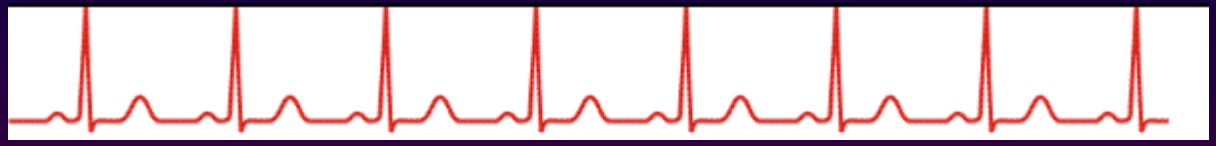
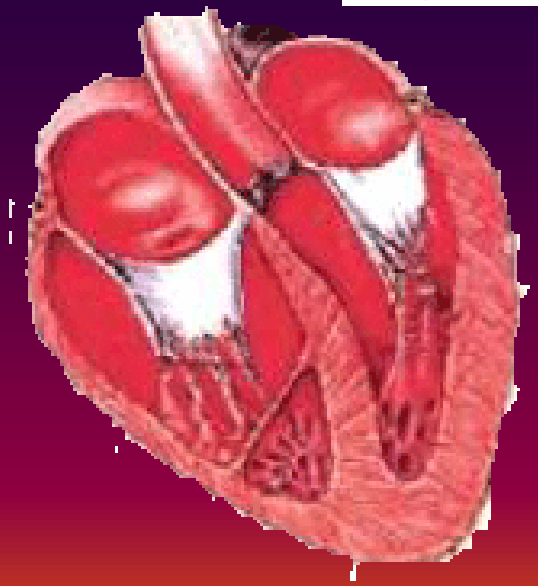
ARRHYTMIAS





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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.

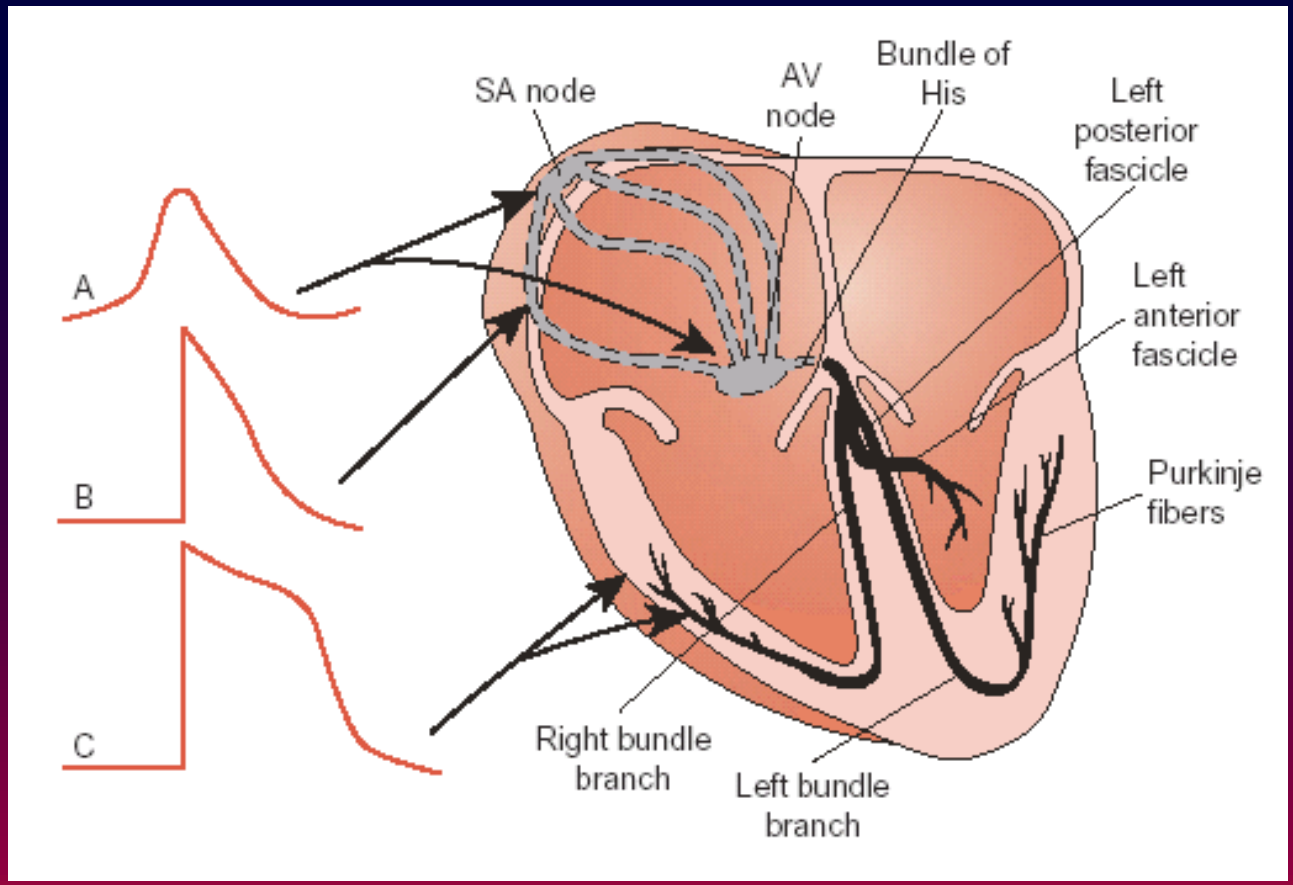
ECG and sometimes longer follow-up might be necessary.

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


SA node
AV node
Bundle of His
Left posterior fascicle
Left anterior fascicle
Purkinje fibers
Right bundle branch
Left bundle branch
A
B
C

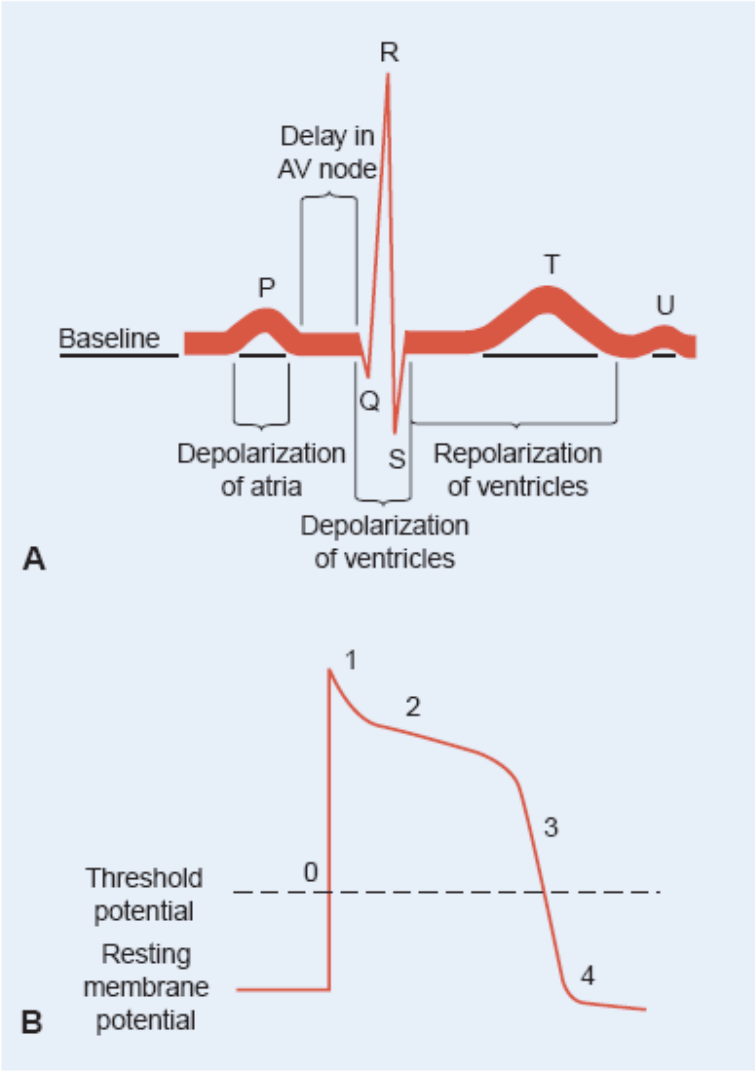
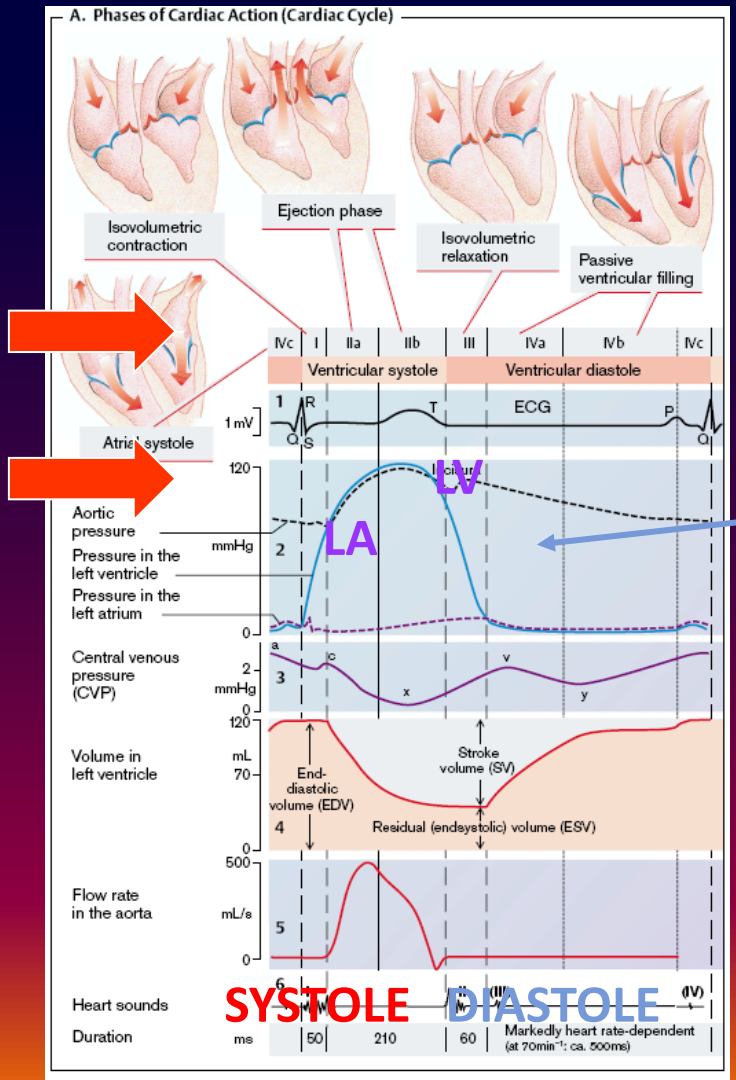


FIGURE 27-3 Relation between (**A**) the electrocardiogram and (**B**) phases of the ventricular action potential.


A. Phases of Cardiac Action (Cardiac Cycle)
Isovolumetric contraction
Ejection phase
Isovolumetric relaxation
Passive ventricular filling
Atrial systole
IVc
I
IIa
IIb
III
IVa
IVb
IVc
Ventricular systole
Ventricular diastole
ECG
1 mV
Aortic pressure
Pressure in the left ventricle
Pressure in the left atrium
mmHg
LV
LA
Central venous pressure (CVP)
Volume in left ventricle
mL
End-diastolic volume (EDV)
Stroke volume (SV)
Residual (endsystolic) volume (ESV)
Flow rate in the aorta
mL/s
Heart sounds
SYSTOLE
DIASTOLE
Duration
ms
50
210
60
Markedly heart rate-dependent
Silbernagl/Lang, Color Atlas of Pathophysiology © 2000 Thieme

Main causes of disturbance of conductive system

1. 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

2. Myocardial damage

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

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

*** inflammation (*myocarditis*)**

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.*)

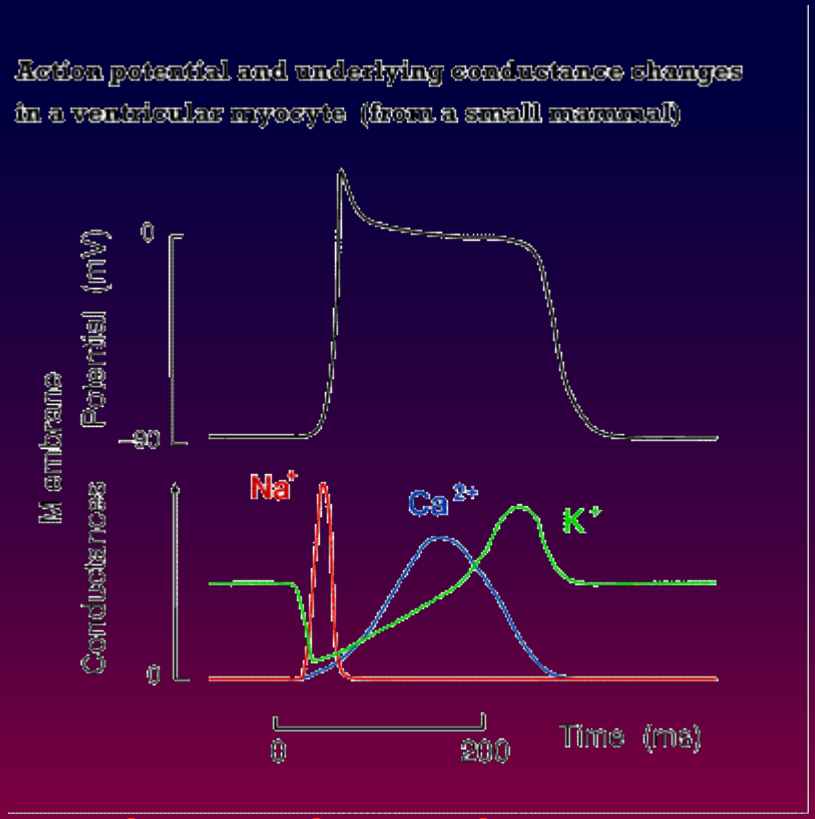
6. genetic causes (*mutation of ionic channels*)

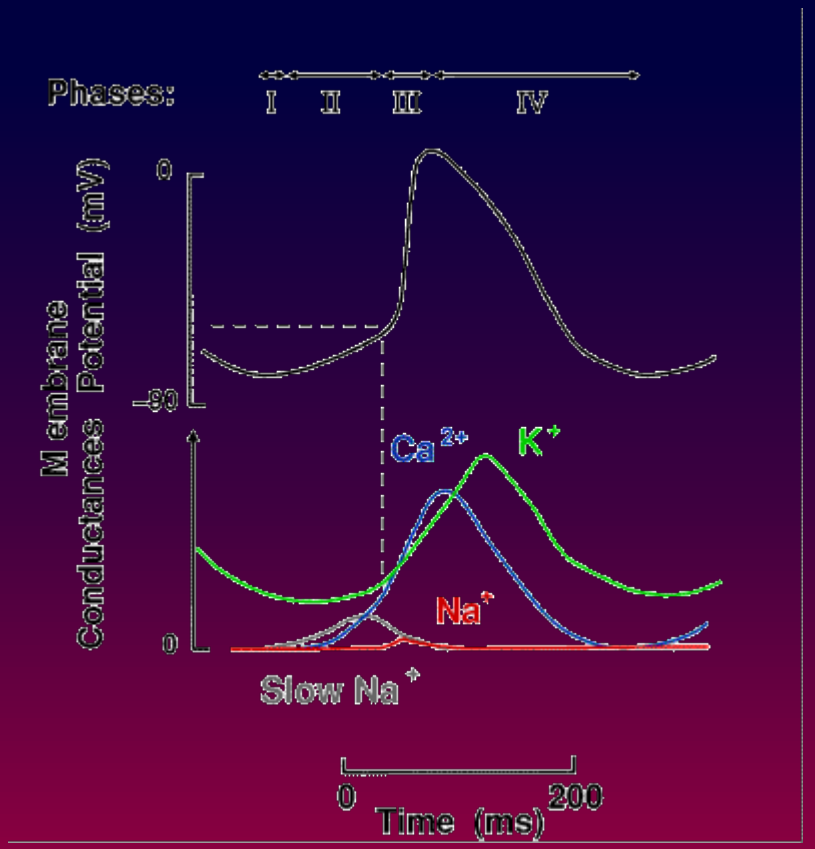
Ion channels

- Sodium channel
- Potassium channels
- Calcium channels

Channel regulation

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





Calcium channels:

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

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

Ion channels pathology

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

Electrical nonhomogenity

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

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

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

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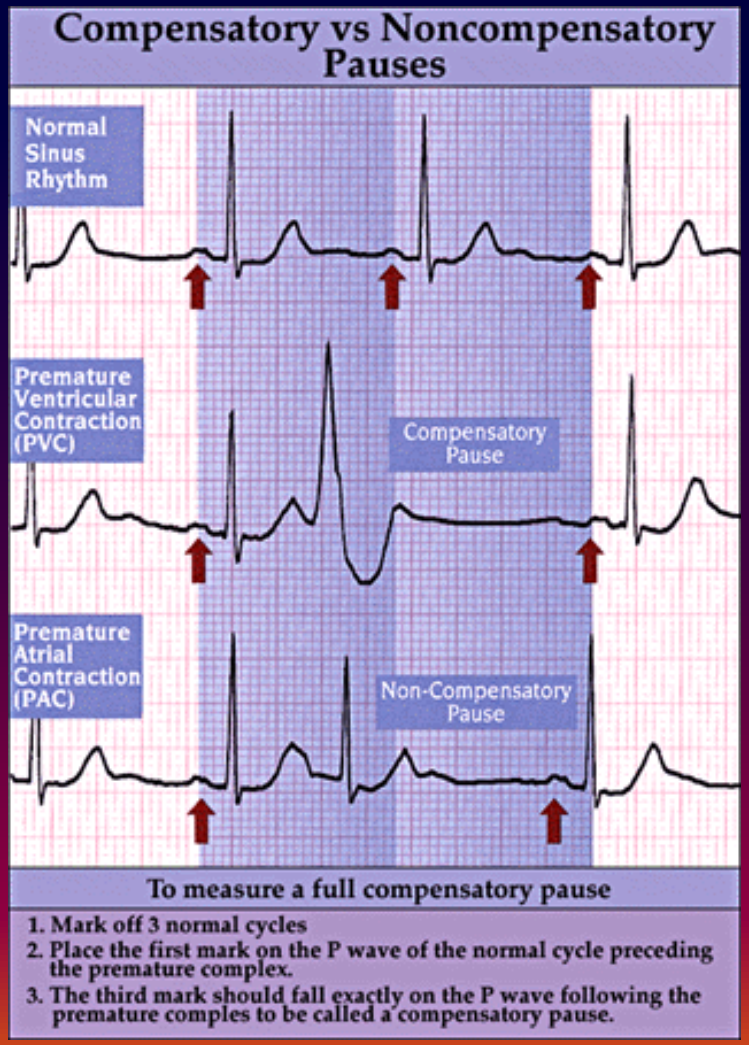
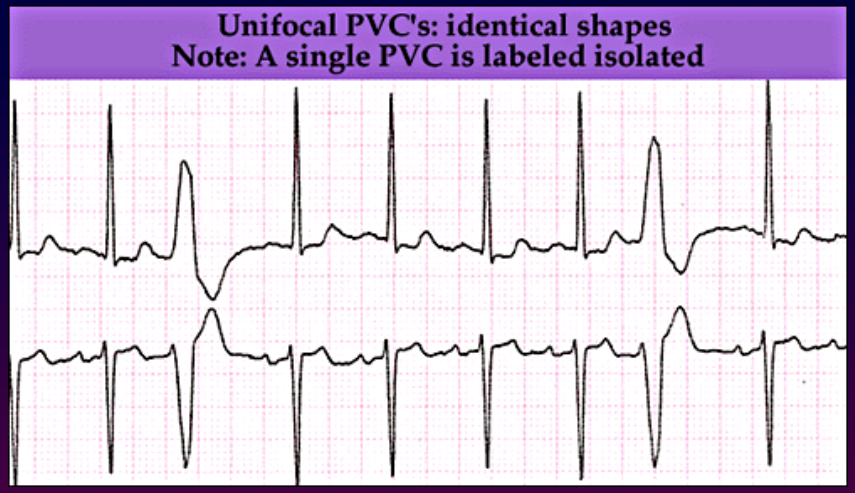
Ventricular extrasystole (VES), premature beat, premature ventricular contraction (PVC)

- 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


Compensatory vs Noncompensatory Pauses
Normal Sinus Rhythm
Premature Ventricular Contraction (PVC)
Compensatory Pause
Premature Atrial Contraction (PAC)
Non-Compensatory Pause
To measure a full compensatory pause
1. Mark off 3 normal cycles
2. Place the first mark on the P wave of the normal cycle preceding the premature complex.
3. The third mark should fall exactly on the P wave following the premature comples to be called a compensatory pause.

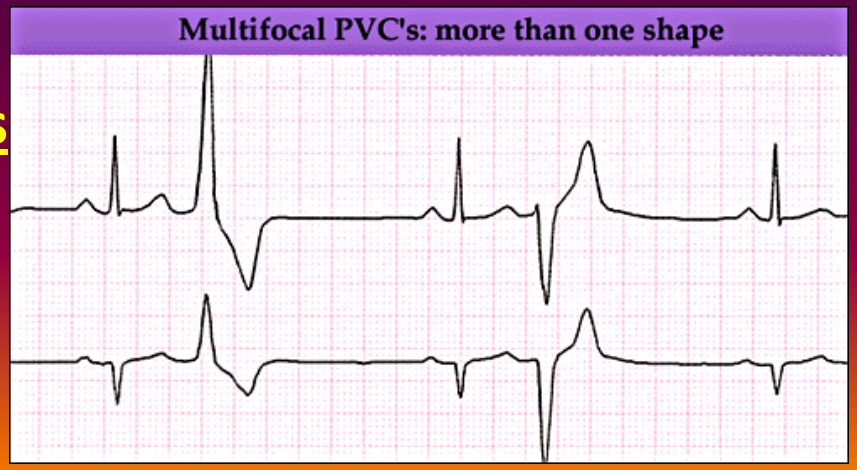
VES

SVES



Spreading from one site – monotopic

Spreading from more sites – polytopic



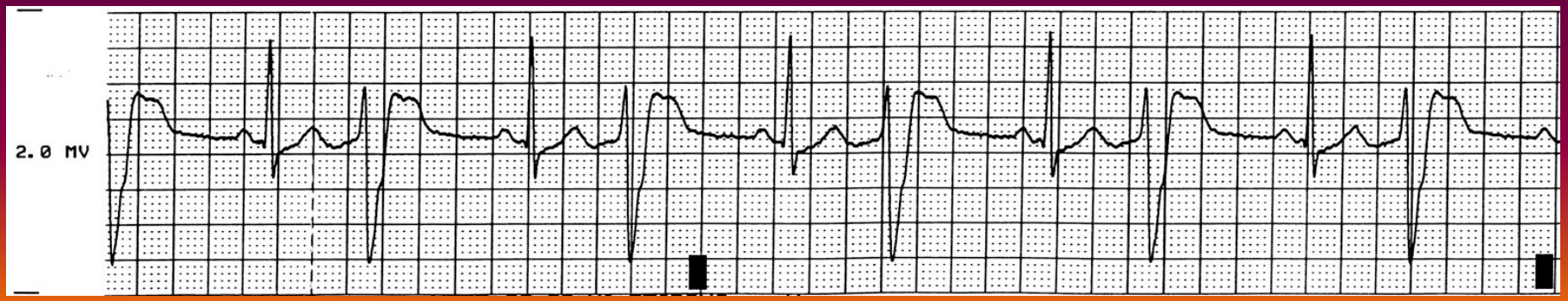
serious are:

- 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

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

SV tachycardia

sometimes in healthy persons

ECG: normal QRS,

changes of P wave and PQ interval

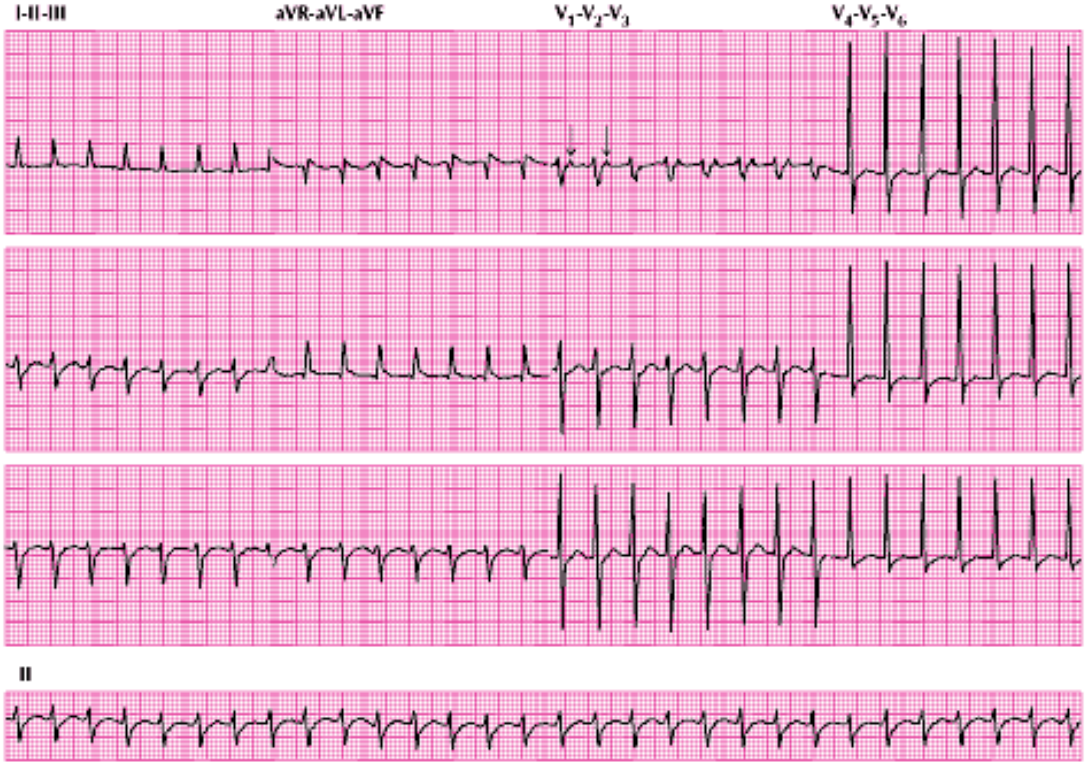
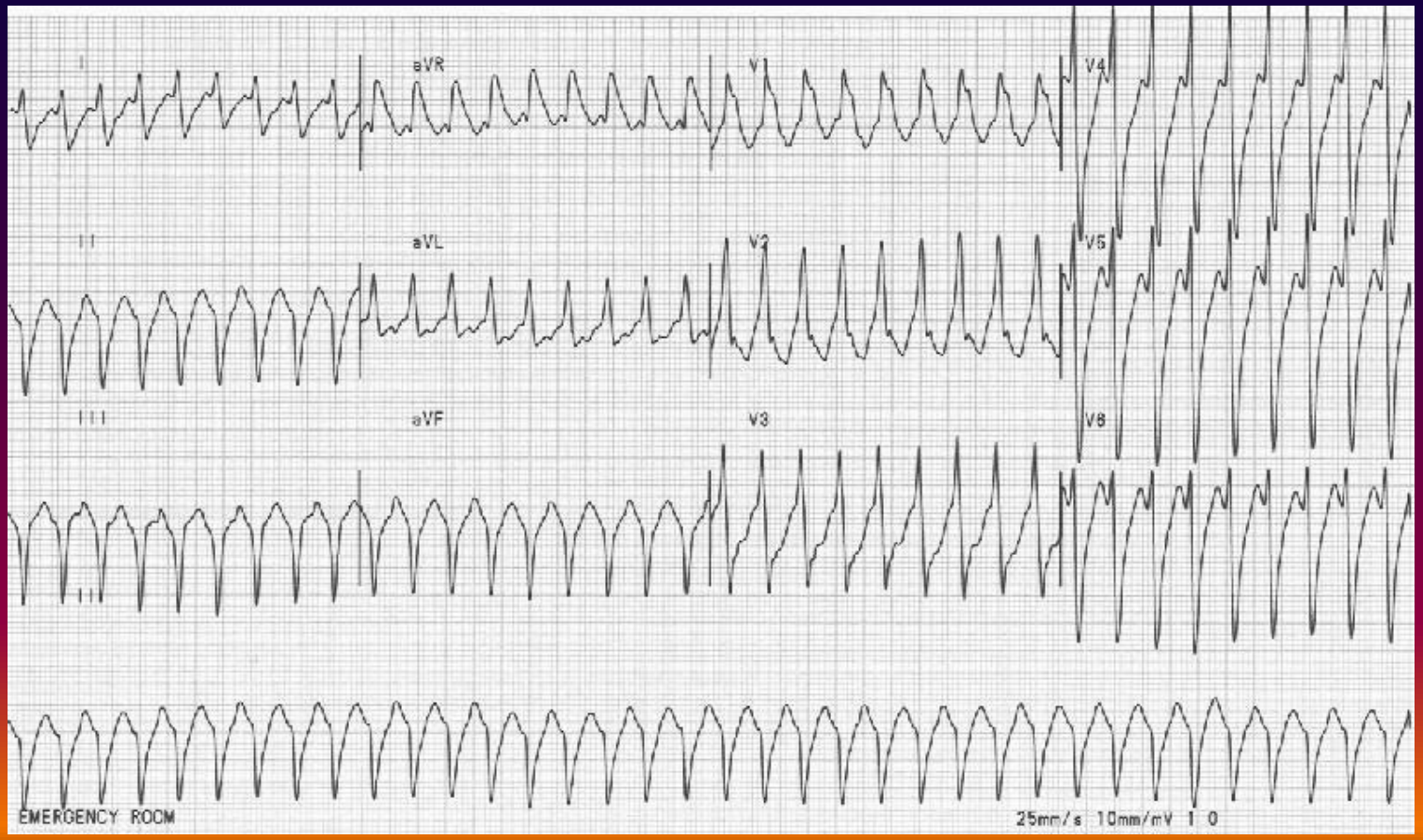


Figure 2. ECG shows supraventricular tachycardia in a 36-year-old woman with frequent episodes of sudden-onset, rapid, and regular heart rate. The ventricular rate is 183 bpm. Note the P waves at the end of the QRS complex (arrows in V_1). Symptoms persisted despite treatment with oral verapamil and 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.

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)

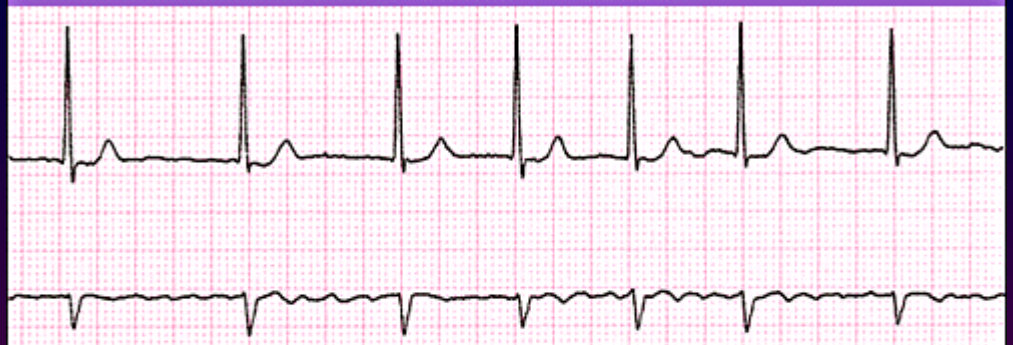
Atrial fibrillation

- absolutely irregular el. activity of atria
  with frequency
  up to >300/min, without efficient contractions

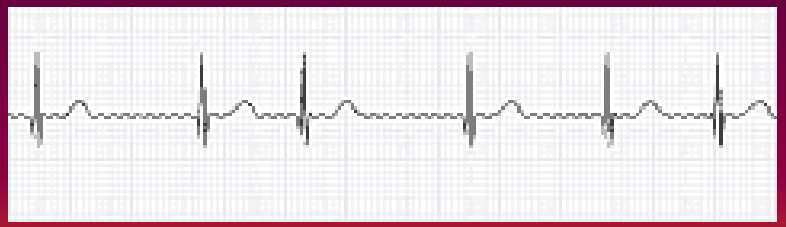
- only some of the impulses are conducted to the ventricles:
  pulse is *absolutely* irregular, the filling of the ventricles is variable
  (pulse deficit can occur)

- ECG: fibrillation waves (*f*) between QRS complexes, QRS complexes have normal shape

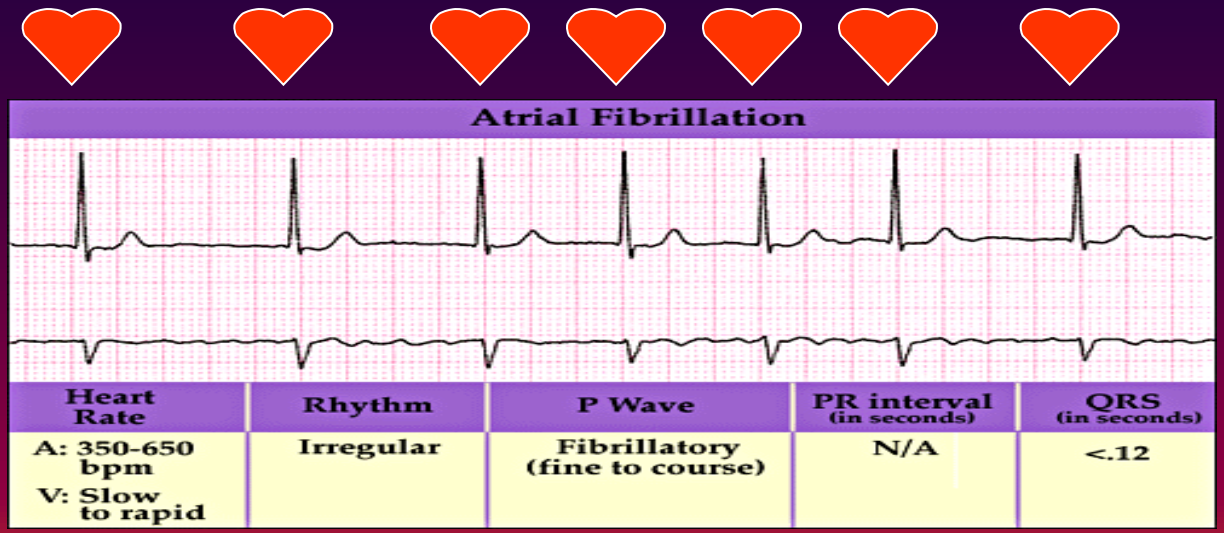
Atrial Fibrillation



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)
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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

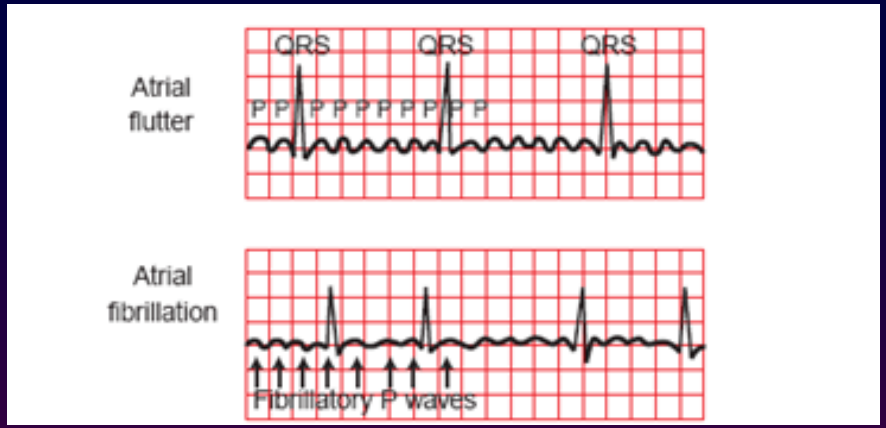

FIGURE 27-10 Electrocardiographic tracings of atrial arrhythmias. Atrial flutter *(first tracing)* is characterized by the atrial flutter (F) waves occurring at a rate of 240 to 450 beats per minute. The ventricular rate remains regular because of the conduction of every sixth atrial contraction. Atrial 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).

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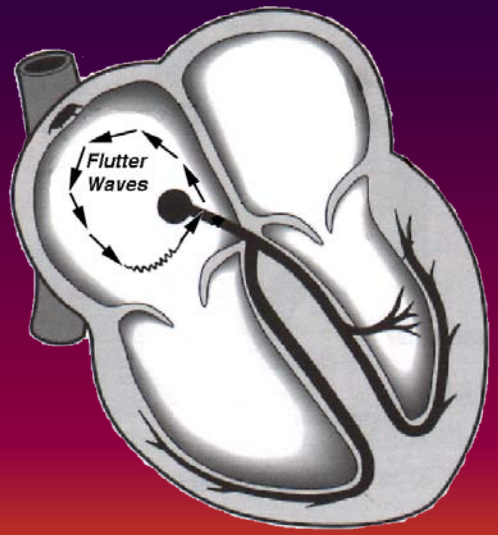
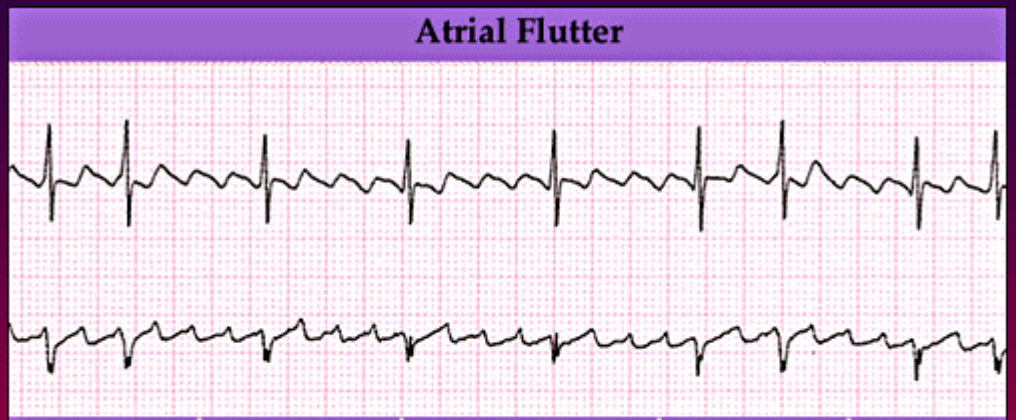
Atrial flutter

less frequent, el. activity in the atria is regular

usually more serious than fibrillation, depending on the resulting HR





Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

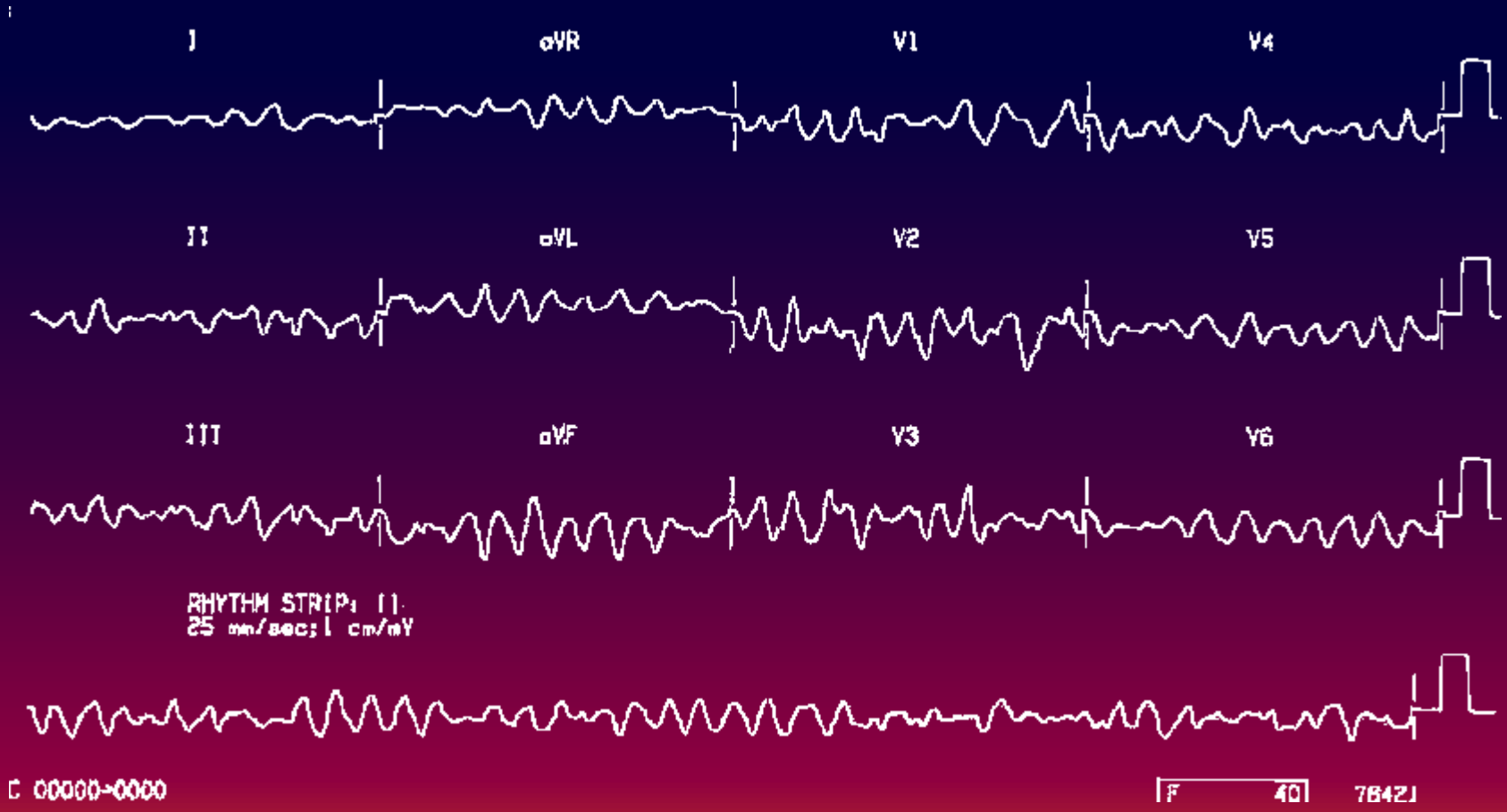
Ventricular fibrillation

acute, life-threatening situation
with complet hemodynamic failure

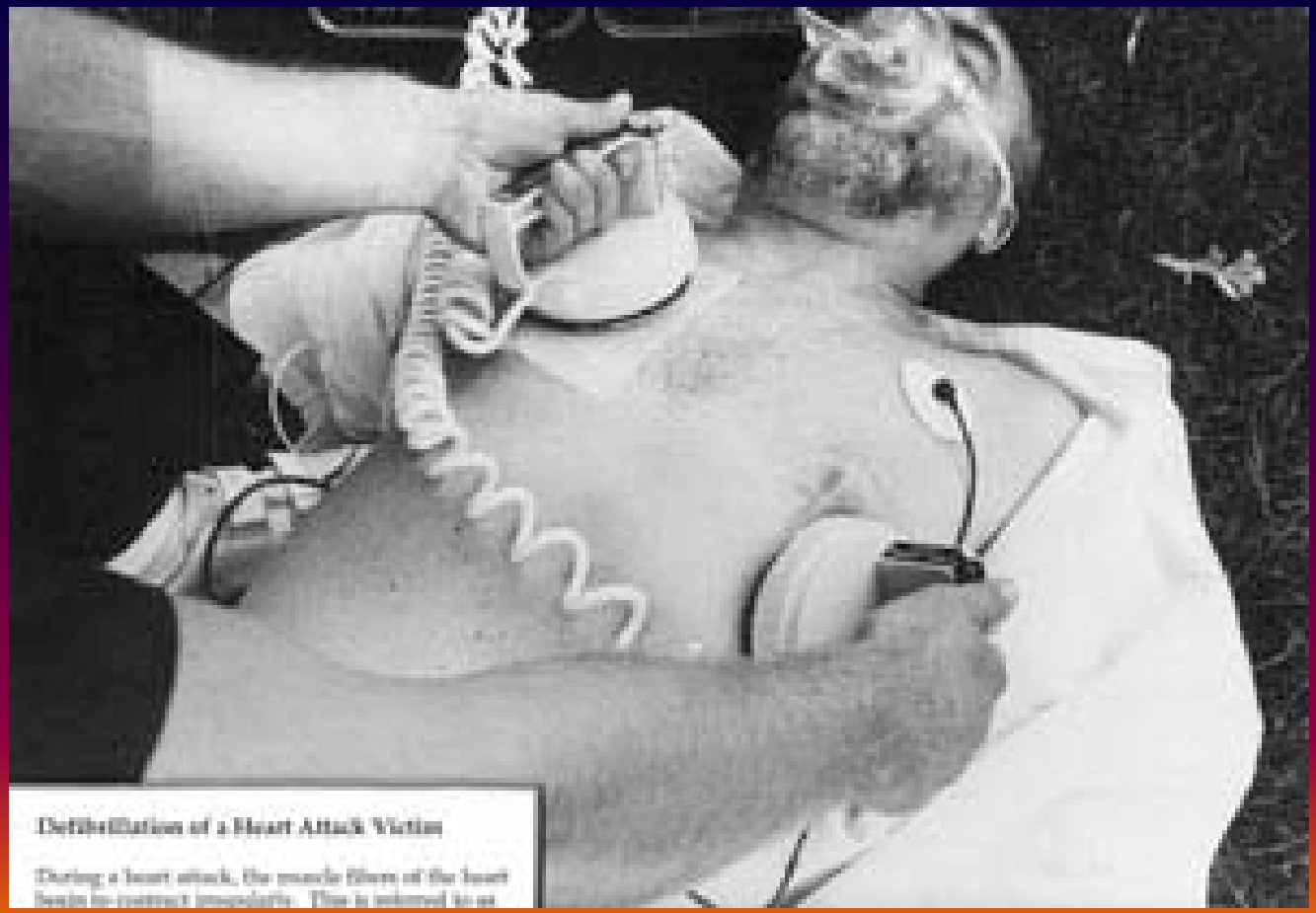
-no cardiac output,
-no pulse,
-unconscioussness,
-reanimation required to save life

frequent cause of death in the early
acute myocardial infarction
cardiomyopathy

defibrillation


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV
C 00000>0000
F 401 7842J

Defibrillation



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Bradyarrhytmias

Sinus bradycardia

vagus

normal: exercise

pathology: acute myocardial infarction
of diaphragmatic wall

cranial hypertension, some infections...

sick sinus syndrome

Sick sinus syndrome

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

bradycardia-tachycardia syndrome

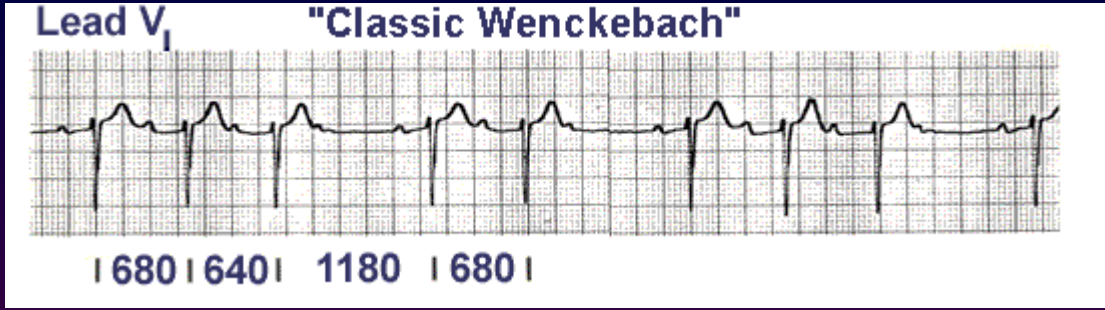
Blocks

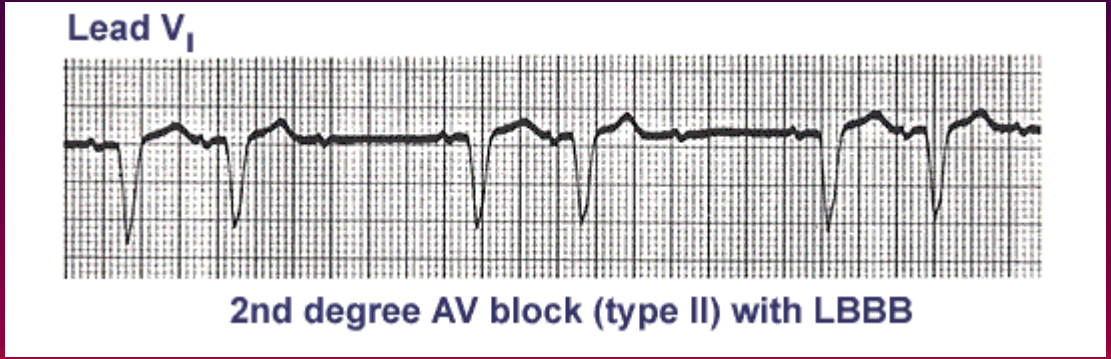
I. slowing, prolongation
II. partial blockade
III. complete blockade

Sinoatrial block

Atrioventricular block

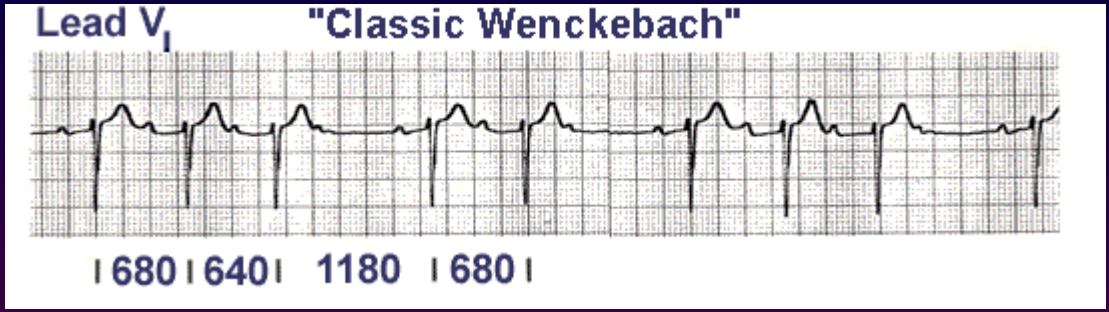
I. degree
II. degree
type Wenckebach (Mobitz I)
type Mobitz (Mobitz II)
III. degree
Adams-Stokes attacks


Lead V1
"Classic Wenckebach"
680
640
1180
680


Lead V1
2nd degree AV block (type II) with LBBB

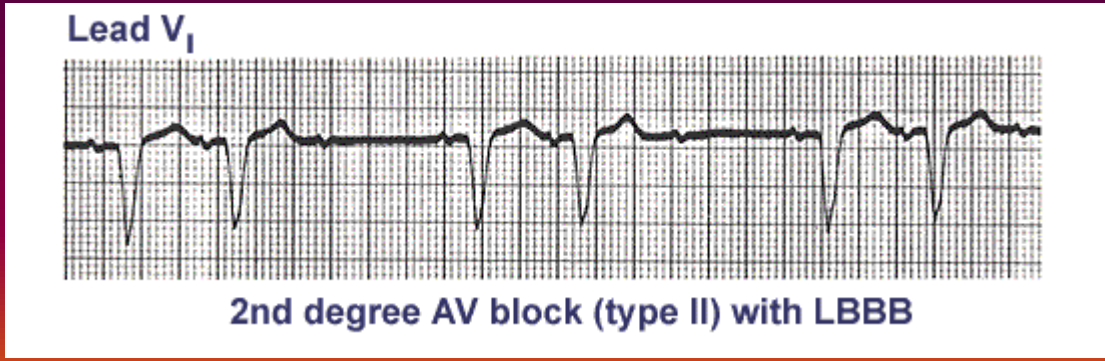
Wenkebach (Mobitz I)

prolongation of PR intervals



Mobitz (Mobitz II)

PR intervals do not change

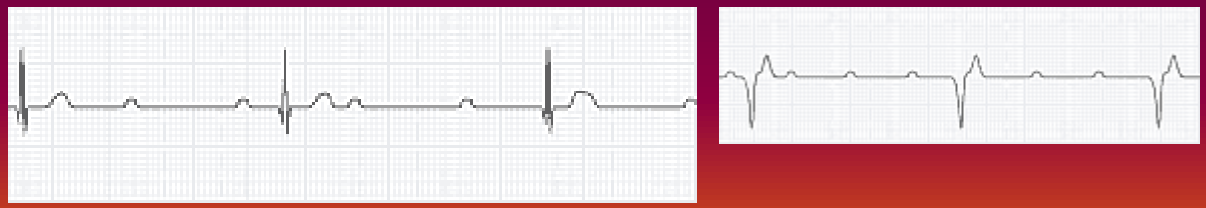


Ratio atria : ventricles (P:QRS)

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



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

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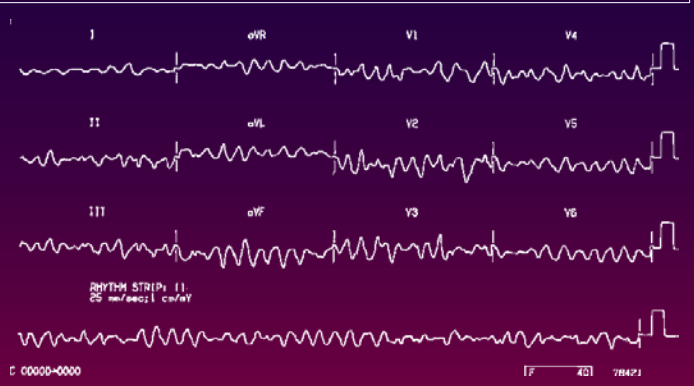
Uncosciousness

- Breathing ?
- Pulse?

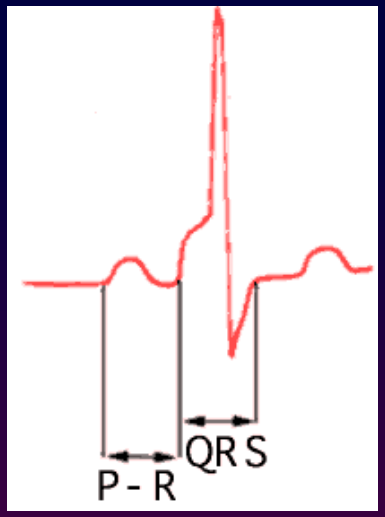
Unconsciousness + no pulse

- Ventricular fibrillation



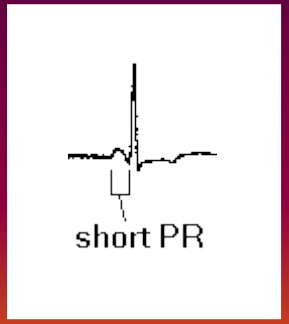
- AV blok III. degree
- Asystoly
- Sick sinus syndrome

Abnormal AV conduct

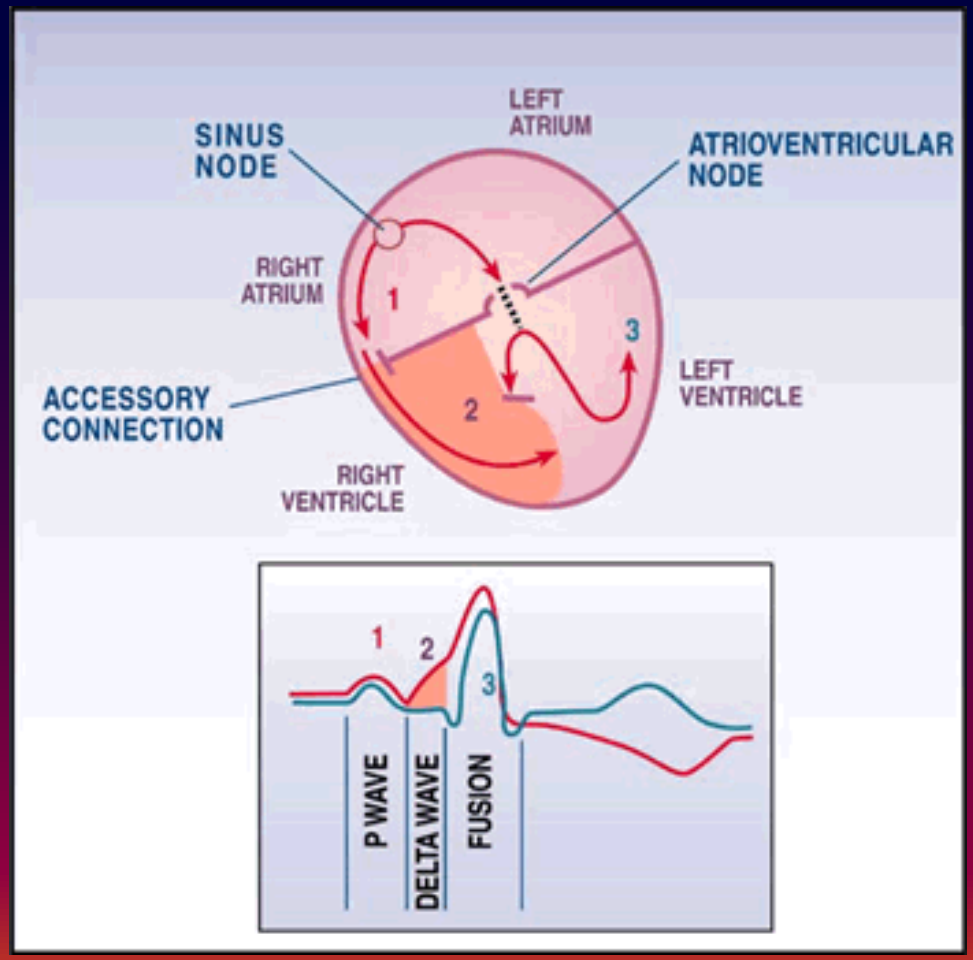


accessory pathways
preexcitation syndromes

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

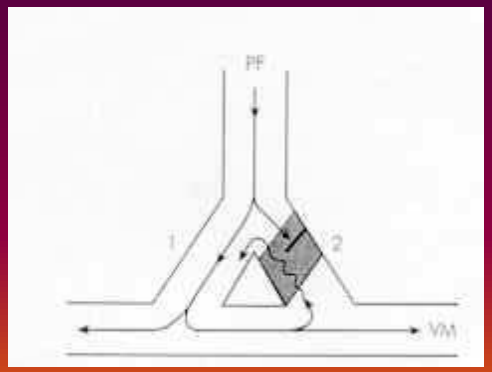

LEFT ATRIUM
SINUS NODE
ATRIOVENTRICULAR NODE
RIGHT ATRIUM
1
3
LEFT VENTRICLE
ACCESSORY CONNECTION
2
RIGHT VENTRICLE
1
2
3
P WAVE
DELTA WAVE
FUSION

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



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)

Mechanisms of arrhytmias

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

ARYTMOGENIC MECHANISMS

* changes in automaticity
* triggered activity
* re-entry

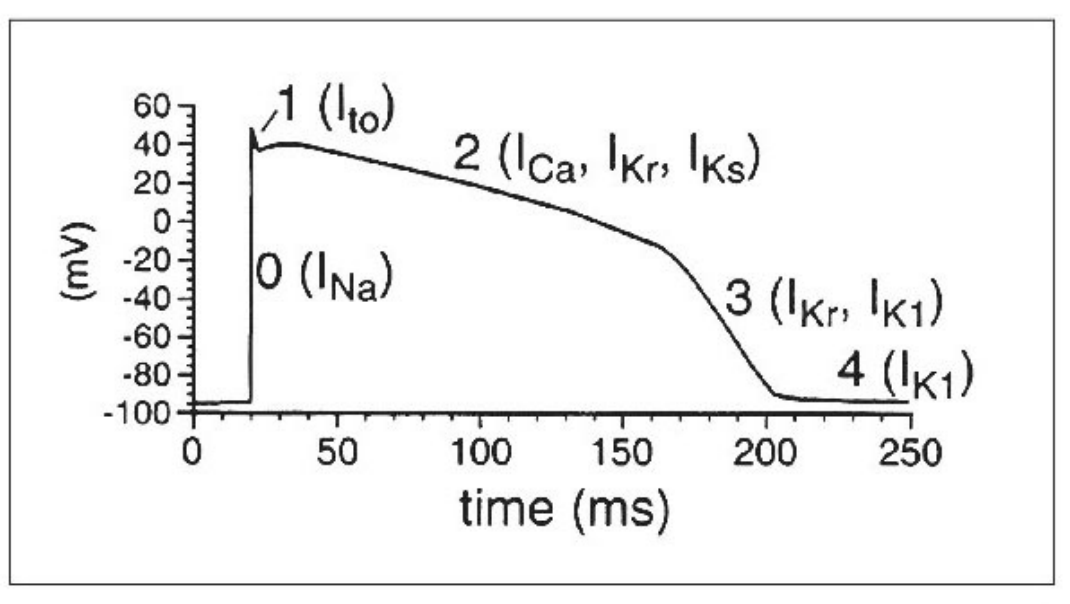
Important parameters in electrical events:

* ***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)



Membrane action potential

I_{Na} – fast sodium current
I_{to} – "transient outward" (potassium from the cell) + chlorides to the cell
I_{ca} – calcium to the cell
I_{K} – potassium from the cell (rapid, slow)
I_{K1} – flow of potassium from the cell

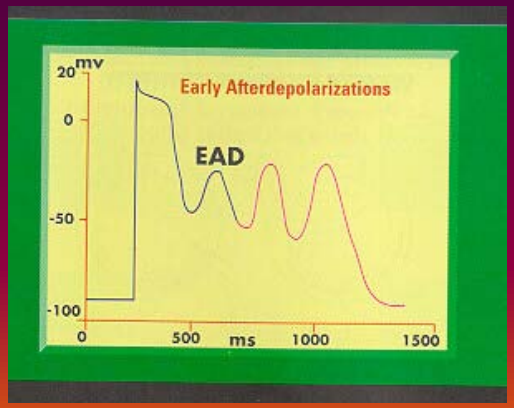
Antiarhytmic drugs

- Influence sodium or calcium channels
- Influence vegetative nerves

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

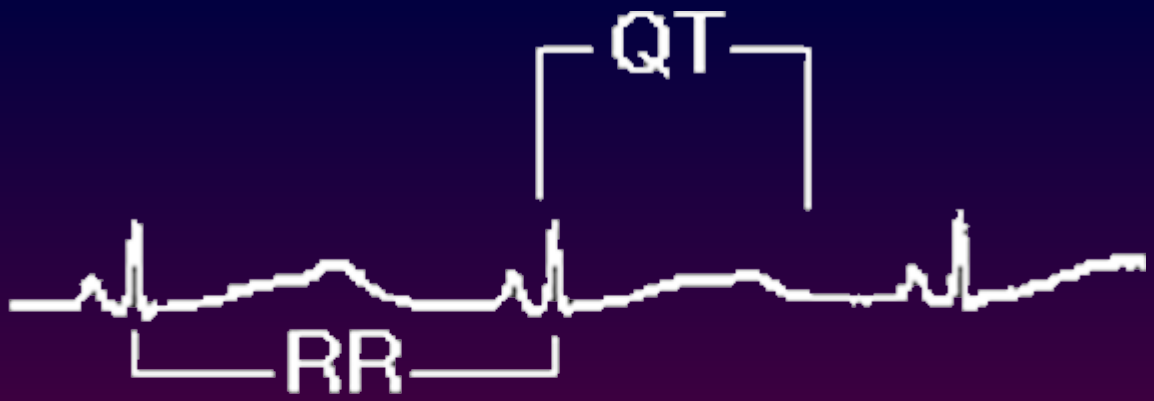
Consequences
-Fast HR (tachyarrhytmia)
-torsade de pointes

Syndrom of long QT

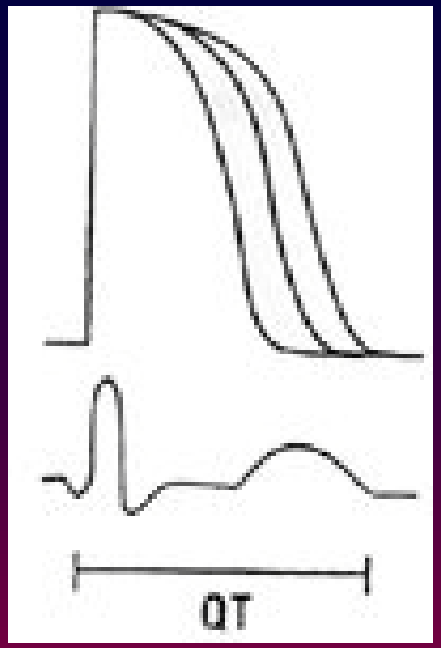
interval QT longer

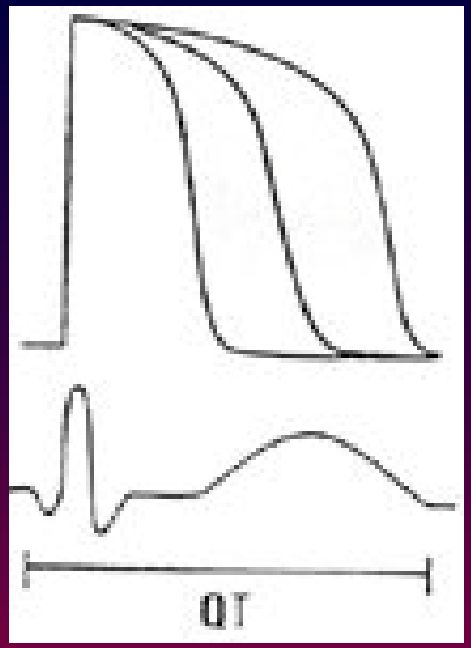
importance: connected to frequent serious ventricular tachyarrhytmias
length and *dispersion*

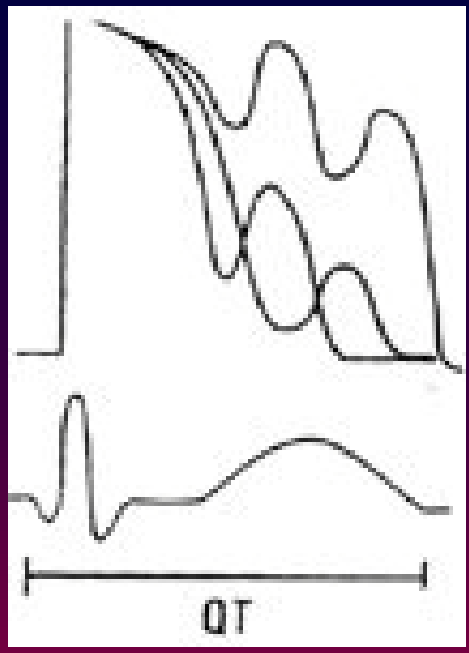
Acquired formes
- electrolyte dysbalance (hypokalemia, hypomagnesemia, hypokalcemia)
- drugs (antiarrhytmic drugs, tricyclic antidepressants, ATB aj.)

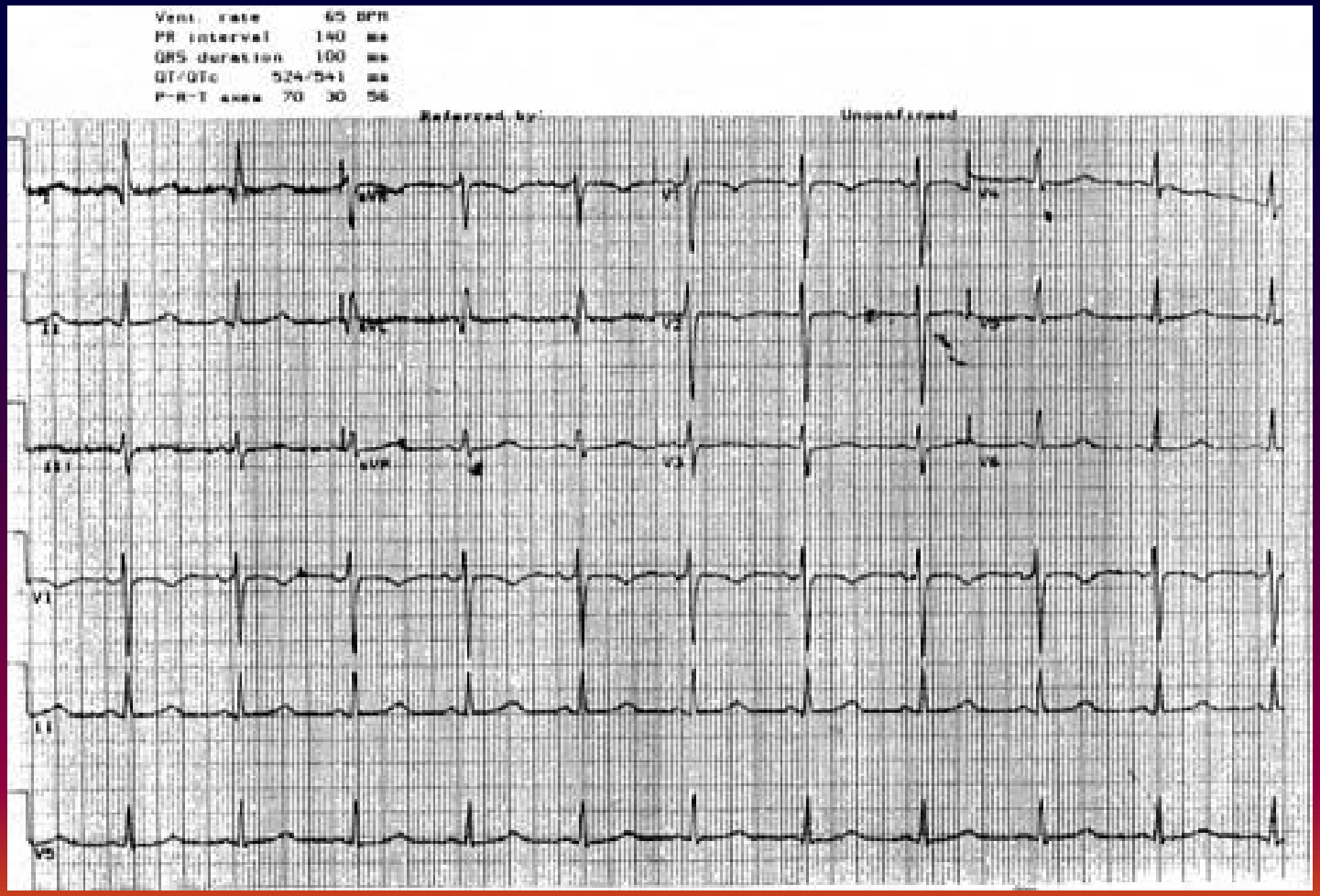


$$QTc = \frac{QT}{\sqrt{RR}}$$


QT

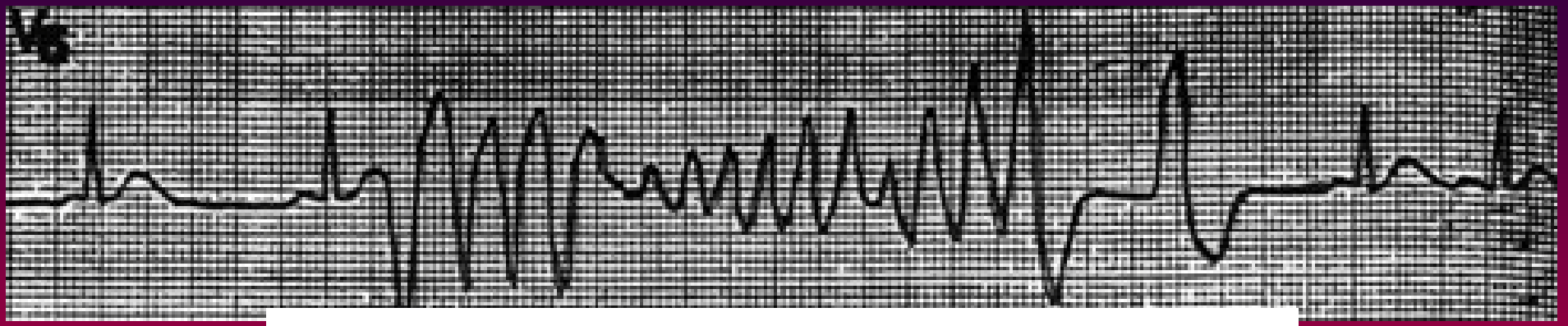

QT

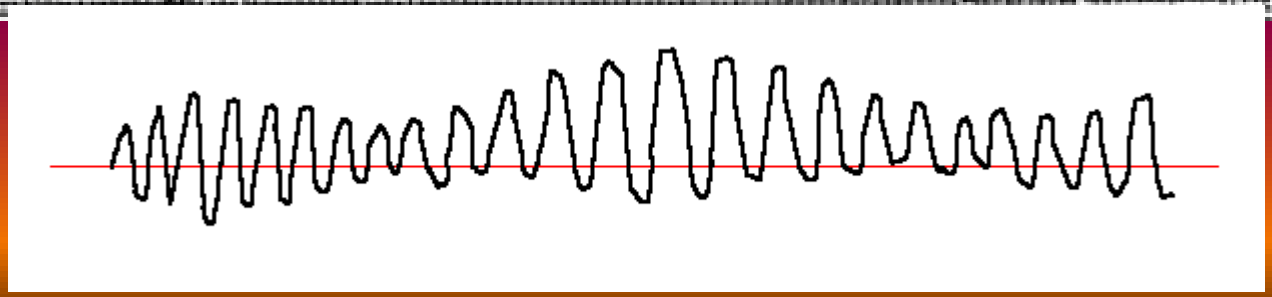

QT


Vent. rate 65 BPM
PR interval 140 ms
QRS duration 100 ms
QT/QTc 524/541 ms
P-R-T axes 70 30 56
Referred by:
Unconfirmed
I
aVR
V1
V4
II
V2
V5
III
aVF
V3
V6
V1
II
V5

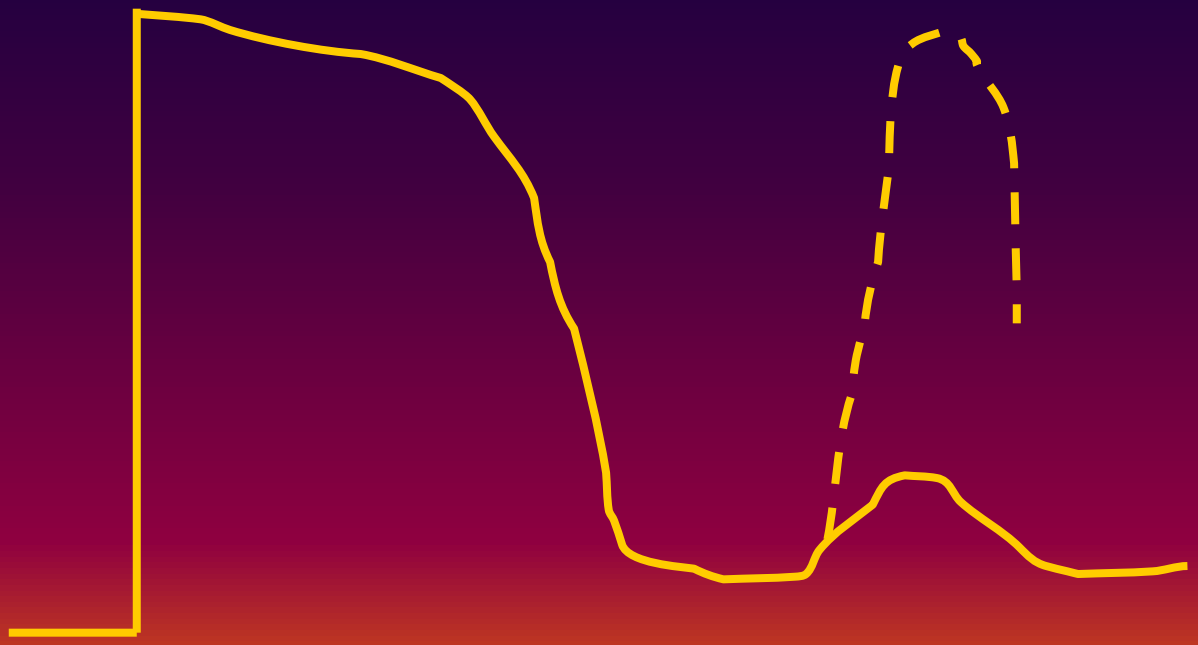
Klasické komorové tachykardie typu „torsades de pointes“, synkopy, nebezpečí náhlé smrti
Podle typu mutace vznikají více v klidu (sodíkový kanál) nebo naopak při námaze (emoci, adrenergní stimulaci)

Mechanismus vzniku souvisí s časnými „afterpotenciály“ (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)

Mutations of ion channels

Roman-Ward sy: AD hereditary, more frequent
Jervel - Lange-Nielsen sy: more serious, deafness, AR hereditary

over 100 mutations of 5 proteins
potassium channels – KvLQT1 (KCNQ1) or HERG (α subunits) or β subunits: decreased function
sodium channel – SCN5A (mutated also in ***Brugada sy***): increased function
$\rightarrow$prolongated repolarization