Department of Pathological Physiology

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Arrhythmia

- Disturbance of heart rhythm: 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-

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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 arrhythmia
2. search for its cause
3. to treat the arrhythmia and its cause
Heart conduction system

- Origin of the impulses
- Impulse conduction
- Hierarchy
FIGURE 27-3 Relation between (A) the electrocardiogram and (B) phases of the ventricular action potential.
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, conduction, excitability and risk of arrhythmias

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, antiarrhythmic 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:
\( I_{Ca-L} \): long-lasting, plateau
\( I_{Ca-T} \): transient – only in pacemaker cells, diast. depolarization
Ion channels pathology

- mutation (hereditary)
- Pathological voltage
- Electrolyte concentration
- Influence of neurotransmitters (including 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 arrhythmias

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

General symptoms of arrhythmias
• electrical: ECG
• hemodynamic
• subjective
Types of arrhythmias

I. electrical events
disturbance in origin of the impulse
disturbance in conduction
combined

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

III. resulting heart rate (effect on hemodynamics, ev. therapy)
bradyarrhythmia
tachyarrhythmia
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, occurs 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 interval
- 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**

**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 complex to be called a compensatory pause.
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

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**Bigeminy**

Unofficial study material
**Tachyarrhythmia**

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

**Sinus tachycardia**

increased activity of sympathetic nerves / decreased 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 V1). 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 fibrillation),
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 electric 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

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: 350-650 bpm V: Slow to rapid</td>
<td>Irregular</td>
<td>Fibrillatory (fine to course)</td>
<td>N/A</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>
THE PATIENTS HAVE ABSOLUTELY IRREGULAR PULSE

![Atrial Fibrillation ECG](image)

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<tr>
<td>V: Slow to rapid</td>
<td></td>
<td></td>
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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

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<tbody>
<tr>
<td>A: 220-430 bpm</td>
<td>Regular or variable</td>
<td>Sawtoothed appearance</td>
<td>N/A</td>
<td>&lt;.12</td>
</tr>
<tr>
<td>V: &lt;300 bpm</td>
<td></td>
<td></td>
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</table>
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
Defibrillation
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Bradyarrhythmias

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
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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Uncossciousness

- Breathing?
- Pulse?
Unconsciousness + no pulse

- Ventricular fibrillation
- AV block 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 arrhythmias
(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
REENTRY

main cause of tachyarrhythmias

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

ischemia, fibrosis
typically accessory pathways
Treatment of arrhythmias

1. **Vegetative nervous system**
   *stimulation of n. vagus* – increase of parasympathetic tone –
   treatment of SV tachycardia: massage of carotic sinus, Valsalve,
   pressure on eye bulb
   *drugs* – sympatholytic drugs (betablockers), sympathomimetic (epinefrin),
   parasympatholytic drugs (atropin)

2. **Antiarrhythmic drugs**
   acting on ionic channels

3. **Electrical** treatment
   - defibrillation
   - implantable defibrillators (ICD)
   - cardioversion
   - cardiostimulation

4. **Other** treatment
   ablation or surgery (e.g. surgery of the accessory pathway)
Mechanisms of arrhythmias

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

ARYTMOCGENIC MECHANISMS

* changes in automaticity
* triggered activity
* re-entry
Important parameters in electrical events:

* **excitability**: capacity of cells to respond to the stimulus of certain intensity (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 impulses to the neighbouring cells amplitude, start of the MAP, cellular junctions, size, shape of the cells

* **refractority**: 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
Antiarhythmic 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 (tachyarrhythmia)
- torsade de pointes
Syndrom of long QT

interval QT longer

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

Acquired formes
- electrolyte dysbalance (hypokalemia, hypomagnesemia, hypokalcemia)
- drugs (antiarrhythmic drugs, tricyclic antidepressants, ATB aj.)
QTc = \frac{QT}{\sqrt{RR}}
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

→ prolonged repolarization