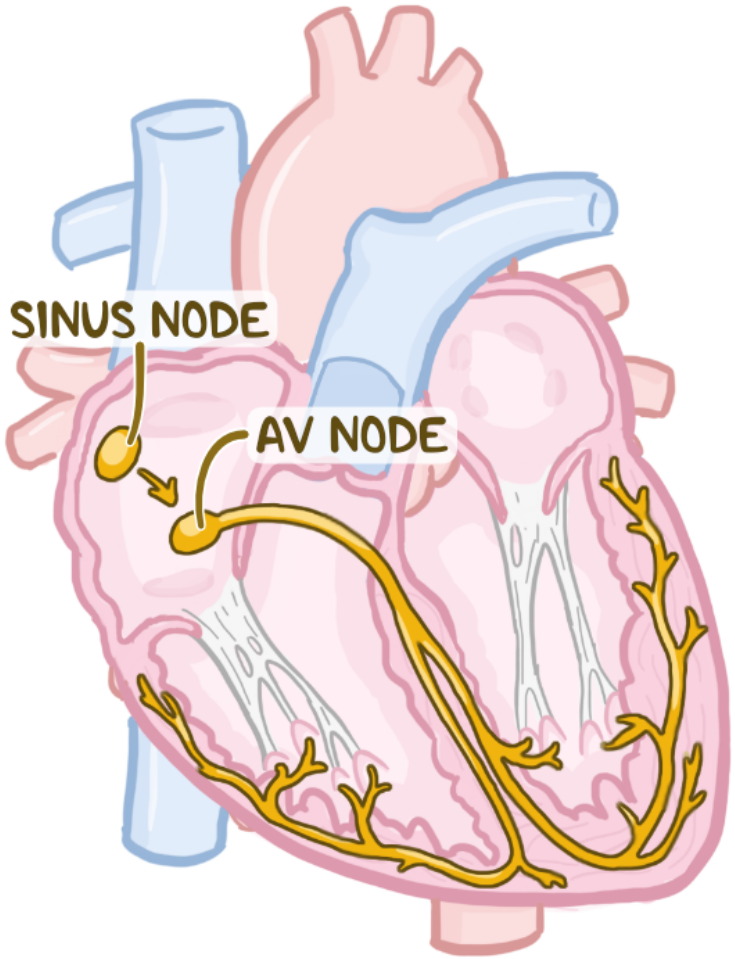
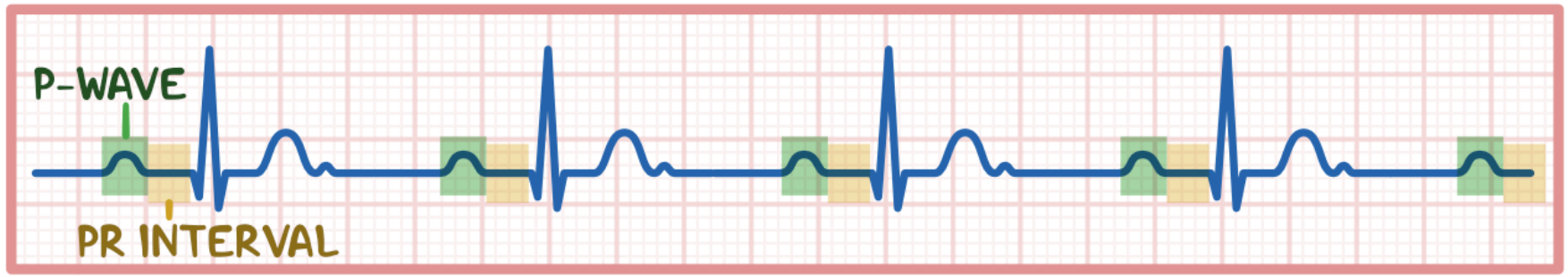
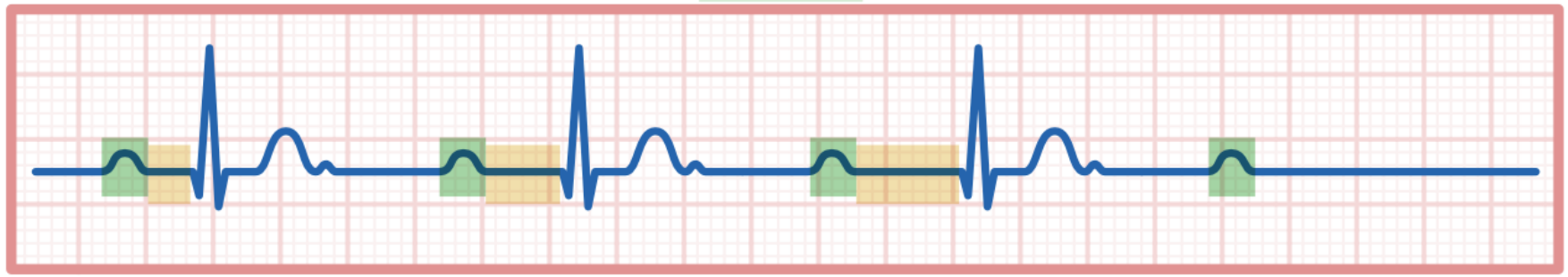


HEALTHY ECG



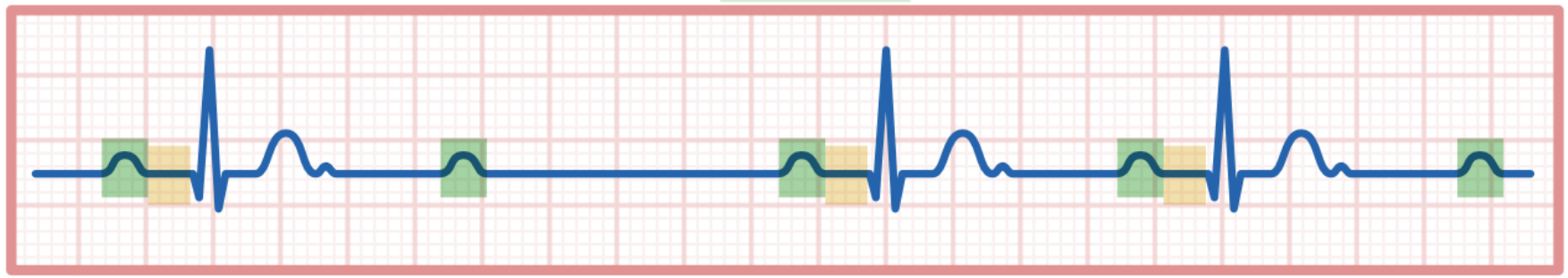
MOBITZ TYPE I

PR INTERVALS GRADUALLY ELONGATE UNTIL a P-WAVE is COMPLETELY BLOCKED



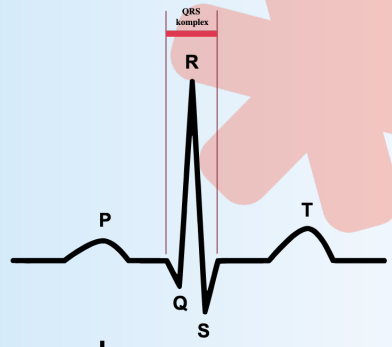
MOBITZ TYPE II

PR INTERVALS are CONSISTENT, but SOME P-WAVES DON'T CONDUCT

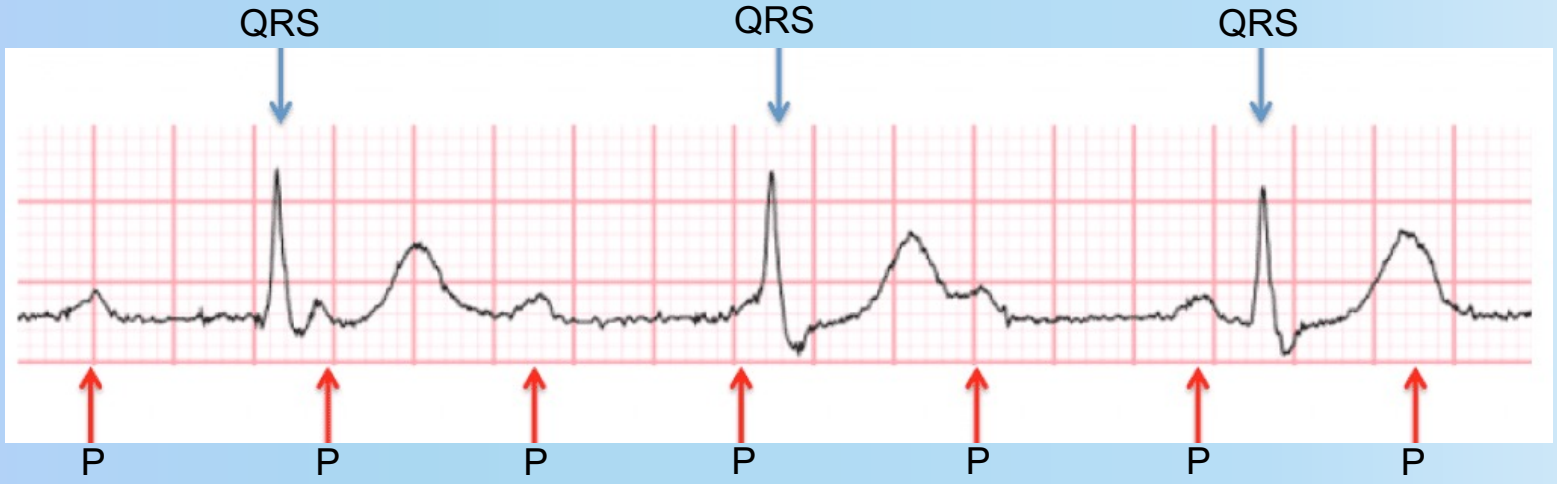


Conduction block

3rd degree AV-block



- No conduction between atria & ventricles → P & QRS are independent
  - QRS complexes may arise from AV junction → *normal width*
  - QRS complexes arise from His-purkinje system → *wide QRS*
- HR: 40-60 BPM
- High risk of sudden cardiac death
- Treated by contemporary pacing until implantation of pacemaker



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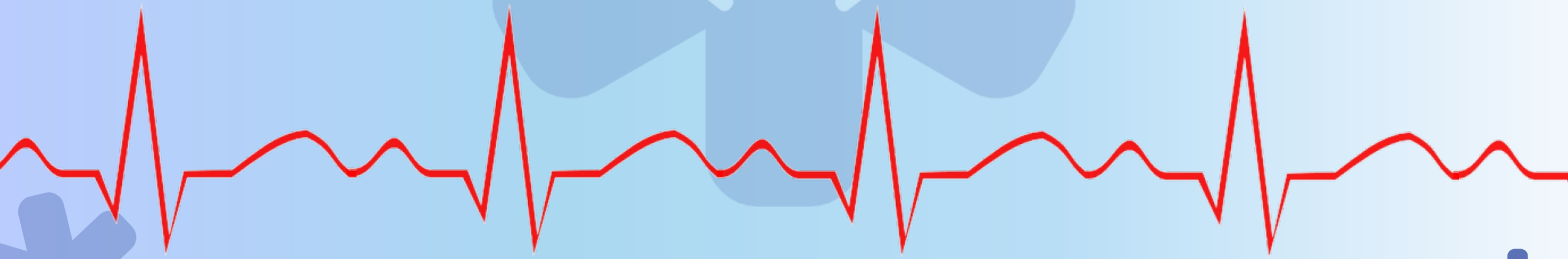
Tachyarrhythmias

Tachy = fast



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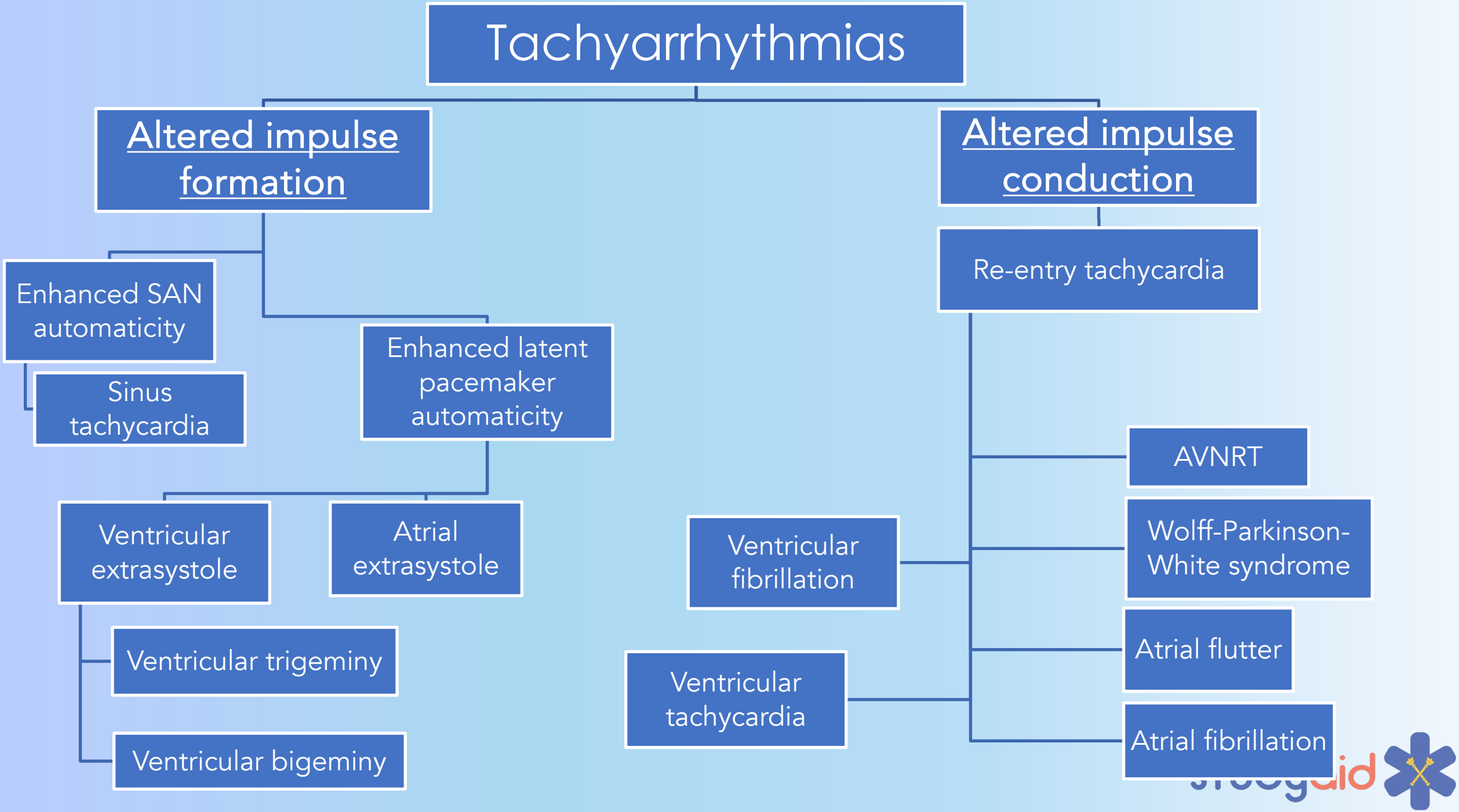
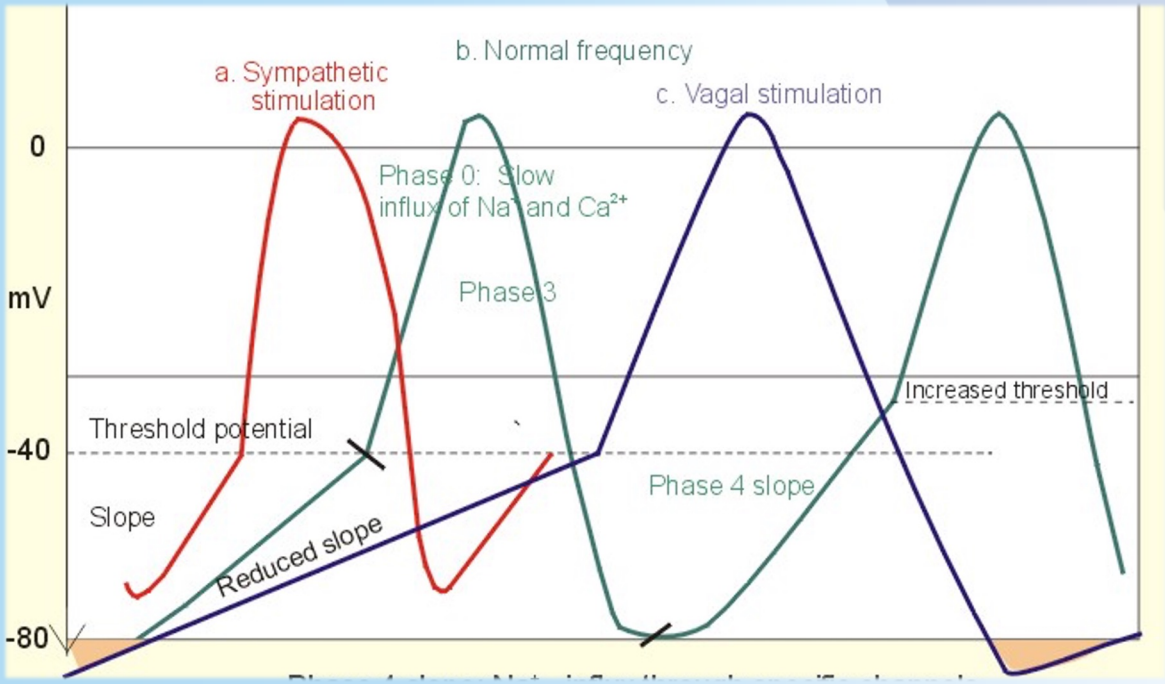
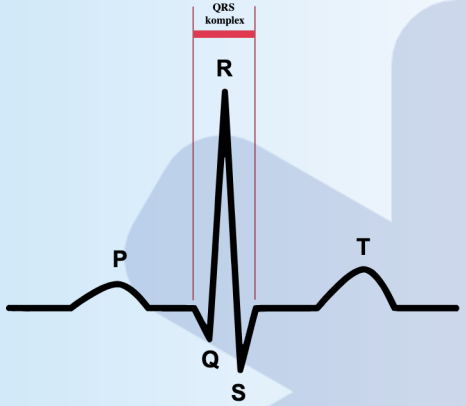

Tachyarrhythmias
Altered impulse formation
Altered impulse conduction
Enhanced SAN automaticity
Sinus tachycardia
Enhanced latent pacemaker automaticity
Ventricular extrasystole
Atrial extrasystole
Ventricular trigeminy
Ventricular bigeminy
Re-entry tachycardia
Ventricular fibrillation
Ventricular tachycardia
AVNRT
Wolff-Parkinson-White syndrome
Atrial flutter
Atrial fibrillation

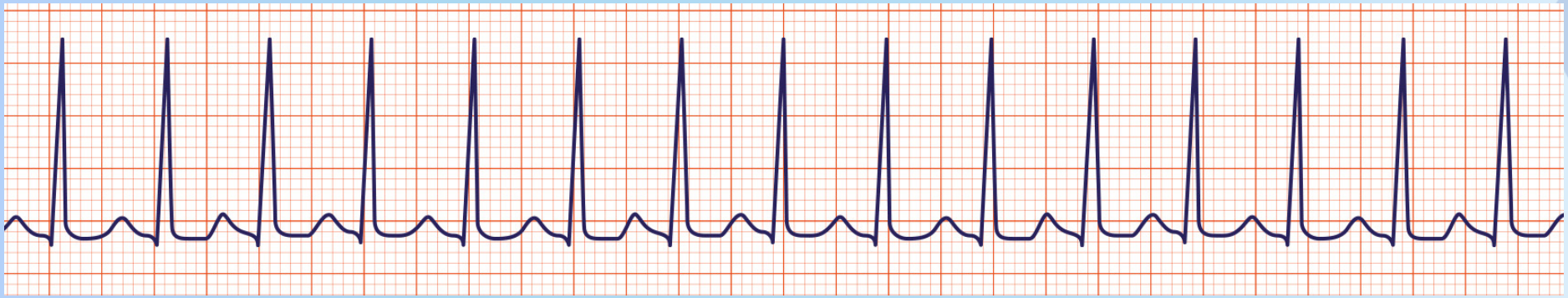
Sinus tachycardia

- Decreased vagal tone and/or increased sympathetic tone → changes in pacemaker potential:
  - Increased funny current → increased slope in phase 4
  - Less negative resting potential (-60mV to -30mV)
  - More negative threshold potential
- Physiological tachycardia: Exercise, stress, pregnancy
- Pathological tachycardia: Anemia, hyperthyroidism, liver disease



Sinus tachycardia





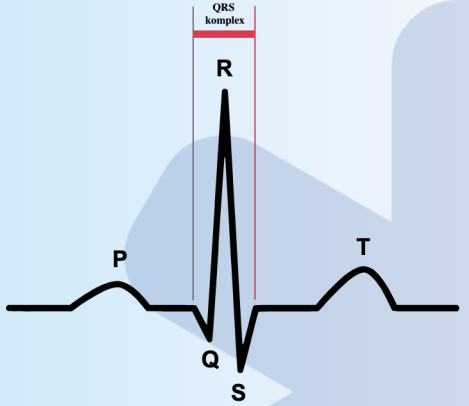
- Defined as HR 100-160 BPM with no other ECG changes
- May be symptom of other pathology (sepsis, hypovolemia, hypoxia)

Ectopic beats

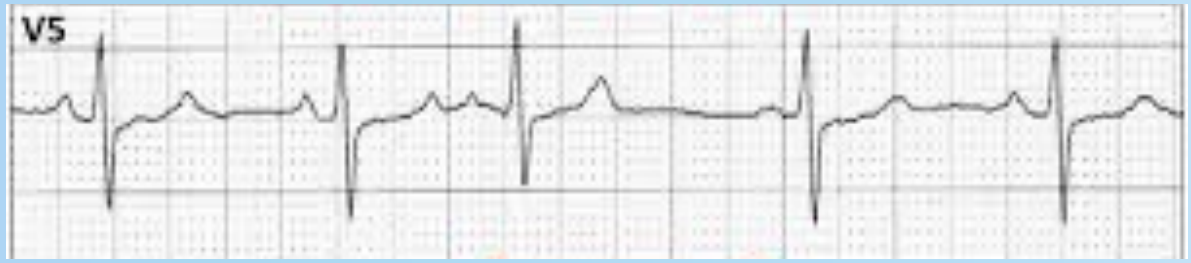
Premature beats, atrial/ventricular extrasystole

- Increased automaticity by latent pacemakers, or afterdepolarizations
  - Can arise in both atria and ventricles
- Etiology:
  - Caffeine, alcohol, stress, beta-blockers
  - Hypoxemia, electrolyte imbalances, drug use
- Present in all individuals, usually not treated.
  - May cause palpitations, which need treatment
  - Many extrasystoles in a row predisposes ventricular tachycardia/fibrillation, and needs treatment

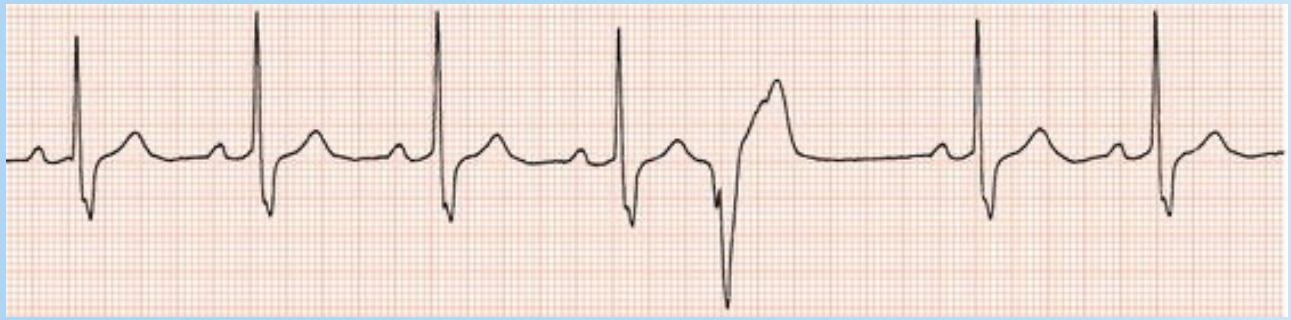
Ectopic beats



Atrial extrasystole

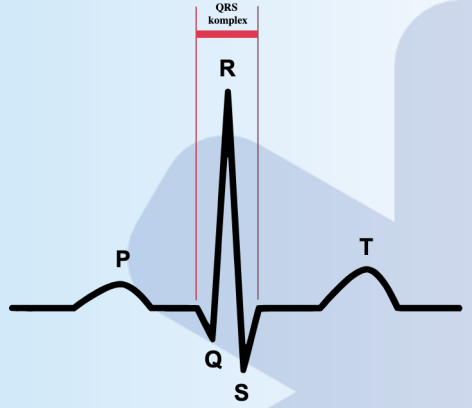


Ventricular extrasystole

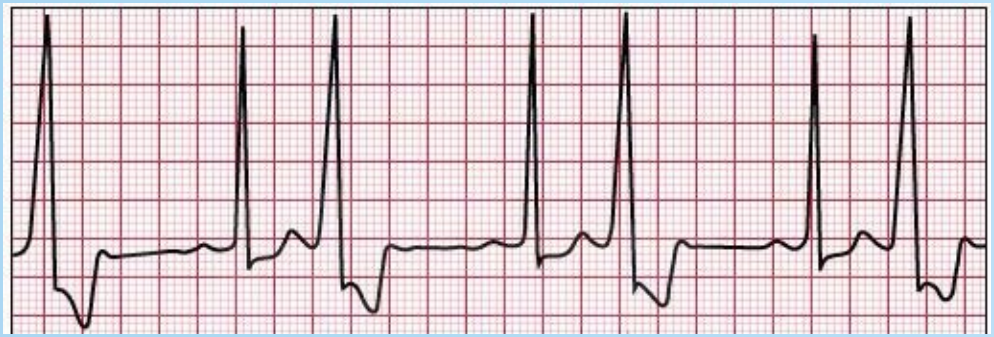


- Beats are asynchronous with otherwise normal rhythm
  - Atrial extrasystole has different P-wave, but normal QRS
  - Ventricular extrasystole has no P-wave, and a wide QRS
- Often followed by a compensatory pause for repolarization

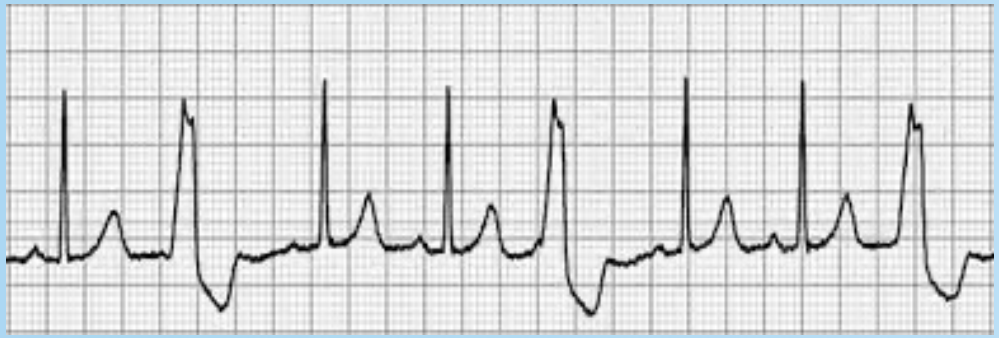
Ectopic beats



Ventricular bigeminy

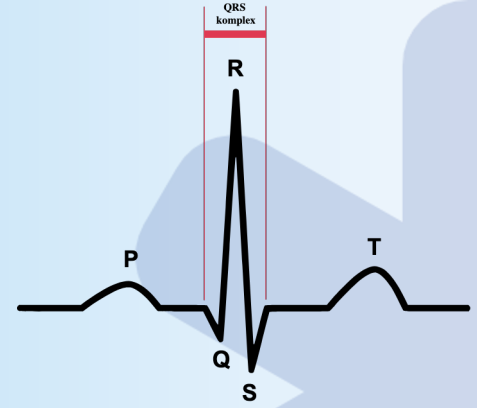


Ventricular trigeminy

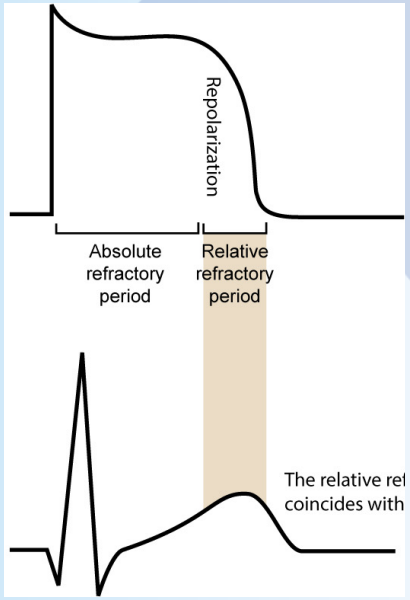


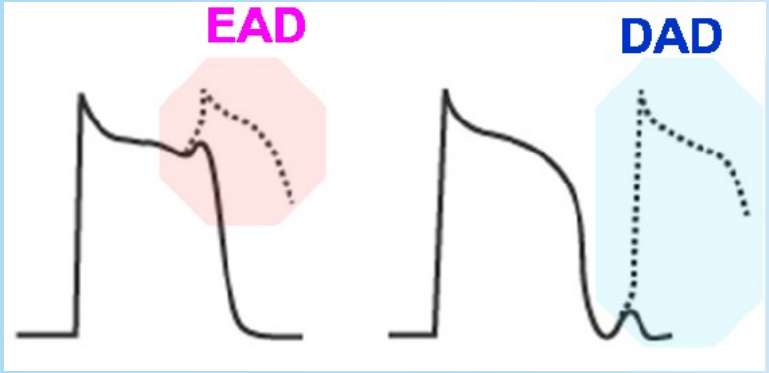
- Ventricular bi-/trigeminy: every 2nd/3rd beat is ventricular extrasystole
- Usually benign, but may predispose to other dangerous arrhythmias
- Associated with ischemia and drug toxicities

Afterdepolarizations



- Abnormal depolarizations of cardiomyocytes
- <u>Early afterdepolarizations (EAD)</u>
  - Electricity changes during repolarization
  - Occur during plateau phase (2) or repolarization phase (3)
- <u>Delayed afterdepolarizations (DAD)</u>
  - Spontaneous Ca^{2+} release during relaxation phase
  - Associated with states causing high intracellular Ca^{2+} (i.e. Digoxin use)
- Lead to:
  - Ectopic beats
  - Re-entry tachycardia





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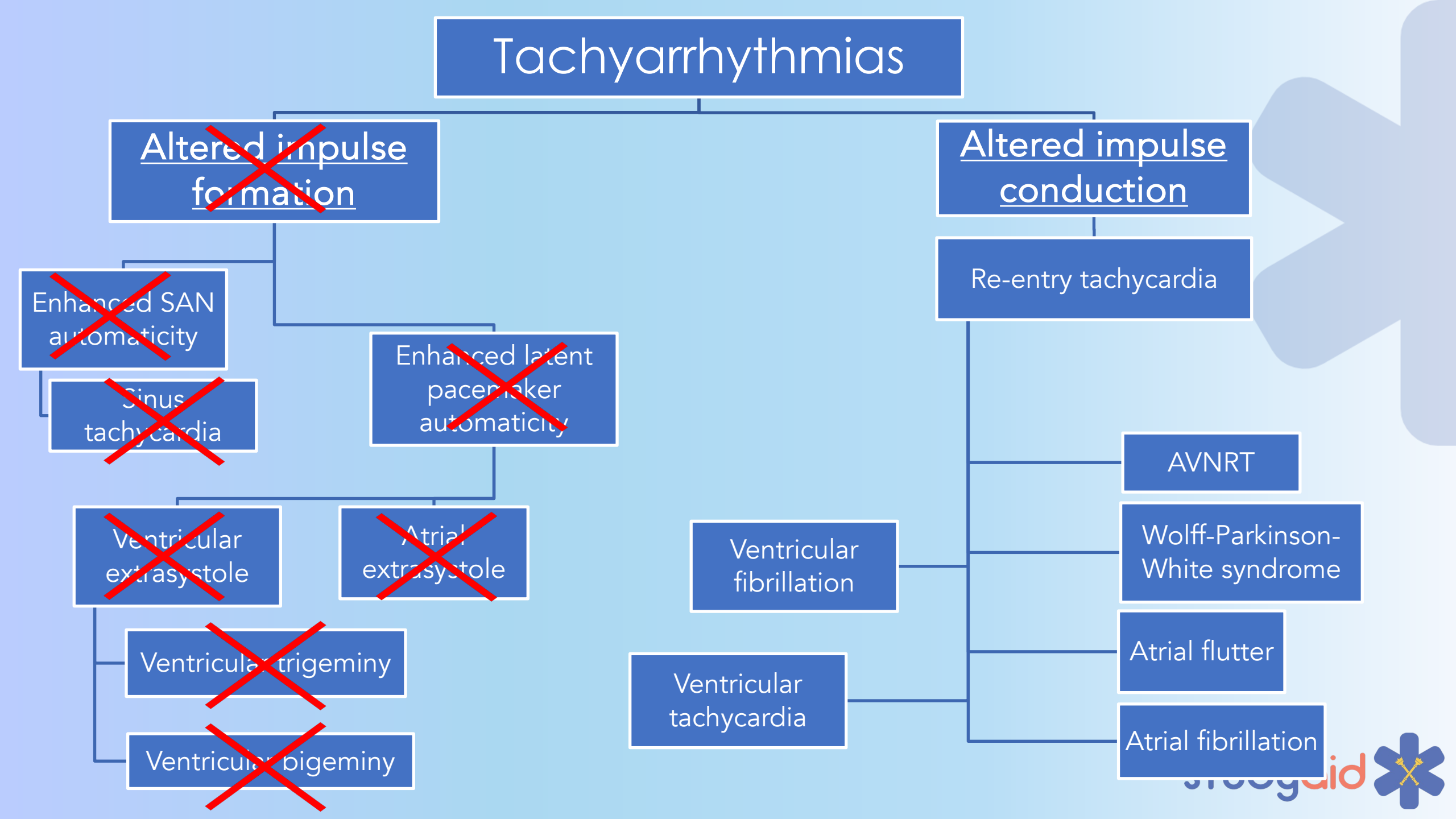

Tachyarrhythmias
Altered impulse formation
Enhanced SAN automaticity
Sinus tachycardia
Enhanced latent pacemaker automaticity
Ventricular extrasystole
Atrial extrasystole
Ventricular trigeminy
Ventricular bigeminy
Altered impulse conduction
Re-entry tachycardia
AVNRT
Wolff-Parkinson-White syndrome
Atrial flutter
Atrial fibrillation
Ventricular fibrillation
Ventricular tachycardia

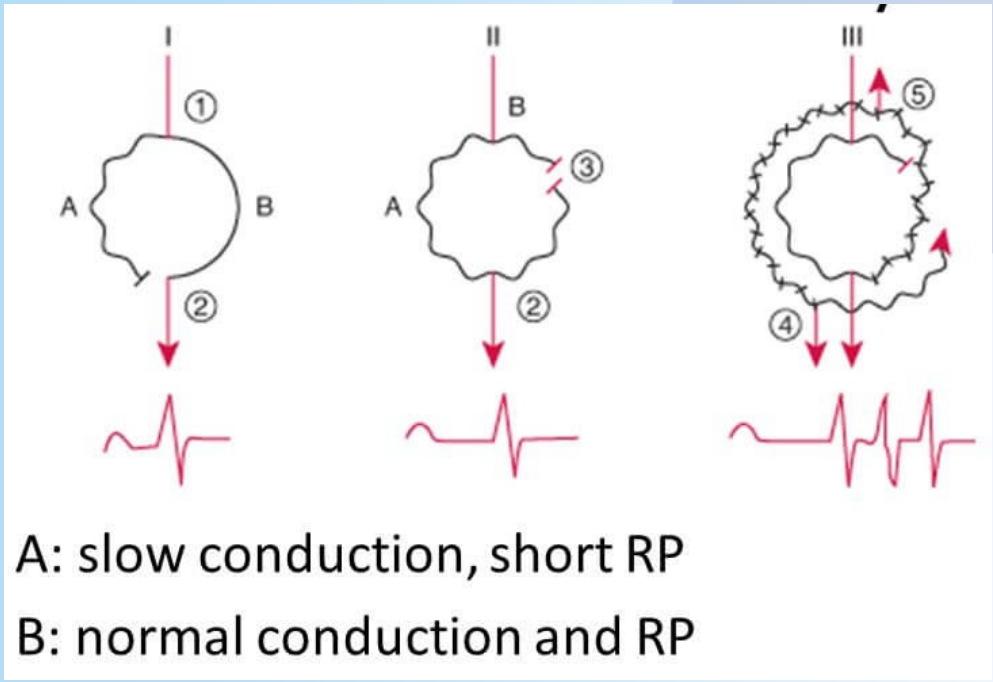
Re-entry tachycardia

- Most common mechanism for tachyarrhythmia
  - Slow-fast AVNRT is most common subtype

- Excitable tissue with slow conduction, has a short refractory period
- Excitable tissue with fast conduction, has a long refractory period

This allows for:

- Unidirectional conduction block
- Slowed conduction through the re-entry loop



https://youtu.be/j8pVU9snSH4?t=121

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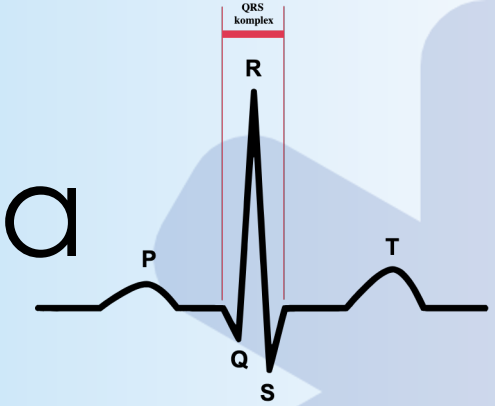
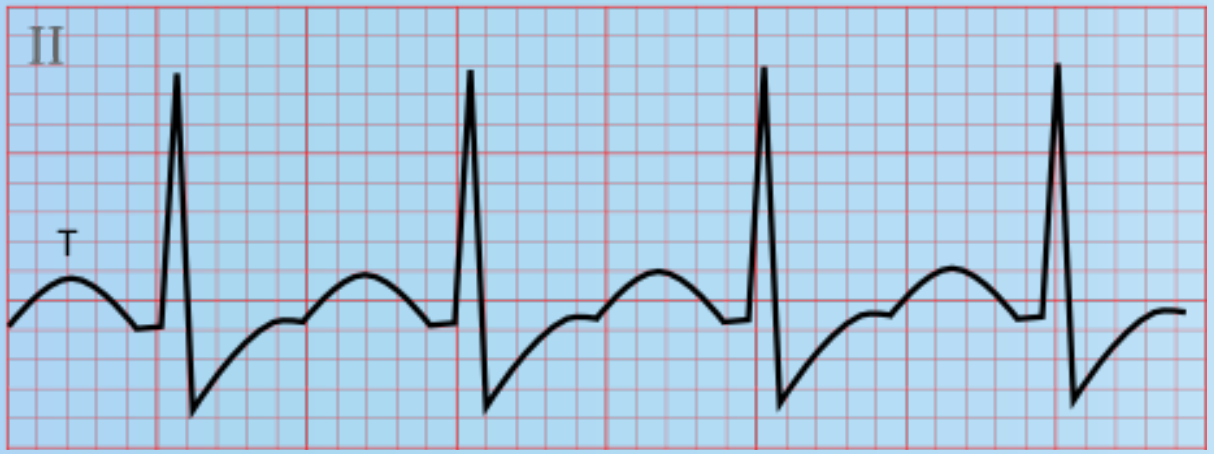
Nodal re-entry tachycardia

Slow-fast AtrioVentricular Nodal Re-entry Tachycardia (AVNRT)





- Normal QRS-complexes with no preceding P-waves
- Heart rate 120-240 BPM
- Treated with beta-blockers, Ca^{2+}-channel blockers, ablation therapy

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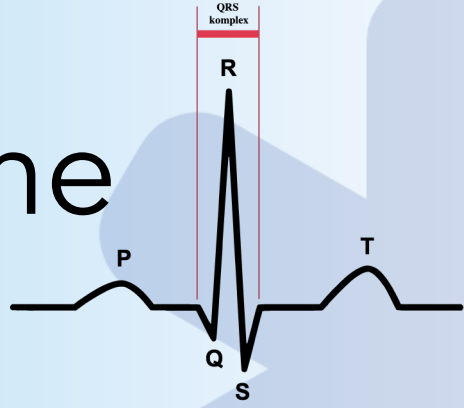
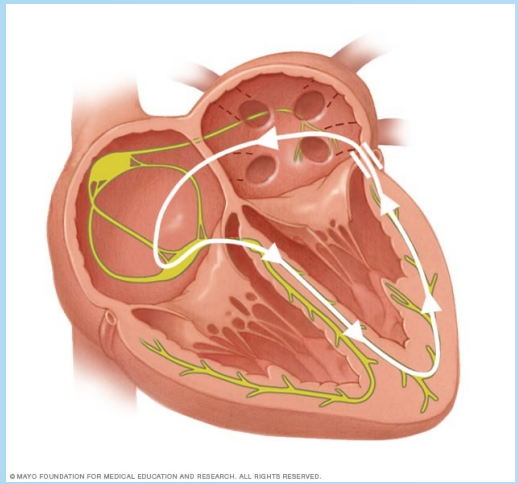
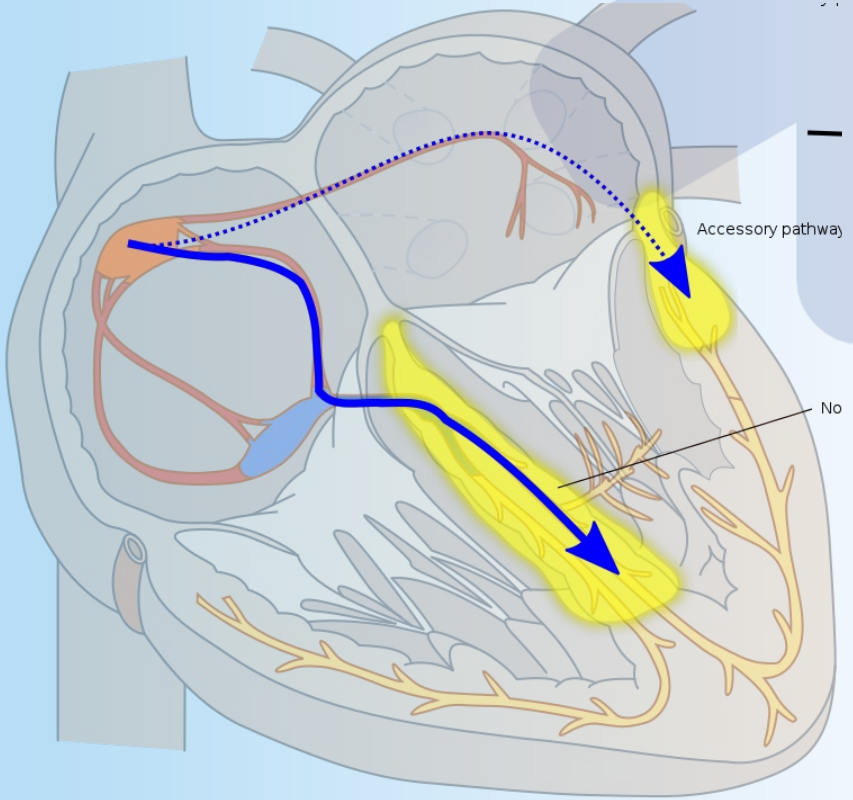
Wolff-Parkinson White syndrome



- Presence of an accessory atrioventricular pathway: Bundle of Kent
  - Present in ≈ 1 in 1000 people
- Bypasses AV-node → premature contraction of ventricles → shortened PR interval (< 0.12ms)
- Can lead to **atrioventricular re-entry tachycardia (AVRT)**





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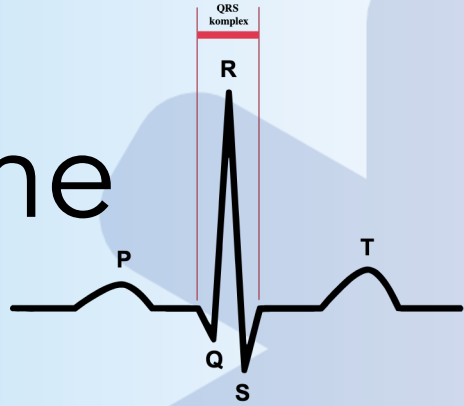
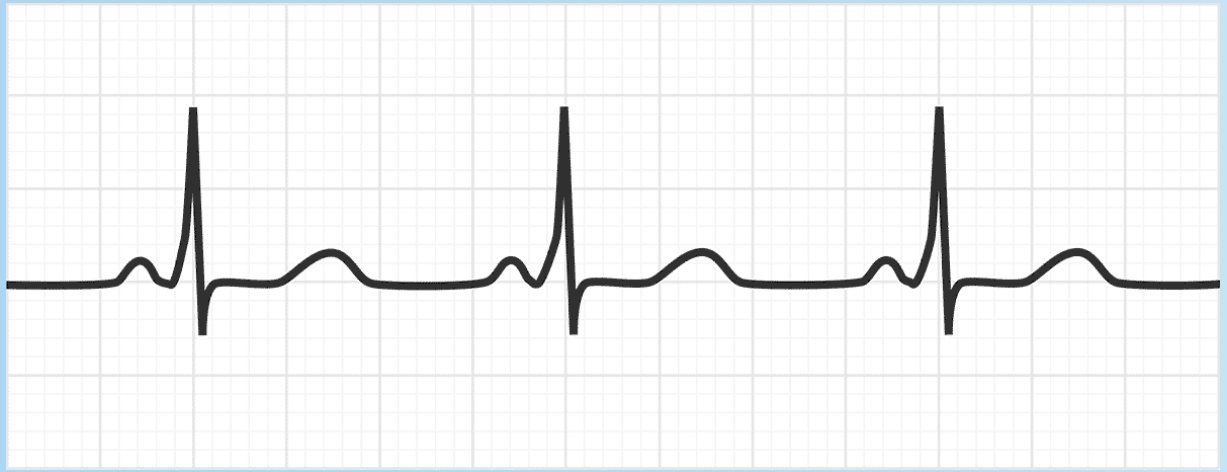
Wolff-Parkinson White syndrome

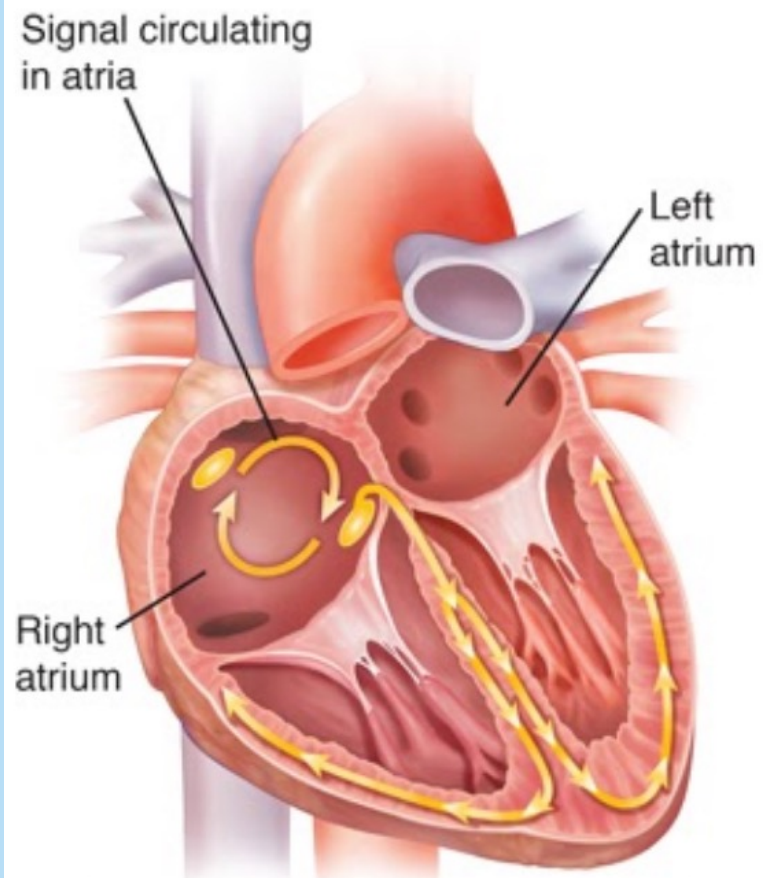




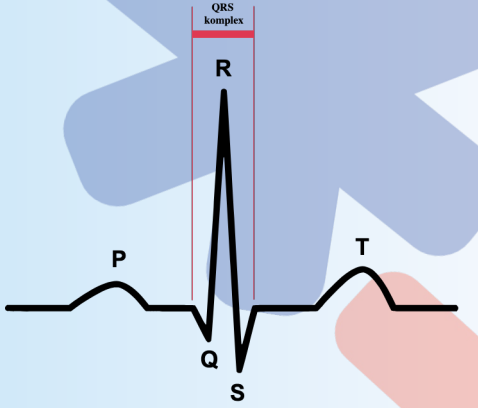
- Premature ventricular contraction → short PR interval & early upstroke of the QRS complex
- Early upstroke + normal upstroke of QRS seen as a **delta wave**

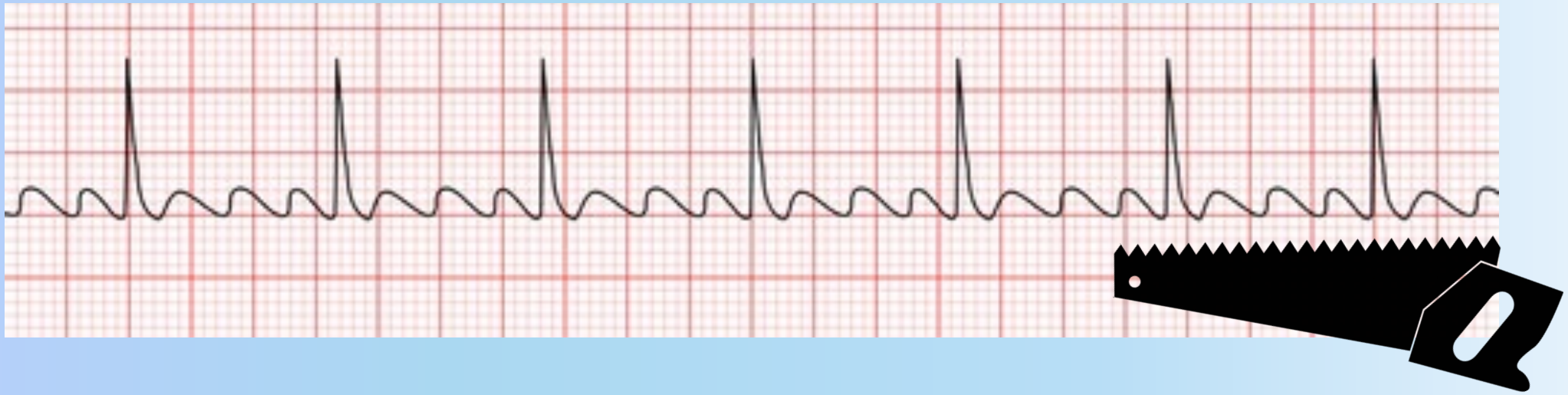
Atrial flutter

- Intra-atrial re-entry tachycardia
- Atrial rate: regular 180-350 BPM
- Impulses reach AV-node during refractory period → ventricular rate is slower (80-180 BPM)
- Predisposes for blood-clot formation in the atria
- Treated with beta-blockers, Ca^{2+}-channel blockers, ablation therapy, blood thinners



Atrial flutter





- P-waves (flutter waves) are regular with a high frequency; «sawtooth appearance»
- QRS-complexes are regular with normal morphology, but may be fast

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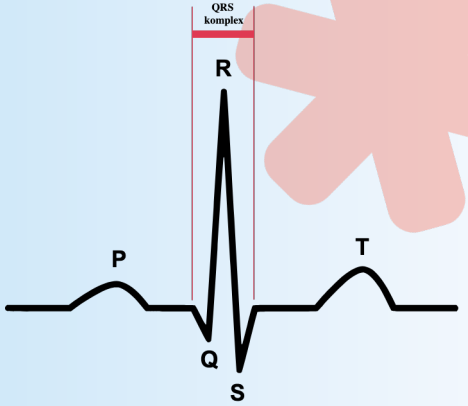
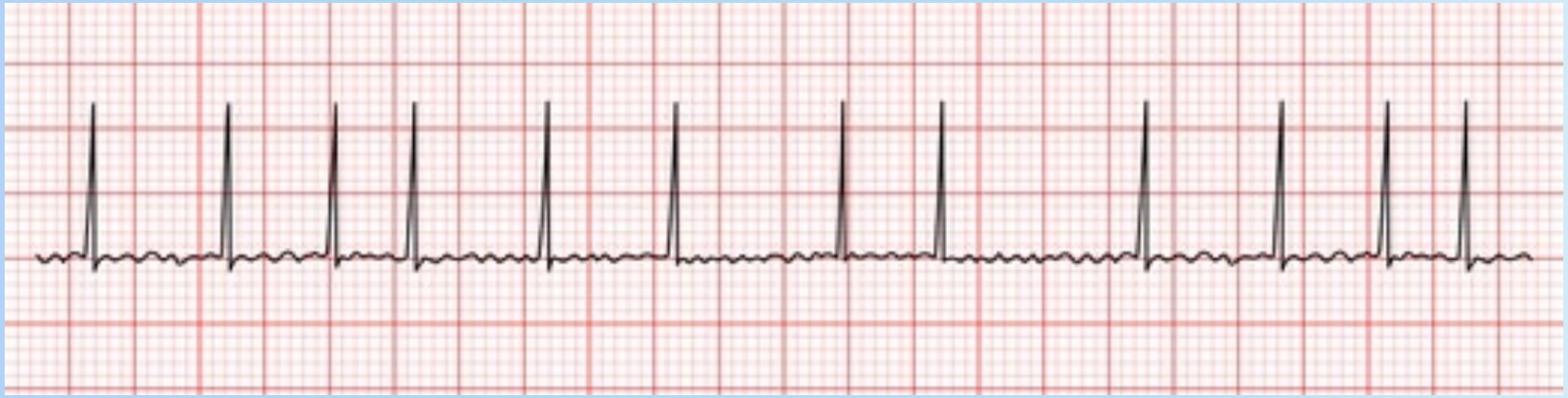
Atrial fibrillation

- Intra-atrial re-entry tachycardia (most common)
- Atrial rate: irregular 350-600 BPM
    - So fast and chaotic that P-waves are not distinguishable
- Ventricular rate: irregular 160 BPM
- Associated with enlarged atria (eccentric hypertrophy)
- Predisposes for blood-clot formation in the atria
- Treated with beta-blockers, Ca^{2+}-channel blockers, ablation therapy, blood thinners

Atrial fibrillation



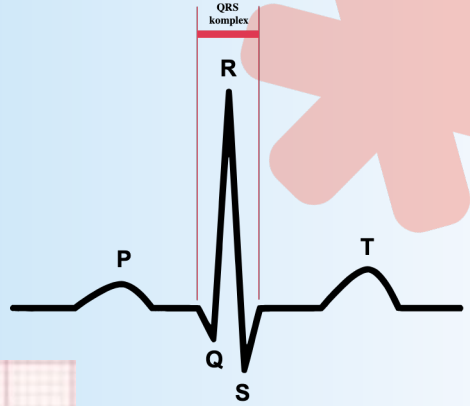


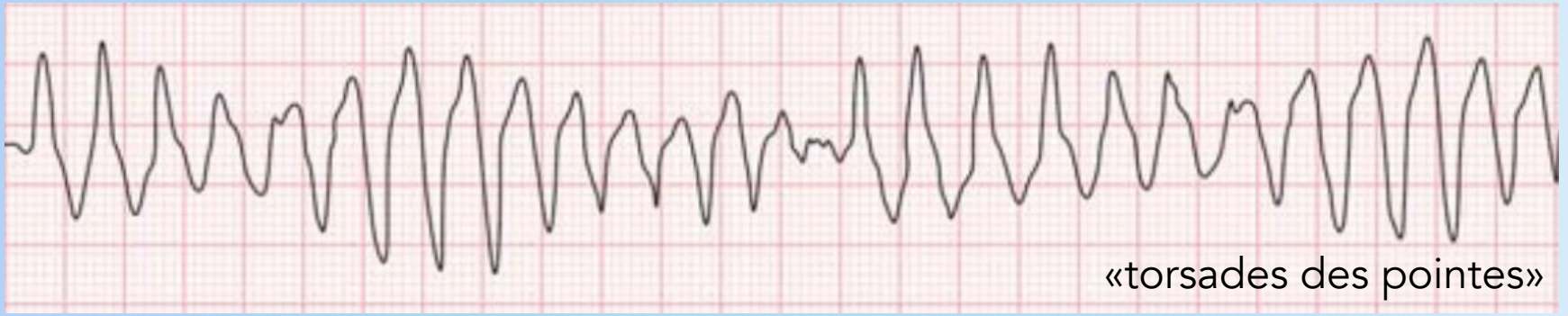
- P-waves are barely visible
- Irregular and fast QRS-complexes with normal morphology

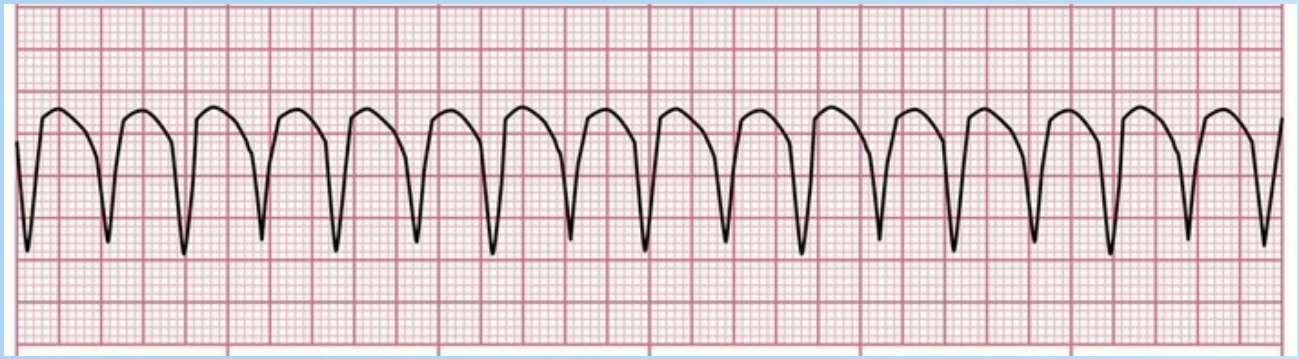
Ventricular tachycardia

- Ventricles are pumping ineffectively → cell death
- Symptoms: Dyspnoea, chest pain, syncope, sudden cardiac death
- Etiology
  - Increased automaticity in latent ventricular pacemaker cells
  - Ventricular re-entry tachycardia
    - Myocardial scarring, afterdepolarizations (long QT-syndrome)
- Needs urgent treatment
  - Defibrillation
  - Cardioversion
  - Implantation of ICD (implantable cardioverter-defibrillator)

Ventricular tachycardia







- Broad, continously repeating QRS complexes
- HR: 110-250 BPM

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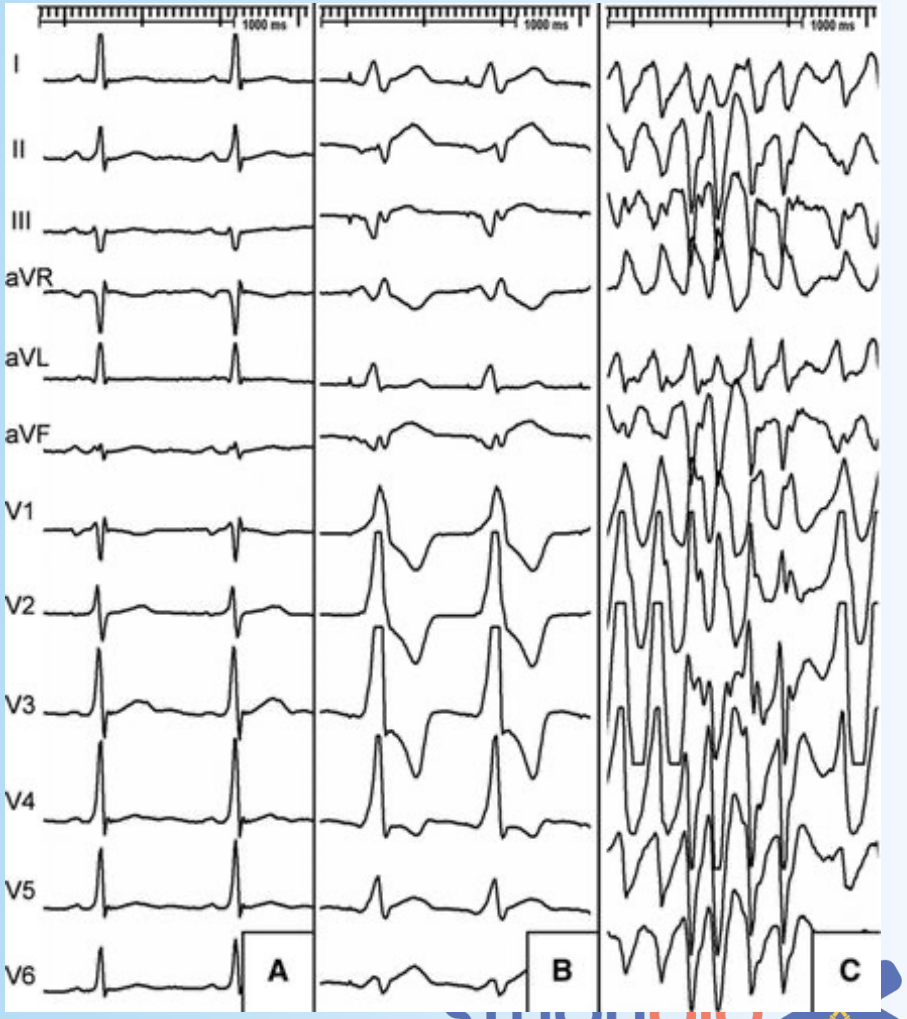
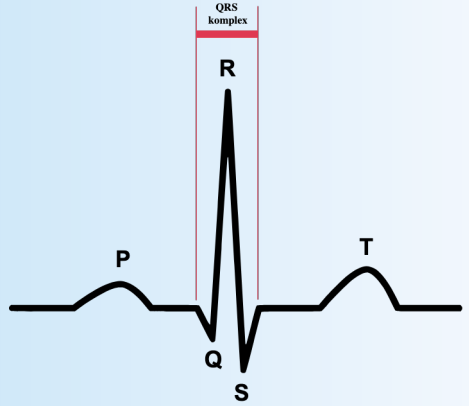
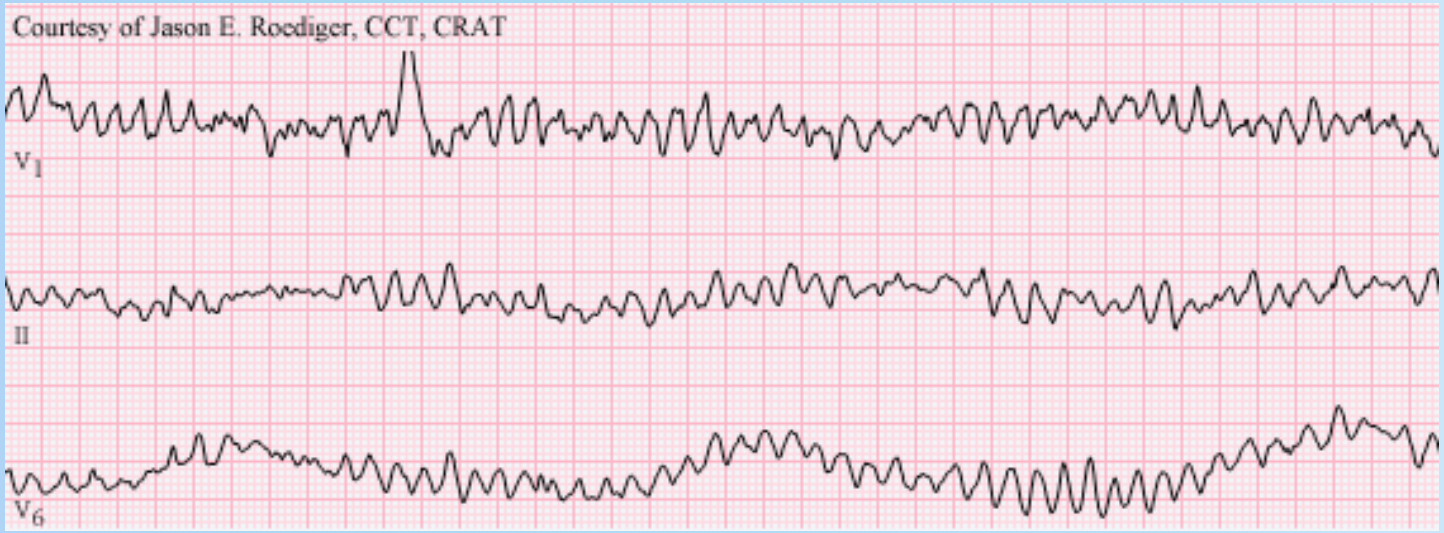
Ventricular fibrillation

- Chaotic ventricular rhythm, 150-500 BPM
- Heart «vibrating» → **no blood is pumped**
- Associated with ventricular tachycardia, myocardial ischemia
  - Major cause of death in MI
- Immediate intervention:
  - External defibrillation, antiarrhythmics, adrenaline



Ventricular fibrillation





- No distinguishable features visible on ECG
- Heart rate 150-500 BPM
- Mortality rate 90-95%