



# Cardiovascular Pathophysiology 3

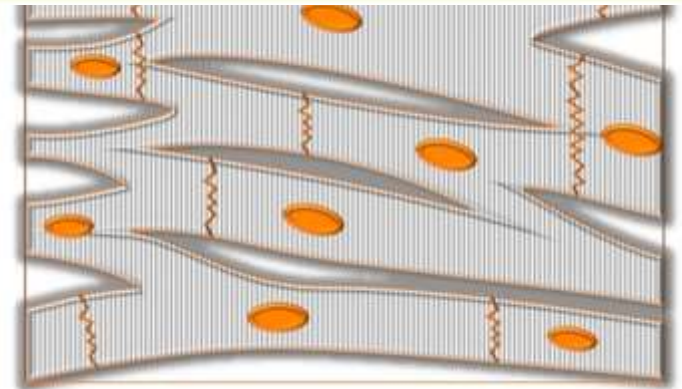
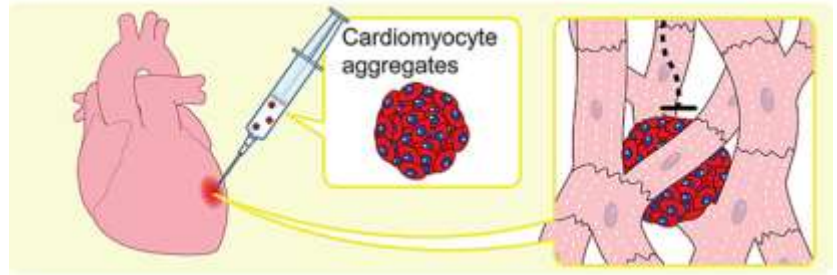
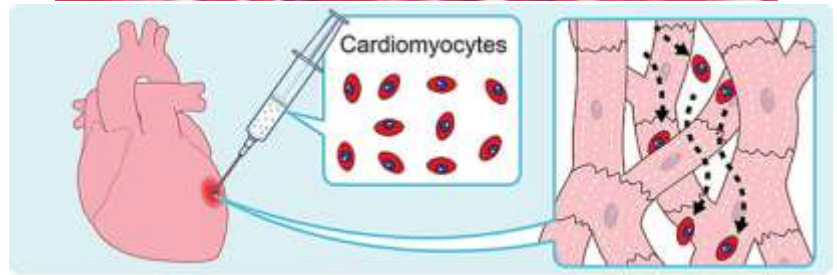
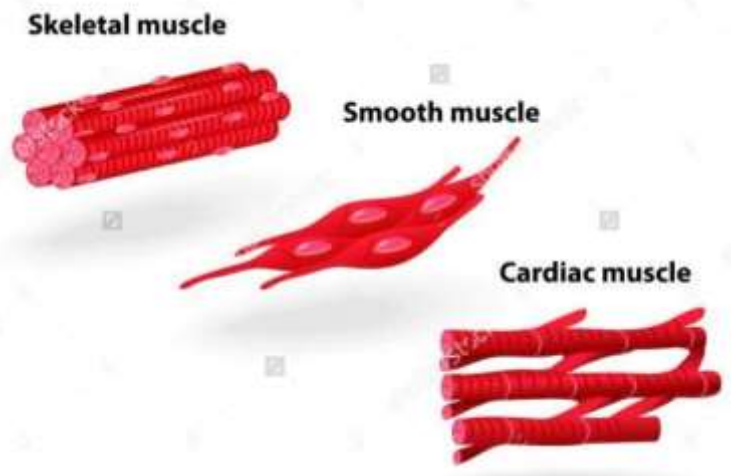
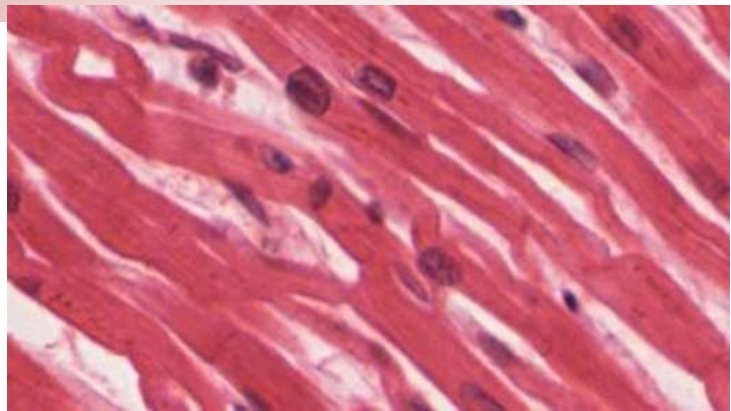
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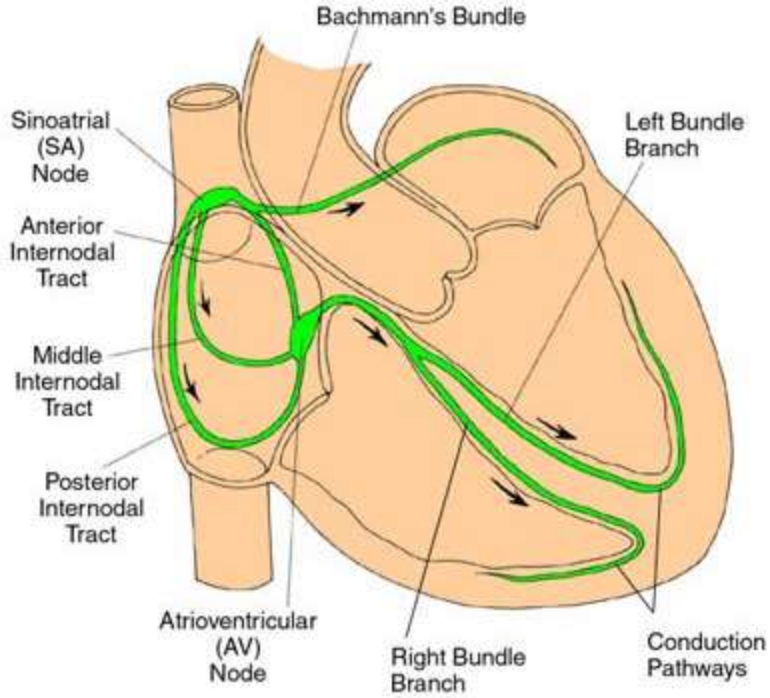
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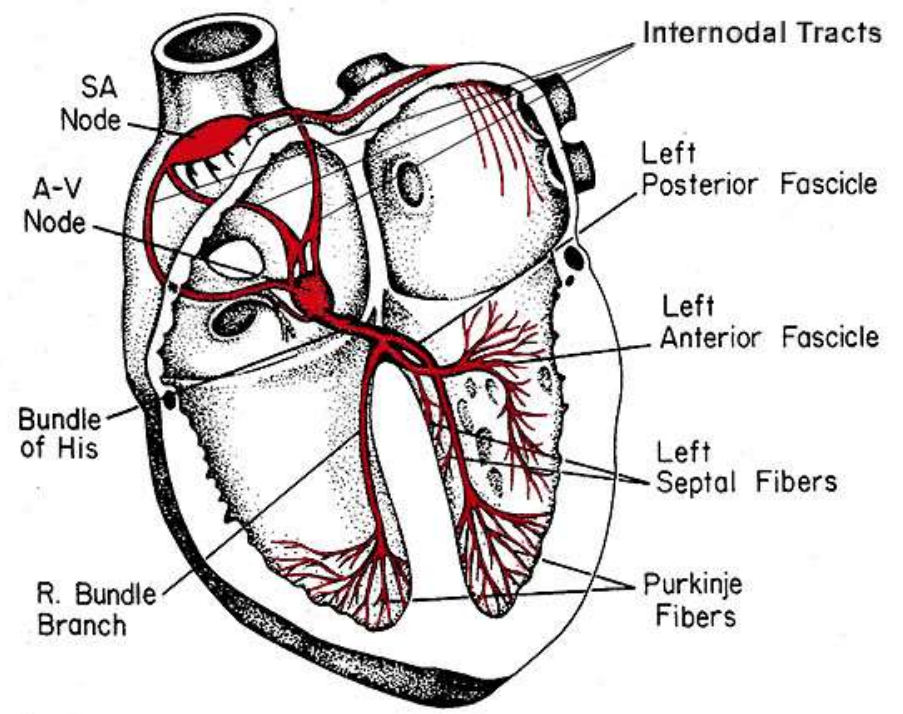
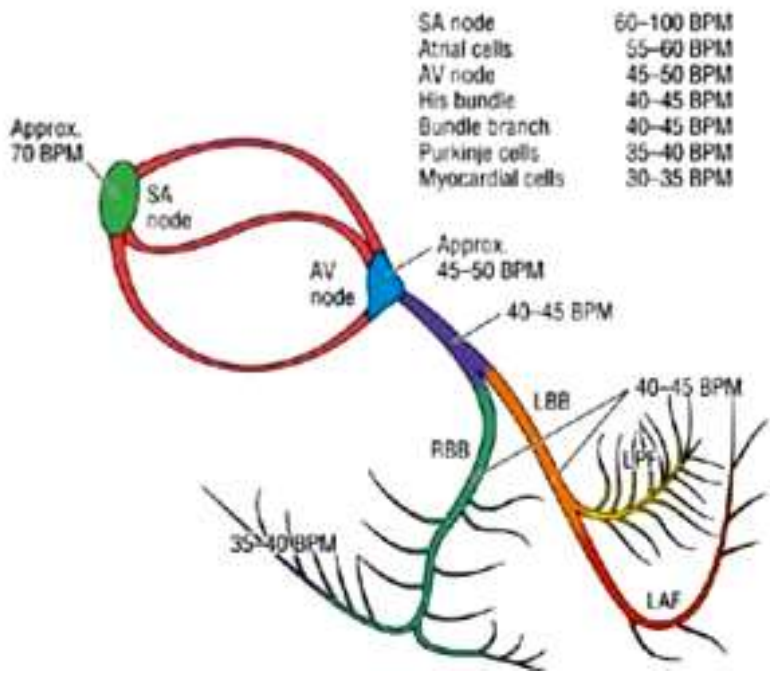
# Physiological review

- Heart – special volume-pressure pump with self organized pacing & conductive system
- Cardiomyocytes - reticular organisation differing from smooth + skeletal muscle

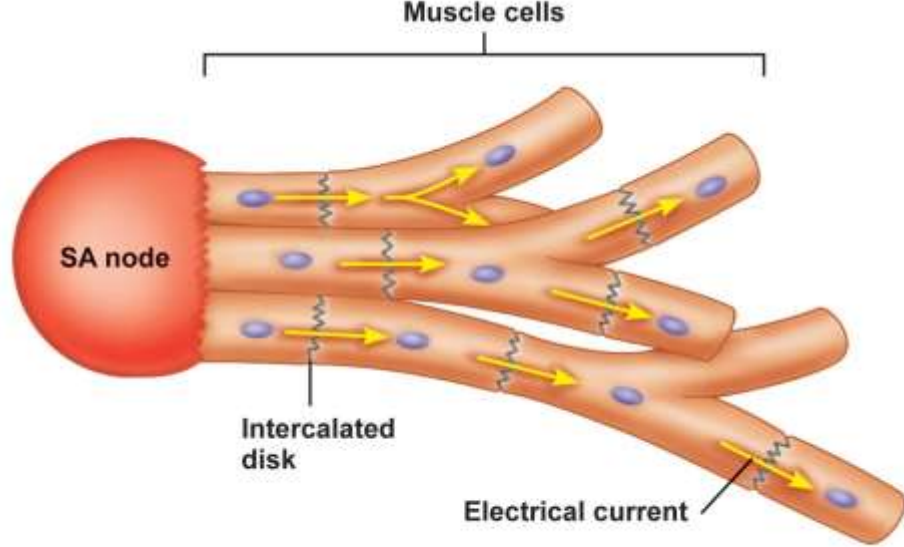
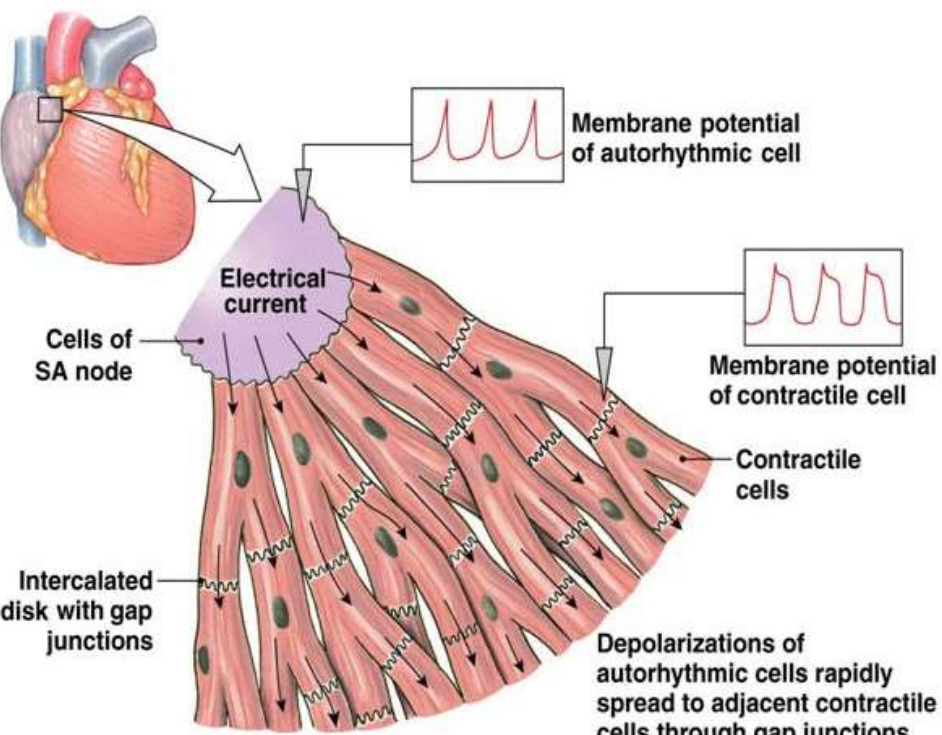




- Cardiomyocytes can conduct electric currents from the cell to cell; intercalated discs electrical synapses
- Conductive system = not nerves but preformed muscle cells; specific anatomy to organize (direction, speed) heart excitability = basic rhythm
- Atria and ventricles are electrically relatively isolated; AV gateway control (+ abnormal bypasses)
- Vegetative nerves + hormones modulate chronotropy, dromotropy, batmotropy



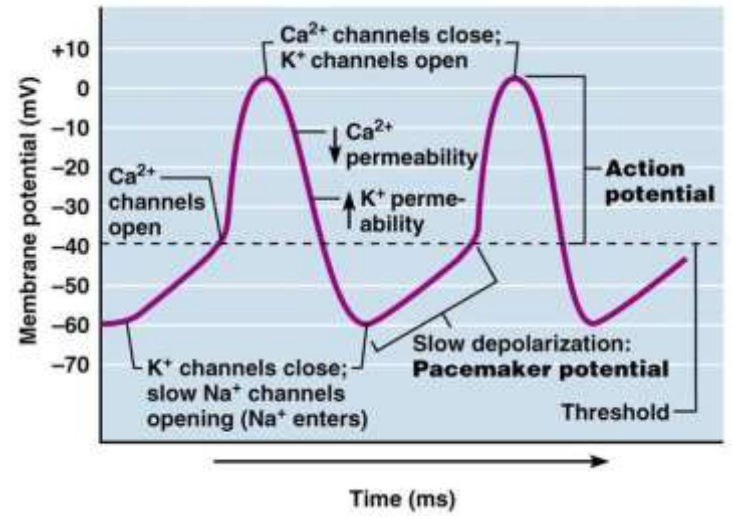
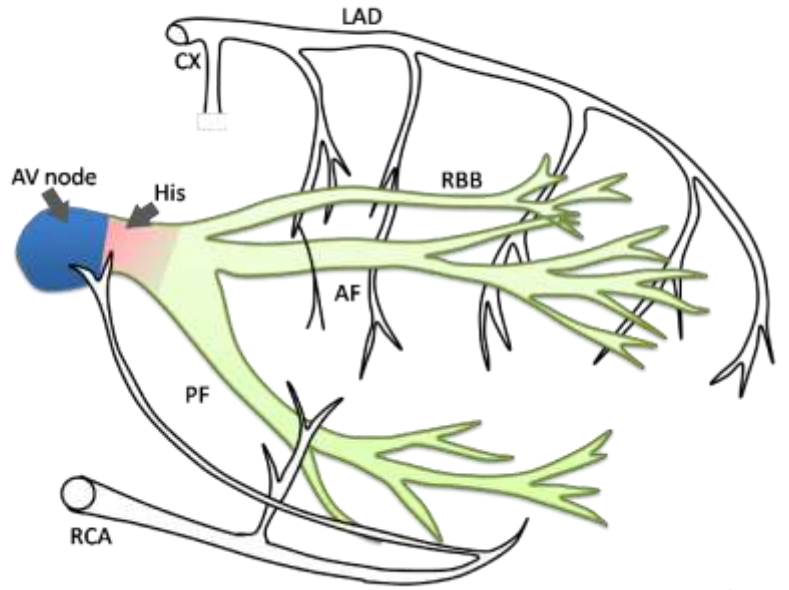


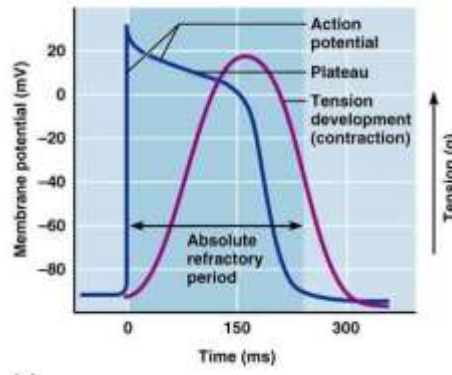
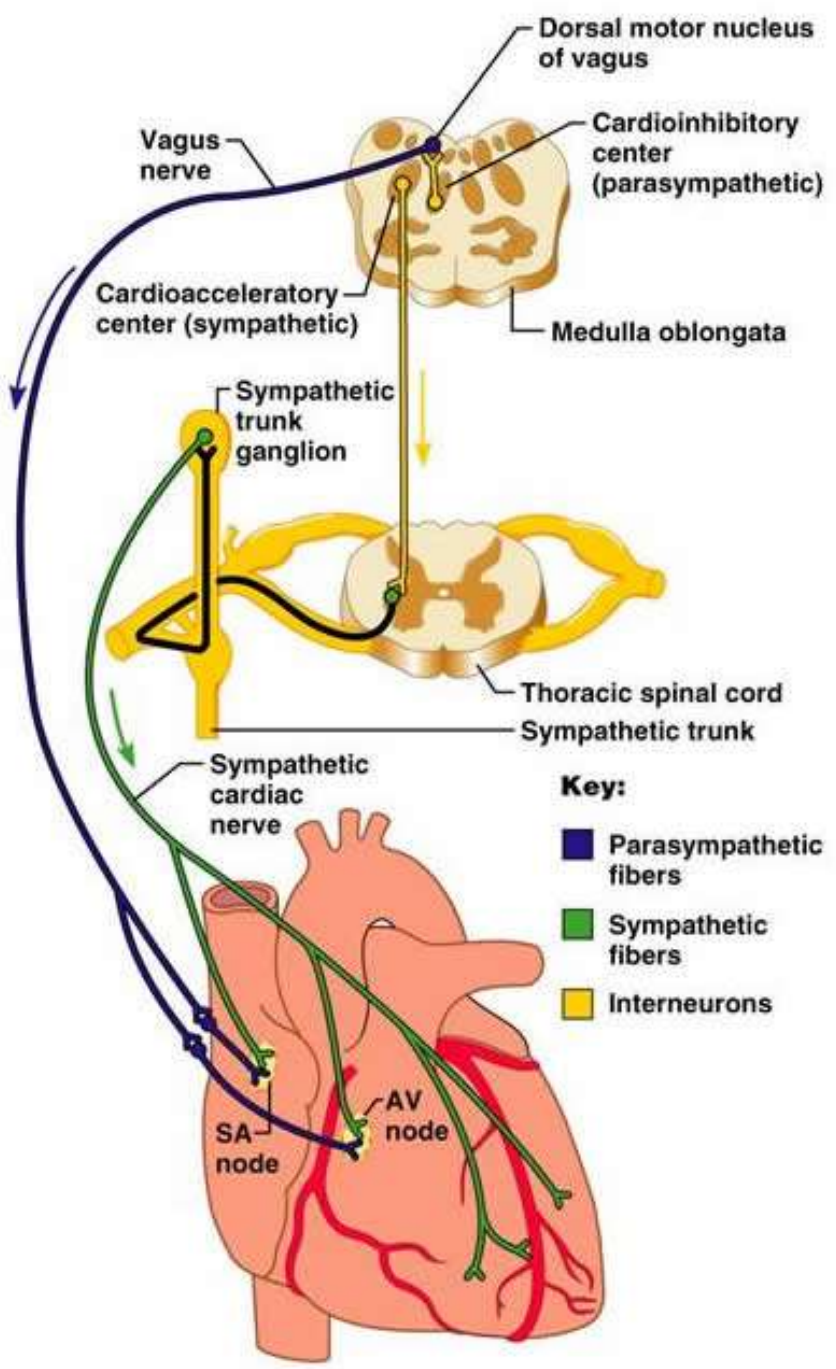


(a)

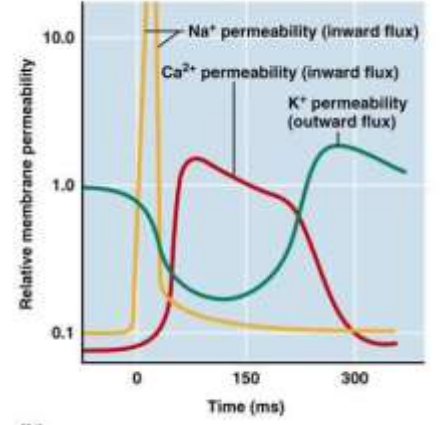
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Coronary blood supply to the cardiac conduction system

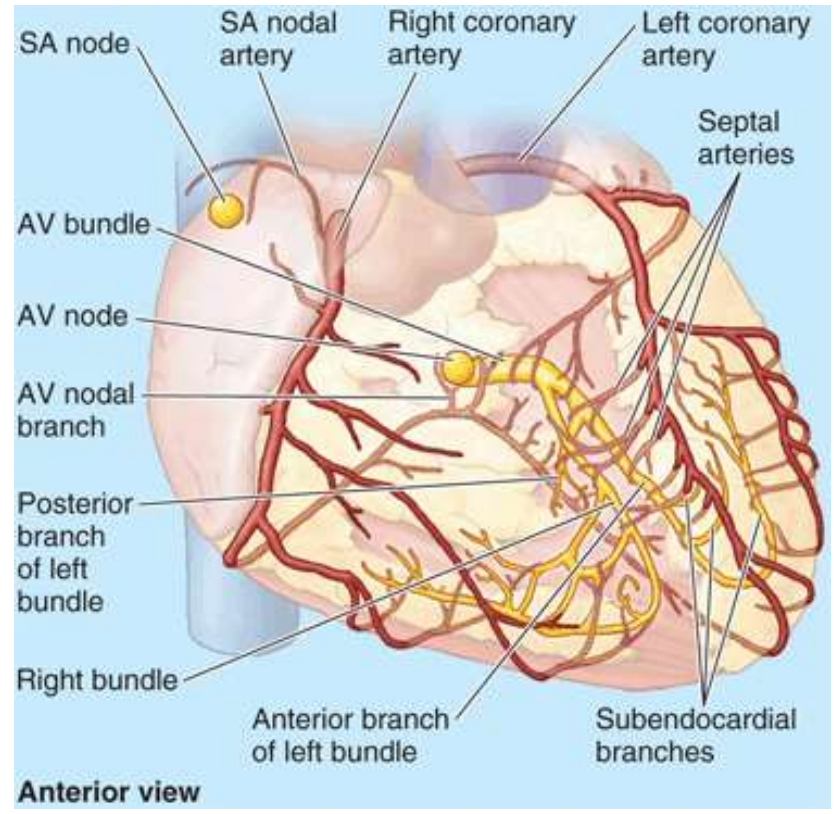




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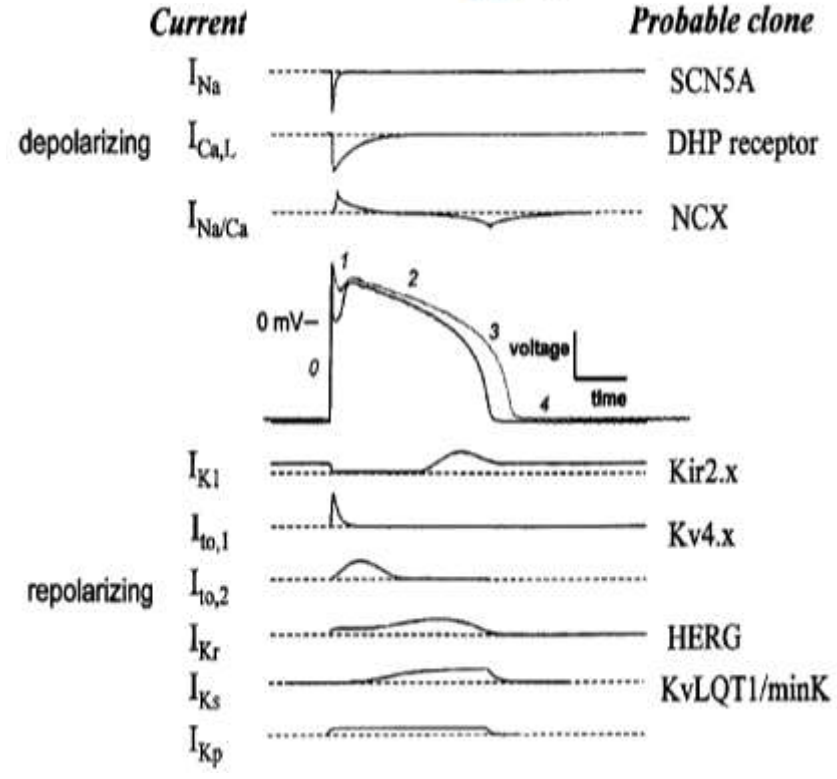
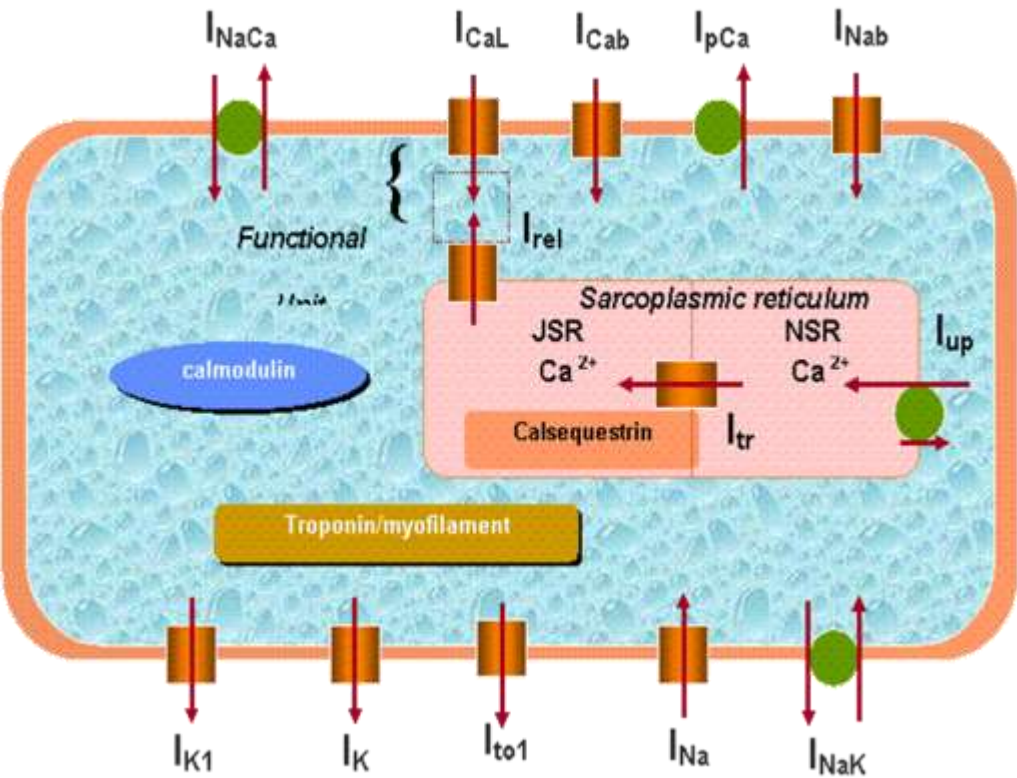
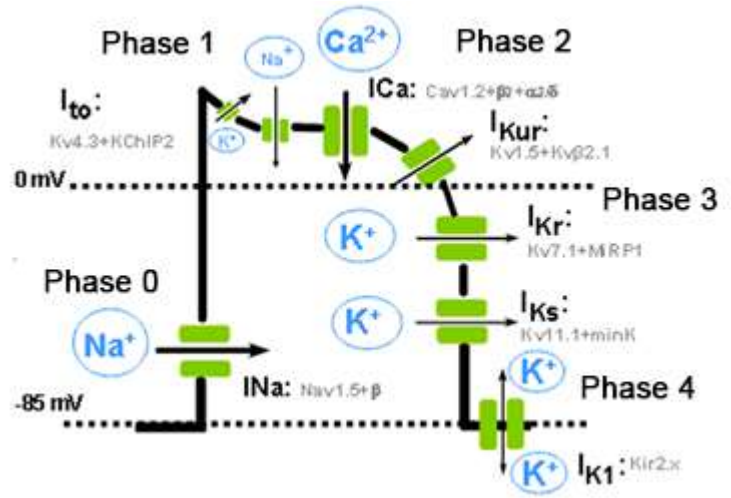
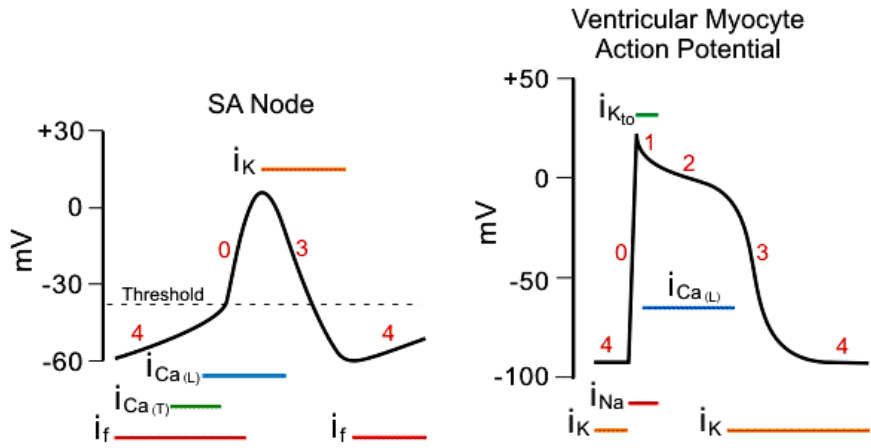


(b)





# Principal ion channels

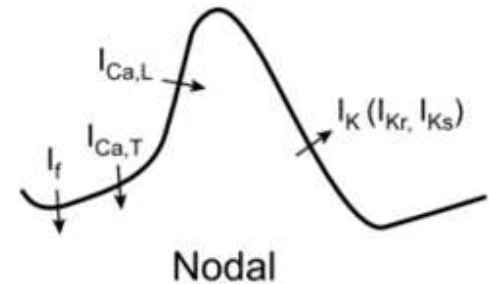
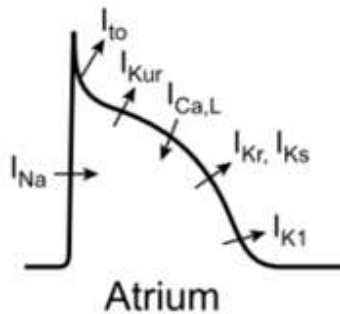
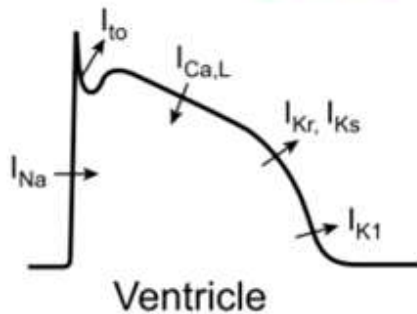


# Principal ion channels in myocardium

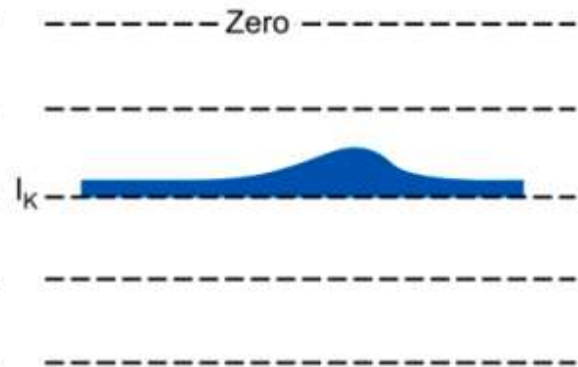
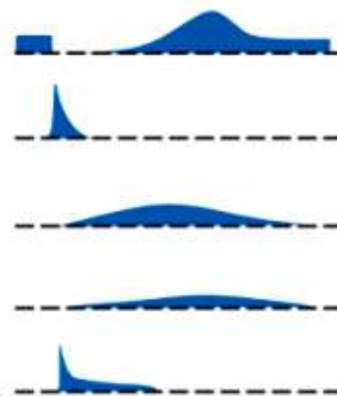
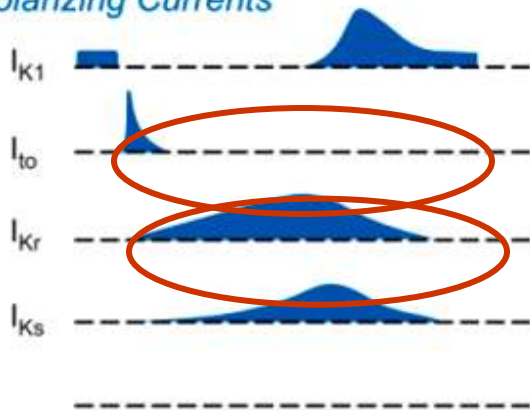
Sodium spikes, Plateau channel

Calcium spikes, Pacemaker channel

Depolarizing Currents



Repolarizing Currents

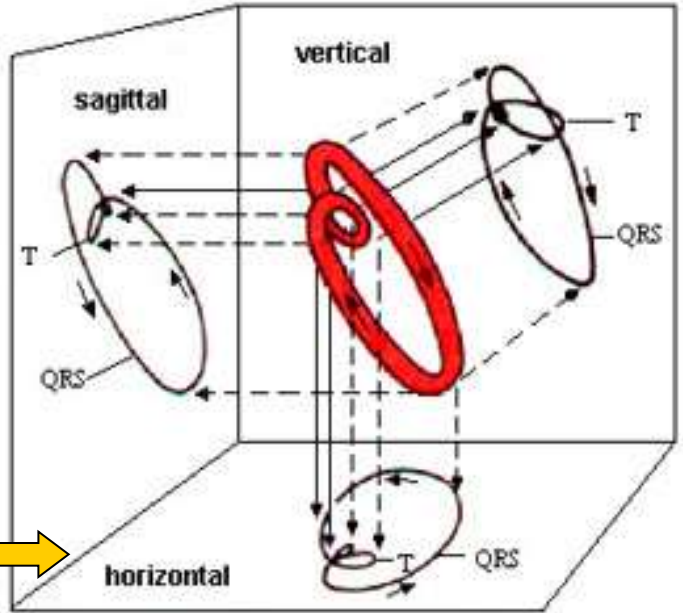






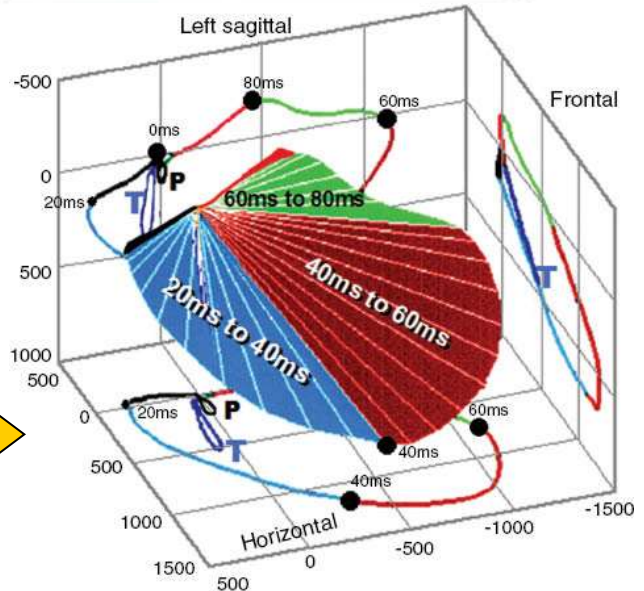
# Principles of ECG

- **Vectorcardiography (VCG)** (developed by E. Frank, 1950s) = method of recording the magnitude and direction of the electrical field in the heart by a continuous series of vectors that form curving lines around a central point.
- Summary vector of heart electrical field describes **3 loops** during the heart cycle in 3D space : (1) **P - atrial depolarization loop** (smallest ), (2) **QRS - ventricular depolarization loop** (biggest), (3) **T- repolarization loop**



- **Electrocardiography (ECG)** is an 2-Dimensional projection of VCG loopings according to given arbitrary system

- **Arbitrarily:** a) **Wave is positive** if the vector runs towards the electrode, b) **Maximal amplitude** is recorded if the vector goes straightly towards the electrode (positive deflection) or out of the electrode ( negative deflection) c) **Amplitude is zero** if the vector rubs in 90 angle with respect to electrode

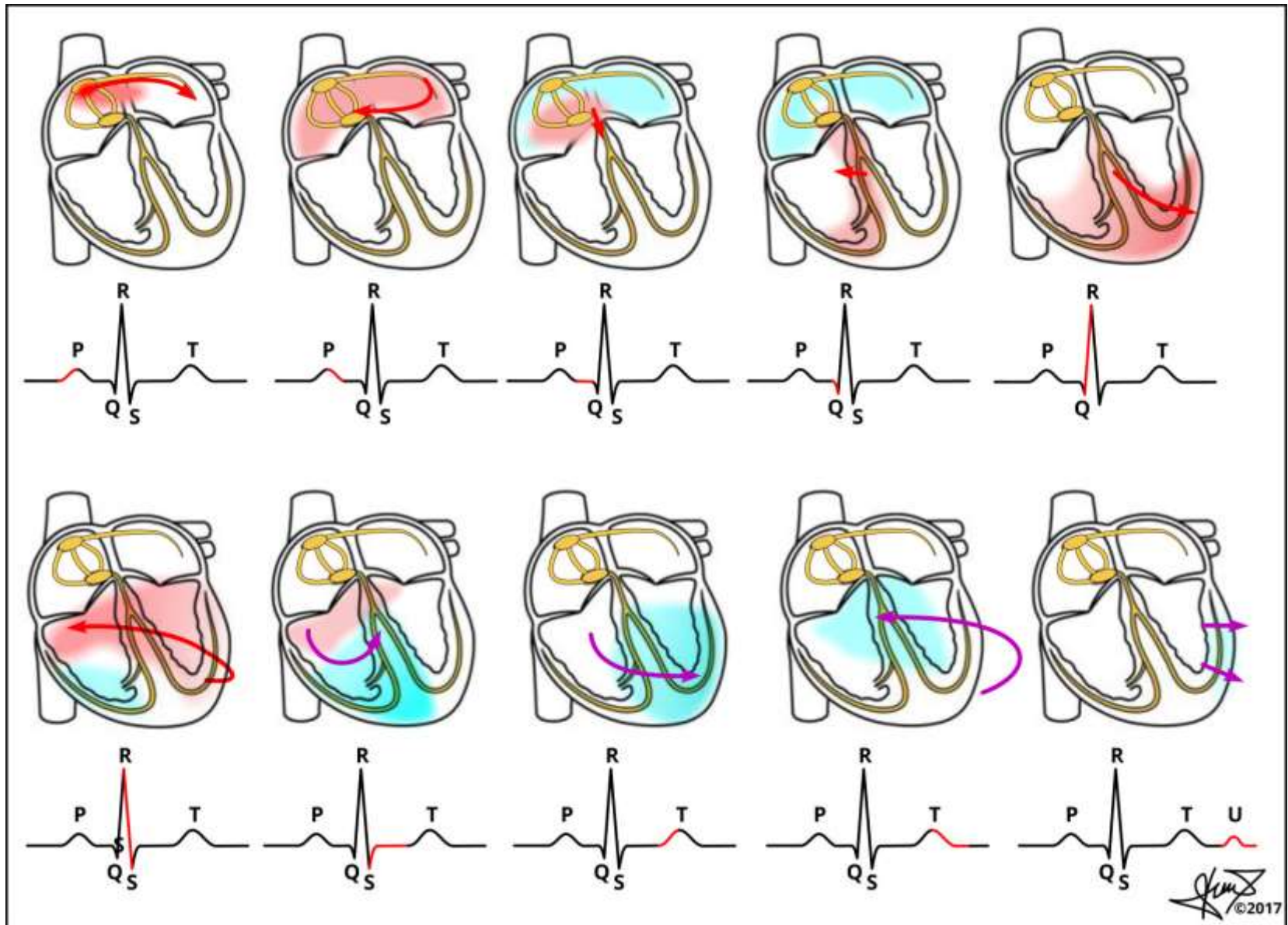


Electrical activity of the heart in the three perpendicular directions X, Y, and Z. The P, QRS, and T wave loops are observed in three different planes (sagittal, frontal, and horizontal)

Color map for time	
0ms to 20ms	60ms to 80ms
20ms to 40ms	80ms to 100ms
40ms to 60ms	T Loop color

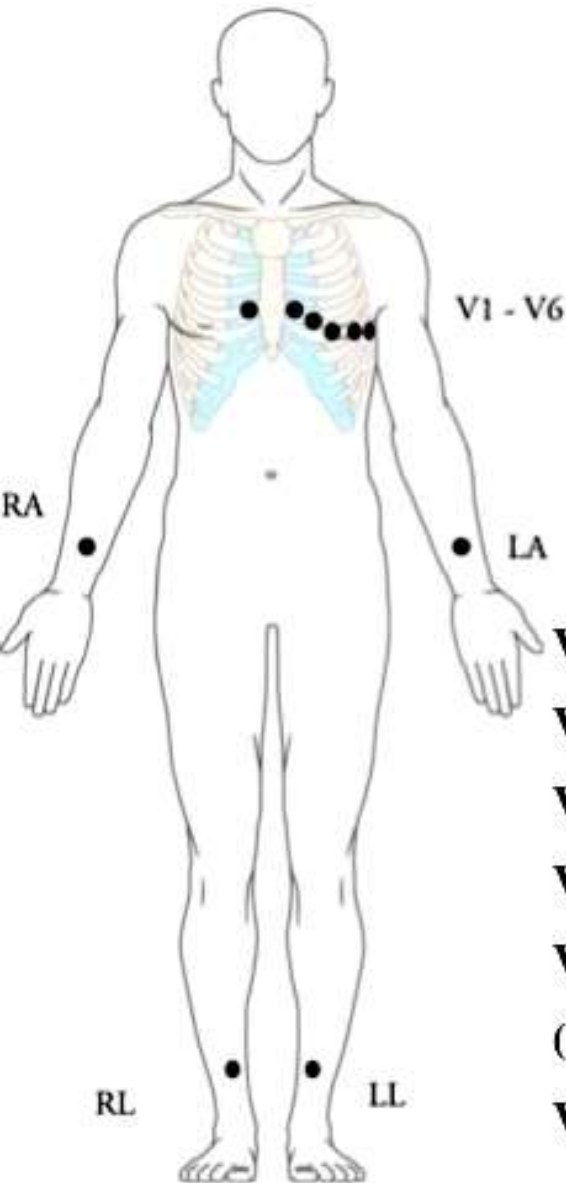
*Hasan, M.A., Abbott, D.: A review of beat-to-beat vectorcardiographic (VCG) parameters for analyzing repolarization variability in ECG signals DOI: <https://doi.org/10.1515/bmt-2015-0005>*

# Generation of ECG waves

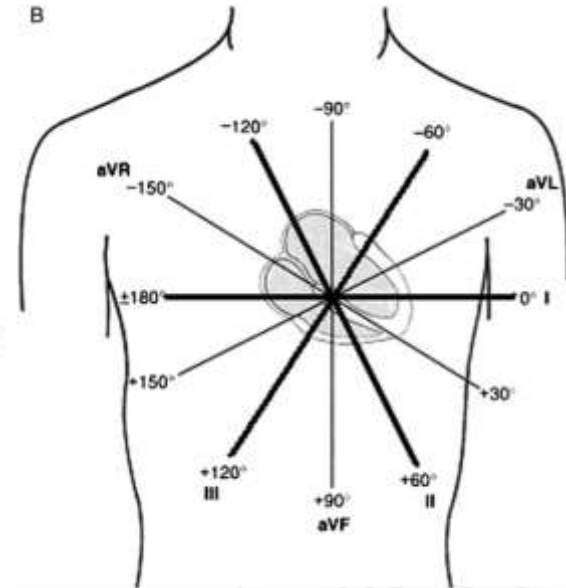
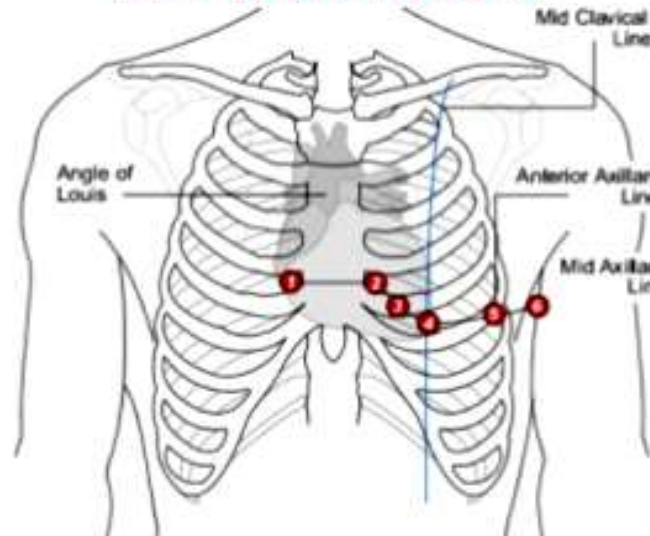




# Electrode placement



## 12 lead ECG



**V1 - 4th Intercostal space, right of Sternum**

**V2 - 4th Intercostal space, left of sternum**

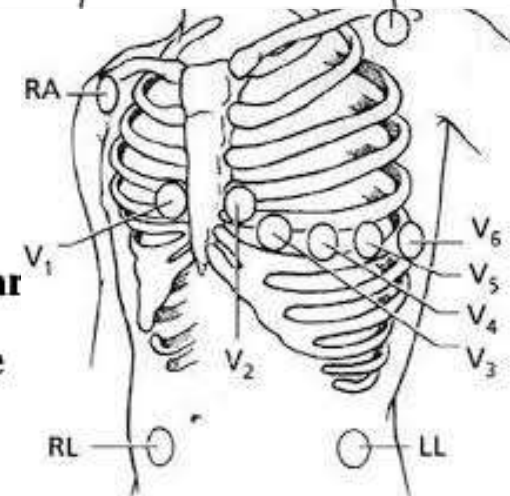
**V3 - Midway between V2 and V4**

**V4 - 5th Intercostal space, in the Midclavicular**

**V5 - same level as V4, at anterior Axillary line**

**(between V4 and V6)**

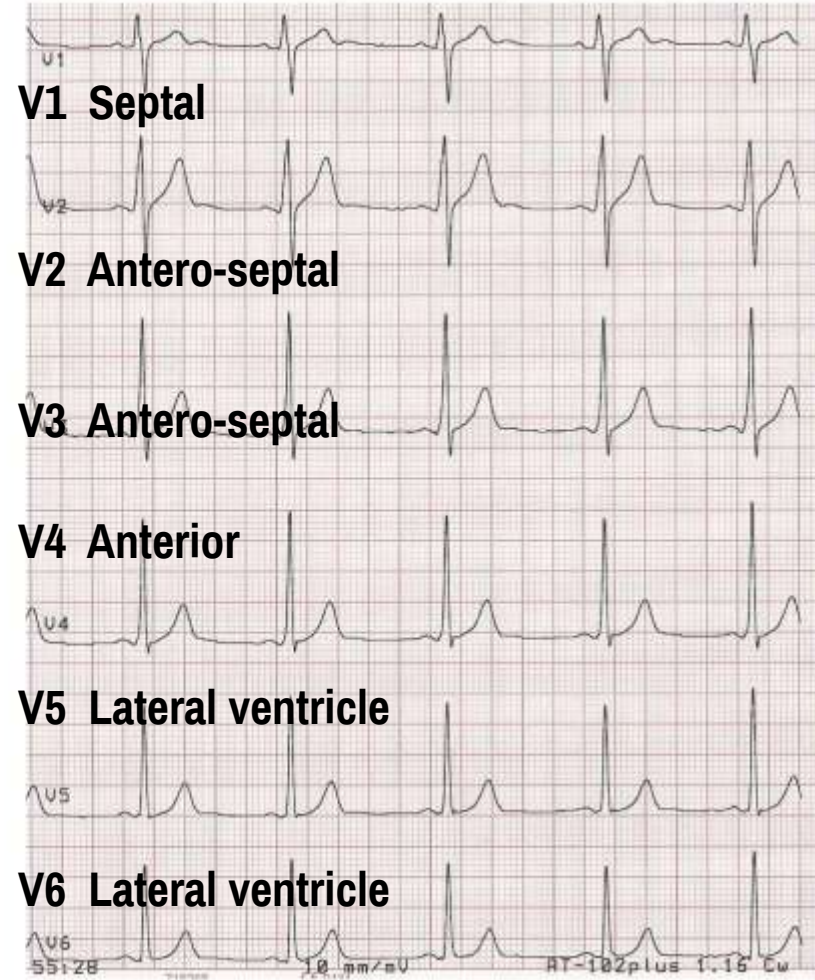
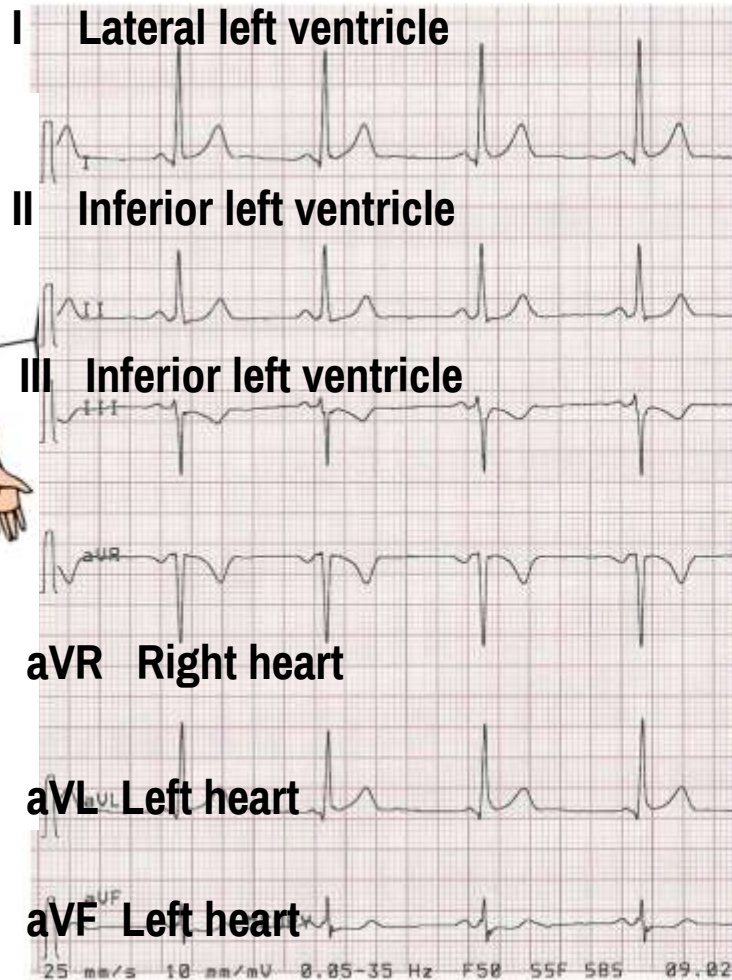
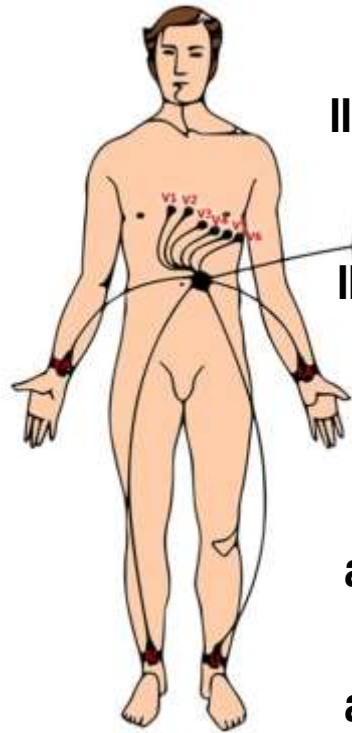
**V6 - in 5th Intercostal space, in the Mid axillary line**



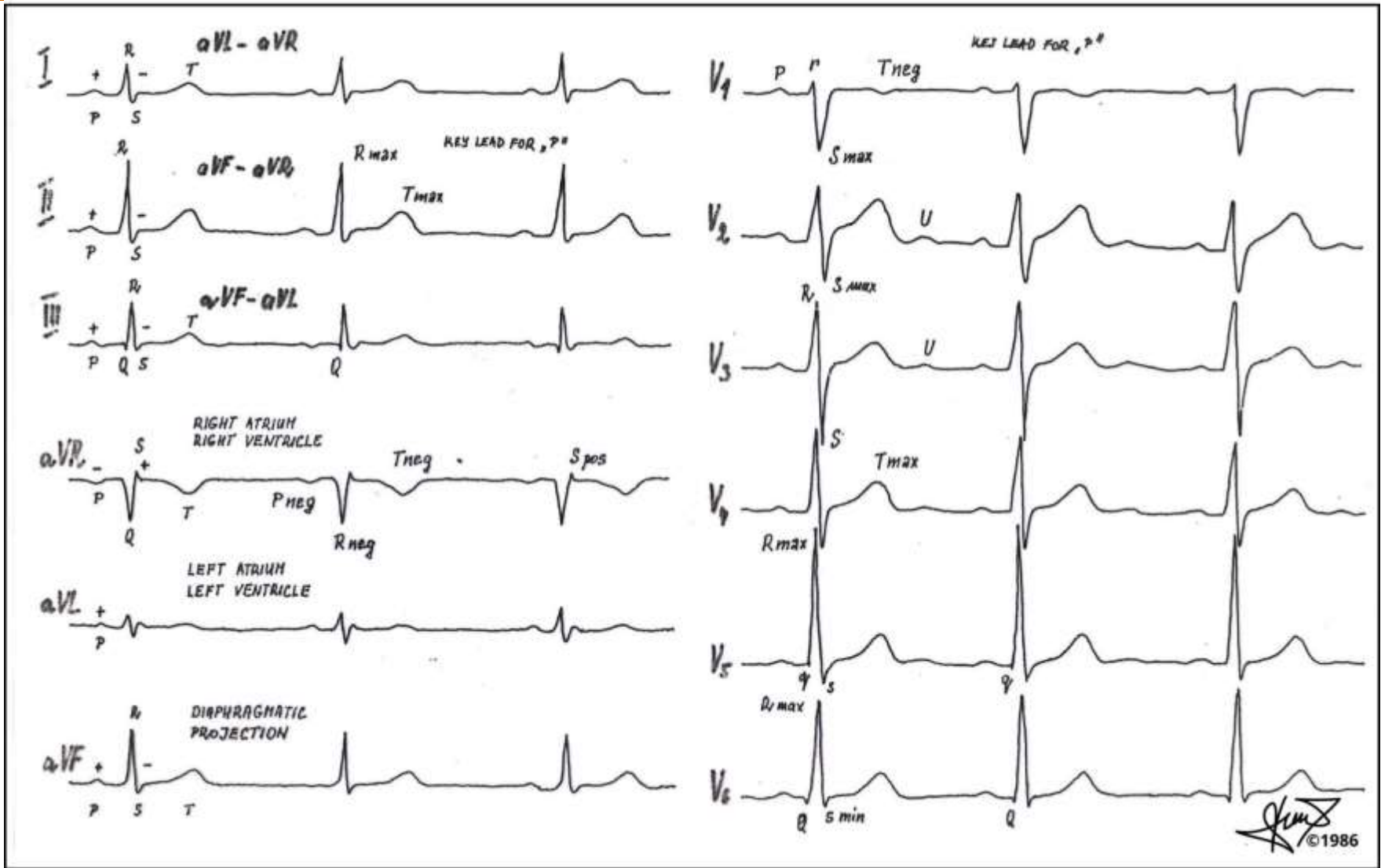
# Standard 12-lead ECG recording

## Limb leads

## Precordial leads



25 mm/s 10 mm/1mV



1. Rhythm regularity and normality, 2. Heart rate,, 3. Electric heart axis, trasmient zone, 4. Normality of wave composition, intervals, segments analysis – P wave, PQ interval , QRS complex, ST segment, T wave, QT interval



# Evaluation of ECG

## 1. Rhythm

- **SA rhythm (physiological):** P wave is present before QRS, PQ interval is constant; depolarisation proceeds from rostral to caudal parts of the heart
- **Supraventricular rhythm:** P wave is not normal, PQ shorter (atrial rhythms) or P wave and PQ are absent before QRS; morphology of QRS may be normal; depolarisation may proceed both rostrally and caudally ( e.g. AV nodal rhythm) thus atrial depolarisation may be hidden
- **Ventricular rhythms:** distinct from normal rhythms

2. **Axis** – left- oriented or right oriented

3. **Waves**

4. **Intervals and segments**

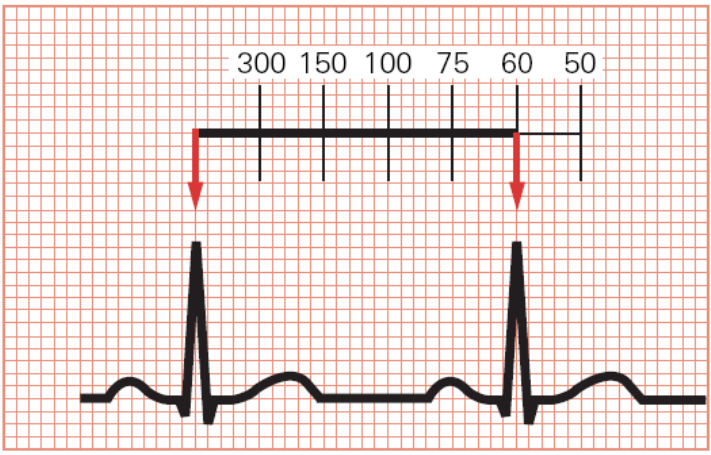
# Electrocardiography – Frequency calculation

1. **Counting large boxes.** If a sweep speed is 0,2 s/large box (LB) = 300 LB/min; **HR** = 300/number of LB in between RR intervals

2. **Counting R waves in 6 sec strip x 10.**



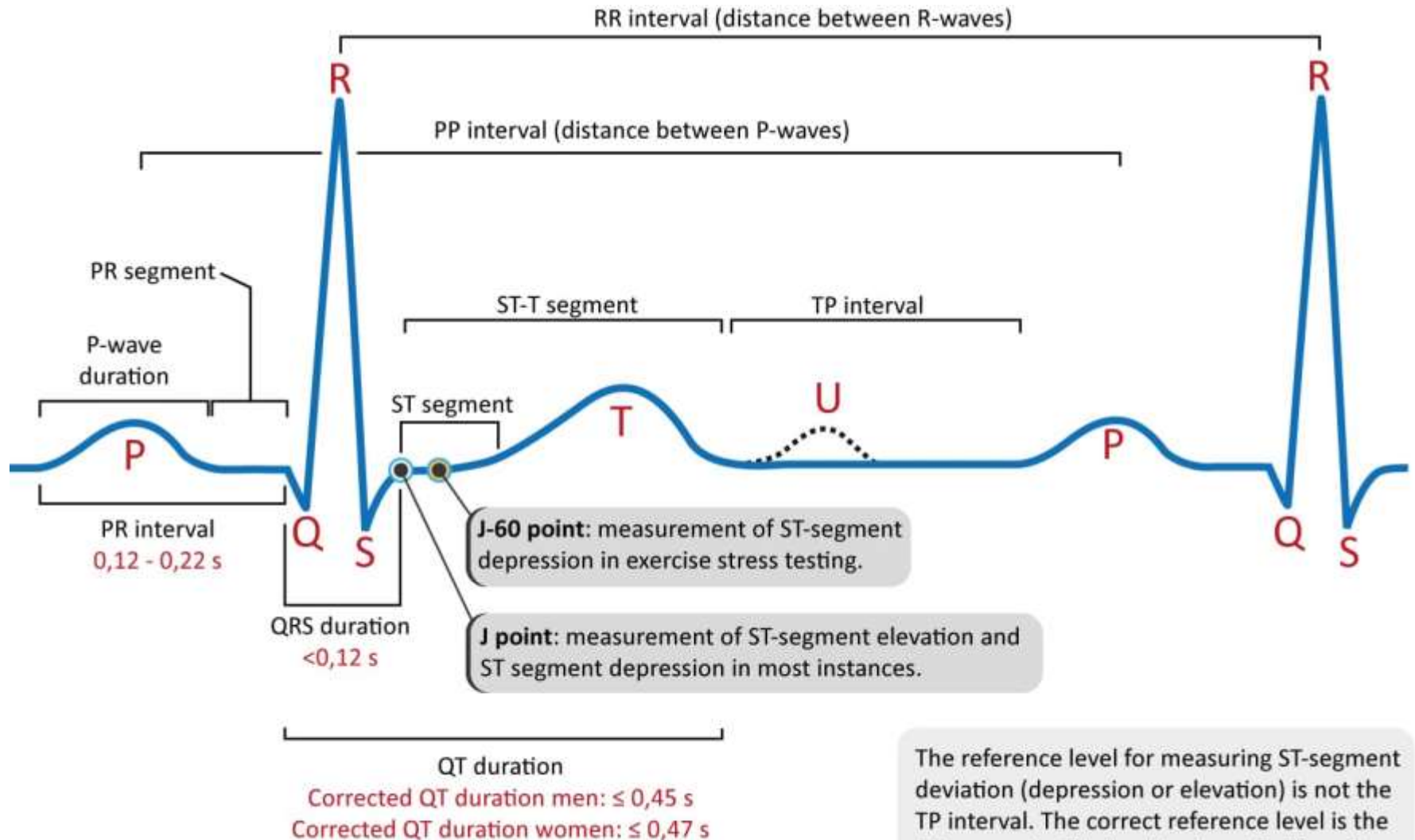
HR = 300/5 = 60 b/min



3. **Counting large & small boxes.**

Number of Large Boxes	Rate/Min	Number of Small Boxes	Rate/Min
1	300	2	750
2	150	3	500
3	100	4	375
4	75	5	300
5	60	6	250
6	50	7	214
7	43	8	186
8	38	9	167
9	33	10	150
10	30	11	136
11	27	12	125
12	25	13	115
13	23	14	107
14	21	15	100
15	20	16	94

# ECG waves and intervals



The reference level for measuring ST-segment deviation (depression or elevation) is not the TP interval. The correct reference level is the **PR segment**. This level is also called **baseline level** or **isoelectric level**.





# Arrhythmias

# Cardiac arrhythmias (dysrhythmias)

- **Definition: Cardiac dysrhythmias** = group of disorders of cardiac electrical rhythm aut pacing and distribution in which the heartbeat may show irregularities or ECG abnormalities with no change in normal frequency, or too fast or too slow.
- **Epidemiology:** affect millions of people, occur at any age incl. children; more common among older people; Sudden cardiac death is the cause 1/2 of deaths due to cardiovascular disease or about 15% of all deaths globally. About 80% of sudden cardiac death ← ventricular arrhythmias. (atrial fibrillation and atrial flutter = 112,000 deaths (2013))
- **Clinical manifestations:**
  - Many types of arrhythmia have **no symptoms, are not serious**
  - Typical symptoms include - **palpitations, feeling a pause** between heartbeats, lightheadedness, shortness of breath, chest pain
  - Sudden serious complications – **heart failure, cardiac arrest.**

# Cardiac arrhythmias (cont.)

## ■ Etiology:

- Specific cardiac and non-specific **channelopathies**;
- **Congenital/acquired defects** in electrical **conduction system** of the heart ( e-g. abnormalities of resting ECG, pre excitation (short PR interval)
- **Structural cardiac diseases** - mitral valve dis., LV aneurysm, congenital heart diseases
- **Ischemic heart disease** = mother of many arrhythmias (conductive system & myocard);, (angina, recent myocardial infarction)
- **Internal milieu disturbances** = ↓ or ↑ K<sup>+</sup> hyper-/hypokalemia; ↓ or ↑ Ca<sup>2+</sup> hypo/hypercalcemia; ↓ Mg<sup>2+</sup>, acidosis/alkalosis; hypoxia, hypercarbia ↓ PaO<sub>2</sub>, ↑ PaCO
- **Miscellaneous:** Febrile illness, Emotional stress, Smoking, Fatigue
- **Hormonal dysbalance** (thyroid hormones = hyper-/hypothyroidism, growth hormone, estrogens, testosterone)
- **Vegetative dystonia** - sympathetic hyperreactors (tachycardic arrhythmias; vagal hyperresponsiveness (bradycardia) Pheochromocytoma
- **Drugs** Anti-arrhythmics, Para/ sympathomimetics (β<sub>2</sub> agonists, cocaine), antidepressants, caffeine), Alcohol



# Cardiac arrhythmias (dysrhythmias)

## ■ Classification:

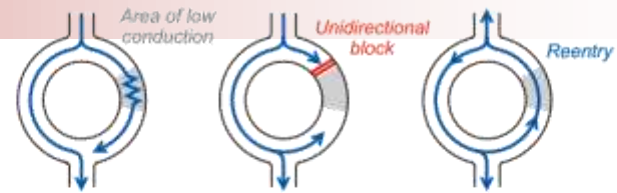
- **According to origin:** a) **Nomotopic** (sinus rhythm) = generated in sinoatrial node ; b) **Ectopic** = released from locations from elsewhere
- **According to ectopic location:** a) **Supraventricular arrhythmias** (incl. **Atrial arrhythmias + Nodal arrhythmias**= atrioventricular node area) b) **Ventricular dysrhythmia** (generated in conductive system (Hiss bundle, Tawara bundles + myocardium of ventricles))
- **According to stability of pacing :** a) **Rhythms** (= paroxysms/ or longer periods (minutes) with out of normal rhythmicity, ECG wave composition, etc.) b) **Extrabeats (captured beats,** short periods, several or individual QRST complexes)
- **Extra beats** include premature **atrial**, premature **ventricular** contractions and premature **junctional** contractions.
- **According to regularity:** a) **regular** (equal RR intervals), (e.g. sinus bradycardia, tachycardia) **irregular** (non-equal RR int.), e.g. sinus arrhythmia, extrasystoles
- **According to contraction frequency:** a) **normocardic rhythms** = 60 - 100 b/ min in adults; b) **tachycardic rhythms** >100 b/ min (hypoxia, ischemia to the heart !!) c) **bradycardic rhythms** <60 b/ min in adults

# Cardiac arrhythmias

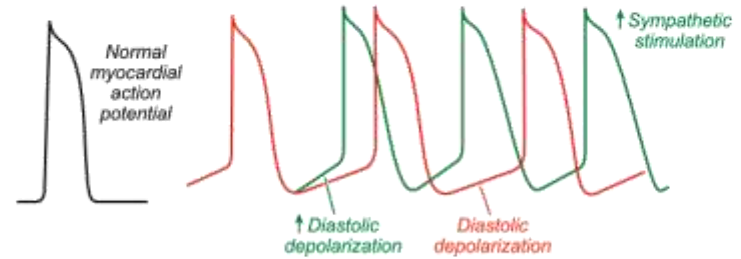
## Mechanisms:

- Abnormal / hidden / revediced pacemakers (excuted by pathological condtirions)
- Abnormal automaticity (effects of hormones, nervous drive)
- Triggered activity – EAD, DAD (tetanic activity, refractory phases)
- Reentry circuits – small or long loop reentries,
- Channelopathies – specific disorders

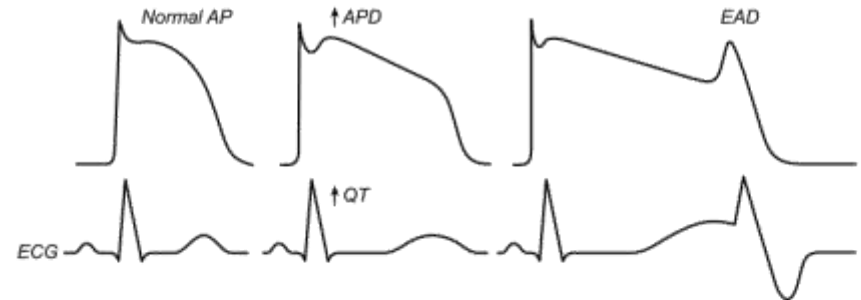
### Reentry



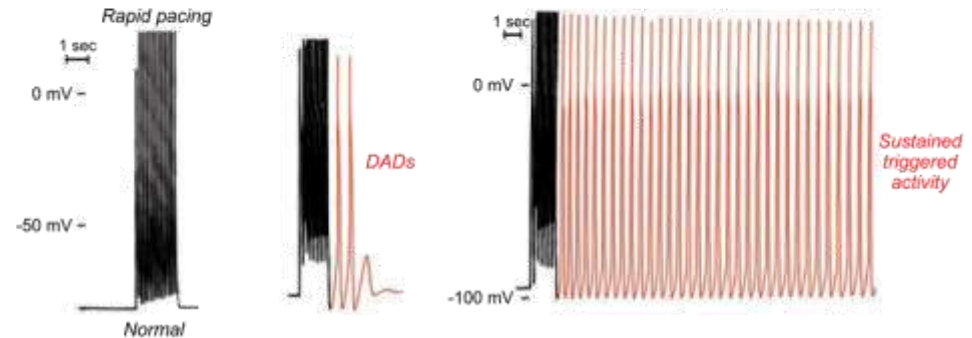
### Automaticity



### Triggered Activity–EAD



### Triggered Activity–DAD





# Sinus Arrhythmias

- P wave has normal morphology
- QRS and T wave are of normal morphology

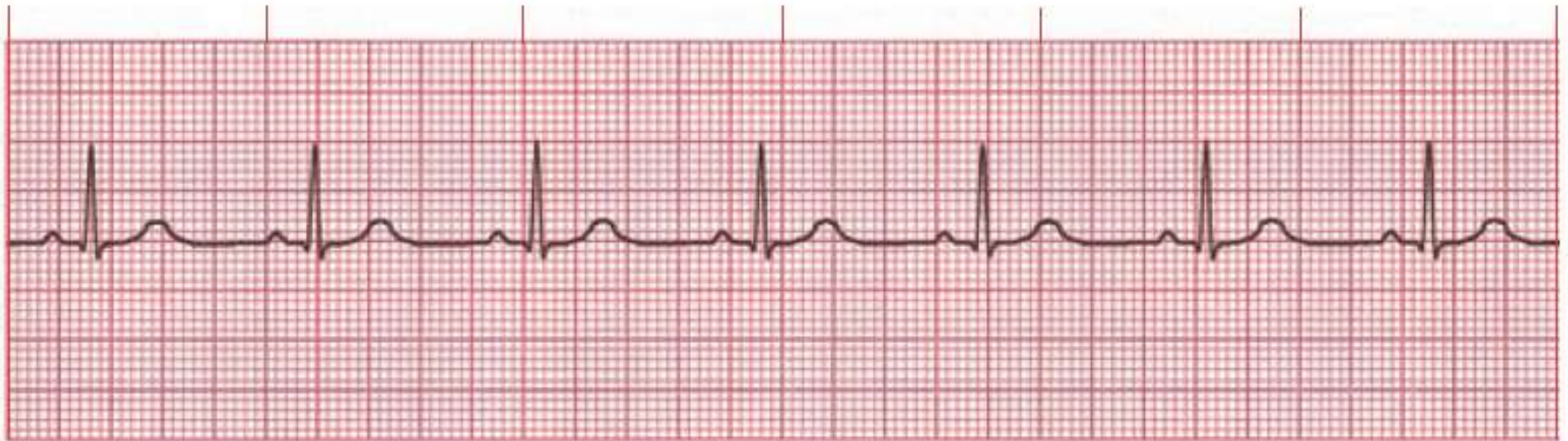


## Sinoatrial (SA) Node Arrhythmias

- Upright P waves all look similar.
- PR intervals and QRS complexes are of normal duration.

**Note:** All ECG strips in this tab were recorded in lead II.

### Normal Sinus Rhythm (NSR)



**Rate:** Normal (60–100 bpm)

**Rhythm:** Regular

**P Waves:** Normal (upright and uniform)

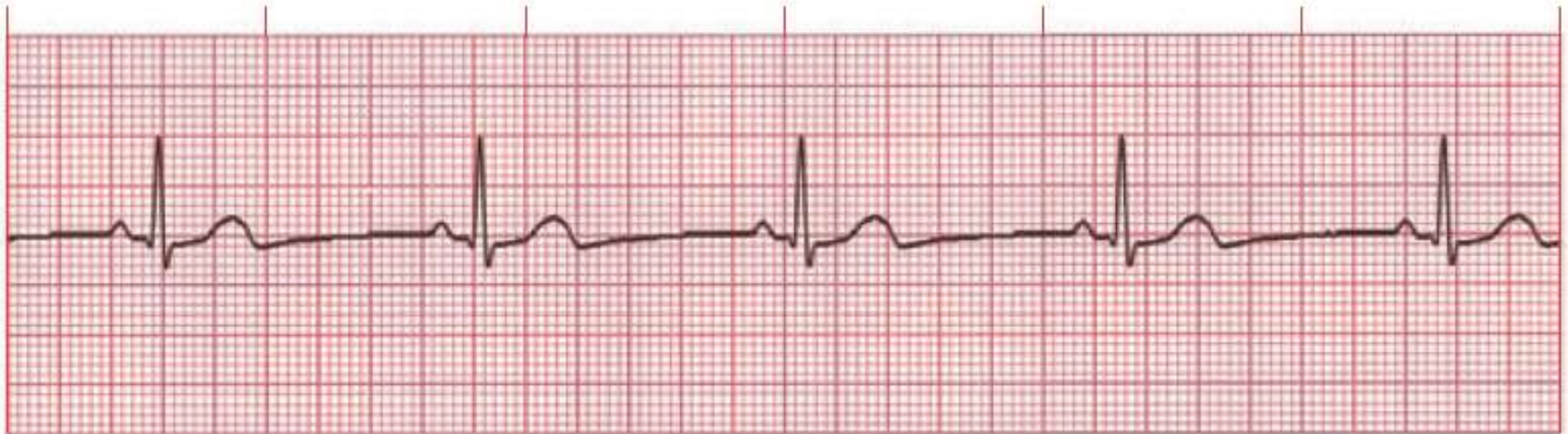
**PR Interval:** Normal (0.12–0.20 sec)

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** A normal ECG does not exclude heart disease.

## Sinus Bradycardia

- Results from slowing of the SA node.



**Rate:** Slow (<60 bpm)

**Rhythm:** Regular

**P Waves:** Normal (upright and uniform)

**PR Interval:** Normal (0.12–0.20 sec)

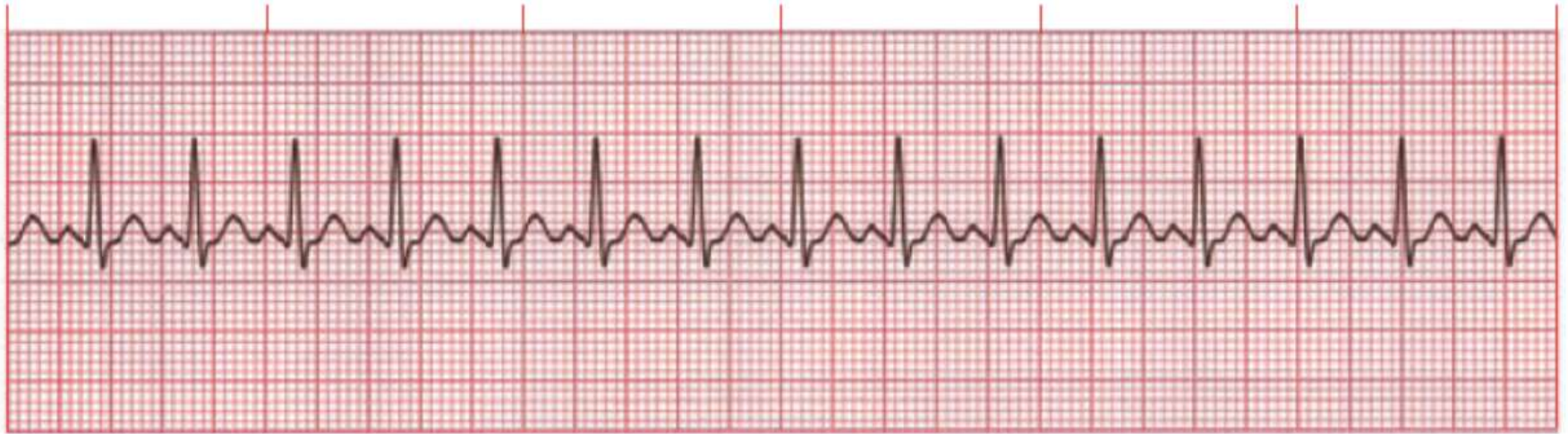
**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Sinus bradycardia is normal in athletes and during sleep. In acute MI, it may be protective and beneficial or the slow rate may compromise cardiac output. Certain medications, such as beta blockers, may also cause sinus bradycardia.



## Sinus Tachycardia

- Results from increased SA node discharge.



**Rate:** Fast ( $>100$  bpm)

**Rhythm:** Regular

**P Waves:** Normal (upright and uniform)

**PR Interval:** Normal (0.12–0.20 sec)

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Sinus tachycardia may be caused by exercise, anxiety, fever, hypoxemia, hypovolemia, or cardiac failure.



## Sinus Arrhythmia

- The SA node discharges irregularly.
- The R-R interval is irregular.



**Rate:** Usually normal (60–100 bpm); frequently increases with inspiration and decreases with expiration

**Rhythm:** Irregular; varies with respiration

**P Waves:** Normal (upright and uniform)

**PR Interval:** Normal (0.12–0.20 sec)

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** The pacing rate of the SA node varies with respiration, especially in children and elderly people.

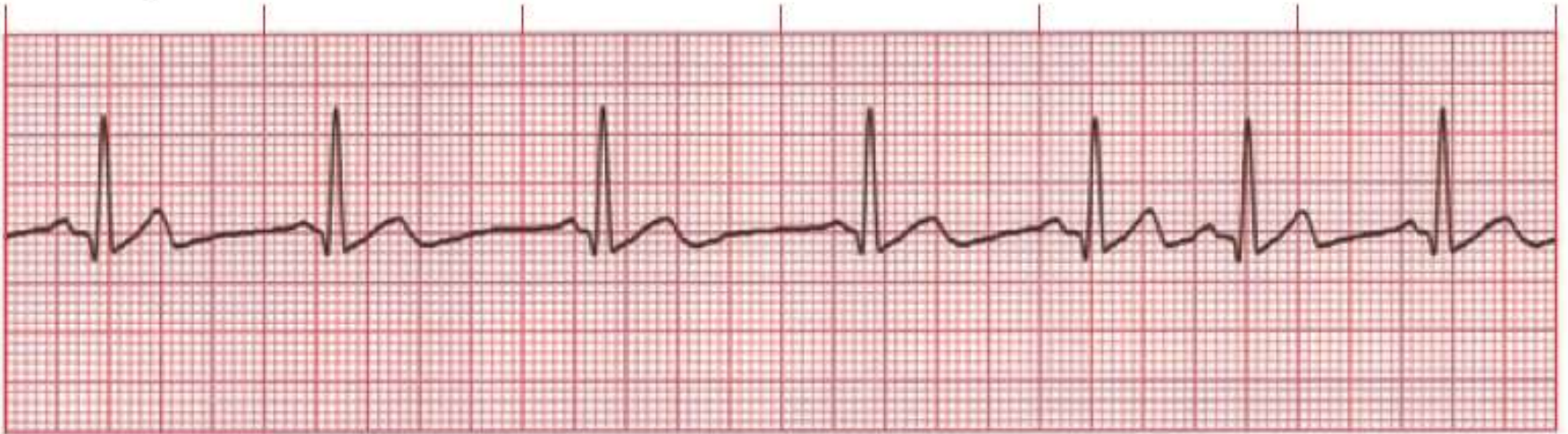


# Atrial Arrhythmias

- P wave is different from that generated in SA node
- QRS and T wave are of normal morphology

## Wandering Atrial Pacemaker (WAP)

- Pacemaker site transfers from the SA node to other latent pacemaker sites in the atria and the AV junction and then moves back to the SA node.



**Rate:** Normal (60–100 bpm)

**Rhythm:** Irregular

**P Waves:** At least three different forms, determined by the focus in the atria

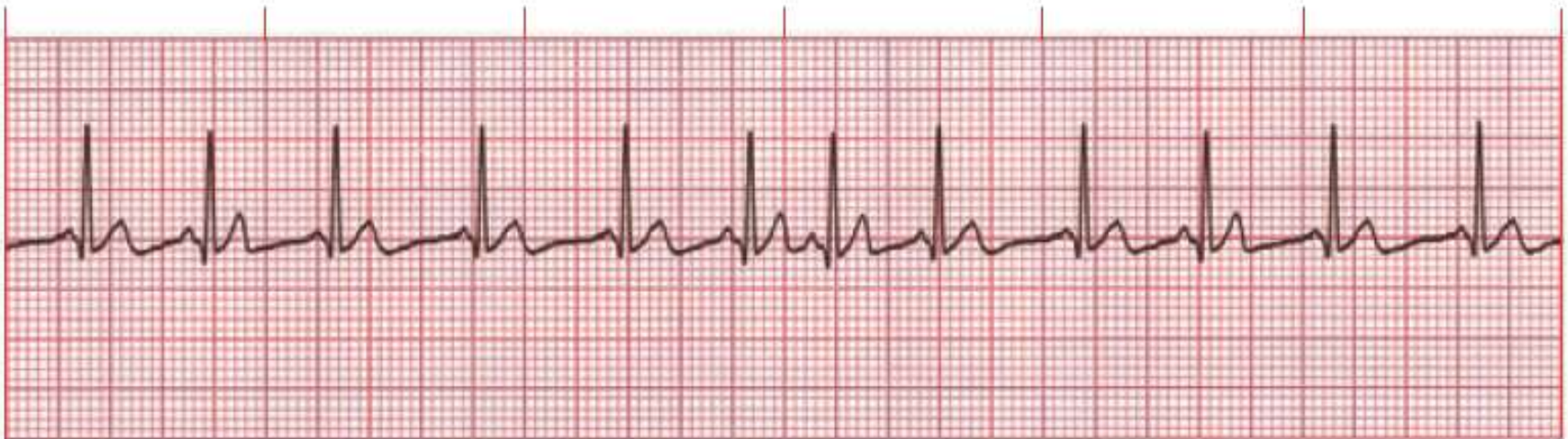
**PR Interval:** Variable; determined by focus

**QRS:** Normal (0.06–0.10 sec)



## Multifocal Atrial Tachycardia (MAT)

- This form of WAP is associated with a ventricular response of  $>100$  bpm.
- MAT may be confused with atrial fibrillation (A-fib); however, MAT has a visible P wave.



**Rate:** Fast ( $>100$  bpm)

**Rhythm:** Irregular

**P Wave:** At least three different forms, determined by the focus in the atria

**PR Interval:** Variable; depends on focus

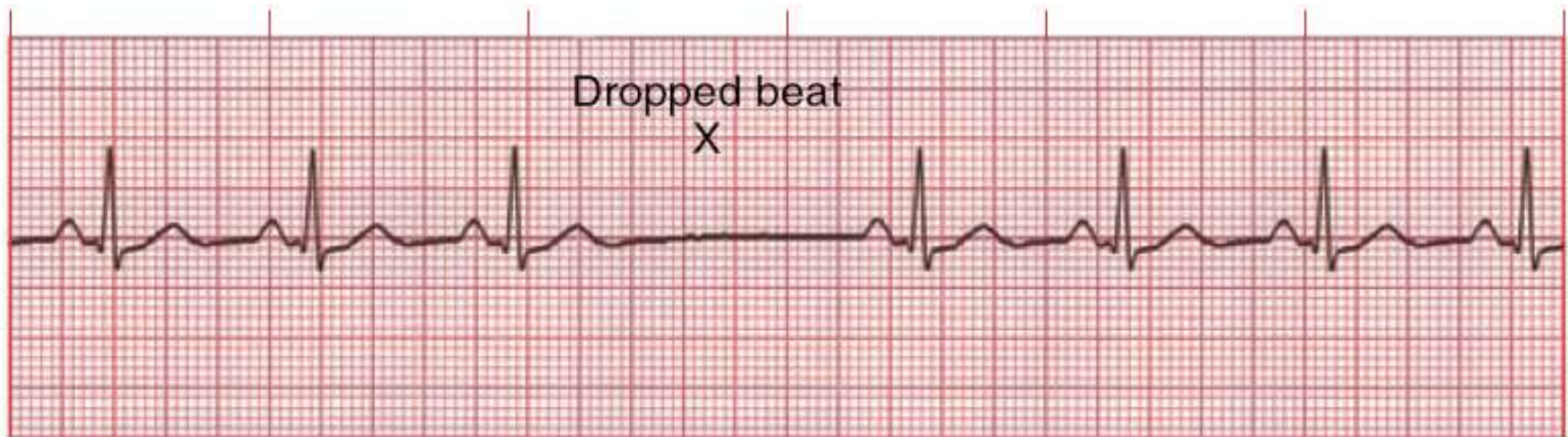
**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** MAT is commonly seen in patients with COPD but may also occur in acute MI.



## Sinoatrial (SA) Block

- The block occurs in some multiple of the P-P interval.
- After the dropped beat, cycles continue on time.



**Rate:** Normal to slow; determined by duration and frequency of SA block

**Rhythm:** Irregular whenever an SA block occurs

**P Waves:** Normal (upright and uniform) except in areas of dropped beats

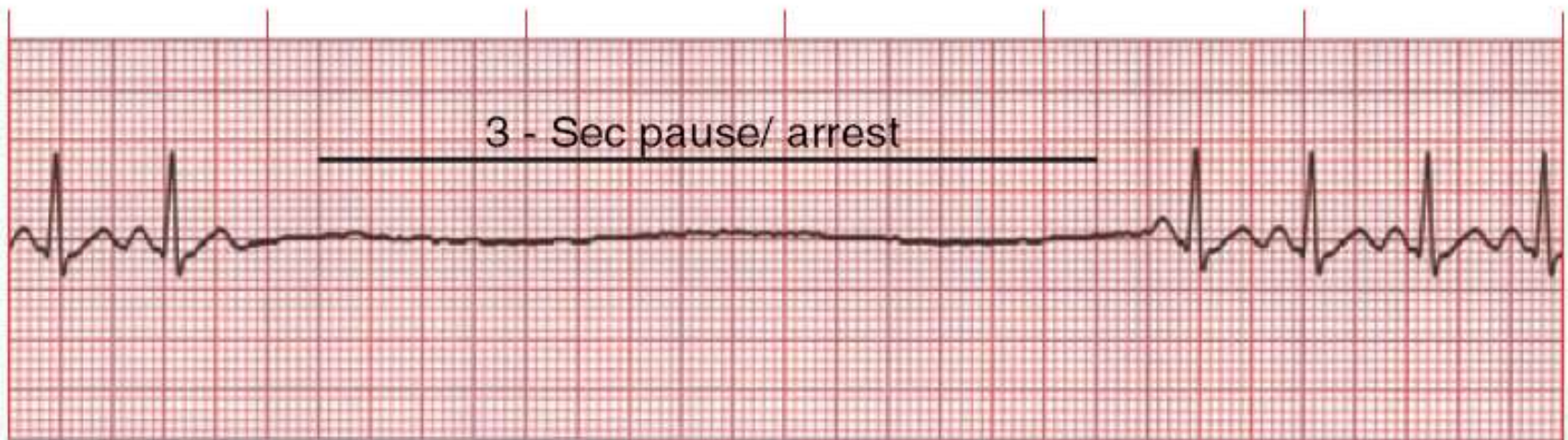
**PR Interval:** Normal (0.12–0.20 sec)

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Cardiac output may decrease, causing syncope or dizziness.

## Sinus Pause (Sinus Arrest)

- The SA node fails to discharge and then resumes.
- Electrical activity resumes either when the SA node resets itself or when a lower latent pacemaker begins to discharge.
- The pause (arrest) time interval is not a multiple of the normal P-P interval.



**Rate:** Normal to slow; determined by duration and frequency of sinus pause (arrest)

**Rhythm:** Irregular whenever a pause (arrest) occurs

**P Waves:** Normal (upright and uniform) except in areas of pause (arrest)

**PR Interval:** Normal (0.12–0.20 sec)

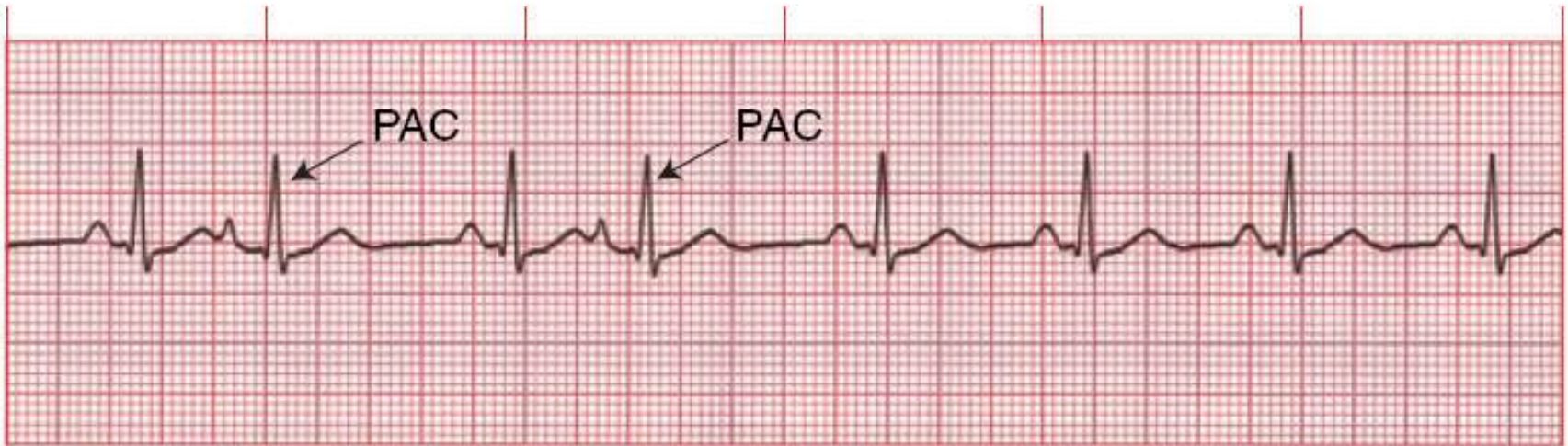
**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Cardiac output may decrease, causing syncope or dizziness.



## Premature Atrial Contraction (PAC)

- A single complex occurs earlier than the next expected sinus complex.
- After the PAC, sinus rhythm usually resumes.



**Rate:** Depends on rate of underlying rhythm

**Rhythm:** Irregular whenever a PAC occurs

**P Waves:** Present; in the PAC, may have a different shape

**PR Interval:** Varies in the PAC; otherwise normal (0.12–0.20 sec)

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** In patients with heart disease, frequent PACs may precede paroxysmal supraventricular tachycardia (PSVT), A-fib, or A-flutter.

## Atrial Tachycardia

- A rapid atrial rate overrides the SA node and becomes the dominant pacemaker.
- Some ST wave and T wave abnormalities may be present.



**Rate:** 150–250 bpm

**Rhythm:** Regular

**P Waves:** Normal (upright and uniform) but differ in shape from sinus P waves

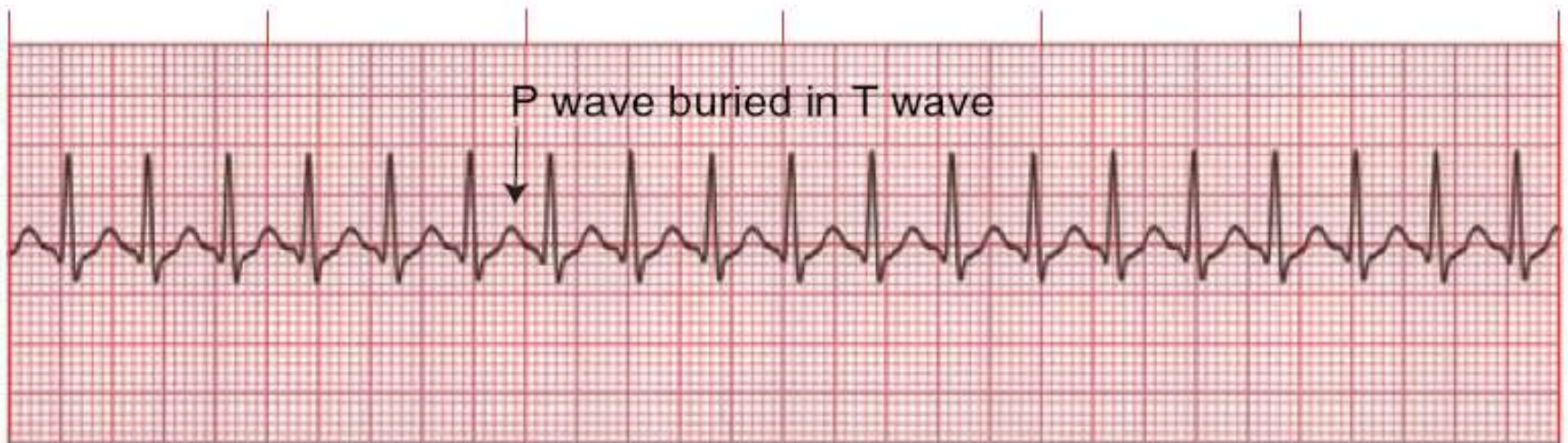
**PR Interval:** May be short (<0.12 sec) in rapid rates

**QRS:** Normal (0.06–0.10 sec) but can be aberrant at times



## Supraventricular Tachycardia (SVT)

- This arrhythmia has such a fast rate that the P waves may not be seen.



**Rate:** 150–250 bpm

**Rhythm:** Regular

**P Waves:** Frequently buried in preceding T waves and difficult to see

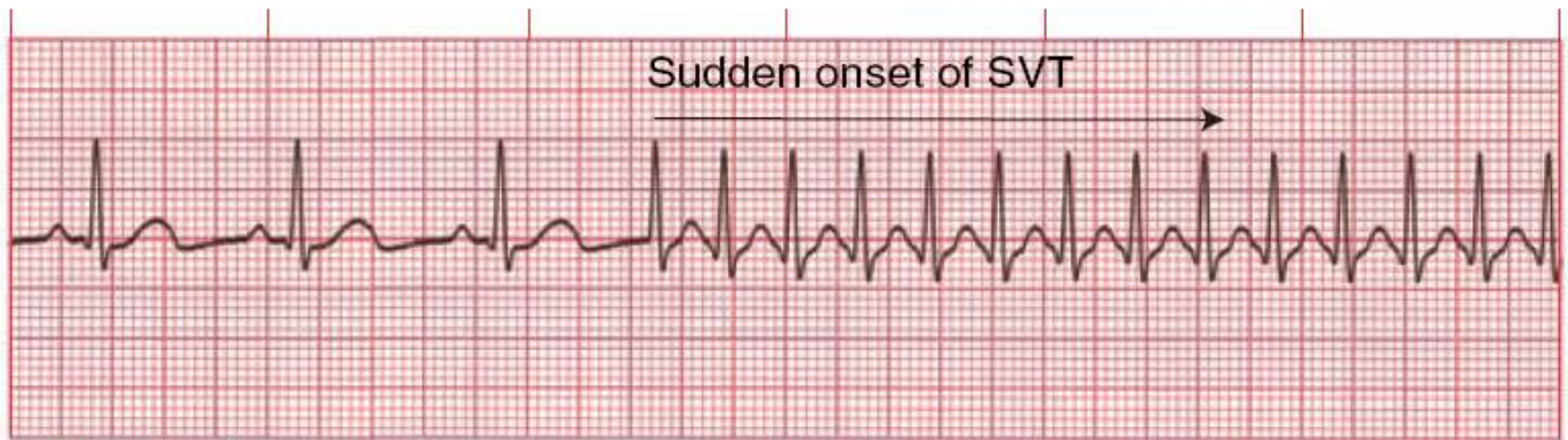
**PR Interval:** Usually not possible to measure

**QRS:** Normal (0.06–0.10 sec) but may be wide if abnormally conducted through ventricles

♥ **Clinical Tip:** SVT may be related to caffeine intake, nicotine, stress, or anxiety in healthy adults.

## Paroxysmal Supraventricular Tachycardia (PSVT)

- PSVT is a rapid rhythm that starts and stops suddenly.
- For accurate interpretation, the beginning or end of the PSVT must be seen.
- PSVT is sometimes called paroxysmal atrial tachycardia (PAT).



**Rate:** 150–250 bpm

**Rhythm:** Regular

**P Waves:** Frequently buried in preceding T waves and difficult to see

**PR Interval:** Usually not possible to measure

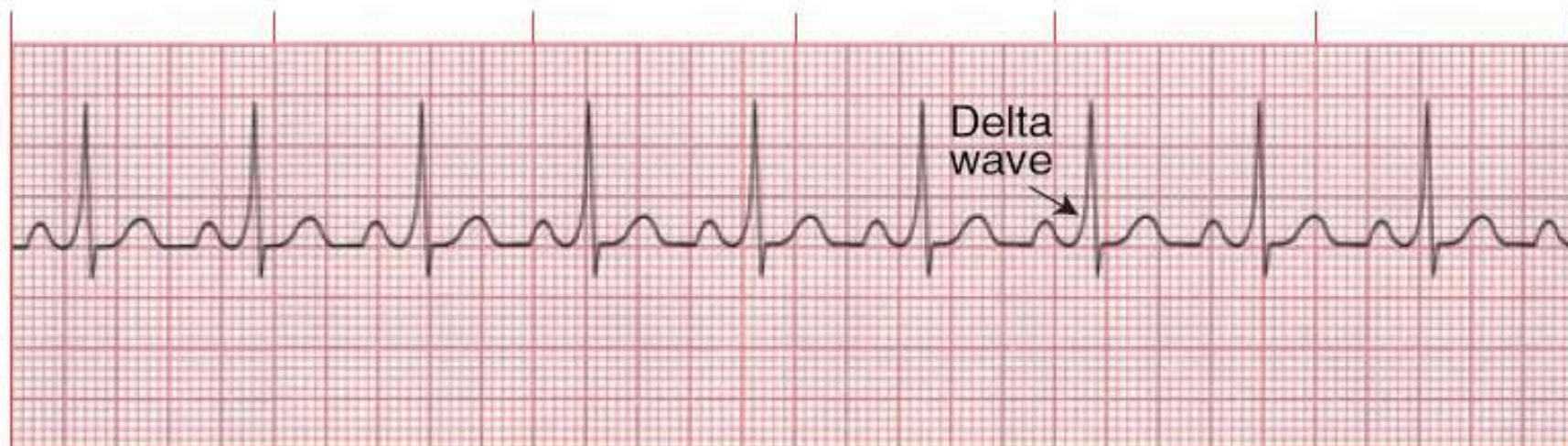
**QRS:** Normal (0.06–0.10 sec) but may be wide if abnormally conducted through ventricles

♥ **Clinical Tip:** The patient may feel palpitations, dizziness, lightheadedness, or anxiety.



## Wolff-Parkinson-White (WPW) Syndrome

- In WPW an accessory conduction pathway is present between the atria and the ventricles. Electrical impulses are rapidly conducted to the ventricles.
- These rapid impulses create a slurring of the initial portion of the QRS called the delta wave.



**Rate:** Depends on rate of underlying rhythm

**Rhythm:** Regular unless associated with A-fib

**P Waves:** Normal (upright and uniform) unless A-fib is present

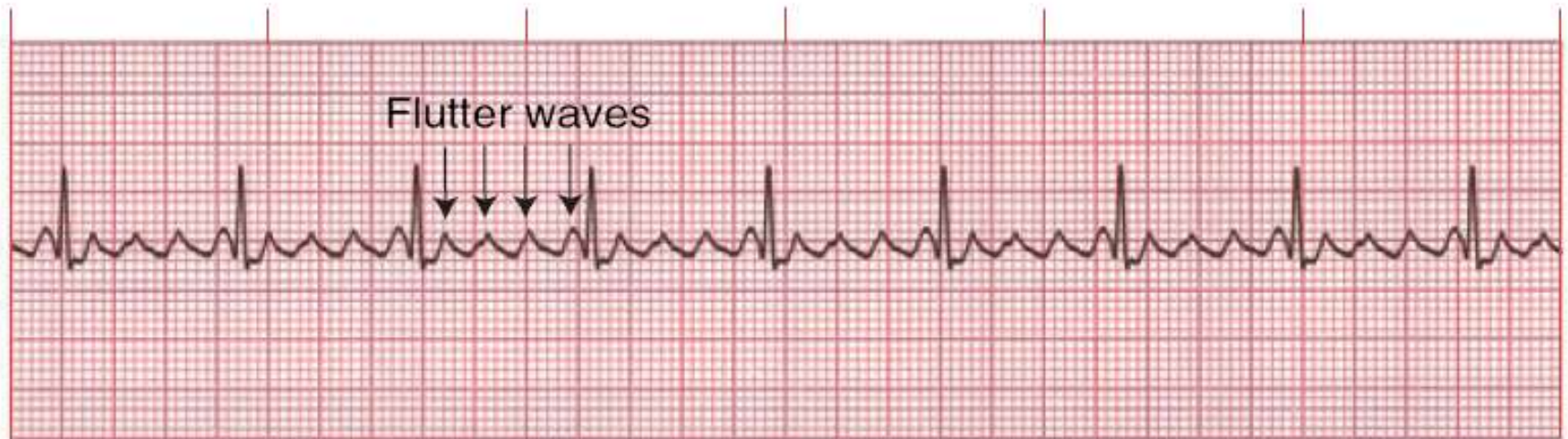
**PR Interval:** Short ( $<0.12$  sec) if P wave is present

**QRS:** Wide ( $>0.10$  sec); delta wave present

♥ **Clinical Tip:** WPW is associated with narrow-complex tachycardias, including A-flutter and A-fib.

## Atrial Flutter (A-flutter)

- AV node conducts impulses to the ventricles at a 2:1, 3:1, 4:1, or greater ratio (rarely 1:1).
- Degree of AV block may be consistent or variable.



**Rate:** Atrial: 250–350 bpm; ventricular: slow or fast

**Rhythm:** Usually regular but may be variable

**P Waves:** Flutter waves have a saw-toothed appearance

**PR Interval:** Variable

**QRS:** Usually normal (0.06–0.10 sec), but may appear widened if flutter waves are buried in QRS

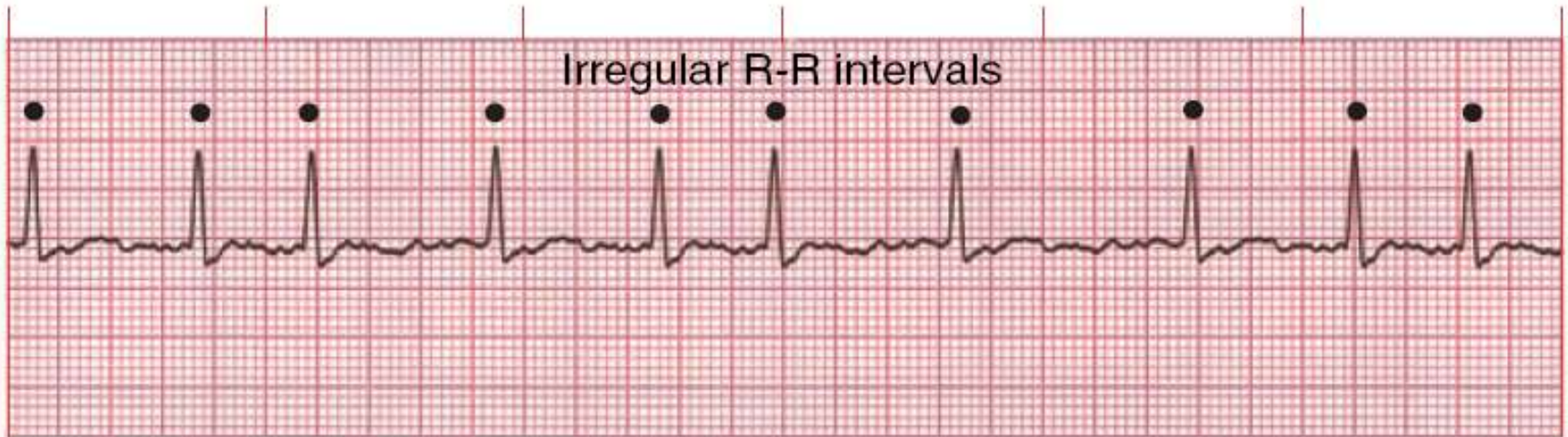
♥ **Clinical Tip:** The presence of A-flutter may be the first indication of cardiac disease.

♥ **Clinical Tip:** Signs and symptoms depend on ventricular response rate.



## Atrial Fibrillation (A-fib)

- Rapid, erratic electrical discharge comes from multiple atrial ectopic foci.
- No organized atrial contractions are detectable.



**Rate:** Atrial: 350 bpm or greater; ventricular: slow or fast

**Rhythm:** Irregular

**P Waves:** No true P waves; chaotic atrial activity

**PR Interval:** None

**QRS:** Normal (0.06–0.10 sec)

- ♥ **Clinical Tip:** A-fib is usually a chronic arrhythmia associated with underlying heart disease.
- ♥ **Clinical Tip:** Signs and symptoms depend on ventricular response rate.

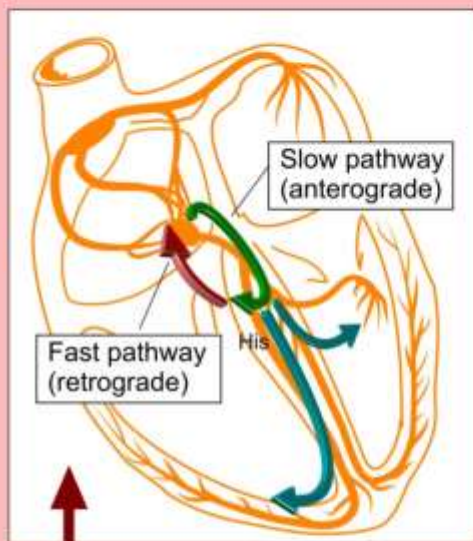


# Junctional Arrhythmias

- The atria and SA node lose their pacemaking functions
- A junctional escape rhythm begins

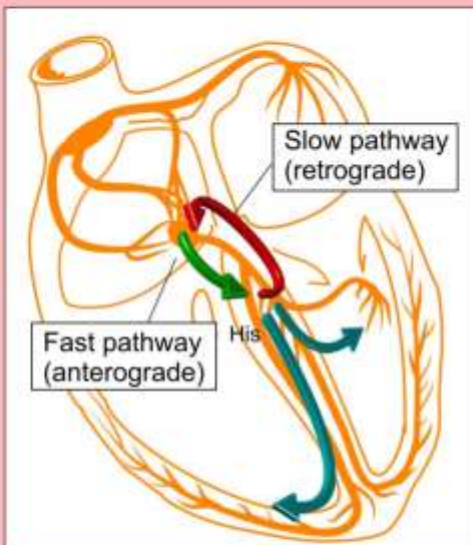


**Typical AVNRT**  
(slow-fast)  
90-95% cases

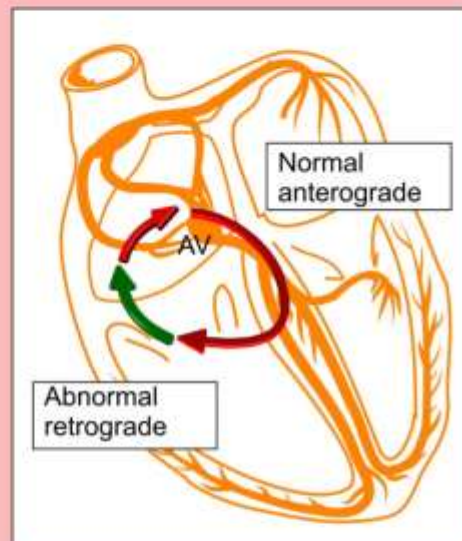


*Slow pathway* – slow conductance, short refract-erity, Localized: inferiorly and posteriorly to com-pact AV- nodal tissue running along the edge of tricuspidal anulus close to the sinus coronarius  
*Fast pathway* – fast conductance, long refrac-terity, Localized: close to the apex of Koch triangle

**Atypical AVNRT**  
(fast-slow)  
5-10% cases



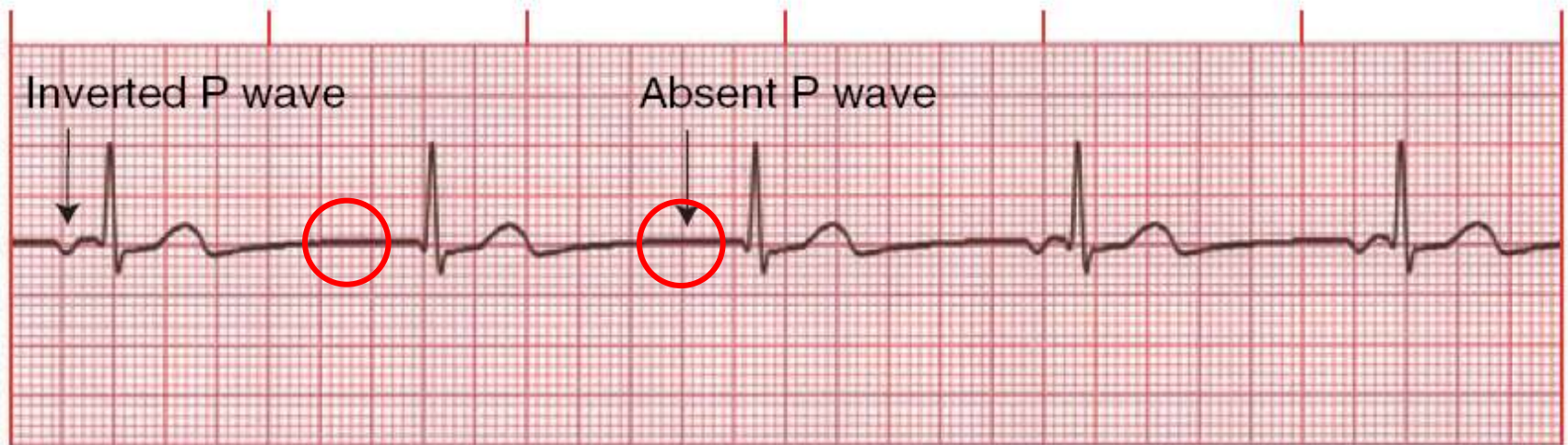
**Ortodromic AVRT**



*antegrade limb* - impulse propagates in the usual fashion ;  
*retrograde limb* - an abnormal accessory pathway reexiting atrium



# Junctional Rhythm



**Rate:** 40–60 bpm

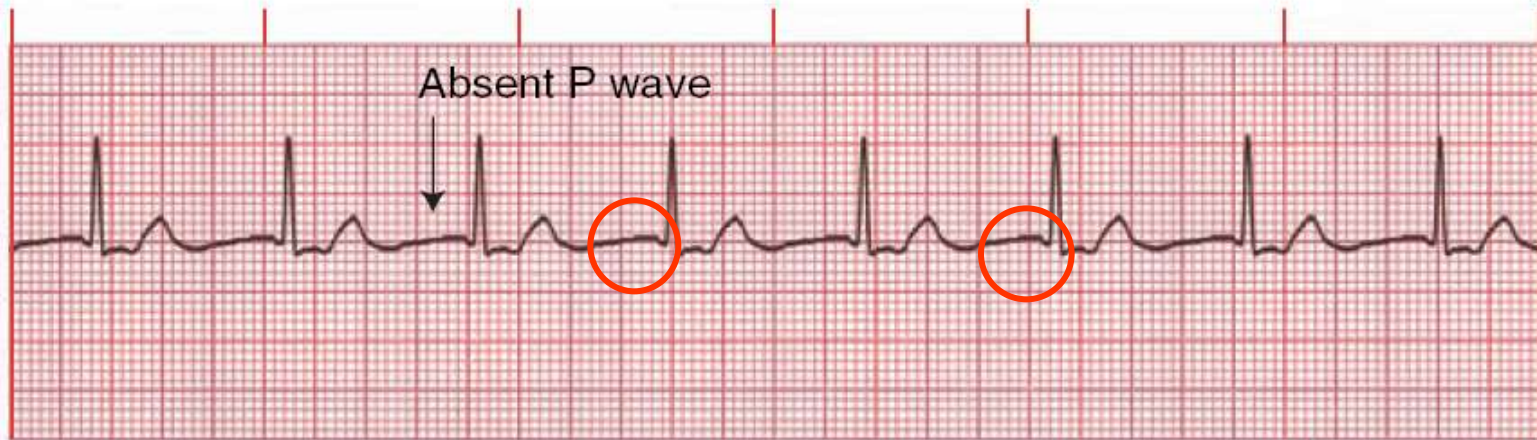
**Rhythm:** Regular

**P Waves:** Absent, inverted, buried, or retrograde

**PR Interval:** None, short, or retrograde

**QRS:** Normal (0.06–0.10 sec)

# Accelerated Junctional Rhythm



**Rate:** 61–100 bpm

**Rhythm:** Regular

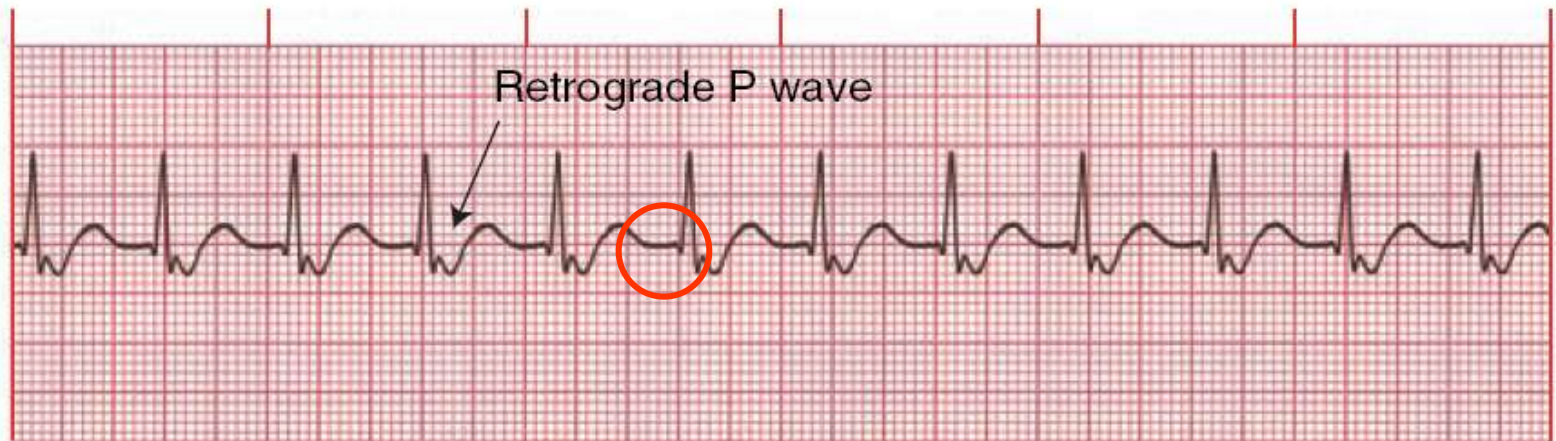
**P Waves:** Absent, inverted, buried, or retrograde

**PR Interval:** None, short, or retrograde

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Monitor the patient, not just the ECG, for clinical improvement.

# Junctional Tachycardia



**Rate:** 101–180 bpm

**Rhythm:** Regular

**P Waves:** Absent, inverted, buried, or retrograde

**PR Interval:** None, short, or retrograde

**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Signs and symptoms of decreased cardiac output may be seen in response to the rapid rate.



# Junctional Escape Beat

- An escape complex comes later than the next expected sinus complex.



**Rate:** Depends on rate of underlying rhythm

**Rhythm:** Irregular whenever an escape beat occurs

**P Waves:** None, inverted, buried, or retrograde in the escape beat

**PR Interval:** None, short, or retrograde

**QRS:** Normal (0.06–0.10 sec)

# Premature Junctional Contractions (PJC)

- Enhanced automaticity in the AV junction produces PJCs.



**Rate:** Depends on rate of underlying rhythm

**Rhythm:** Irregular whenever a PJC occurs

**P Waves:** Absent, inverted, buried, or retrograde in the PJC

**PR Interval:** None, short, or retrograde

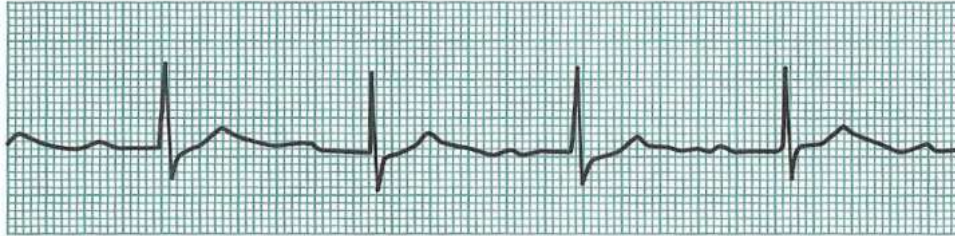
**QRS:** Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Before deciding that isolated PJCs may be insignificant, consider the cause.



# Atroventricular blockade

## AV block 1rd degree



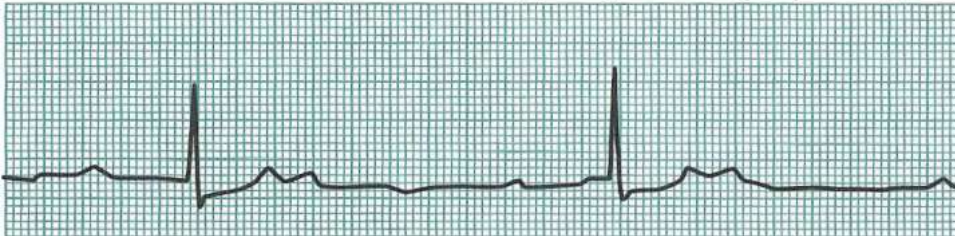
**ECG:** lengthening of PR > 0.2 s; HR regular, slower  
**SY:** asymptomatic,  
**E:** physiologic block in tachyarrhythmias

## AV block 2nd degree (A. Mobitz type 1, Wenckebach's phenomenon)



**ECG:** progressive  $\leftrightarrow$  PR with each beat until one beat is totally blocked  
**SY:** well tolerated, asymptomatic  
**E:** common; block is high in AV-junction; ischemia physiologic block in tachyarrhythmias

## AV block 2nd degree (B. Mobitz type 2) - 1. degree, 2.degree



**ECG:** no cycle, intermittently dropped beats (uniform  $\leftrightarrow$  PR); often QRS malformation due to bundle branch block  
**2. degree** block of 2-3 consecutive P waves  
**SY:** congestive heart failure if ventricular rhythm is slow in ischemic myocardium,  
**E:** less common; block is low in AV-junction, often in excessive myocardial damage  
block of 2-3 consecutive P waves

## AV block 3rd degree (AV - dissociation)



**SY:**  
a) sudden (in MI) - occurs unless AV-node or Ven-pacemaker start to pace  
b) gradual occurrence - most common - if latent pacemaker is weak

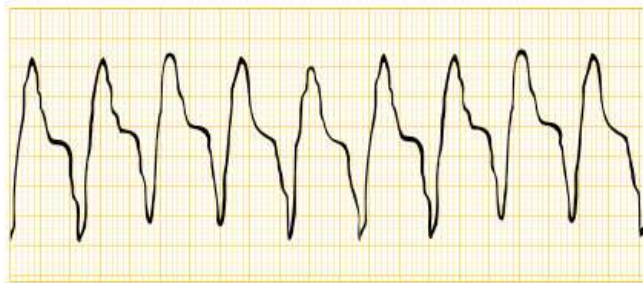




# Ventricular Arrhythmias

- The atria and SA node lose their pacemaking functions
- Ventricular loci drive the rhythm

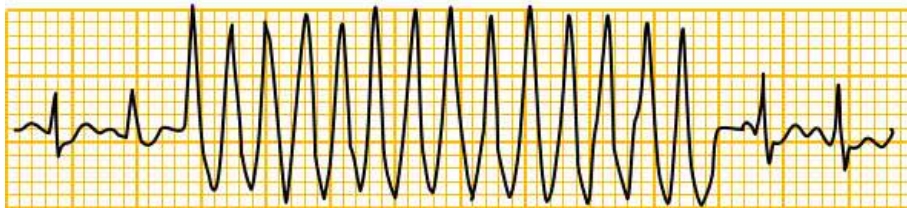
## Idioventricular rhythm



## Ventricular tachycardia (ectopic beats)

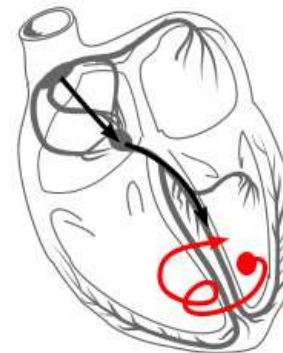
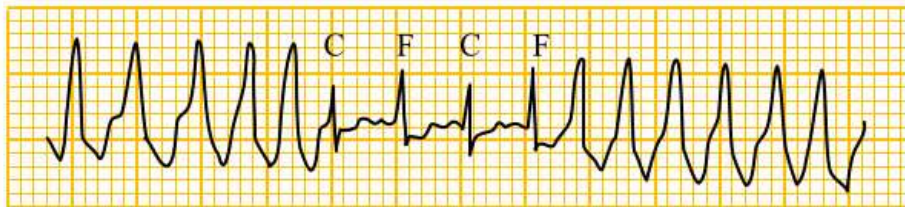


## Ventricular tachycardia (paroxysmal)



regular, 100-250/min, wide & bizarre QRS, atria beat independently (AV-dissociation), often sudden onset and termination

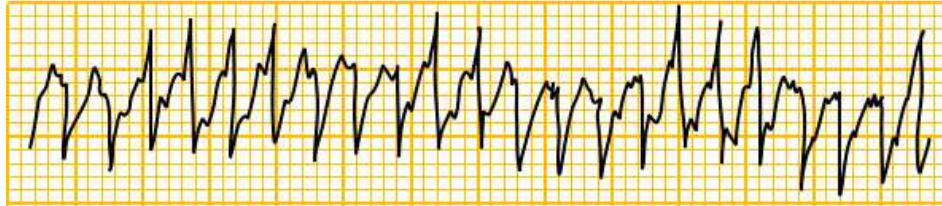
## Ventricular tachycardia with capture and fusion beats



slightly irregular, 130/min, wide & bizarre QRS, AV-dissociation), *capture beat* sinus beat between ventricular beats (SA captured by SA node, *fusion beat* - simultaneous activation of ventricles from SA node and ectopic source)

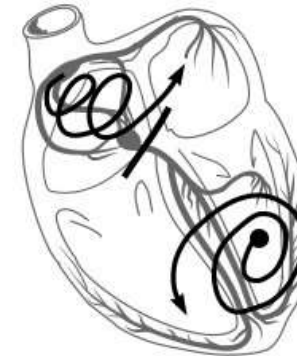
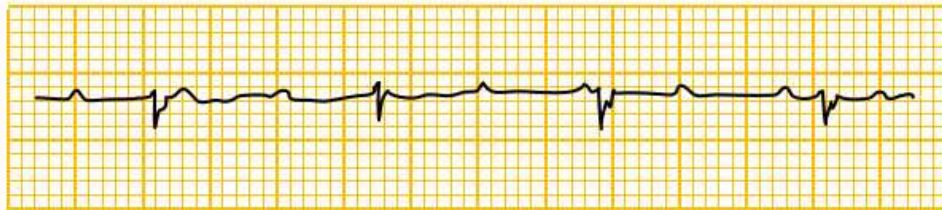


## Torsade de pointes (polymorphous tachycardia)



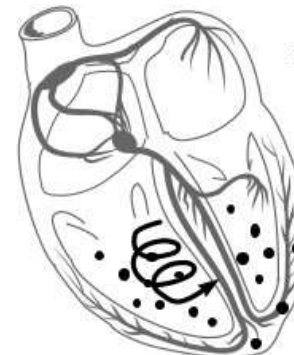
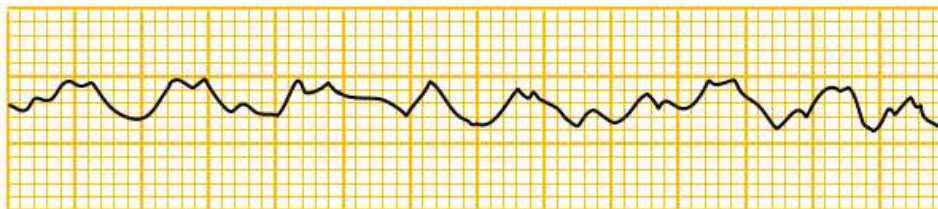
irregular, 200-250 /min,  
progressive changes in  
amplitude and polarity of  
QRS  
"twisting around  
isoelectric line", occurs in  
patients with impaired  
ventricular  
repolarisation

## Ventricular escape beats and rhythms



irregular, 35-40 /min, wide  
bizzare QRS complexes

## Ventricular fibrillation



irregular, circulatory failure




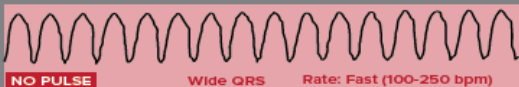




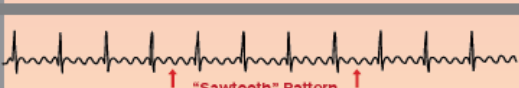

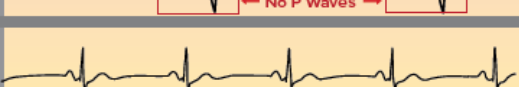

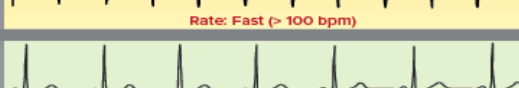
# Sources

■ <http://www.medicalestudy.com/>



## 11 Rhythms Nurses Need to Know

### Basic EKG/ECG Rhythms

Common & Formal Rhythm Names	6 Second Rhythm Strip	Identifiers
<b>S</b> <b>H</b> <b>O</b> <b>C</b> <b>K</b> <b>A</b> <b>B</b> <b>L</b> <b>E</b>	<b>V-Fib</b> Ventricular Fibrillation  <p>NO PULSE      Rate: Unmeasurable</p>	Irregular, No P Wave, No QRS
	<b>V-Tach</b> Ventricular Tachycardia  <p>NO PULSE      Wide QRS      Rate: Fast (100-250 bpm)</p>	Regular, No P Wave, Wide QRS
	<b>Torsade de Pointes</b> Type Of Ventricular Tachycardia  <p>NO PULSE      Rate: Very Fast (200-250 bpm)      Tall and Short Waves</p>	Irregular, No P Wave, Wide QRS
*Synchronized Cardioversion possible for SVT if medication ineffective.		
<b>SVT*</b> Supraventricular Tachycardia  <p>Rate: Very Fast (150-250 bpm)</p>	Regular, P Wave Hidden, Normal QRS	
<b>STEMI</b> ST Elevation Myocardial Infarction  <p>↑      ST Elevation      ↑</p>	Reg or Irreg, P Wave, ST Elevated	
<b>A-Fib</b> Atrial Fibrillation  <p>↑ Erratic Waves      ↑ * QRS normally narrow but not always</p>	Irregular, No P Wave, Normal QRS*	
<b>A-Flutter</b> Atrial Flutter  <p>↑ "Sawtooth" Pattern      ↑</p>	Reg or Irreg, No P Wave, Normal QRS	
<b>PVC</b> Premature Ventricular Contraction  <p>PVC      PVC ← No P Waves →</p>	Irregular, No P Wave, Wide QRS	
<b>Sinus Brady</b> Sinus Bradycardia  <p>Rate: Slow (&lt;60 bpm)</p>	Regular, P Wave, Normal QRS	
<b>Sinus Tach</b> Sinus Tachycardia  <p>Rate: Fast (&gt; 100 bpm)</p>	Regular, P Wave, Normal QRS	
<b>NSR</b> Normal Sinus Rhythm  <p>Rate: Normal (60-100 bpm)</p>	Regular, P Wave, Normal QRS	

## Cardiac channelopathies

- The atria and SA node loss their pacemaking functions
- Ventricular loci drive the rhythm



# Cardiac channelopathies

- **Def.:** Disorders caused by spontaneous or hereditary mutations of genes coding subunits of ionic channels or transporters involved in creation of cardiac electrical excitation or conduction or electro-mechanical coupling in cardiomyocytes
- Channels are multimeric proteins, where each subunit is encoded by various genes in different locuses..
- **Inherited forms of cardiac channelopathies**
- **Acquired forms of cardiac channelopathies**

## Inherited forms of cardiac channelopathies

- Long QT syndrome (LQTS)
- Short QT Syndrome (SQTs)
- Brugada brothers syndrome (BrS)
- Catecholaminergic polymorphic ventricular tachycardia (CPVT)
- Arrhythmogenic right-sided ventricular cardiomyopathy (ARVC)
- Familial forms of atrial fibrillation

## Acquired forms of cardiac channelopathies

- Atrial fibrillation
- Heart failure
- Sick sinus syndrome
- Cardiac hypertrophy
- myocardial infarction



# Cardiac channelopathies

## Inherited cardiac channelopathies

Condition	Mutation	Channel or protein affected	Effects of mutations	Relative frequency
Long QT syndrome				
LQT1	<i>KCNQ1</i>	KvLQT1	↓ I <sub>Ks</sub>	+++
LQT2	<i>KCNH2</i>	hERG	↓ I <sub>Kr</sub>	++
LQT3	<i>SCN5A</i>	Na <sup>+</sup> channel	↑ I <sub>Na</sub> with noninactivating Na <sup>+</sup> currents	+
LQT4	<i>ANK2</i>	Ankyrin B		Rare
LQT5	<i>KCNE1</i>	MinK	↓ I <sub>Ks</sub>	Rare
LQT6	<i>KCNE2</i>	MiERP1	↓ I <sub>Kr</sub>	Rare
LQT7 (Andersen syndrome)	<i>KCNJ2</i>	Kir2.1	↓ I <sub>K1</sub>	Rare
LQT8 (Timothy syndrome)	<i>CACNA1C</i>	Cav1.2	↑ I <sub>Ca,L</sub>	Rare
Short QT syndrome				
	<i>KCNH2</i>	hERG	↑ I <sub>Kr</sub>	Rare
	<i>KCNQ1</i>	KvLQT1	↑ I <sub>Ks</sub>	Rare
	<i>KCNJ2</i>	Kir2.1	↑ I <sub>K1</sub>	Rare
Brugada syndrome	<i>SCN5A</i>	Na <sup>+</sup> channel	↓ I <sub>Na</sub>	+
Catecholaminergic polymorphic VT	<i>RyR2</i>	Ryanodine receptor	↑ Abnormal Ca <sup>2+</sup> release from SR	Rare
	<i>CASQ2</i>	Calsequestrin		Rare
Familial AF				
	<i>KCNQ1</i>	KvLQT1	↑ I <sub>Ks</sub>	Rare
	<i>KCNE2</i>	MiRP1	↑ I <sub>Ks</sub>	Rare
	<i>KCNJ2</i>	Kir2.1	↑ I <sub>K1</sub>	Rare
	<i>SCN5A</i>	Na <sup>+</sup> channel	↓ I <sub>Na</sub>	Rare
Conduction disease				
	<i>SCN5A</i>	Na <sup>+</sup> channel	↓ I <sub>Na</sub>	Rare
Sinus node dysfunction				
	<i>SCN5A</i>	Na <sup>+</sup> channel	↓ I <sub>Na</sub>	Rare
	<i>HCN4</i>	Pacemaker channel	↓ I <sub>f</sub>	Rare

# Cardiac channelopathies

## Acquired cardiac channelopathies

Condition	Expression of channel or protein	Electrical activity	Effect
Atrial fibrillation	<ul style="list-style-type: none"> <li>↓ <math>I_{Ca,L}</math></li> <li>↓ <math>I_{to}</math></li> <li>↑ <math>I_{K1}</math></li> <li>↑ <math>I_{KACH}</math></li> <li>↓ Connexin 40</li> </ul>	<ul style="list-style-type: none"> <li>↓ AERP</li> <li>↑ AERP</li> <li>↓ AERP</li> <li>↓ AERP</li> <li>↓ Conduction</li> </ul>	Overall net effect: ↓ wave-length of tachycardia & loss of rate adaptation
Heart failure	<ul style="list-style-type: none"> <li>↓ <math>I_{to}</math></li> <li>↓ <math>I_{Kr}</math></li> <li>↓ <math>I_{Ks}</math></li> <li>↓ <math>I_{K1}</math></li> <li>↑ <math>Na^+/Ca^{2+}</math> exchange</li> <li>↑ <math>I_{Ca,L}</math> inactivation</li> <li>Connexin-43 redistribution</li> </ul>	<ul style="list-style-type: none"> <li>↑ VERP</li> <li>↑ VERP</li> <li>↑ VERP</li> <li>↑ VERP</li> </ul>	<ul style="list-style-type: none"> <li>↓ Rate adaptation</li> <li>↑ APD</li> <li>↑ EAD</li> <li>↑ Automaticity</li> <li>↑ DAD</li> <li>↓ APD</li> </ul>
Cardiac hypertrophy	<ul style="list-style-type: none"> <li>↑ <math>I_{Ca,L}</math></li> <li>↓ <math>Na^+/Ca^{2+}</math> exchange</li> </ul>	<ul style="list-style-type: none"> <li>↓ Conduction</li> <li>↑ APD</li> <li><math>Ca^{2+}</math> overload</li> </ul>	<ul style="list-style-type: none"> <li>↑ Reentry</li> <li>↑ EAD</li> <li>↑ DAD</li> </ul>
Myocardial infarction	<ul style="list-style-type: none"> <li>↓ Connexin 43</li> <li>↓ <math>I_{Na}</math></li> <li>↓ <math>I_{to}</math></li> <li>↓ <math>I_{Kr}</math></li> <li>↓ <math>I_{Ks}</math></li> <li>↓ <math>I_{Ca,L}</math></li> </ul>	<ul style="list-style-type: none"> <li>↓ Conduction</li> <li>↓ Conduction</li> <li>↑ VERP</li> <li>↑ VERP</li> <li>↑ VERP</li> <li>↓ Plateau</li> </ul>	<ul style="list-style-type: none"> <li>↑ Reentry</li> </ul>

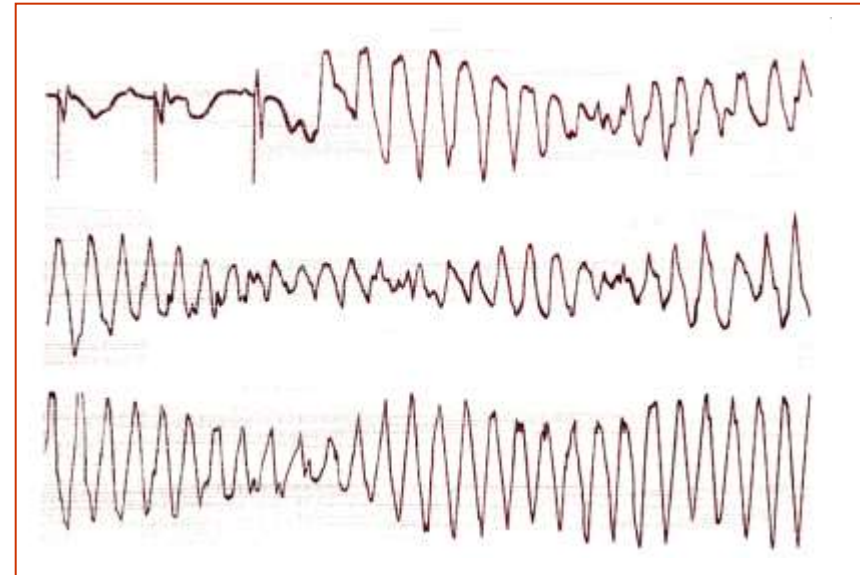
AERP, atrial effective refractory period; APD, action potential duration; Ca, calcium; DAD, delayed after depolarization; EAD, early afterdepolarization;  $I_{Ca,L}$ , L-type calcium current;  $I_{K1}$ , strong inward rectifier potassium current;  $I_{KACH}$ , acetylcholine-sensitive potassium current;  $I_{Kr}$ , rapid component of the delayed rectifier potassium current;  $I_{Ks}$ , slow component of the delayed rectifier potassium current;  $I_{Na}$ , sodium current;  $I_{to}$ , transient outward potassium current; VERP, ventricular effective

## Long QT interval syndrome (LQTS)

- **Def:** Group of cardiac channelopathies typical by prolongation of repolarisation in cardiomyocytes **due to gene defects in mostly potassium, sodium, or calcium channels**
- QT interval (start of Q till end of T) shows interpersonal and intrapersonal variability => corrected QT (QTc) 0.35 to 0.46 sec. ; 95% percentil = 0.38 to 0.44 s
- **Etio:** 14 subtypes of disease exist (LQT1, LQT2, & LQT3 ~ 80-90% of known cases)
  - KCNQ1, KCNH2, & SCN5A cardiac K<sup>+</sup> a Na<sup>+</sup> channels; loss-of-function mutations
  - polymorfisms (postmedical LQTS)
- **Occ:** common 1/2000 - 1/3000 of cardiac patients

### ■ **Clinical manifestations:**

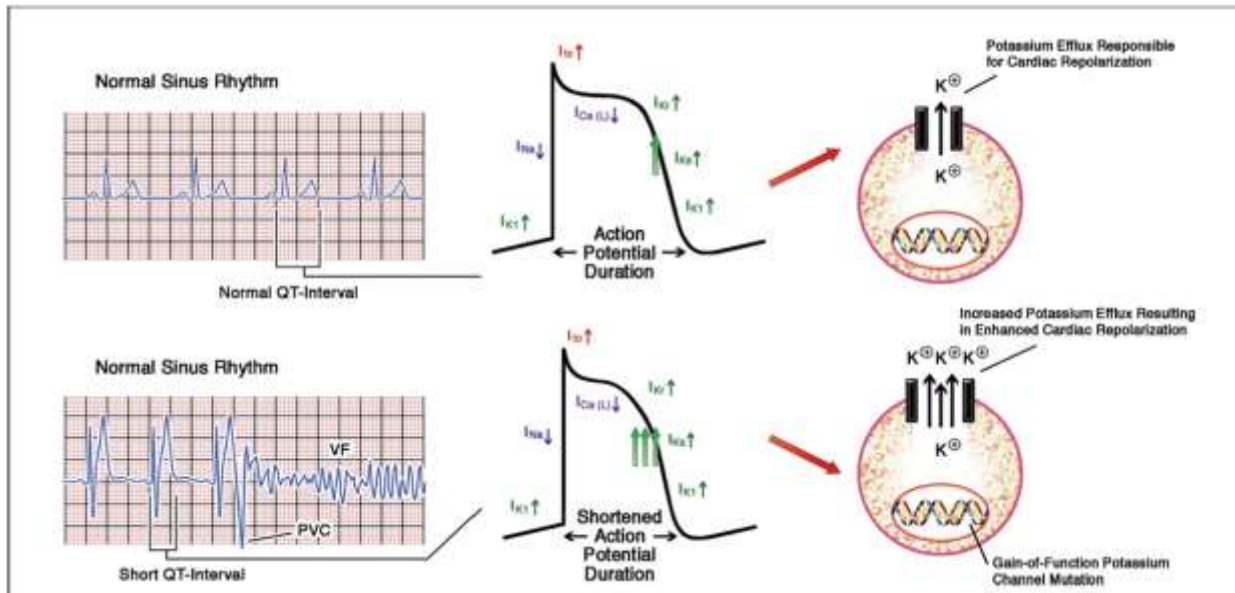
- palpitations, fainting, sudden death due to ventricular fibrillation
- risk of episodes of torsades de pointes (polymorphic ventricular tachyarrhythmias)
- induction by **hypokalemia**, heart attack and heart ischemia, hypothermia, subarachnoidal bleeding etc.





## Short QT interval syndrome (LQTS)

	Gene	Current	Phenotype
SQT1	KCNH2	IKr	
SQT2	KCNQ1	IKs	
SQT3	KCNJ2	IK1	
SQT4	CACNA1C	ICaL	
SQT5	CACNB2B	ICaL	



## Brugada sy. (BRGDA)

- **Def.:** Group of hereditary arrhythmias leading to sudden unexpected death (ventricular fibrillation) (Pedro and Joseph Brugada, 1992)
- One of reasons of unexplained cardiac death (**sudden unexplained death syndrome, SUDS**); most common reason of death in young man without previous cardiac disease in Thailand and Laos
- **Etiology:**
  - (a) **Na<sup>+</sup> channel in cardiomyocytes (SCN5A)** 20% cases ; 160 types of mutations
  - (b) **Ca<sup>2+</sup> channels L-type (CACNA1C and CACNB2)** leading to elevation of ST and shortening of QT (<360 ms).

Type	Gene	Locus
BS1	<u>SCN5A</u>	3p22.2
BS2	<u>GPD1L</u>	3p22.3
BS3	<u>CACNA1C</u>	12p13.33
BS4	<u>CACNB2</u>	10p12.33- p12.31
BS5	<u>SCN1B</u>	19q13.12
BS6	<u>KCNE3</u>	11q13.4
BS7	<u>SCN10A</u>	3p22.2
BS8	<u>HEY2</u>	6q22.31

# Cardiac channelopathies

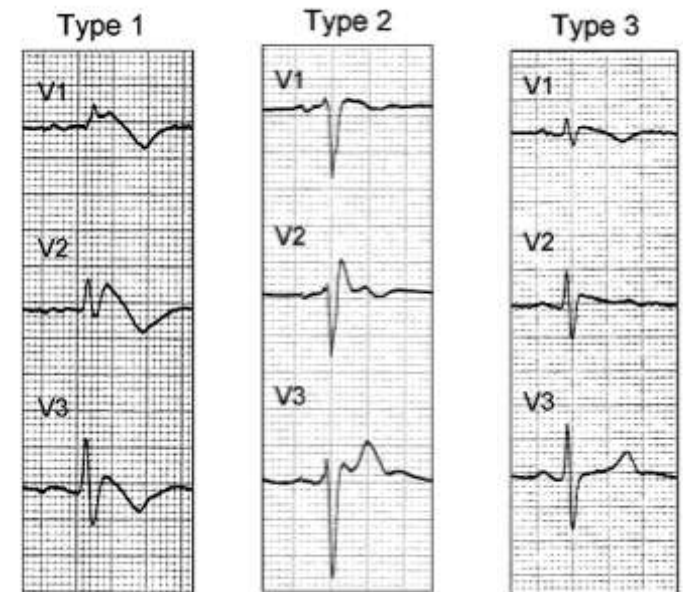
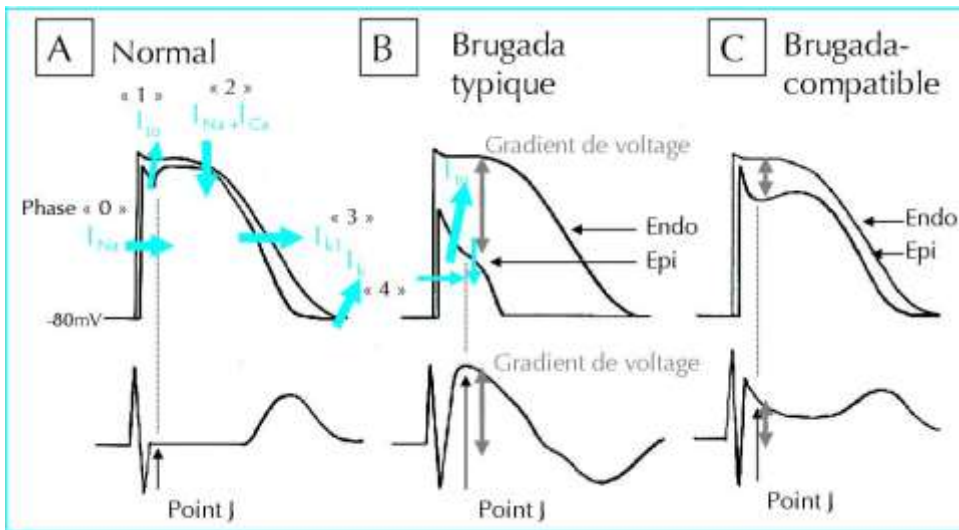
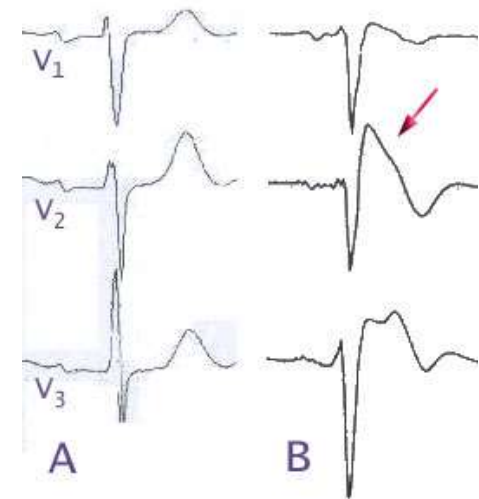
**Dg.** typical spontaneous changes in ECG or induced by antiarrhythmic drugs blocking Na<sup>+</sup> channels in 3 various ECG patterns:

**Type 1:** ST elevation >2 mm (0.2 mV), J-point elevation; decrease of ST segment with negative T-wave.

**Type 2:** > 2 mm elevation of J-point and > elevation 1 mm ST with positive biphasic T-wave

**Type 3:** similar to type 1 or type 2 with elevation of J point < 2 mm + ST elevation < 1 mm.

[http://upload.wikimedia.org/wikipedia/commons/0/05/Brugada\\_EKG\\_Schema.jpg](http://upload.wikimedia.org/wikipedia/commons/0/05/Brugada_EKG_Schema.jpg)





# Cardiac channelopathies

## Examples of allelic heterogeneity of various arrhythmias

SODIUM CHANNEL, VOLTAGE-GATED,  
TYPE V, ALPHA SUBUNIT; SCN5A  
SCN5A 3p22.2

Atrial fibrillation, familial, 10  
Brugada syndrome 1  
Cardiomyopathy, dilated, 1E  
Heart block, nonprogressive  
Heart block, progressive, type IA  
Long QT syndrome-3  
Sick sinus syndrome 1  
Ventricular fibrillation, familial, 1  
Sudden infant death syndrome

POTASSIUM CHANNEL,  
VOLTAGE-GATED, SHAKER FAMILY,  
MEMBER 9; KCNA9 KVLQT1  
11p15.5-p15.4

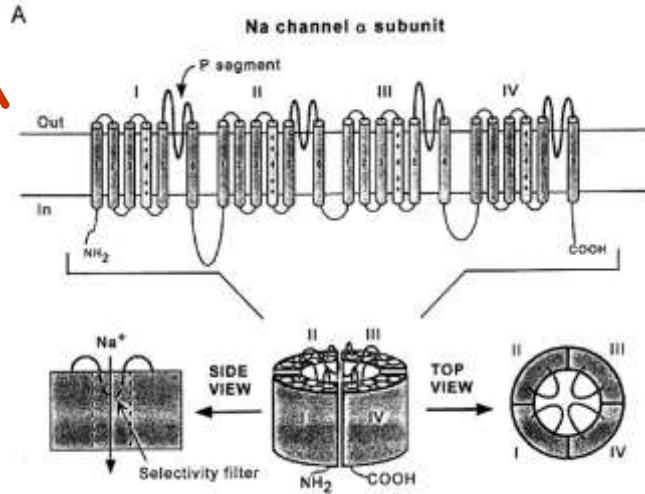
Atrial fibrillation, familial, 3  
Jervell and Lange-Nielsen syndrome  
Long QT syndrome-1  
Short QT syndrome-2

POTASSIUM CHANNEL, VOLTAGE  
GATED, SUBFAMILY H, MEMBER 2;  
KCNH2 7q36.1

Long QT syndrome-2  
Short QT syndrome-1

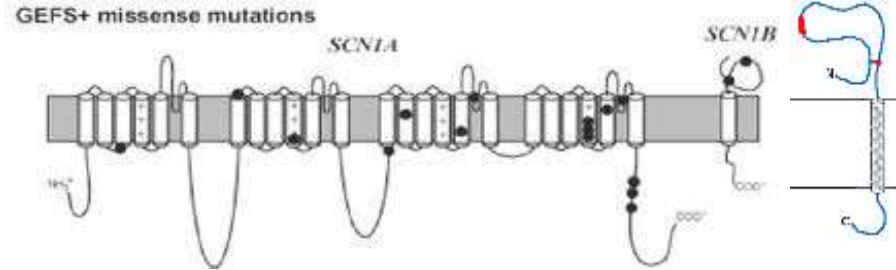
# Cardiac channelopathies

## Alfa- subunite of cardiac Na<sup>+</sup> channel (SCN5A) Ch3p22.2



- Long QT syndrome-3
- Cardiomyopathy, dilated, 1E
- Atrial fibrillation, familial,
- Brugada syndrome 1
- Heart block, nonprogressive
- Heart block, progressive, type IA
- Sick sinus syndrome 1
- Ventricular fibrillation, familial, 1
- Sudden infant death syndrome, susceptibility

## Alpha subunite of cardiac Na<sup>+</sup> channel SCN1B 19q13.12



- Atrial fibrillation, familial, 13
- Brugada syndrome 5
- Cardiac conduction defect, nonspecific
- Epilepsy, generalized, with febrile seizures plus, type 1