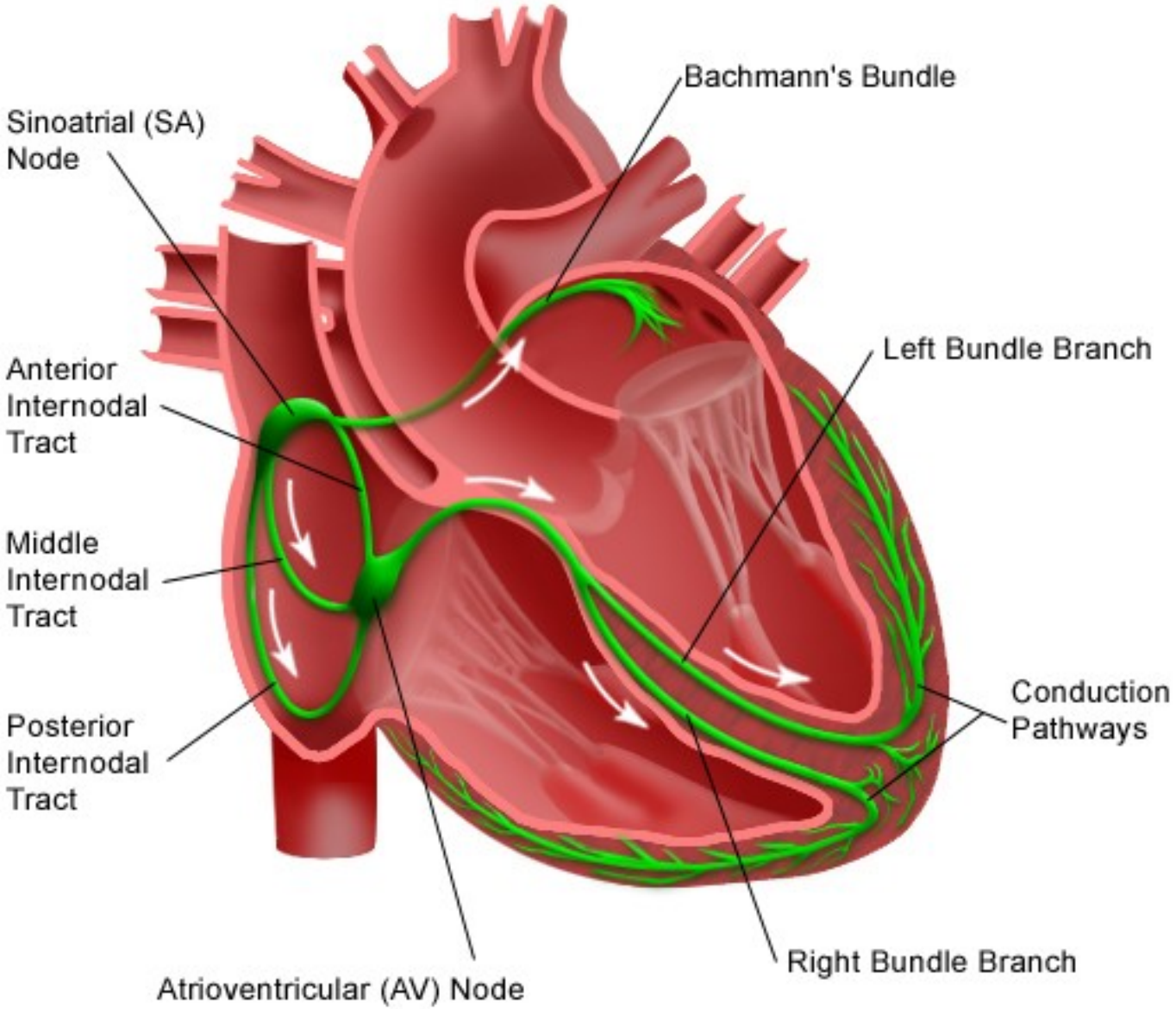
Arrhythmology

Electrical System of the Heart



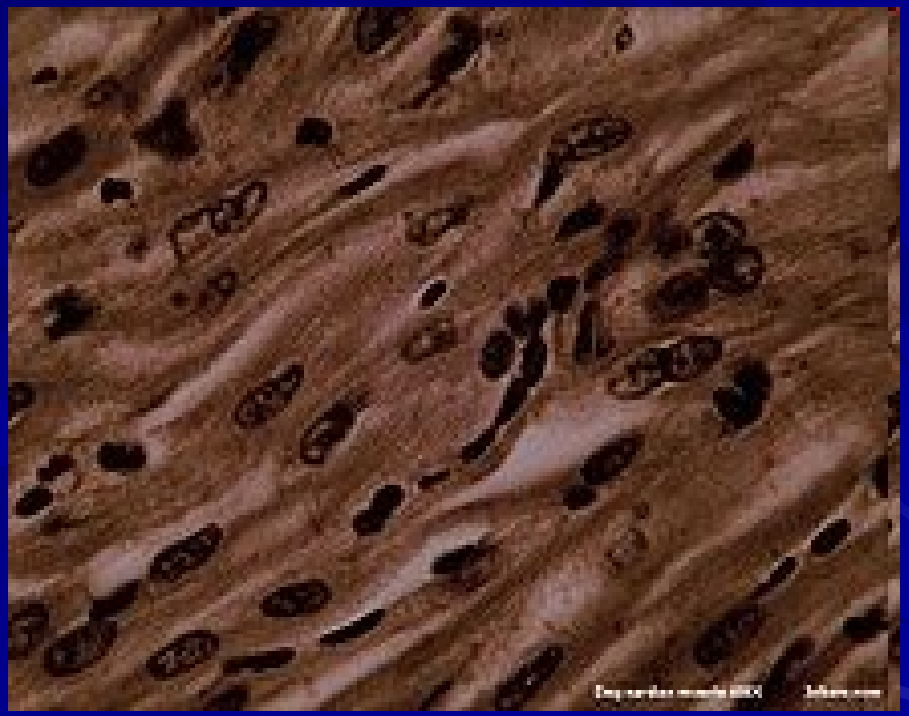
Cardiomyocytes

Heart muscle consists of three types of cells:

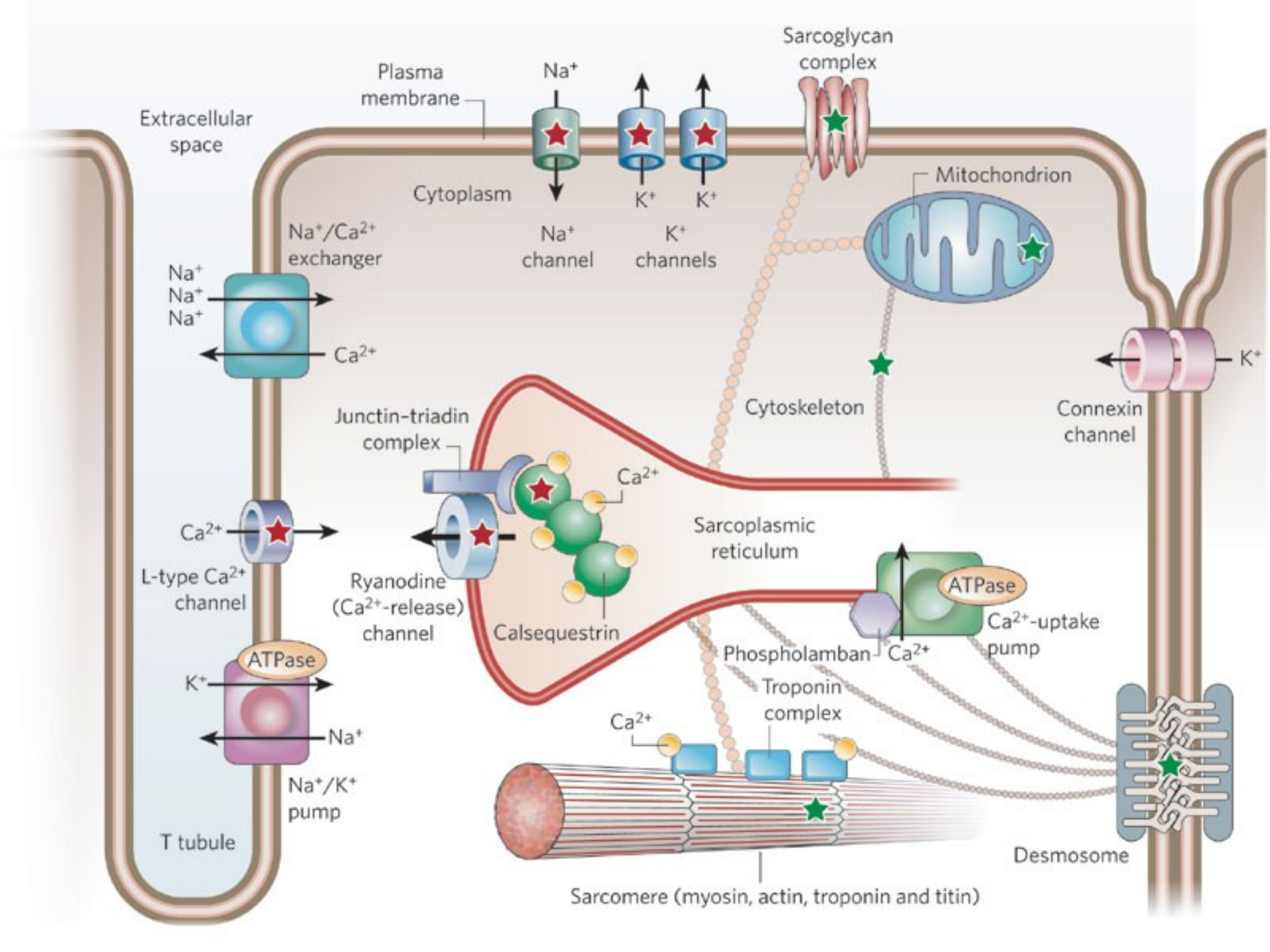
1) „Fast cells“ of working myocardium that make a contraction as a response to electric signal created in pacemaker cells – most common type

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

3) „Pacemaker cells“ that create the electric signal.



Connection between two cells is maintained by desmosomes

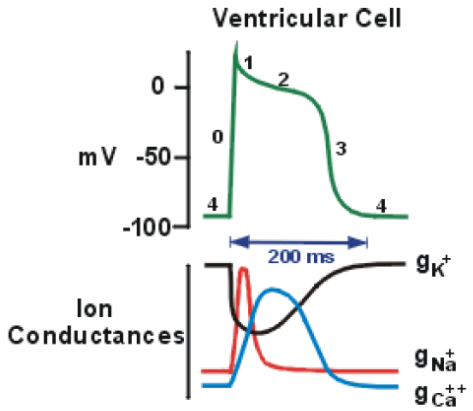

Sarcoglycan complex
Plasma membrane
Na+
Extracellular space
Cytoplasm
K+
K+
Mitochondrion
Na+/Ca2+ exchanger
Na+ channel
K+ channels
Na+
Na+
Na+
Ca2+
K+
Cytoskeleton
Connexin channel
Junctin-triadin complex
Ca2+
Sarcoplasmic reticulum
Ca2+
L-type Ca2+ channel
Ryanodine (Ca2+-release) channel
Calsequestrin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

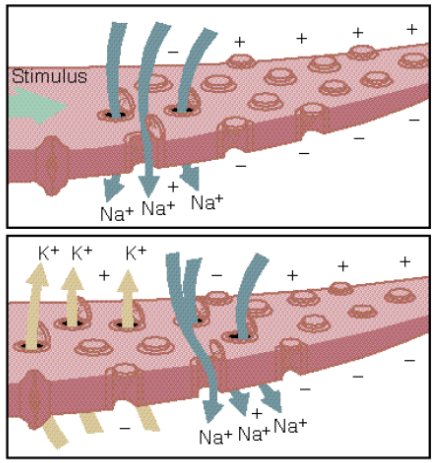
Mechanism of cardiomyocyte activity 1

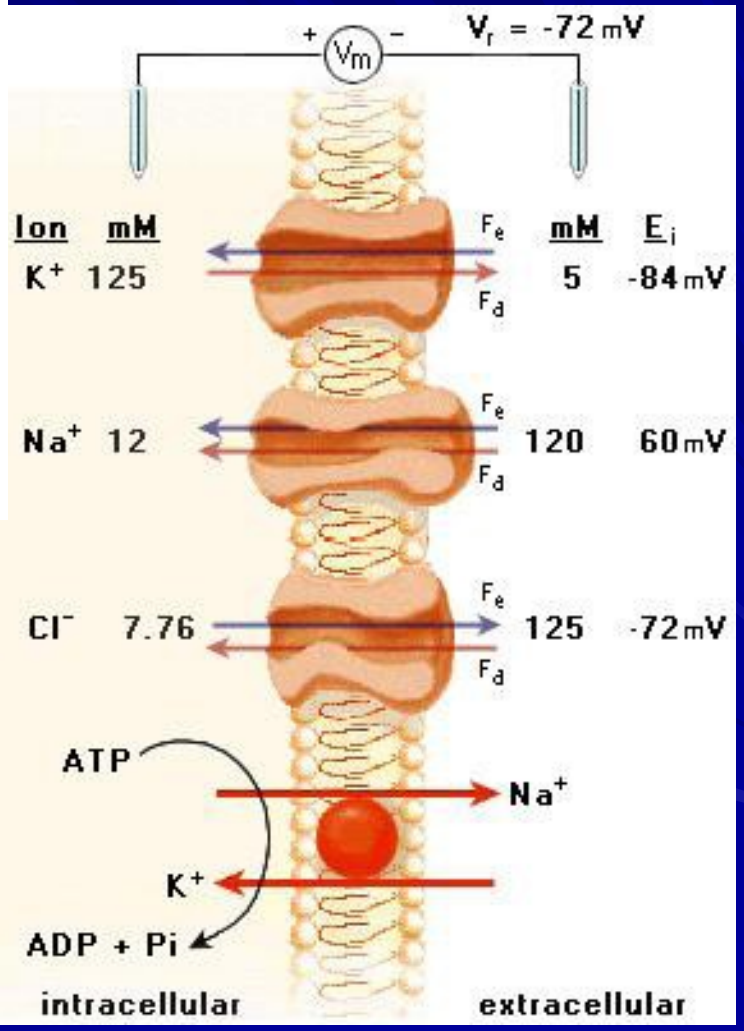
- Three cations present in both extra- and intracelular fluid participate in electrical activity of heart muscle: Na^+, K^+ and Ca^{2+}. Na^+ and Ca^{2+} are present mainly in ECF (Ca^{2+} also in endoplasmic reticulum) , K^+ in ICF
- During fast depolarisation of a cardiomyocyte (phase 0), voltage-gated 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 – refractory period

Mechanism of cardiomyocyte activity 2

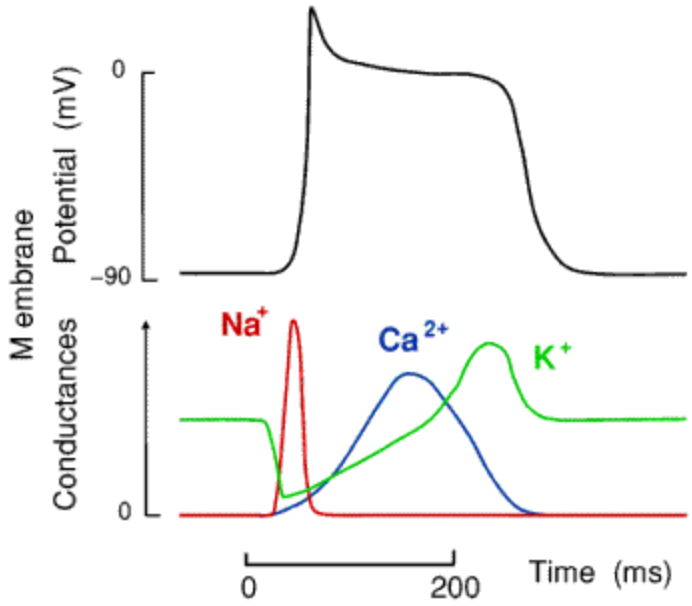
- In phase 2 („plateau“), prolonged depolarisation is maintained by the influx of Ca^{2+} through $I_{Ca\text{-}L}$ channels. Unlike I_{Na} or I_{to}, $I_{Ca\text{-}L}$ channel is gated both by voltage and receptor mechanism, that responds to vegetative nervous signalisation. Ca^{2+} binds to ryanodin 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

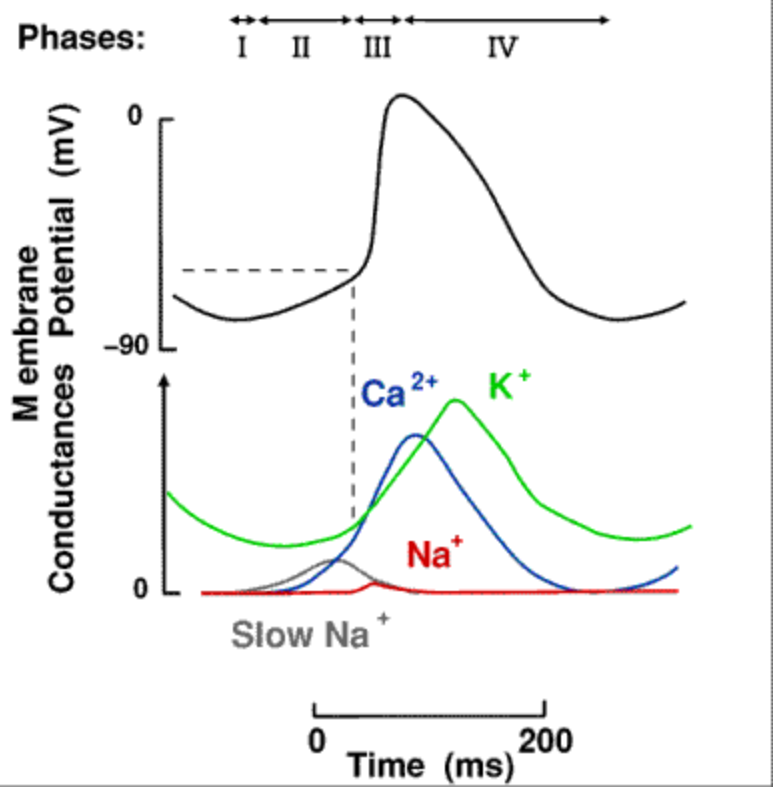

Ventricular Cell
0
mV -50
-100
1
2
0
3
4
4
200 ms
Ion
Conductances
g_{K^+}
g_{Na^+}
$g_{Ca^{++}}$

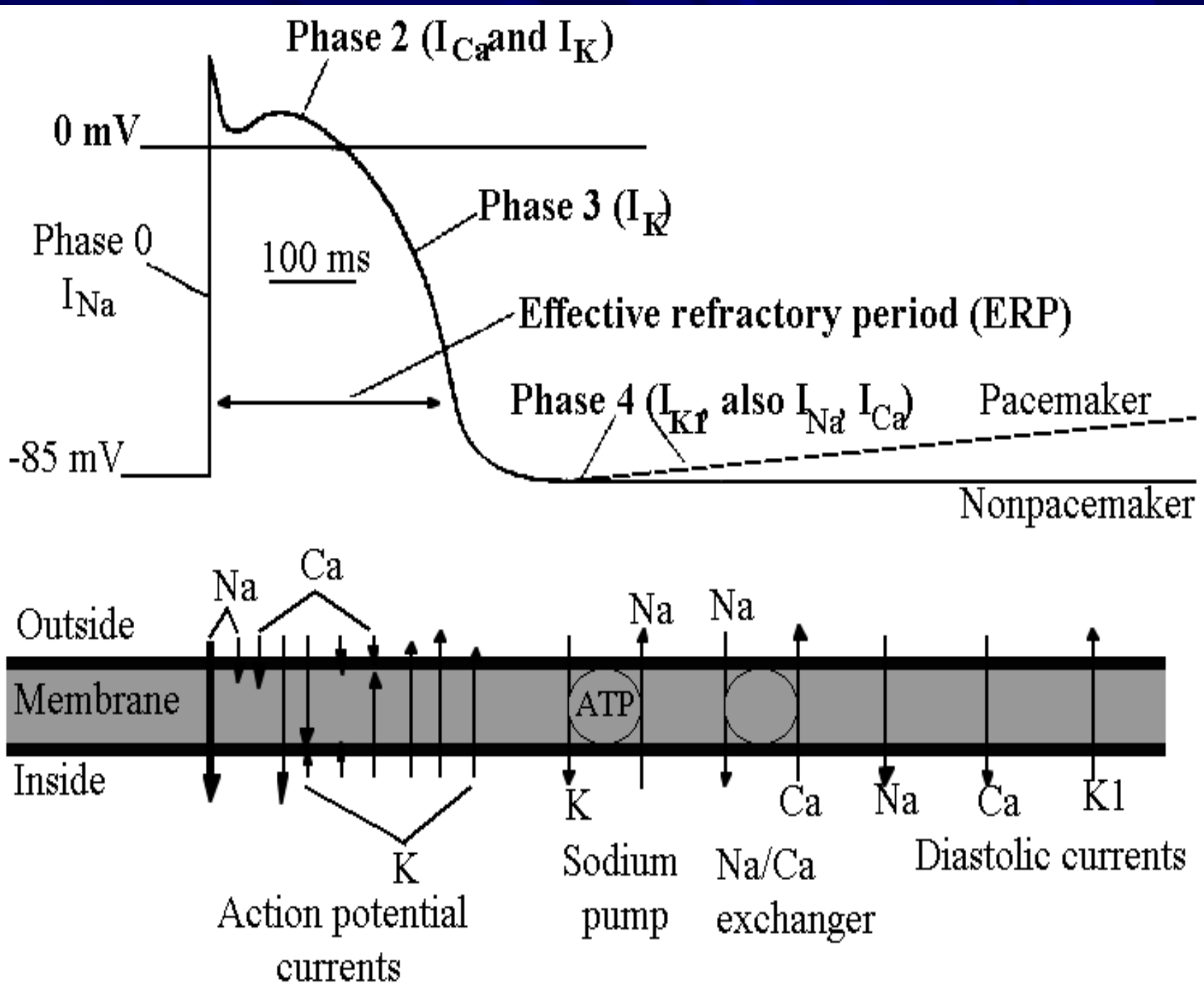

Stimulus
Na+ Na+ Na+
K+ K+ K+
Na+ Na+ Na+


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

Action potential and underlying conductance changes in a ventricular myocyte (from a small mammal)





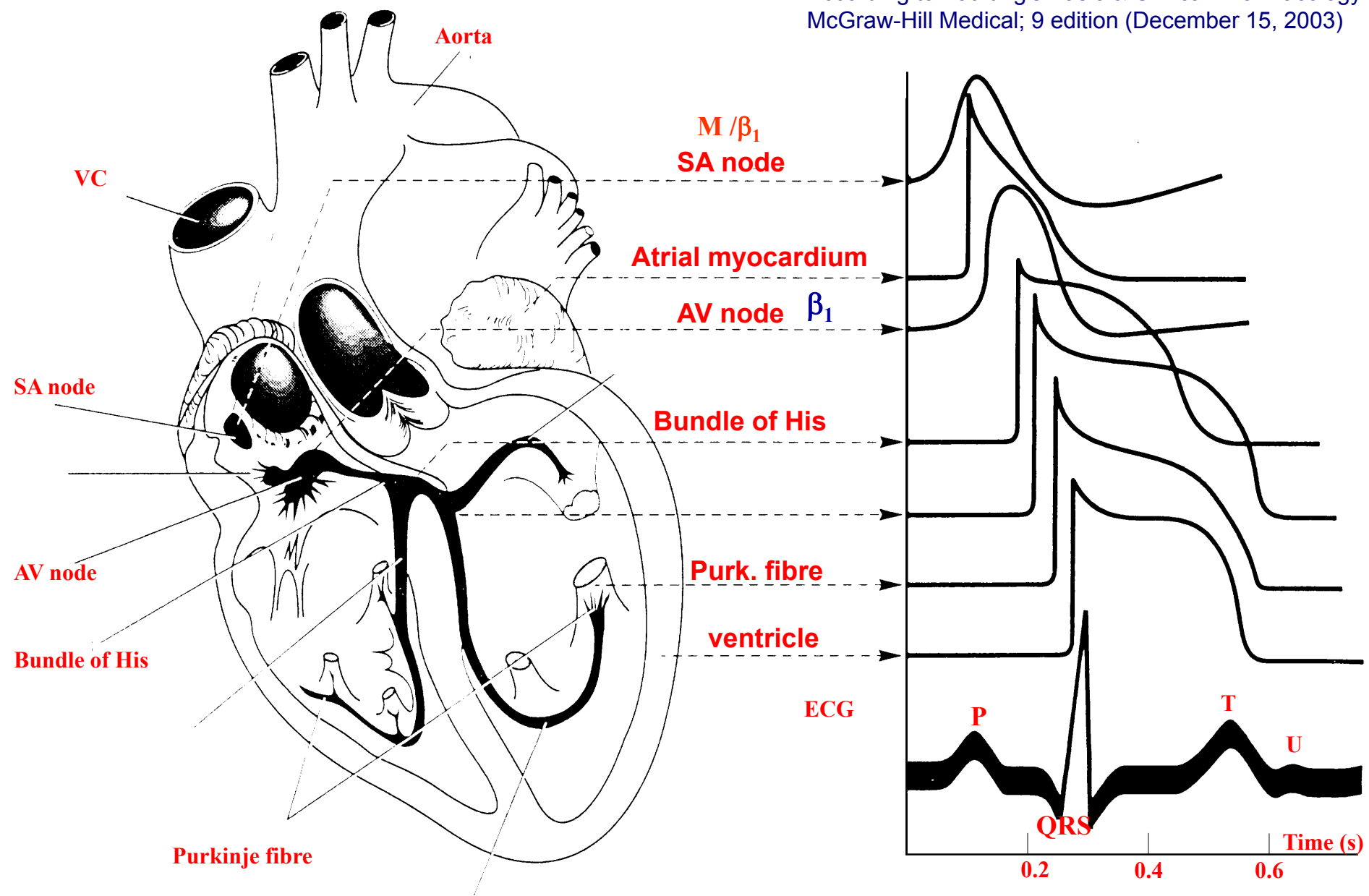

Phase 2 (I_{Ca} and I_K)
0 mV
Phase 3 (I_K)
Phase 0
I_{Na}
100 ms
Effective refractory period (ERP)
Phase 4 (I_{K1} also I_{Na}, I_{Ca})
Pacemaker
-85 mV
Nonpacemaker
Na
Ca
Outside
Membrane
Inside
ATP
Na
Na
K
Ca
Na
Ca
K1
K
Action potential currents
Sodium pump
Na/Ca exchanger
Diastolic currents

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

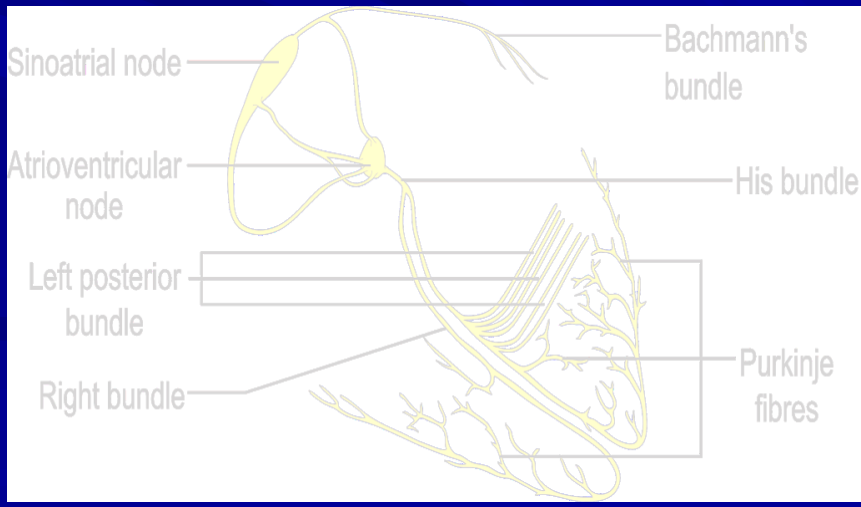
Normal conduction within the heart

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



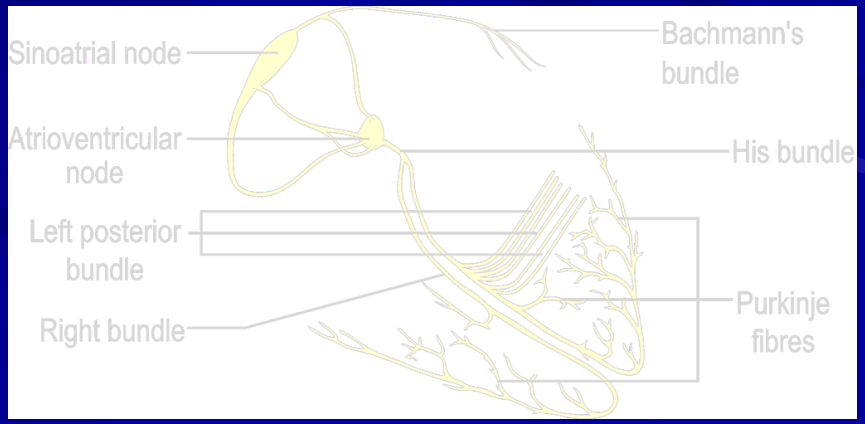
Sinoatrial (SA) node

- 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

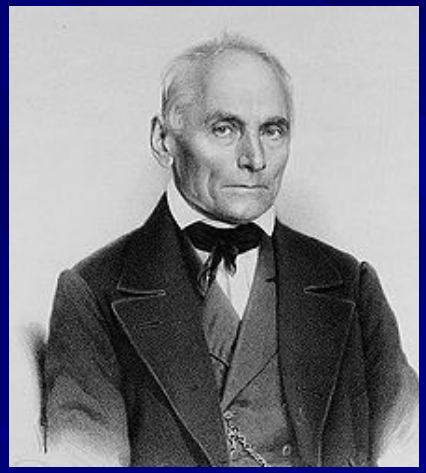
- Area of specialized tissue located between atria and ventricles, near the coronary sinus and tricuspid valve. It serves as secondary pacemaker and is the only way of electric connection between the atria and the ventricles under normal circumstances.
- AV node consists of 3 zones: AN (atria-nodus), 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“. These cells do not contain sodium channels, their depolarisation is then mediated by Ca^{2+}. The conduction delays by about 0,12s there. The Ca^{2+} I_{Ca-L} receptors are influenced by the sympathicus and the parasympathicus.
- 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

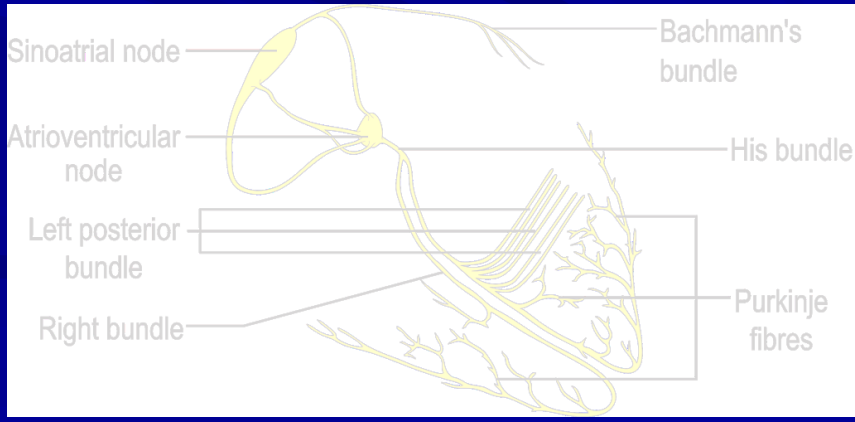
- Part of cardiac tissue specialized for fast electrical conduction that leads the signal from AV-node to working myocardium of the ventricles.
- After its short course, the Bundle of His branches ito right and left bundle branch (Tawara branches). Right bundle branch is long and thin, thus more vulnerable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

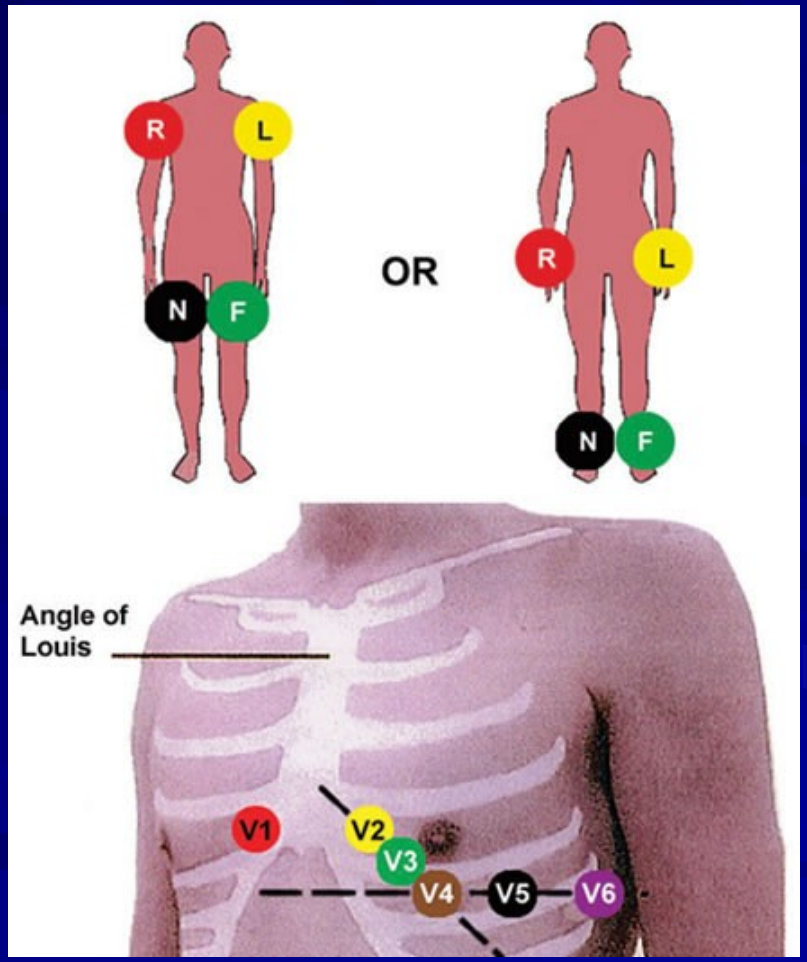


12-leads ECG (uses 10 electrodes)

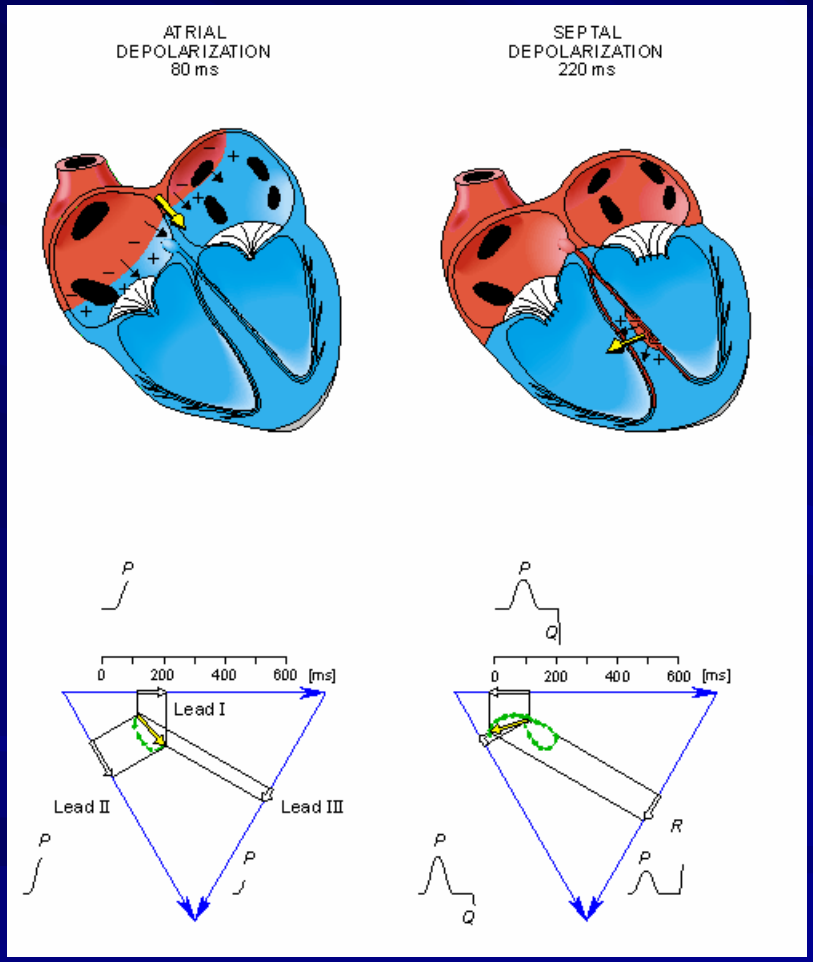
Electrode placement:

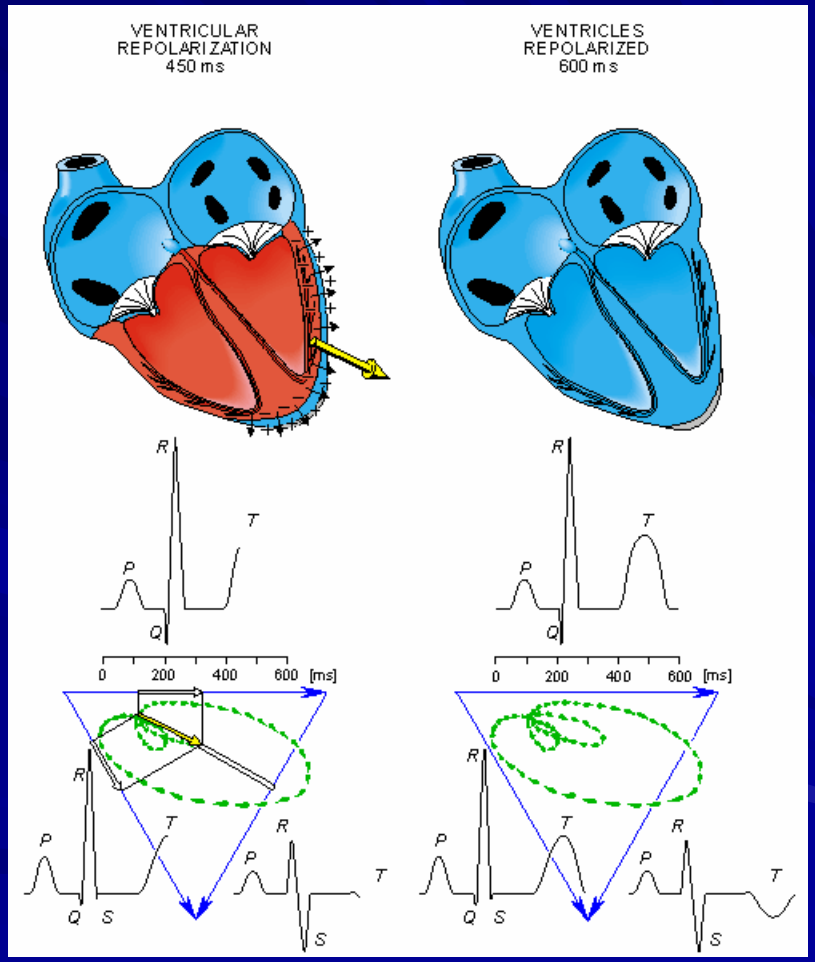
RA: On the right arm, avoiding bony prominences.
LA: In the same location that RA was placed, but on the left arm this time.
RL: On the right leg, avoiding bony prominences.
LL: In the same location that RL was placed, but on the left leg this time.
V1: In the *fourth* intercostal space (between ribs 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-leads ECG – electrode placement

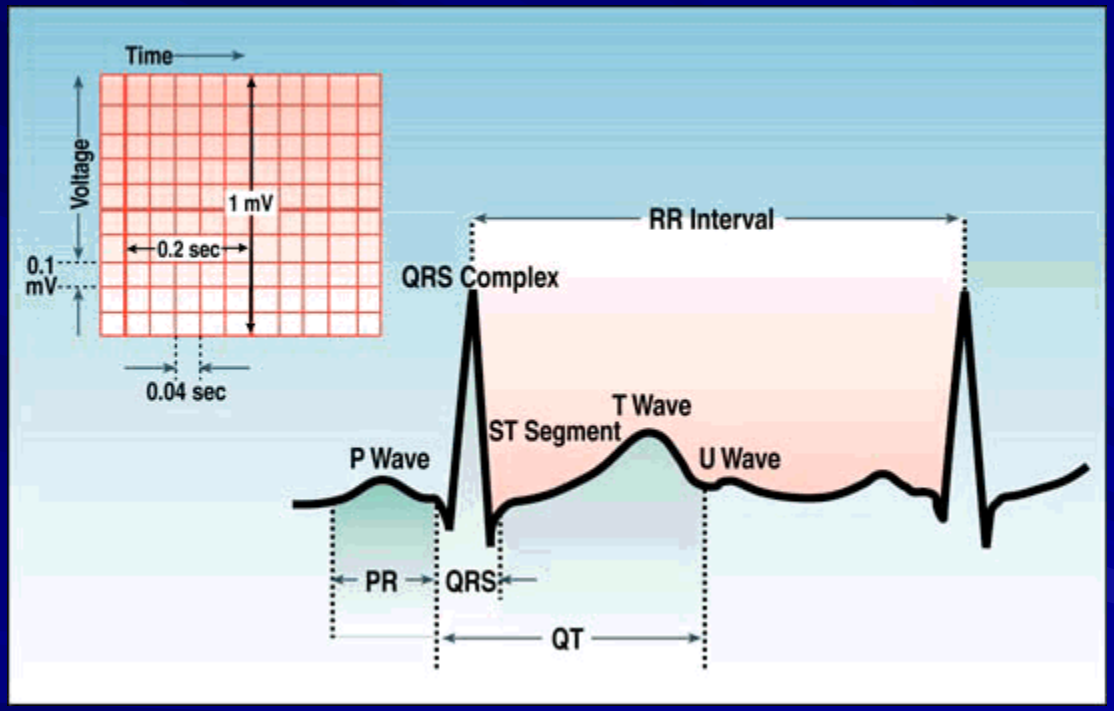


Evaluation of electrical signal: Eindhoven‘s triangle

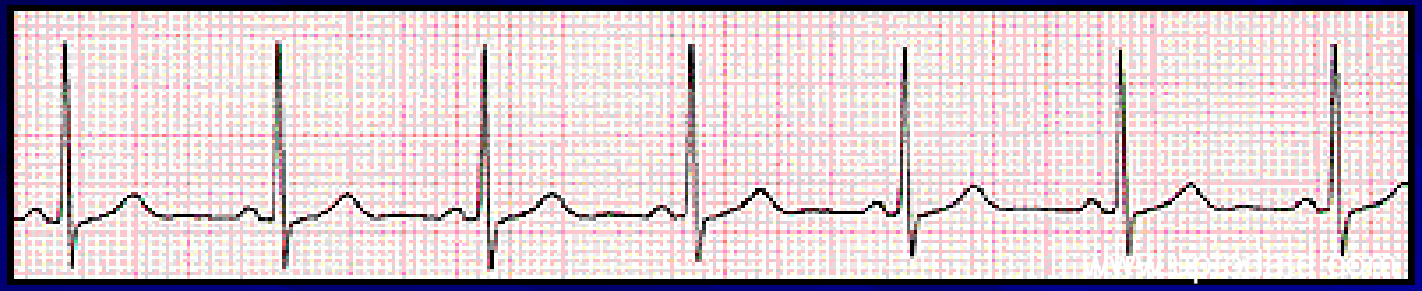




Normal ECG curve



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.

EKG Characteristics:

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

Description of ECG

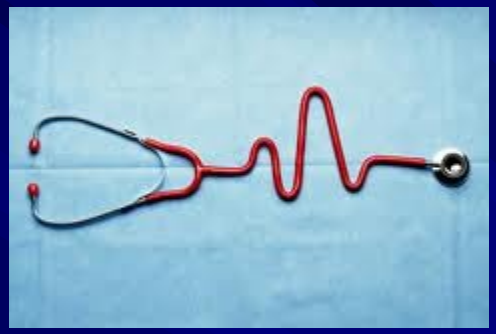
- rhythm
  - – sinus
    - 60-90/min
  - – other
- junctional
  - – 40-60/min
- idioventricular
  - – 30-40/min
- atrial fibrilation
- atrial flutter

- action
- – regular
- – irregular

- frequency
- – normal
- 60 – 90/min
- – tachycardia
- >90/min
- – bradycardia
- <60/min

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

Arrhythmias:



- Electrophysiological abnormalities arising from the impairment of the impulse

  1. genesis (origin), 2. conduction, 3. both previous

- Arrhythmias are defined by exclusion - i.e., any rhythm that is not a normal sinus rhythm (NSR, 60-100 bpm) is an arrhythmia

- With respect to the
  - Frequency – bradyarrythmias vs. tachyarrhythmias
  - Localization – supraventricular (SV), ventricular (V)
  - Mechanism – early after depolarisation (EAD), delayed after depolarisation (DAD), re-entry

Mechanism of Arrhythmia

- **Abnormal heart pulse formation**

1. Sinus pulse
2. Ectopic pulse
3. Triggered activity

- **Abnormal heart pulse conduction**

1. Reentry
2. Conduct block

Possible causes of arrhytmia

- Vegetative nervous system disorder (nervous lability, compensation of heart failure, shock, anxiety)
- Ischaemia, hypoxia and reperfusion, pH disorders
- Disorders of iont balance
- Disorders of myocardium – hypertrophy, dilatation, amyloidosis, scar aftar acute myoacrdial infarction
- Inflammation
- Drugs (β-blockers, digitalis, antiarrhytmics)
- General state (trauma, endokrinopathy..)
- Genetic causes (ion channel mutations)
- Aberrant conduction – bundle of KENT (WPW syndrom – aberrant track between the atria and the ventricles bypassing the AV-node

Brady- and tachyarrhythmias:

1. Bradyarrhythmias

- SA block
- sick-sinus syndrome
- AV block

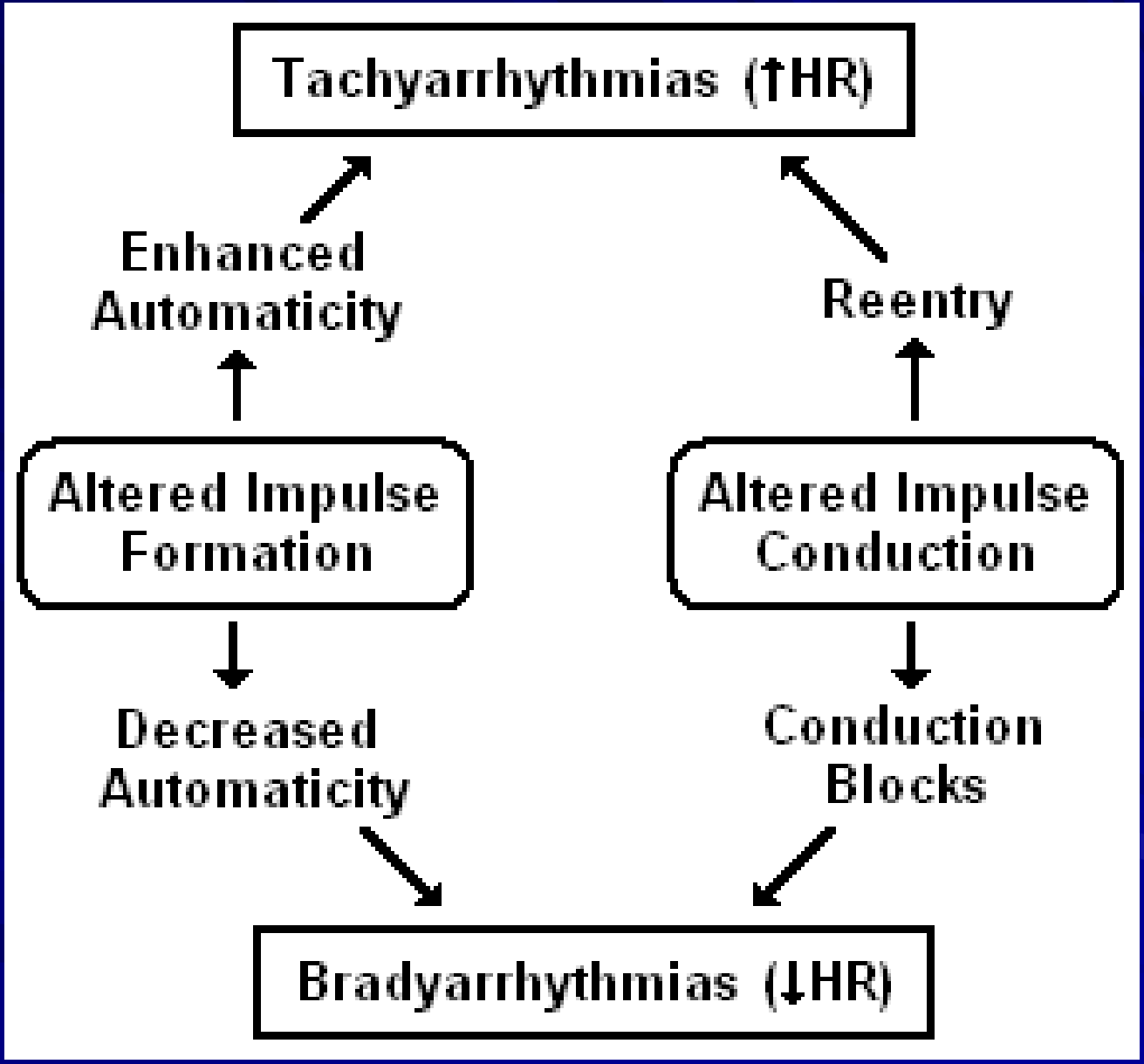
2. Tachyarrhythmias

a) Supraventricular (SV)

- SV extrasystoles – 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…

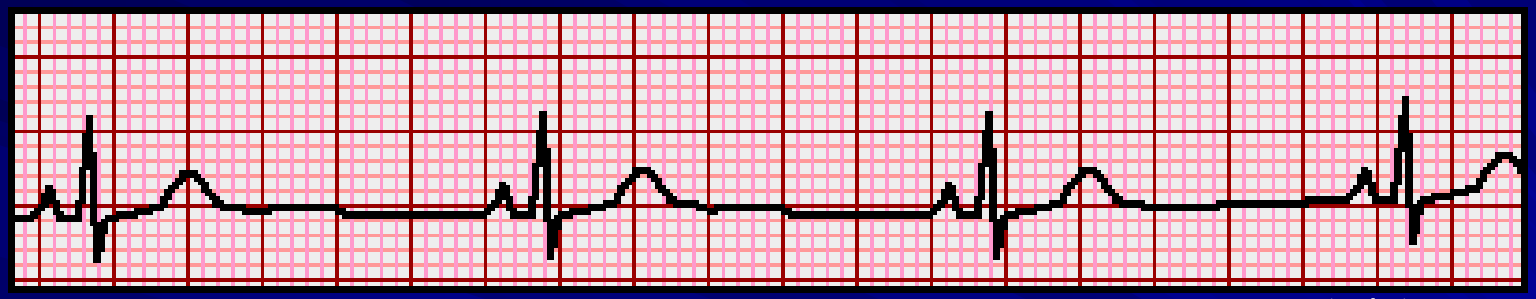

Tachyarrhythmias (↑HR)
Enhanced Automaticity
Reentry
Altered Impulse Formation
Altered Impulse Conduction
Decreased Automaticity
Conduction Blocks
Bradyarrhythmias (↓HR)

Recognizing altered automaticity on EKG

Gradual onset and termination of the arrhythmia.

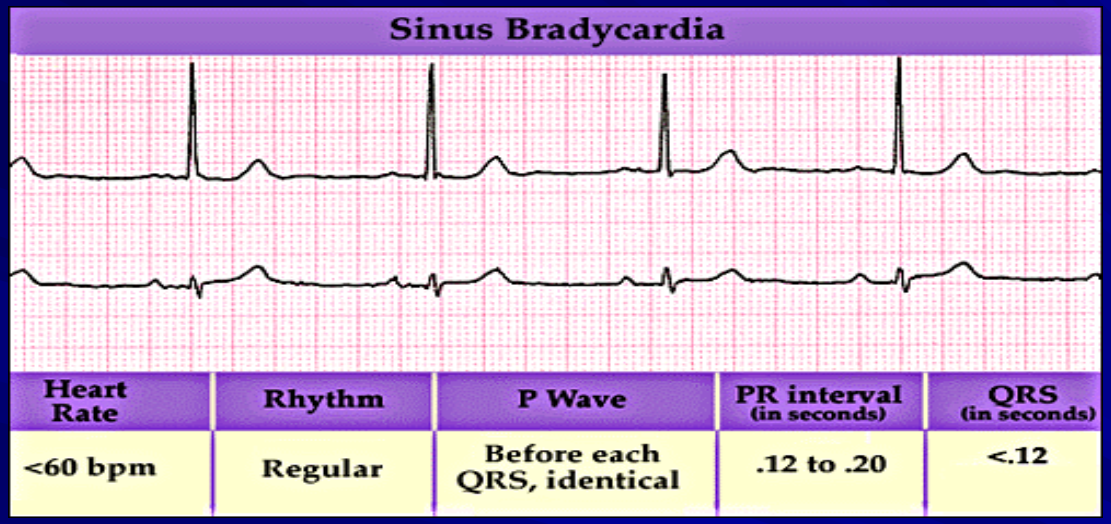
The P wave of the first beat of the arrhythmia is typically the same as the remaining beats of the arrhythmia (if a P wave is present at all).

Decreased Automaticity



Sinus Bradycardia

Sinus Bradycardia

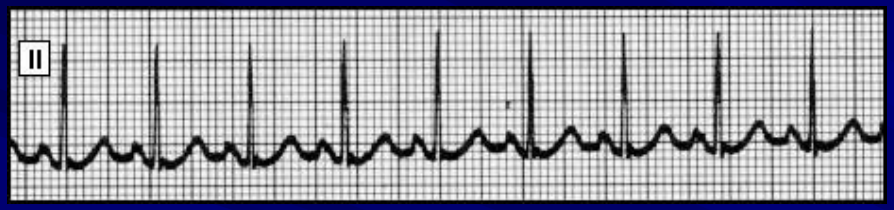


- HR< 60 bpm; every QRS narrow, preceded by p wave
- Can be normal in well-conditioned athletes
- HR can be<30 bpm in children, young adults during sleep, with up to 2 sec pauses

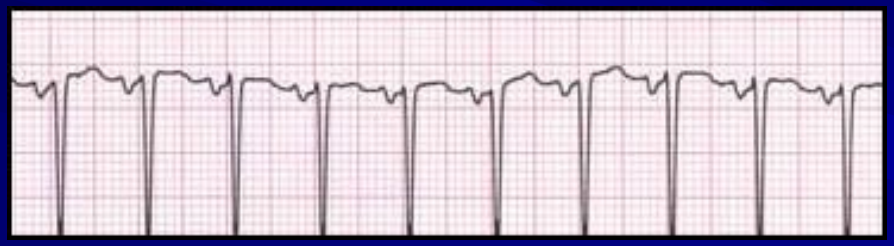
Sinus bradycardia - etiologies

- Normal aging
- 15-25% Acute MI, esp. affecting inferior wall
- Hypothyroidism, infiltrative diseases (sarcoid, amyloid)
- Hypothermia, hypokalemia
- SLE, collagen vasc diseases
- Situational: micturation, coughing
- Drugs: beta-blockers, digitalis, calcium channel blockers, amiodarone, cimetidine, lithium

Increased/Abnormal Automaticity



Sinus tachycardia



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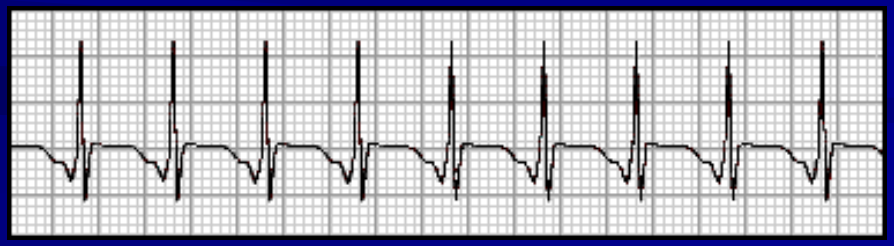
Ectopic atrial tachycardia

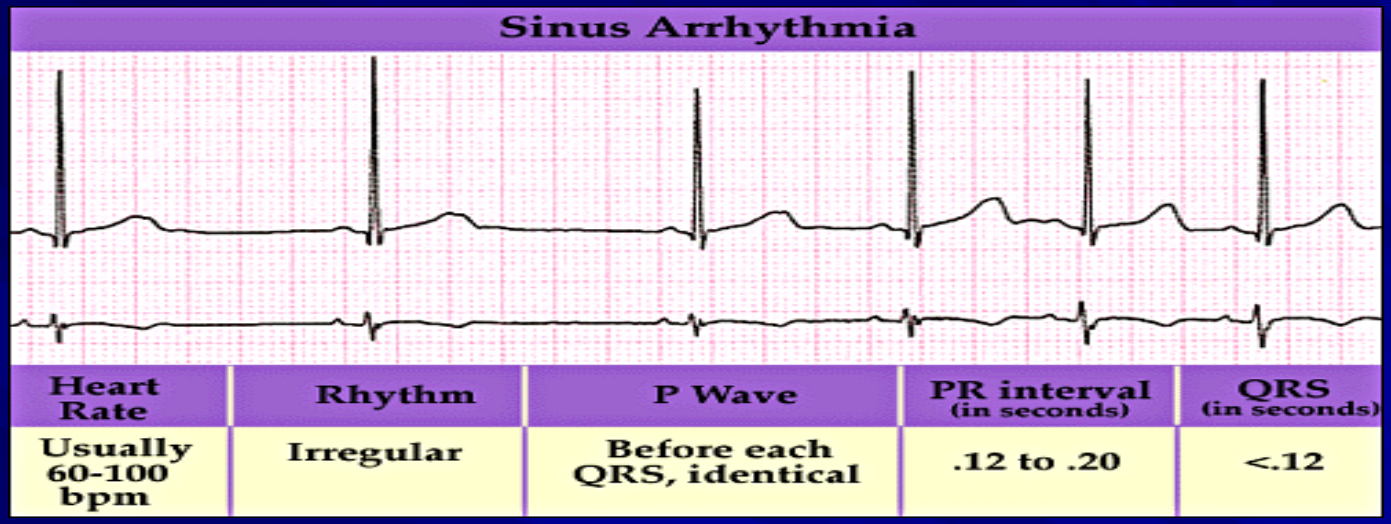


Junctional tachycardia

Sinus tachycardia - etiologies

- Fever
- Hyperthyroidism
- Effective volume depletion
- Anxiety
- Pheochromocytoma
- Sepsis
- Anemia
- Exposure to stimulants (nicotine, caffeine) or illicit drugs
- Hypotension and shock
- Pulmonary embolism
- Acute coronary ischemia and myocardial infarction
- Heart failure
- Chronic pulmonary disease
- Hypoxia

Sinus Arrhythmia

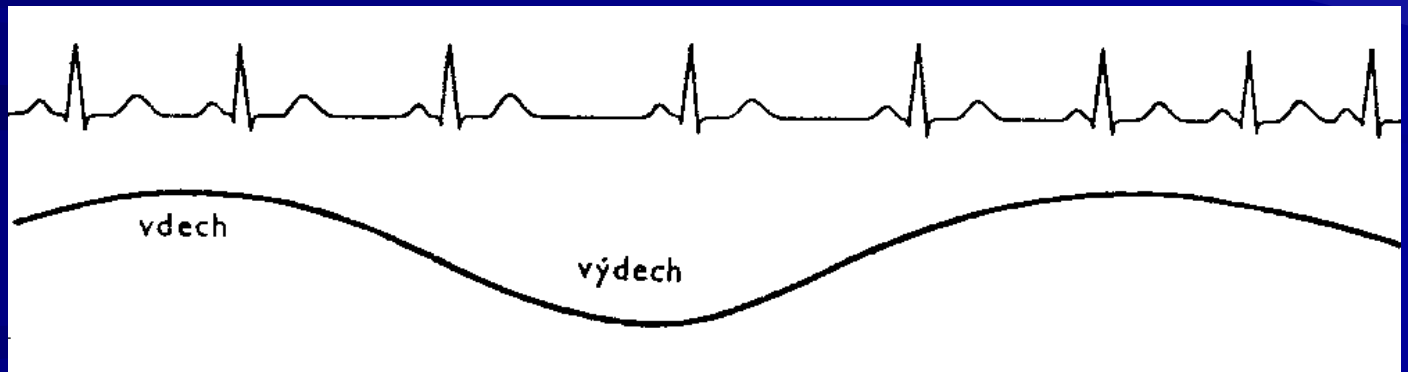


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
Usually 60-100 bpm	Irregular	Before each QRS, identical	.12 to .20	<.12

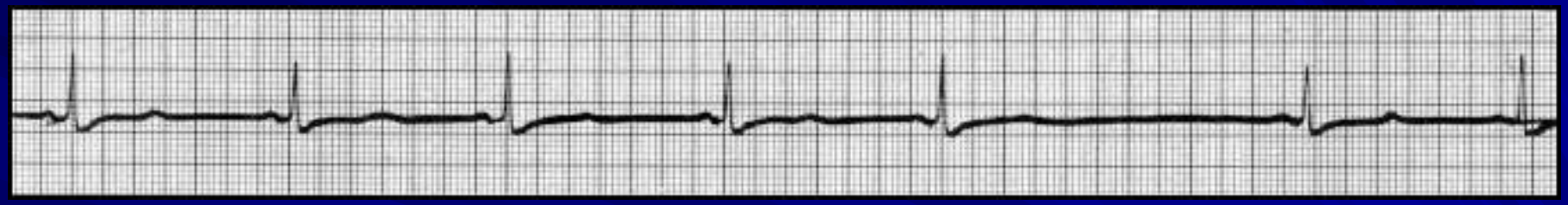
- Variations in the cycle lengths between p waves/ QRS complexes
- Will often sound irregular on exam
- Normal p waves, PR interval, normal, narrow QRS

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



Sick Sinus Syndrome



- All result in bradycardia
- Sinus bradycardia (rate of ~43 bpm) with a sinus pause
- Often result of tachy-brady syndrome: where a burst of atrial tachycardia (such as afib) is then followed by a long, symptomatic sinus pause/arrest, with no breakthrough junctional rhythm.

Sick Sinus Syndrome - etiology

- Often due to sinus node fibrosis, SNode arterial atherosclerosis, inflammation (Rheumatic fever, amyloid, sarcoid)
- Occurs in congenital and acquired heart disease and after surgery
- Hypothyroidism, hypothermia
- Drugs: digitalis, lithium, cimetidine, methyldopa, reserpine, clonidine, amiodarone
- Most patients are elderly, may or may not have symptoms

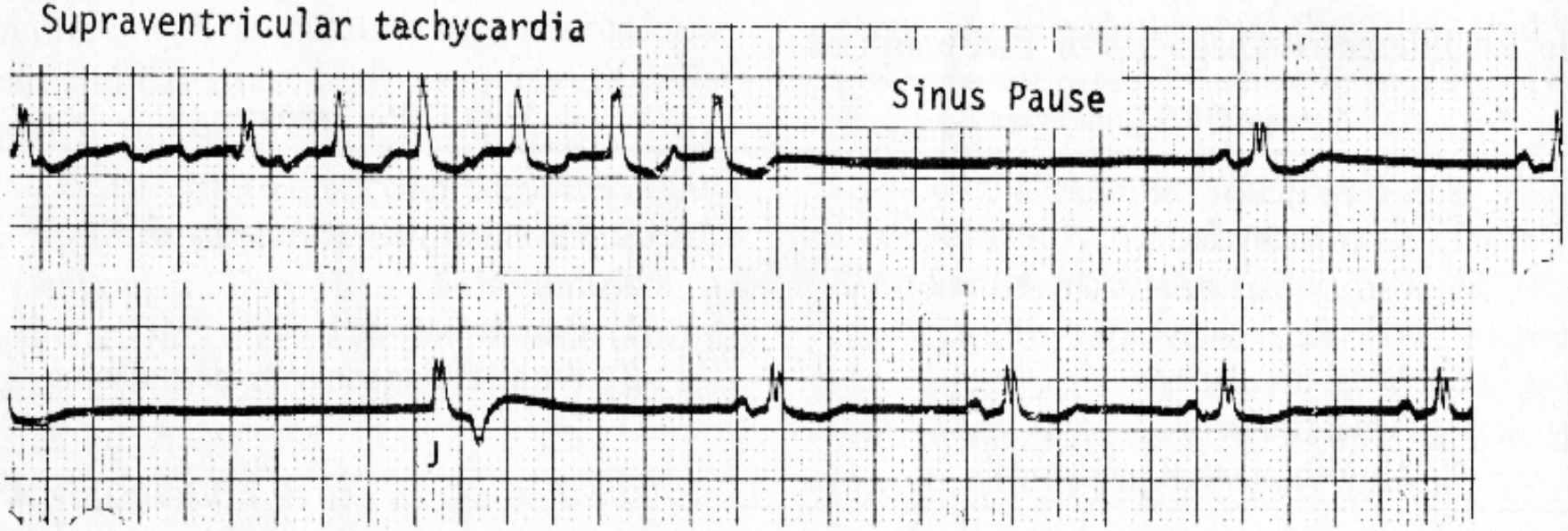
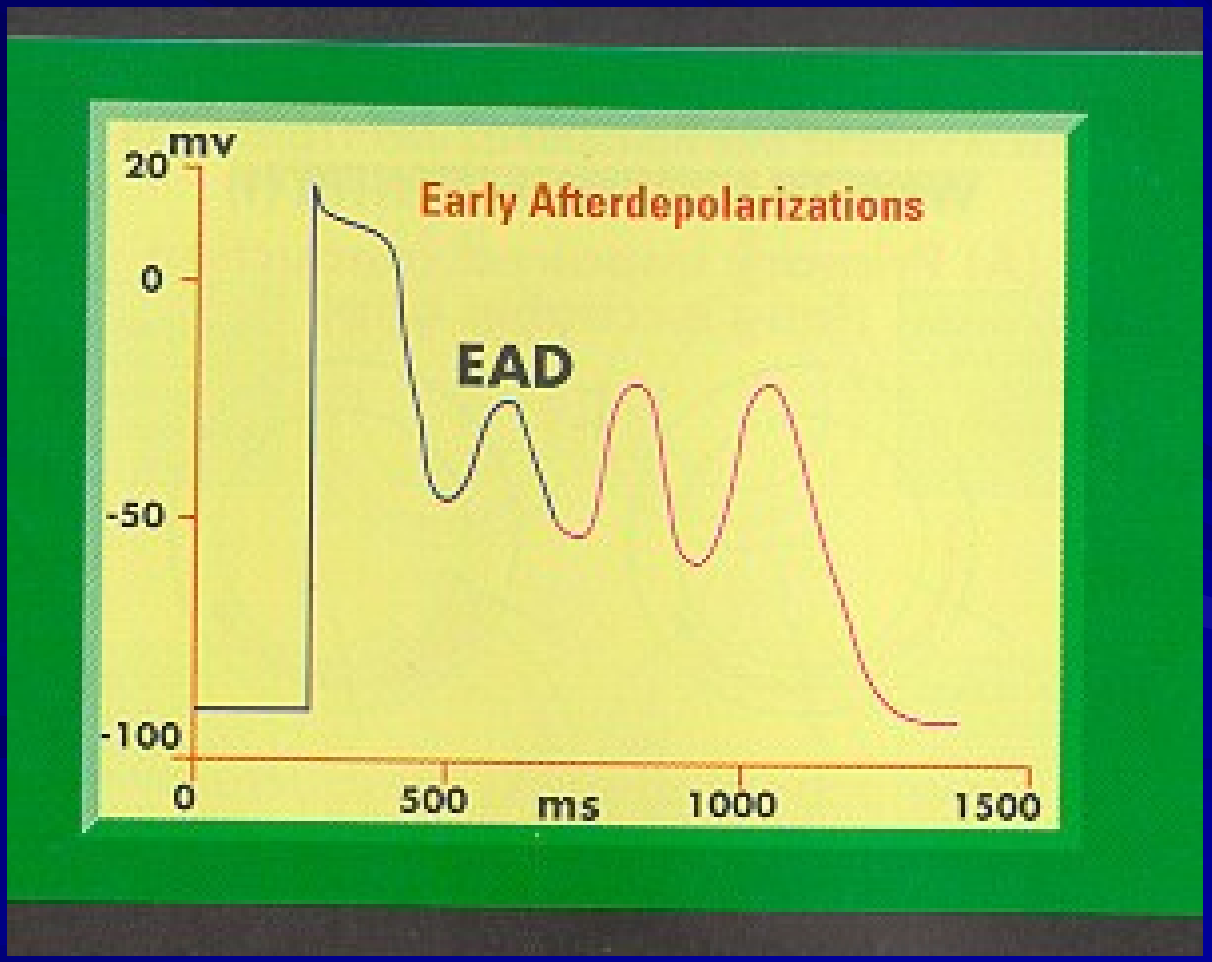


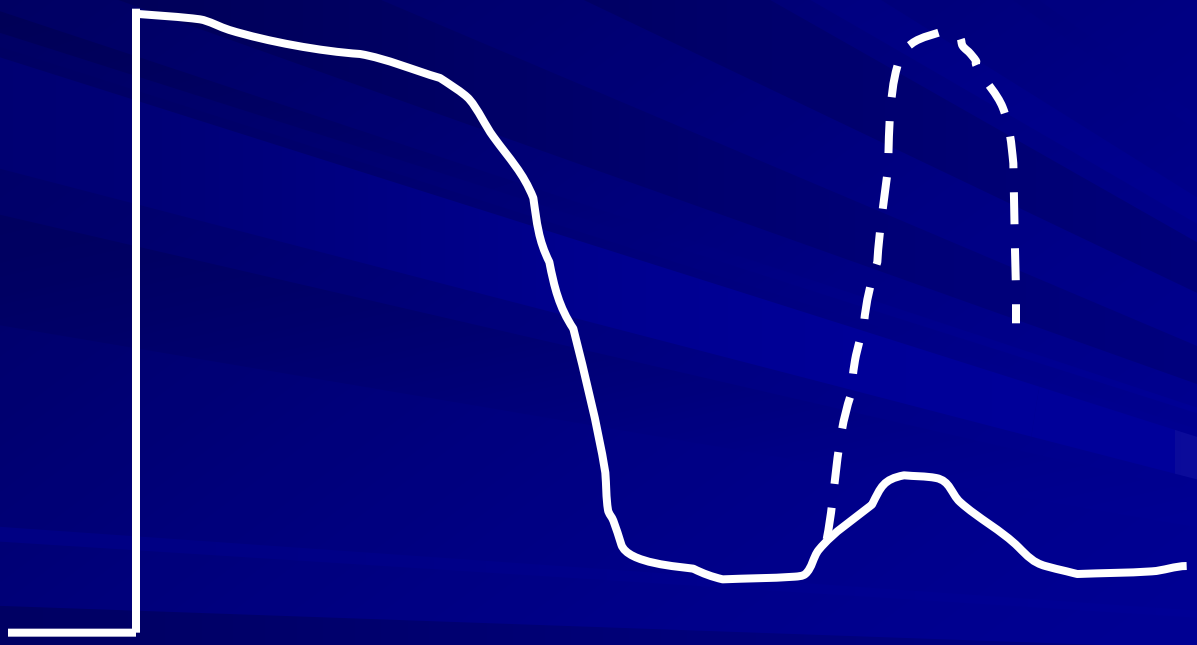
Fig. 18-13. Brady-tachy (sick sinus) syndrome. This rhythm strip shows supraventricular tachycardia (probably atrial flutter) followed by a sinus pause, an AV junctional escape beat *(J)*, and then sinus rhythm.

Triggered activity

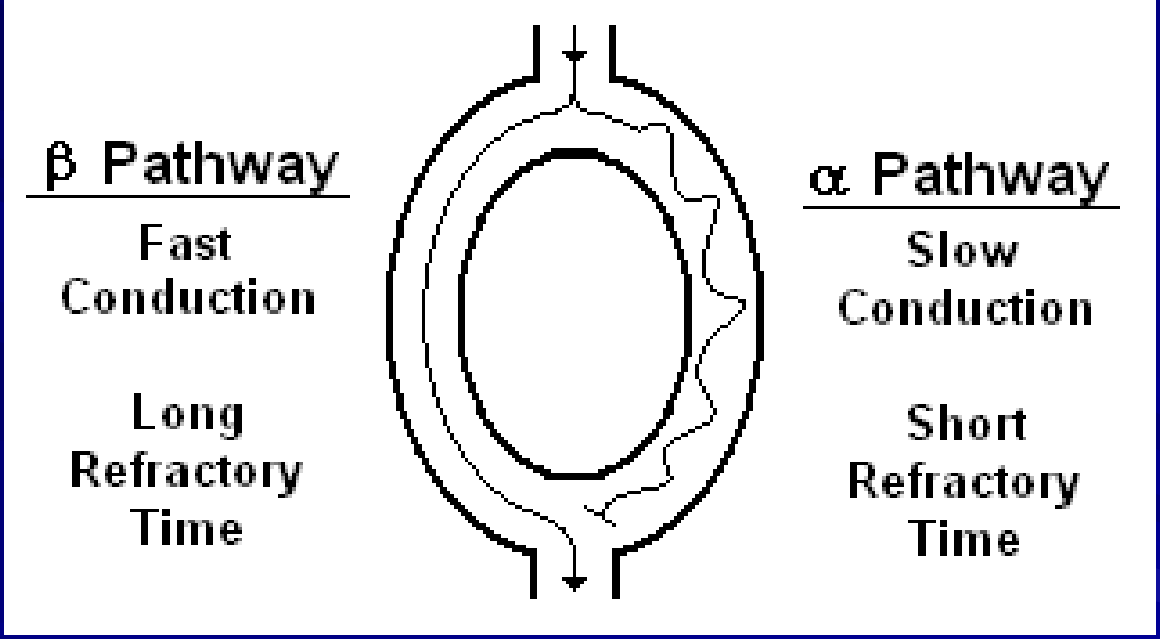
Long QT a bradycardia



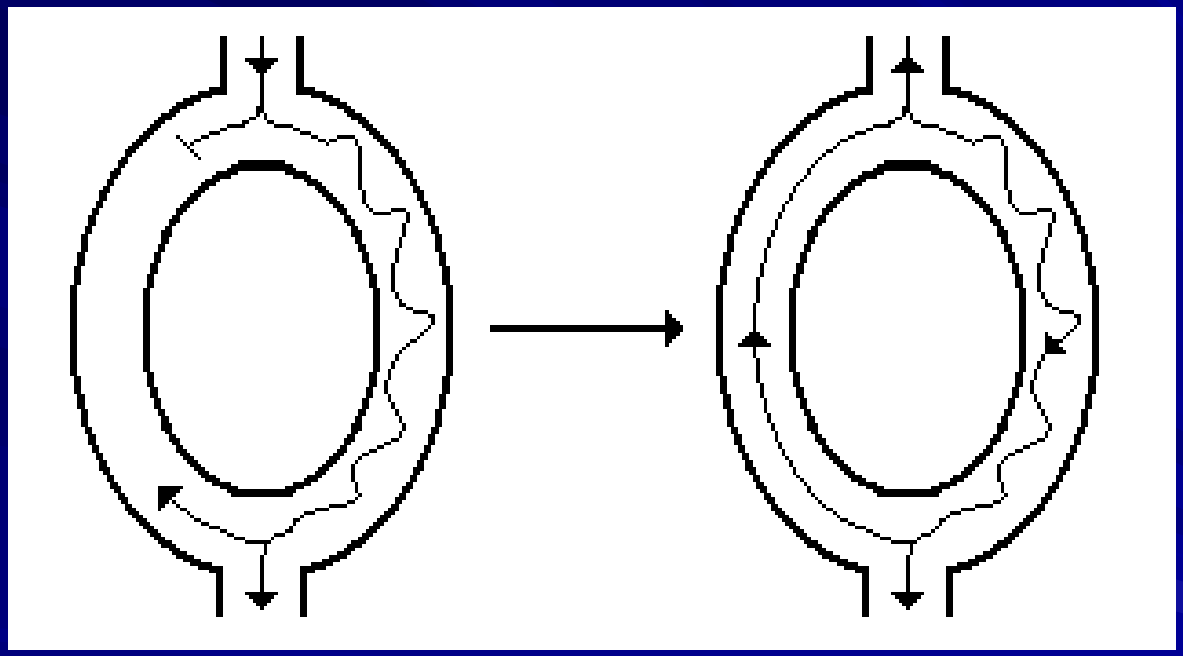
- 2. Delayed afterdepolarization (DAD)



Mechanism of Reentry



Mechanism of Reentry



Reentrant Rhythms

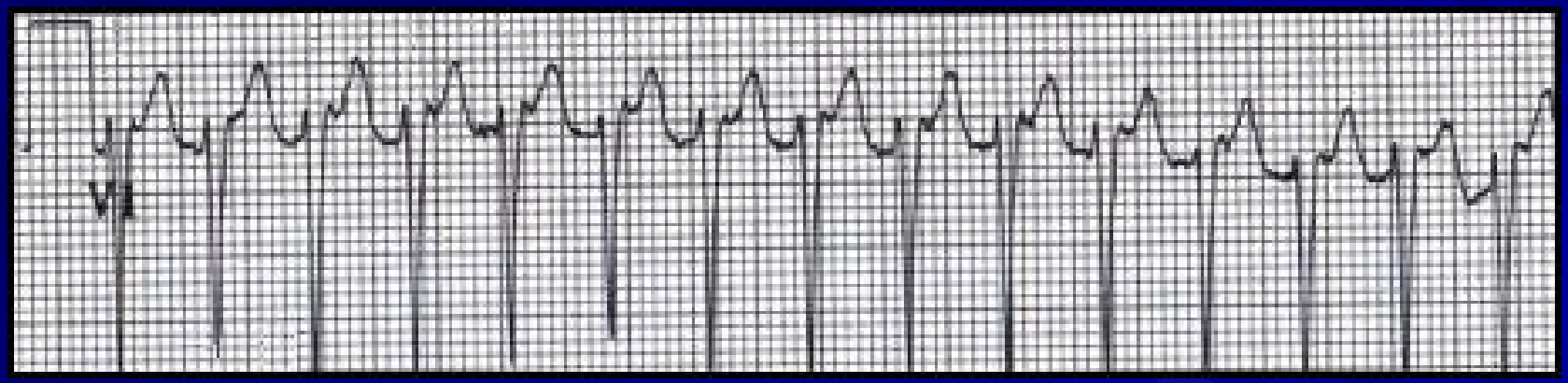
- AV nodal reentrant tachycardia (AVNRT)
- AV reentrant tachycardia (AVRT)
  - Orthodromic
  - Antidromic
- Atrial flutter
- Atrial fibrillation
- Ventricular tachycardia

Recognizing reentry on EKG

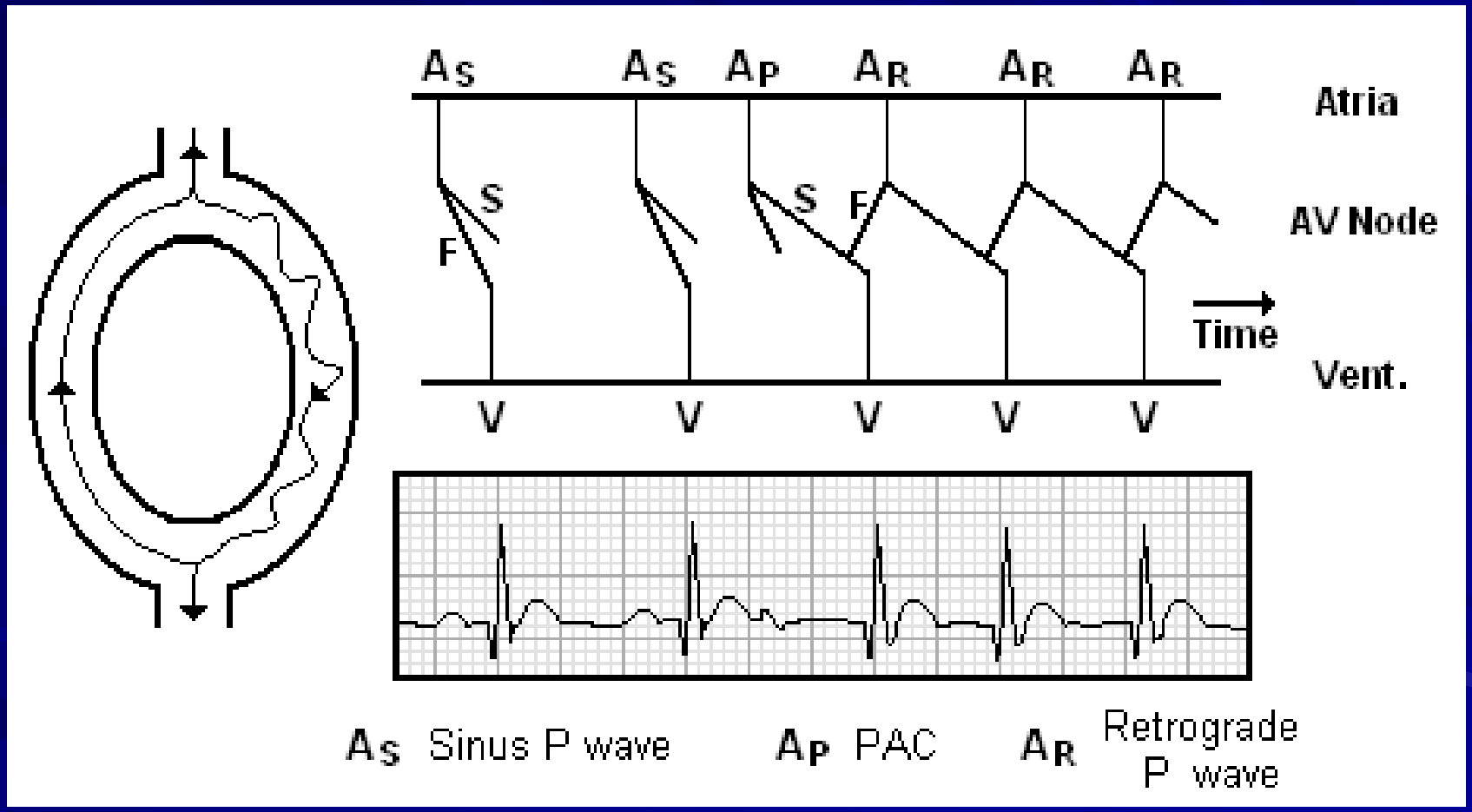
Abrupt onset and termination of the arrhythmia.

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).

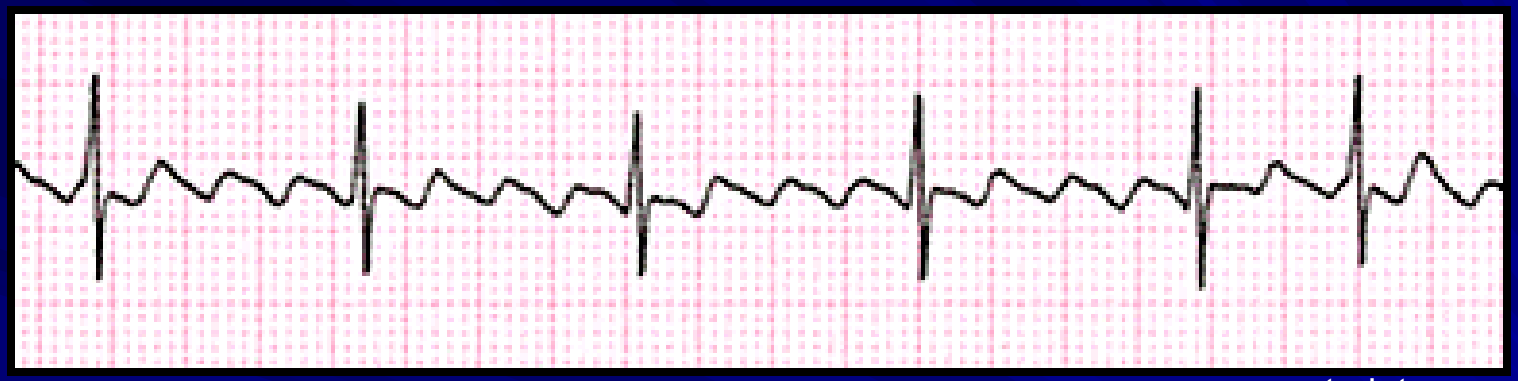
Example of AVNRT



Mechanism of AVNRT



Atrial Flutter



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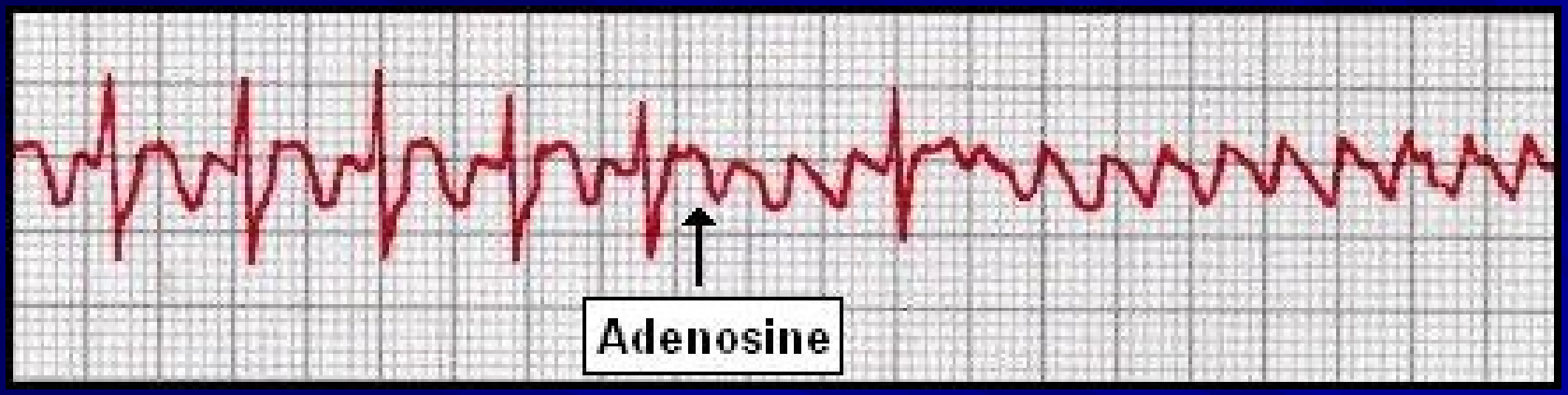
Most cases of atrial flutter are caused by a large reentrant circuit in the wall of the right atrium

EKG 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, resulting in ventricular rates of either 150 or 75 bpm

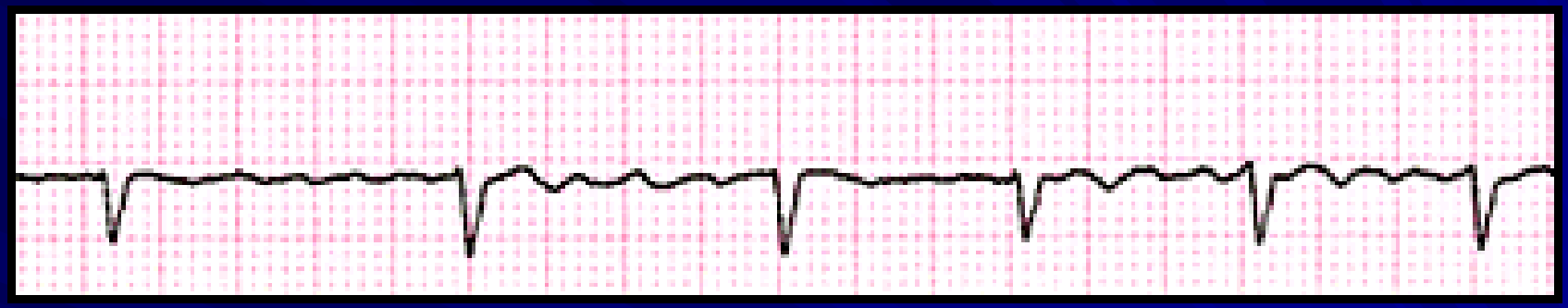
Unmasking of Flutter Waves



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.

Atrial Fibrillation



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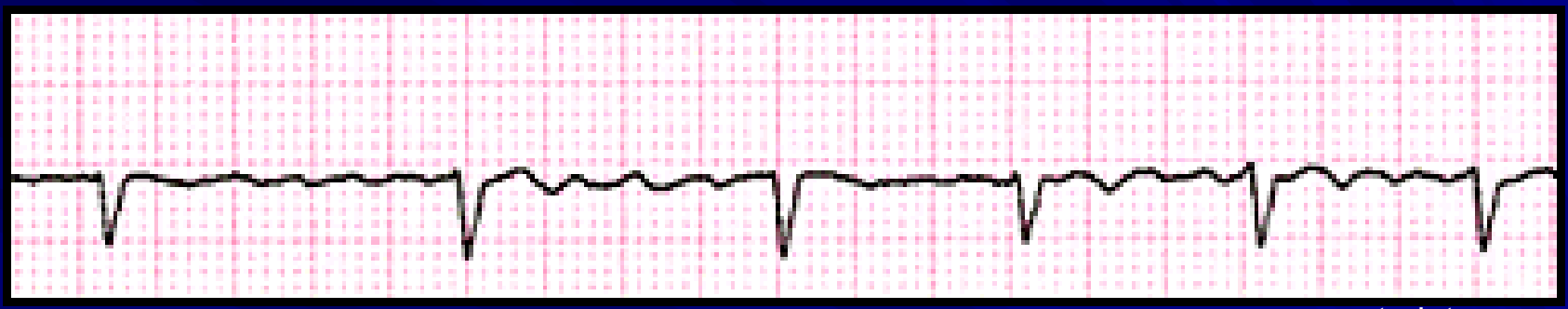
Atrial fibrillation is caused by numerous wavelets of depolarization spreading throughout the atria simultaneously, leading to an absence of coordinated atrial contraction.

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.

Atrial fibrillation is important because it can lead to:

- Hemodynamic compromise
- Systemic embolization
- Symptoms

Atrial Fibrillation



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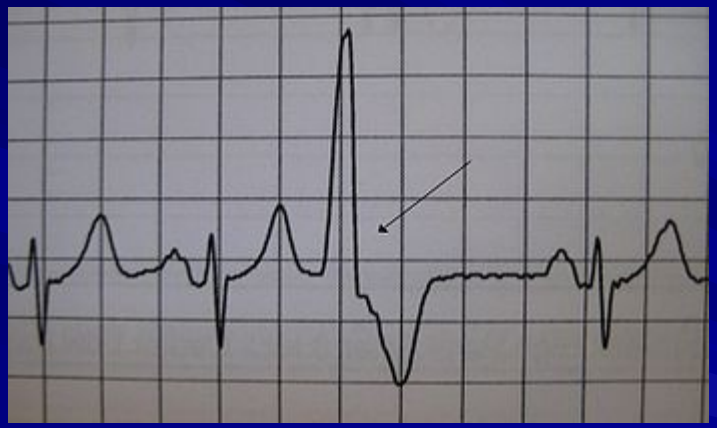
ECG Characteristics:

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

Ventricular arrhythmia

Ventricular extrasystoles (VES)

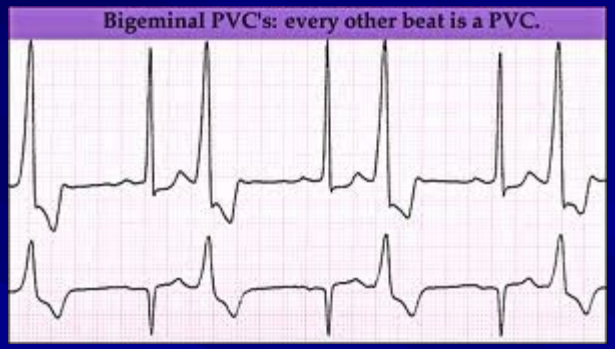
- Is caused by either reentrant signaling or enhanced automaticity in some ectopic focus



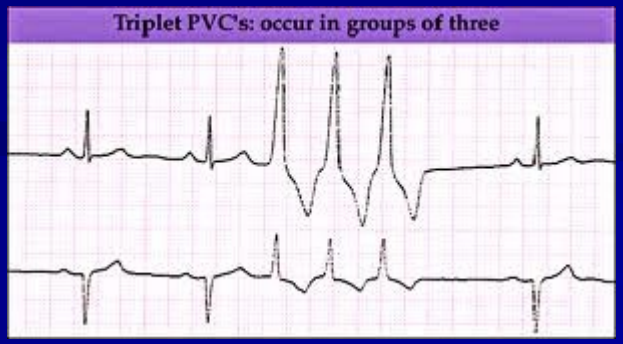
- The QRS complex is enlarged (>120ms) and has different shape

Coupling of VES

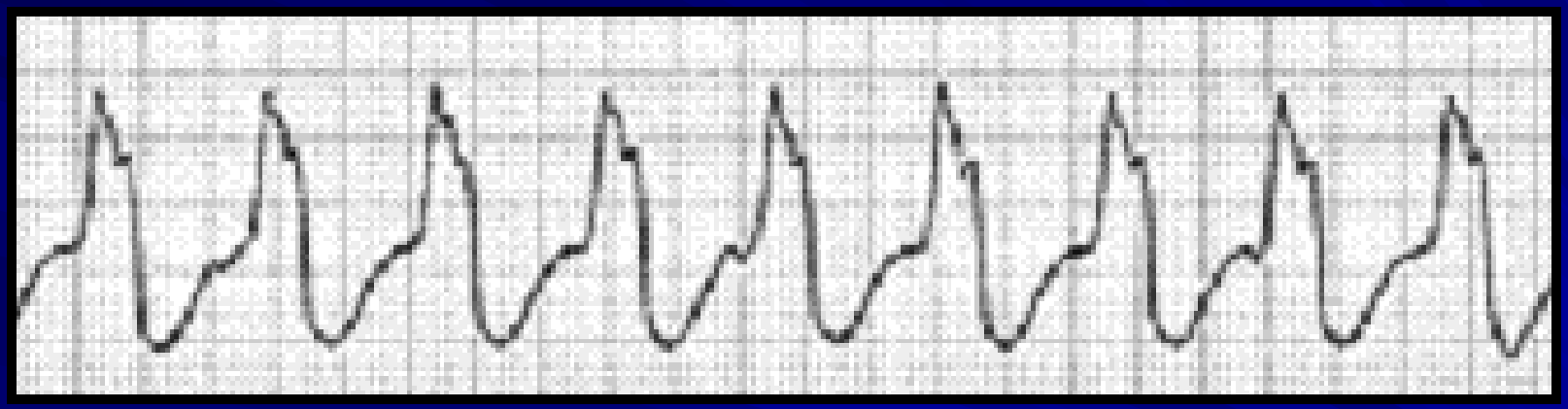
- Premature ventricular beats occurring after every normal beat are termed ventricular bigeminy, if 2 normal QRS complexes are folloved by VES, we speak of ventricular trigeminy.



- Two VES grouped together are called a couplet, three a triplet. Runs longer than 3 VES is referred as ventricular tachycardia



What is this arrhythmia?

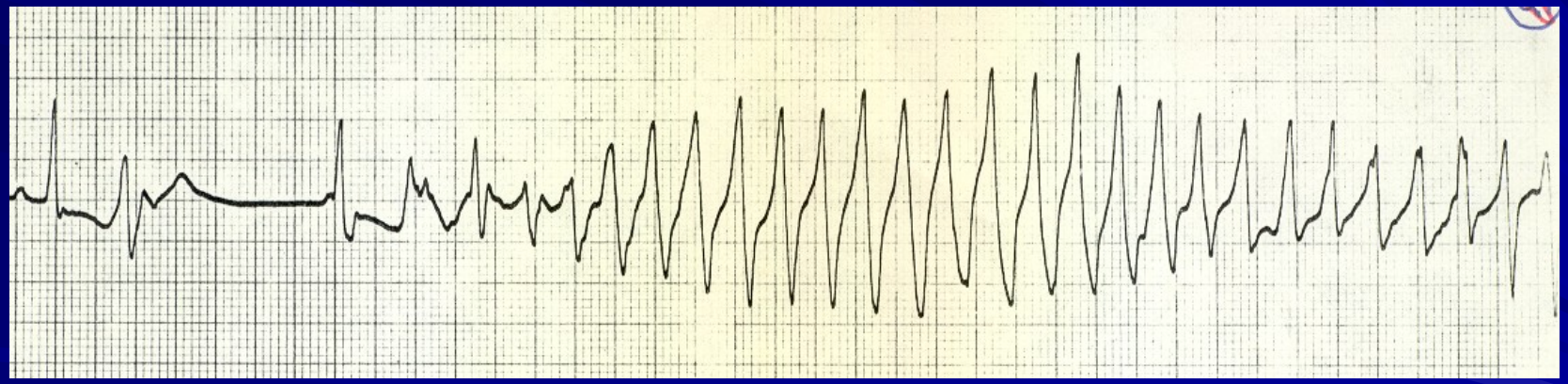


Ventricular tachycardia

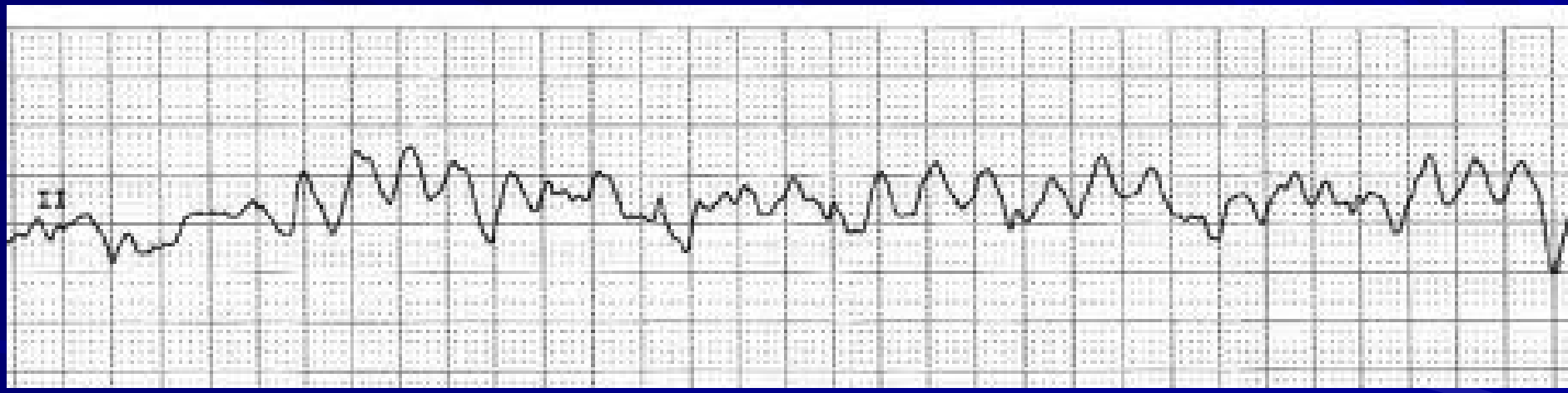
Ventricular tachycardia is usually caused by reentry, and most commonly seen in patients following myocardial infarction.

Polymorphic ventricular tachycardia – torsades de pointes

- Is connected with prolonged QT interval.
- The place of origin of the beats is moving – that leads into different shape of QRS



Ventricular fibrillation (lethal condition)



Conduction Block

Rhythms Produced by Conduction Block

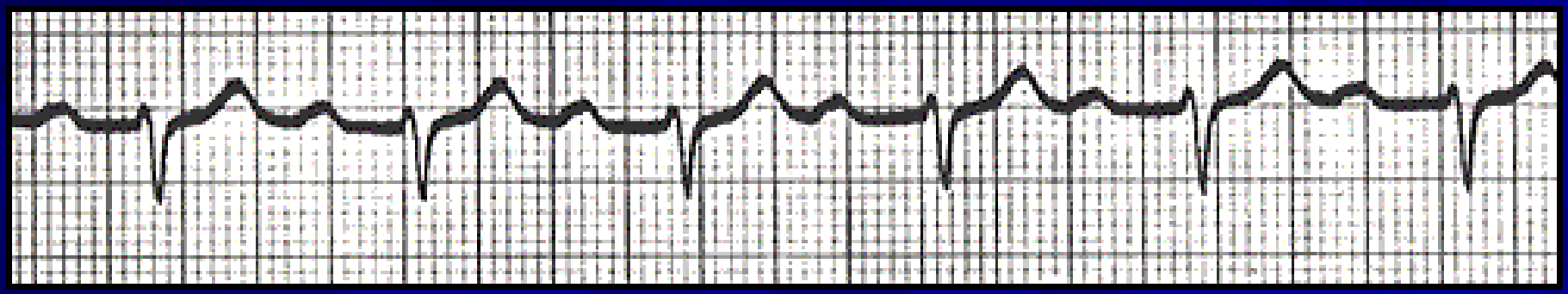
- AV Block (relatively common)
  - 1st degree AV block
  - Type 1 2nd degree AV block
  - Type 2 2nd degree AV block
  - 3rd degree AV block
- SA Block (relatively rare)

Atrioventricular Block

- AV block is a delay or failure in transmission of the cardiac impulse from atrium to ventricle.
- **Etiology:**

  Atherosclerotic heart disease; myocarditis; rheumatic fever; cardiomyopathy; drug toxicity; electrolyte disturbance, collagen disease, lev's disease.

1st Degree AV Block

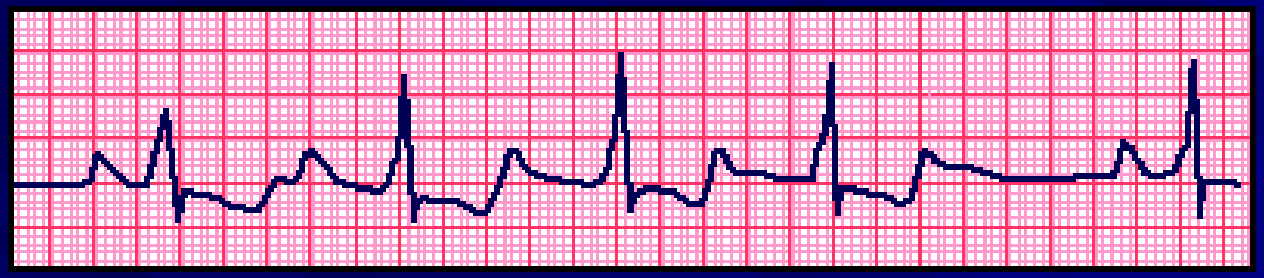


The Alan E. Lindsay ECG Learning Center ; http://medstat.med.utah.edu/kw/ecg/

ECG Characteristics: Prolongation of the PR interval, which is constant

All P waves are conducted

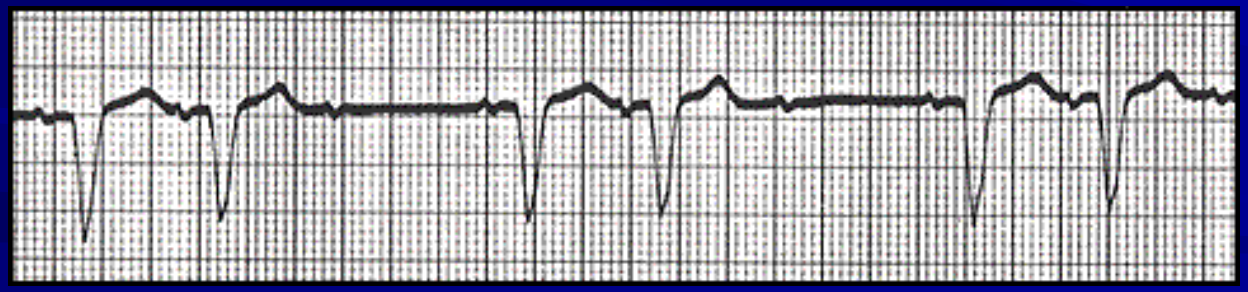
2nd Degree AV Block



Type 1
(Wenckebach)

EKG Characteristics: Progressive prolongation of the PR interval until a P wave is not conducted.

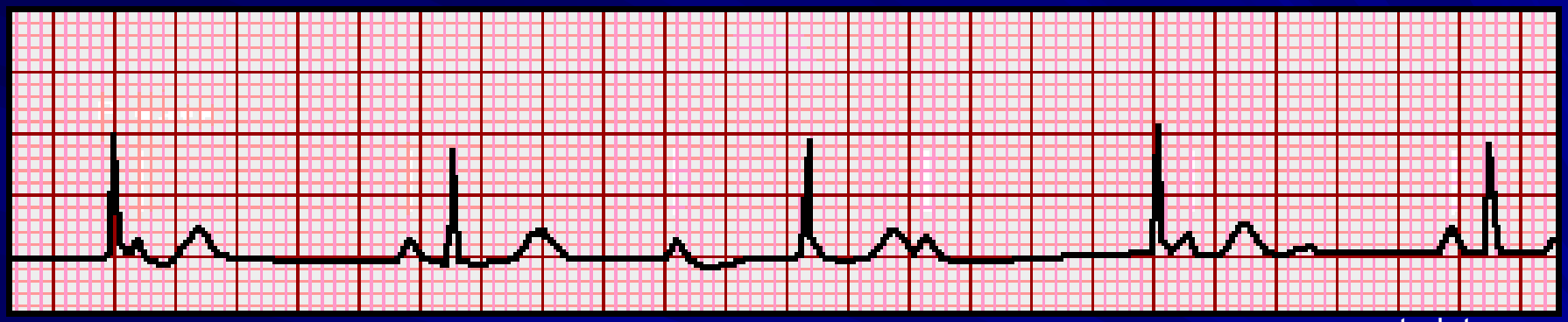
As the PR interval prolongs, the RR interval actually shortens



Type 2

EKG Characteristics: Constant PR interval with intermittent failure to conduct

3rd Degree (Complete) AV Block



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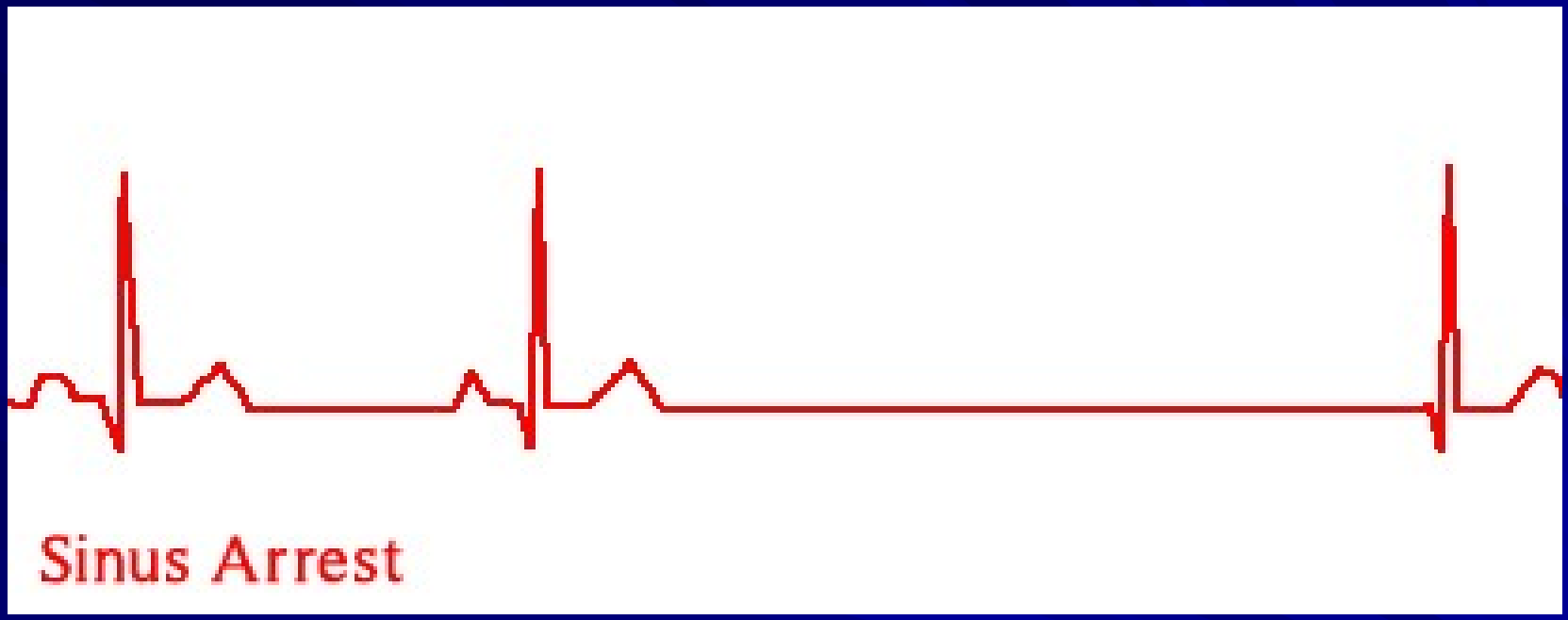
EKG Characteristics:

- No relationship between P waves and QRS complexes
- Relatively constant PP intervals and RR intervals
- Greater number of P waves than QRS complexes

SA arrest with compensatory AV activity



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

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

Intraventricular Block

- **Intraventricular conduction system:**

1. Right bundle branch
2. Left bundle branch
3. Left anterior fascicular
4. Left posterior fascicular

Intraventricular Block

- **Etiology:**
- Myocarditis, valve disease, cardiomyopathy, CAD, hypertension, pulmonary heart disease, drug toxicity, Lenegre disease, Lev's disease et al.
- **Manifestation:**
- Single fascicular or bifascicular block is asymptom; tri-fascicular block may have dizziness; palpitation, syncope and Adams-stokes syndrome

Premature contractions

- The term “premature contractions” are used to describe non sinus beats.
- Common arrhythmia
- The morbidity rate is 3-5%

Atrial premature contractions (APCs)

- APCs arising from somewhere in either the left or the right atrium.
- **Causes:** rheumatic heart disease, CAD, hypertension, hyperthyroidism, hypokalemia
- **Symptoms:** many patients have no symptom, some have palpitation, chest incomfortable.
- **Therapy:** Needn't therapy in the patients without heart disease. Can be treated with ß-blocker, propafenone, moricizine or verapamil.

Ventricular Premature Contractions (VPCs)

- **Etiology:**

1. Occur in normal person
2. Myocarditis, CAD, valve heart disease, hyperthyroidism, Drug toxicity (digoxin, quinidine and anti-anxiety drug)
3. electrolyte disturbance, anxiety, drinking, coffee

Pre-excitation syndrome (W-P-W syndrome)

- There are several type of accessory pathway

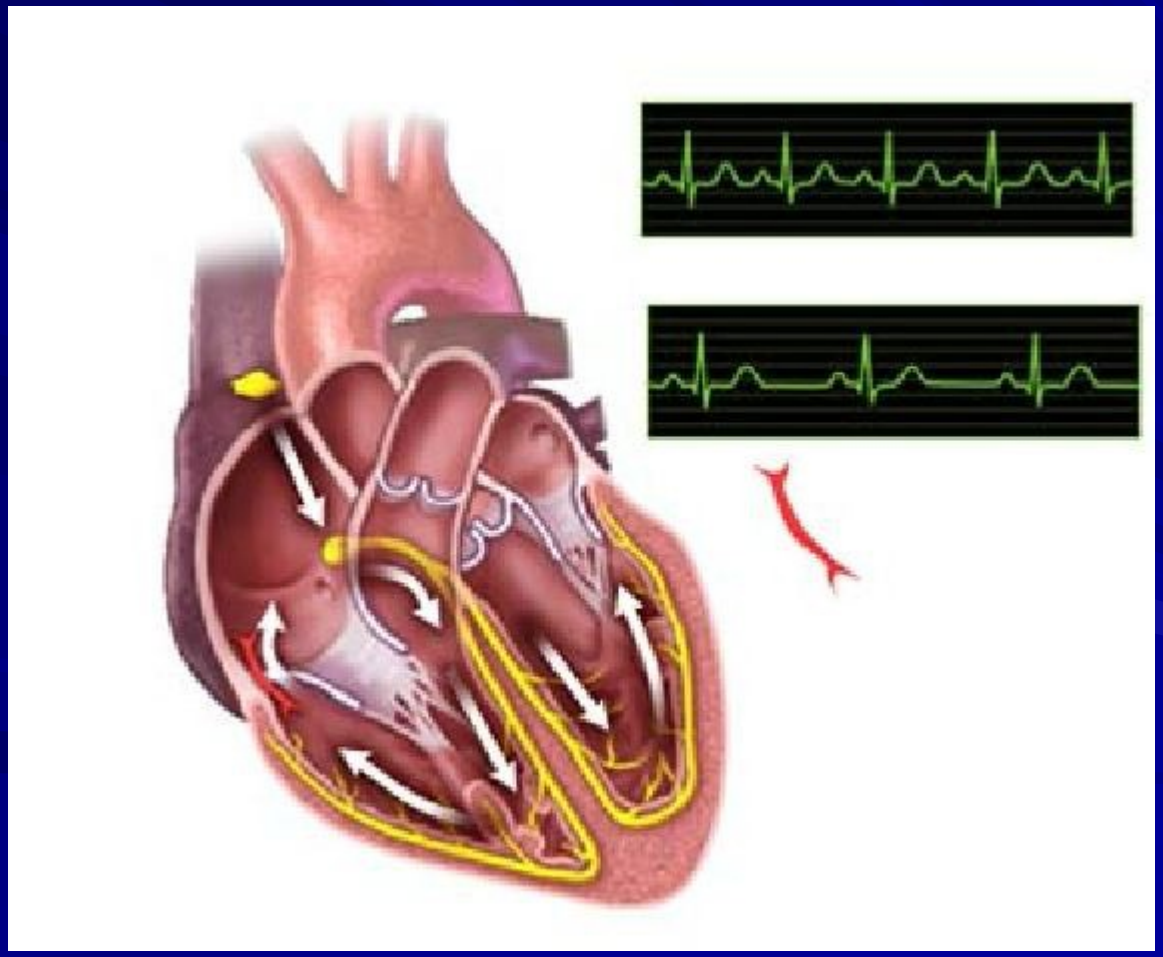
1. Kent: adjacent atrial and ventricular
2. James: adjacent atrial and his bundle
3. Mahaim: adjacent lower part of the AVN and ventricular

- Usually no structure heart disease, occur in any age individual

WPW syndrome

- **Manifestation:**
- Palpitation, syncope, dizziness
- Arrhythmia: 80% tachycardia is AVRT, 15-30% is AFi, 5% is AF,
- May induce ventricular fibrillation

Wolff-Parkinson White Syndrome (WPW) is a condition in which the heart beats too fast due to abnormal, extra electrical pathways between the heart's atrium and ventriculum .



Thank you for your attention