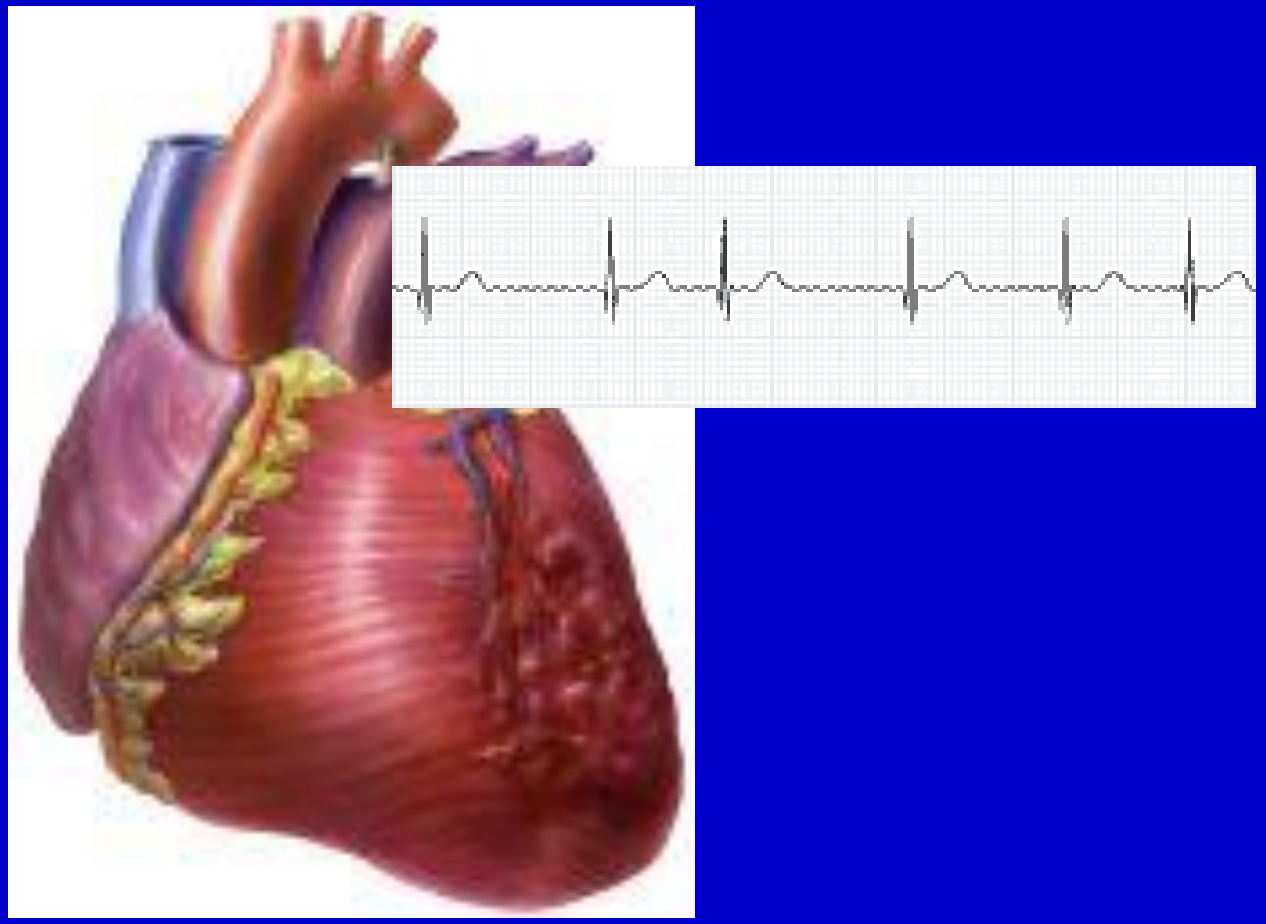


ARRHYTMIAS

Department of Pathological Physiology

Martin Vokurka
mvoku@lf1.cuni.cz

Arrhytmia

- Disturbance of heart rhytm:
  heart rate
  regularity

CASE REPORTS

A) The patient feels irregularities in heart beat (palpitations), sometimes faster, sometimes slower. At times he feels weak and is about fainting.

B) The patient repeatedly looses consciousness, is without puls. After a while his consciousness restores-

C) The patient suddenly looses consciousness, without puls, no breathing. Without reanimation he dies.

D) Young healthy person feels sometimes irregularities of heart beat w/o any other problem.

CASE REPORT

ALL PATIENTS HAVE DISTURBANCE OF HEART RHYTHM OF VERY DIFFERENT IMPORTANCE.

ECG and sometimes longer follow-up might be necessary (HOLTER).

Further the causes of irregularities should be searched for.

1. **to find and name the type of arrhytmia**
2. **search for its cause**
3. **to treat the arrythmia and its cause**

Heart conduction system

- **Origin of the impuls**
- **Impuls coduction**
- **hierarchy**

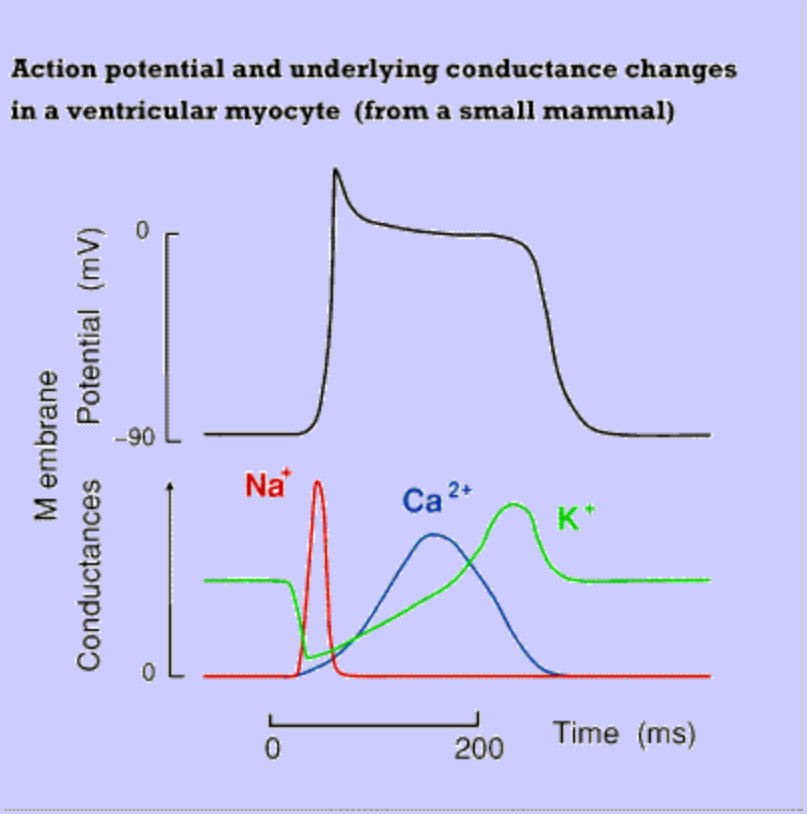
Important parameters in electrical events:

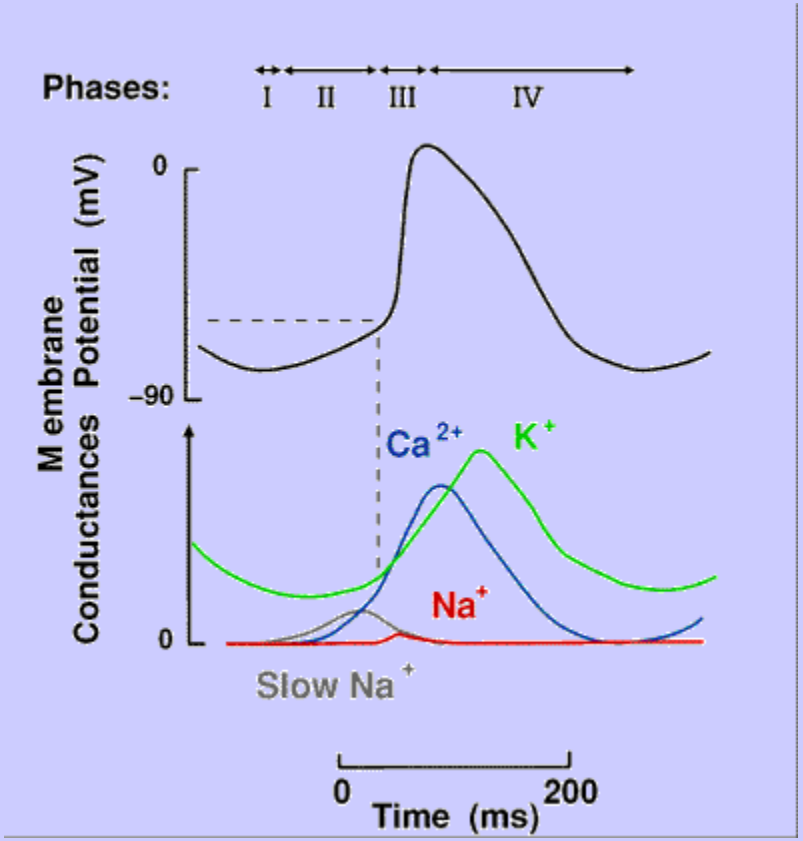
* ***excitability***: capacity of cells to respond to the stimulus of certain insenzity (by depolarization, MAP)

* ***automaticity***: capacity to produce spont. impulses
  diastolic depolarization (special phase 4 of MAP, threshold potential, influence of nerve stimulation)

* ***conductibility***: capacity to transfer impuls to the neighbouring cells
  amplitude, start of the MAP, cellular junctions, size, shape of the cells

* ***refracterity***: incapacity to excitation after previous activation (absolute, relative)





Calcium channels:

$I_{Ca\text{-}L}$: long-lasting, plateau

$I_{Ca\text{-}T}$: transient – only in pacemaker cells, diast. depolarization (+ funny current)

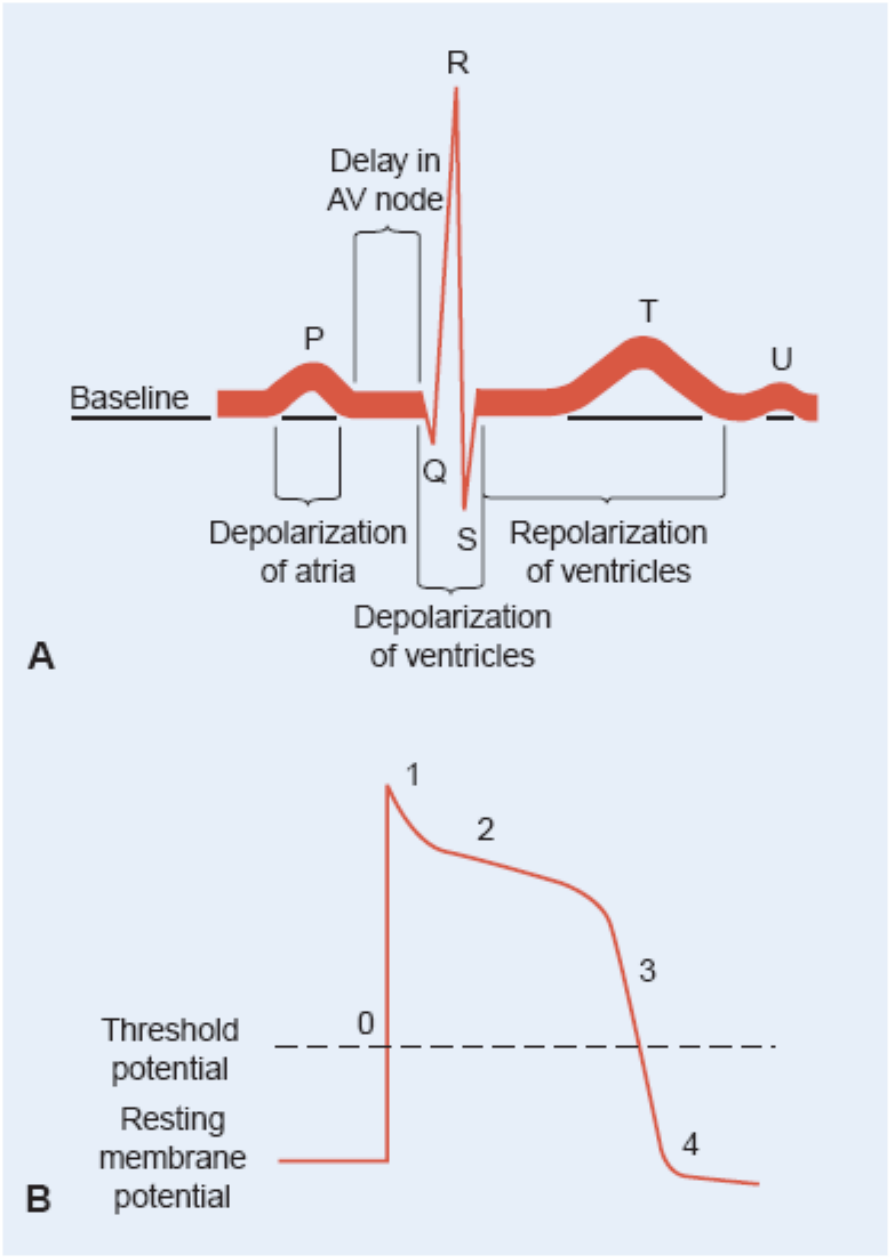
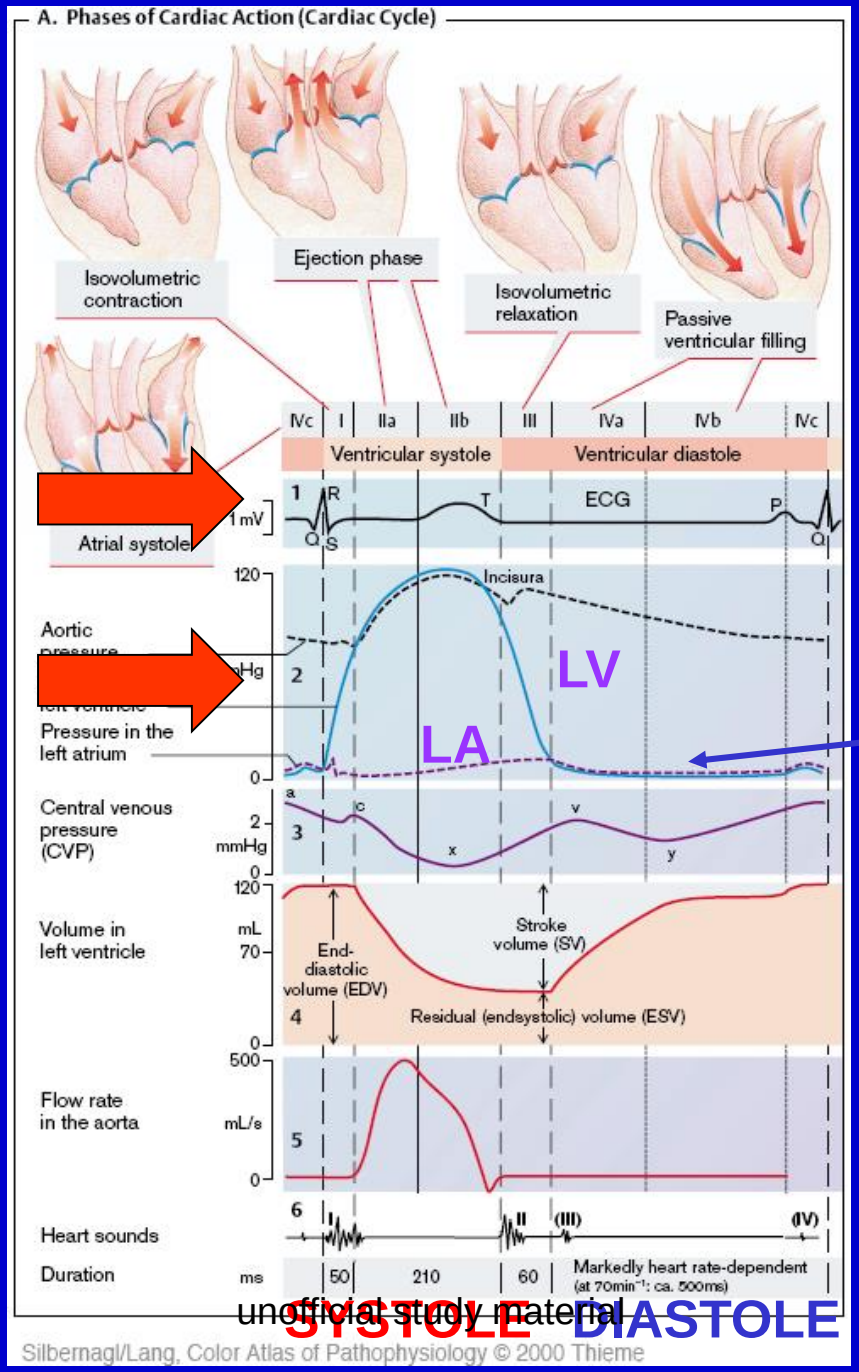


FIGURE 27-3 Relation between (**A**) the electrocardiogram and (**B**) phases of the ventricular action potential.



Ion channels

- **Sodium channel**
- **Potassium channels**
- **Calcium channels – T, L…**

Channel regulation

* voltage
* chemicals (incl. drugs)
* mechanical deformation

Ion channels pathology

- Pathological voltage
- Electrolyte concentration
- Influence of neurotransmitters (inlc. Vegetative nerves)
- Lack of energy (ATPase pumps)
- mutation (hereditary)

Main causes of disturbance of conductive systém - HEART

1. Myocardial damage

* ischemia, hypoxia, acidosis (*CHD*) + reperfusion

* *mechanical tension,hypertrophy, excessive dilatation, cardiomyopathy, fibrosis, amyloidosis, postinfarction scarring*)
– „electrical remodelation“

* inflammation (*myocarditis*)

Electrical nonhomogenity

- Focus of ischemia
- Focus of fibrotization and scarrin
- Local dilatation and/or hypertrophy

Main causes of disturbance of conductive systém - EXTRACARDIAL

2. vegetative nervous system (*compensation of heart failure, shock, but also e.g. anxiety, pain in acute MI*)

Sympathetic nerves – increase heart rate, condution, excitability and risk of arrhytmias

Parasympathetic nerves – decrease HR and conduction

Drugs influencing VNS (adrenalin, atropin, betablockers…)

Main causes of disturbance of conductive system

3. Electrolyte disturbances (*potassium, calcium*)

4. drugs, toxins (*influencing VNS, antiarrhytmic drugs, digitalis etc.*)

5. Electrical current (*trauma, endokrinopathies etc.*)

6. genetic causes (*mutation of ionic channels*)

General consequences of arrhytmias

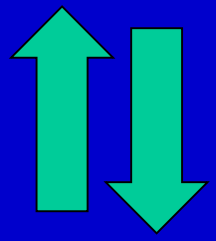
- negligent (vegetative influences)
- predictor of disease (e.g. ischemia)
- electrical instability – progression of arrhytmias
- hemodynamic consequences
- impact on myocardial perfusion and metabolism

General symptoms of arrhytmias

- electrical: ECG
- hemodynamic
- subjective

Types of arrhytmias

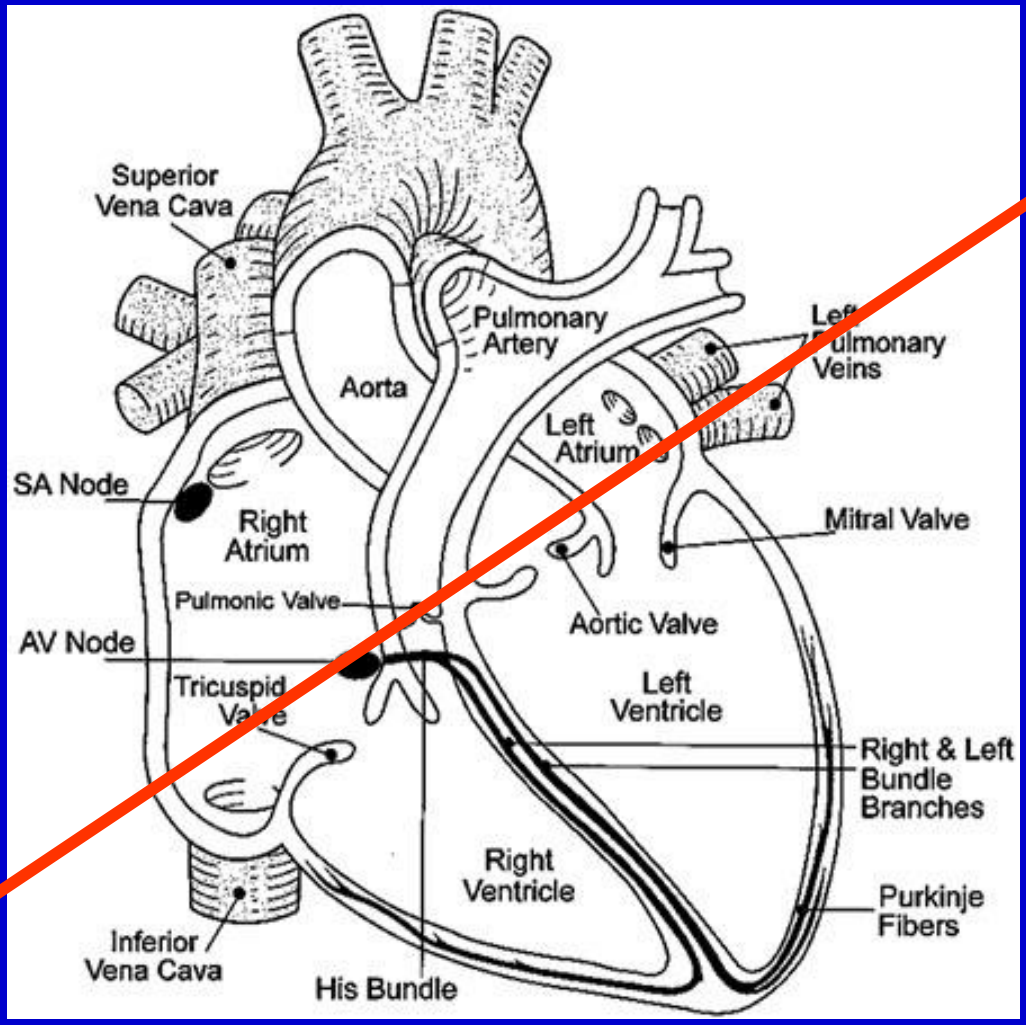
I. *electrical events*
disturbance in origin of the impuls
disturbance in conduction
combined



II. *localization* (*clinical importance !*)
supraventricular (SV) – atrial, junctional
ventricular (V)

III. *resulting heart rate* (*effect on hemodynamics, ev. therapy*)
bradyarrhytmia
tachyarrhytmia

SV



V

Ectopy

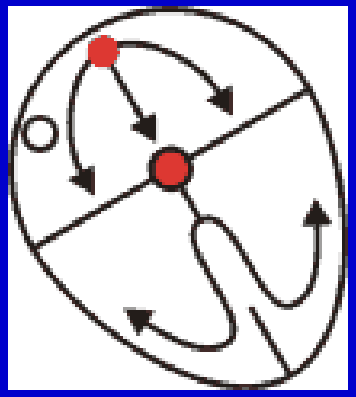
- **Area out of sinoatrial (SA) node which becomes the trigger of electrical activity**

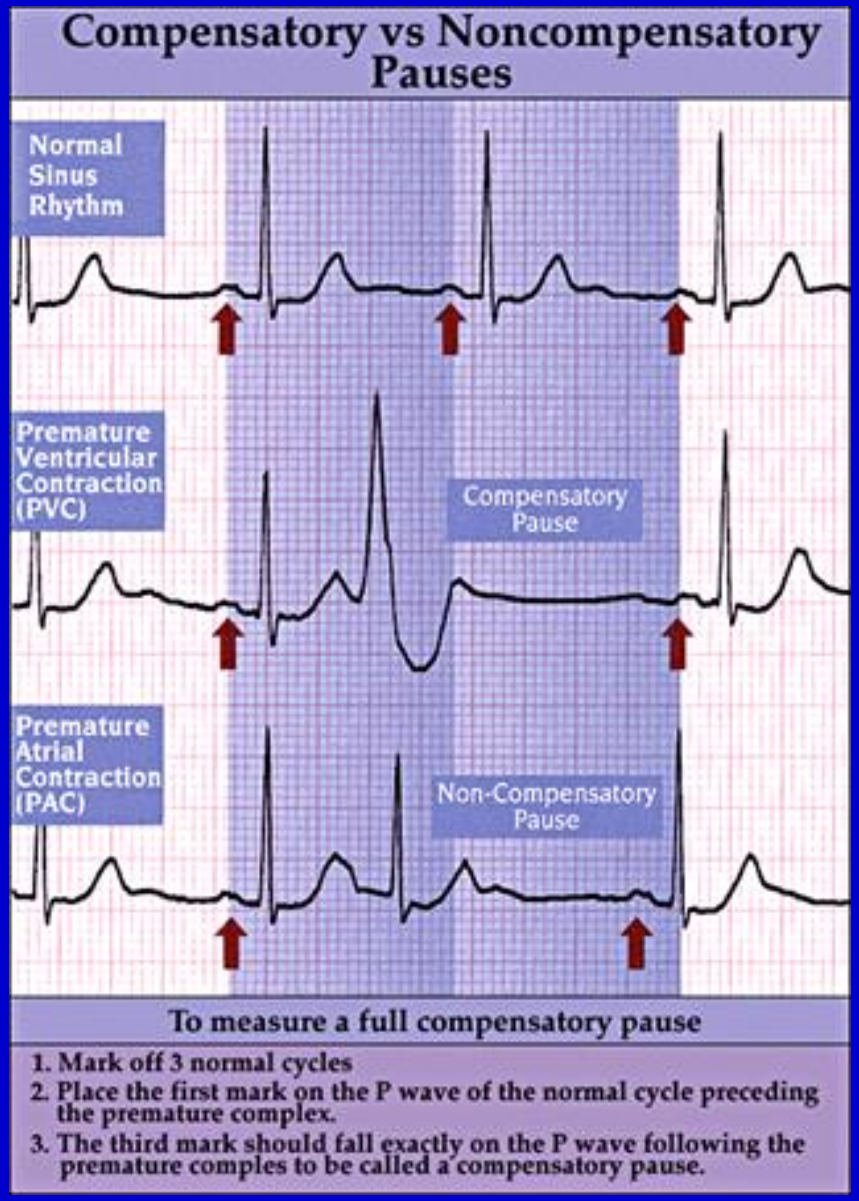
Extrasystole (premature beat, premature contraction)

- Heart beat is initiated by other parts of the heart than SA node, occurrs before the expected sinus beats

Supraventricular extrasystole (SVES)

- normal pathway to the ventricle – QRS complex has normal shape
- the impulse can spread in a retrograde way – negative P wave with aberrant PQ intervale
- retrograde spreading can discharge SA node
- new impulse in SA node follows after „normalní“ time after its discharge from retrograde spreading





VES

SVES

CASE REPORTS

A) The patient feels irregularities in heart beat (palpitations), sometimes faster, sometimes slower. At times he feels weak and is about fainting.

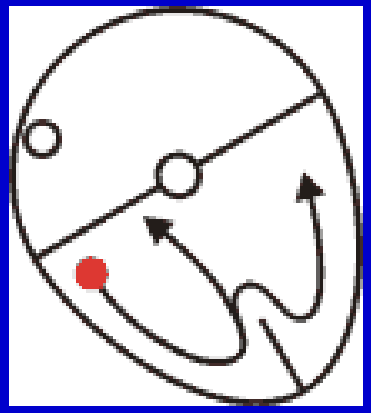
B) The patient repeatedly looses consciousness, is without puls. After a while his consciousness restores-

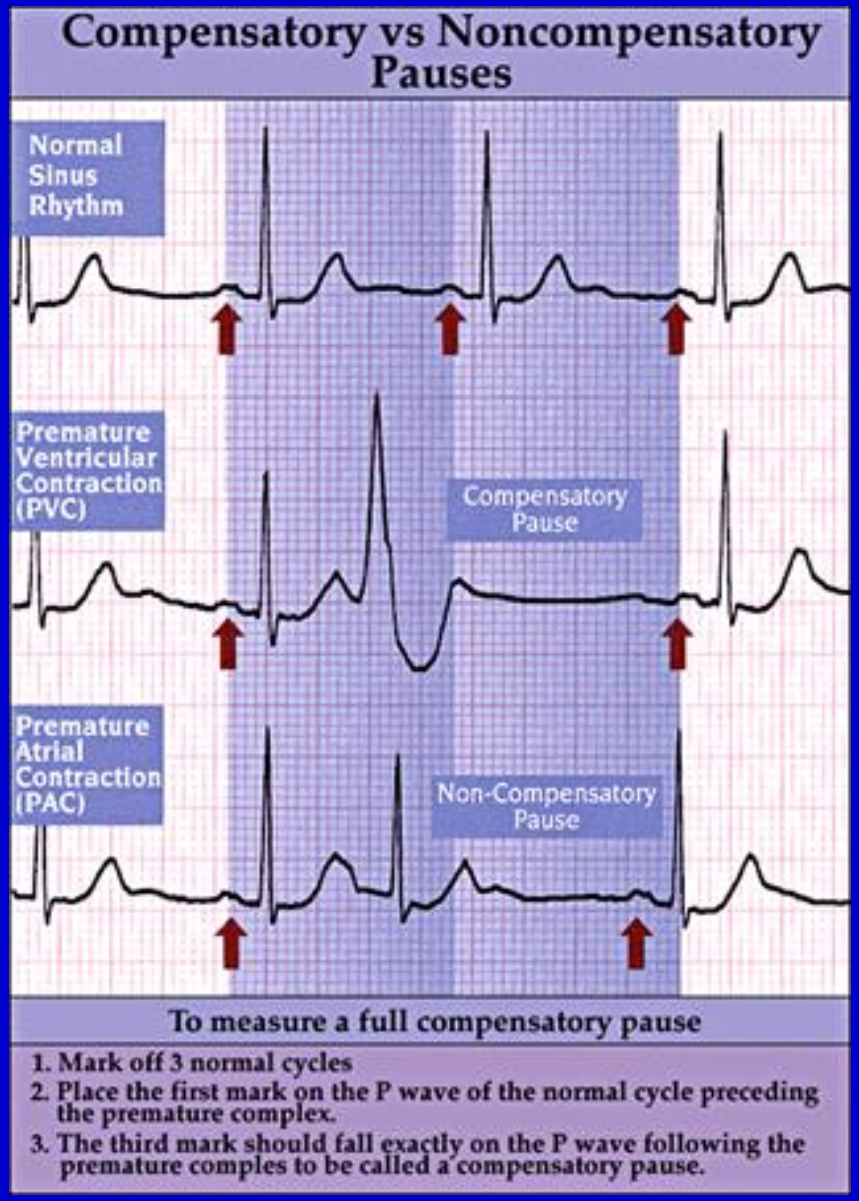
C) The patient suddenly looses consciousness, without puls, no breathing. Without reanimation he dies.

D) Young healthy person feels sometimes irregularities of heart beat w/o any other problem.

Ventricular extrasystole (VES), premature beat, premature ventricular contraction (PVC)

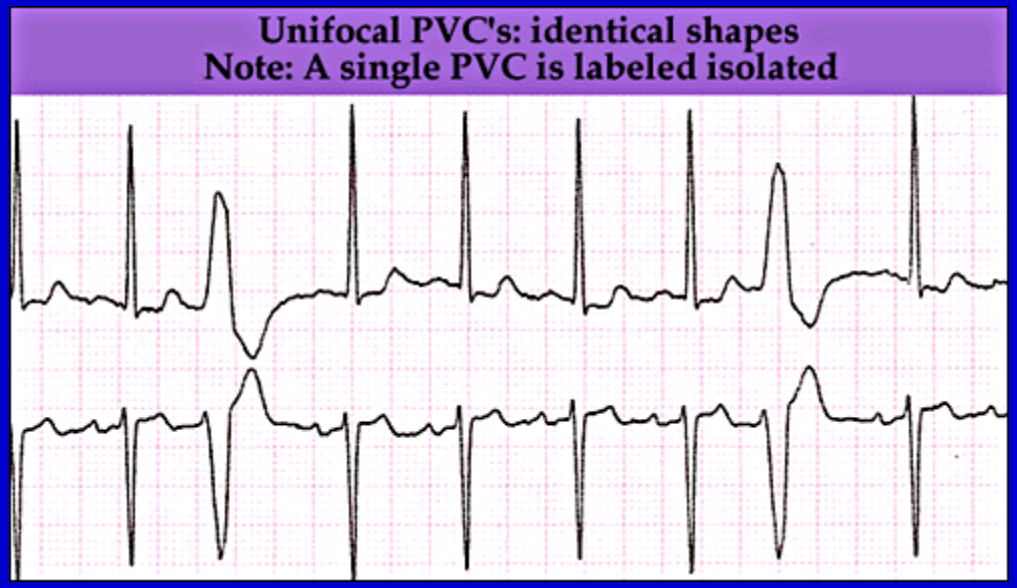
- the spreading in the ventricle is aberrant – QRS complex has *abnormal* shape
- it cannot spread to the atria in a retrograde way
- SA node has unchanged frequency of impulse formation, the impulse, however, cannot be conducted by AV node because of the refractory period in the ventricles
- the ventricles will be activated only by the next impulse from the SA node





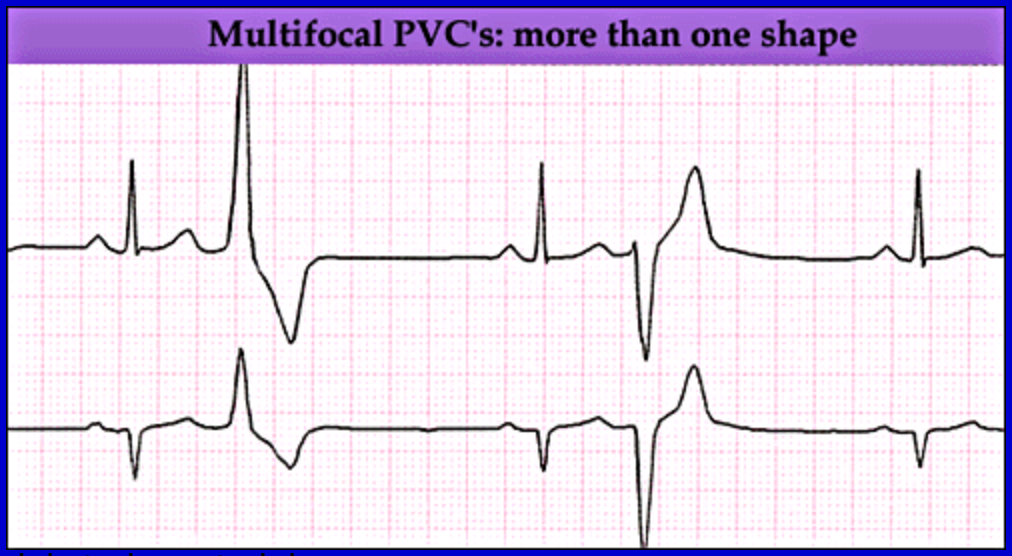
VES

SVES



Spreading from one site – monotopic

Spreading from more sites – polytopic



serious **are:**

- frequent

- polytopic

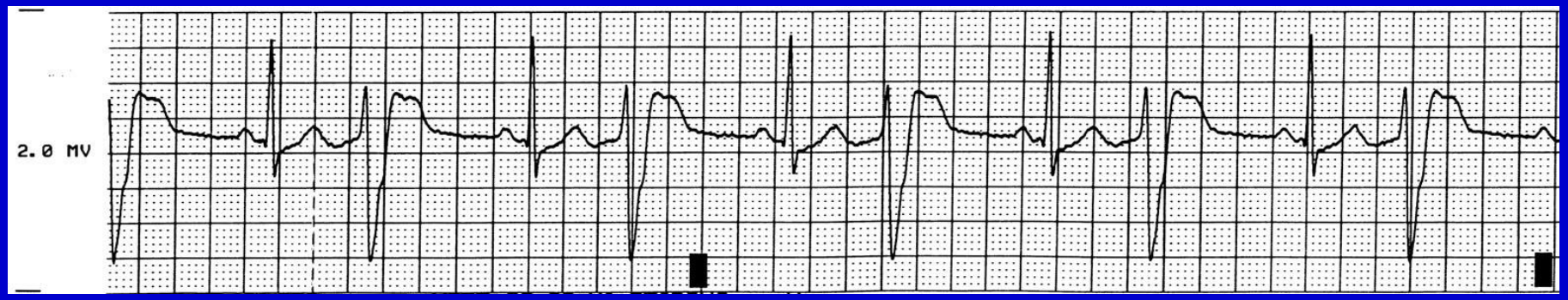
- two or more following each other

- paired to normal beat:

bigeminy (1+1), trigeminy (1+2)

- fenomen R/T (vulnerabile phase)

! predisposes to the ventricular tachycardia/fibrillation



Bigeminy

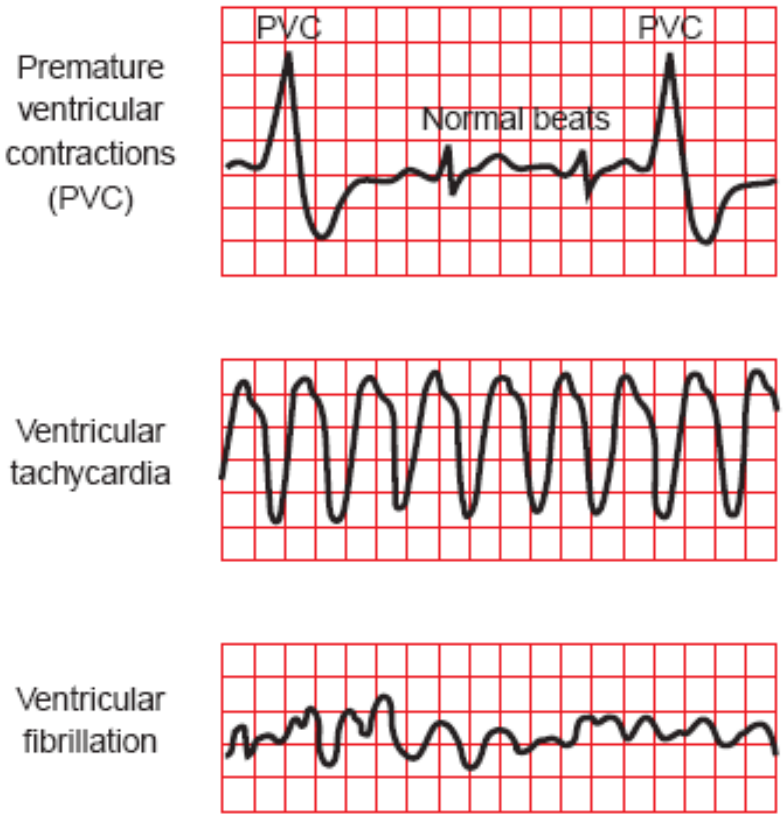


FIGURE 27-12 Electrocardiographic (ECG) tracings of ventricular arrhythmias. Premature ventricular contractions (PVCs) (*top tracing*) originate from an ectopic focus in the ventricles, causing a distortion of the QRS complex. Because the ventricle usually cannot repolarize sufficiently to respond to the next impulse that arises in the sinoatrial node, a PVC frequently is followed by a compensatory pause. Ventricular tachycardia (*middle tracing*) is characterized by a rapid ventricular rate of 70 to 250 beats per minute and the absence of P waves. In ventricular fibrillation (*bottom tracing*), there are no regular or effective ventricular contractions, and the ECG tracing is totally disorganized.

Tachyarrhytmia

importance of high HR for the circulation (preload, perfusion of the myocardium, energy and oxygen consumption)

Sinus tachycardia

increased activity of sympathetic nerves / decreasted activity of parasympathetic n. (atropin), catecholamines,
drugs influencing VNS, psychic influences, exercise, fever, anemia,
thyreotoxicosis etc.
ECG is normal

Importance of heart rate for the heart function: duration of diastole

1. *filling of the ventricles* (preload) – decreased in high HR, increased in bradycardia

2. *cardiac output* – increased HR × decrease of preload in high tachycardia, very slow HR decreases CO

3. *perfusion of myocardium* – high HR impaires perfusion

4. *blood pressure*

5. *contractility* – tachycardia increases contractility (calcium entry)

6. *oxygen and energy consumption* – increased in tachycardia

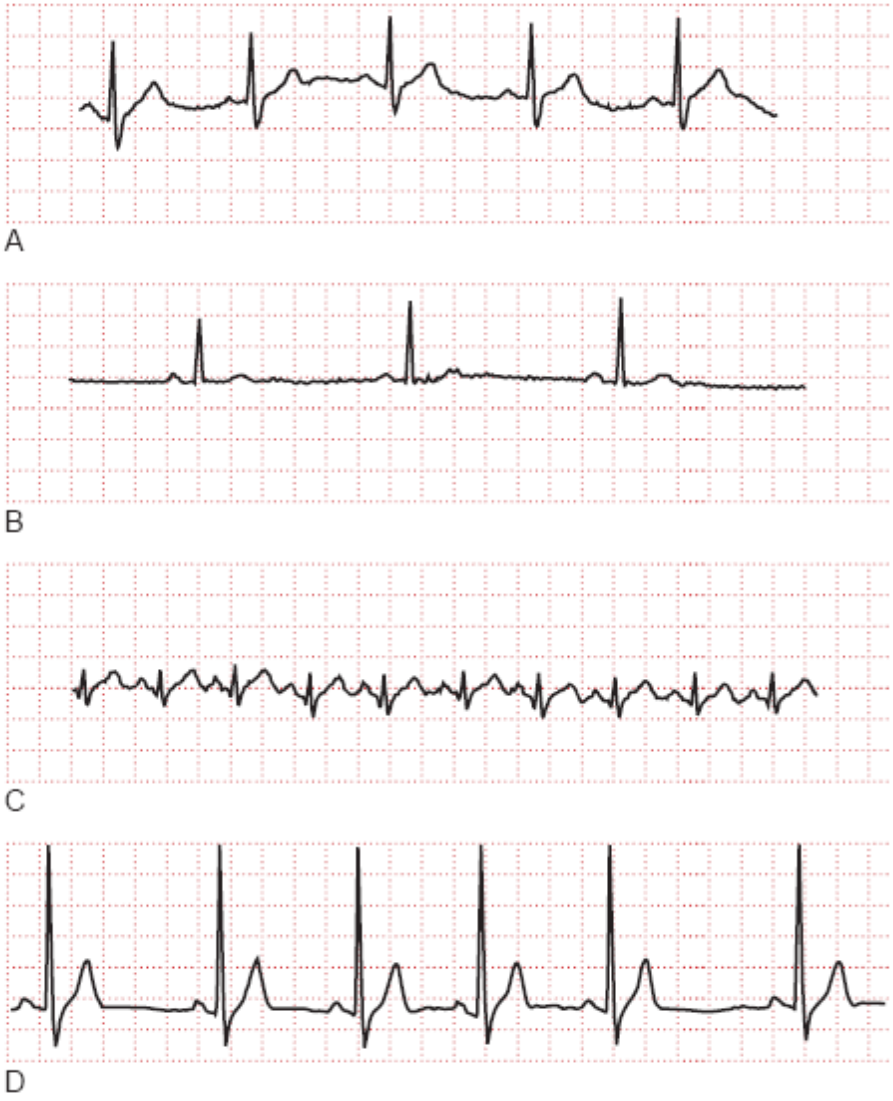


FIGURE 27-9 Electrocardiographic (ECG) tracings of rhythms originating in the sinus node. (**A**) Normal sinus rhythm (60 to 100 beats per minute). (**B**) Sinus bradycardia (<60 beats per minute). (**C**) Sinus tachycardia (>100 beats per minute). (**D**) Respiratory sinus arrhythmia, characterized by gradually lengthening and shortening of RR intervals.

SV tachycardia

sometimes in healthy persons

ECG: normal QRS,

changes of P wave and PQ interval

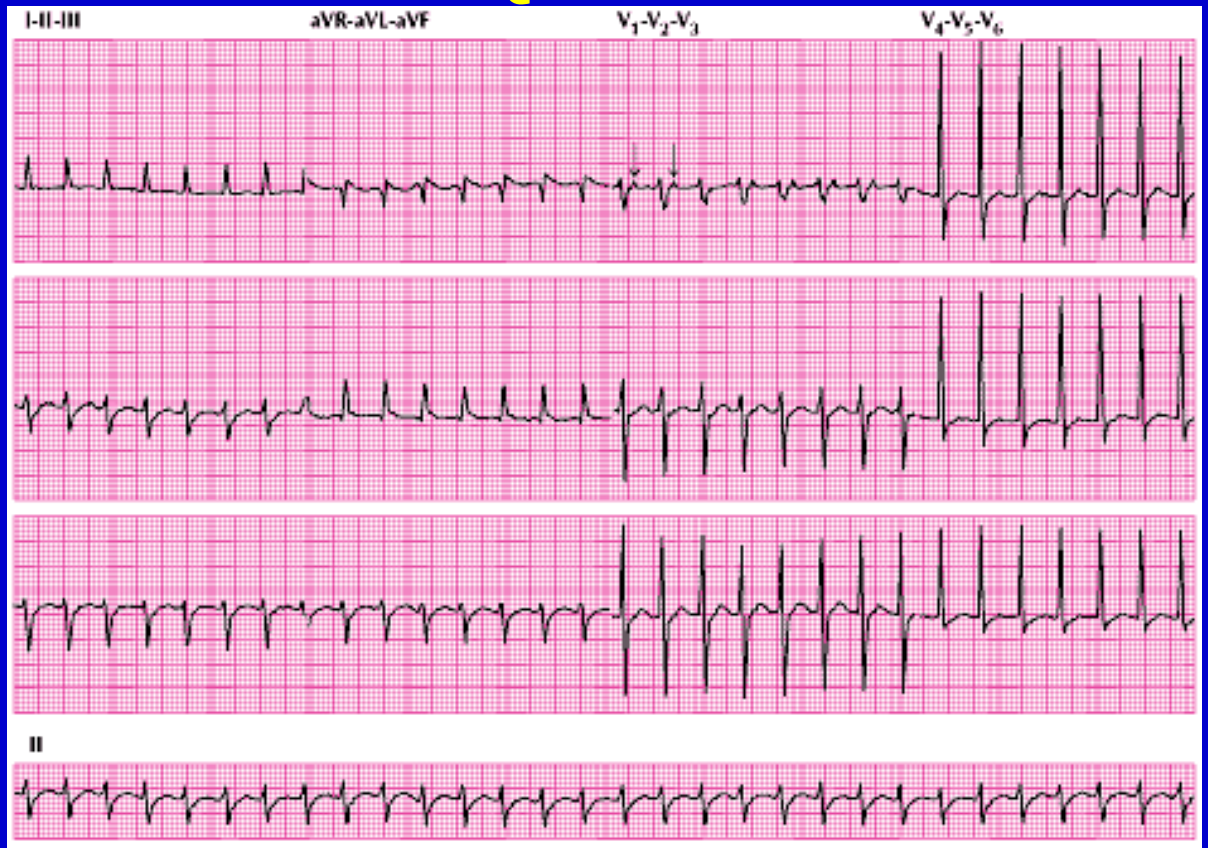


Figure 2. ECG shows supraventricular tachycardia in a 36-year-old woman with frequent episodes of sudden onset, rapid, and regular heart rate. The ventricular rate is 183 bpm. Note the P waves at the end of the QRS complex (arrows in V_1). Symptoms persisted despite treatment with oral verapamil and metoprolol, and the patient was referred for radiofrequency ablation. AV-node reentry tachycardia was diagnosed on electrophysiologic testing. The patient underwent successful ablation of the "slow pathway" with resolution of symptoms.

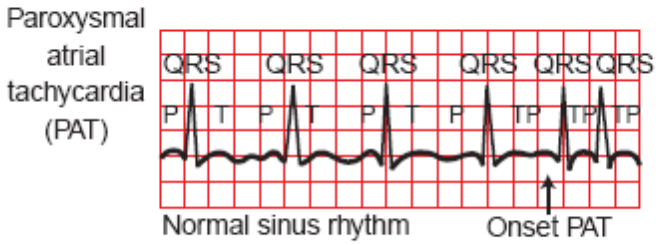
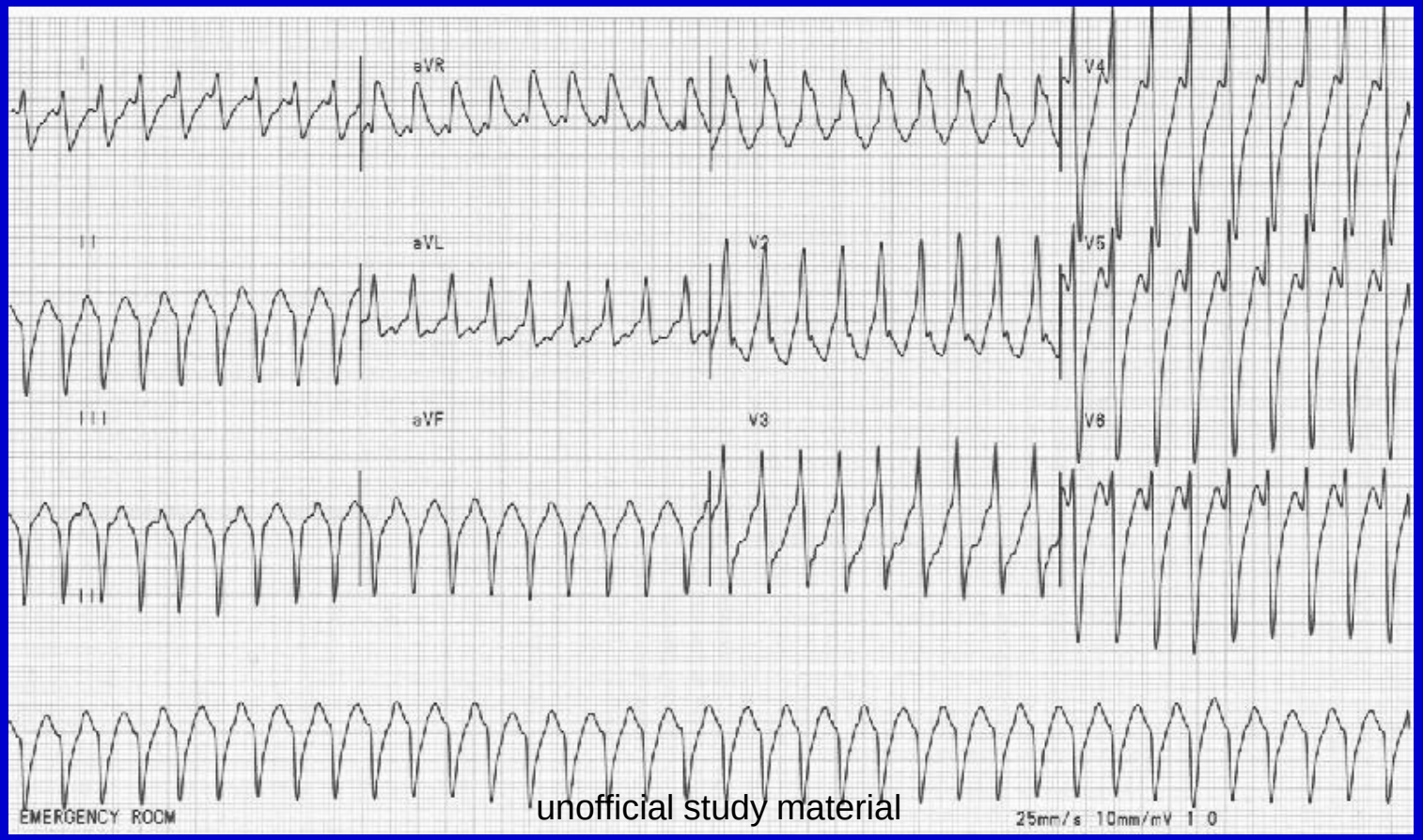


FIGURE 27-10 Electrocardiographic tracings of atrial arrhythmias. Atrial flutter *(first tracing)* is characterized by the atrial flutter (F) waves occurring at a rate of 240 to 450 beats per minute. The ventricular rate remains regular because of the conduction of every sixth atrial contraction. Atrial fibrillation *(second tracing)* has grossly disorganized atrial electrical activity that is irregular with respect to rate and rhythm. The ventricular response is irregular, and no distinct P waves are visible. The *third tracing* illustrates paroxysmal atrial tachycardia (PAT), preceded by a normal sinus rhythm. The *fourth tracing* illustrates premature atrial complexes (PAC).

Ventricular tachycardia

urgent!! hemodynamically and electrically (development of ventricular fibrillatio),
ECG: fast, irregular, bizarre QRS



Fibrillation

- rapid, irregular, and unsynchronized contraction of muscle fibers
- Chaotical electrical events
- Inadequate mechanical response – virtually no output
- Atrial
- Ventricular

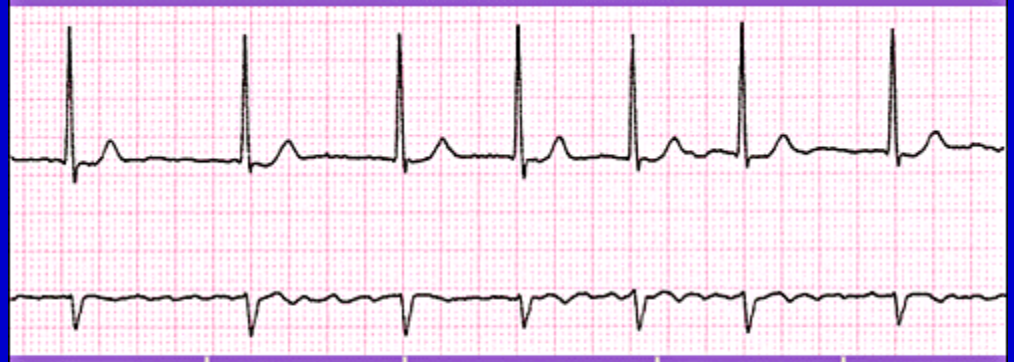
Atrial fibrillation

very frequent, mainly in elderly people (CHD), in younger more often in thyreotoxicosis or postrheumatical mitral valve disease (mainly stenosis)

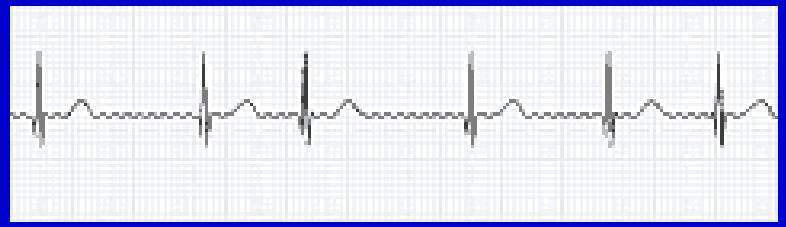
Atrial fibrillation

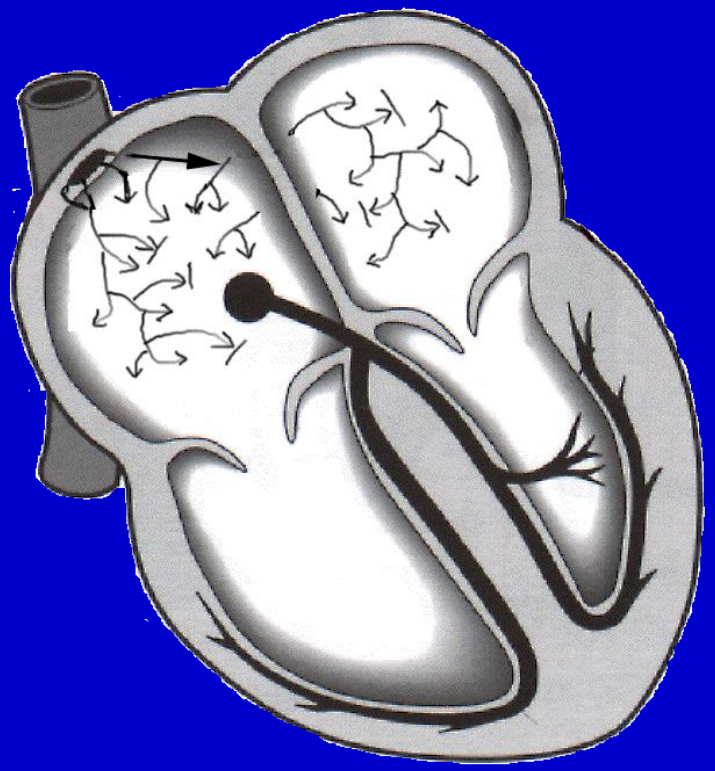
- **absolutely irregular el. activity of atria with frequency up to >300/min, without efficient contractions**
- **only some of the impulses are conducted to the ventricles: pulse is *absolutely* irregular, the filling of the ventricles is variable (pulse deficit can occur)**
- **ECG: fibrillation waves (*f*) between QRS complexes, QRS complexes have normal shape**

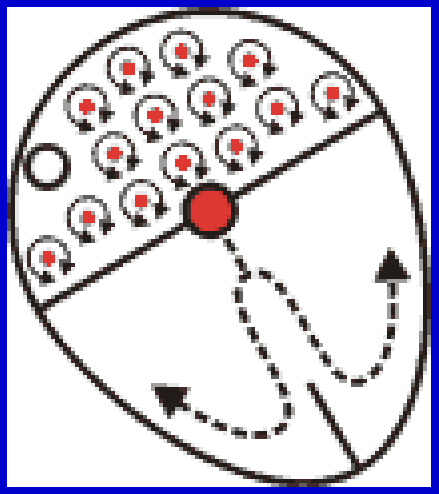
Atrial Fibrillation



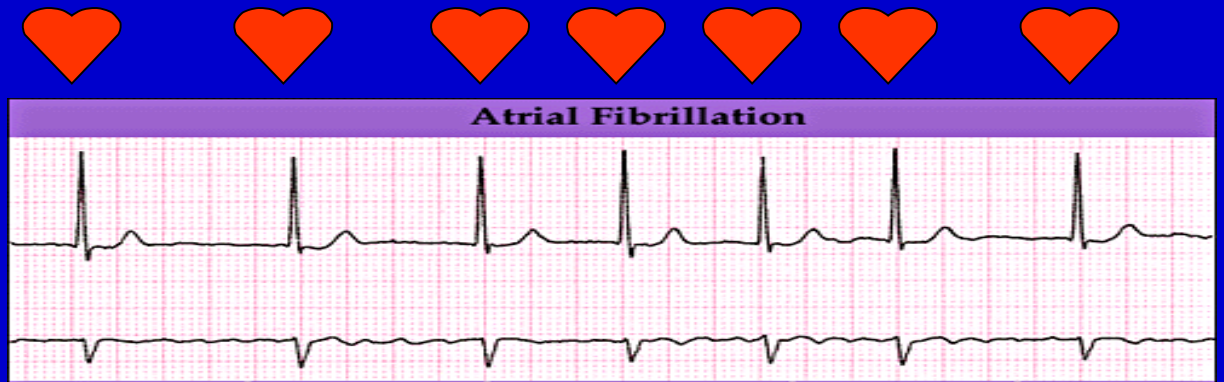
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 350-650 bpm V: Slow to rapid	Irregular	Fibrillatory (fine to course)	N/A	<.12







THE PATIENTS HAVE ABSOLUTELY IRREGUALAR PULSE

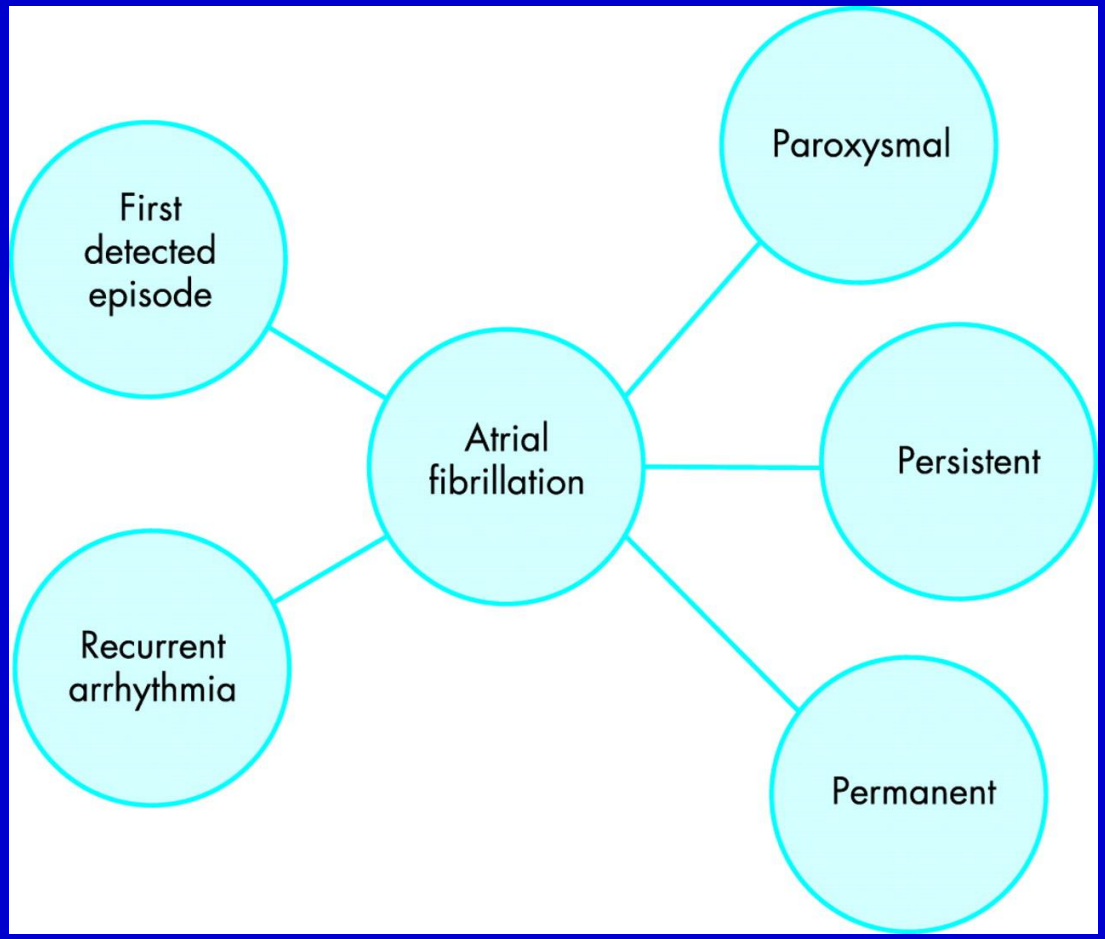


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 350-650 bpm V: Slow to rapid	Irregular	Fibrillatory (fine to course)	N/A	<.12

Atrial fibrillation – hemodynamics

- **no contraction of atria, their contribution to the ventricular filling is missing**
  preload can be decreased
  (important mainly in heart failure)
- **Variable preload in the ventricles (pulse deficit)**
- **frequently thrombi in atria (embolism !): anticoagulation therapy**

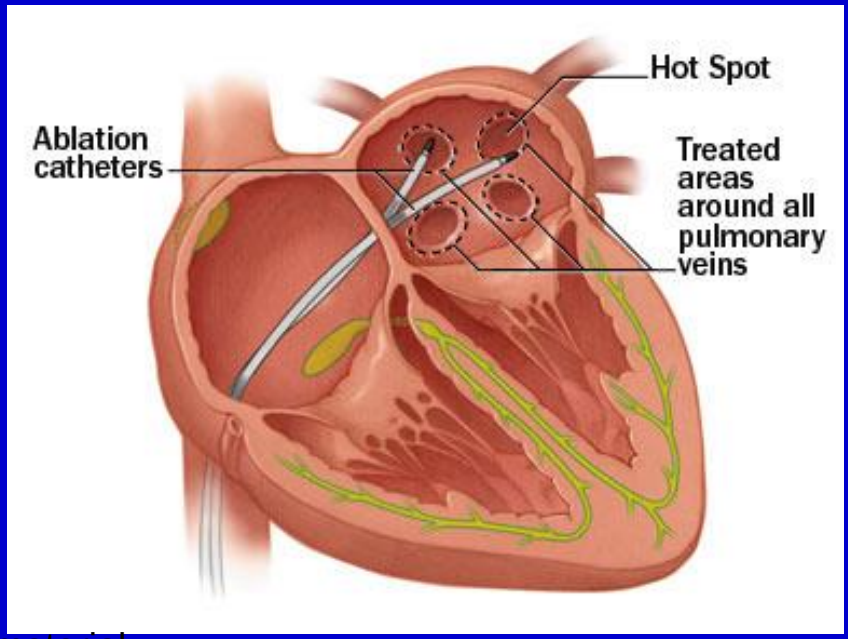
AF – forms



AF – pathogenesis

- In most cases the structural changes / disease of the heart is present
- Often the ectopic foci with fast impulses in the area of the pulmonary veins estuary
- Sometimes even in healthy heart but if lasting longer it can lead to the structural changes of the atrium
- In some patients alcohol can trigger the AF

AF – ablation therapy



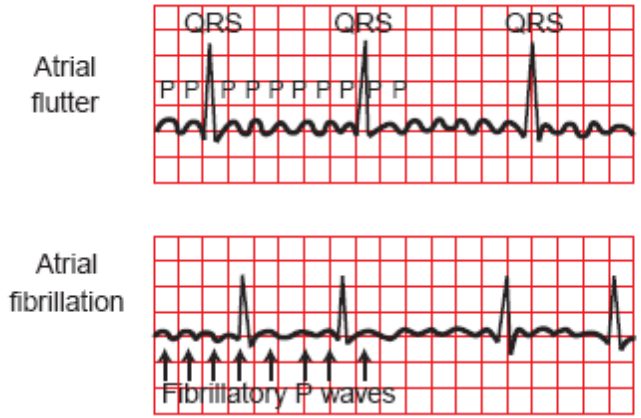


FIGURE 27-10 Electrocardiographic tracings of atrial arrhythmias. Atrial flutter *(first tracing)* is characterized by the atrial flutter (F) waves occurring at a rate of 240 to 450 beats per minute. The ventricular rate remains regular because of the conduction of every sixth atrial contraction. Atrial fibrillation *(second tracing)* has grossly disorganized atrial electrical activity that is irregular with respect to rate and rhythm. The ventricular response is irregular, and no distinct P waves are visible. The *third tracing* illustrates paroxysmal atrial tachycardia (PAT), preceded by a [illegible] *fourth tracing* illustrates premature atrial complexes (PAC).

CASE REPORTS

A) The patient feels irregularities in heart beat (palpitations), sometimes faster, sometimes slower. At times he feels weak and is about fainting.

B) The patient repeatedly looses consciousness, is without puls. After a while his consciousness restores-

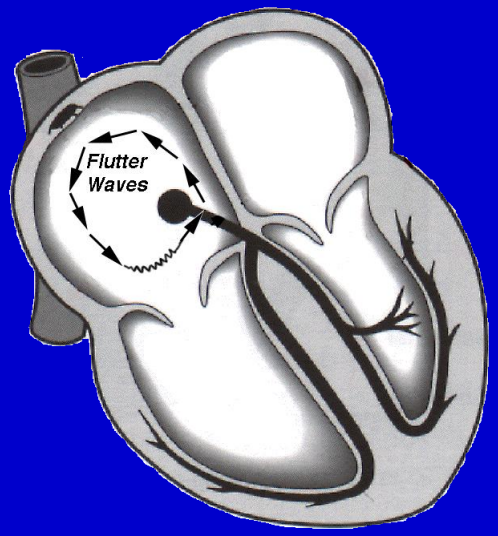
C) The patient suddenly looses consciousness, without puls, no breathing. Without reanimation he dies.

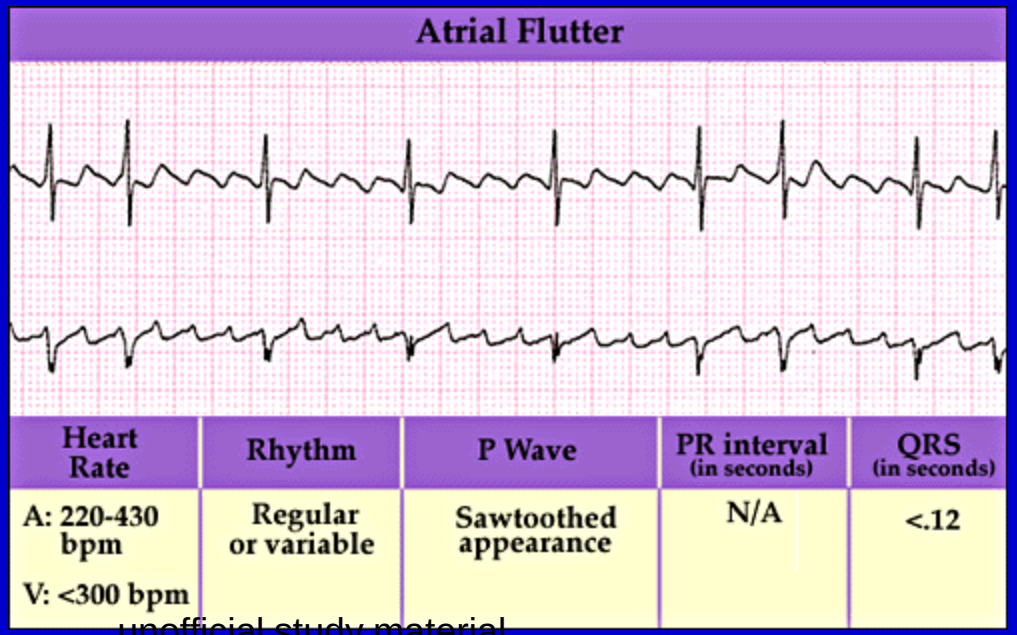
D) Young healthy person feels sometimes irregularities of heart beat w/o any other problem.

Atrial flutter

less frequent, el. activity in the atria is regular

usually more serious than fibrillation, depending on the resulting HR





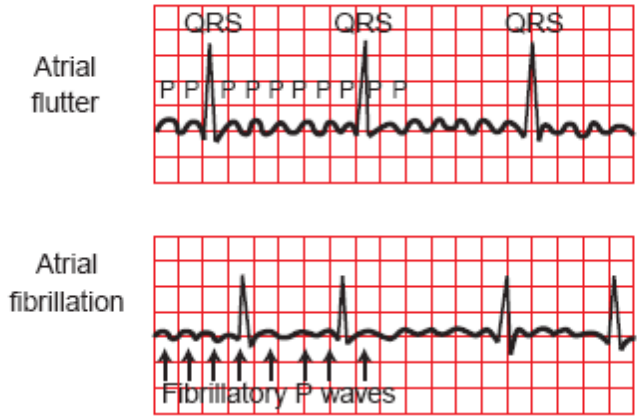


FIGURE 27-10 Electrocardiographic tracings of atrial arrhythmias. Atrial flutter *(first tracing)* is characterized by the atrial flutter (F) waves occurring at a rate of 240 to 450 beats per minute. The ventricular rate remains regular because of the conduction of every sixth atrial contraction. Atrial fibrillation *(second tracing)* has grossly disorganized atrial electrical activity that is irregular with respect to rate and rhythm. The ventricular response is irregular, and no distinct P waves are visible. The *third tracing* illustrates paroxysmal atrial tachycardia (PAT), preceded by a [illegible] *fourth tracing* illustrates premature atrial complexes (PAC).

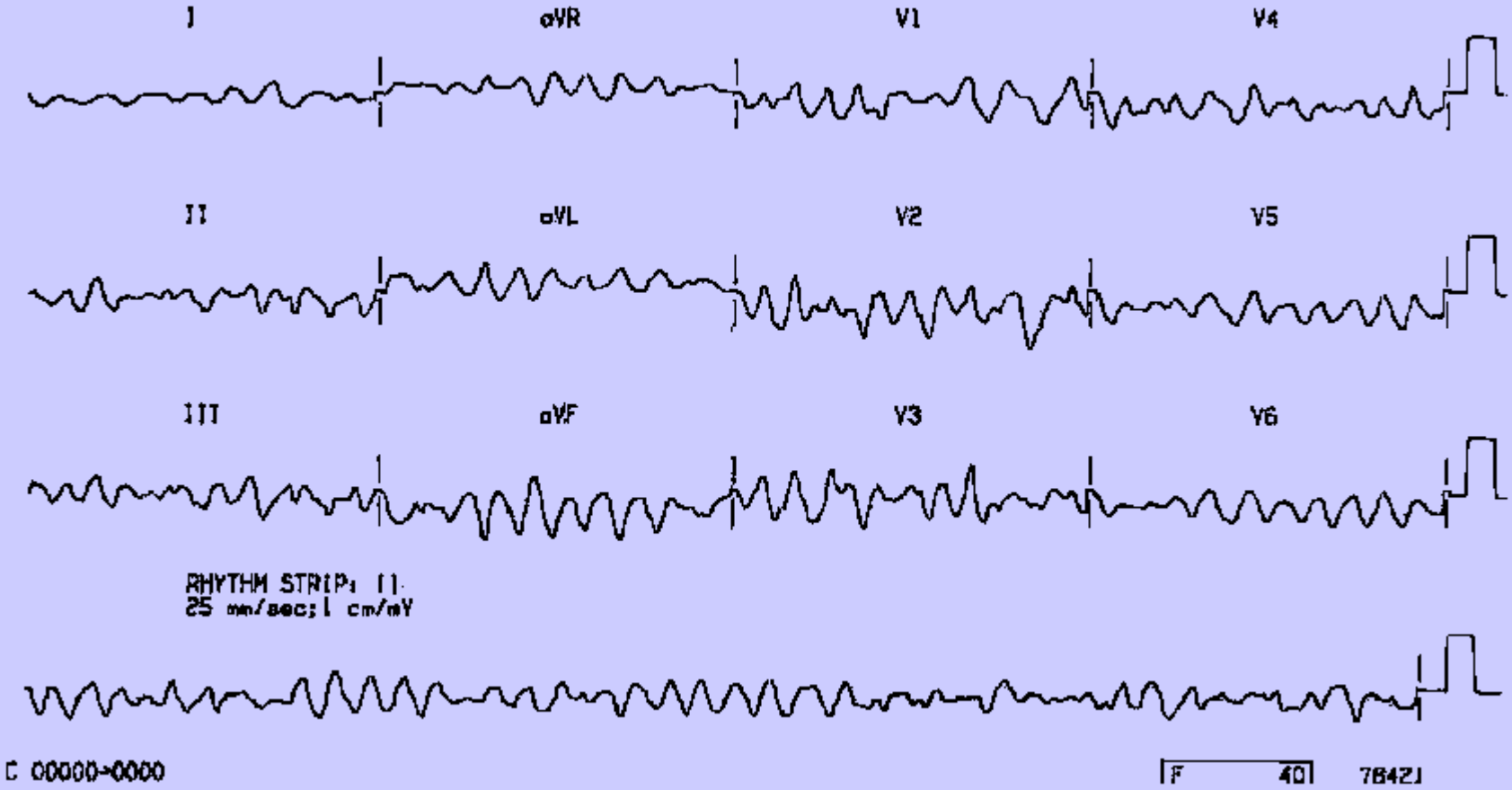
Ventricular fibrillation

acute, life-threatening situation with complet hemodynamic failure

-no cardiac output,
-no pulse,
-unconsciousness,
-reanimation required to save life

frequent cause of death in the early acute myocardial infarction cardiomyopathy

defibrillation



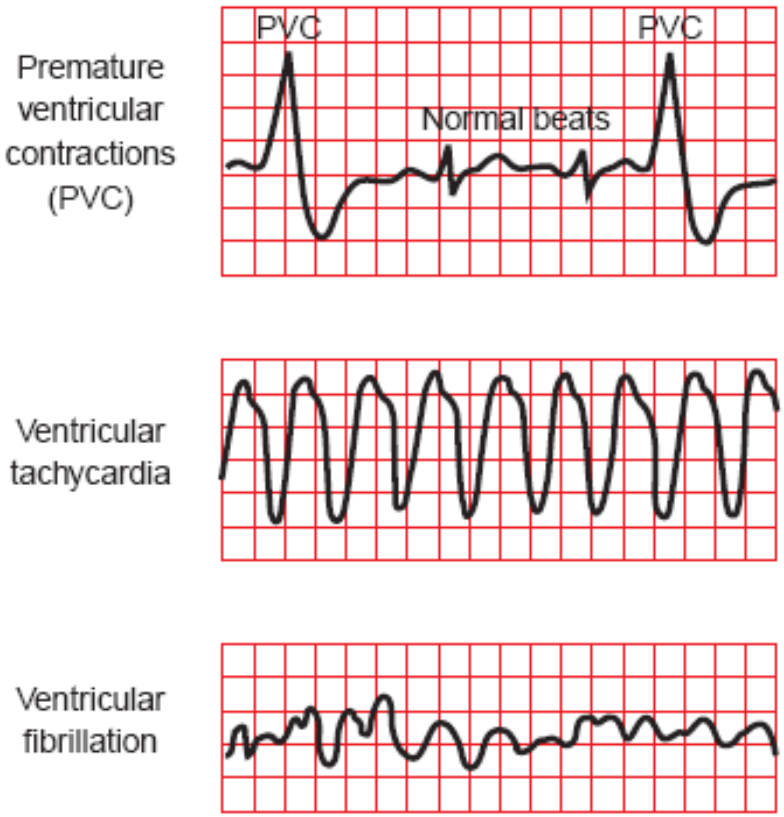
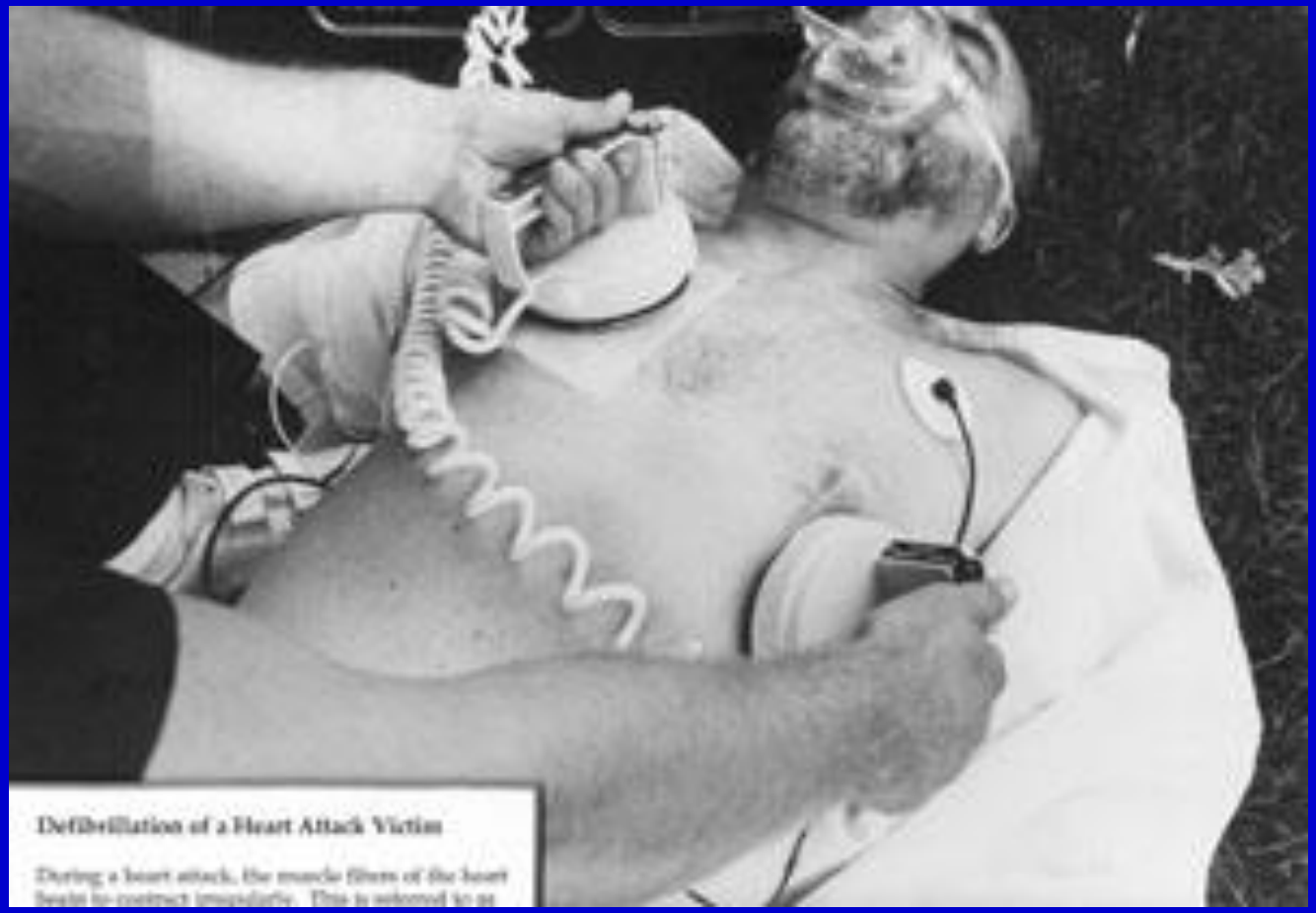


FIGURE 27-12 Electrocardiographic (ECG) tracings of ventricular arrhythmias. Premature ventricular contractions (PVCs) (*top tracing*) originate from an ectopic focus in the ventricles, causing a distortion of the QRS complex. Because the ventricle usually cannot repolarize sufficiently to respond to the next impulse that arises in the sinoatrial node, a PVC frequently is followed by a compensatory pause. Ventricular tachycardia (*middle tracing*) is characterized by a rapid ventricular rate of 70 to 250 beats per minute and the absence of P waves. In ventricular fibrillation (*bottom tracing*), there are no regular or effective ventricular contractions, and the ECG tracing is totally disorganized.

Defibrillation



CASE REPORTS

A) The patient feels irregularities in heart beat (palpitations), sometimes faster, sometimes slower. At times he feels weak and is about fainting.

B) The patient repeatedly looses consciousness, is without puls. After a while his consciousness restores-

C) The patient suddenly looses consciousness, without puls, no breathing. Without reanimation he dies.

D) Young healthy person feels sometimes irregularities of heart beat w/o any other problem.

Bradyarrhytmias

Sinus bradycardia

vagus

normal: exercise

pathology: acute myocardial infarction
of diaphragmatic wall

cranial hypertension, some infections...

sick sinus syndrome

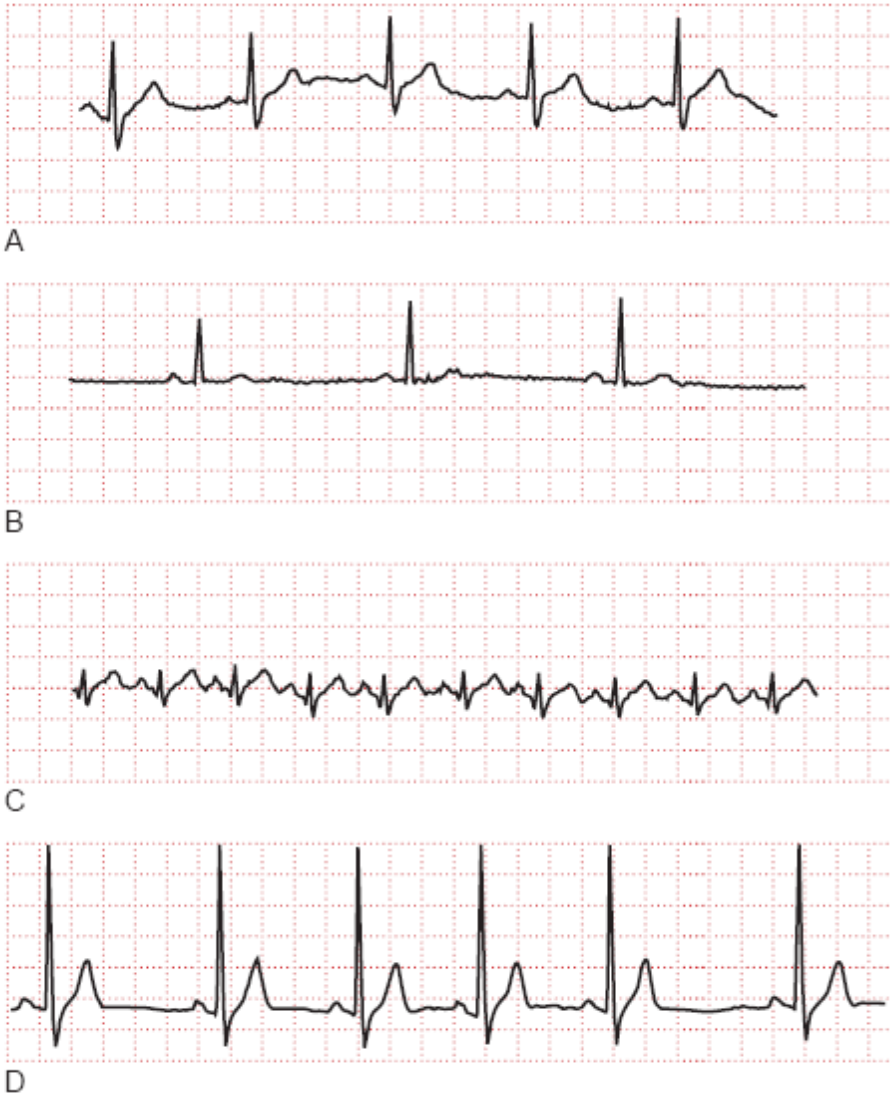


FIGURE 27-9 Electrocardiographic (ECG) tracings of rhythms originating in the sinus node. (**A**) Normal sinus rhythm (60 to 100 beats per minute). (**B**) Sinus bradycardia (<60 beats per minute). (**C**) Sinus tachycardia (>100 beats per minute). (**D**) Respiratory sinus arrhythmia, characterized by gradually lengthening and shortening of RR intervals.

Sick sinus syndrome

-sinus bradycardia
-insufficient increase in HR during exercise
-sinoatrial blocks
-paroxysmal SV tachycardias or
atrial fibrillation

bradycardia-tachycardia syndrome

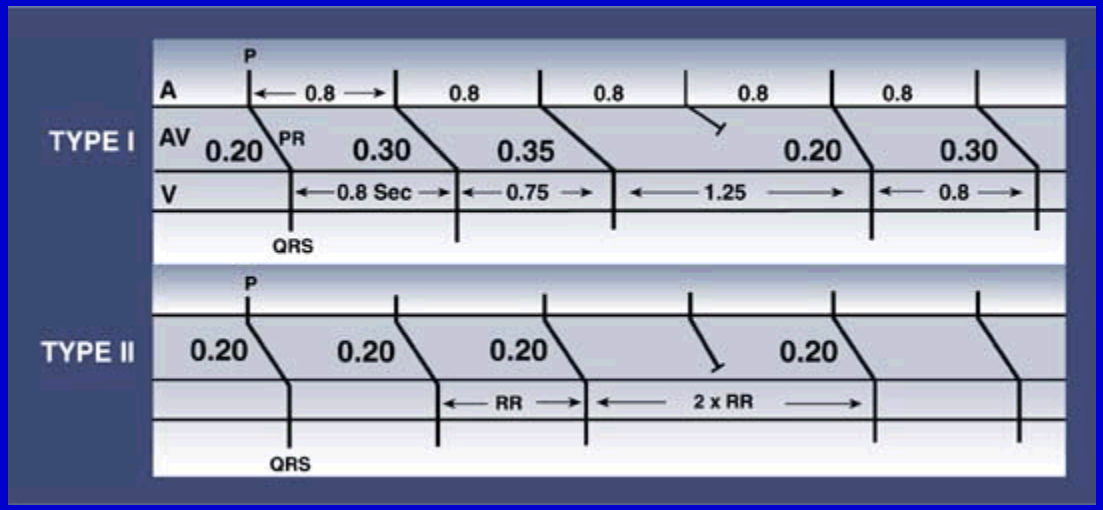
Blocks

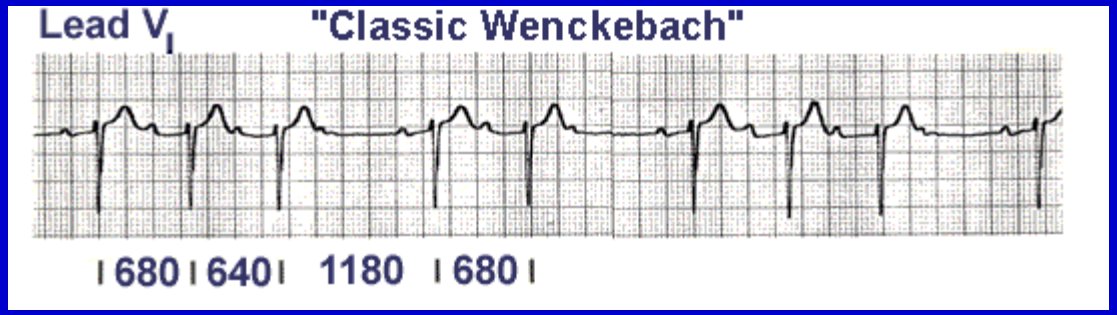
I. slowing, prolongation
II. partial blockade
III. complete blockade

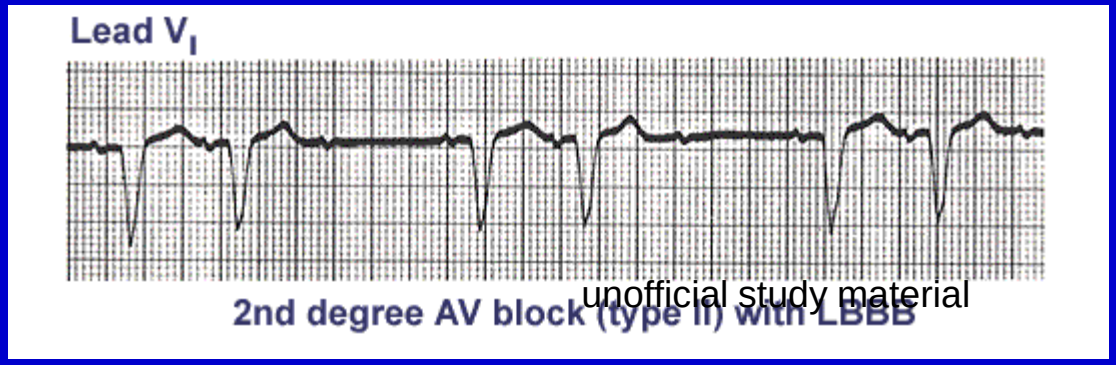
Sinoatrial block

Atrioventricular block

I. degree
II. degree
type Wenckebach (Mobitz I)
type Mobitz (Mobitz II)
III. degree
Adams-Stokes attacks

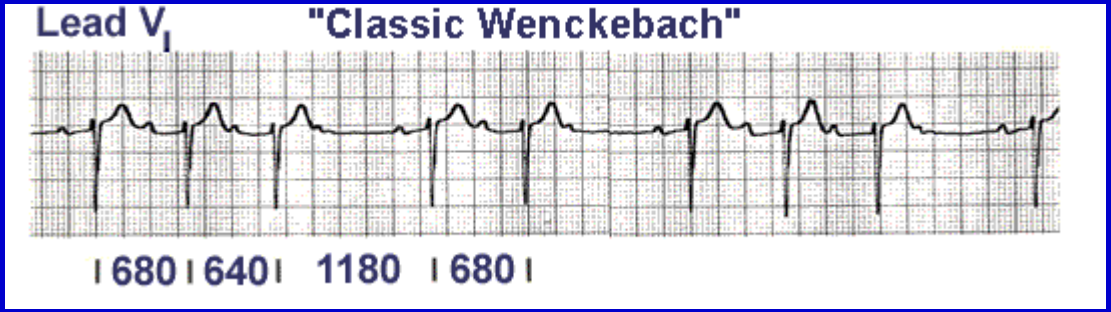






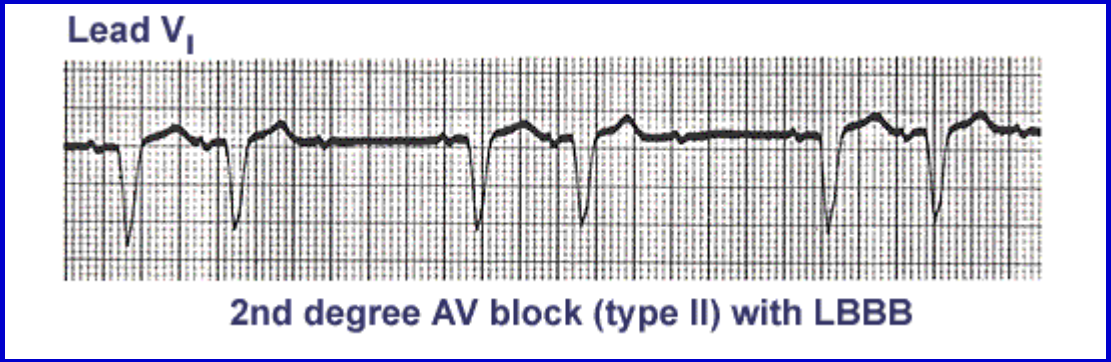
Wenkebach (Mobitz I)

prolongation of PR intervals



Mobitz (Mobitz II)

PR intervals do not change



Ratio atria : ventricles (P:QRS)

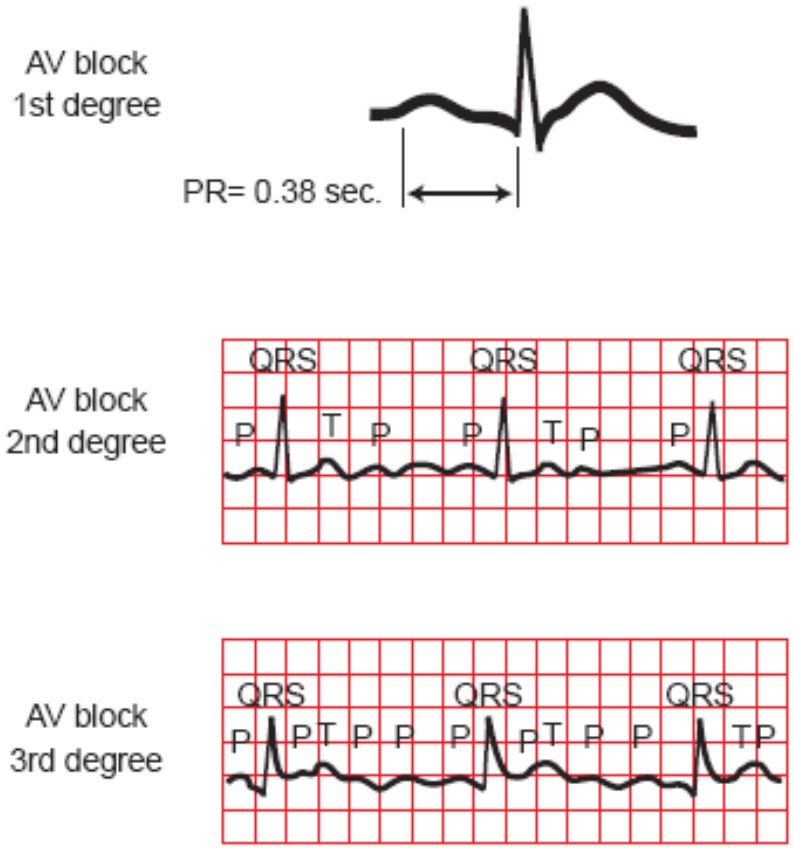
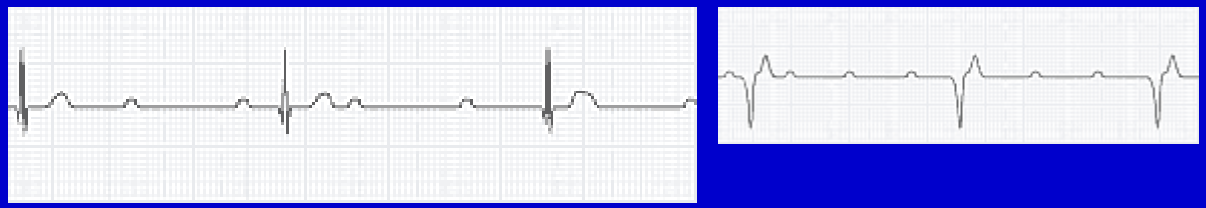


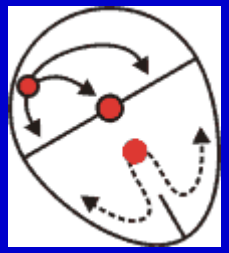
FIGURE 27-13 Electrocardiographic changes that occur with alterations in atrioventricular (AV) node conduction. The *top tracing* shows the prolongation of the PR interval, which is characteristic of first-degree AV block. The *middle tracing* illustrates Mobitz type II second-degree AV block, in which the conduction of one or more P waves is blocked. In third-degree AV block (*bottom tracing*), complete block in conduction of impulses through the AV node occurs, and the atria and ventricles develop their own rates of impulse generation.

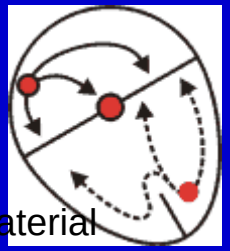
Block III. degree - AV dissociation

Complete block, no propagation to the ventricles
No ventricular complexes and contractions
No cardiac output
Unconsciousness, no puls

Escaped ventricular rhythm







CASE REPORTS

A) The patient feels irregularities in heart beat (palpitations), sometimes faster, sometimes slower. At times he feels weak and is about fainting.

B) The patient repeatedly looses consciousness, is without puls. After a while his consciousness restores (Adams-Stokes)

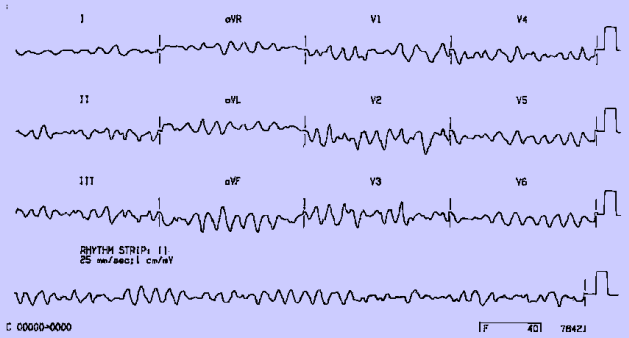
C) The patient suddenly looses consciousness, without puls, no breathing. Without reanimation he dies.

D) Young healthy person feels sometimes irregularities of heart beat w/o any other problem.

Uncosciousness

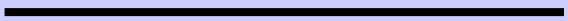
- **Breathing ?**
- **Pulse?**

Unconsciousness + no pulse



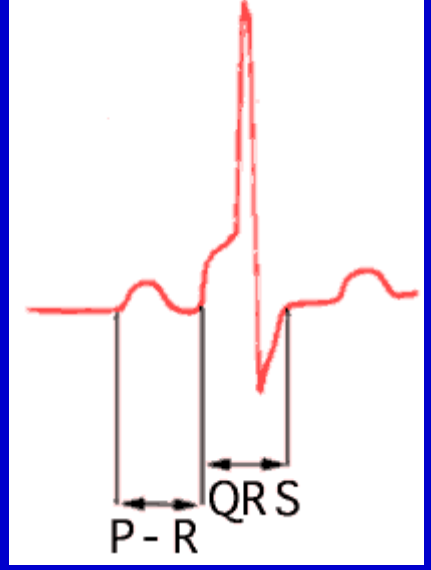
- Ventricular fibrillation
- AV blok III. degree

- Asystoly



- Sick sinus syndrome

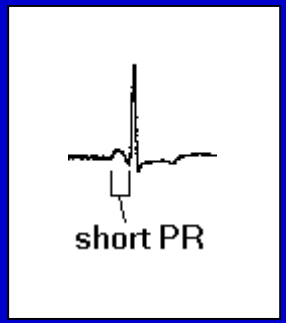
Abnormal AV conduct

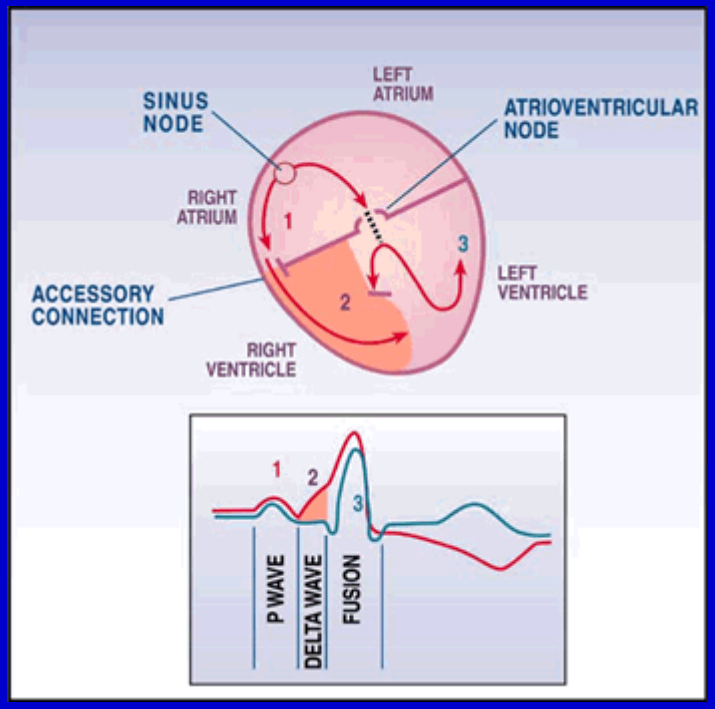


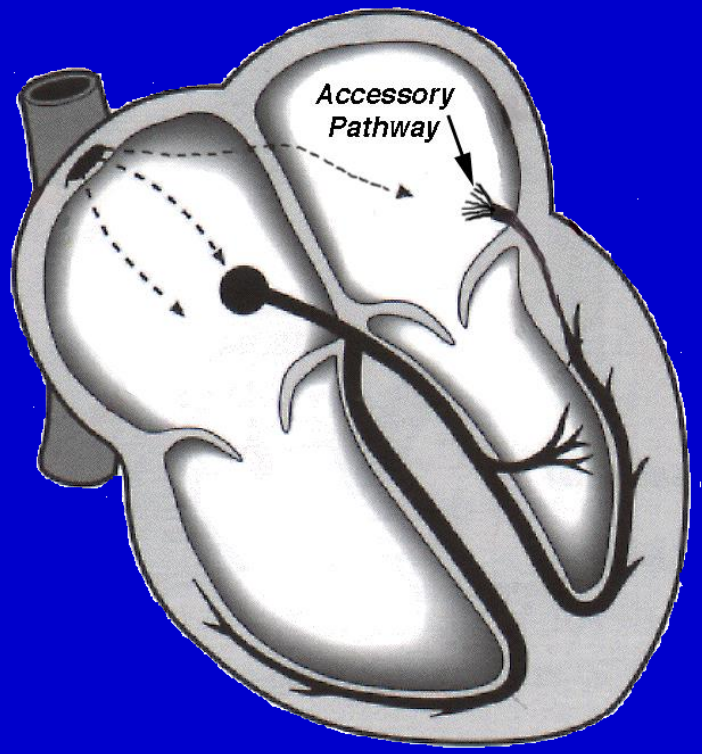
accessory pathways
preexcitation syndromes

WPW syndrome (Wolff-Parkinson-White)
abnormal *Kent* pathway (out of AV node)
PQ interval shorter and changed, changed QRS complex
re-entry mechanisms can lead to more serious arrhytmias (SV tachycardia, atrial fibrillation or flutter)

LGL syndrom (Lown-Ganong-Levin)
accessory pathway connected to the distal part of AV node (*James fibres*) or to the His bundle (*Brechenmacher* fibers)
PQ shortened, QRS of normal shape







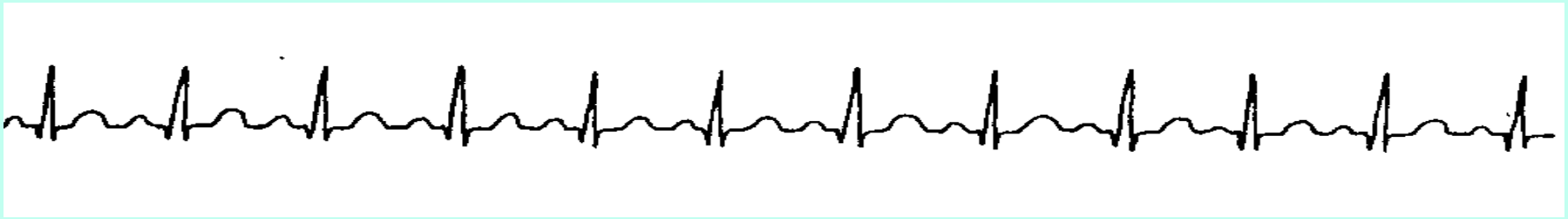
ARRHYTMIA

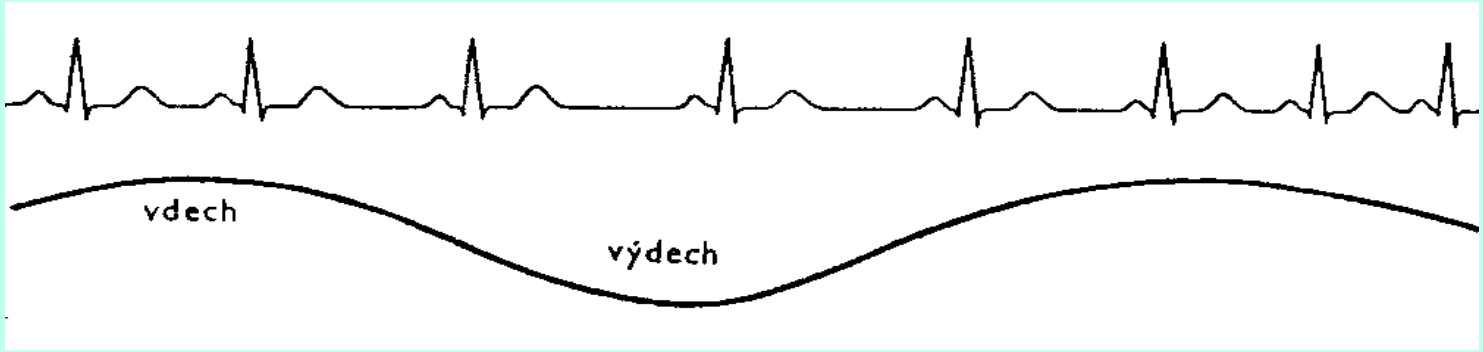
Frequent in childhood,

On structurally normal heart mostly benign

Normal ***heart rate in children***

Sinus rhytm, sinus (respiratory) arrhytmia due to changes of vagotony during the respiration





Other physiological arrhytmias in children

Escaped junction rhytm due to vagotony – during the sleep in 19% of neonatals and 45% children

A-V block II. dg. – during the sleep even in 11% healthy children

Atrial ES – 14% of healthy sucklings

Ventricular ES – 10-20% children (not frequent uniform isolated premature ventricular contractions which disappear during the effort)

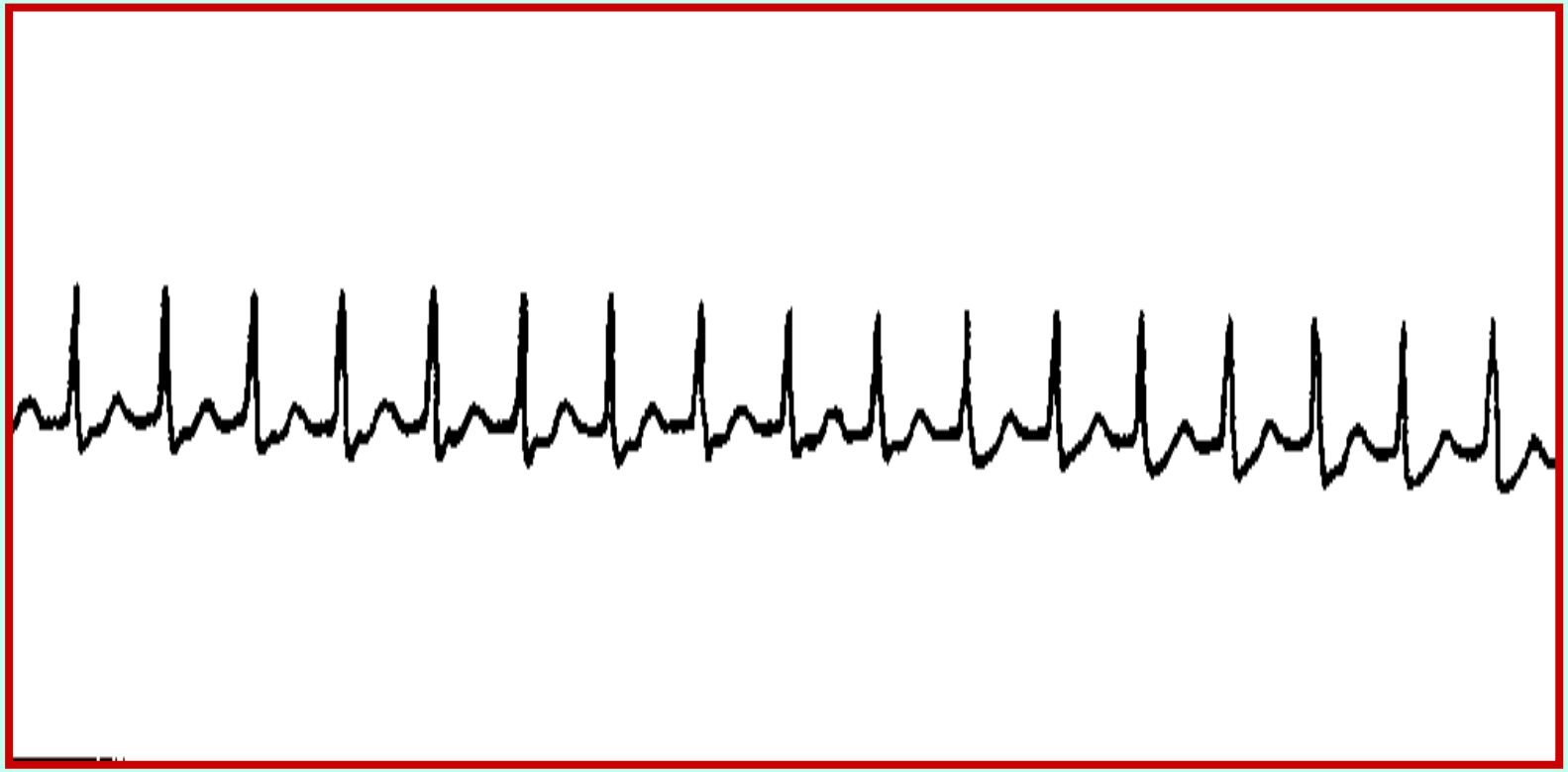
Most frequent arrhytmias in pediatrics

Bradyarrhytmias

Hypoxia
Dysfunction of sinus node (vagotony, hypothyresis, drugs, postoperative status)
S-A block
A-V block (congenital, postoperative, postinflammatory)

Most frequent arrhytmias in pediatrics

Supraventricular tachycardia



Mechanisms of arrhytmias

- changes in action potential
- re-entry
- electrical nonhomogenity

ARYTMOGENIC MECHANISMS

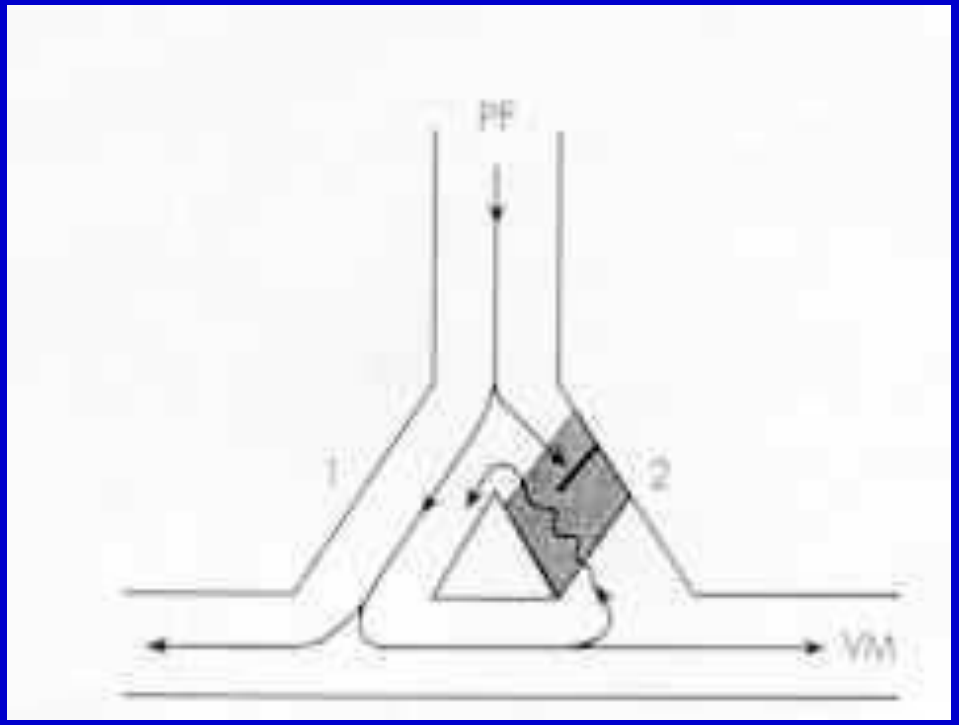
* changes in automaticity
* triggered activity
* re-entry

REENTRY

main cause of tachyarrhytmias

- two pathways proximally and distally connected
- different conductivity (slow)
- unidirectional conduction block of 1 pathway

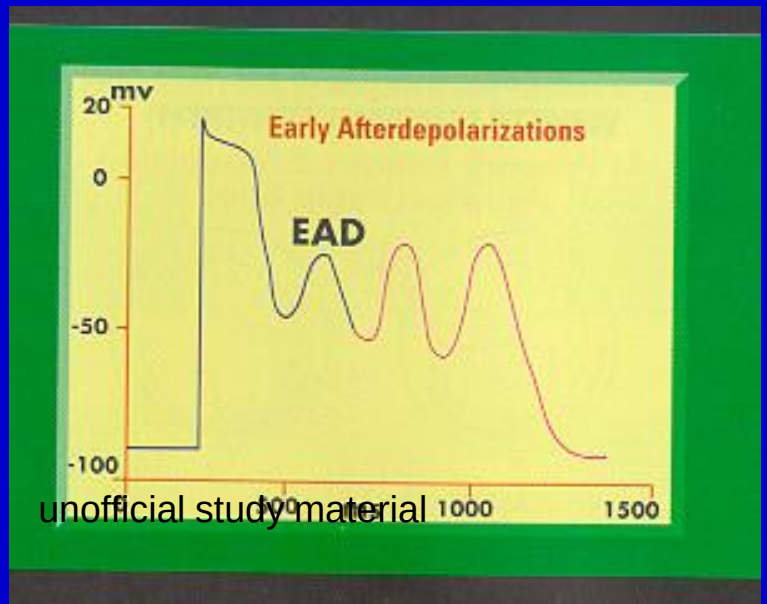
ischemia, fibrosis
typically accessory pathways



TRIGGERED ACTIVITY

abnormal repolarization
repeated spontaneous depolarization

1. Early afterdepolarization (EAD)
Before the end of repolarization (phase 3) new depolarization occurs due to opening of channels for Na^{+} and Ca^{++}.
Often in long QT, bradycardia, hypokalemia (long MAP)



Occurrs mainly in

long QT,
bradycardia,
hypokalemia (long MAP),
hypoxia

Consequences

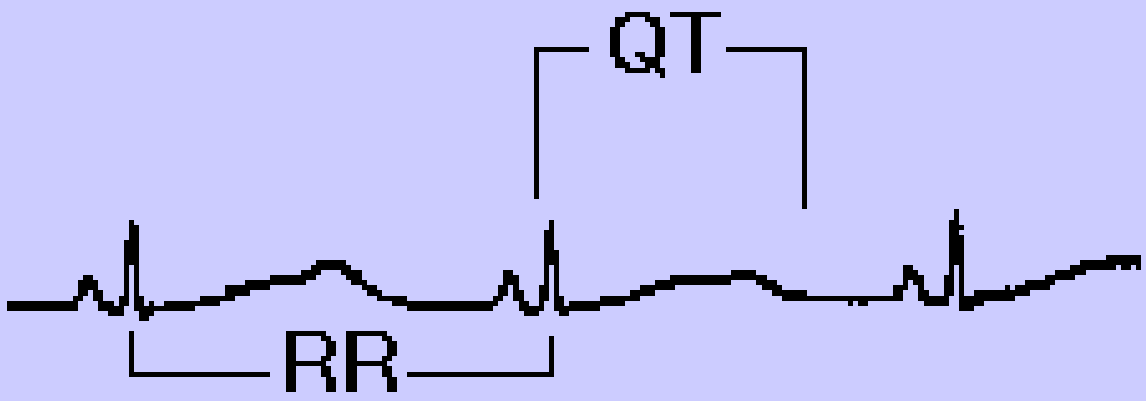
-Fast HR (tachyarrhytmia)
-torsade de pointes

Syndrom of long QT

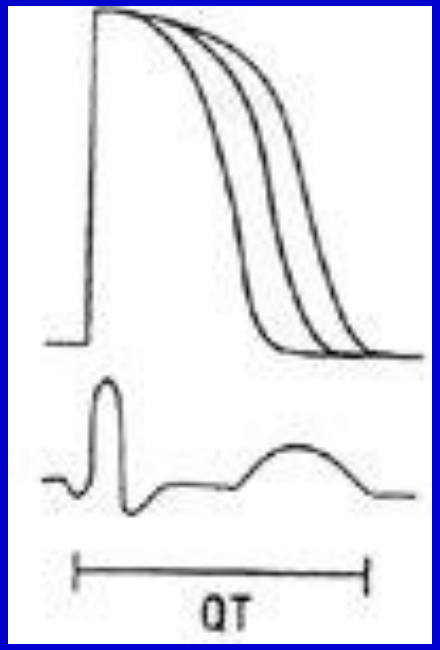
interval QT longer

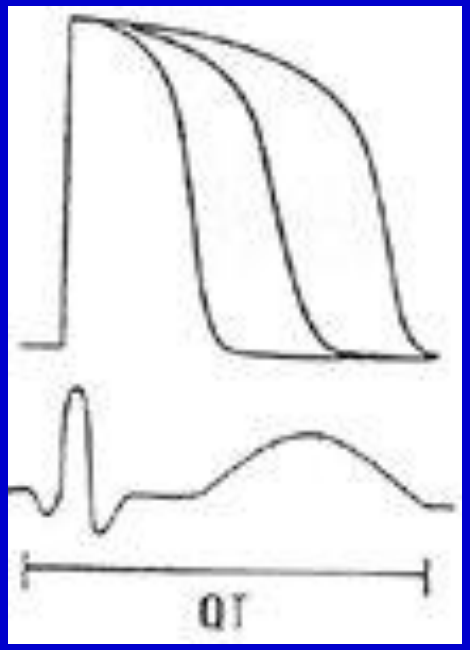
importance: connected to frequent serious ventricular tachyarrhytmias
length and *dispersion*

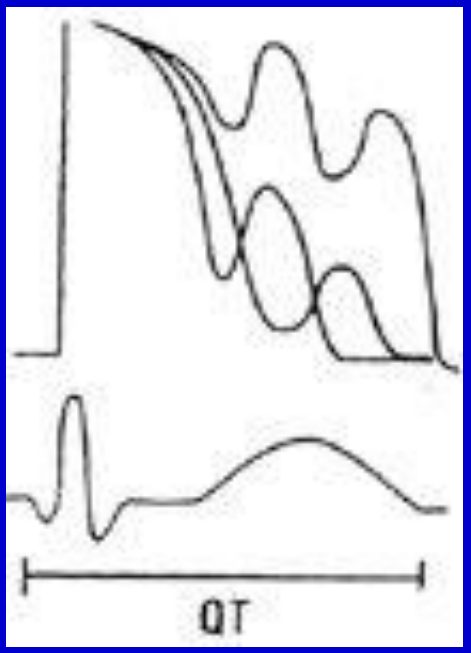
Acquired formes
- electrolyte dysbalance (hypokalemia, hypomagnesemia, hypokalcemia)
- drugs (antiarrhytmic drugs, tricyclic antidepressants, ATB aj.)

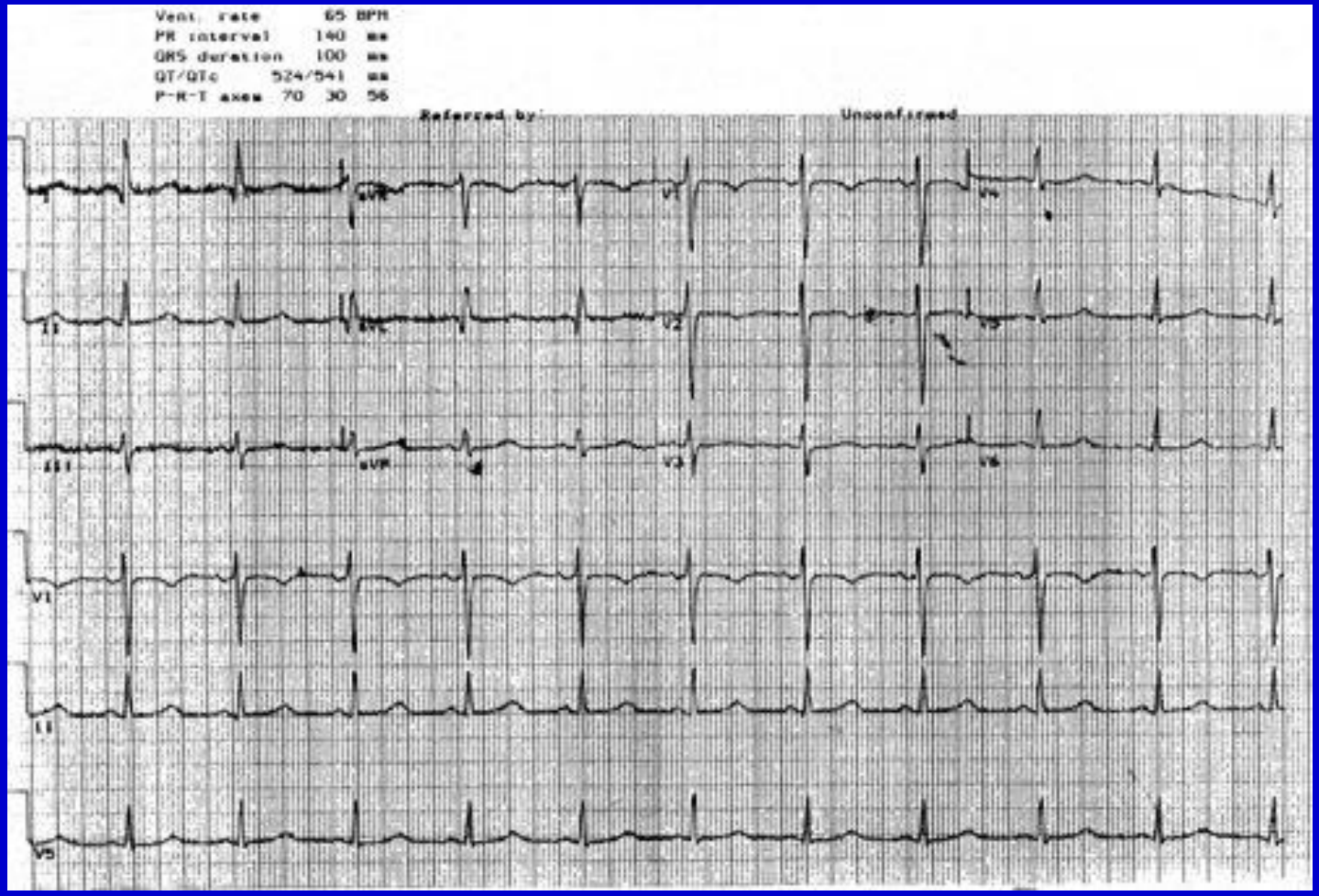


$$QTc = \frac{QT}{\sqrt{RR}}$$



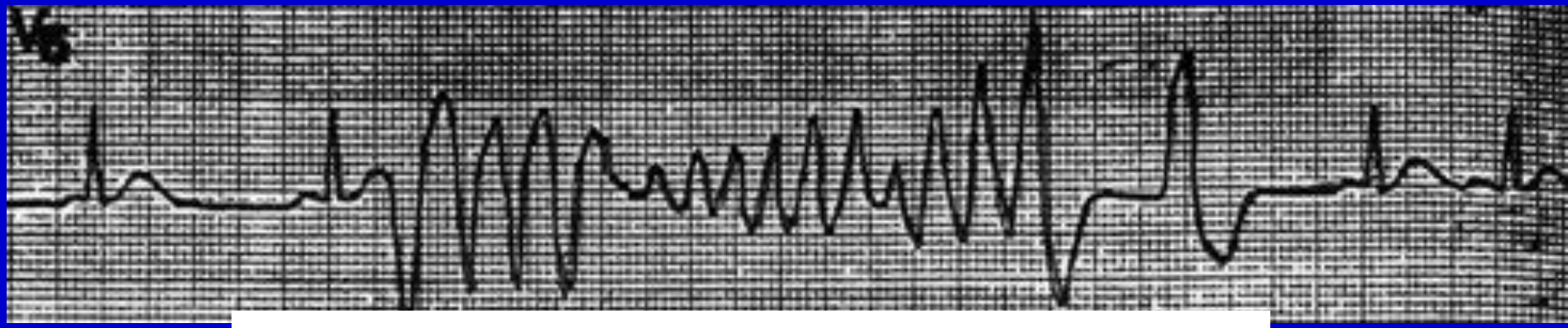


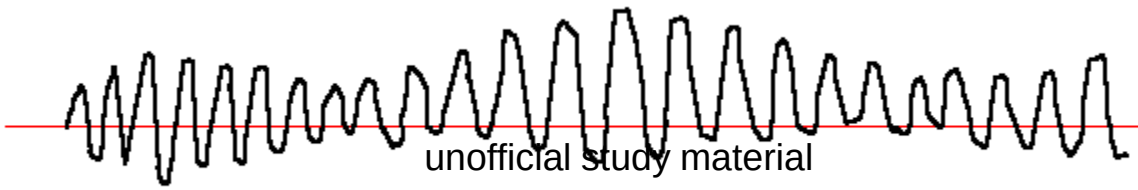




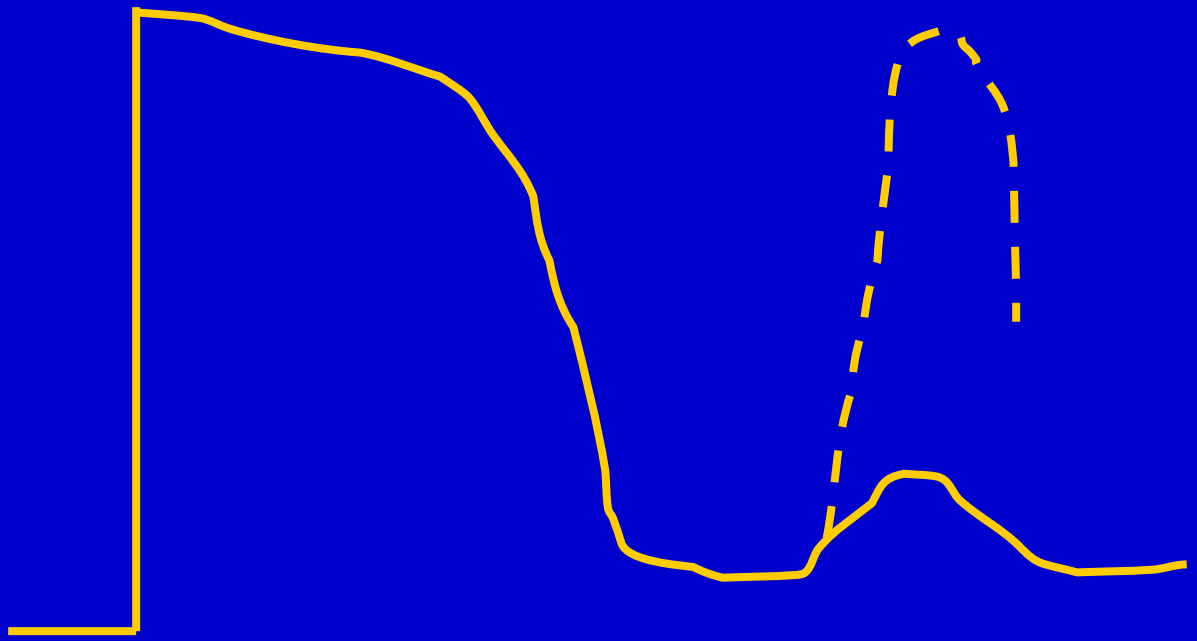
Ventricular tachycardias type „torsades de pointes“, syncopes, danger of sudden death
According to the type of mutation they can occur rather at rest (sodium channel)
or rather during the exercise or stress (adrenergic stimulation)

Mechanisms is linked to early depolarizations „afterpotencials“ (EAD)





2. Delayed afterdepolarization (DAD)
After the repolarization



Vulnerable phase

between phae 3 and 4 of MAP,
sensitivity to the stimuli with low intensity

on ECG declining part of T wave
extrasystole can provoke ventricular tachycardia of fibrillation (phenomenon R/T)

Inherited – ion channels mutations

***Roman-Ward sy*: AD hereditary, more frequent**
***Jervel - Lange-Nielsen sy*: more severe, spojen deafness, AR inheritance**

Over 100 mutations, 5 proteins
Potassium channels – KvLQT1 (KCNQ1) or HERG (α subunits) or β subunits: decreased functions
Sodium channel – SCN5A (mutated also in *Brugada sy*): increased function
$\rightarrow$prolongated repolarization

Treatment of arrhytmias

1. Vegetative nervous system

***stimulation of n. vagus* – increase of parasympatethic tone – treatemnt of SV tachycardia: massage of carotic sinus, Valsalve, pressure on eye bulbes**

***drugs* – sympatolytic drugs (betablockers), sympathomimetic (epinefrin), parasympatolytic drugs (atropin)**

2. Antiarrhytmic drugs

acting on ionic channels

3. Electrical treatment

- defibrillation

- implantable defibrillators (ICD)

- cardiovesion

- cardiostimulation

4. Other treatment

ablation or surgery (e.g. surgery of the accessory pathway)

Antiarhytmic drugs

- Influence sodium or calcium channels
- Influence vegetative nerves

The End