

Supraventricular tachyarrhythmias (SVT)

Appropriate cardiac output is determined by:

- ♣ heart rate
- ♣ synergy of myocardial contraction

The both are controlled by the conducting system of the heart.

SVT

- disorders of the heart rhythm (brady- and tachyarrhythmias)
- disorders of the synergy of the contraction (branch blocks)

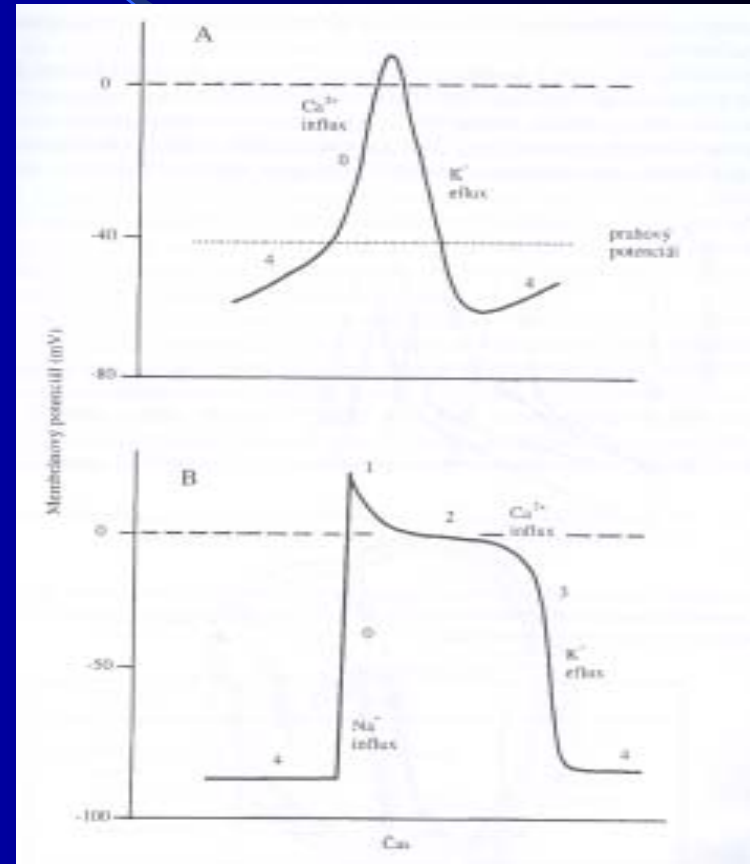
bradycardia $<60/\text{min}$

tachycardia $>100/\text{min}$

Conducting system of the heart

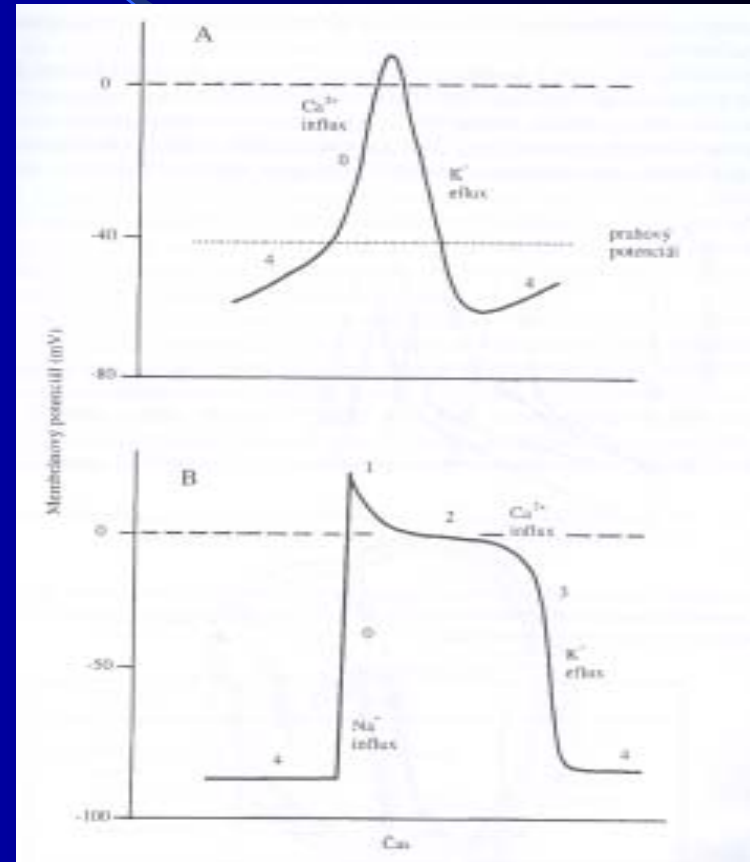
SA node

- physiological pacemaker located in the right atrium (near the opening of the superior vena cava).
- comprises cardiomyocytes with lower resting membrane potential, capable of spontaneous depolarization leading to threshold potential



The spontaneous depolarization leads to automatic repetitive generation of depolarization waves spreading across the myocardium and causing muscle contraction.

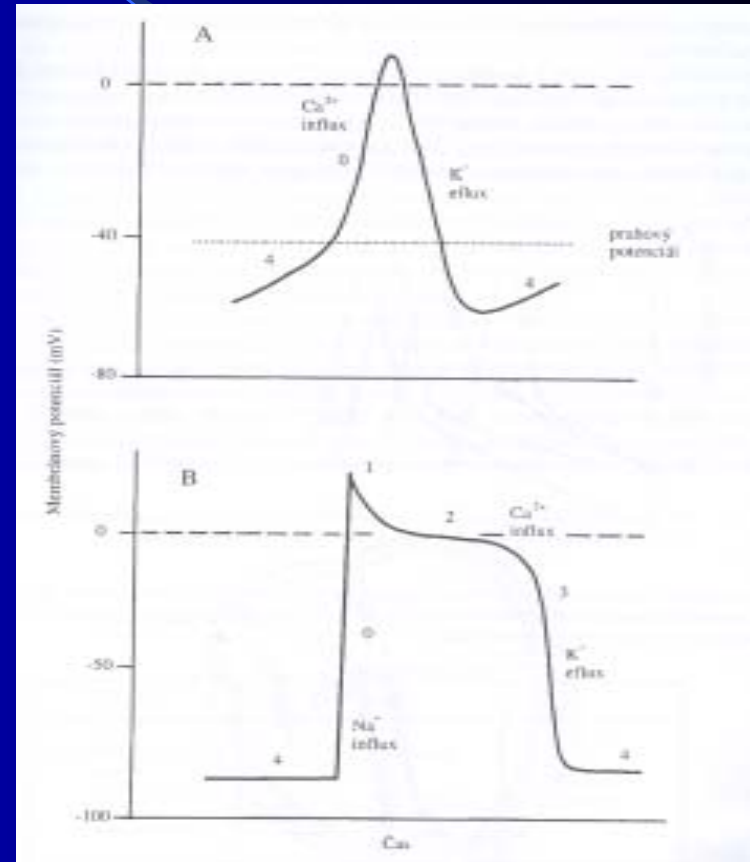
- regulated by sympathetic and parasympathetic nerve fibres



AV node – myocytes capable of independent depolarization (similar to SA node) – „reserve (standby) pacemaker“

Conduction of the impulse:

Generation of the impulse in SA node – conduction across the atria (0.1s) – AV node (0.1s) – bundle of His – Tawar branches – Purkinje fibres



Possible pharmacological actions on the heart rhythm

- effect on the balance of the autonomic nervous system: sympathomimetics, parasympatholytics – atropine; sympatholytics – beta-blockers)
- direct influence on cardiomyocytes (conducting system): chronotropic, dromotropic, bathmotropic drugs (antiarrhythmics, sympathomimetics, parasympatholytics – atropine; sympatholytics – beta-blockers)

General haemodynamic and cardiac consequences of tachycardias/tachyarrhythmias

- shortening of the period of filling of ventricles at diastole (decrease of preload) – decrease of cardiac output – cardiac decompensation (atrial fibrillation – potentiation of the lower preload due to absence of the atrial contribution)
- shortening of the period of the diastolic perfusion of the myocardium – myocardial ischaemia – deterioration of the arrhythmogenic field

Classification of tachyarrhythmias

- ventricular (arising from the distal parts of the conducting system – distal of AV node)
- supraventricular (arising from the proximal parts of the conducting system – above the AV node, incl.)

Mechanisms of genesis of tachyarrhythmias

1/ Reentry

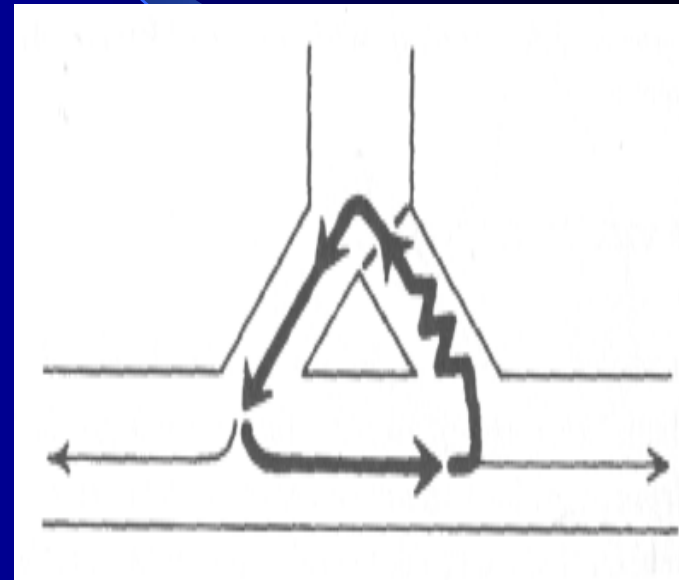
2/ Triggered activity

3/ Increased automaticity

1/ Reentry

pathological reexcitant loop
generated on the basis of macro-
or microreentry circuit

- functional - ischaemia
- morphological — wound, accessory pathways



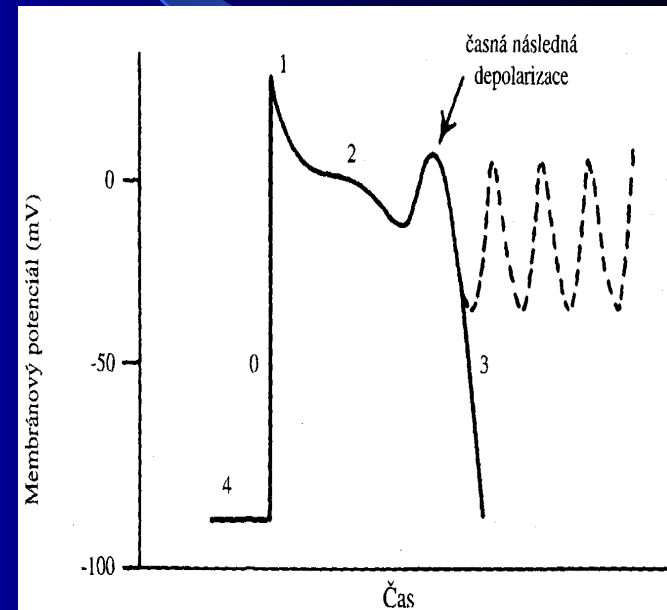
2/ Triggered activity

instability of the membrane potential of a group of cardiomyocytes

- early afterdepolarization
- delayed afterdepolarization

3/ Increased automaticity

(hypoxia, sympathetic, hyperthyreosis)



Supraventricular tachycardias / tachyarrhythmias

- 1/ Sinus tachycardia
- 2/ Atrial tachyarrhythmia
- 3/ AV junctional tachyarrhythmia
- 4/ AV reentry tachyarrhythmia
- 5/ Atrial flutter
- 6/ Atrial fibrillation

1/ Sinus tachycardia

- influence of the sympathetic/parasympathetic nerves
- physical activity, psychological stress, vagal depression, metabolic influences – hyperthyreosis, hypoxia
- RR variability *vs* respiratory arrhythmia

2/ Atrial tachyarrhythmias

- **A/ Non-paroxysmal – ectopic** (increased automaticity or triggered activity)
- **B/ Paroxysmal** (reentry)
- **C/ Multifocal atrial tachycardia** (wandering pacemaker) – different morphology of P waves – need not be regular (diff. dg. atrial fibrillation)

Aetiology:

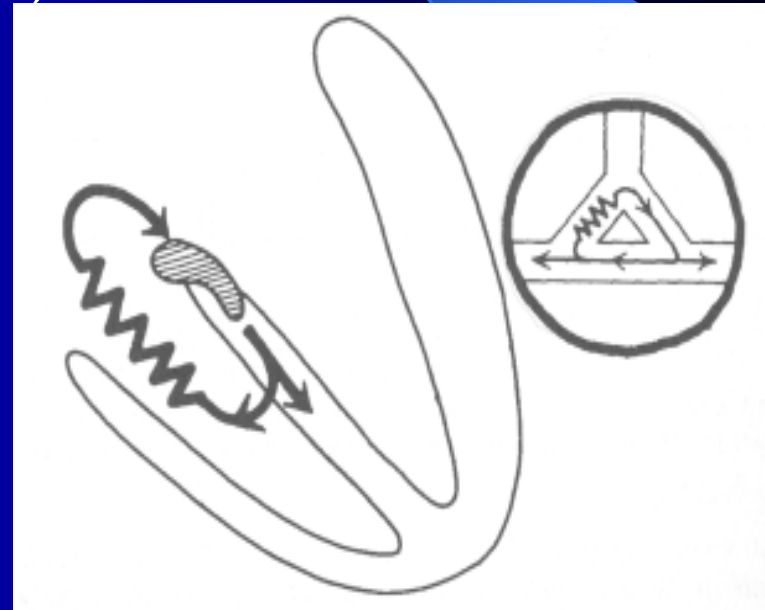
ischaemic heart disease, valvular diseases, pulmonary embolization, cor pulmonale

3/ AV junctional tachyarrhythmia

- **paroxysmal (reentry)**
- **non-paroxysmal (increased automaticity)**

4/ AV reentry tachyarrhythmia

accessory AV bundle (Kent`s)



5/ Atrial flutter

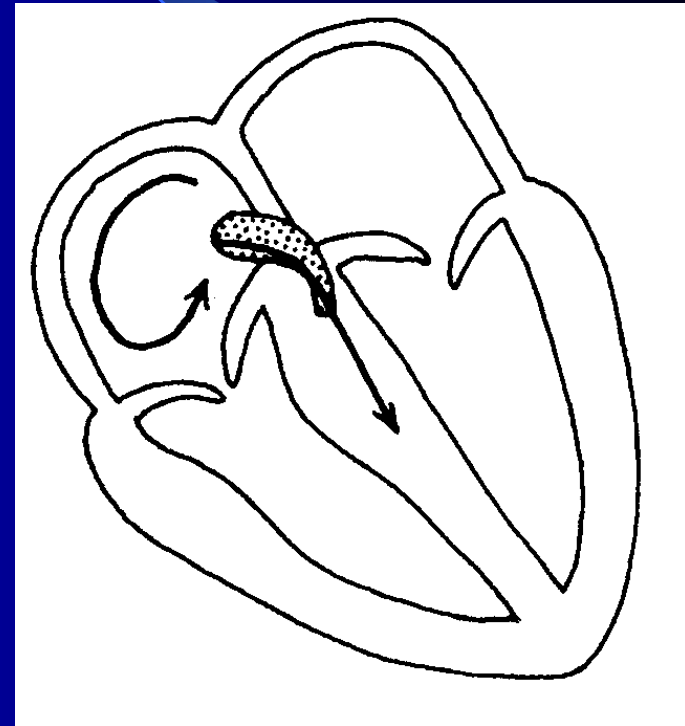
- very fast and regular activity of the atria
- impulse circles in a macro-reentry circuit of the right atrium with the subsequent activation of the left atrium

ECG: regular flutter waves (F); rate 250-350/min; conduction to ventricles usually blocked 2-4:1; usually a regular block; ventricular rate regular around 150/min.

Every regular SVT with a rate of 150/min is suspicious to be atrial flutter (blok 2:1 – hidden in QRS complexes)

5/ Atrial flutter

- **typical** (type I) – macro-reentry circuit in the right atrium (isthmus between the inferior vena cava and the three valves)
- **atypical** (type II) – macro-reentry circuit anywhere in the right or left atrium (after surgical treatment of inborn errors of the heart)



5/ Atrial flutter

Deblocked flutter

life-threatening state due to haemodynamic collapse

- existence of accessory pathways that do not slow down the conduction
- acceleration of conduction in AV junction (quinidine)

5/ Atrial flutter - therapy

- **1/ pharmacological:** amiodarone, verapamil, propafenone, used only in haemodynamically stable patients, when electric cardioversion cannot be performed
- **2/ electric cardioversion** (synchronic discharge of 50-200 J)
- **3/ atrial cardiostimulation** (overdriving)

Long-term therapy:

1/ pharmacological

2/ catheterization-mediated radiofrequency-response ablation

5/ Atrial flutter – case report

A 55-year-old patient (without a personal history) was admitted for hospitalization for suddenly developed dyspnoea provoked by physical activity.

Objective finding:

eupnoea at rest, auscultation of the lungs without pathol. findings, pulse 150/min, no murmur on the chest, boundary filling of jugular veins.

ECG: SVT 150/min, without focal ischaemic changes

Posuv 25 mm/s
Amplituda 10 mm/mV
Filtry 0.2 s, 50 Hz, Fuzzy +
Tepová frekv. 157 [1/min]

Poznámka:





Differential diagnosis:

- 1/ left-ventricle decompensation at SVT
- 2/ acute coronary syndrome
- 3/ pulmonary embolization

Echocardiography: sings of pulmonary hypertension, tricuspid regurgitation, left ventricle without kinetic disorders, with good systolic function (ejection fraction)

Differential diagnosis:

suspicious pulmonary embolization

X-ray of the chest: atelectases in the base of the right lung
without signs of congestion

Differential diagnosis:

suspicious pulmonary embolization

Lab tests: Troponin I 0.2

CPK normal

CK-MB negative

blood count normal

D-dimers 1230 $\mu\text{g/l}$ (significantly positive)

Differential diagnosis:

suspicious pulmonary embolization

Spiral angioCT:

embolus in the lobar bronchial branch for the right inferior lobe

Diagnosis:

Pulmonary embolization

Therapy:

- 1/ Anticoagulation (heparin i.v.)
- 2/ Volumexpansion (increase of preload of the right ventricle)
- 3/ Electric cardioversion (50-100-150 J to restore the sinus rhythm)

- EKG (1), 11.2.2004, 7:51 +00:00

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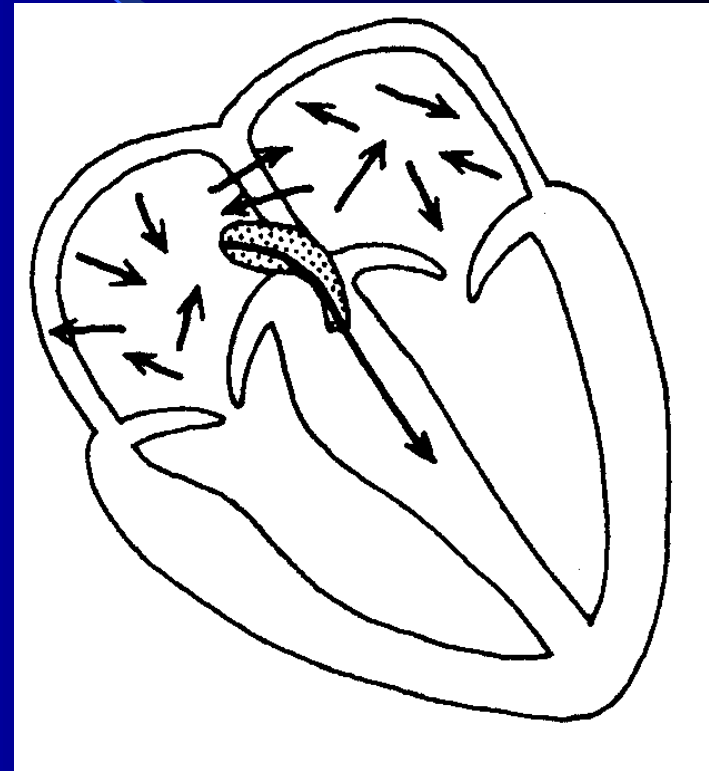
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Tepová frekv. 88 [1/min]

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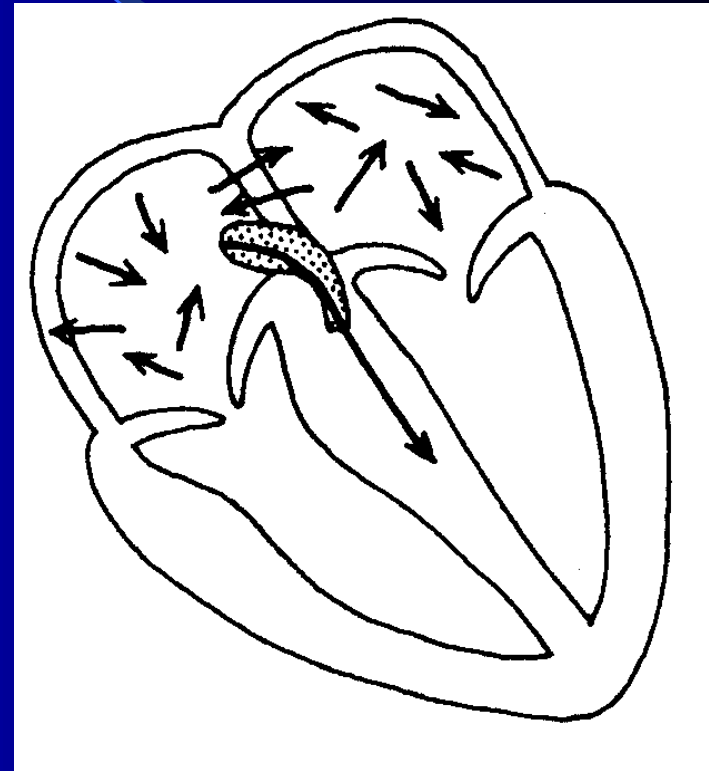
6/ Atrial fibrillation (AF)

- very frequent tachyarrhythmia
- incidence increases with age
- develops on the basis of many micro- or macroreentry circuits, largely in the left atrium (opening of the pulmonary veins)



6/ Atrial fibrillation

- functional reentry circuits often generate paroxysmal forms of AF with spontaneous version to sinus rhythm
- morphological reentry circuits with a tendency to persistent AF



Aetiology of atrial flutter and fibrillation:

- 1/ Valvular disorders (Mi stenosis and/or regurgitation) – dilation of the left atrium – electric remodeling – generation of microentry circuits on the basis of different depolarization features of cardiomyocytes in remodeled myocardium
- 2/ Ischaemic heart disease
- 3/ Morphological changes of the heart (dilated cardiomyopathy)
- 4/ Degenerative changes of the conducting tissues of the heart (sick sinus syndrome)
- 5/ Inflammatory diseases of the heart (pericarditis, myocarditis)

Aetiology of atrial flutter and fibrillation:

- 6/ Metabolic reasons (hyperthyreosis)
- 7/ Arterial hypertension
- 8/ Alcoholism

Haemodynamic consequences of atrial fibrillation:

1/ Decrease in cardiac output:

shortening of the period of filling of the ventricles at diastole +
absence of atrial contribution – decrease of preload

The faster the rate of the ventricles, the larger the decrease of preload
- this potentiates the loss of the atrial contribution to total diastolic
filling of the left ventricle.

2/ Arrhythmias:

unequal duration of the diastole – different volume of the filling of
the left ventricle – variations of the cardiac output – unevenly filled
peripheral pulse (peripheral deficit)

3/ Shortening of the period of diastolic nutritional perfusion of the myocardium (myocardial ischaemia):

- decrease of contractility of the left ventricle (failure)
- deterioration of the arrhythmogenic field
- acute coronary syndrome

**Sum: left ventricle decompensation (failure)
coronary insufficiency (myocardial infarction)
cerebrovascular insufficiency (brain stroke)**

ECG in atrial fibrillation:

- **fibrillation waves “f” with a rate of 400-600/min** (usually they cannot be differentiated)
- **absence of the P waves**
- **irregular rhythm of QRS complexes**
- **rate of the ventricles depending on the AV conduction**
(AF with slow x adequate x rapid ventricular response)

Example: AF with a rate of QRS complexes on ECG 150/min
correct description: AF with RVR (rapid ventricular response)

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Posuv 25 mm/s
 Amplituda 10 mm/mV
 Filtry 0.2 s, 50 Hz, Fuzzy +
 Tepová frekv. 131 [1/min]

Poznámka:



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Posuv 25 mm/s
Amplituda 10 mm/mV
Filtry 0.2 s, 25 Hz, Fuzzy +
Tepová frekv. 75 [1/min]

Poznámka:



Clinical picture of atrial fibrillation:

- symptom-free
- palpitations
- dyspnoea
- angina pectoris
- fatigue
- cardiac decompensation – pulmonary congestion
- clinical expression of atherosclerotic encephalopathy/
ischaemic stroke

Therapy of atrial fibrillation:

1/ stabilization of circulation (slowing down of ventricular response pharmacologically – verapamil, beta-blockers, digoxin), electric version in case of instability of circulation

2/ anticoagulation (heparin, Warfarin)

3/ version to sinus rhythm

(in acute cases may be applied immediately, in long-term fibrillation taking more than 3 weeks we apply anticoagulation therapy prior to electric cardioversion to prevent thromboembolization – e.g. stroke)

- **electric version** (150-300 J)
- **pharmacological version** (propafenone, sotalol, amiodarone)

4/ Prevention of recurrence of AF after version –

- **pharmacological** - propafenone, amiodarone, sotalol
- **catheterization**
 - **radiofrequency ablation** of the AV node with the establishment of permanent cardio stimulation
 - **radiofrequency isolation** of the locus producing AF in the left atrium (based on the electric mapping), **linear radiofrequency incision** in the atrial musculature

5/ Pharmacological control of the ventricular response

in persistent/chronic AF

(beta-blockers, digoxin, verapamil, amiodarone)

6/ Prevention of thromboembolism

permanent anticoagulation in patients with chronic AF