ABC of clinical ECG

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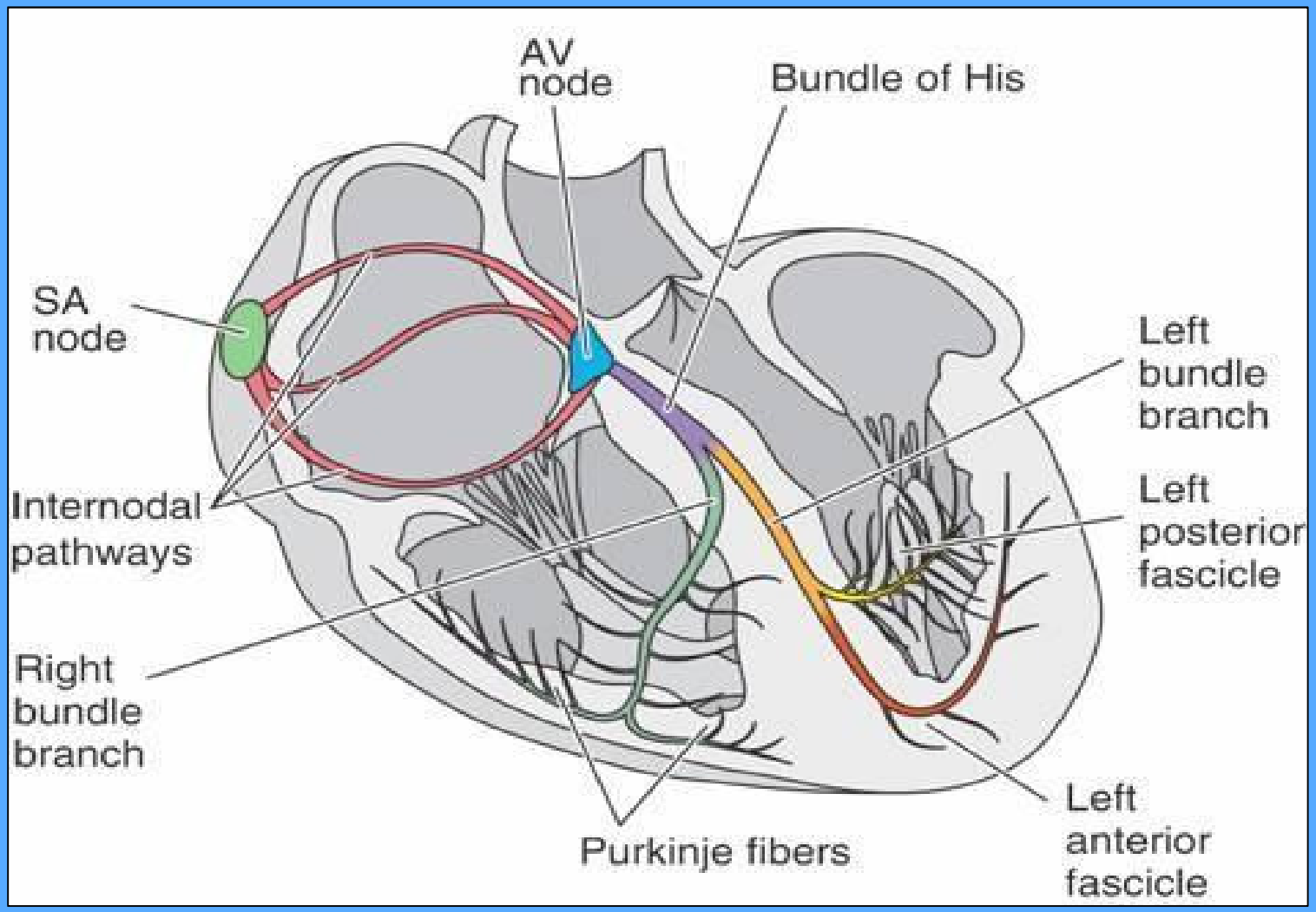
Goal with this seminar

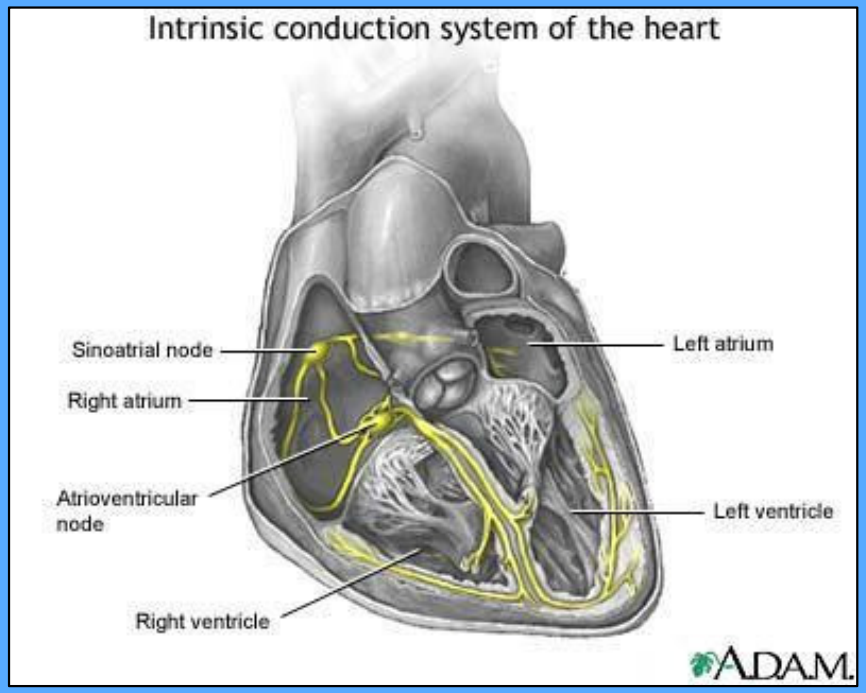
- Learn a systematic approach for analysing ECGs:” 5+1”
- Recognize and understand normal ECGs
- Interpret abnormalitets in ryhtm, conduction or morphology

Anatomy

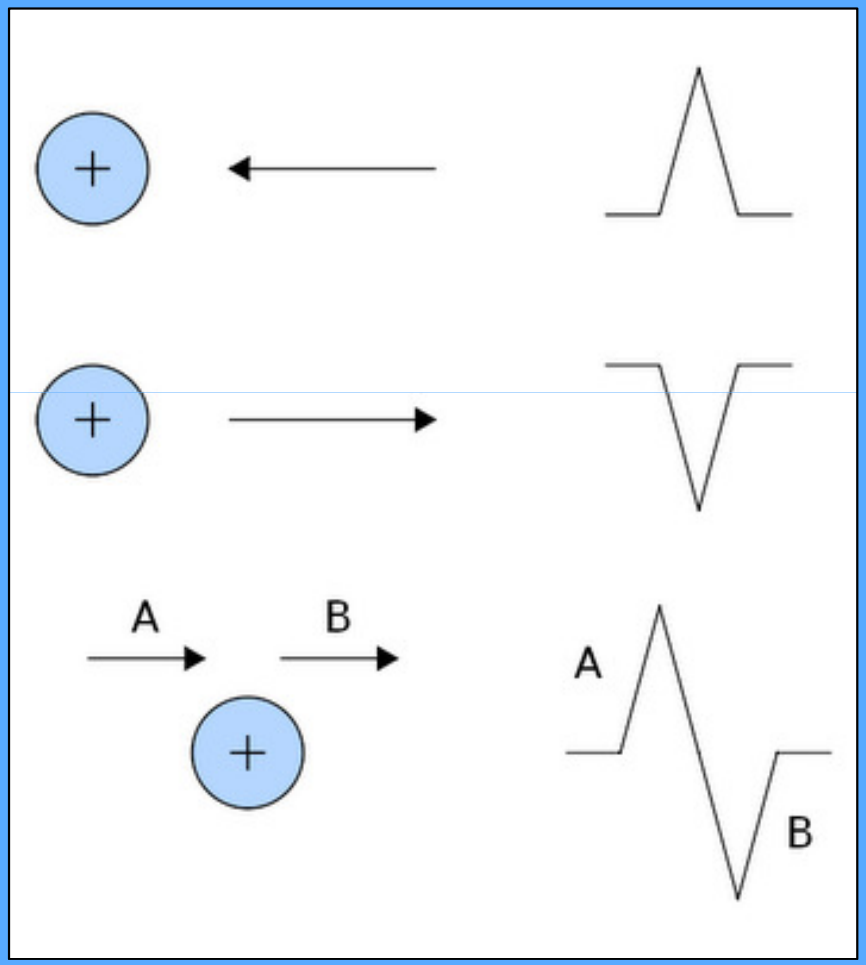

AV node
Bundle of His
SA node
Left bundle branch
Internodal pathways
Left posterior fascicle
Right bundle branch
Purkinje fibers
Left anterior fascicle

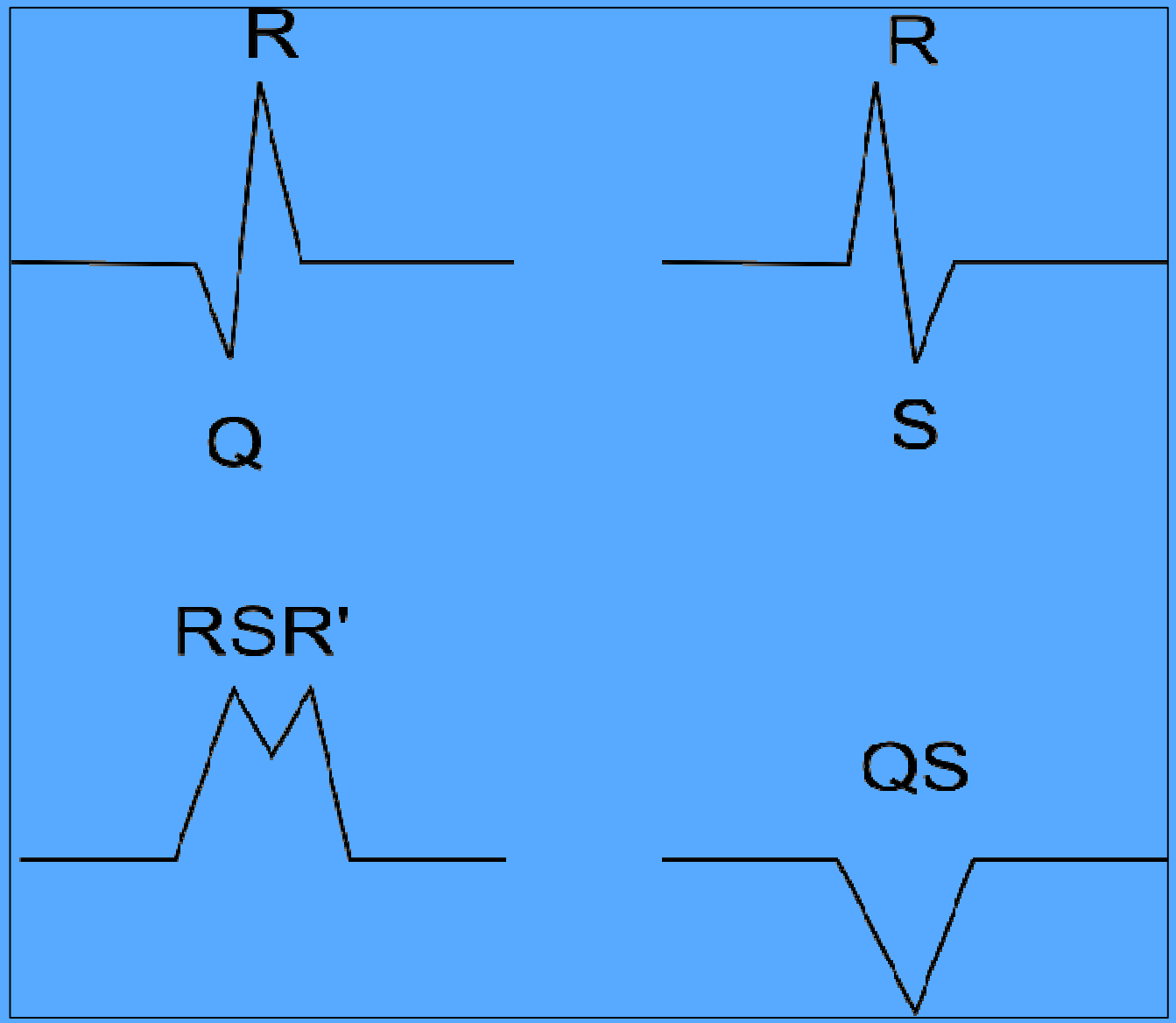
Physiology

- The sinoatrial node (SA node) contains the fastest physiological pacemaker cells of the heart; therefore, they determine the heart rate
- SA-node initiates depolarization, first atria, than ventricles by help of specialized conduction system
- Repolarization follows depolarization



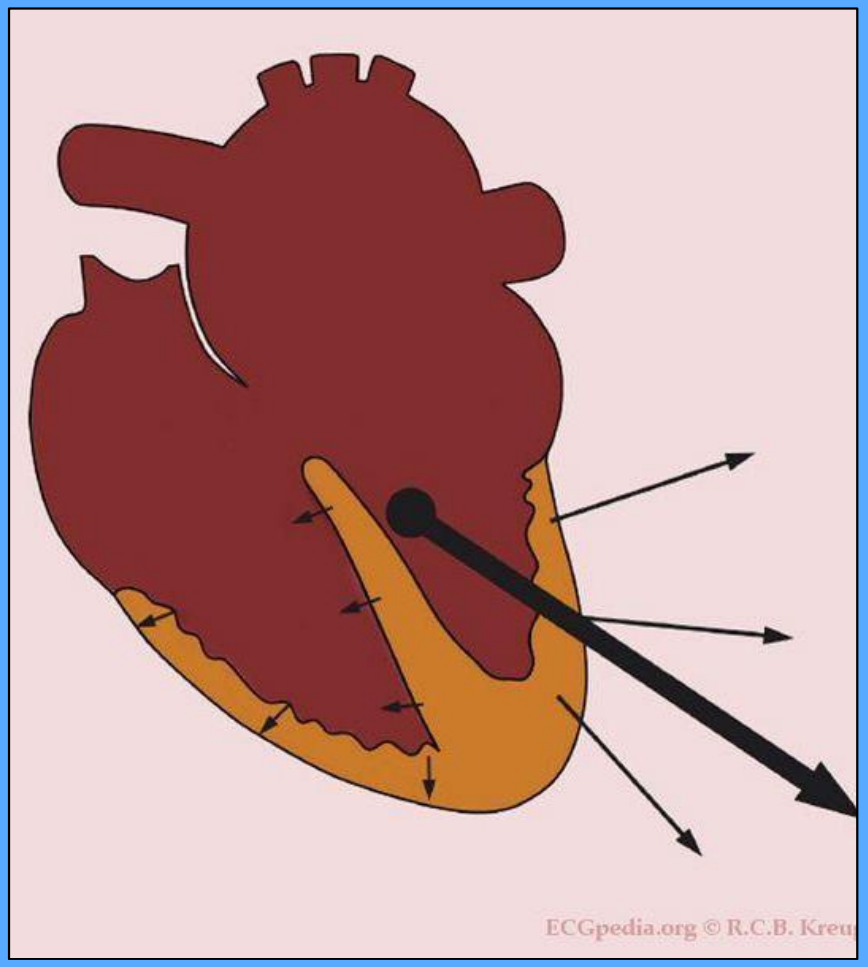
- Depolarization og myocardial cells causes pos. electrical charges which can be measured be electrodes
- Electrical charges moving towards an electrode causes positive deflection relative to the isoelectric line
- Electrical charges moving away from an electrode causes negative deflection



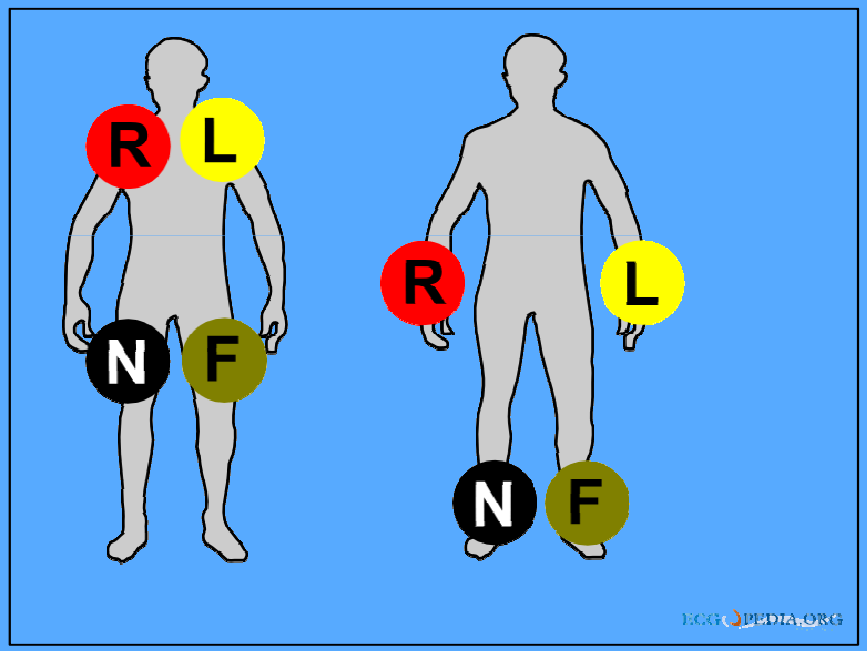

R
Q
R
S
RSR'
QS

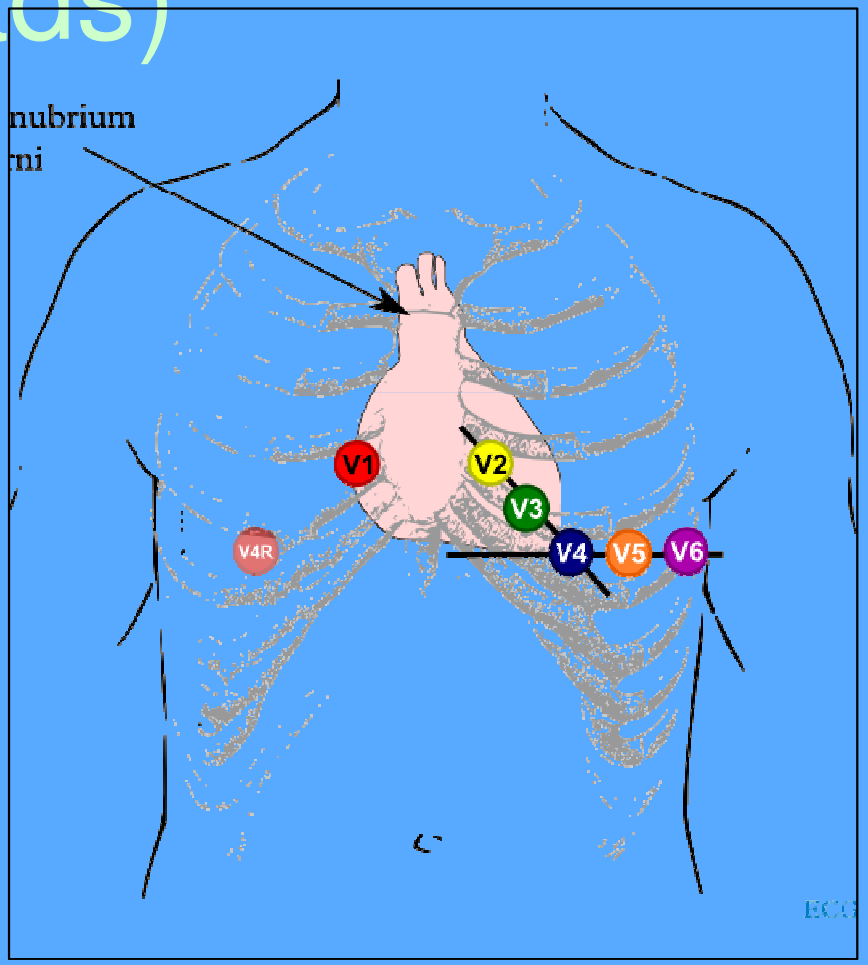
Electrical axis of heart

- The electrical heart axis is an average of all depolarizations in the heart.
- The depolarization wave begins in the right atrium and proceeds to the left and right ventricle.
- Because the left ventricle wall is thicker than the right wall, the arrow indicating the direction of the depolarization wave is directed to the left.



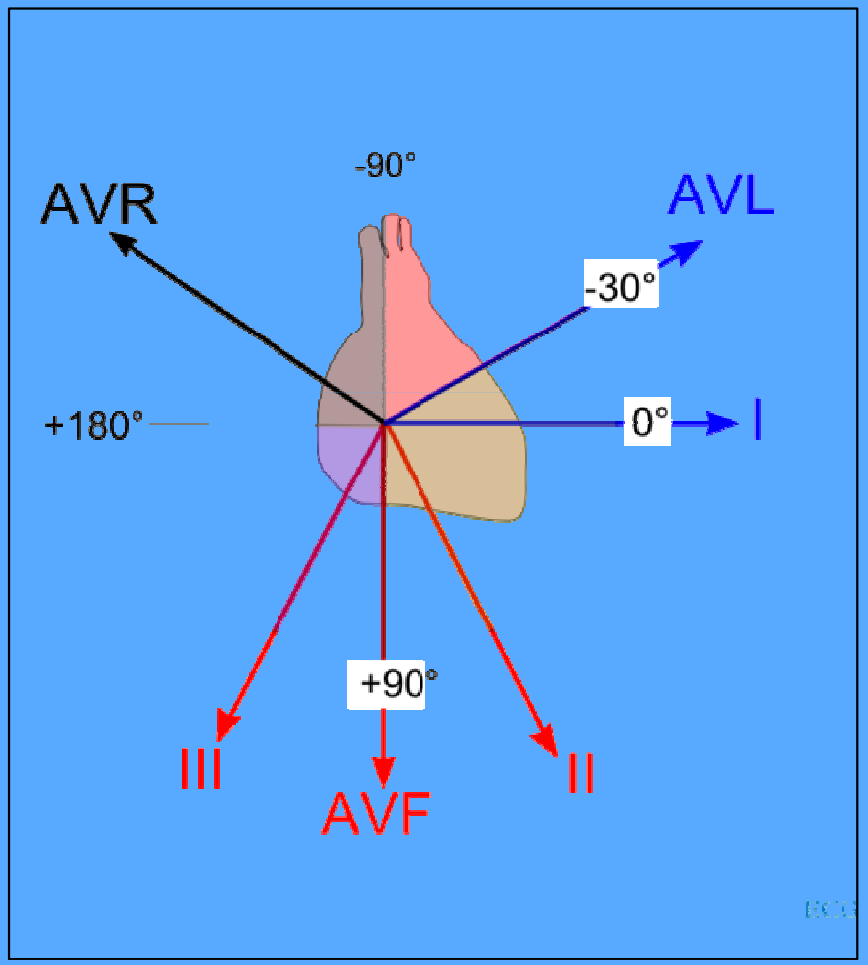
Placements of electrodes (leads)



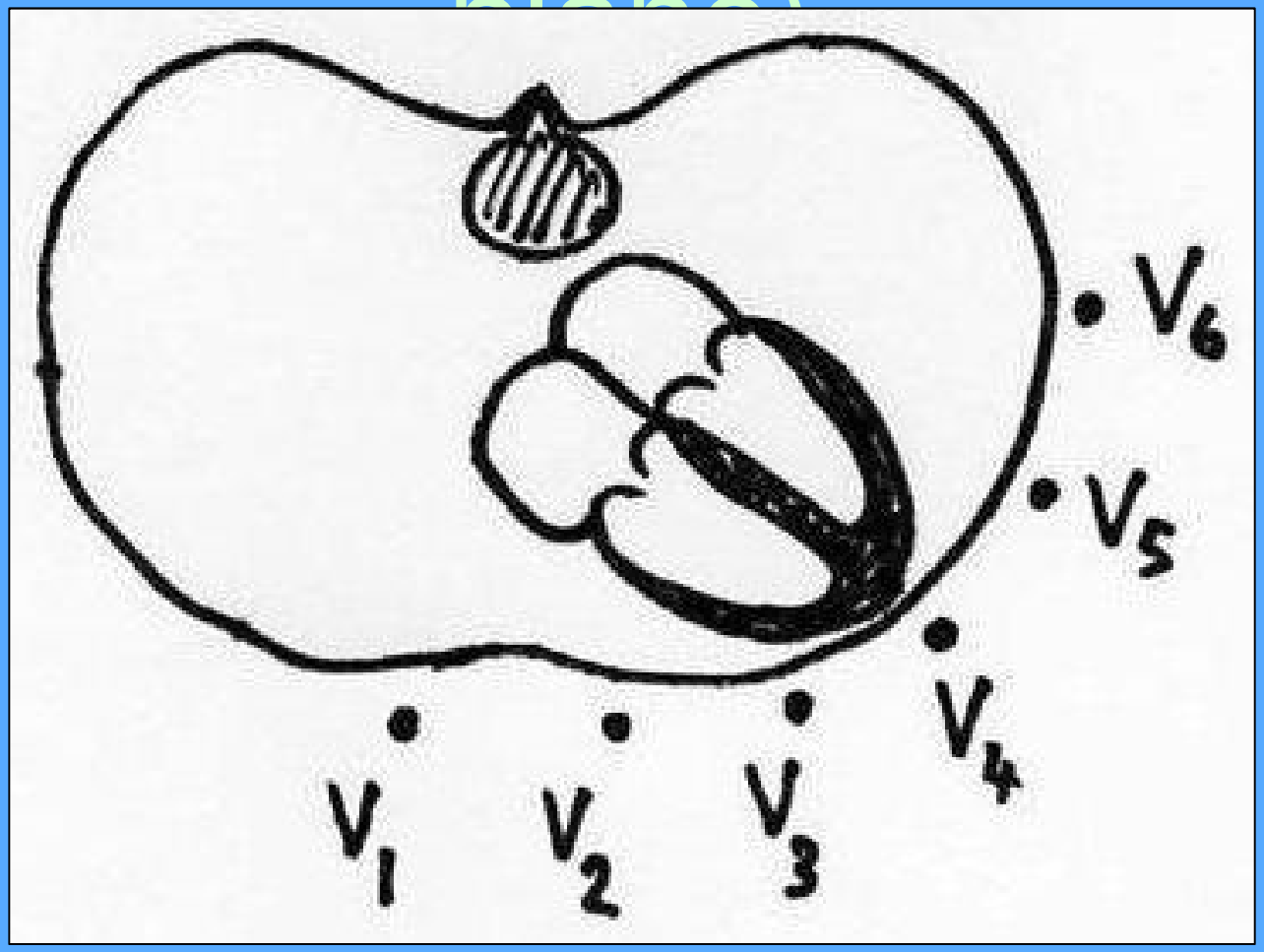


Limb leads (Vertical plane)

- Info from limb electrodes are combined to produce six limb leads
- Try to look at the leads as eyes ”looking” at the heart from different angles
- Group the leads together into right, left and inferior-posterior

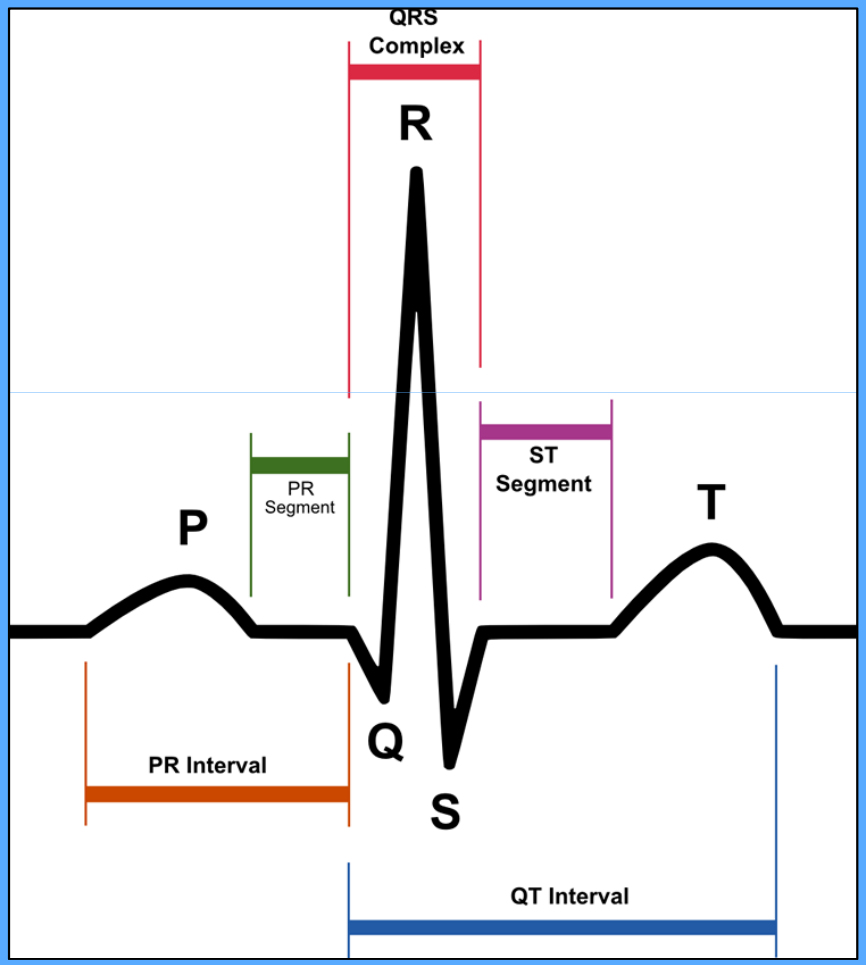


Chest leads (horizontal plane)



Normal ECG intervalls

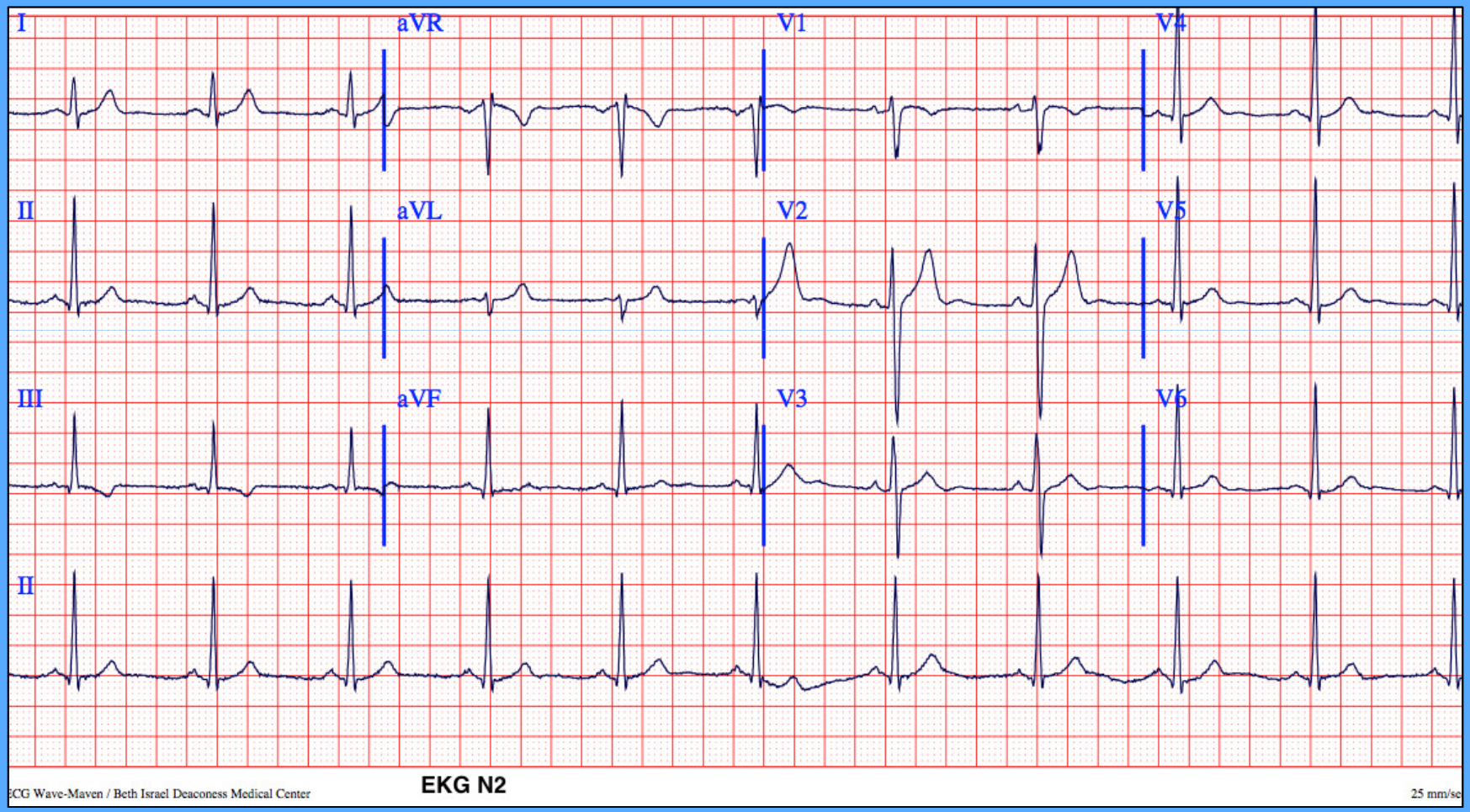
- The PQ interval starts at the beginning of the atrial contraction and ends at the beginning of the ventricular contraction (0,12 – 0,20 seconds)
- The QRS duration indicates how fast the ventricles depolarize (normal < 0,10 seconds)
- The normal QTc (corrected) interval indicates how fast the ventricles are repolarized, becoming ready for a new cycle. (below 0.45 seconds in men and below 0,46 in women)



Normal ECG – ”Sinus ryhtm”

- A P-wave (atrial contraction) precedes every QRS complex
- The rhythm is regular, but varies slightly during respirations
- The rate ranges between 60 and 100 beats per minute
- The P waves maximum height at 2.5 mm in II and/or III
- The P wave is positive in I and II, and biphasic in V1

Normal ECG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ECG Wave-Maven / Beth Israel Deaconess Medical Center
EKG N2
25 mm/se

Arrhytmias

Classifications based on heart rate:

1. Tachycardia (HR > 100 bpm)
2. Bradycardia (HR < 60 bpm)

Classification based on origin of impulse:

1. Supraventricular – ”Narrow QRS complex” (< 0.12 ms)
2. Ventricular - ”Wide QRS complex” (> 0.12 ms)

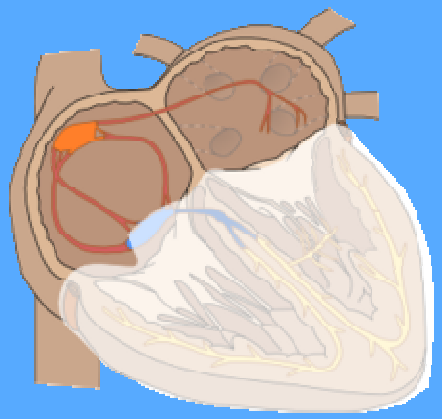
Tachyarrhytmias

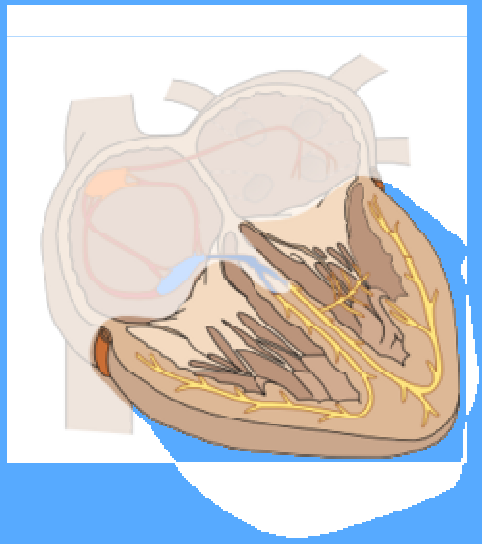
Supraventricular

Atrial

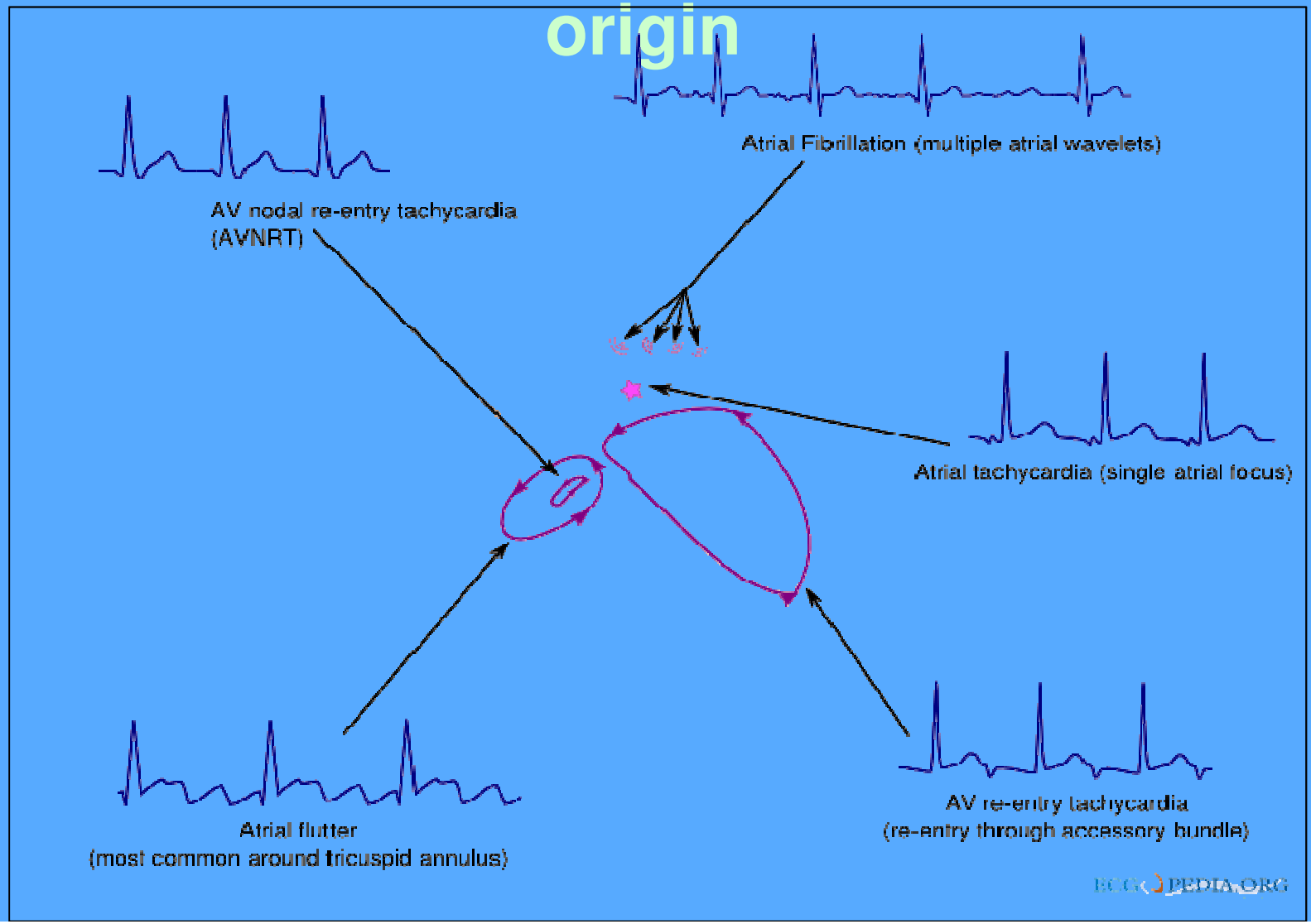
Nodal

Ventricular

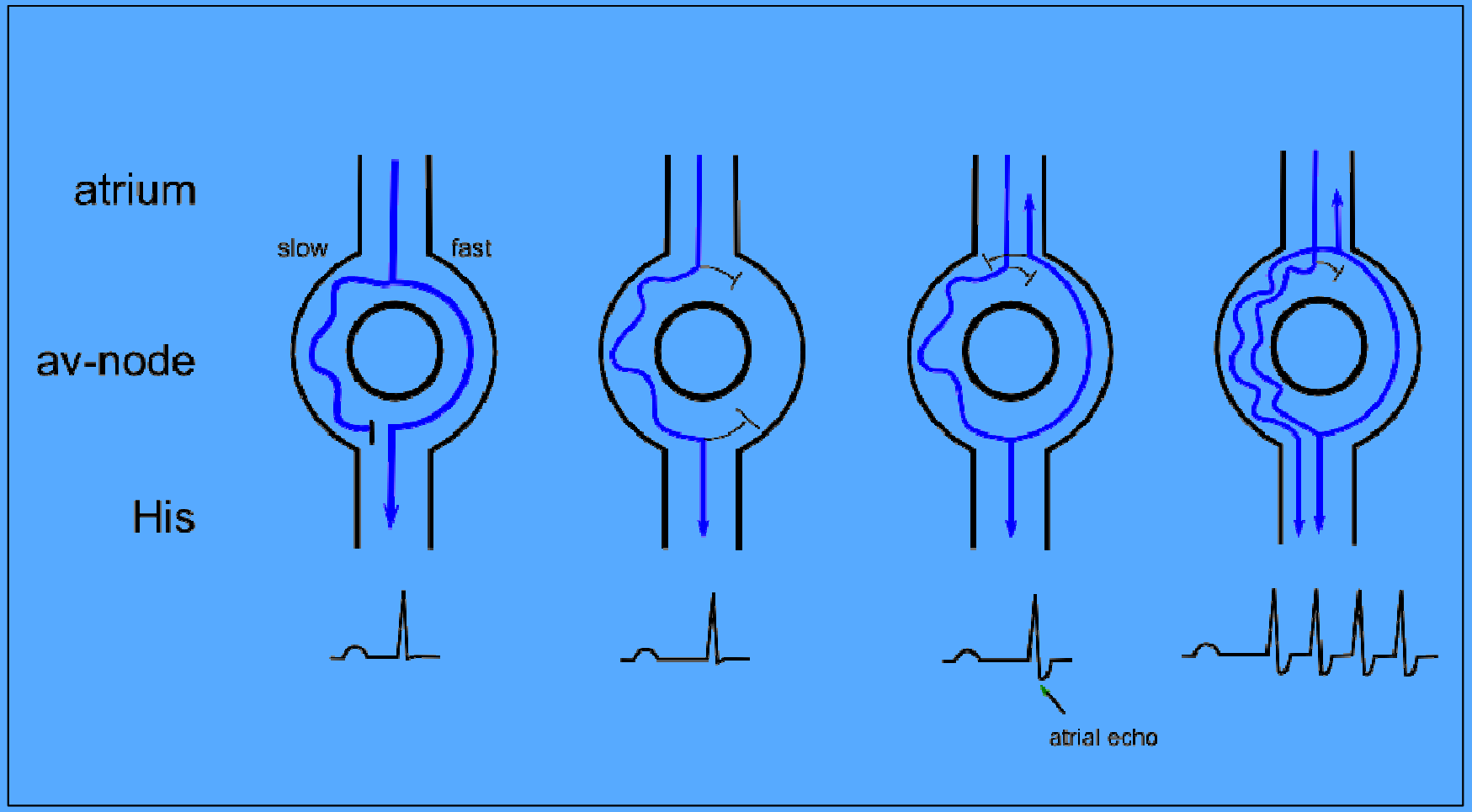




An overview of pathological supraventricular arrhythmias and their origin



Re-entry mechanism



Sinustachycardia

Atrial frequency	100 – 180 bpm
Ventricular frequency	100 – 180 bpm
Regularity	Regular
Origin	Sinus node
P-wave	Positive in II, aVF
Effect of Adenosine	No (can lead to temporary AV block

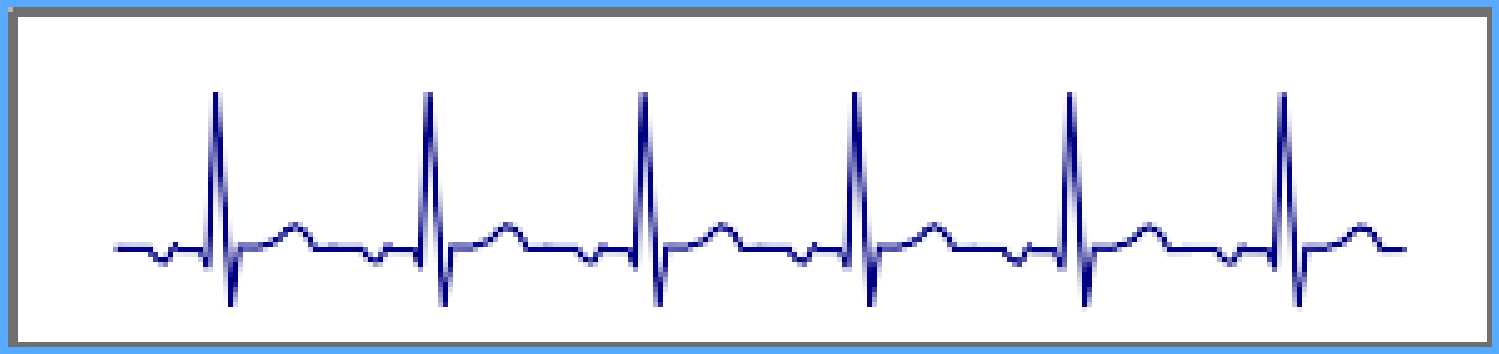
Sinustachycardia

Causes:

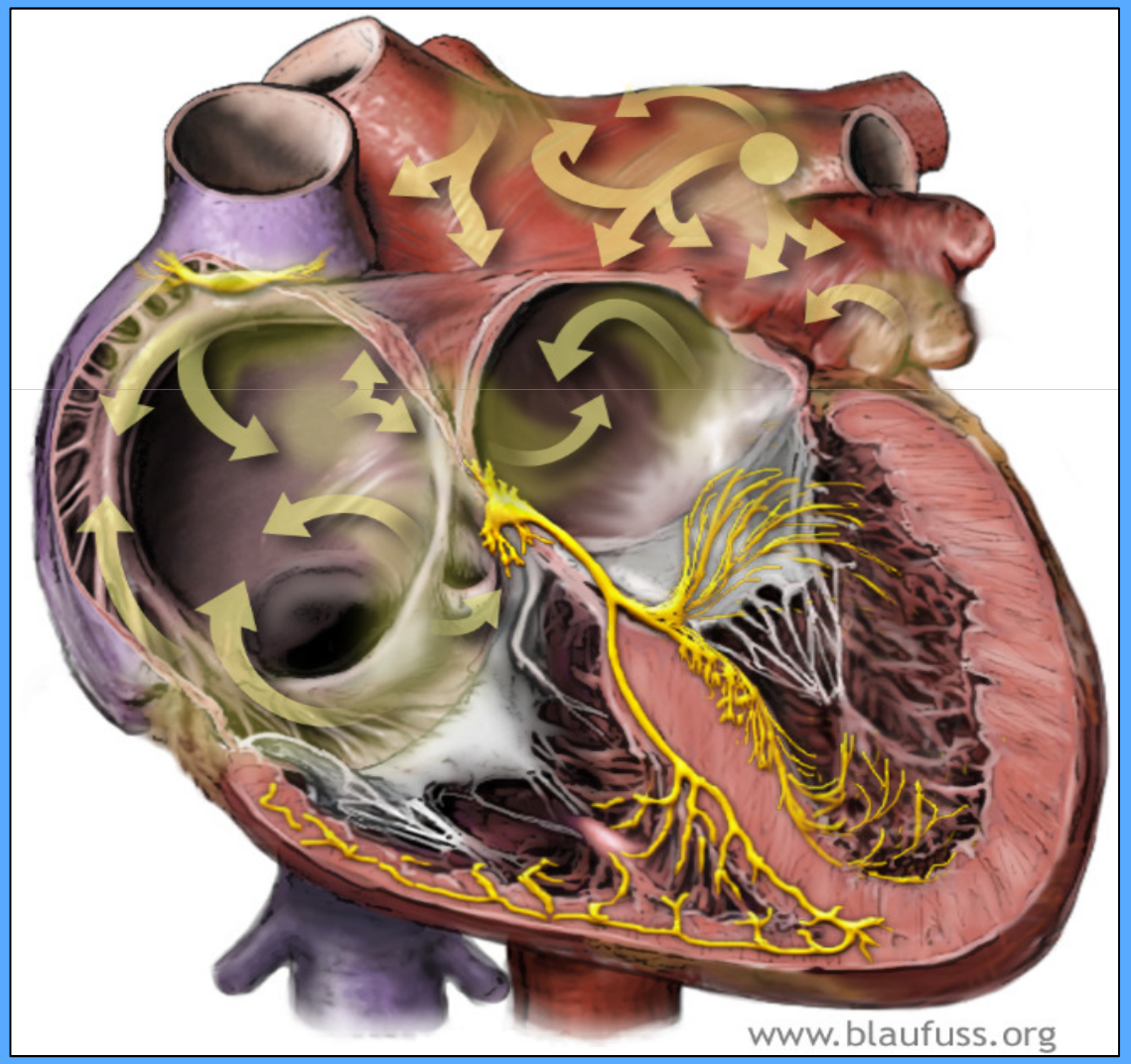
- Exercise, anxiety, alcohol, caffeine, drugs
- Fever
- Hypoxia
- Bleeding
- Anemia
- Hyperthyroidism
- +++++

Atrial tachycardia

Atrial frequency	> 100
Ventricular frequency	>100
Regularity	Regular
Origin	Ectopic foci in atrium (re-entry)
P-wave	Negative in I, aVF (different morphology)
Effect of Adenosine / Vagal stimulation	Slow down rythm (AV-conduction)

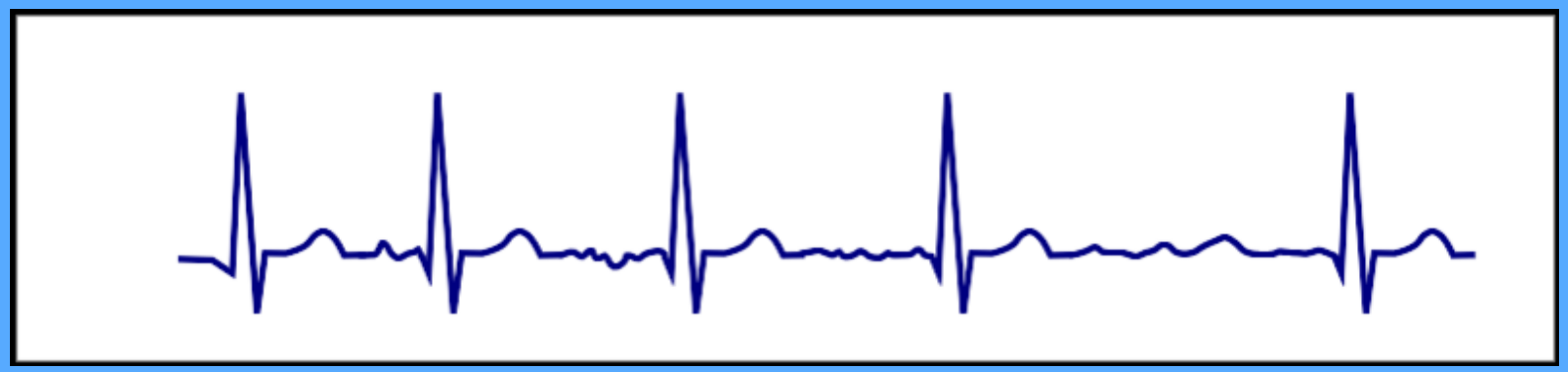


Atrial fibrillaton (AF)

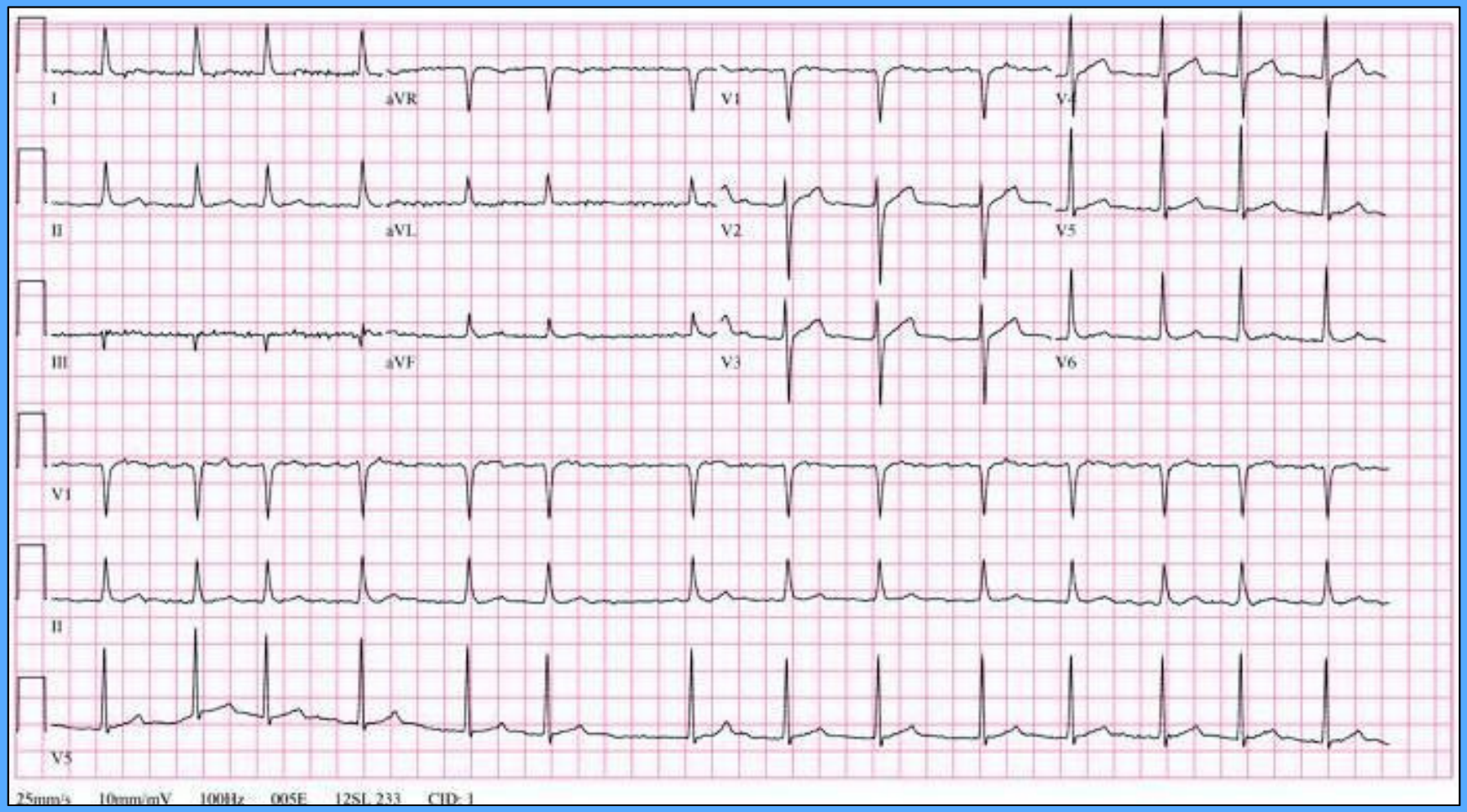


Atrial Fibrillation (AF)

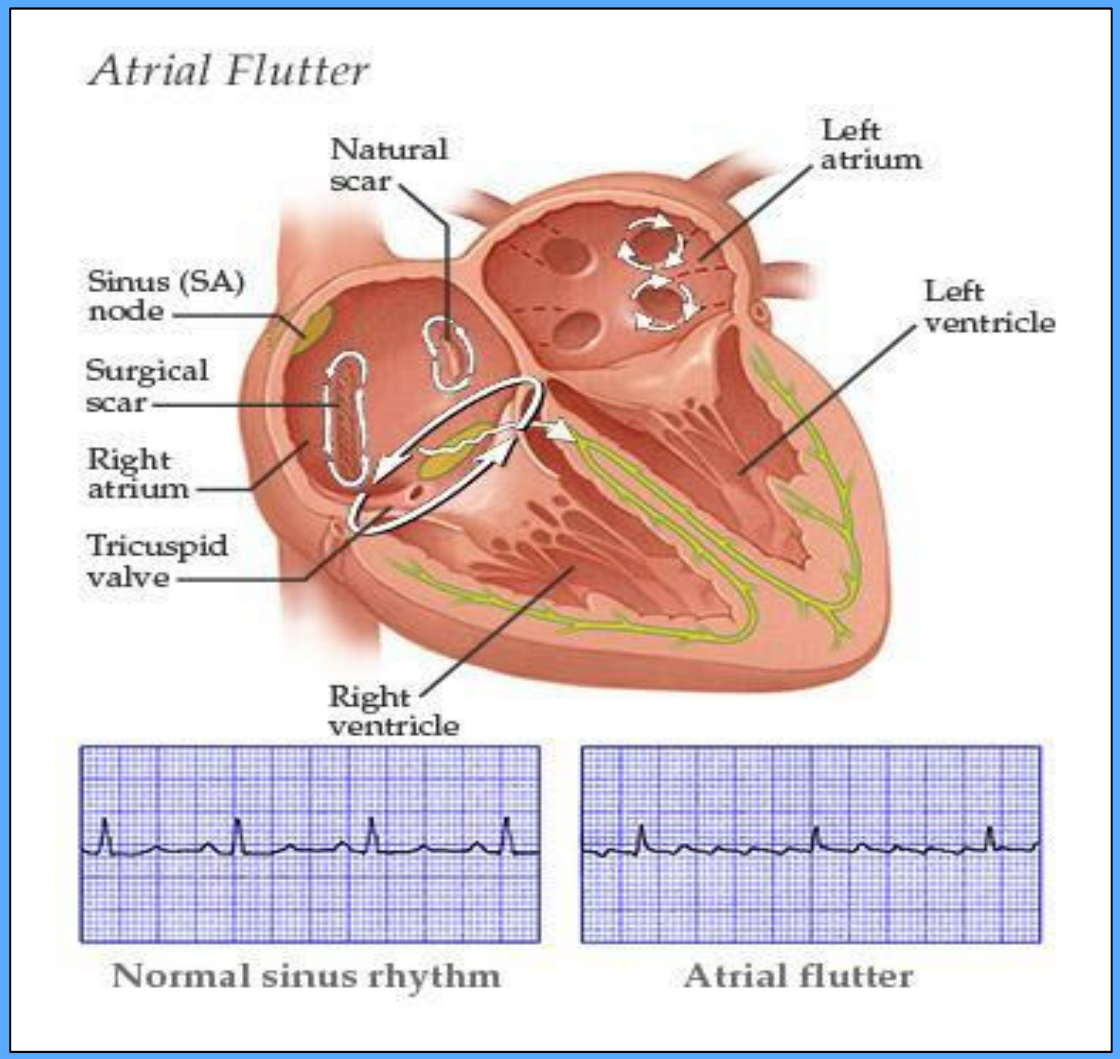
Atrial frequency	**400-600 bpm**
Ventricular frequency	**75-175 bpm**
Regularity	**Irregular**
Origin	**Atria (SVT)**
P-wave	**Absent**
Effect of Adenosine	**Reduces heart rate**



Atrial Fibrillation (AF)

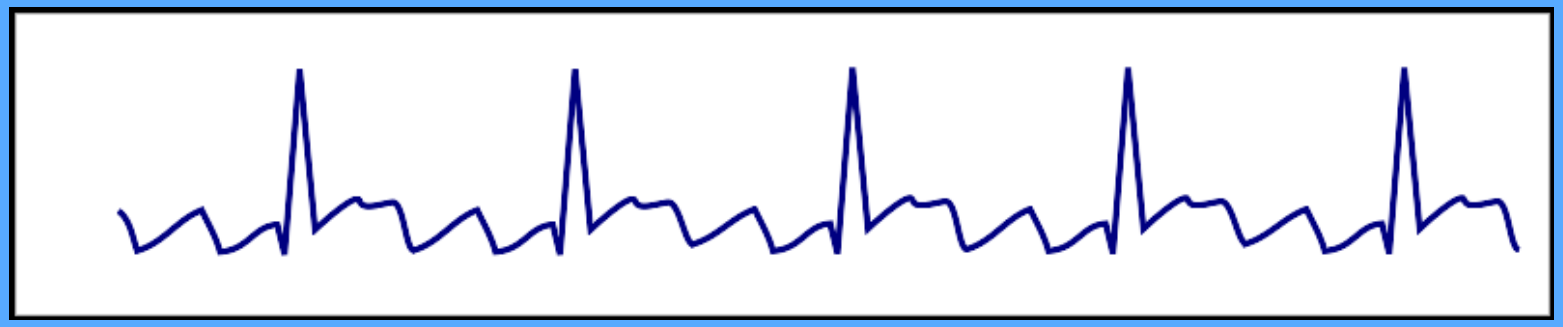


Atrial flutter

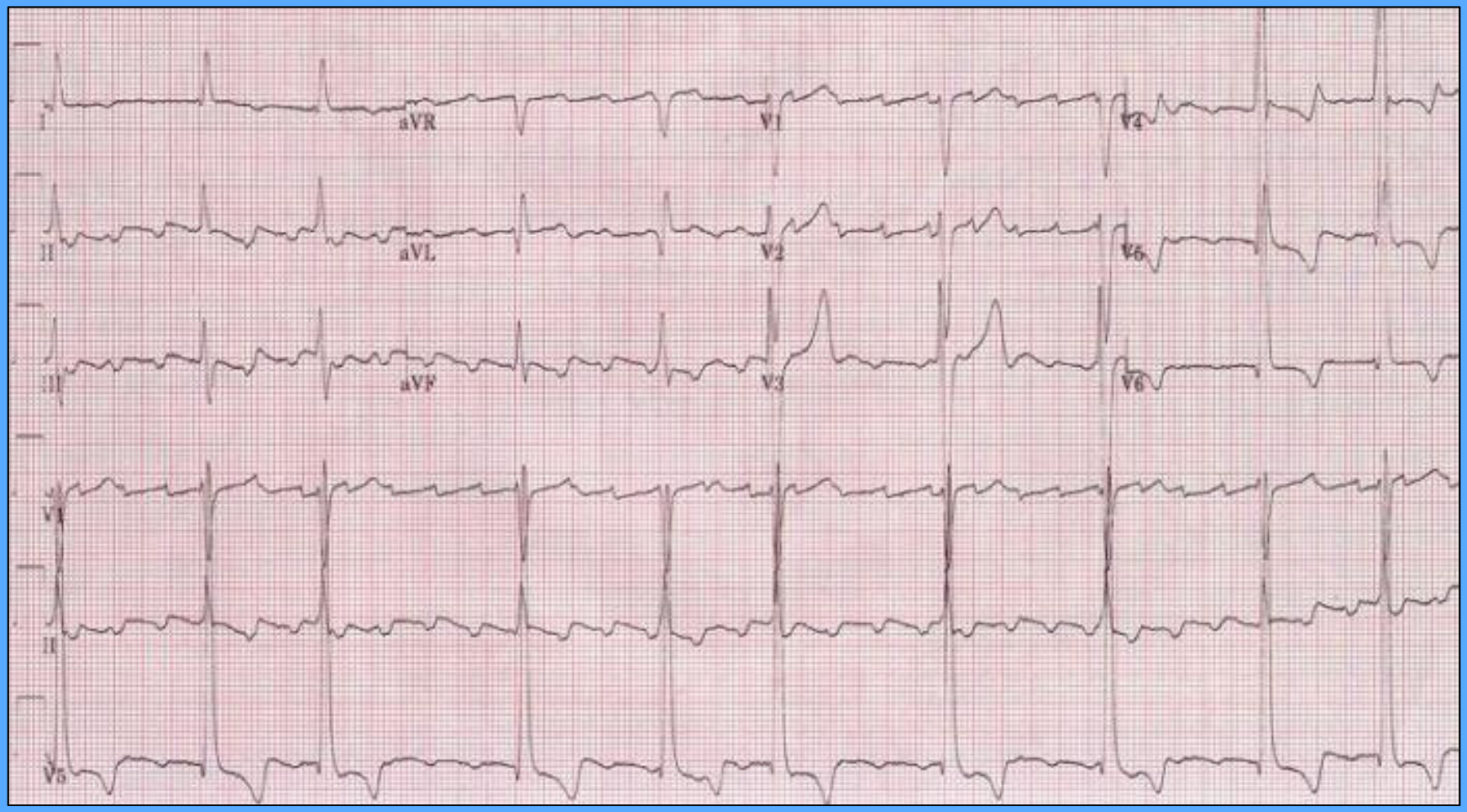


Atrial flutter

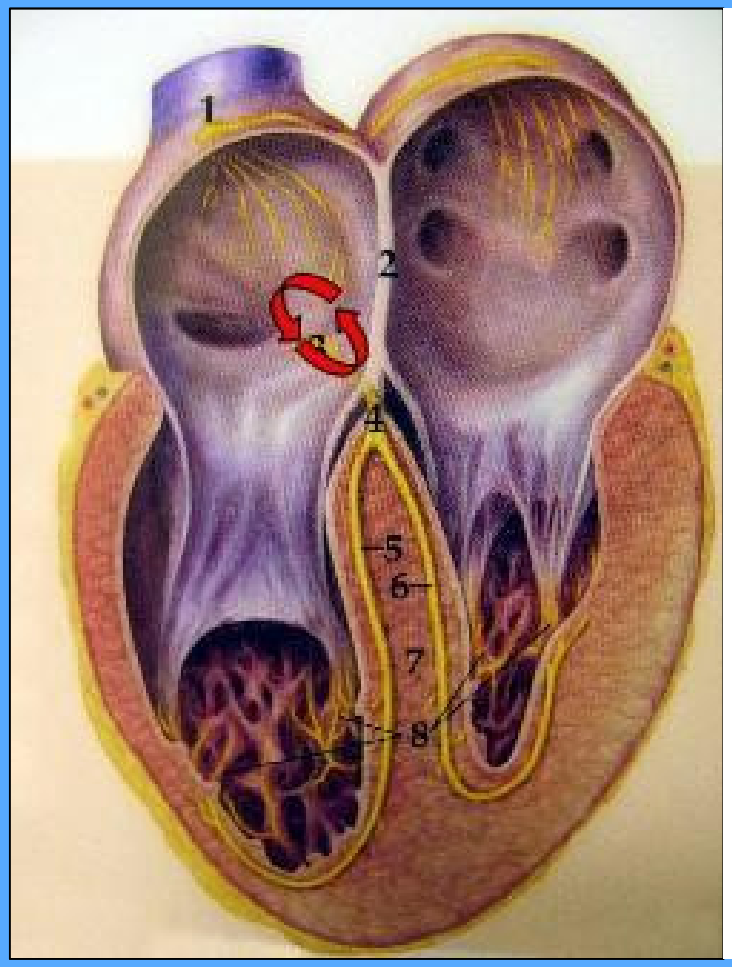
Atrial frequency	250-350 bpm
Ventricular frequency	75-150 bpm (3:1 or 2:1 block)
Regularity	Regular
Origin	Atria (SVT)
P-wave	Negative sawtooth in lead II
Effect of Adenosine	Temporary reduced AV conduction

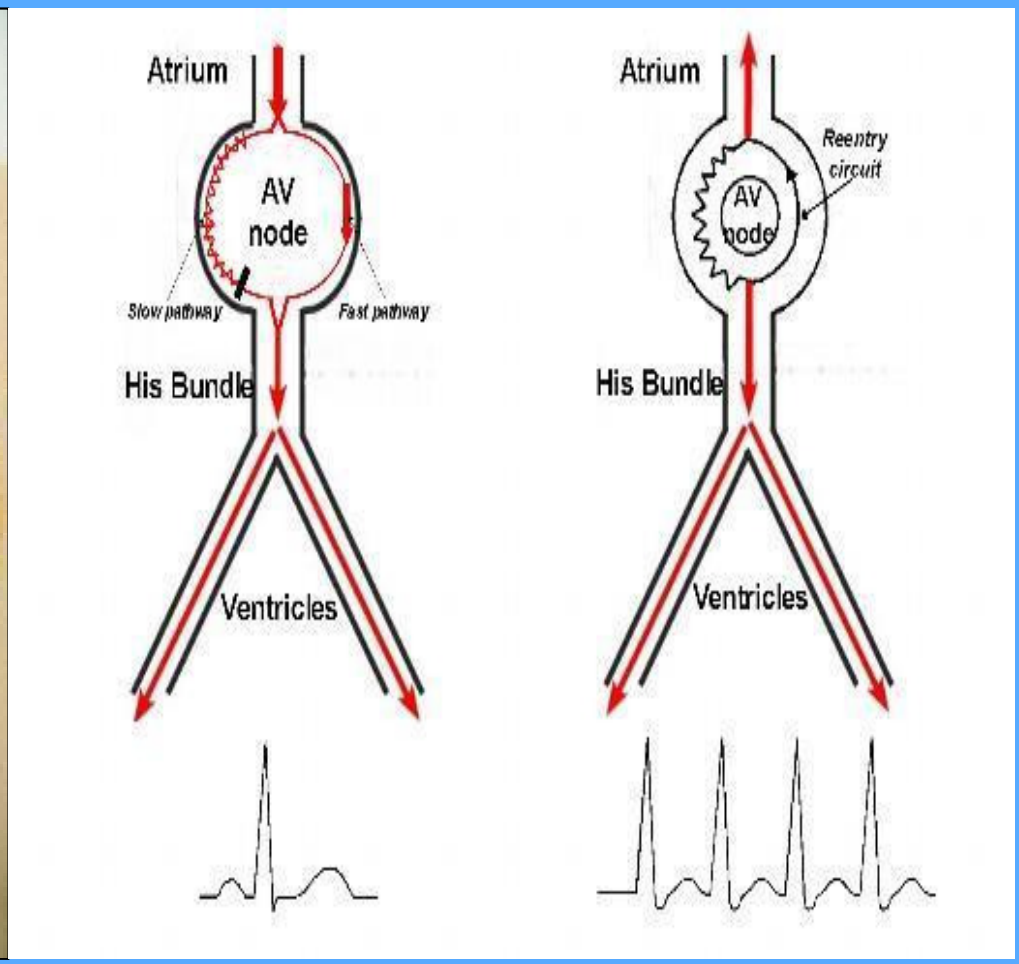


Atrial flutter



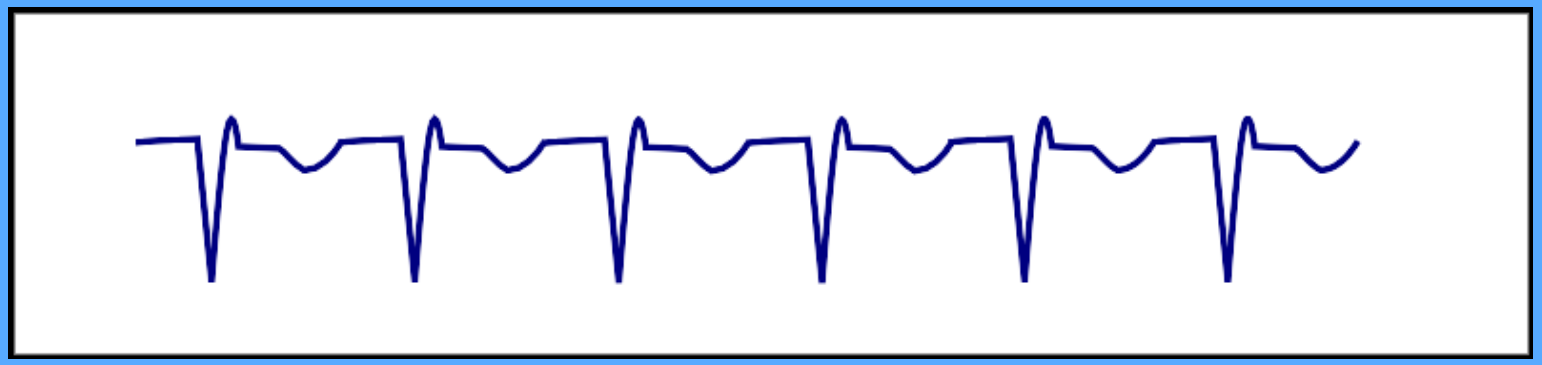
AV-nodal re-entry tachycardia (AVNRT)



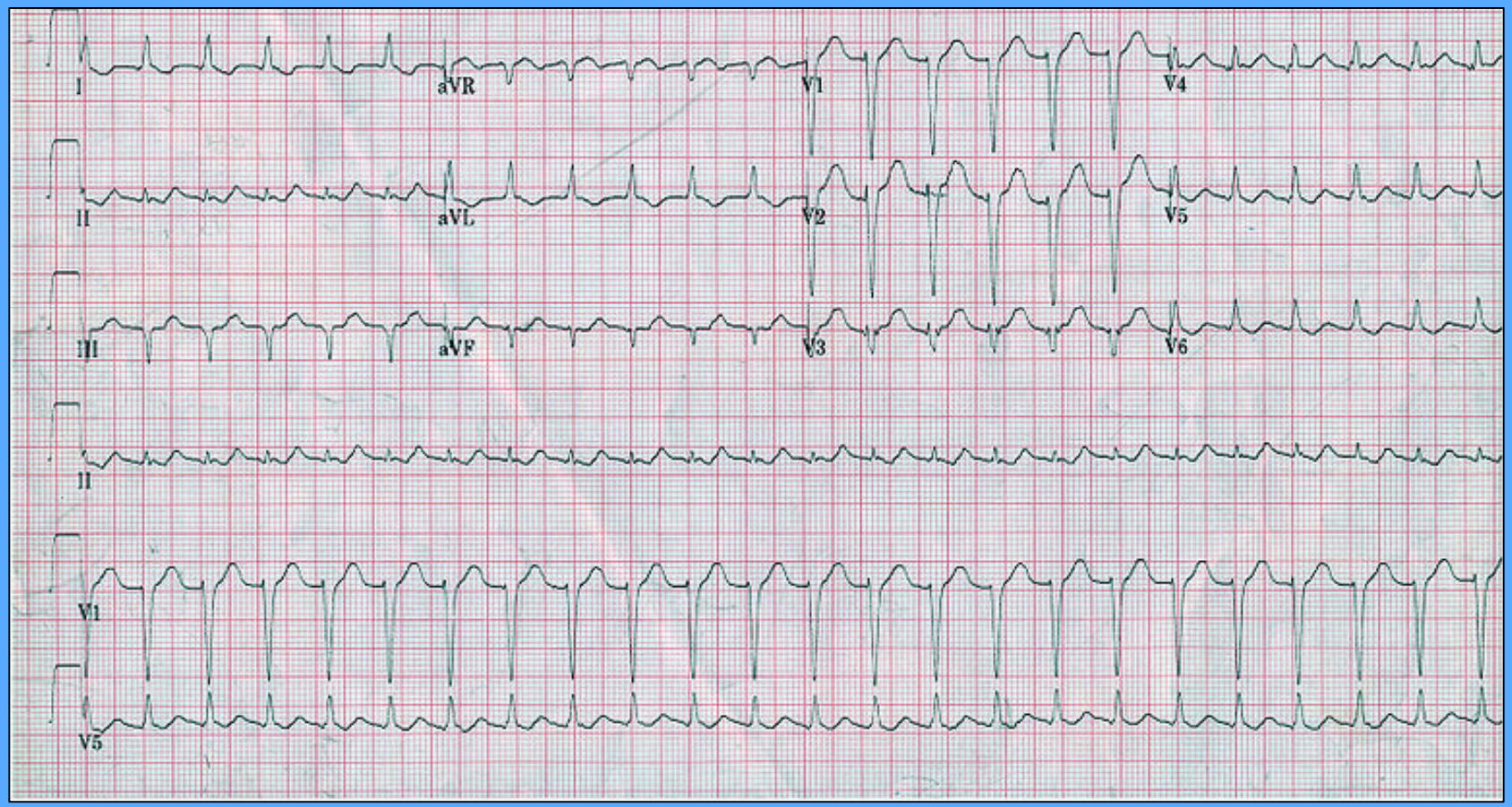


AVNRT

Atrial frequency	180-250 bpm
Ventricular frequency	180-250 bpm
Regularity	Regular
Origin	AV-node
P-wave	Inside or right after QRS-complex
Effect of Adenosine	Terminates arrhytmia



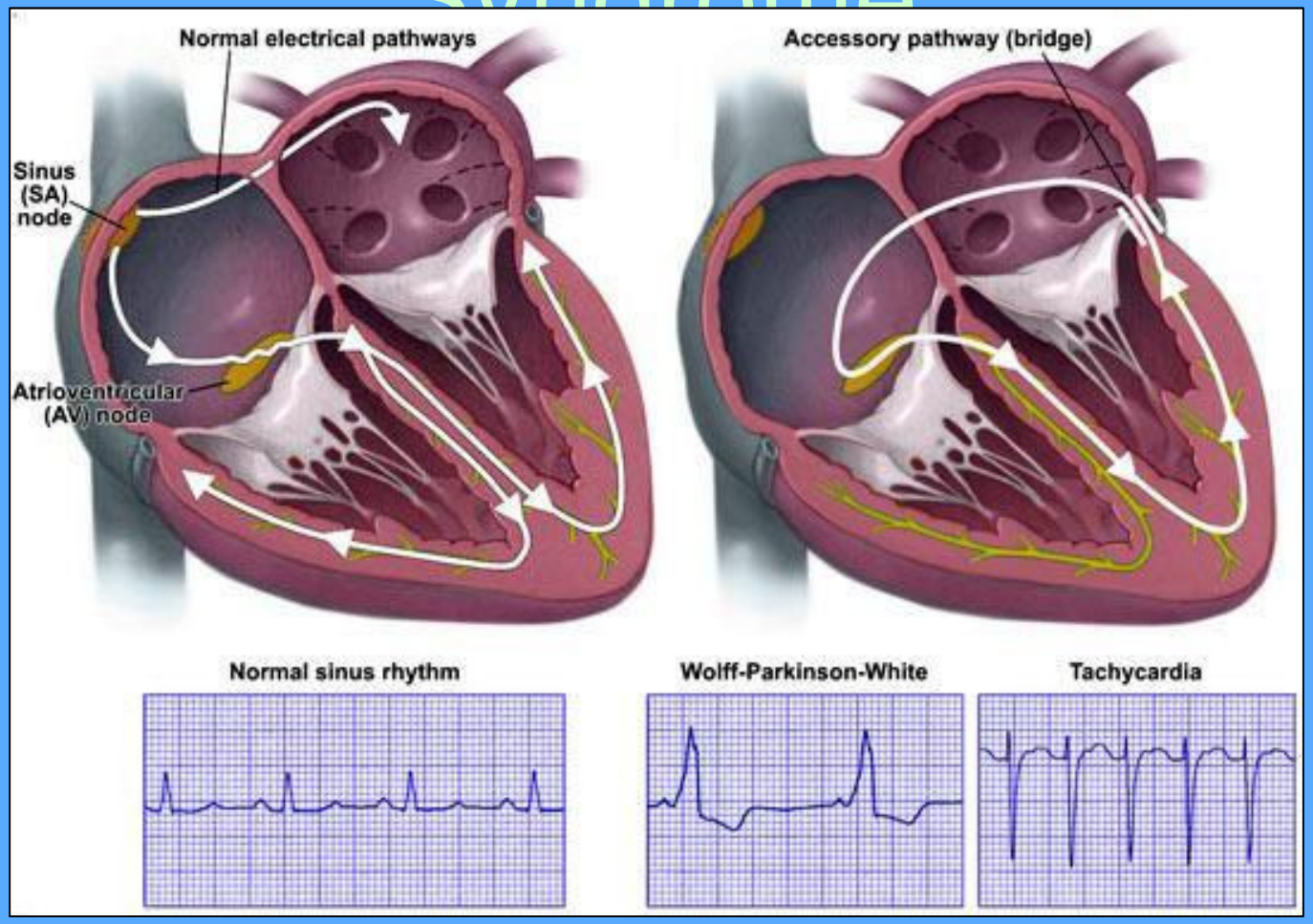
AVNRT



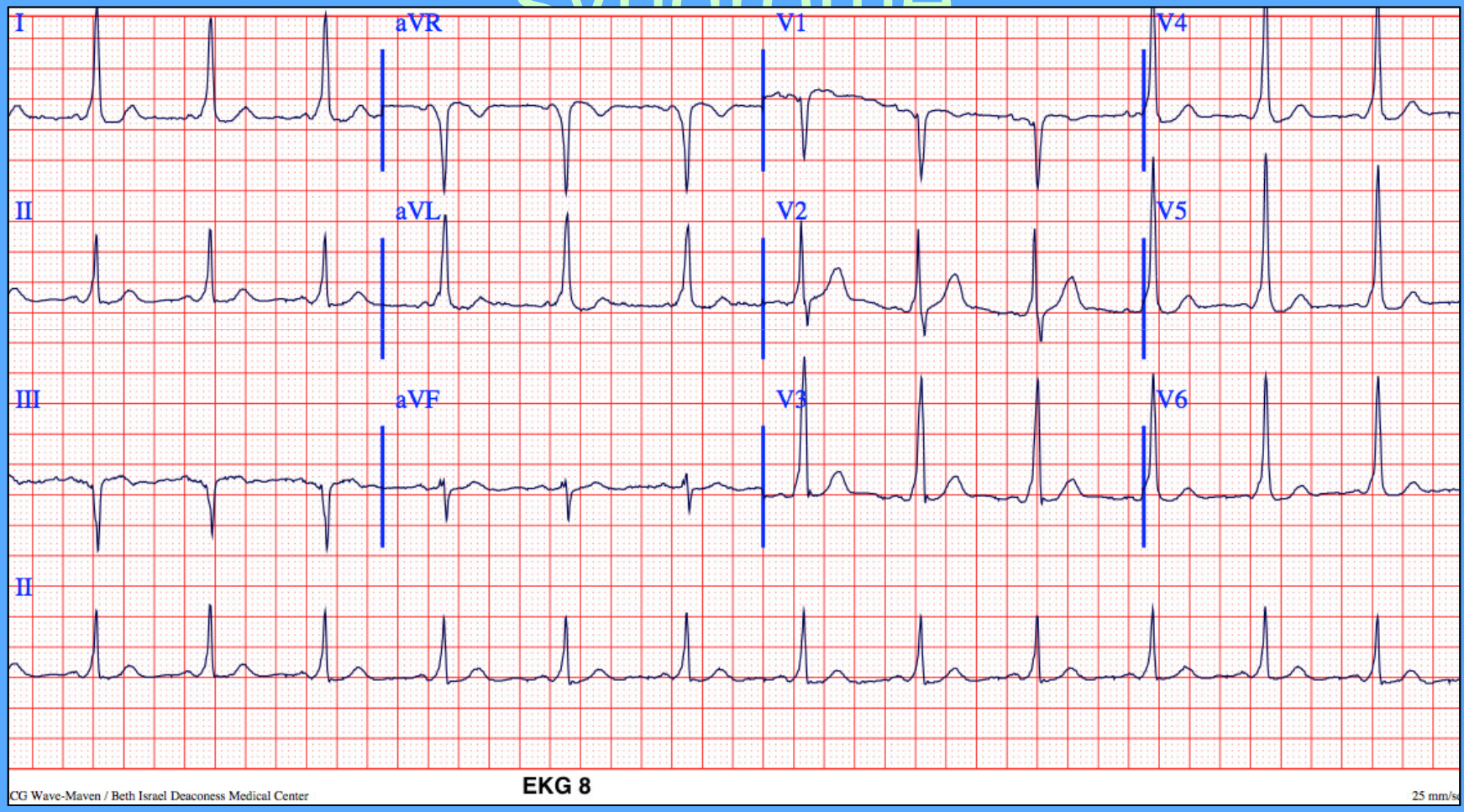
Atrioventricular Re-entrant Tachycardia (AVRT)

- Also called ”Wolf-Parkinson-White-syndrome” (WPS) and is a part of ”Preexitation syndromes”
- Caused by an abnormal accessory conduction pathway between atria and ventricles
- Ventricles might be stimulated prematurely, resulting in an atriventricular re-entry tachycardia
- Can also cause diffuse ECG-changes resembling ischaemia

Wolf-Parkinson-White-Syndrome



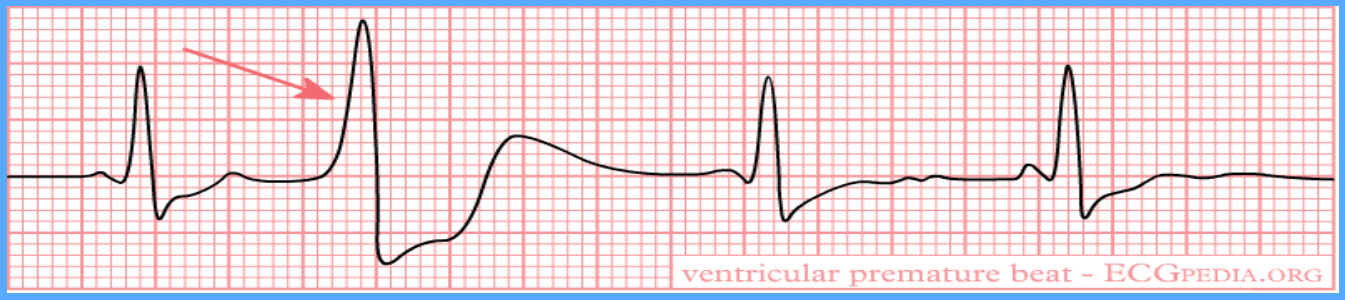
Wolf-Parkinson-White syndrome



Ventricular tachycardias

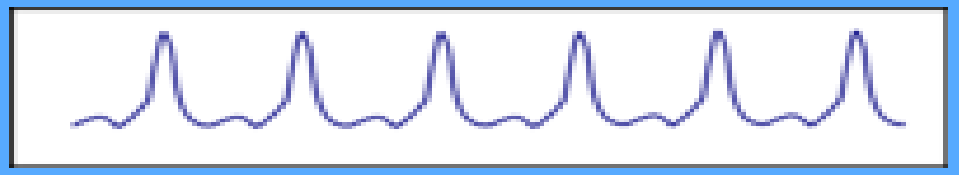
Premature ventricular contractions (PVC)

- Most common of ventriuclar arrhytmias



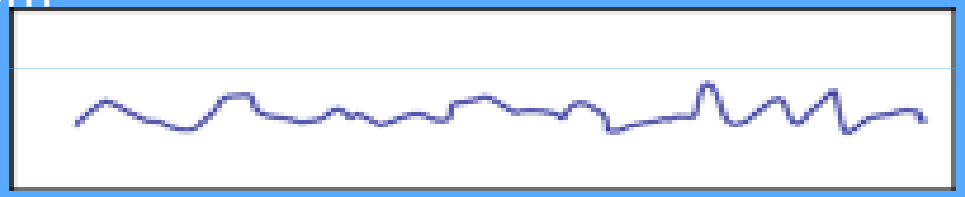
Ventricular tachycardia (VT)

- Regular, HR 110-250



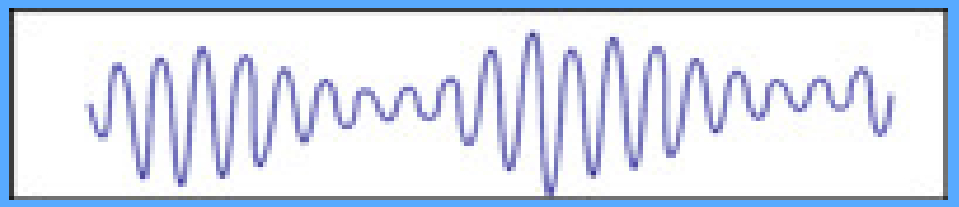
Ventricular fibrillation (VF)

- Irregular, HR 400-600 bpm



Torsade de pointes

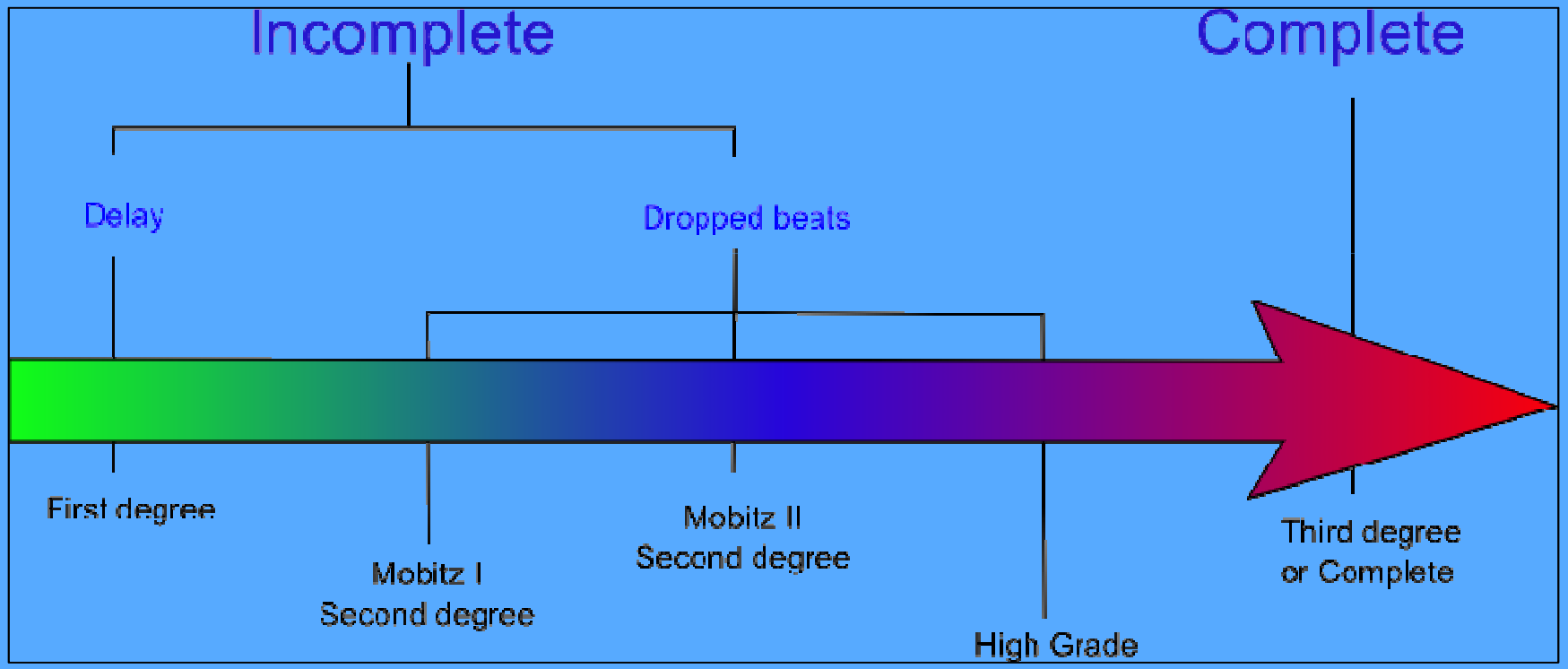
- Regular, HR 150-300



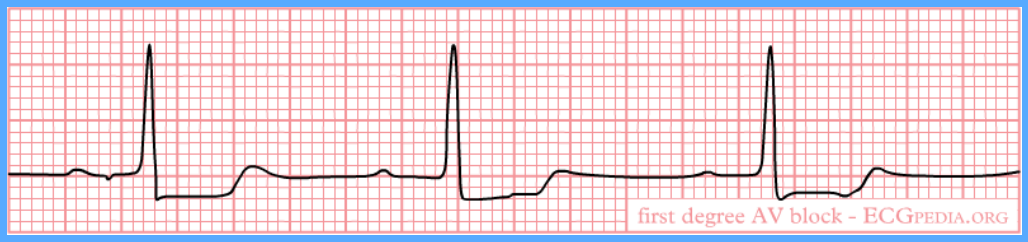
Bradyarrhytmias

- AV-block 1st degree
- AV-block 2nd degree (Wenckebach and Mobitz type II)
- AV-block 3rd degree (complete AV-block)
- AV-blocks
- Sick sinus syndrome

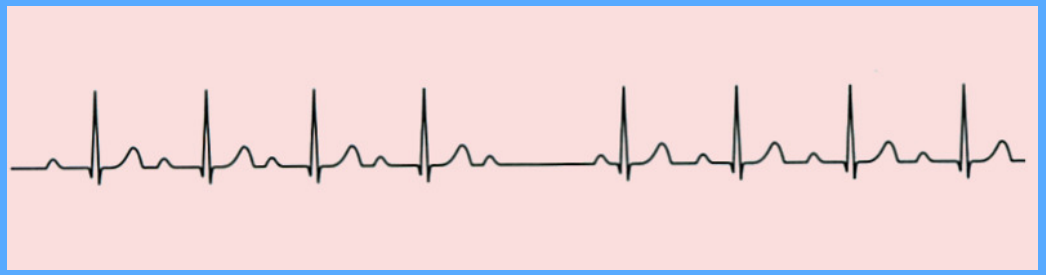
AV-blocks


Incomplete
Complete
Delay
Dropped beats
First degree
Mobitz I
Second degree
Mobitz II
Second degree
High Grade
Third degree
or Complete

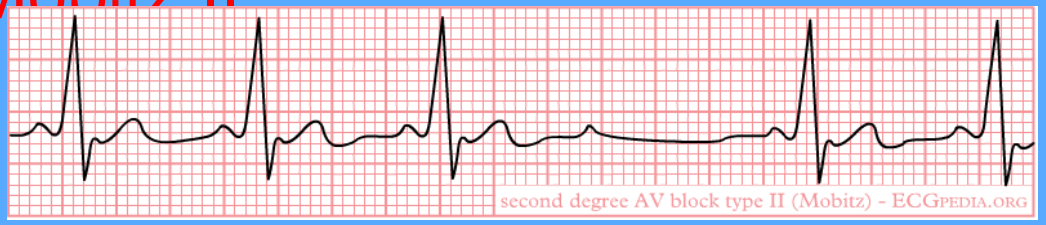
- AV-block type 1



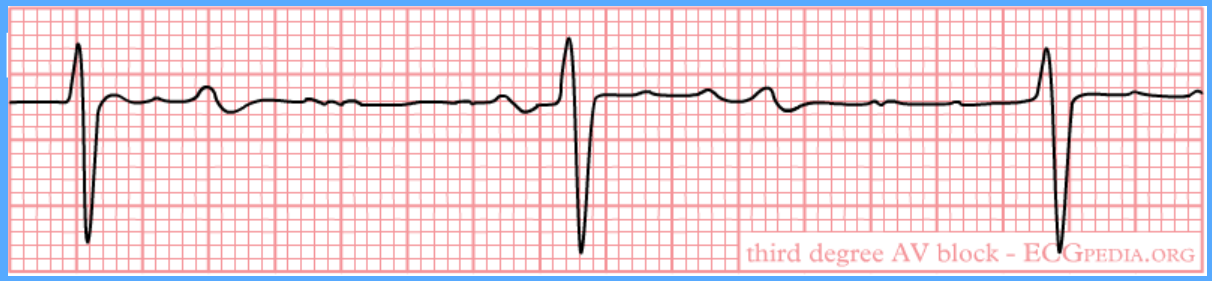
- AV-block type 2 – Mobitz I (Wenchebach)



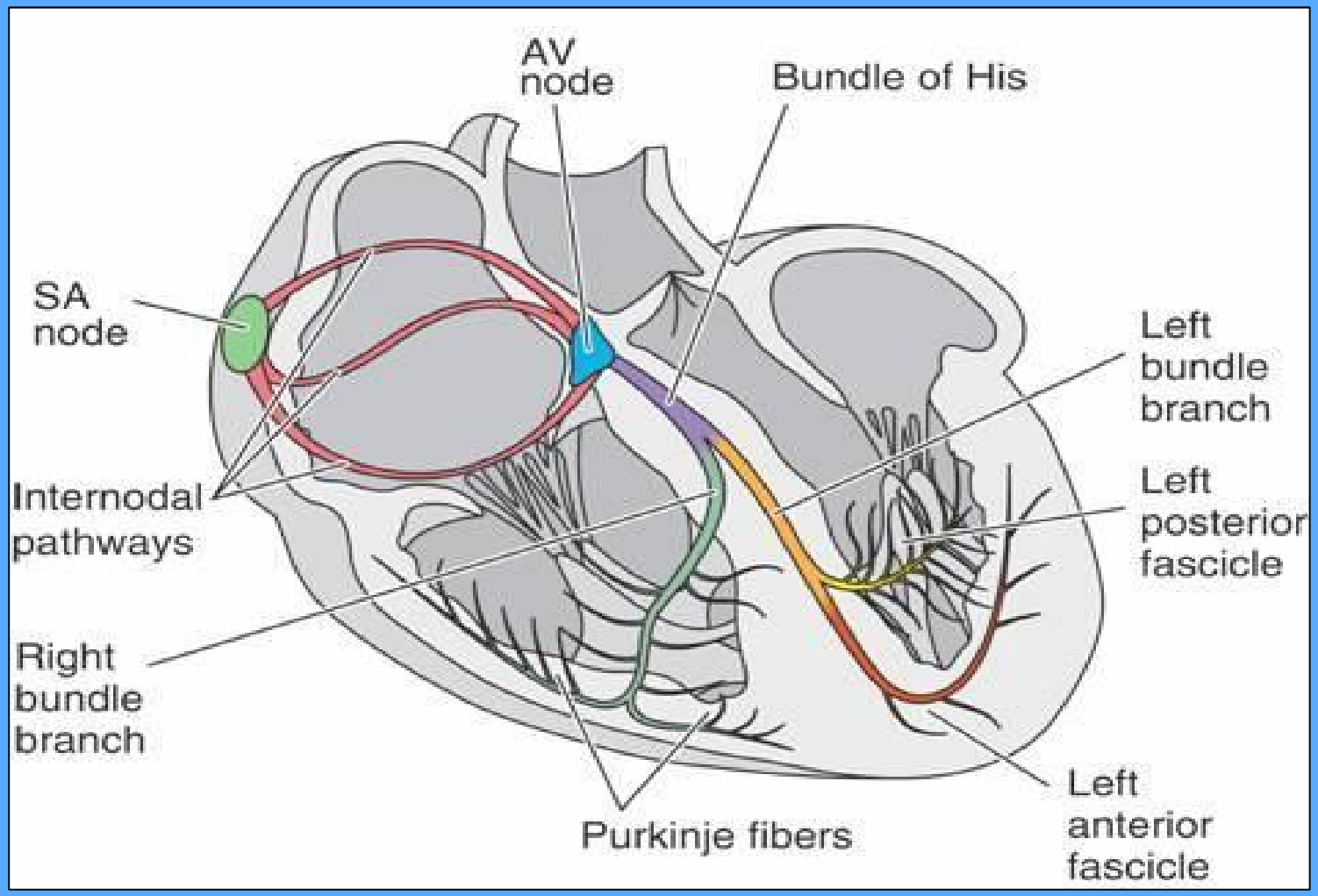
- AV-block type 2 – Mobitz II



- AV-block type III (complete block)
- No relation between P-waves and QRS complexes
- Atrial rythm 60-100 bpm (or AF)
- Ventricular ryhtm might be nodal, ventricular o

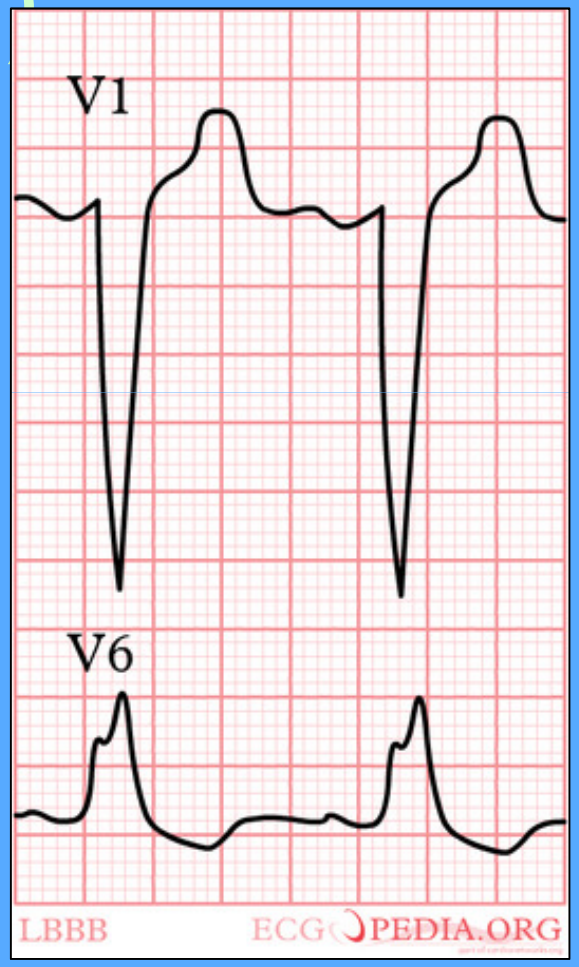


Conduction system



Left bundle branch block (LBBB)

- Slowed conduction in left bundle, causing delayed depolarization of left ventricle
- QRS > 0,12 sec
- Deep S-waves in V1-V3
- Late R-waves in V5-V6
- ST-segment depression in lateral leads (I, aVL, V5-V6)
- Always a pathological finding in patients!



Right bundle branch block (RBBB)

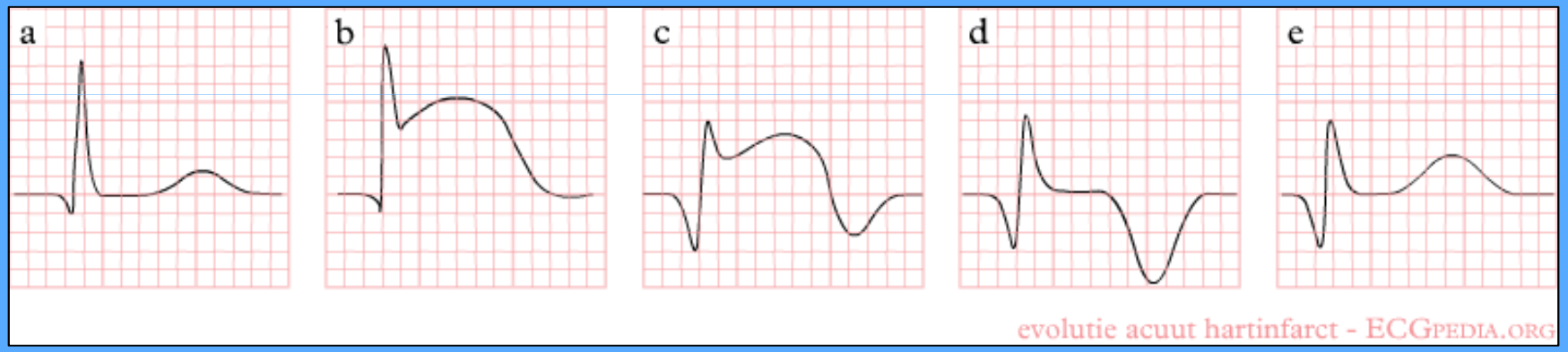
- Conduction in right bundle branch is slow, causing late depolarization of right ventricle
- QRS > 0,12 sec
- rSR (”rabbit ears”) in V1-V2
- Late deep S-waves in lateral leads (I, aVL, V5-V6)
- Commonly a physiological finding in patients!

Myocardial ischemia and infarction

Evolution of the ECG during a myocardial infarct

Time from onset of symptoms	ECG	Changes in the heart
minutes	hyperacute T waves (tall T waves), ST-elevation	reversible ischemic damage
hours	ST-elevation, with terminal negative T waves, negative T waves (these can last for days to months)	onset of myocardial necrosis 
days	Pathologic Q Waves	 scar formation

Evolution of ECG changes in Myocardial infarction

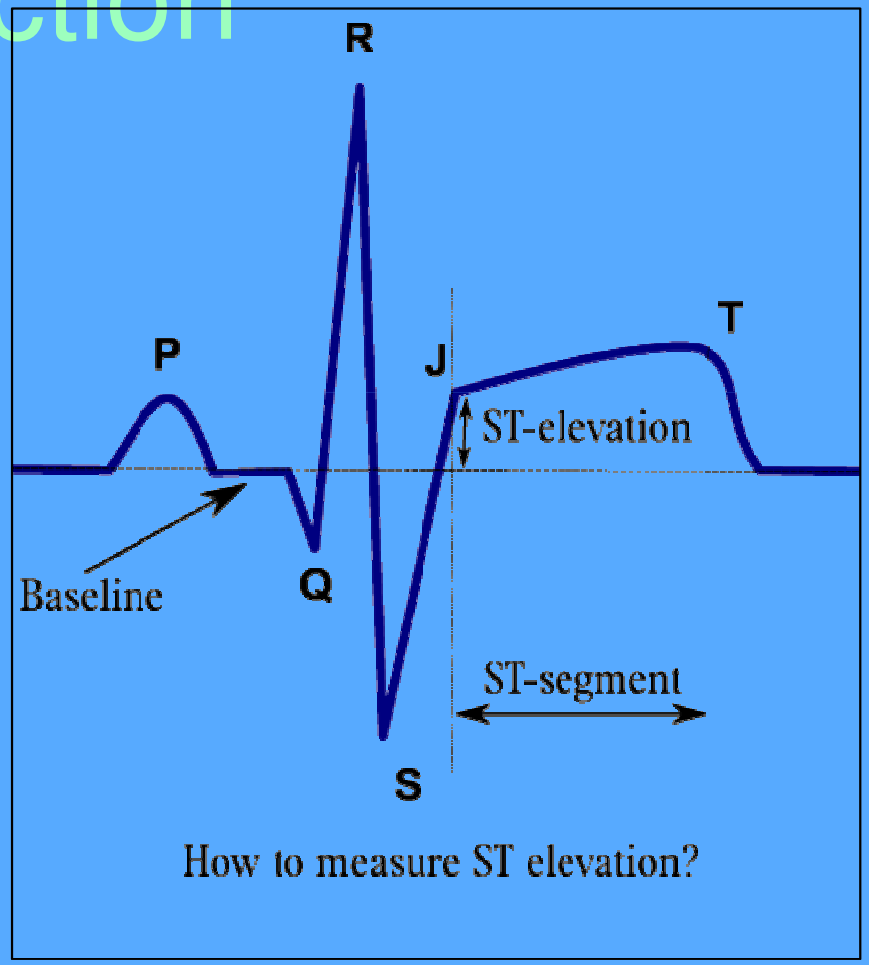


Diagnosis of myocardial infarction

- Elevated cardiac enzymes in blood (Troponin T, C and CKMB)

AND on of the following:

- Typical symptoms (chest pain > 20 min)
- ECG changes (ST elevation, ST depression or pathological Q-waves)



ECG-changes

ST-elevation (STEMI)

- Men > 0.2 mV in V2-V3, and/or > 0.1 mV in other leads
- Women > 0.1 mV in two or more leads

ST-depression

- New horizontal or downsloping ST-depression > 0.05 mV in two contiguous leads

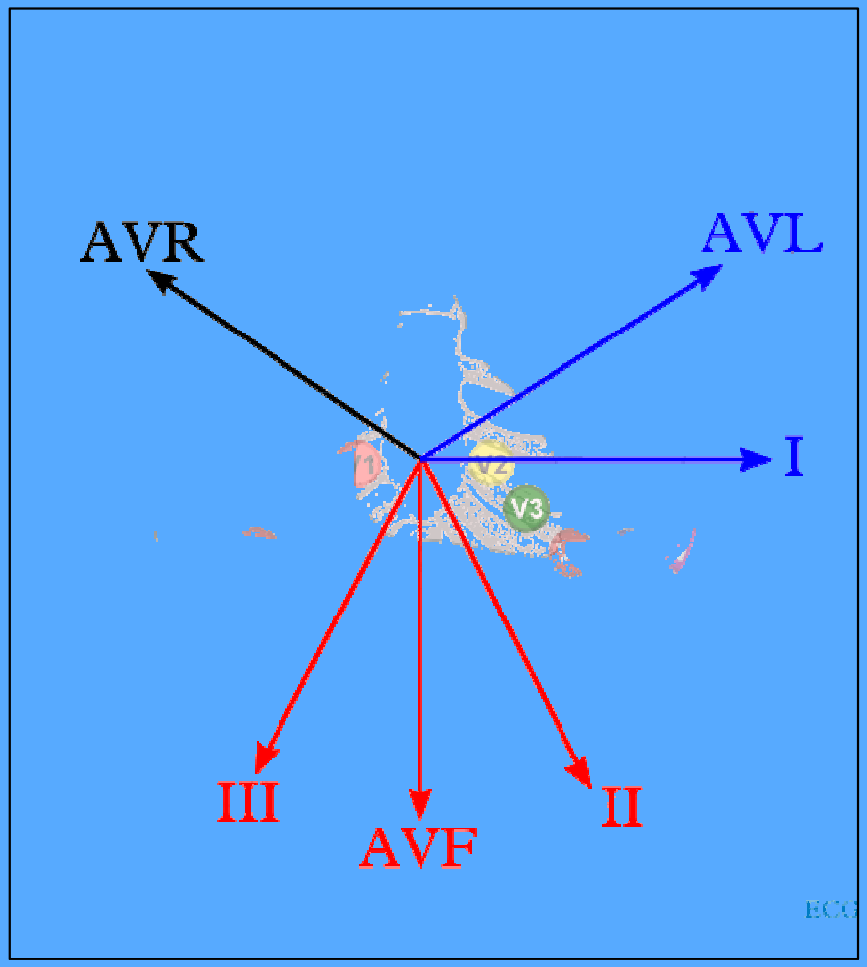
T-wave inversion

- > 0.1 mV in two contiguous leads

Q-wave (old infarction, develops after hours/days)

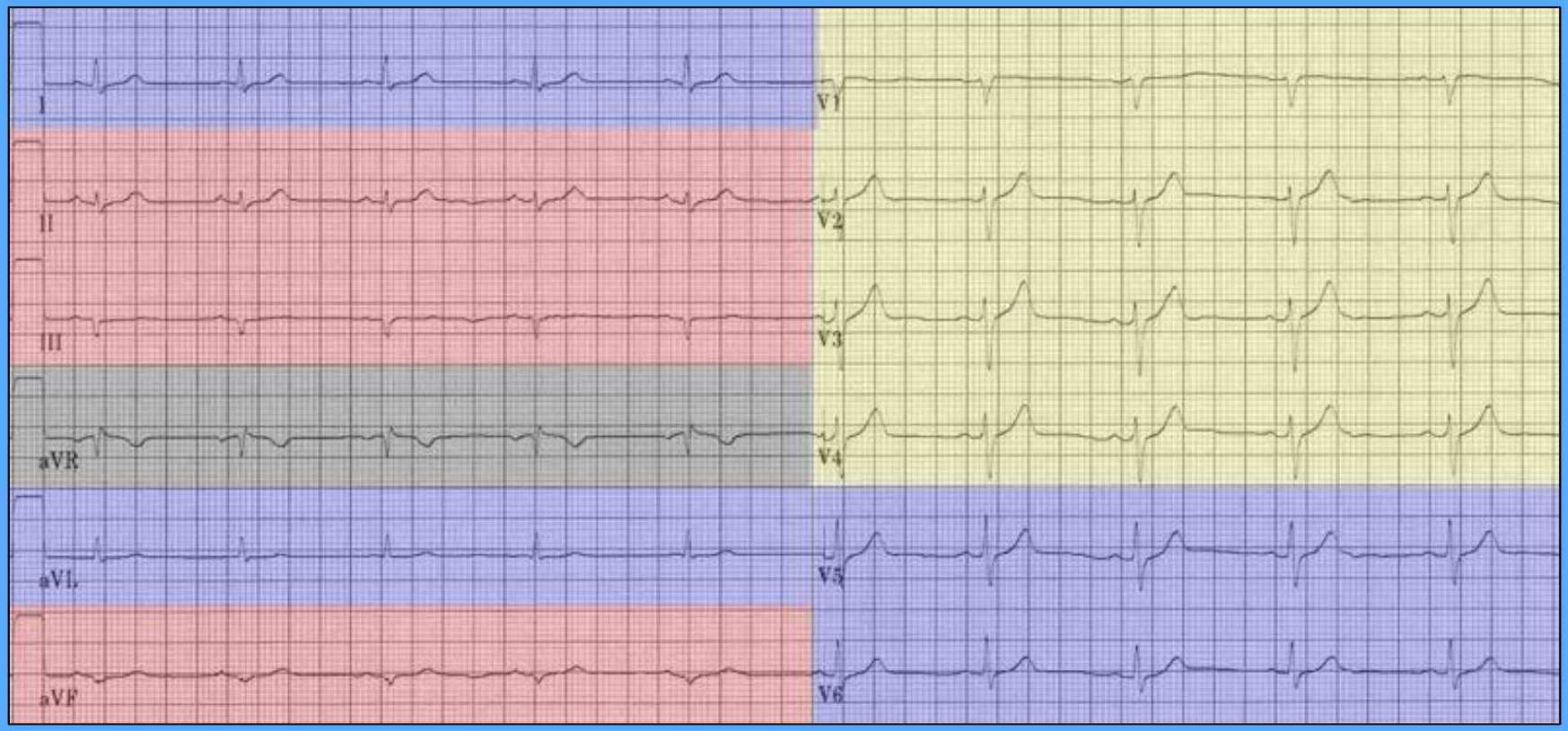
New LBBB!!

Location of ECG-changes

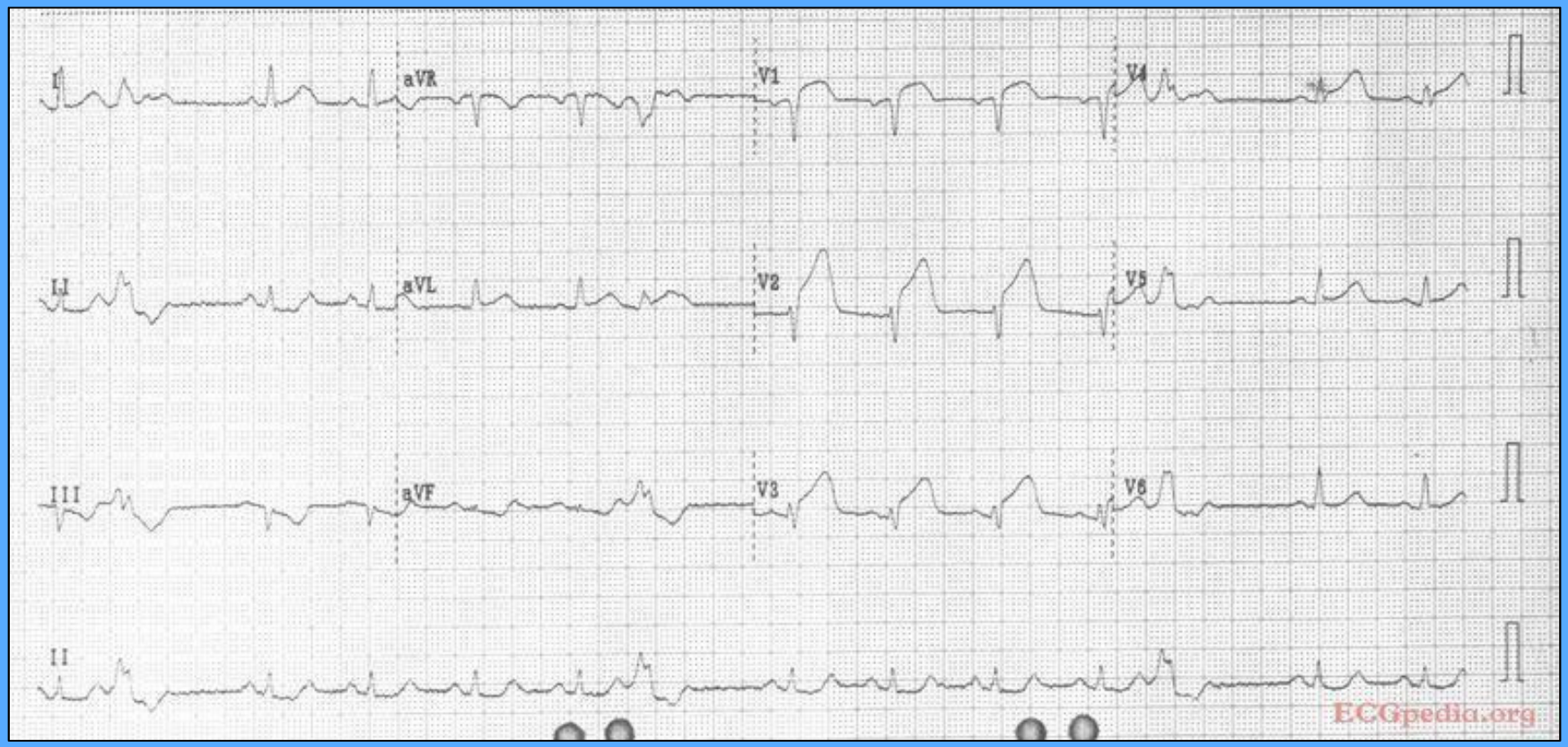


I Lateral	V1 Septal
II Inferior	V2 Septal
III Inferior	V3 Anterior
aVR Left Main	V4 Anterior
aVL Lateral	V5 Lateral
aVF Inferior	V6 Lateral

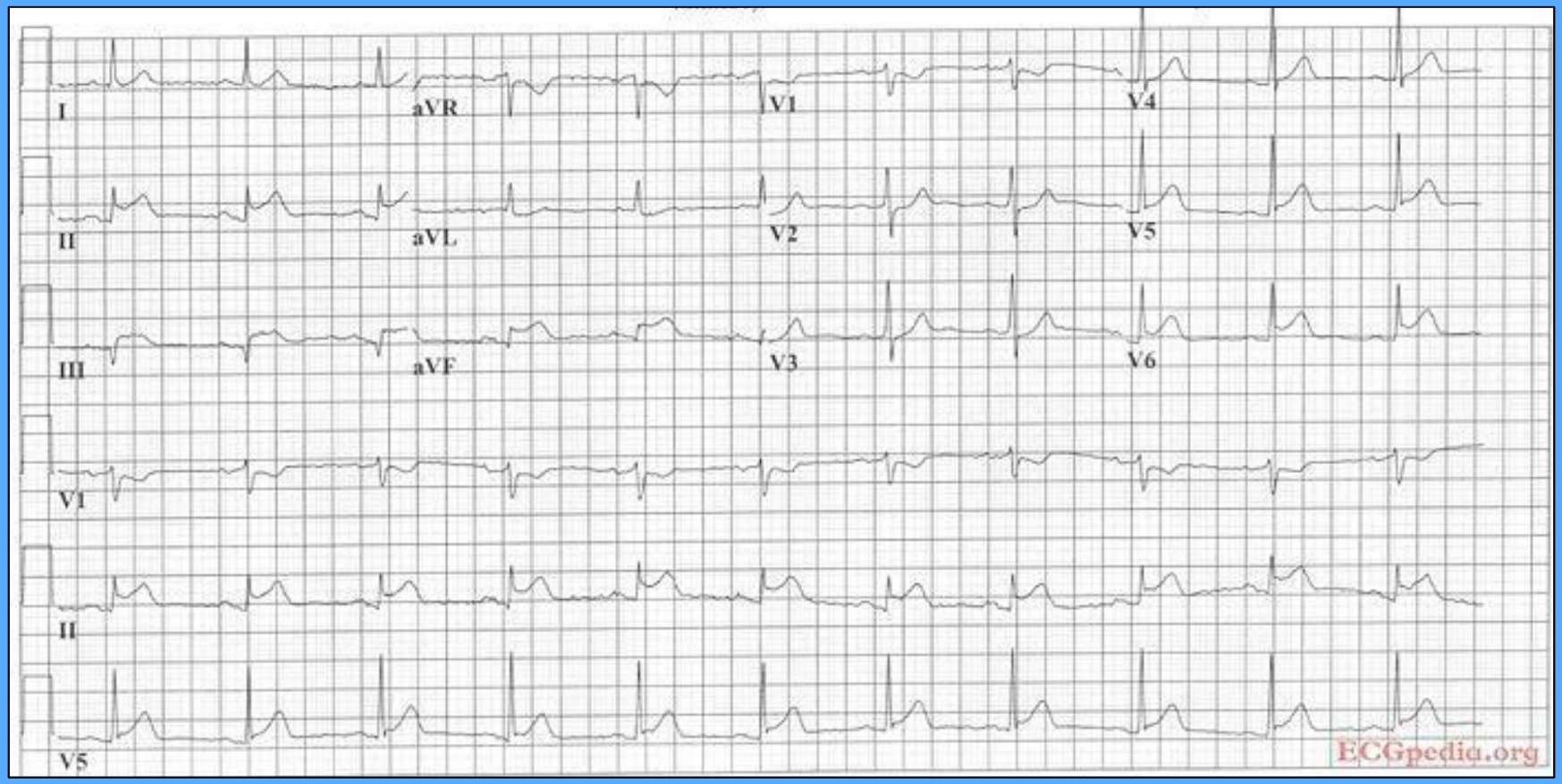
Location of ECG-changes



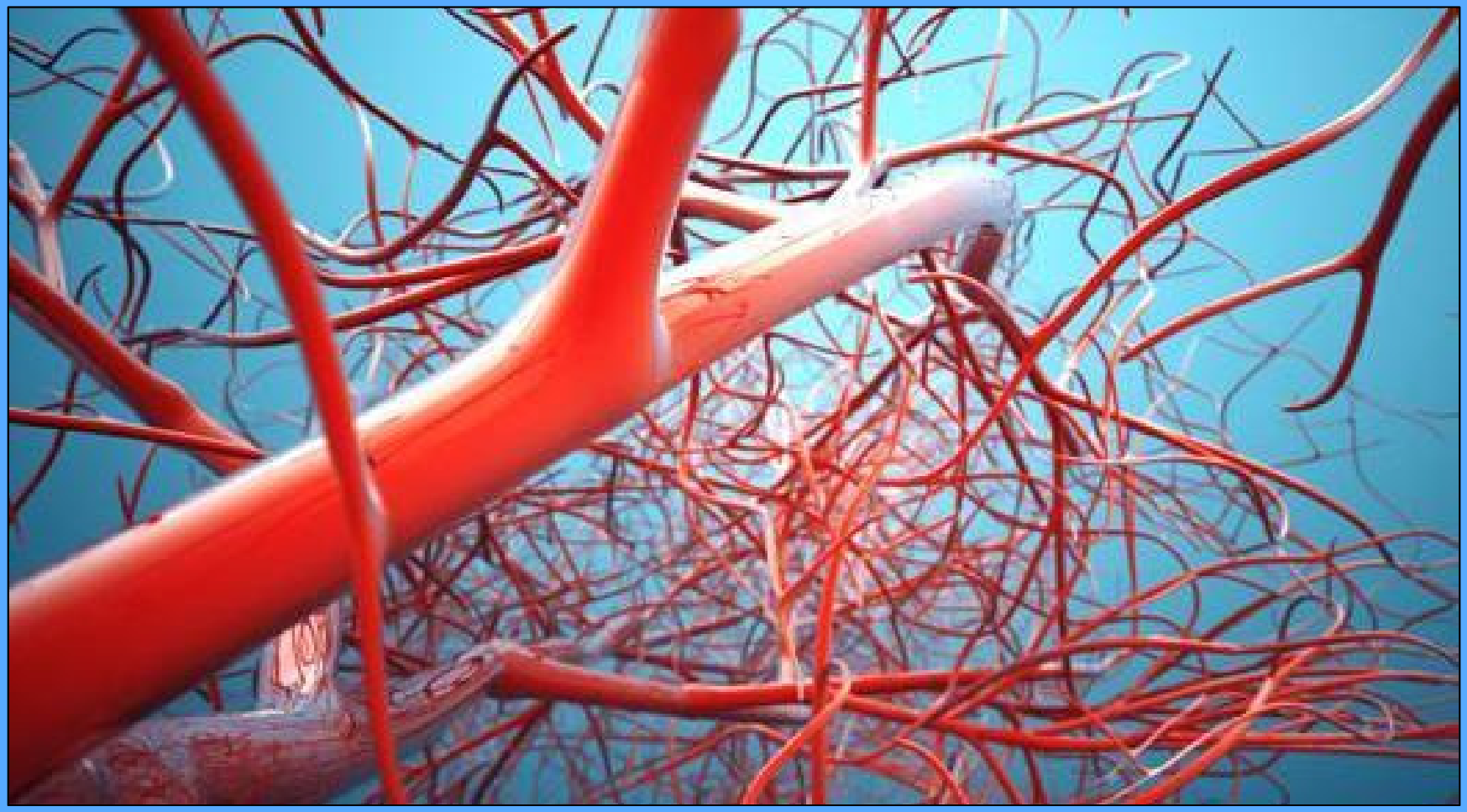
Where is MI located?



Where is MI located?



PAUSE!



How to read ECG

”5+1”:

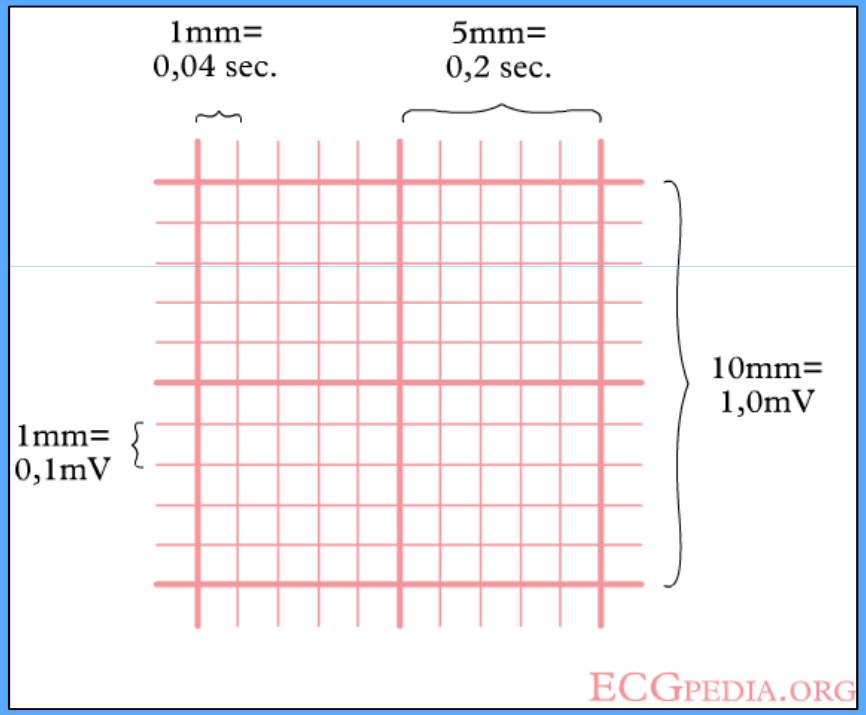
1. Rhytm
2. Rate
3. Conduction (PQ, QRS, QT)
4. Heart axis
5. Morphology (P wave, QRS, ST-segment)
6. Compare current ECG with a previous one

1. Ryhtm

- Is it sinus ryhtm?
- Regular or irregular?
- Prolonged recording from one lead is used to provide a rhytm strip (usually lead II)

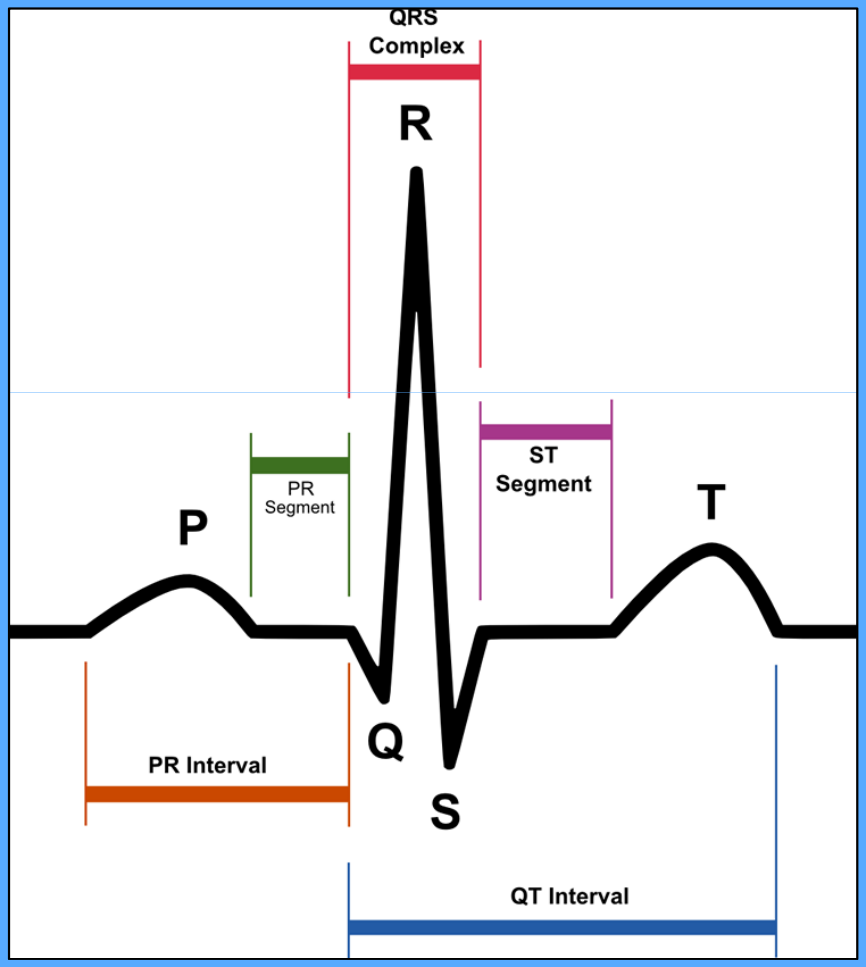
2. Heart rate

- Determine the time between two QRS complexes (RR)
- IF paper speed is 25 mm/second, count number of big squares and divide with 300 (only in regular ryhtm)
- If paperspeed is 50 mm/second, divide with 600 (only in regular ryhtm)
- ”ECG-rulers”



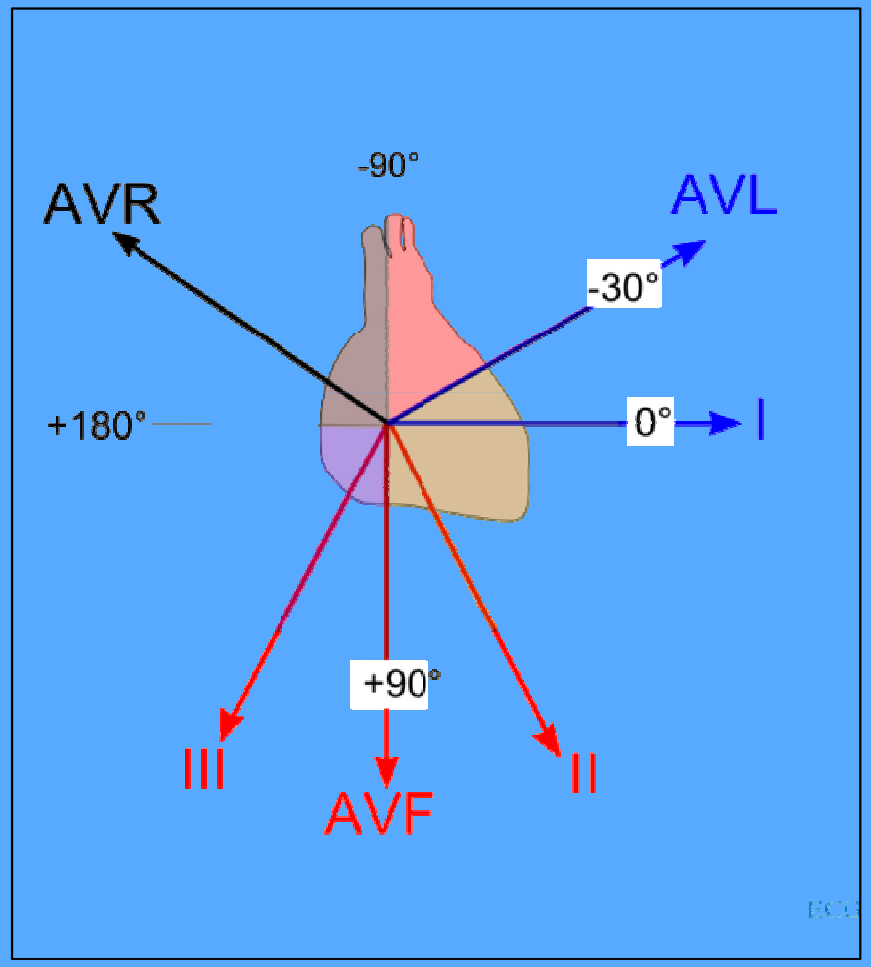
3. Conduction

- The PQ interval starts at the beginning of the atrial contraction and ends at the beginning of the ventricular contraction (0,12 – 0,20 seconds)
- The QRS duration indicates how fast the ventricles depolarize (normal < 0,10 seconds)
- The normal QTc (corrected) interval indicates how fast the ventricles are repolarized, becoming ready for a new cycle. (below 0.45 seconds in men and below 0,46 in women)



4. Heart axis

- Look at lead aVF and I, should normally be positive
- Lead II is also postive, lead III can be pos. or neg.
- In normal axis both has predominant positive deflections

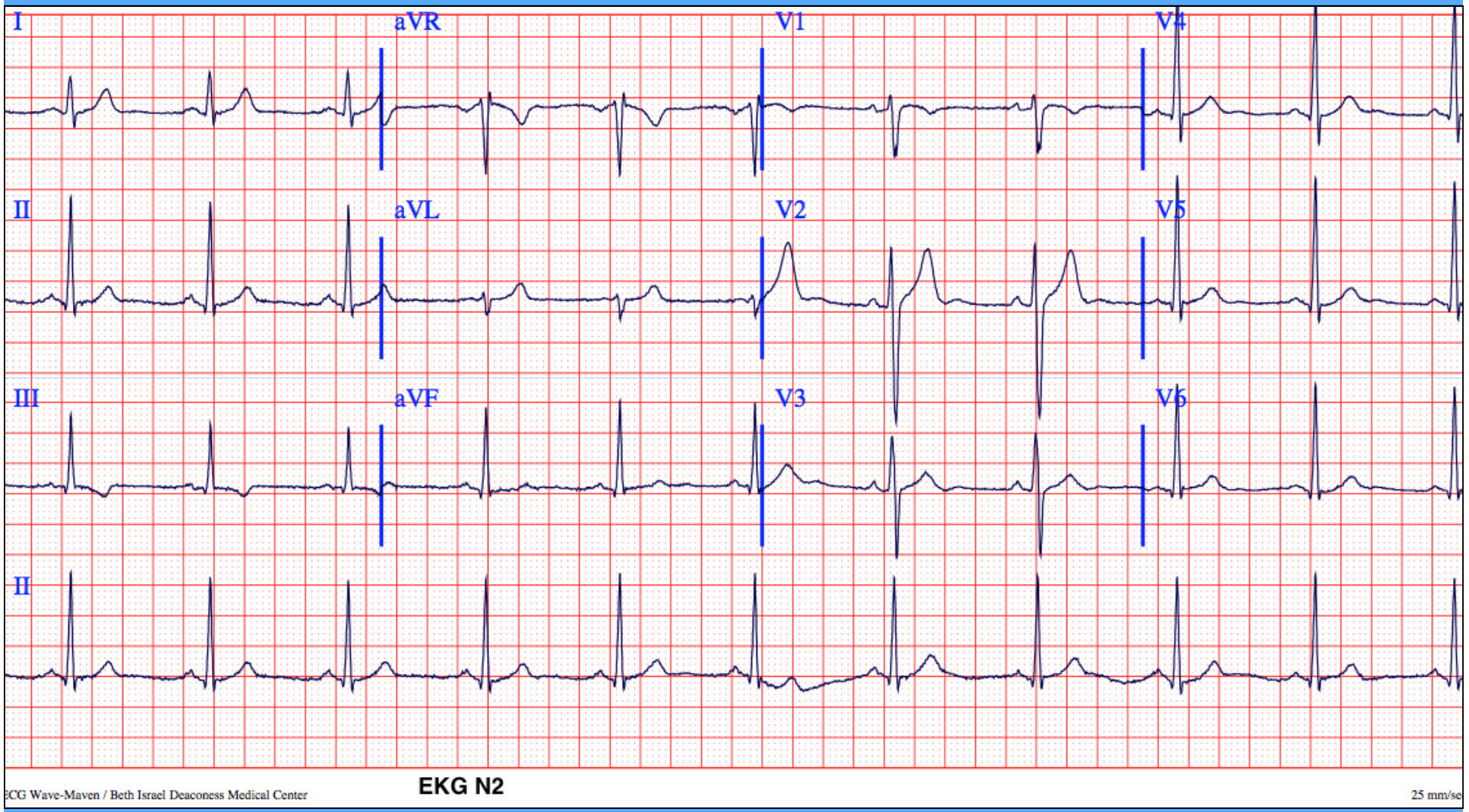


5. Morphology

- Normal p wave?
- No pathological Q-waves?
- Prolonged QRS?
- Normal R-wave progression in V1-V6?
- ST elevation or depression?
- Abnormal T-wave?

6. Compare to old ECG

- Are the presenting ECG-changes new?
- Remember that new LBBB is treatet as STEMI..


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ECG Wave-Maven / Beth Israel Deaconess Medical Center
EKG N2
25 mm/se

For more ECG traces click:

http://ecg.bidmc.harvard.edu/maven/mavenmain.asp