

**MINISTRY OF HEALTHCARE OF UKRAINE  
HSEEU "Ukrainian Medical Stomatological Academy"**

**"Approved"**  
at the meeting of internal  
medicine №1 department  
Head of Department  
**Prof. Skrypnyk I.M.**

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**Protocol № 1 from 29.08.2016**

**GUIDELINES  
FOR STUDENTS  
INDEPENDENT WORK  
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Emergency conditions in clinic of Internal Medicine
<i>Content module</i>	Emergency conditions in clinic of Internal Medicine
<i>Study subject</i>	<b>Curation of the patients with paroxysmal cardiac arrhythmias</b>
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

## 1. Actuality of the topic

### Topic actuality:

Disorders of cardiac rhythm is one of the most dangerous pathologies of cardiovascular disease according to WHO. The incidence of arrhythmias among people of working age in Ukraine have increased in recent years. Sudden cardiac death may be the result of the variety of forms, sometimes lack of effective treatment. The incidence of sudden cardiac death in different countries is about more than 1 case per 1000 people per year. Thus, it's very important to know the mechanisms of arrhythmias, clinical manifestations, diagnostic methods and its treatment to prevent complications.

### 2. The aims of the training course:

#### To Know:

- analyze the prevalence of disorders of cardiac rhythm;
- Determine the etiology and pathogenesis of arrhythmias;
- classify the cardiac rhythm and analyze the typical clinical picture;
- create an individual scheme of diagnostic search, identify and propose the necessary diagnostic testing of patients with different forms of arrhythmias;

#### To be able to:

- to conduct physical examination of the patient (survey, inspection, palpation, percussion, auscultation) and justify a preliminary diagnosis;
- make a plan for additional examination of the patient with disorders of cardiac rhythm;
- justify the use of basic invasive and noninvasive diagnostic methods applied in the patients, indications and contraindications;
- interpret the results of additional research methods: blood biochemical analysis, electrocardiography (ECG), echocardiography, daily monitoring of ECG and others.
- to explain differential diagnosis and clinical diagnosis;
- know the principles of treatment, rehabilitation and prevention of cardiac rhythm disorders;

3. Basic knowledge, abilities and skills necessary for studying theme.

### Interdisciplinary integration:

No	The names of previous sciences	The received skills
1.	Anatomy, topographic anatomy	Describe the anatomical and topographical characteristics of cardiovascular system
2.	Normal and Pathological Physiology	Know the physiology of circulation and conduction system of the heart, pathophysiological basis of cardiac rhythm disorders

3.	Pharmacology	To know pharmacokinetics and pharmacodynamics of drugs that are appointed to the cardiac rhythm disorders. To be able to prescribe proper treatment, calculate the dose of antiarrhythmic drugs
4.	Propedeutics of Internal Medicine	To master the methods of examination of the patient with disorders of cardiac rhythm (palpation, percussion, auscultation of the heart). A survey of the patient, evaluate the results obtained survey data of laboratory and instrumental methods
5.	Intra-subject integration	To know the signs of arrhythmia differential between themselves and other disorders of the cardiovascular system. Be able to determine the nature of cardiac rhythm disturbance, diagnose arrhythmias

The contents of the topic:

## SINOATRIAL (SA) NODE ARRHYTHMIAS

### Normal Sinus Rhythm (NSR)



**Rate:** Normal (60–100 bpm)

**Rhythm:** Regular

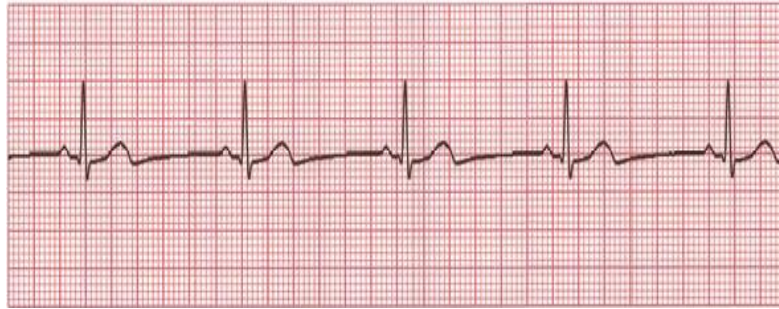
**P Waves:** Normal (upright and uniform)

**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 Bradicardia



**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



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

**Rhythm:** Regular

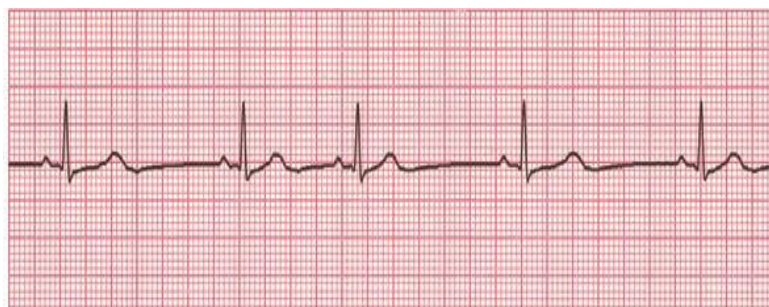
**P Waves:** Normal (upright and uniform)

**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



**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)

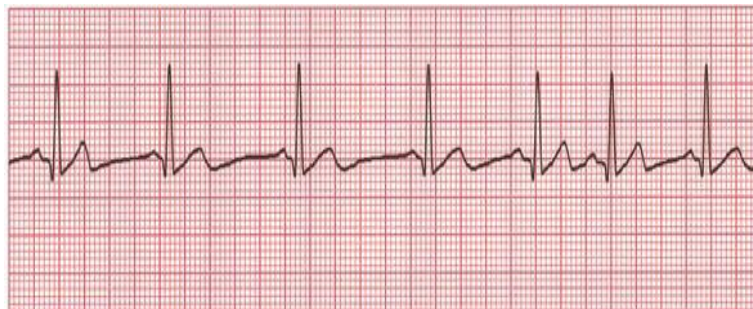
**PR Interval:** Normal (0.12–0.20 sec)

**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

### Wandering Atrial Pacemaker (WAP)



**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)



**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.



## Premature Atrial Contraction (PAC)



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

**Rhythm:** Irregular whenever a PAC occurs

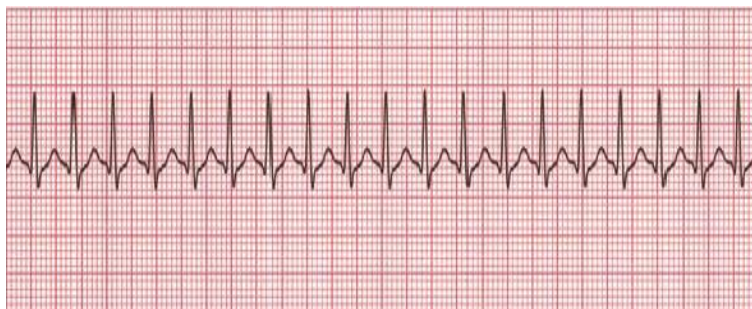
**P Waves:** Present; in the PAC, may have a different shape

**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.

## Supraventricular Tachycardia (SVT)



**Rate:** 150–250 bpm

**Rhythm:** Regular

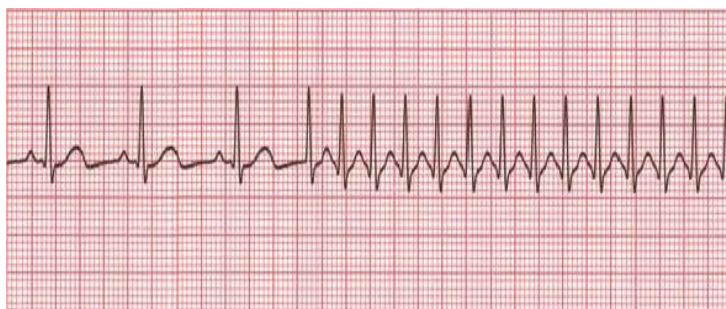
**P Waves:** Frequently buried in preceding T waves and difficult to see

**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)



**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.

## Atrial Flutter (A-flutter)



**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)



**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.

## Wolf-Parkinson-White (WPW) Syndrome





**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 ( $\leq 0.12$  sec) if P wave is present

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

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

## JUNCTIONAL ARRHYTHMIAS

### 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



**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 Contraction (PJC)



**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.

## VENTRICULAR ARRHYTHMIAS

### Idioventricular Rhythm



**Rate:** 20–40 bpm

**Rhythm:** Regular

**P Waves:** None

**PR Interval:** None

**QRS:** Wide ( $>0.10$  sec), bizarre appearance

♥ **Clinical Tip:** Idioventricular rhythm may also be called agonal rhythm.

### Accelerated Idioventricular Rhythm



**Rate:** 41–100 bpm

**Rhythm:** Regular

**P Waves:** None

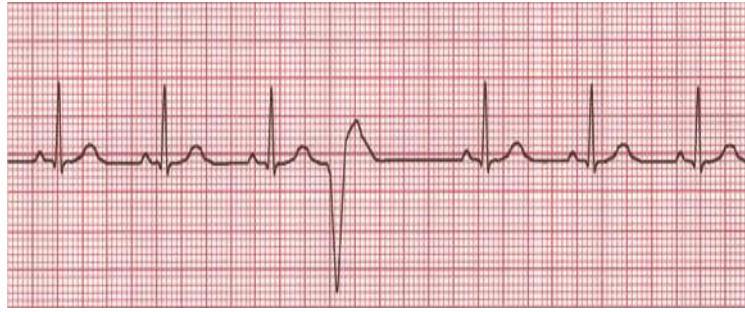
**PR Interval:** None

**QRS:** Wide ( $\geq 0.10$  sec), bizarre appearance

♥ **Clinical Tip:** Idioventricular rhythms appear when supraventricular pacing sites are depressed or absent. Diminished cardiac output is expected if the heart rate is slow.



## Premature Ventricular Contraction (PVC)



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

**Rhythm:** Irregular whenever a PVC occurs

**P Waves:** None associated with the PVC

**PR Interval:** None associated with the PVC

**QRS:** Wide ( $>0.10$  sec), bizarre appearance

♥ **Clinical Tip:** Patients may sense the occurrence of PVCs as skipped beats. Because the ventricles are only partially filled, the PVC frequently does not generate a pulse.

## Premature Ventricular Contraction (same form)



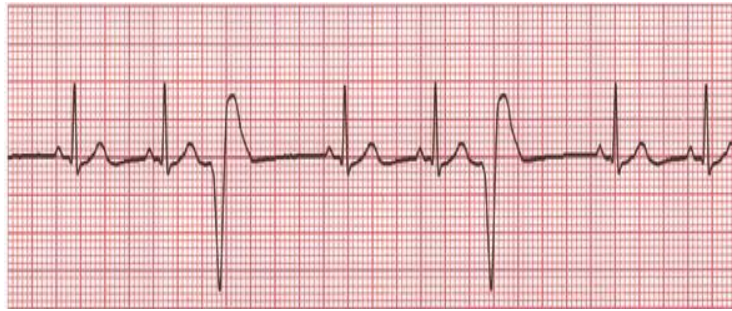
## Premature Ventricular Contraction: Multiform (different forms)



## Premature Ventricular Contraction: Ventricular Bigeminy (PVC every other)



## Premature Ventricular Contraction: Ventricular Trigeminy (PVC every 3rd)



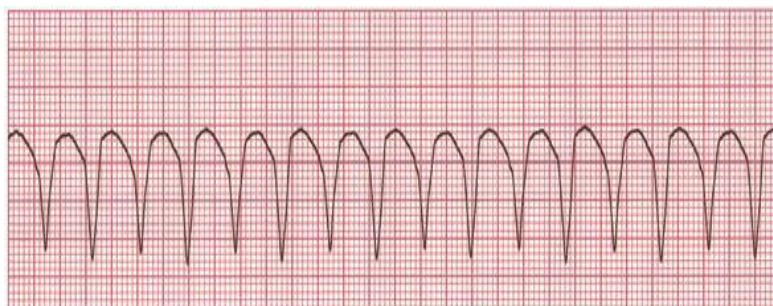
## Premature Ventricular Contraction: Ventricular Quadrigeminy (PVC every 4th beat)



## Premature Ventricular Contraction: Couplets (paired PVCs)



## Ventricular Tachycardia (VT): Monomorphic





**Rate:** 100–250 bpm

**Rhythm:** Regular

**P Waves:** None or not associated with the QRS

**PR Interval:** None

**QRS:** Wide ( $\geq 0.10$  sec), bizarre appearance

♥ **Clinical Tip:** It is important to confirm the presence or absence of pulses because monomorphic VT may be perfusing or nonperfusing.

♥ **Clinical Tip:** Monomorphic VT will probably deteriorate into VF or unstable VT if sustained and not treated.

## Ventricular Tachycardia (VT): Polymorphic



**Rate:** 100–250 bpm

**Rhythm:** Regular or irregular

**P Waves:** None or not associated with the QRS

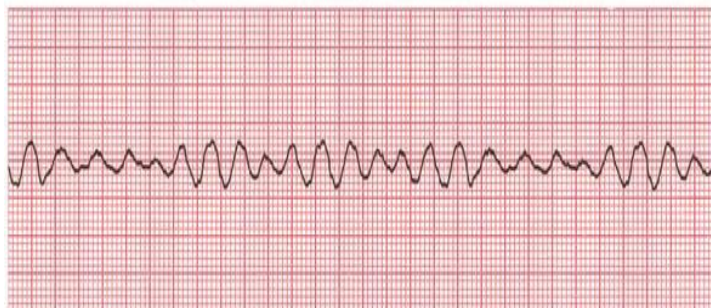
**PR Interval:** None

**QRS:** Wide ( $\geq 0.10$  sec), bizarre appearance

♥ **Clinical Tip:** It is important to confirm the presence or absence of pulses because polymorphic VT may be perfusing or nonperfusing.

♥ **Clinical Tip:** Consider electrolyte abnormalities as a possible etiology.

## Ventricular Fibrillation (VF):



**Rate:** Indeterminate

**Rhythm:** Chaotic

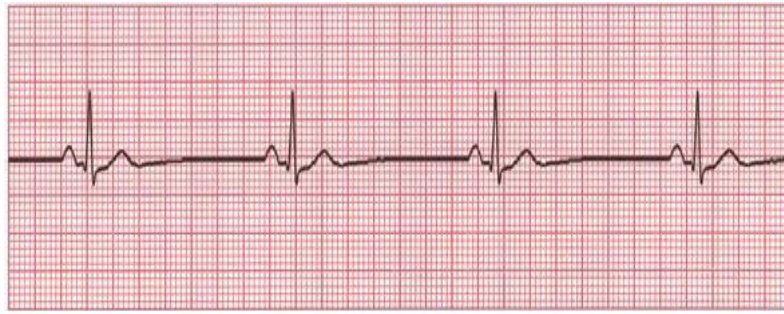
**P Waves:** None

**PR Interval:** None

**QRS:** None

♥ **Clinical Tip:** There is no pulse or cardiac output. Rapid intervention is critical. The longer the delay, the less the chance of conversion.

# Pulseless Electrical Activity (PEA)



**Rate, rhythm, P waves, P-R interval, and QRS: Reflect underlying rhythm.**

♥ **Clinical Tip:** Potential causes of PEA are pulmonary embolism, MI, acidosis, tension pneumothorax, hyper- and hypokalemia, cardiac tamponade, hypovolemia, hypoxia, hypothermia, and drug overdose (i.e., cyclic antidepressants, beta blockers, calcium channel blockers, digoxin).

## ANTIARRHYTHMIC DRUGS: DOSES AND SIDE EFFECTS

ANTIARRHYTHMIC DRUG AND COMMON USE	DOSE/METABOLISM	SIDE EFFECTS AND REQUIRED MONITORING	SELECTED DRUG INTERACTIONS
Quinidine	Hepatic CYP 3A4 (70%), renal (30%) Dose: sulfate—600 mg tid, gluconate—324 to 648 mg q8h Dose reduced for renal failure	Thrombocytopenia Cinchonism Pruritus, rash QT prolongation/torsades de pointes	↑ Digoxin and amiodarone concentrations Quinidine inhibits CYP 2D6 and may increase drugs metabolized by this enzyme, e.g., ↑ effect of tricyclic antidepressants, haloperidol, some β-blockers, fluoxetine, narcotics Quinidine metabolism inhibited by cimetidine Quinidine metabolism increased by phenobarbital, phenytoin, and rifampicin
Procainamide	Mostly hepatic—rapid acetylators produce more NAPA; NAPA renally cleared PO dose: 50 mg/kg/24 hr IV dose: 1 g over 25 min, then 20-60 µg/kg/min infusion Reduce dose for renal dysfunction or low cardiac output	Rash, fever, arthralgias, drug-induced lupus, particularly in slow acetylators Agranulocytosis QT prolongation/torsades de pointes	Procainamide clearance reduced by trimethoprim, cimetidine, and ranitidine
Disopyramide	Renal, hepatic (CYP 3A4) Dose: 100-400 mg q8-12h; max dose, 800 mg/24 hr Reduce dose for renal or hepatic dysfunction	Anticholinergic (contraindicated for narrow-angle glaucoma): dry mouth, urinary retention, constipation, blurry vision QT prolongation/torsades de pointes	None
Propafenone	Hepatic: 150-300 mg q8h or sustained release 225-425 mg bid	Metallic taste, dizziness, SIADH Atrial flutter, ventricular tachycardia	May decrease the metabolism of warfarin Increase digoxin levels
Flecainide	Renal, hepatic CYP 2D6 50-100 mg bid; max dose, 300-400 mg/day	Dizziness, headache, visual blurring Atrial flutter, ventricular tachycardia	May increase digoxin levels Flecainide levels increased by amiodarone, haloperidol, quinidine, cimetidine, and fluoxetine
β-Blockers (selected)	Hepatic, renal Only renal (atenolol, nadolol) IV esmolol: 250-500 µg over 1 min, then 50-300 µg/kg/min over 4 min Acebutolol, 200-600 mg bid; atenolol, 25-100 mg qd; carvedilol, 3.125-50 mg bid; metoprolol, 25-150 mg bid; nadolol, 20-120 mg qd; nebivolol, 5-40 mg qd; propranolol, 10-120 mg bid	Fatigue, depression, bronchospasm, impotence	Minimal, except for carvedilol and metoprolol, whose levels may be increased by amiodarone, propafenone, quinidine, fluoxetine, haloperidol, paroxetine, and cimetidine
Sotalol	Renal: 80-120 mg bid Max dose, 240 mg bid	Bronchospasm QT prolongation/torsades de pointes	No significant interactions

ANTIARRHYTHMIC DRUG AND COMMON USE	DOSE/METABOLISM	SIDE EFFECTS AND REQUIRED MONITORING	SELECTED DRUG INTERACTIONS
Dofetilide	Renal, hepatic CYP 3A4 CrCl > 60 (500 µg bid), CrCl 40-60 (250 µg bid), CrCl 20-39 (125 µg bid)	QT prolongation and torsades de pointes Three days of in-hospital monitoring is required during drug initiation	Contraindicated with verapamil, ketoconazole, cimetidine, megestrol, prochlorperazine, and trimethoprim Hydrochlorothiazide increases dofetilide levels Must discontinue amiodarone at least 3 mo before dofetilide initiation
Ibutilide	Hepatic CYP 3A4 1 mg IV over 10 min, repeat after 10 min if necessary	Nausea QT prolongation and torsades de pointes Must monitor for 4 hr after drug initiation	None
Amiodarone	Hepatic half-life 50 days PO load 10 g over 7-10 days, then 400 mg for 3 wk, then 200 mg/day for atrial fibrillation Maintenance dose of 400 mg/day for VT Dose reduce load for bradycardia or QT prolongation IV: 150-300 mg bolus, then 1 mg/min infusion for 6 hr, followed by 0.5 mg/min thereafter	Pulmonary (acute hypersensitivity pneumonitis, chronic interstitial infiltrates), hepatitis Thyroid (hypo- or hyperthyroidism) Photosensitivity, blue-gray discoloration with chronic high dose, nausea, ataxia, tremor, alopecia Avoid if identified thyroid nodule LFTs two to three times a year, TFTs twice yearly, PFTs and CXR at initiation and CXR yearly thereafter. QT prolongation expected; reduce dose if exceeds 500 msec	Inhibits CYP 450 enzymes—increases concentrations of warfarin, digoxin, cyclosporine, alprazolam, carbamazepine, HMG-CoA inhibitors, phenytoin, and quinidine
Dronedarone	Hepatic CYP 3A4 half-life 30 hr PO 400 mg bid Improved absorption with food	Reduces the secretion of creatinine without a reduction in GFR Hepatic failure Avoid in heart failure	Increases digoxin levels (dose reduce digoxin by half) May increase myositis with simvastatin Avoid grapefruit
Calcium-channel blocker (nondihydropyridine)	Hepatic Inhibit CYP 3A4 IV diltiazem, 20 mg bolus over 2 min, then 5-15 mg per hour maintenance infusion Verapamil long-acting 120-480 mg qd Diltiazem long-acting 180-300 mg qd	Constipation, rash, peripheral edema	Inhibits CYP 3A4—will increase levels of alprazolam, carbamazepine, dihydropyridine, cyclosporine, HMG-CoA inhibitors. Verapamil (but not diltiazem) increases digoxin levels

#### ANTIARRHYTHMIC DRUGS: DOSES AND SIDE EFFECTS—cont'd

ANTIARRHYTHMIC DRUG AND COMMON USE	DOSE/METABOLISM	SIDE EFFECTS AND REQUIRED MONITORING	SELECTED DRUG INTERACTIONS
Adenosine	Erythrocyte, endothelial cell 6-mg IV push, followed if necessary by 12 mg after 1-2 min	Nausea, headache, flushing, chest pain, bronchospasm (contraindicated if asthma)	Methylxanthines compete for adenosine receptors with adenosine Dipyridamole decreases the metabolism of adenosine
Digoxin	Renal, hepatic, gastrointestinal, 0.125-375 mg/day	Anorexia, nausea, fatigue, confusion, altered vision with green-yellow halos	Levels of or sensitivity to digoxin increased by hypokalemia, quinidine, verapamil, amiodarone, propafenone, renal failure, hypoxia, decreased muscle mass Levels of or sensitivity to digoxin decreased by malabsorption, hyperkalemia, hypocalcemia

CXR = chest x-ray; CYP = cytochrome P-450; GFR = glomerular filtration rate; HMG-CoA = 3-hydroxy-3-methylglutaryl coenzyme A; IV = intravenous administration; LFT = liver function test; NAPA = N-acetyl procainamide; PFT = pulmonary function test; PO = oral administration; SIADH = syndrome of inappropriate diuretic hormone; TFT = thyroid function test; VT = ventricular tachycardia.

## 4. Materials for self-training

### 4.1. The main terms, subjects and its introductions:

Term	Determination
1. Disorders of cardiac rhythm	change the normal frequency, regularity and sources of stimulation of the heart, and violation of conduction and consistency between ventricular and atrial activation
2. Extrasystole	Premature stimulation and reduction of part or the whole heart

3. Fibrillation, or atrial fibrillation	very often (from 350 to 700 per minute), chaotic stimulation and reduction of atrial muscle fibers
4. Atrial flutter	considerable atrial reductions becoming more frequent (up to 200-400 per minute) while retaining the correct regular atrial rhythm
5. Ventricular fibrillation	frequent (up to 200-500 beats per minute) chaotic stimulation and reduction of certain groups of ventricular muscle fibers
6. Ventricular flutter	frequent (up to 200-300 per minute) rhythmic stimulation and reduction of certain groups of ventricular muscle fibers

#### 4.2. Self-control materials Questions to be answered:

- determination of disorders of cardiac rhythm;
- modern views on etiology and pathogenesis of arrhythmias;
- Classification of disorders of cardiac rhythm;
- Basic clinical and laboratory syndromes in different types of arrhythmias;
- criteria for diagnosis of disorders of cardiac rhythm;
- differential diagnosis;
- complications of arrhythmias;
- indications and contraindications to the use of antiarrhythmic drugs and class I;
- indications and contraindications to the use of antiarrhythmic drugs class II;
- indications and contraindications for the appointment of class III antiarrhythmic drugs;
- indications and contraindications for the appointment of class IV antiarrhythmic drugs;
- Basic principles of therapy, rehabilitation, prevention of disorders of cardiac rhythm; Weather and efficiency.

#### ***A. The questions for self-control:***

1. Name the main aetiological factors of cardiac rhythm disorders.
2. Make the plan of additional investigation of the patient with cardiac rhythm disorders.
3. Name the main principles and ways of treatment of cardiac rhythm disorders.

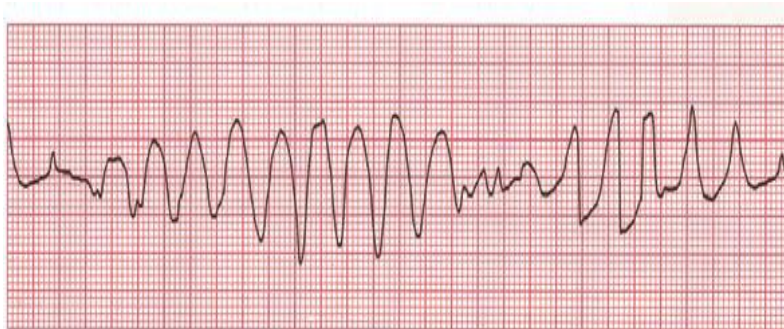


### ***B. Tests for self-control:***

#### ***Questions:***

What are cardiac rhythm disorders in these cases, by the given ECG data?

ECG Number 1



ECG Number 2



#### **Recommended literature:**

##### ***A. Main:***

1. "Harrison's principles of internal medicine", Editors: Anthony S. Fauci, Dennis L. Kasper, Stephen L. Hauser, Dan L. Longo, Joseph Loscalzo, McGraw-Hill Education / Medical; 19 edition (April 8, 2015), 1-2 volumes, 3000 p.
2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. Davidson's Principles and Practice of Medicine: With STUDENT CONSULT Online Access, 21e (Principles & Practice of Medicine (Davidson's)) by Nicki R. Colledge BSc FRCP(Ed), Brian R. Walker BSc MD FRCP(Ed) and Stuart H. Ralston MB ChB MD FRCP FMedSci FRSE (Paperback - Mar 11, 2010)Kumar and Clark's Clinical Medicine, 7e (Kumar, Kumar and Clark's Clinical Medicine) by Parveen J. Kumar (Paperback - Jul 2, 2009)
4. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
5. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. – 2012. p.
6. Sonographer's Handbook of Diagnostic Ultrasound by Jason R. Young M.D. (Feb 23, 2011)

##### **Additional literature:**

- 1.Kovalyova O.M., Asheulova T.V. Propedeutics to internal medicine. Part 1, Diagnostics. Vinnytsya, Nova Knyha, 2006, 424 p

##### ***Answers:***

1. **Rate:** 200–250 bpm  
**Rhythm:** Irregular

**P Waves:** None

**PR Interval:** None

**QRS:** Wide ( $\geq 0.10$  sec), bizarre appearance

♥ **Clinical Tip:** Torsade de pointes may deteriorate to VF or asystole.

♥ **Clinical Tip:** Frequent causes are drugs that prolong QT interval and electrolyte abnormalities such as hypomagnesemia.

2. **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 ( $\leq 0.12$  sec) in rapid rates

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

**Methodical recommendations consisted by**

**Kulishov S.K.**