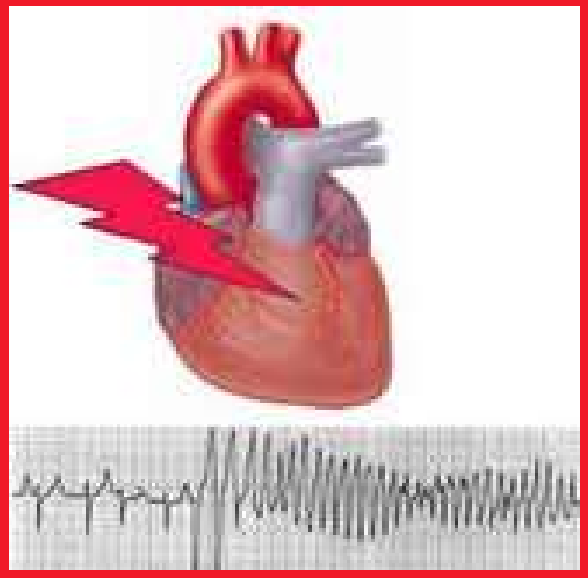
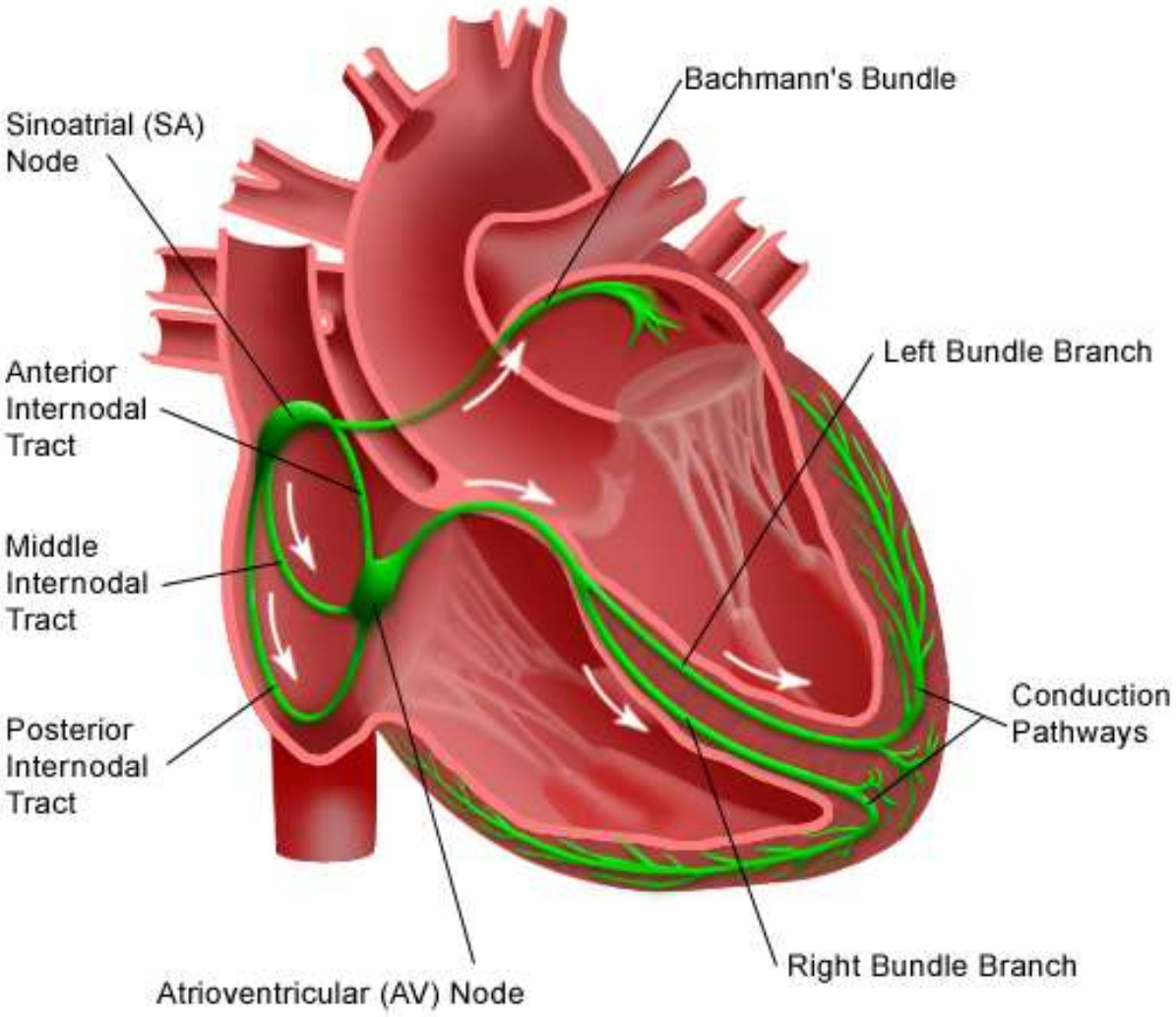
MUNI
MED

Arrhythmology



Electrical System of the Heart



Cardiomyocytes

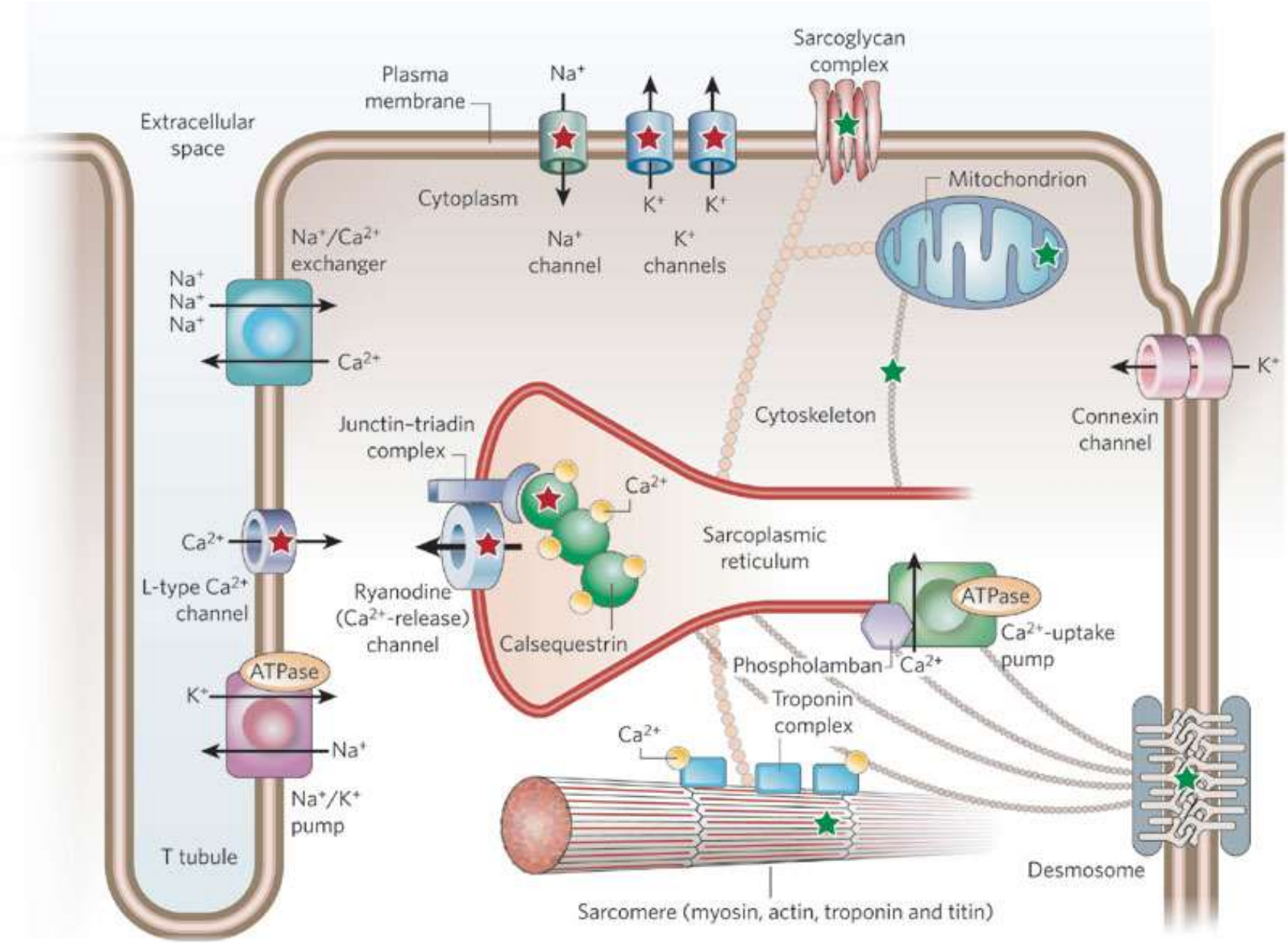
Heart muscle consists of different types of cardiomyocytes:

1) „Fast cells“ of working myocardium that make a contraction as a response to electric signal created in pacemaker cells – most common type

2) „Slow cells“ which participate in conduction through SA and AV node

3) „Pacemaker cells“ that create the electric signal – ability of all the „slow“ cells and some „fast“ cells as well

Connection between two cells is maintained by desmosomes and connexin channels

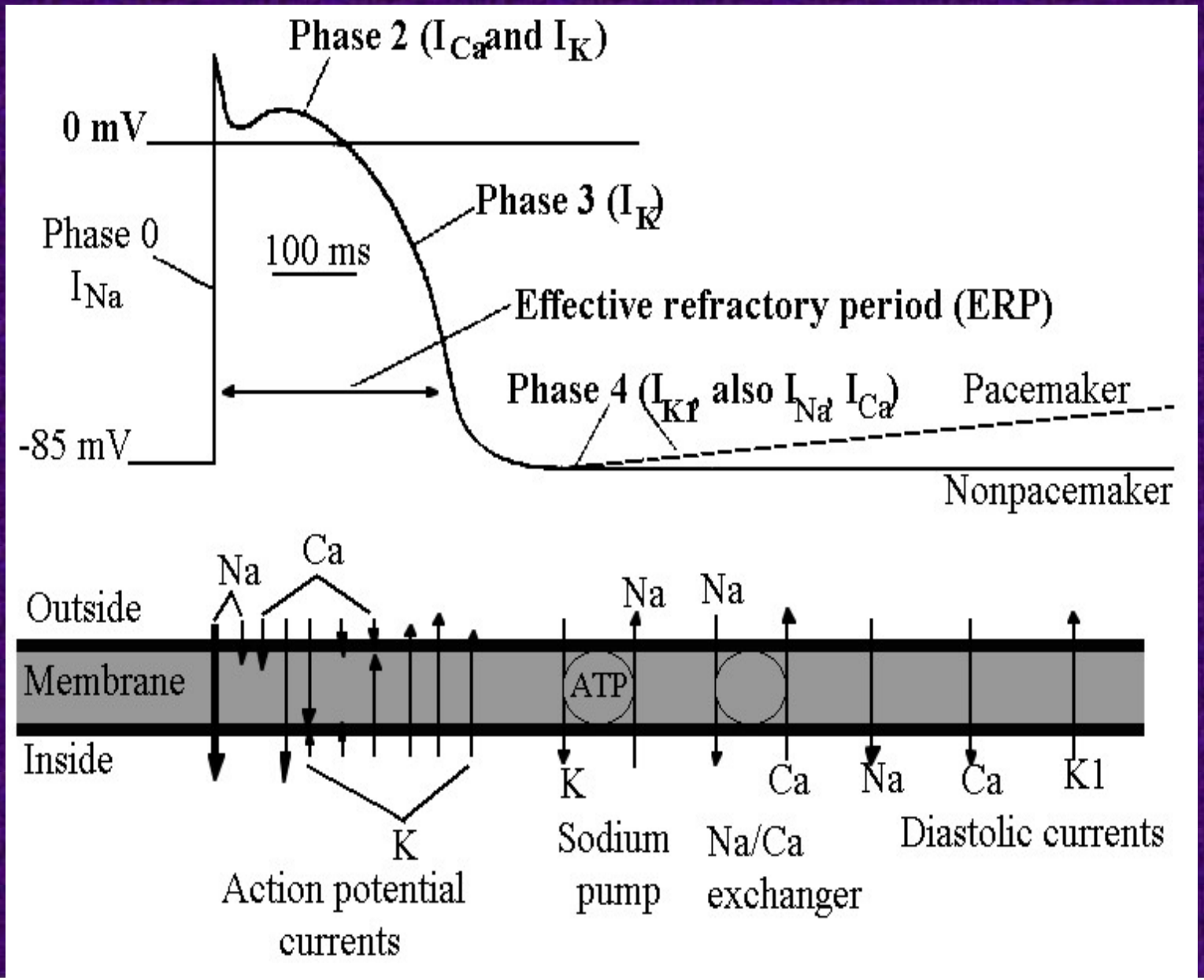

Sarcoglycan complex
Plasma membrane
Na+
Extracellular space
Cytoplasm
K+
K+
Mitochondrion
Na+/Ca2+ exchanger
Na+ channel
K+ channels
Na+
Na+
Na+
Ca2+
K+
Connexin channel
Cytoskeleton
Junctin-triadin complex
Ca2+
Ca2+
Sarcoplasmic reticulum
L-type Ca2+ channel
Ryanodine (Ca2+-release) channel
Calsequestrin
ATPase
Ca2+-uptake pump
Phospholamban
Ca2+
Troponin complex
ATPase
K+
Ca2+
Na+
Na+/K+ pump
T tubule
Desmosome
Sarcomere (myosin, actin, troponin and titin)

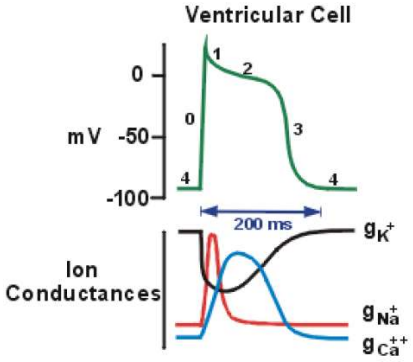
Mechanism of cardiomyocyte activity 1

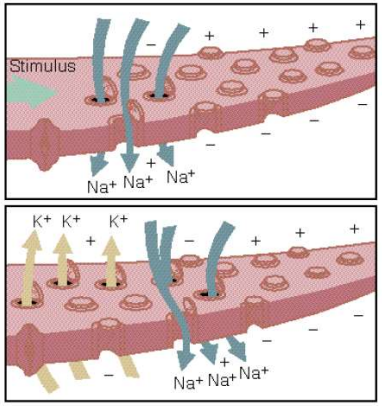
- Three cations present in both extra- and intracelular fluid participate in electrical activity of heart muscle: Na^+, K^+ and Ca^{2+}. Na^+ and Ca^{2+} are present mainly in ECF (Ca^{2+} also in endoplasmic reticulum), K^+ in ICF
- During fast depolarisation of a cardiomyocyte (phase 0), voltage-gated sodium channels (I_{Na}) open at -65 mV. Subsequent influx of Na^+ leads to depolarisation up to +40 mV and closing of Na^+ channels.
- Phase 1 means partial repolarisation carried by diffusion of K^+ through specific ion channels (I_{to} – „transient outward“) K^+ ions diffuse according to both electrical and chemical gradient. In the same time, Ca^{2+} „long-lasting“ ($I_{Ca\text{-}L}$) channels are opened. During phase 0 to 2, heart muscle cell doesn‘t respond to any new electrical signal – absolute refractory period

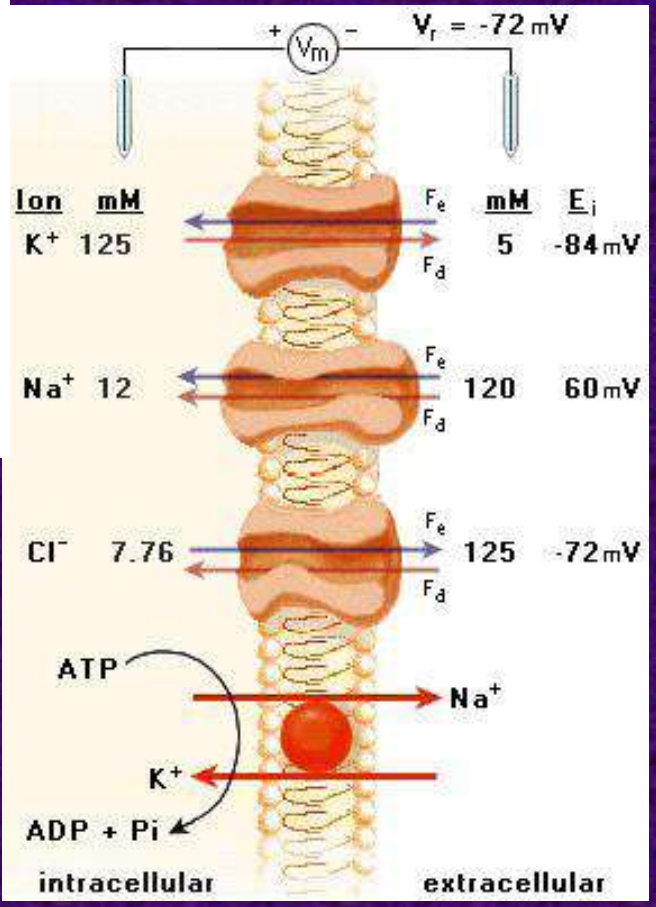
Mechanism of cardiomyocyte activity 2

- In phase 2 („plateau“), prolonged depolarisation is maintained by the influx of Ca^{2+} through $I_{Ca\text{-}L}$ channels. Unlike I_{Na} or I_{to}, $I_{Ca\text{-}L}$ channel is gated both by voltage and receptor mechanism, that responds to vegetative nervous signalisation. Ca^{2+} binds to ryanodin receptor of sarcoplasmic reticulum, where it enhances the release of more Ca^{2+} into the cytoplasm. Ca^{2+} then binds troponin which changes its conformation and stops blocking the actin-myosin interaction. Contraction of muscle fibre follows as in other types of muscles. Another, delayed K^+ channel (I_K) is open.
- Finally, with closing of Ca^{2+} channel, efflux of K^+ lowers the voltage inside the cardiomyocyte to the values during diastole (phase 3)
- Before next repolarisation, Na^+ ions are pumped outside the cell in exchange for K^+ by Na/K ATP-ase (3:2). Some Na^+ ions return inside the cell in change for Ca^{2+} through specific exchanger. Ca^{2+} is also pumped into sarcoplasmic reticulum.The heart muscle gets to diastole


Phase 2 (I_{Ca} and I_K)
0 mV
Phase 3 (I_K)
Phase 0
I_{Na}
100 ms
Effective refractory period (ERP)
Phase 4 (I_{Kf} also I_{Na}, I_{Ca})
Pacemaker
-85 mV
Nonpacemaker
Na
Ca
Outside
Membrane
Inside
ATP
Na
Na
K
Ca
Na
Ca
K1
K
Action potential currents
Sodium pump
Na/Ca exchanger
Diastolic currents


Ventricular Cell
0
mV -50
-100
1
2
0
3
4
4
200 ms
Ion
Conductances
g_{K^+}
g_{Na^+}
$g_{Ca^{++}}$


Stimulus
Na+ Na+ Na+
K+ K+ K+
Na+ Na+ Na+


Vm
V_r = -72 mV
Ion mM
K^+ 125
Na^+ 12
Cl^- 7.76
F_e
F_d
mM E_i
5 -84 mV
120 60 mV
125 -72 mV
ATP
Na^+
K^+
ADP + Pi
intracellular
extracellular

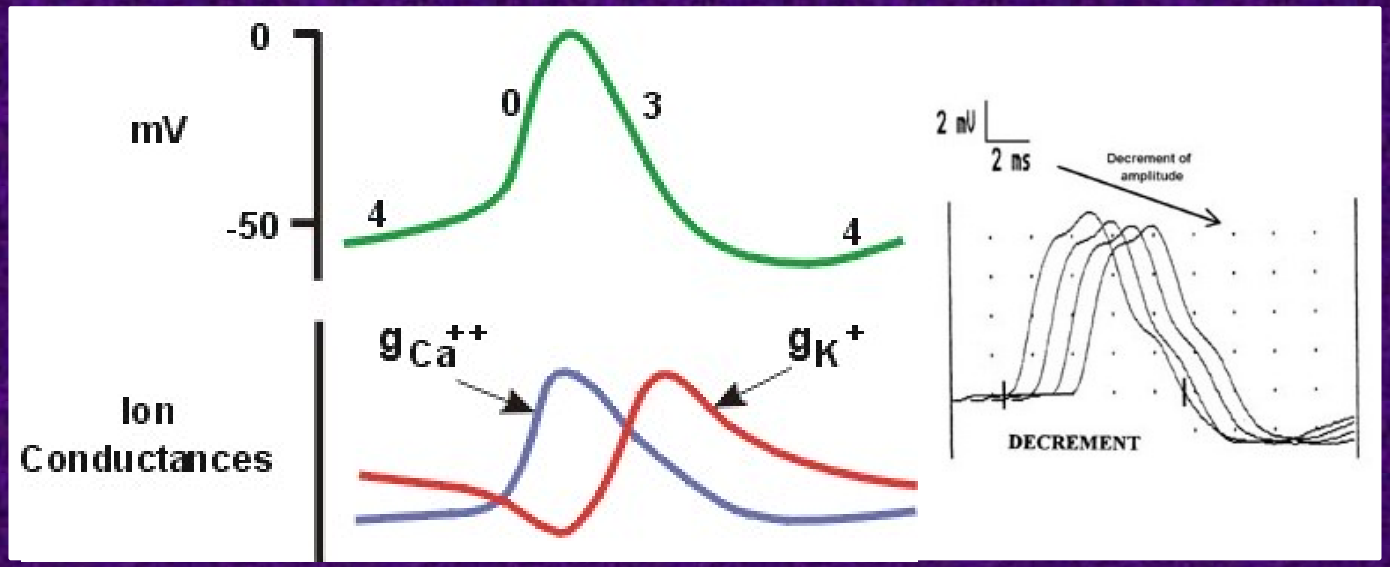
Pacemaker cells

- In pacemaker cells, sympathicus- and parasympathicus-controlled sodium, potassium and calcium channels remain open during the diastole, leading into continual loss of negative voltage up to -65mV, when fast depolarisation begins.
- Pacemaker cells are present in SA node, AV node and Purkinje fibres

Slow cells

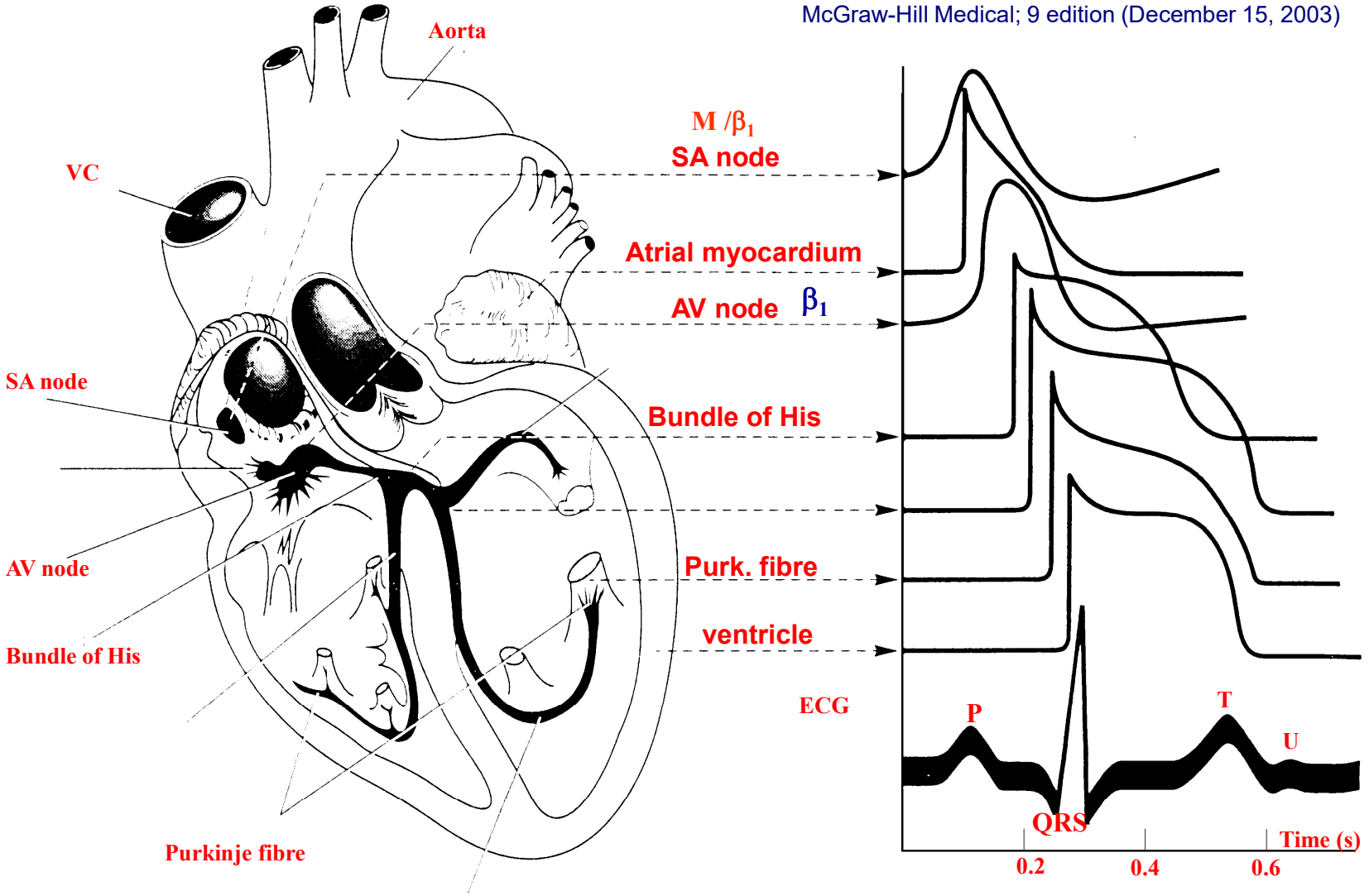
Slow cells do not contain fast sodium channels, their depolarisation is then mediated by Ca2+. The Ca2+ ion channels are influenced by the sympathicus and the parasympathicus.

- Unlike in healthy fast cells, the amplitude of action potentials decreases during consequent depolarization of the slow cells population (decrement)
- Under some circumstances (e.g. repeated stimulation in a short time period), depolarization of the neighbouring cell may become subthreshold, and the depolarization wave stops



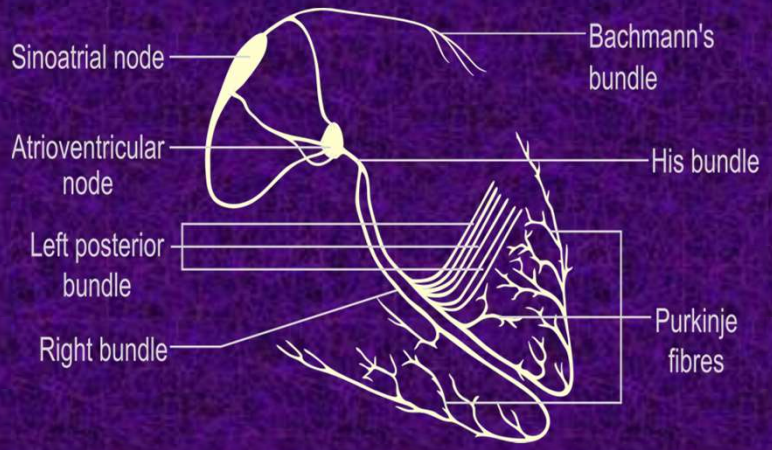
Normal conduction within the heart

According to Katzung's Basic & Clinical Pharmacology.
McGraw-Hill Medical; 9 edition (December 15, 2003)



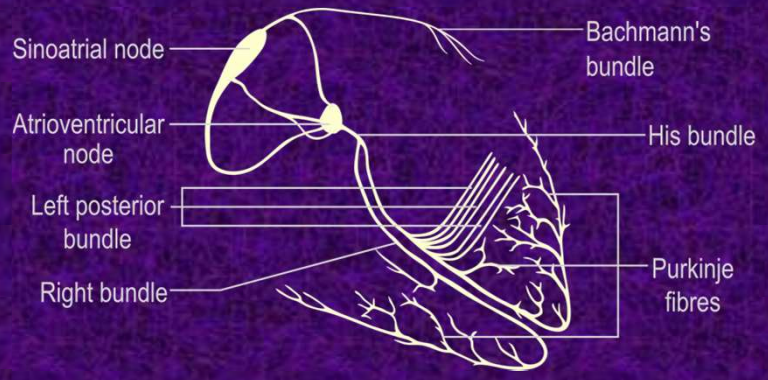
Sinoatrial (SA) node

- Group of pacemaker cells located in the right atrium
- Under normal circumstances it serves as primary pacemaker of the heart
- It spontaneously generates electrical impulses at a rate of 60-90/min
- The SA node is richly innervated by both sympaticus and parasympaticus, which modify the SA node rate and thus heart frequency



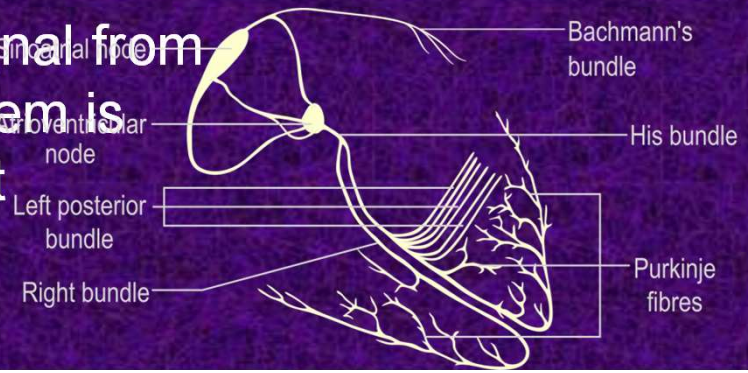
Atrial conduction system

- Bachmann‘s bundle – conducts action potentials to the left atrium
- Internodal tracts (anterior, middle and posterior) – run from SA node to AV node, converging near the coronary sinus. Atrial automacity foci are present within the atrial conduction system



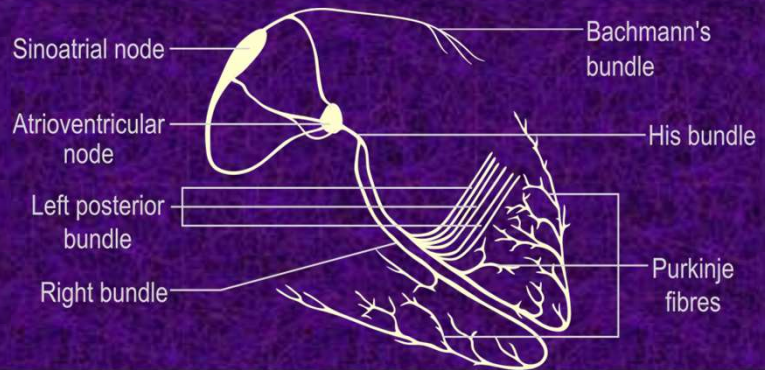
Atrioventricular (AV) node

- Area of specialized tissue located between atria and ventricles, near the coronary sinus and tricuspid valve. It serves as secondary pacemaker and is the only way of electric connection between the atria and the ventricles under normal circumstances.
- AV node consists of 3 zones: AN (atria-nodus), N (nodus) and NH (nodus-His).
- In AN zone, the conduction gets slower, as there is less sodium channels and slower depolarisation
- N zone is formed by nodal cells with low voltage (-50mV) – „slow cells“. In NH zone, the nuber of sodium channels increase again. The cells of NH zone can take over the function of pacemaker, in the case if no signal from upper parts of the conduction system is present. Its rate is slower than that of SA node: 40-60/min



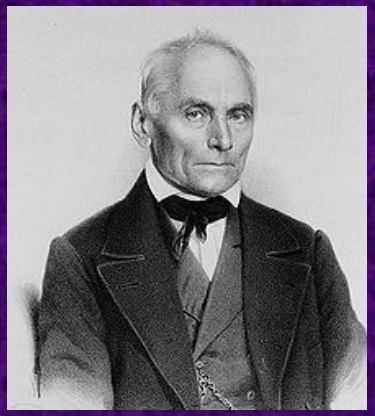
Bundle of His

- Part of cardiac tissue specialized for fast electrical conduction that leads the signal from AV-node to working myocardium of the ventricles.
- After its short course, the Bundle of His branches ito right and left bundle branch (Tawara branches). Right bundle branch is long and thin, thus more vulnerable than the left one
- Left bundle branch is then divided into the left anterior and left posterior fascicle

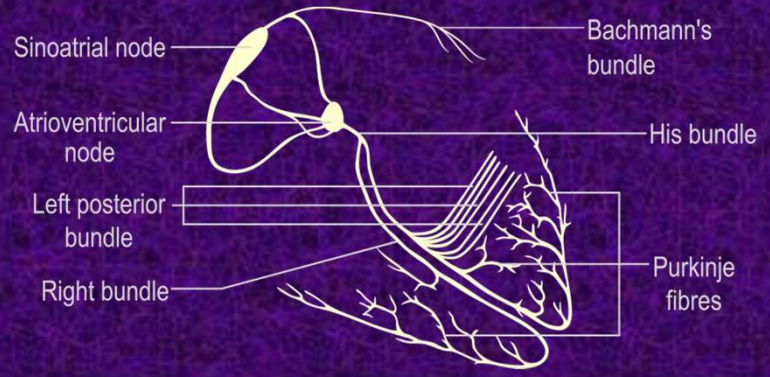


Purkinje fibres

- Terminal part of the conduction system
- Tertiary pacemaker – idioventricular rhythm (20-40/min), without innervation



Jan Evangelista Purkyně (1787-1869),
Czech physiologist



Recording of action potentials

- Action potentials in various parts of the heart can be registered using electrodes
- Besides the electrodes and a recorder, signal amplifier is also a part of the device
- For recording the ECG curve, potential difference between two electrodes is used.
- Thus a dipole with its dipole moment (vector) appears – leading from negatively to positively charged electrode
- The potential difference (i.e., voltage) generates a deviation from the isoelectric line (= condition, when the potential at both electrodes is equal)

Leads

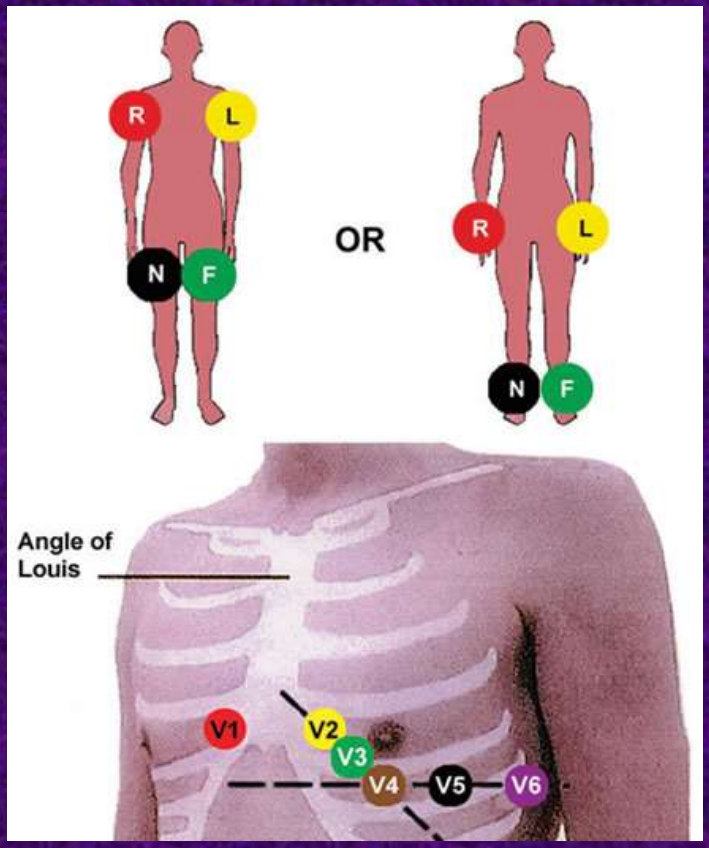
- The direction of the deviation depends on the comparison of the electrode pair (lead), to which a conventional direction is assigned (usually, it points from right, up and back to left, down and forward), and recorded dipole moment vector
- Standardly, 12 leads are defined using 10 electrodes
- Each possible position (lead) matches one ECG curve
- The direction and the magnitude of deviation on the record (for a given lead) depends on:
  A) character of voltage change (depolarization or repolarization
  B) direction of voltage change spreading (in comparison with a dipole moment vector)

12-lead ECG (uses 10 electrodes)

Electrode placement:

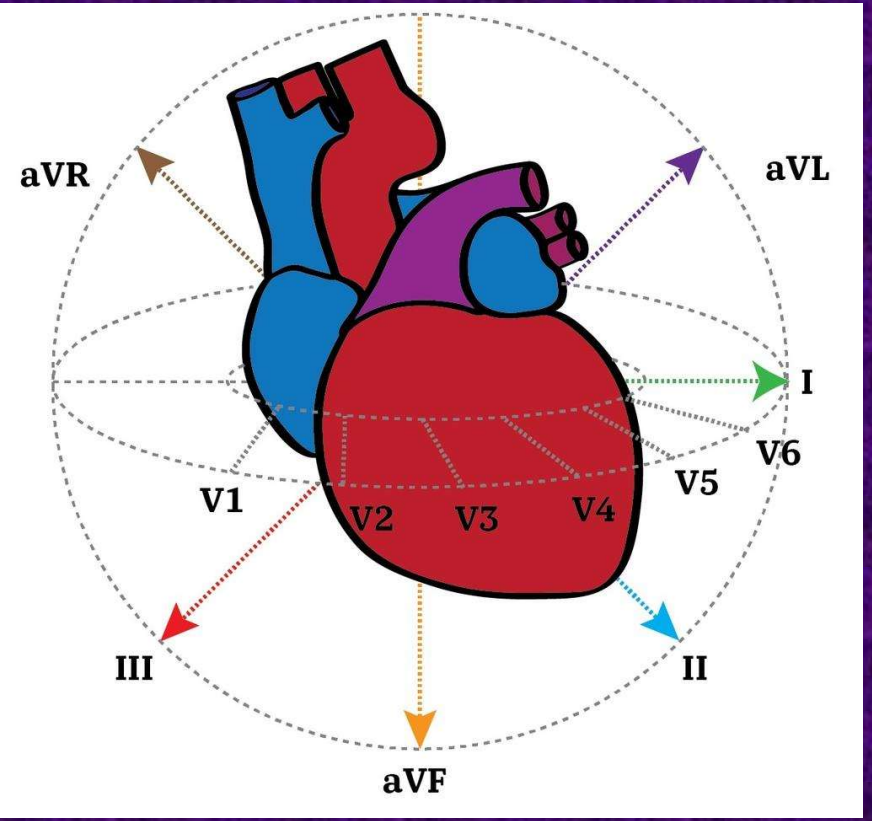
RA: On the right arm, avoiding bony prominences.
LA: In the same location that RA was placed, but on the left arm this time.
RL: On the right leg, avoiding bony prominences – it‘s used as a ground.
LL: In the same location that RL was placed, but on the left leg this time.
V1: In the *fourth* intercostal space (between ribs 4 & 5) just to the *right* of the sternum (breastbone).
V2: In the *fourth* intercostal space (between ribs 4 & 5) just to the *left* of the sternum.
V3: Between leads V2 and V4.
V4: In the fifth intercostal space (between ribs 5 & 6) in the mid-clavicular line (the imaginary line that extends down from the midpoint of the clavicle (collarbone).
V5: Horizontally even with V4, but in the anterior axillary line. (The anterior axillary line is the imaginary line that runs down from the point midway between the middle of the clavicle and the lateral end of the clavicle; the lateral end of the collarbone is the end closer to the arm.)
V6: Horizontally even with V4 and V5 in the midaxillary line. (The midaxillary line is the imaginary line that extends down from the middle of the patient’s armpit.)

12-lead ECG – electrode placement

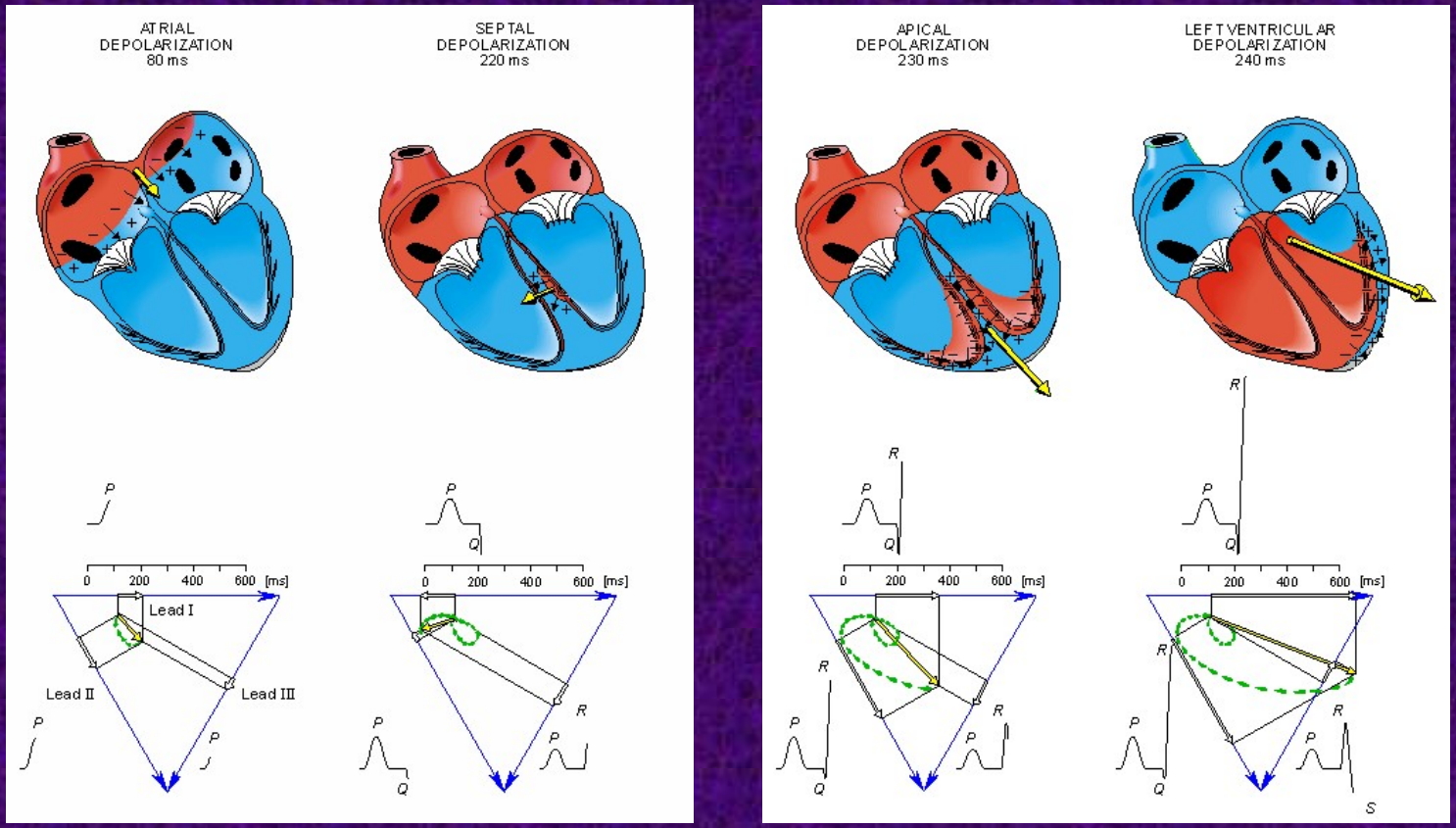


Orientation of the leads

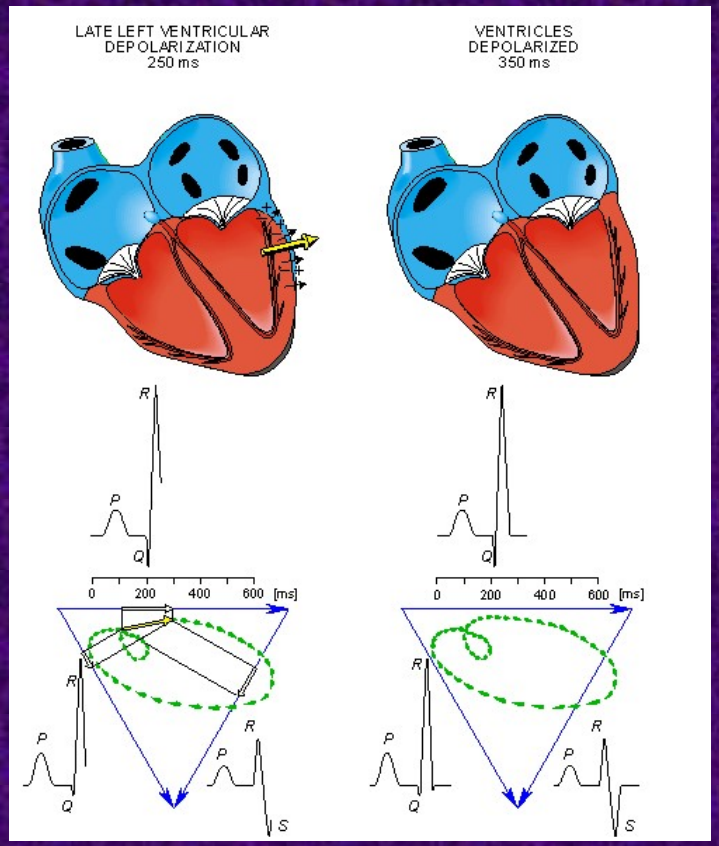
- Einthoven‘s leads
  - A reference electrode is on one limb, an active one on another
- Wilson‘s leads
  - Reference electrode is constructed as the Wilson‘s terminal, an active one is placed on the chest
- Goldberger‘s leads
  - One electrode is detached from the Wilson‘s terminal and serves as an active one, the remaining two together as a reference one

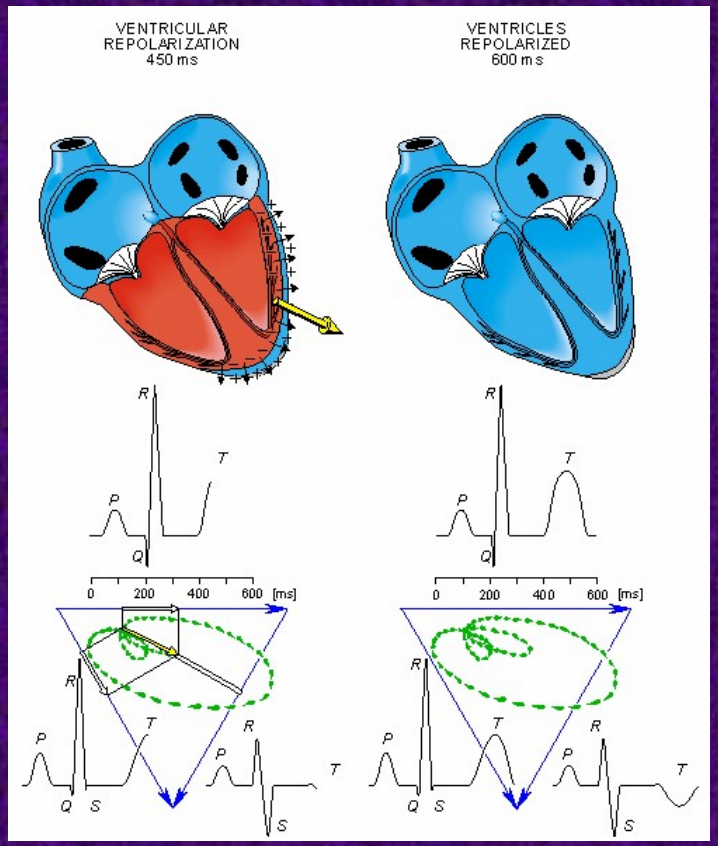


Electric potential changes – summation of vectors (ECG curve in the leads I, II, III)

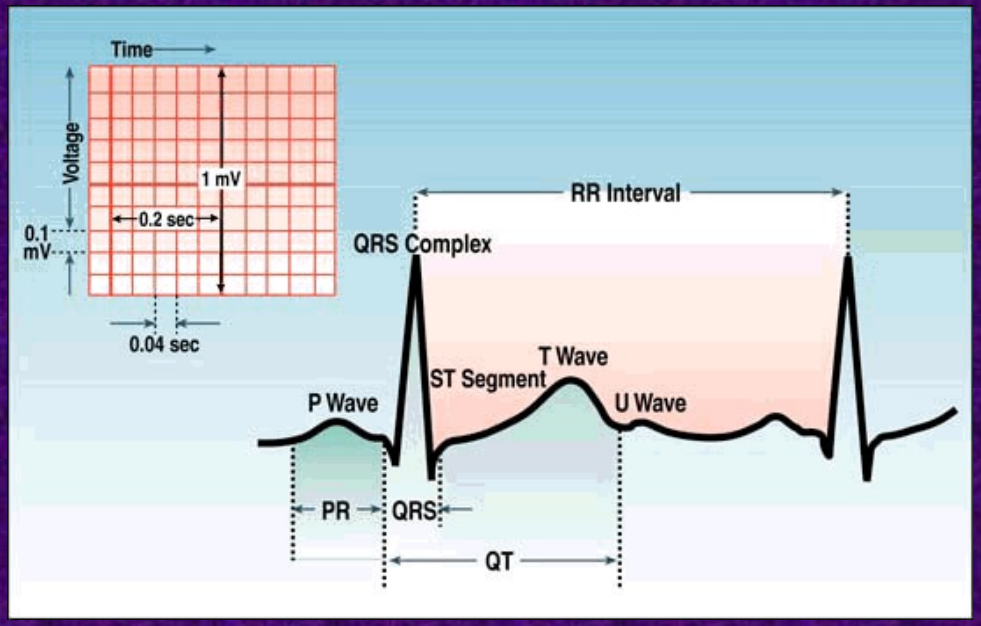


Summation of vectors (ECG curve in the leads I, II, III)





Normal ECG curve



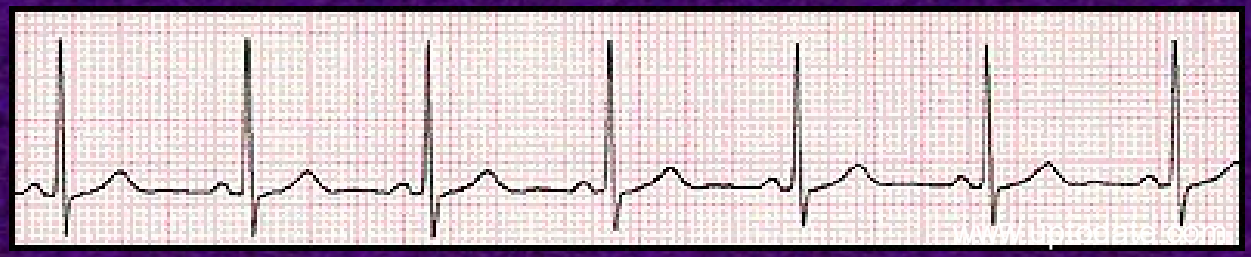
Description of ECG

- rhythm
- sinus
  - 60-90/min
  - other
- junctional
  - 40-60/min
- idioventricular
  - 30-40/min
- atrial fibrilation
- atrial flutter
- ventricular tachycardia

- heart rate
- normal
- 60 – 100/min
- tachycardia
- >100/min
- bradycardia
- <60/min

- description of waves and intervals
- electrical axis of the heart

Normal Sinus Rhythm

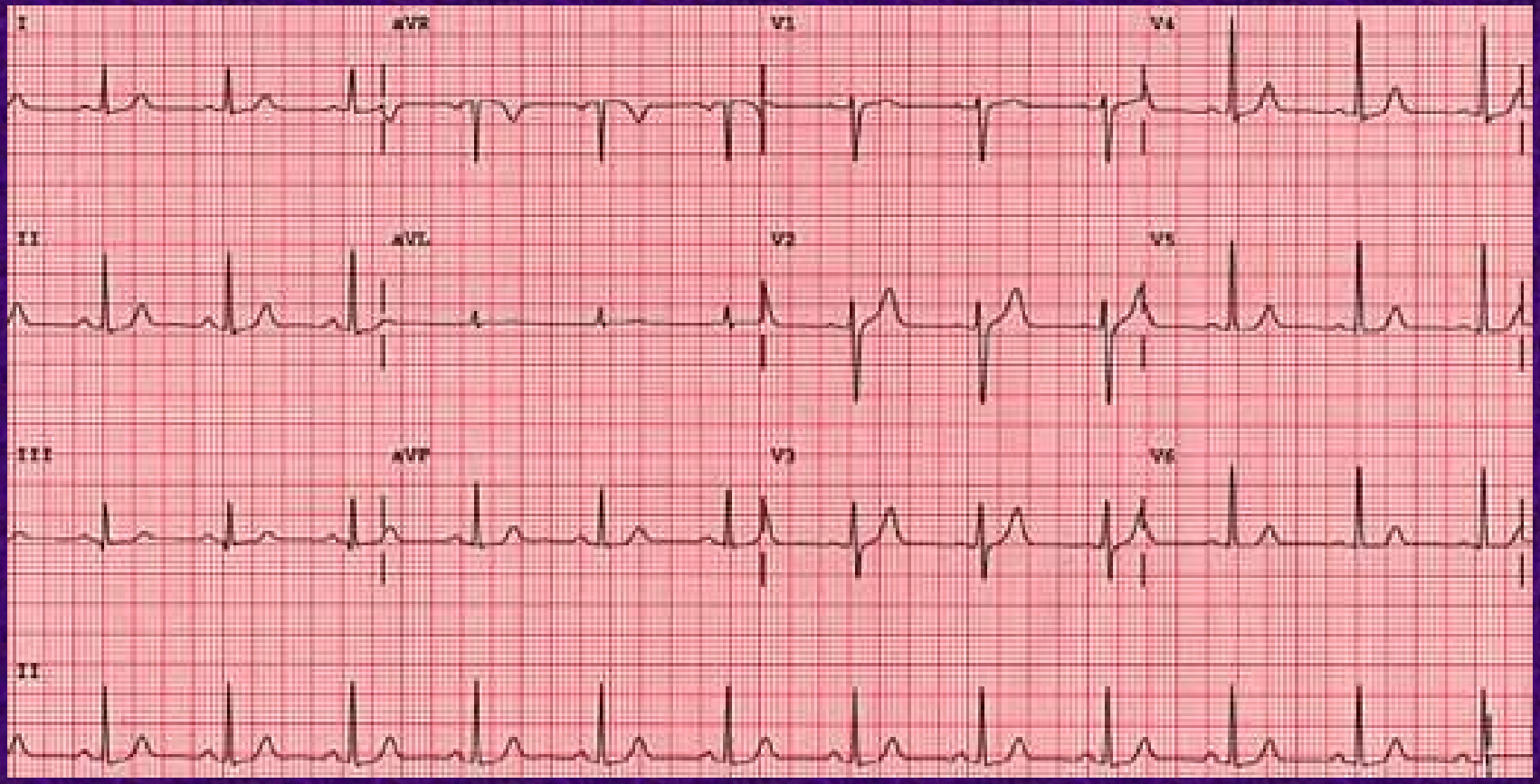


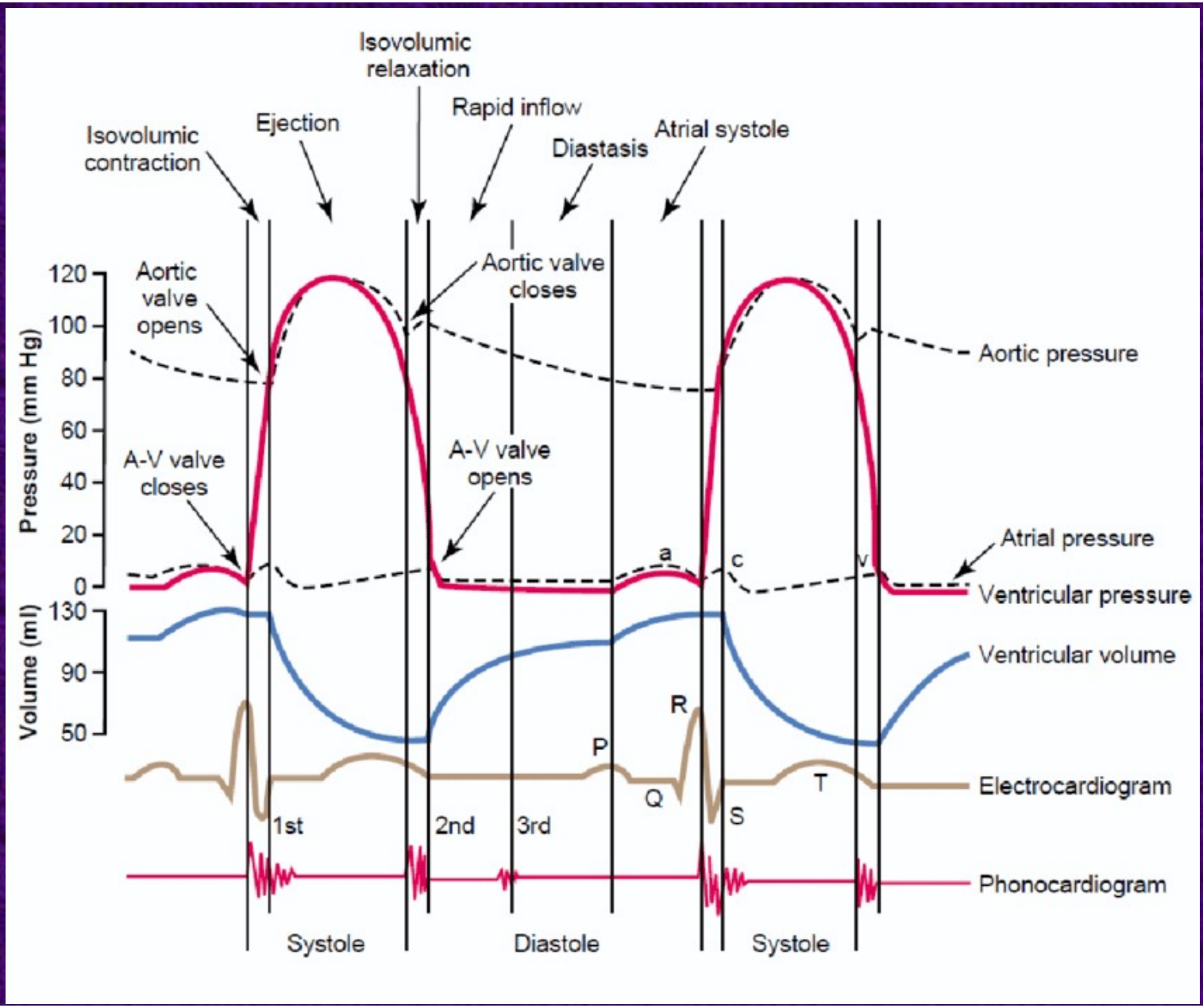
Implies normal sequence of conduction, originating in the sinus node and proceeding to the ventricles via the AV node and His-Purkinje system.

ECG Characteristics:

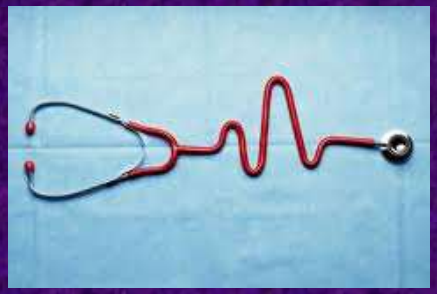
- Regular narrow-complex rhythm
- Rate 60-100 bpm
- Each QRS complex is preceded by a P wave
- P wave is upright in lead II & downgoing in lead aVR

Normal 12-lead ECG




Isovolumic relaxation
Rapid inflow
Ejection
Isovolumic contraction
Diastasis
Atrial systole
Aortic valve closes
Aortic valve opens
Aortic pressure
A-V valve opens
A-V valve closes
Atrial pressure
Ventricular pressure
Ventricular volume
Pressure (mm Hg)
120
100
80
60
40
20
0
Volume (ml)
130
90
50
a
c
v
R
P
Q
S
T
Electrocardiogram
1st
2nd
3rd
Phonocardiogram
Systole
Diastole
Systole

Arrhythmias:



- Electrophysiological abnormalities arising from the impairment of the electric signal
- Arrhythmias are defined by exclusion - i.e., any rhythm that is not a normal sinus rhythm is an arrhythmia

- With respect to the
  - Frequency – bradyarrythmias vs. tachyarrhythmias
  - Localization – supraventricular (SV), ventricular (V)
  - Mechanism – abnormal signal formation / conduction

Possible causes of arrhytmia

- Myocardial ischaemia, hypoxia and reperfusion, pH disorders
- Disorders of myocardium – hypertrophy, dilatation, amyloidosis, scar after acute myocardial infarction
- Inflammation (myocarditis)
- Aberrant conduction – pre-excitation syndromes
- Vegetative nervous system disorder (nervous lability, compensation of heart failure, shock, anxiety)
- Disorders of ion balance
- Drugs (β-blockers, digitalis, antiarrhytmics)
- General state (trauma, endokrinopathy..)
- Genetic causes (ion channel mutations)

Brady- and tachyarrhythmias:

1. Bradyarrhythmias

- SA block
- sick-sinus syndrome
- AV block

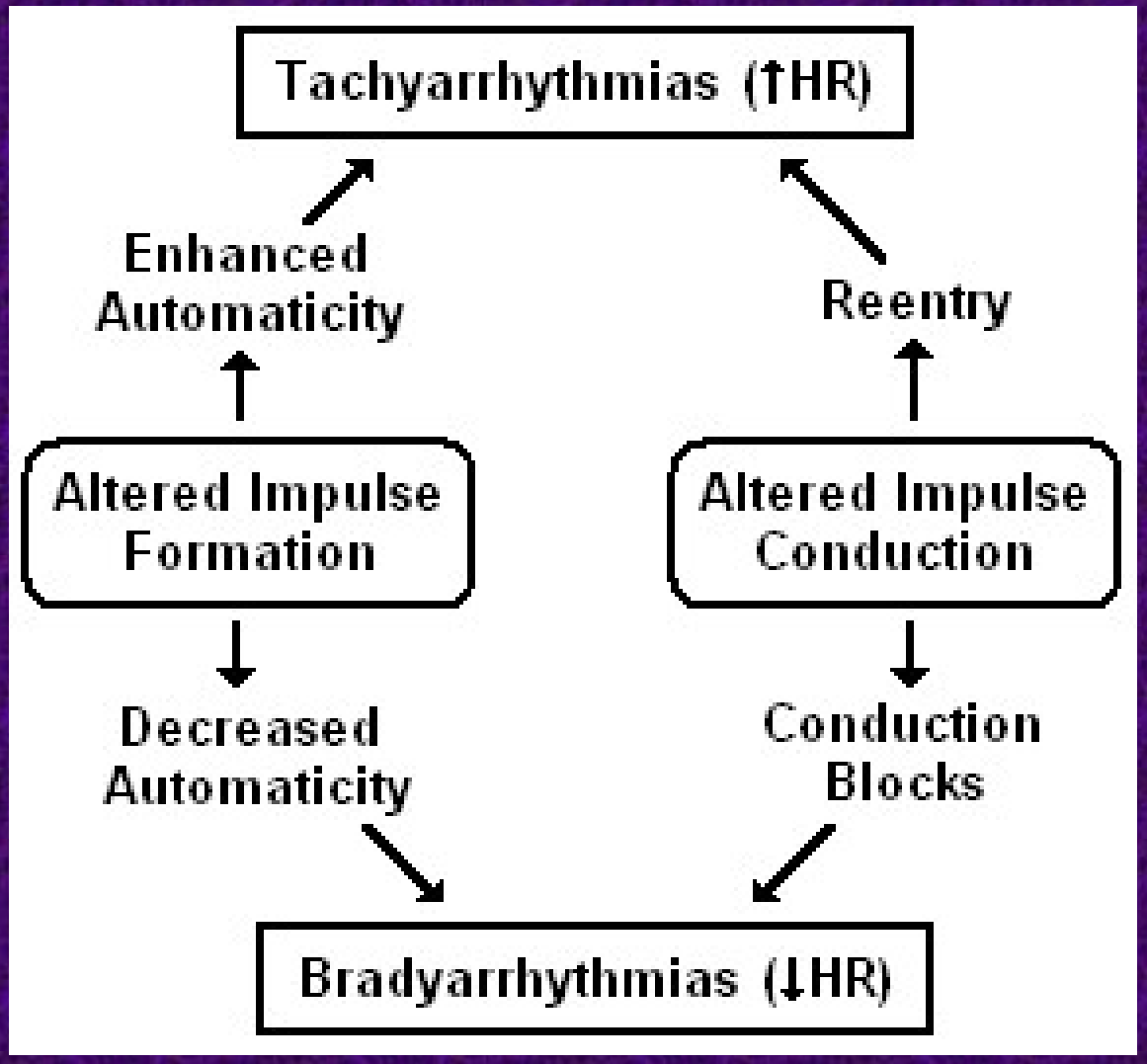
2. Tachyarrhythmias

a) Supraventricular (SV)

- SV premature beat – atrial, junction
- atrial tachycardia, flutter, fibrillation
- AV node re-entry tachycardia (AVNRT)
- AV re-entry tachycardia (Wolf-Parkinson-White syndrome)

b) Ventricular

- ventricular extrasystoles
- ventricular tachycardia
- flutter/fibrillation…


Tachyarrhythmias (↑HR)
Enhanced Automaticity
Reentry
Altered Impulse Formation
Altered Impulse Conduction
Decreased Automaticity
Conduction Blocks
Bradyarrhythmias (↓HR)

Mechanism of Arrhythmia

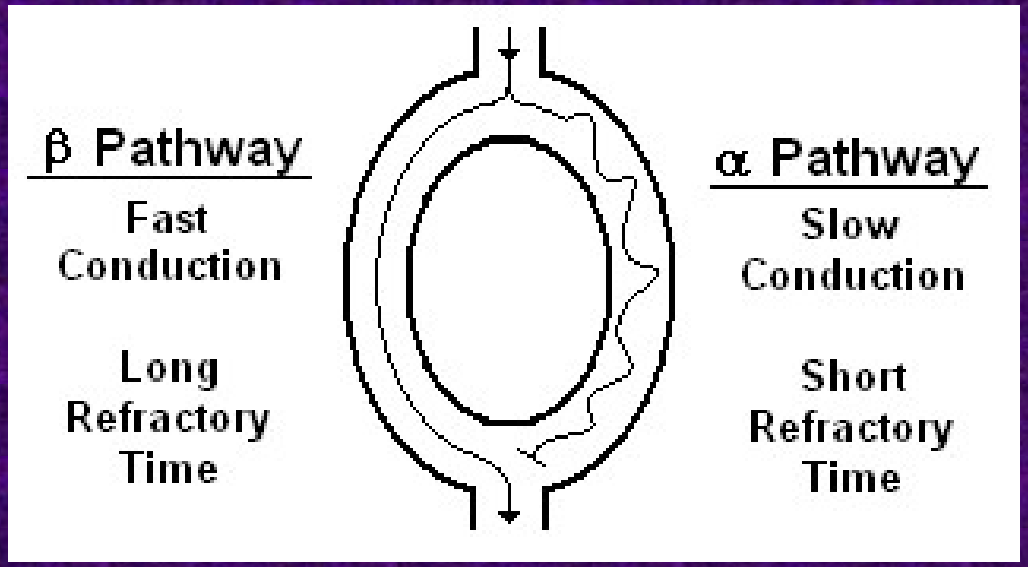
1. **Pacemaker activity disorder**
   (enhanced, depressed, ectopic)
2. **Re-entry**
3. **Triggered activity**
   (early afterdepolarisations, delayed afterdepolarisations)
4. **Conduct block**

Recognizing altered automaticity on ECG

Gradual onset and termination of the arrhythmia.

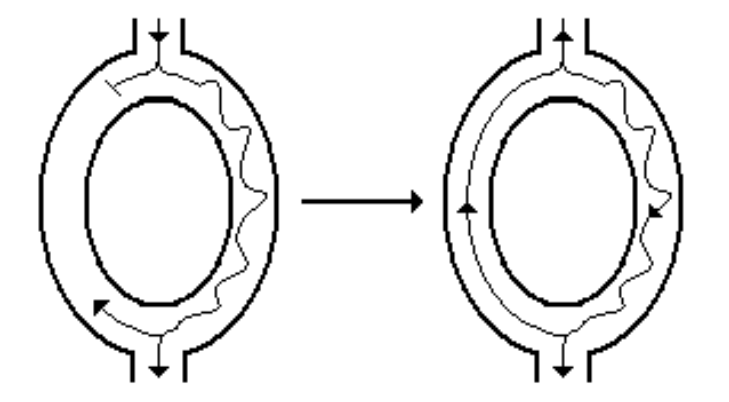
The P wave of the first beat of the arrhythmia is typically the same as the remaining beats of the arrhythmia (if a P wave is present at all).

Mechanism of Re-entry



- The two pathways may be defined both morphologically and functionally
- Substrate:
  - different conduction velocities
  - different refraktory periods
  - unidirectional blocks
  - premature depolarizations

Mechanism of Re-entry 2



- Prior to re-entry formation, new electric impulse is required immediately after the preceding one (e.g. ectopic pacemaker)
- Only the α pathway is depolarized, β is in the refractory period
- If the conduction through α pathway is slow, the signal gets to the second “crossroads“ just after the cells of β pathway are ready for a new depolarisation
- Signal might then return through the β pathway (backwards). A re-entry circuit is formed which depolarizes the surrounding tissue – a high-frequency ectopic focus is formed
- Signal in the re-entry circuit goes down through the slow pathway (!)

Recognizing reentry on ECG

Abrupt onset and termination of the arrhythmia.

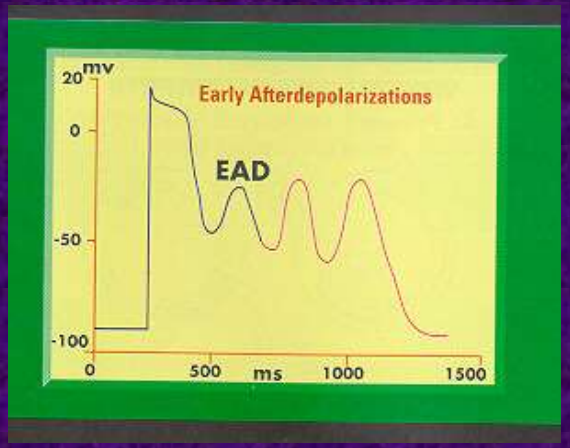
The P wave of the first beat of the arrhythmia is different from the remaining beats of the arrhythmia (if a P wave is present at all).

Reentrant Rhythms

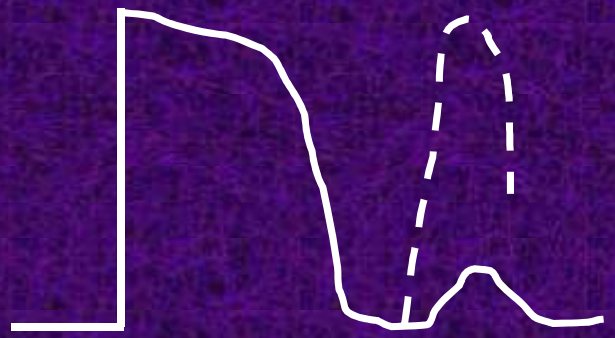
- AV nodal reentrant tachycardia (AVNRT)
- AV reentrant tachycardia (AVRT)
  - Orthodromic
  - Antidromic
- Atrial flutter
- Atrial fibrillation
- Ventricular tachycardia

Triggered activity

- Early afterdepolarization (EAD)
- Under certain circumstances, the action potential can be much longer than absolute refractory period (usually factors lengthening the phase 3 – hypokalemia, genetic causes, some drugs) → long QT
- Such cells can be easily depolarized by local currents
- Their depolarization then induce the depolarization wave in the whole myocardium

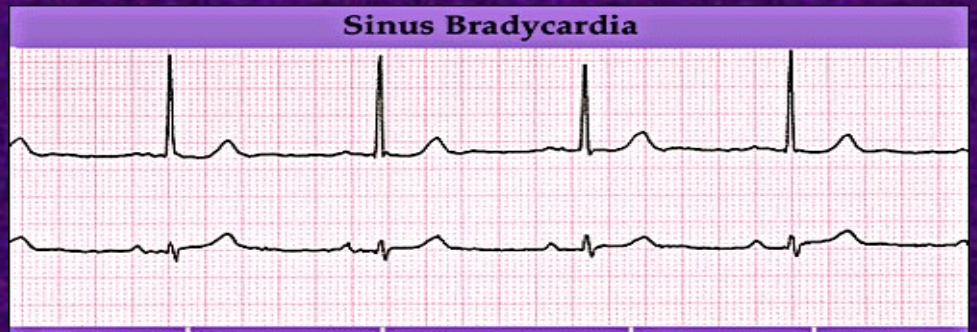


Delayed afterdepolarization (DAD)



- Whenever there is high level of Ca^{2+} in the cytoplasm, ryanodin receptors might be activated, which leads into spilling of Ca^{2+} ions from sarcoplasmic reticulum into the cytoplasm
- The Ca^{2+} ions are then exchanged for Na^{+} via their exchanger in a ratio 1:3 (positive charge 2:3)
- This leads into lower membrane voltage, which can result in the opening of Na^{+} channels and new depolarization

Sinus bradycardia

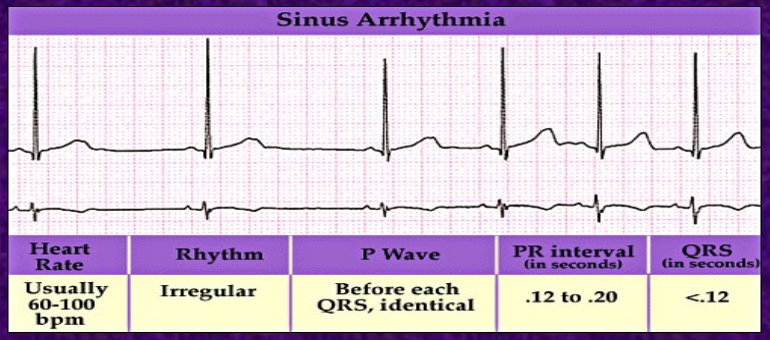


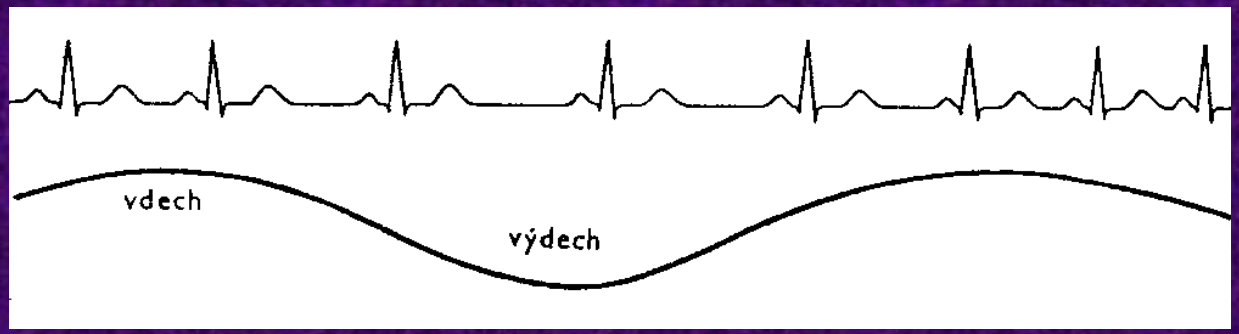
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
<60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

- HR< 60 bpm; every QRS narrow, preceded by p wave
- Can be normal in well-conditioned athletes
- HR can be 30 bpm in children, young adults during sleep, with up to 2 sec pauses

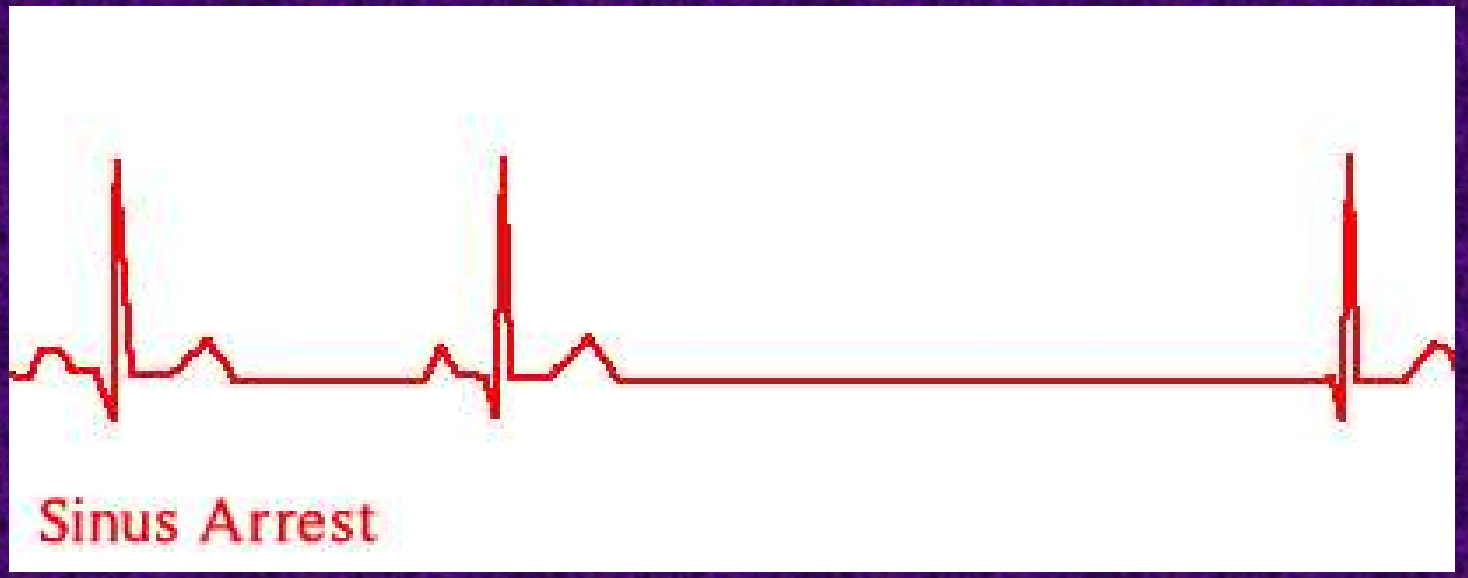
Sinus arrhythmia

- Usually respiratory--Increase in heart rate during inspiration
- Exaggerated in children, young adults and athletes—decreases with age
- Usually asymptomatic, no treatment or referral
- Can be non-respiratory, often in normal or diseased heart, seen in digitalis toxicity
- Referral may be necessary if not clearly respiratory, history of heart disease





SA arrest with compensatory AV activity

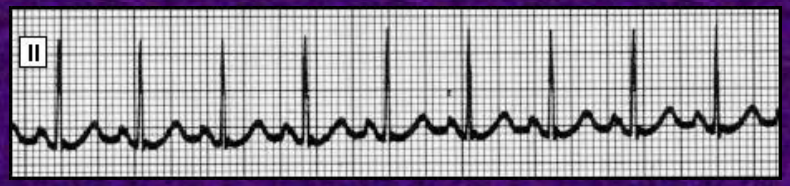


When the activity of SA node is stopped, AV node takes over the role of pacemaker

If the pause is > 3 sec, it can lead into syncope

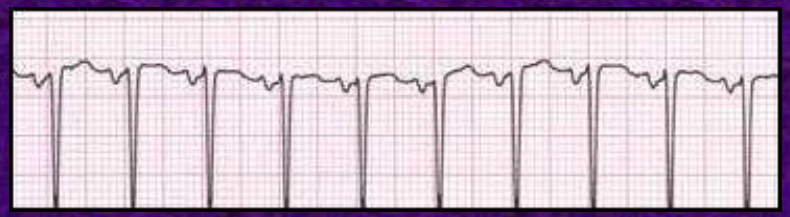
Very similar type of arrhythmia is SA block: pacing in SA node is generated, but not conducted to the myocardium

Increased/Abnormal Automaticity

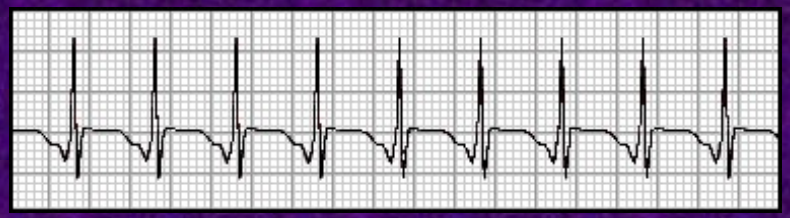


Sinus tachycardia (normal sequence and shape of P and QRS)

- stress reaction, fever, physical effort, hyperthyreoidism, reaction to hypotension



Ectopic atrial tachycardia (P wave is bizzare)

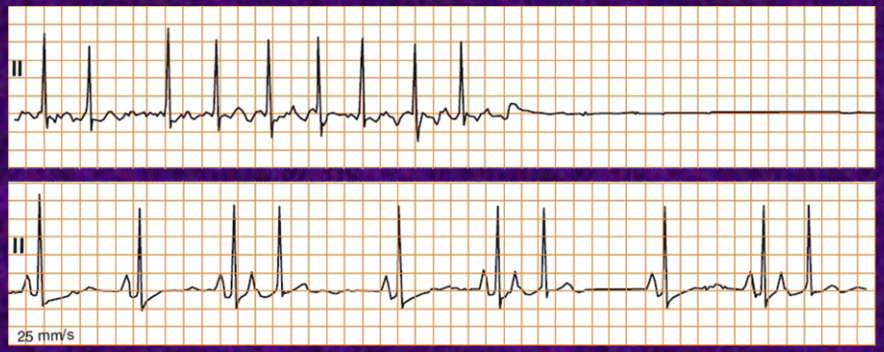


Junctional tachycardia (P wave either follows QRS, or is hidden behind it, or goes immediately before QRS and is bizzare)

www.uptodate.com

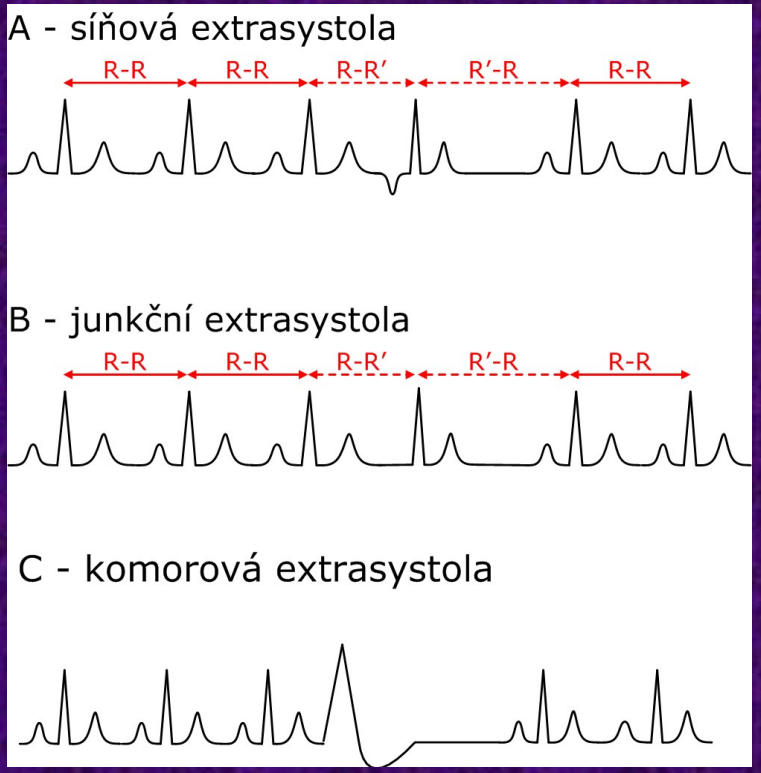
Sick Sinus Syndrome

- Altered function of SA node in older age (fibrosis) or following ischemia
- Other causes: amyloidosis, hypothyreoidism, hypothermia, medication
- Manifests by alternating types of arrhythmia:
  - Sinus bradycardia
  - Wandering pacemaker (ectopic atrial foci)
  - SA arrest or SA block
  - atrial fibriation (brady-tachy) or flutter

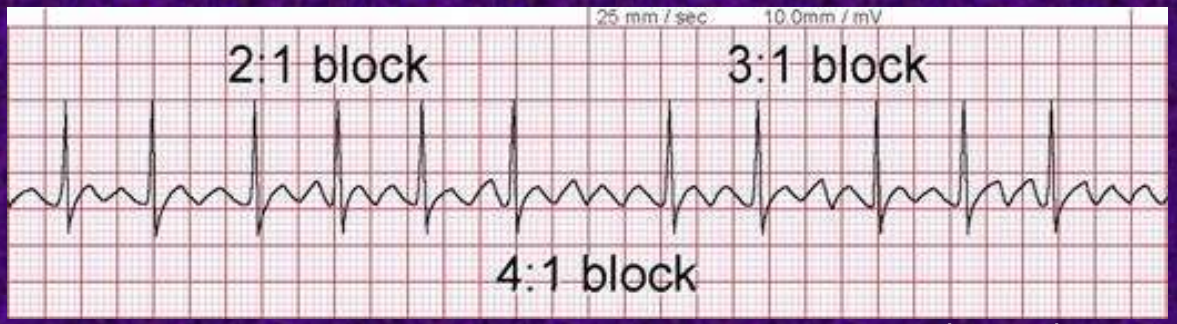


Supraventricular premature beats (supraventricular extrasystoles)

- Narrow QRS complexes.
- In case of premature atrial contractions (PAC), the origin is an ectopic focus in the atria. The QRS is preceded by a bizarre P wave
- In case of premature junctional contraction (PJC), P wave is usually hidden behind the narrow QRS
- Both ectopic automaticity and microre-entry can lead into PAC and PJC
- Supreventricular extrasystoles are usually benign, often present in healthy people



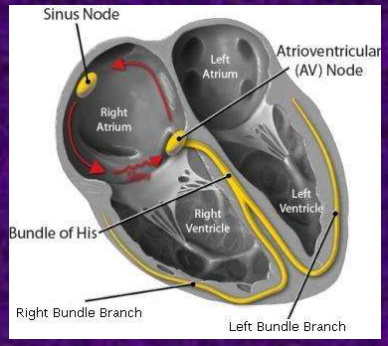
Atrial Flutter



www.spencerknoxmd.com

Most cases of atrial flutter are caused by a large re-entrant circuit in the wall of the right atrium („typical flutter“ – see below)

ECG Characteristics:



Biphasic “sawtooth” flutter waves at a rate of ~ 300 bpm

Flutter waves have constant amplitude, duration, and morphology through the cardiac cycle

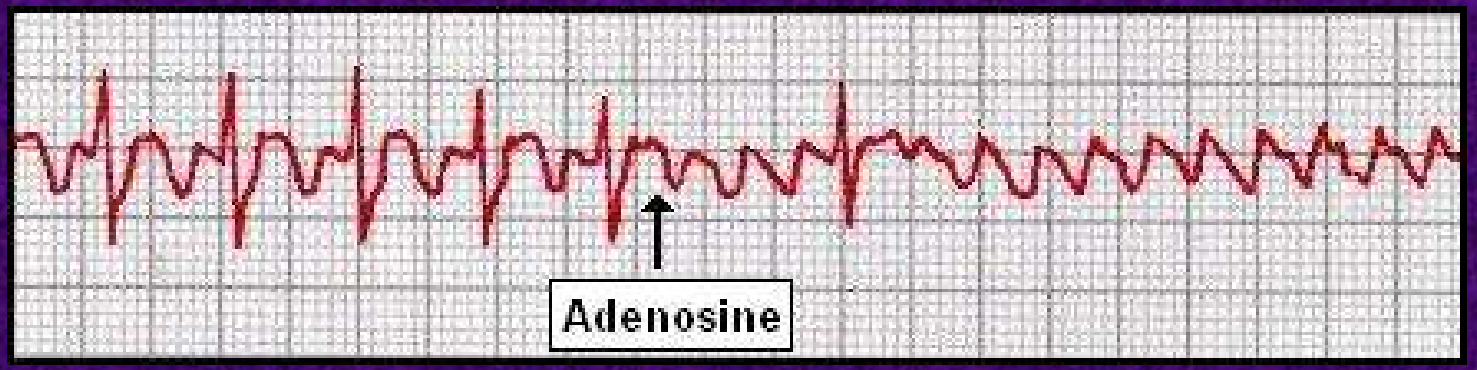
There is usually either a 2:1 or 4:1 block at the AV node

(thanks to its decrement), resulting in

ventricular rates of either 150 or 75 bpm – regular

rhythm (1:1 AV conduction→ circulatory failure)

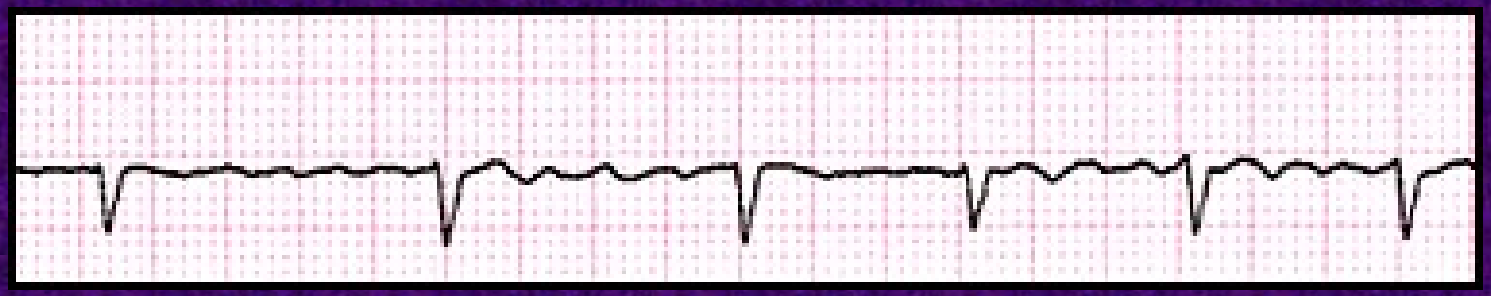
Unmasking of Flutter Waves



Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 7th ed., 2005.

In the presence of 2:1 AV block, the flutter waves may not be immediately apparent. These can be brought out by administration of adenosine (increases the K^+ efflux, which leads into hyperpolarization and disabling the AV node depolarization).

Atrial Fibrillation

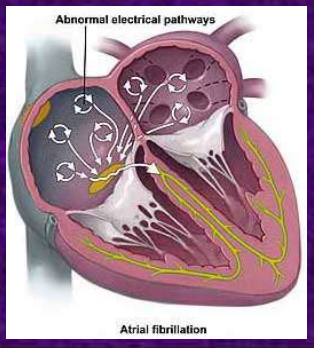


www.uptodate.com

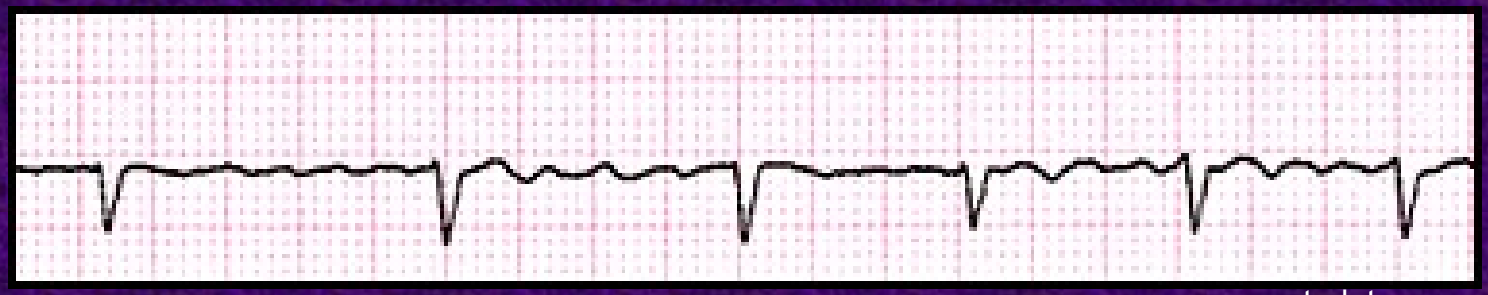
Atrial fibrillation is caused by numerous wavelets of depolarization spreading throughout the atria simultaneously, leading to an absence of coordinated atrial contraction.

Mild tachycardia usually < 150 bpm, when controlled by rate-slowing treatment, it may oscillate between tachy- and bradycardia (tachy-brady syndrome)

This kind of rhythm is present in up to 5% of adult population, mostly in older age. It is often connected with other diseases of the heart (ischaemic haert disease, heart failure, valvular disease.



Atrial Fibrillation



www.uptodate.com

ECG Characteristics:

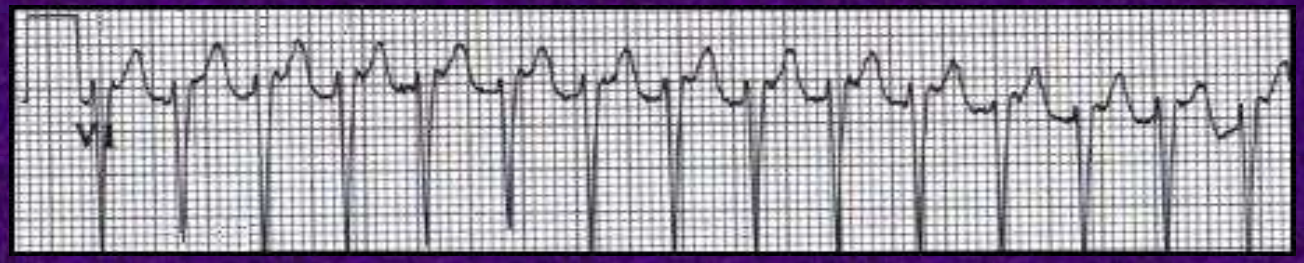
- Absent P waves
- Presence of fine “fibrillatory” waves which vary in amplitude and morphology
- Irregularly irregular ventricular response

Complications:

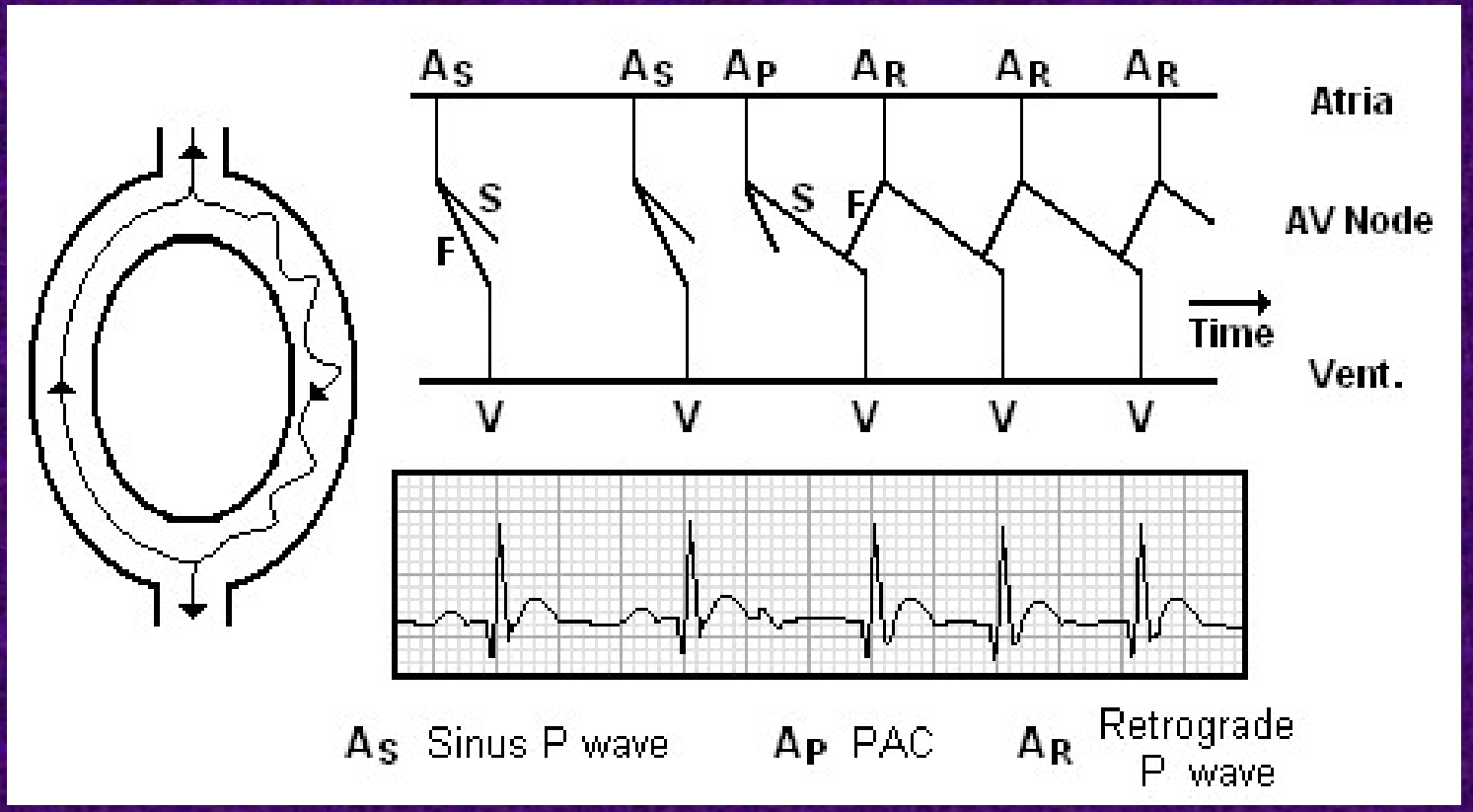
- Missing atrial systole (may constitute a problem in failing heart
- Systemic embolization (thrombus is usually localized in the left atrial appendage)
- Symptoms of tachycardia

AV nodal re-entry tachycardia (AVNRT)

• Most common regular supraventricular tachyarytmia, indistinguishable from escaped junctional rhythm (enhanced AV automaticity), AVNRT has usually higher heart rate (>140/min).
• AVNRT is based on re-entry between slower and faster pathways in the AV node.



Mechanism of AVNRT

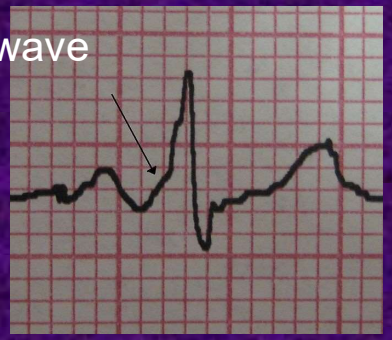


AV re-entry tachycardia (AVRT)

- Re-entry between AV node and an accessory pathway
- There are several type of accessory pathway

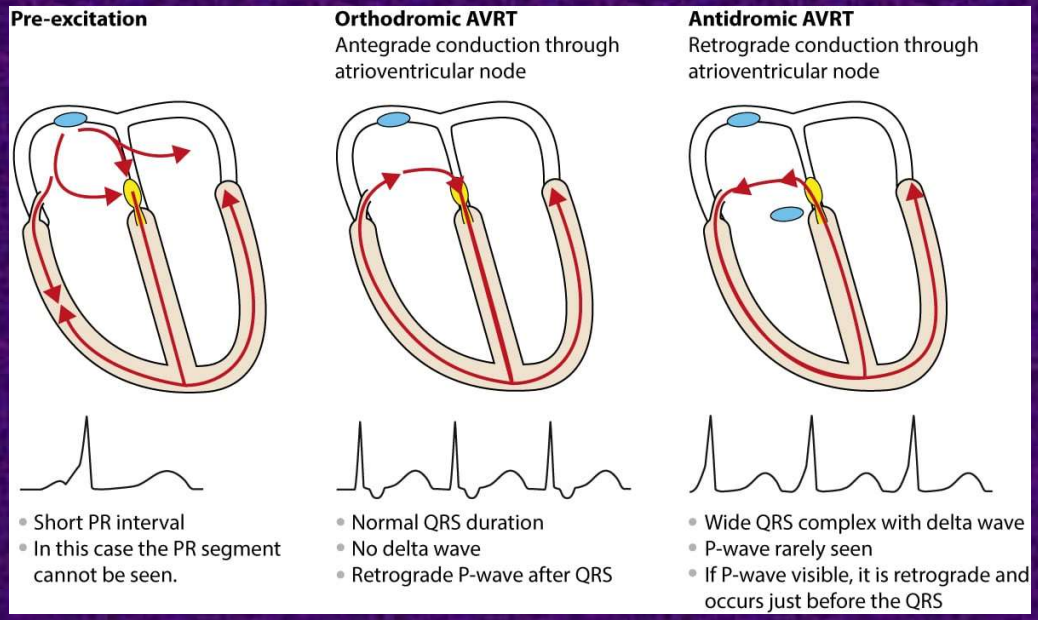
1. Kent: adjacent atrial and ventricular
2. Mahaim: adjacent lower part of the AVN and ventricular (with decrement)

- Usually no structure heart disease, occur in any age individual
- Pre-excitation of ventricular myocardium manifests by δ-wave



AV re-entry tachycardia (AVRT)

- AVRT can be orthodromic (i.e. antegrade conduction through AV node – more often): δ-wave appears during the steady state but disappears during the arrhythmia; or antidromic (i.e. antegrade conduction through accessory pathway): δ-wave is present during the arhythmia

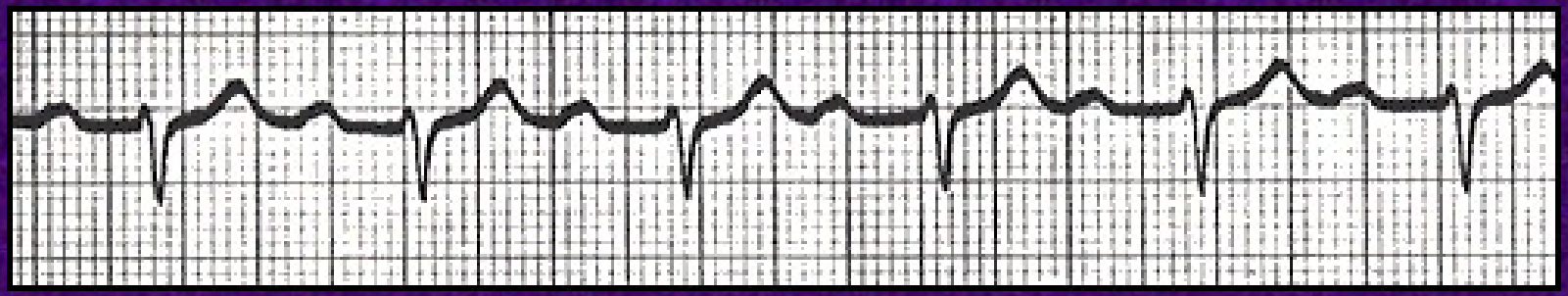


Wolff-Parkinson-White Syndrome (WPW) is a condition in which the heart beats too fast due to abnormal, extra electrical pathway between the heart's atrium and ventricle – bundle of Kent (orthodromic AVRT).

Atrioventricular Block

- AV block is a delay or failure in transmission of the cardiac impulse from atrium to ventricle.
- Three degrees:
  - 1) all the depolarization waves are transmitted to ventricles, but with a delay
  - 2) some signals are transmitted, some not
  - 3) no signals are transmitted

1st Degree AV Block

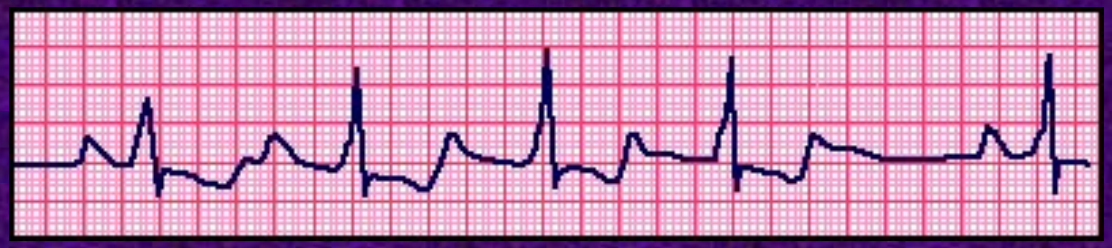


The Alan E. Lindsay ECG Learning Center ; http://medstat.med.utah.edu/kw/ecg/

ECG Characteristics: Prolongation of the PR interval (over 0.22s), which is constant

All P waves are conducted

2nd Degree AV Block

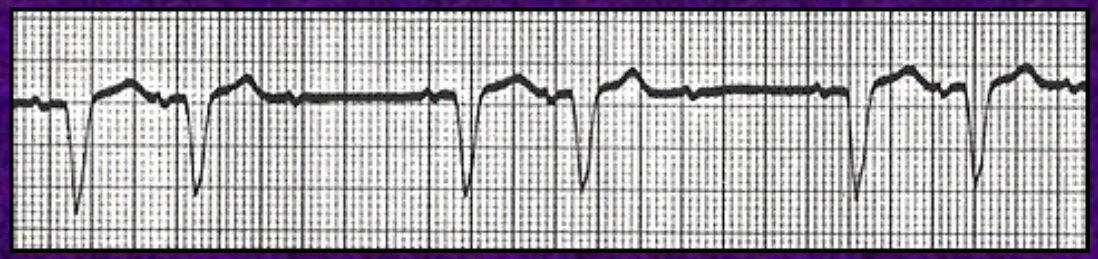


Mobitz 1
(Wenckebach)

ECG Characteristics: Progressive prolongation of the PR interval until a P wave is not conducted (increasing decrement)

As the PR interval prolongs, the RR interval actually shortens

Usually caused by vegetative dysbalance



Mobitz 2

(Hay; or only Mobitz)

ECG Characteristics: Constant PR interval with intermittent failure to conduct

Usually caused by structural disease

3rd Degree (Complete) AV Block

ECG Characteristics:

No relationship between P waves and QRS complexes

Relatively constant PP intervals and RR intervals

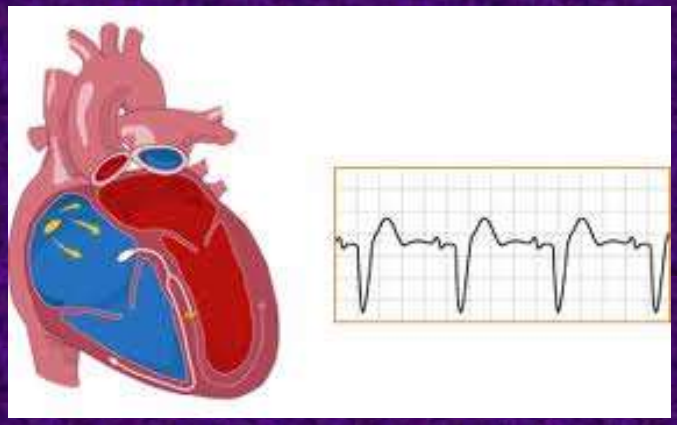
Greater number of P waves than QRS complexes

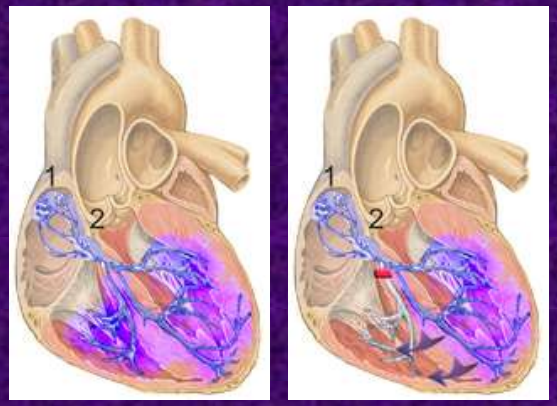
QRS complexes are usually narrow with HR 40-60 bpm (junctional rhythm). When the pacemaker cells in NH zone are destroyed, there might be idioventricular rhythm with wide QRS and HR 20-40 bpm

Intraventricular Block (Bundle Branch Block, BBB)

- Right bundle branch block, RBBB (complete, incomplete)
- Left bundle branch block, LBBB (complete)
- Incomplete LBBB: left anterior hemiblock (LAH), left posterior hemiblock (LPH)
- Trifascicular block

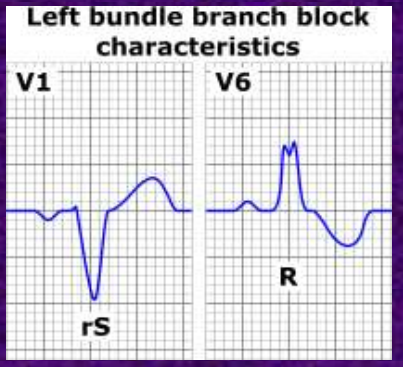
- Usually asymptomatic
- Severe bradycardia in trifascicular block
- Risk of re-entry (Bundle Branch Re-entry Tachycardia)
- Asynchrony of the ventricles (may worsen hemodynamic compromise in heart failure) – may be treated by biventricular pacemaker implantation

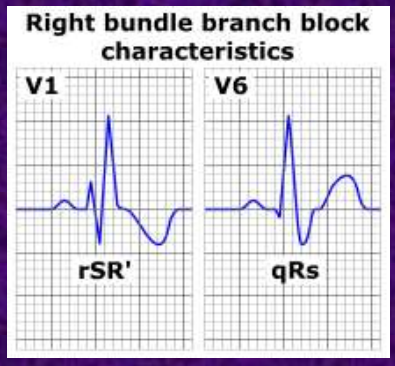




Intraventricular block - ECG

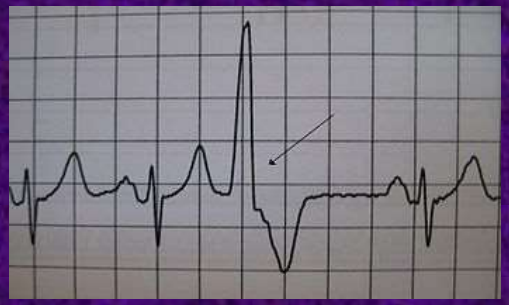
- Widening of QRS (iRBBB, LPH, LAH: 100-120 ms; RBBB, LBBB: ≥120 ms)
- In complete left- or right bundle block, there are also changes in plateau and repolarization synchronization, leading into changes of ST segment and T wave (esp. LBBB)
- In RBBB, most striking changes are in V1 (dipole moment pointing to the right); in LBBB, in V6 (dipole moment pointing to the left)
- In LAH, the axis is deviated into extreme negative values (<-45°); in LPH, into extreme positive values (>120°)
- Trifascicular block leads into complete atrio-ventricular dissociation as in 3rd degree AV block (with idioventricular rhythm)

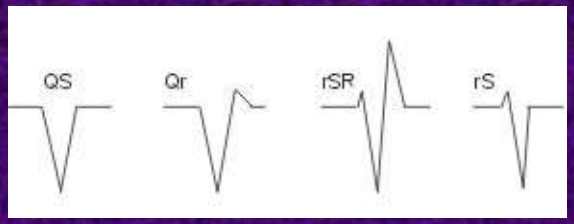




Ventricular Premature contractions (VPC)

- Syn. ventricular extrasystoles (VES)
- Is caused by either reentrant signaling or enhanced automaticity in some ectopic focus or by triggered activity

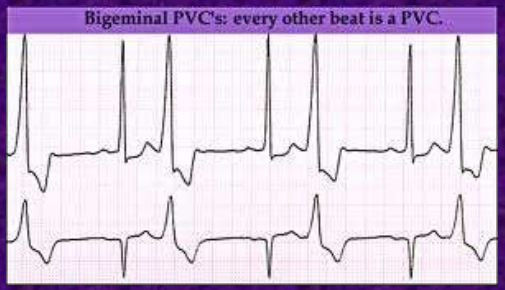




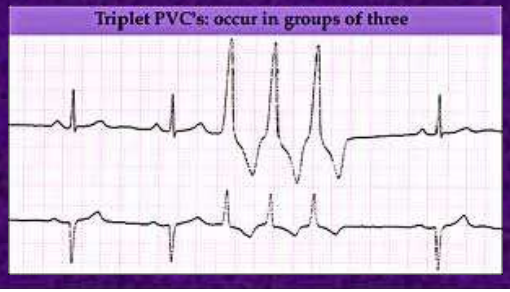
- The QRS complex is enlarged (>120ms) and has different shape, it is accompanied by changes in ST, T wave and compensation pause (SA node is „discharged“)

Coupling of VES

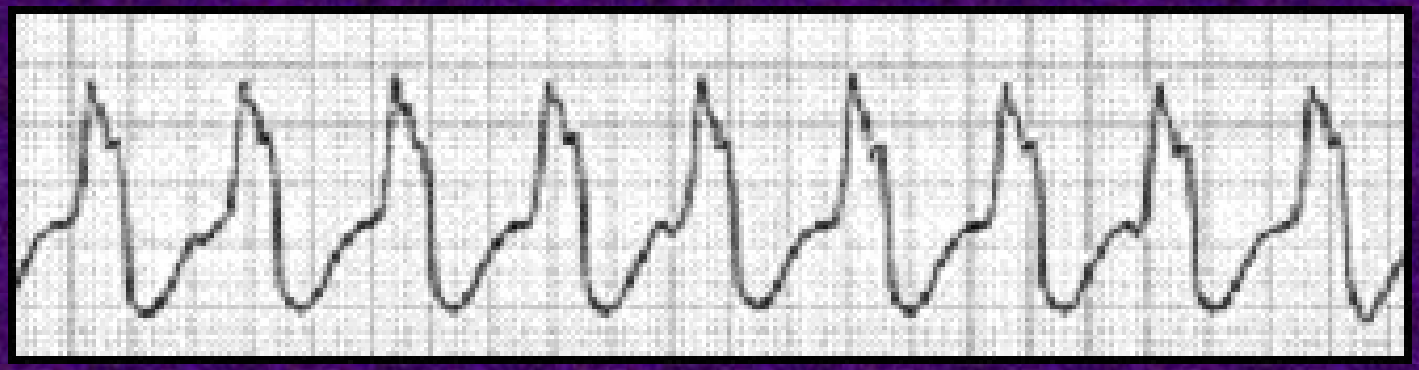
- Isolated VES are usually benign
- Premature ventricular beats occurring after every normal beat are termed ventricular bigeminy, if 2 normal QRS complexes are followed by VES, we speak of ventricular trigeminy (caused by ectopic pacemaker focus)



- Two VES grouped together are called a couplet, three a triplet. Runs longer than 3 VES is referred as non-sustained ventricular tachycardia (nonsustained if <30 sec) - re-entry



What is this arrhythmia?



Monomorphic ventricular tachycardia

Monomorphic = fixed re-entry circuit

Monomorphic ventricular tachycardia is usually caused by re-entry, and most commonly seen in patients following myocardial infarction.

Sometimes, it is called „ventricular flutter“

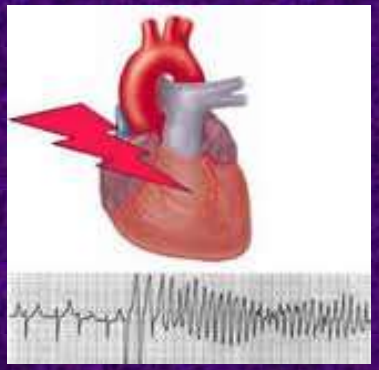
In rarer cases, ventricular tachycardia may be polymorphic – alternating, but stable re-entry circuits, or torsades de pointes

Ventricular or supraventricular?

- While narrow QRS is typical for supraventricular arrhythmias (i.e. origin in SA node, atria, AV node), wide QRS can be caused by ventricular arrhythmia -80% of cases, as well as by supraventricular arrhythmia combined with conduction block or by AVRT -20% of cases
- An older (non-arythmic) ECG of the same person might be checked, if available
- If we don‘t know, it is better to treat the arrhythmia as ventricular

Polymorphic ventricular tachycardia – torsades de pointes

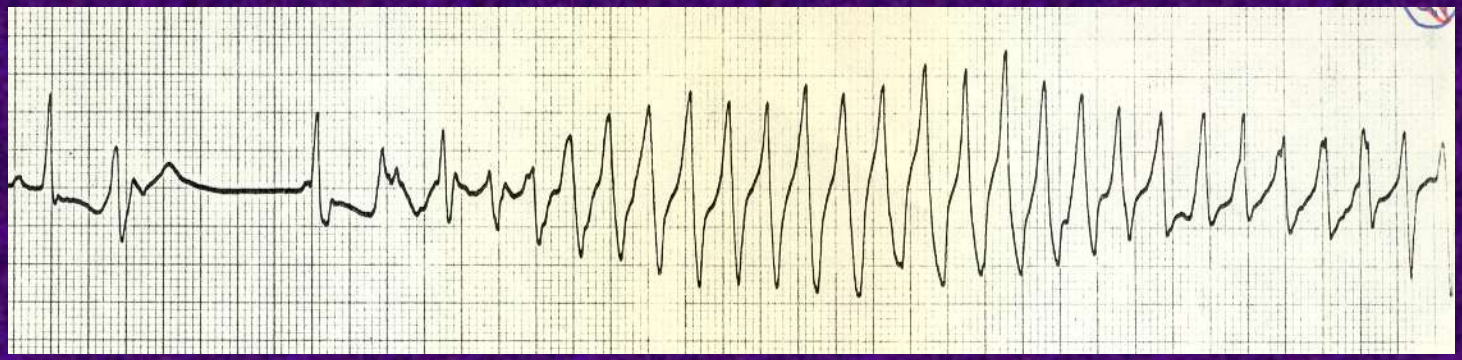
- “Twisting of the points“
- Is connected with the long QT interval – early afterdepolarization (triggered activity)



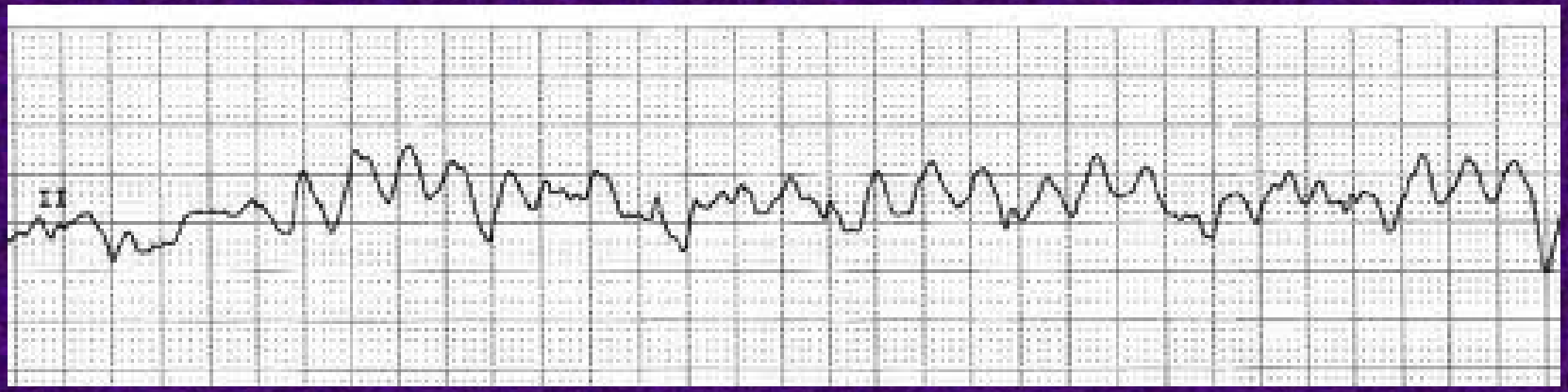
- Unstable cardiomyocytes may be influenced by local currents that induce new depolarization, while the former action potential is still not finished (R-on-T phenomenon). Tachyarhythmia with unstable, rotating circuit may be triggered

Torsades de pointes - ECG

- The rotation of the circuit leads into changing shape of QRS in a given lead
- Causes: hypokalemia, pharmacological/genetic block of K^+ or Na^+ channels



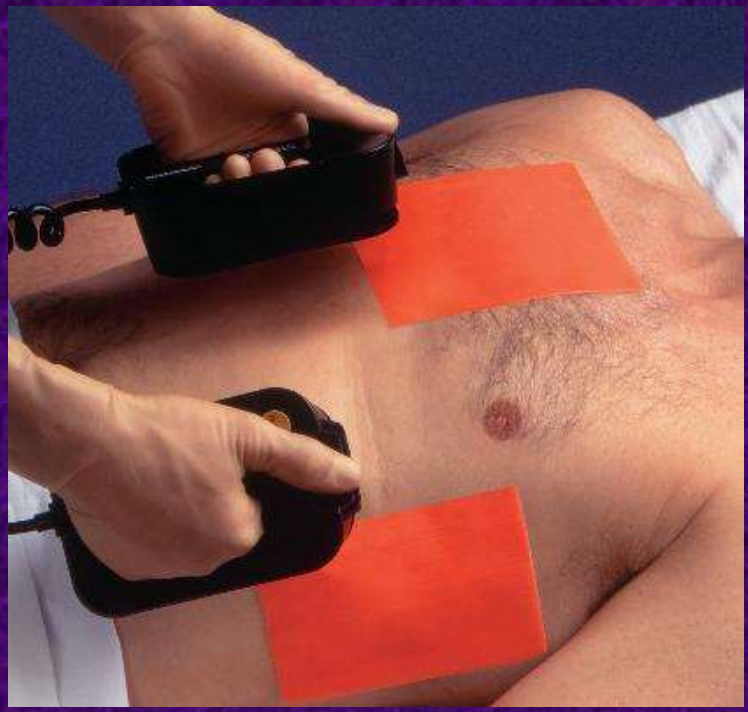
Ventricular fibrillation



- Uncoordinated electric currents and contractions in the ventricles with the frequency of >300bpm
- Lethal arrhythmia, which causes most of the total number of cardiac arrests
- Leads into asystolia (straight line on ECG) when untreated
- It may complicate myocardial infarction or be a result of other ventricular tachyarrhythmia
- VF might be induced by strong electric current or by contrast matter during cardiac catheterization

Defibrillation

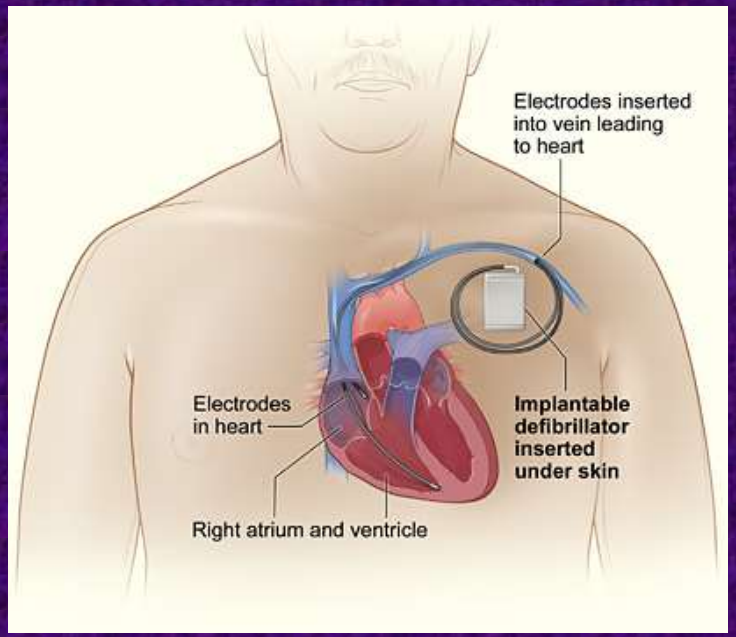
- Electric action of the heart can be re-coordinated by electric discharge (with the energy of 200 – 360 J).
- One electrode is put at upper sternum, the other above the apex of the heart.
- If a defibrillator is unavailable, a precordial thump may be tried (up to 30 sec from the arrest) – limited efficiency.



At public places, automatized external defibrillators (AED) are available. They are capable of automatic analysis of cardiac electric activity in 10-20 sec, and performing discharge, when necessary

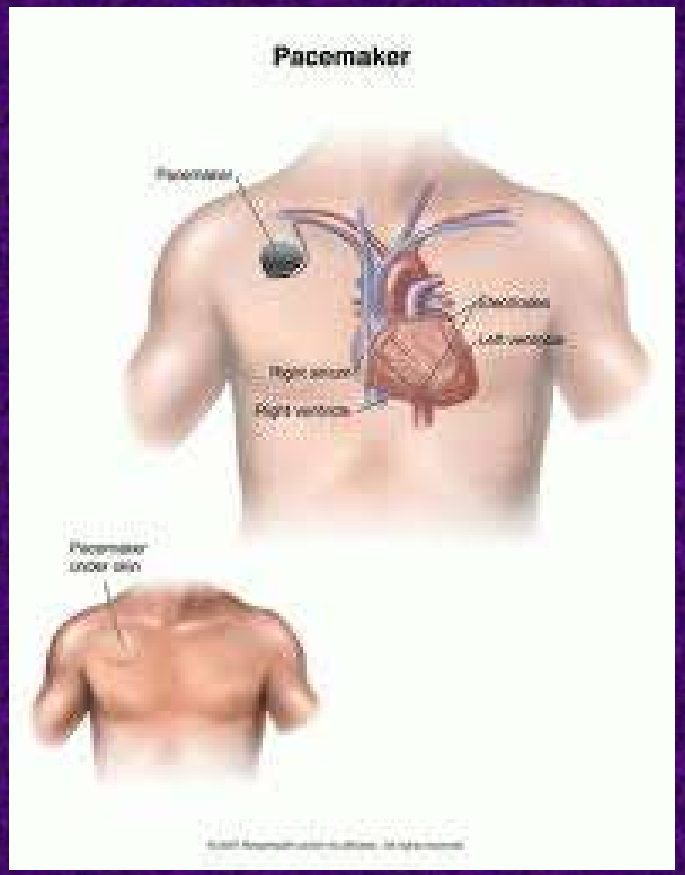
ICD

- Patients at risk may undergo the implantntation of defibrillator (ICD = implantable cardioverter-defibrillator).
- The device will automatically discharge during ventricular fibrillation
- ICD is usually implanted below left clavicle



Cardiac stimulation (arteficial pacemaker - PM)

- PMs are used in manifesting bradyarrhythmia (SA and AV blocks, trifascicular block; in tachy-brady atrial fibrillation and sick sinus syndrom it is used together with pharmacological treatment lowering the HR)
- Usually implanted below right clavicle
- Most often, „on demand“ PMs are used, that register the HR and start discharging only when it is lower than the threshold previously set up
- Combined devices (PM+ICD) also exist

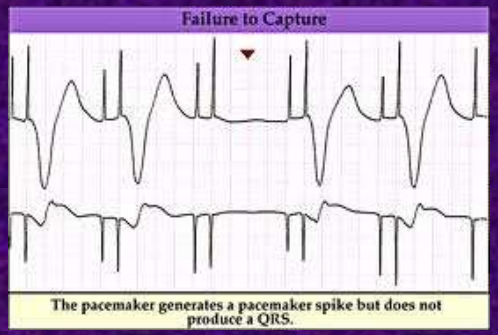


ECG in cardiac stimulation

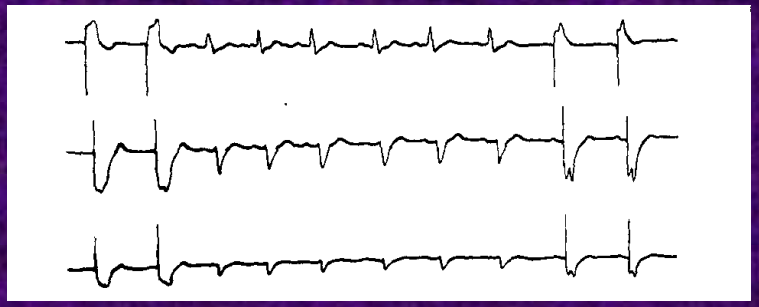
- The stimulating electrodes are installed in stimulated parts of the heart
  - Atria
  - Right ventricle
  - Both ventricles (biventricular)
  - Sequential – atria + ventricles
  - Atrial triggered ventricular stimulation
- The waves on ECG curve are preceded by narrow „spikes“ (according to stimulated part of the heart)

ECG in cardiac stimulation - examples

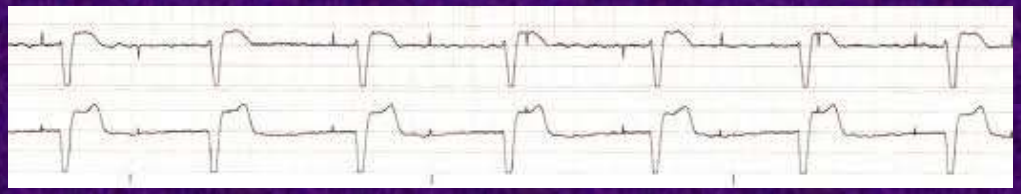
Sequential stimulation of atria and ventricles with a drop out



“On demand“ ventricular stimulation



Ineffective stimulation



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Thank you for your attention