Cardiac arrhythmias

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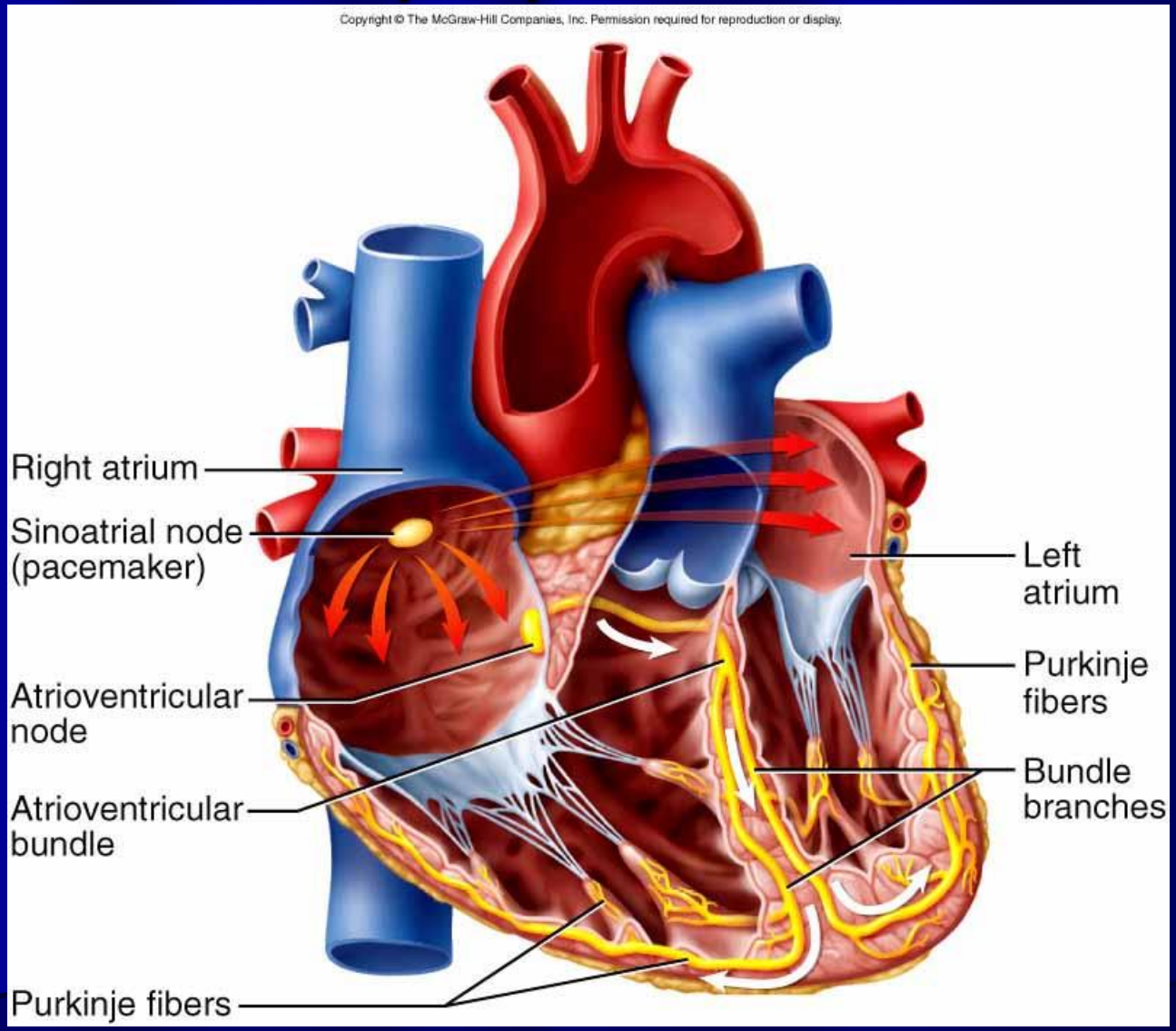
Definition

Cardiac arrhythmia is a group of conditions in which the electrical activity of the heart is irregular or is faster or slower than normal

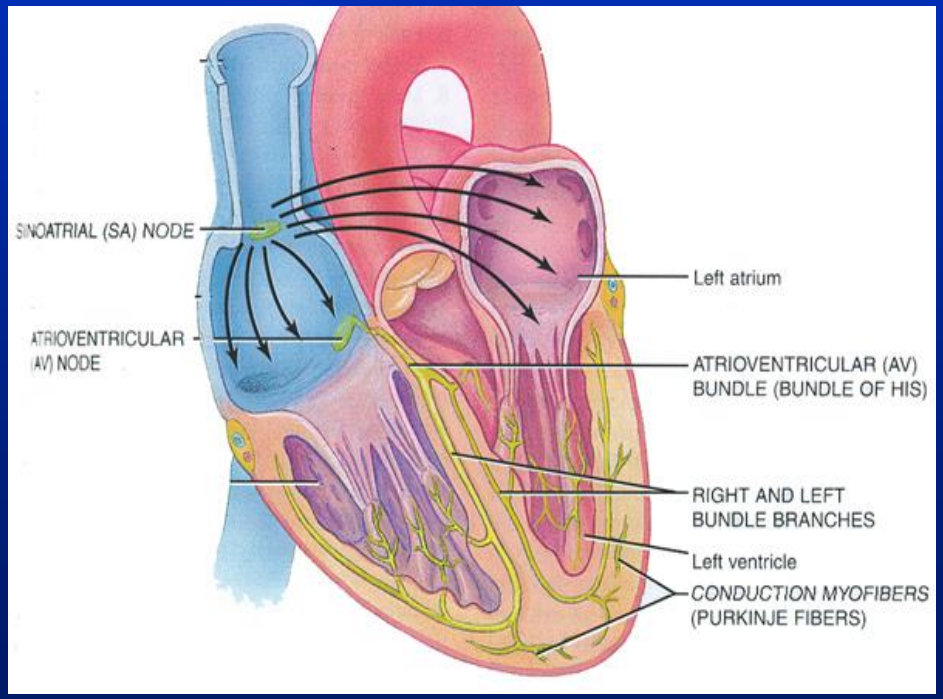
Conductibility system of the heart

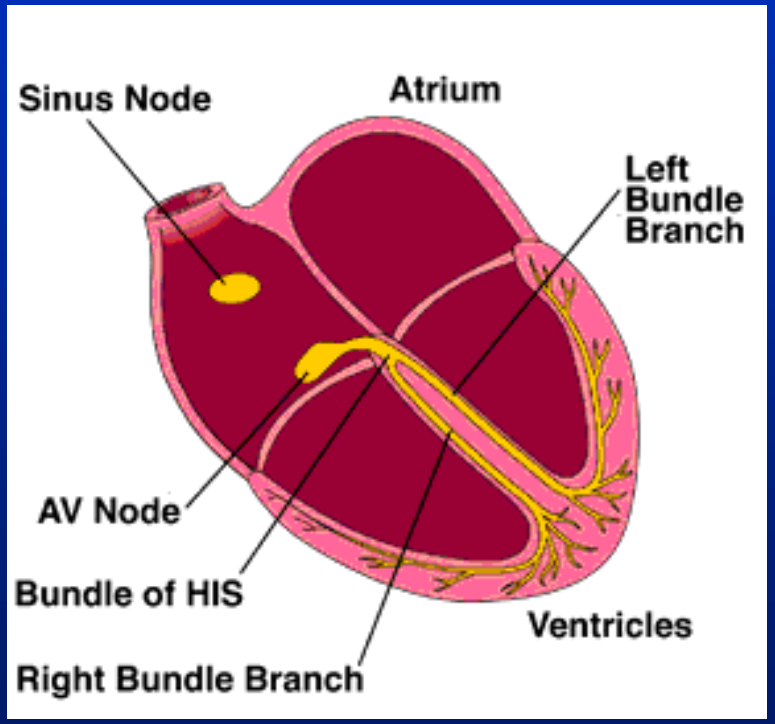


Heart Physiology

Closed system
Supply nutrients/O_2

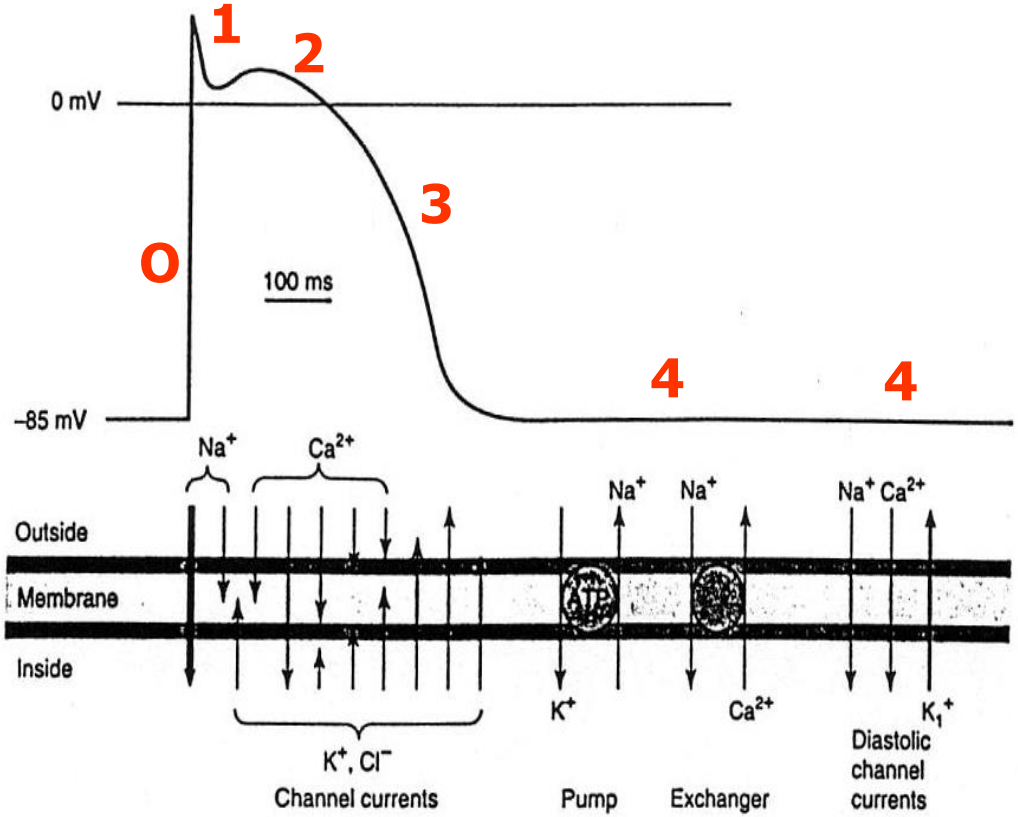
Pressure driven
Remove metabolites





Cardiac Action Potentials
Ion Flow

mM	Na^+	K^+	Ca^{++}
Out	140	4	2.5
In	10	150	0.1

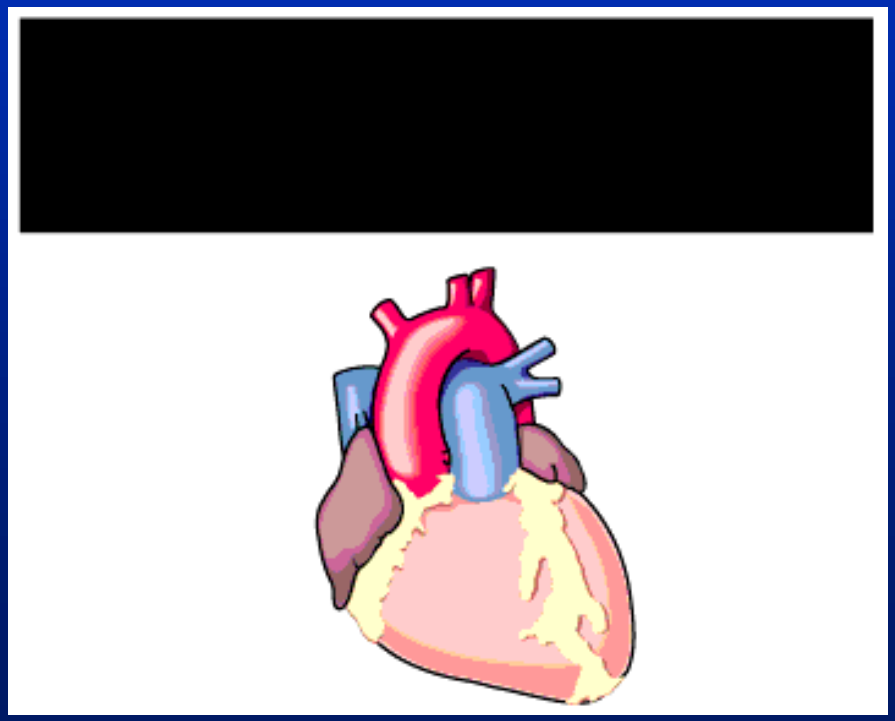


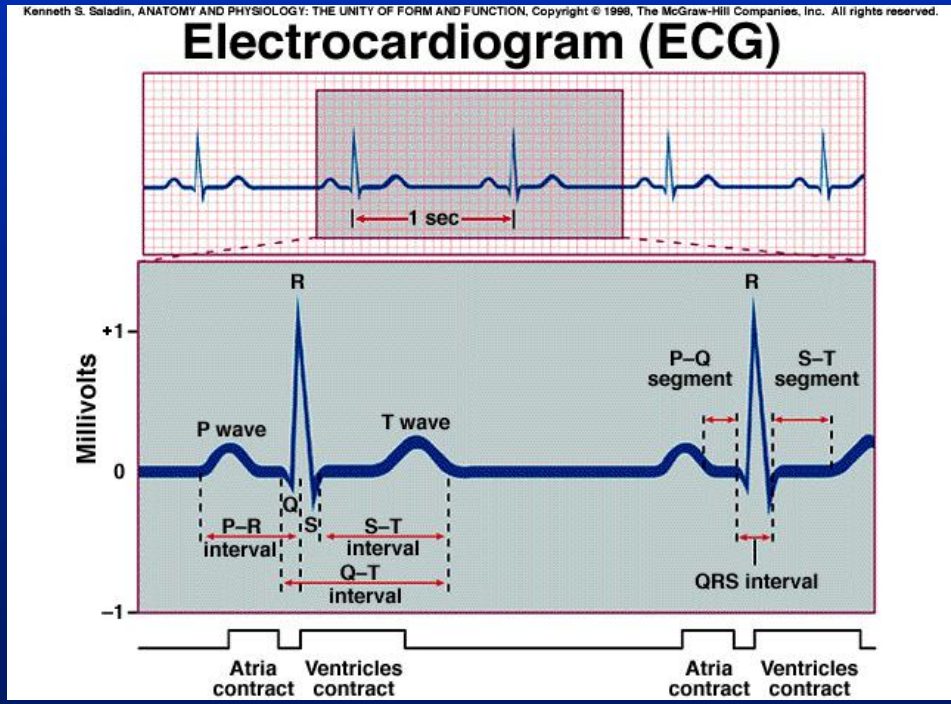
0	Na^+i - open
1	Na^+ - close K^+o - open/close
2	$Ca^{++}i$ - open K^+o - leak
3	Ca^{++} - close K^+o - open
4	K^+ - close

Na^+/Ca^{++} - exchange (3:1)
Na^+/K^+ - ATPase (3:2)

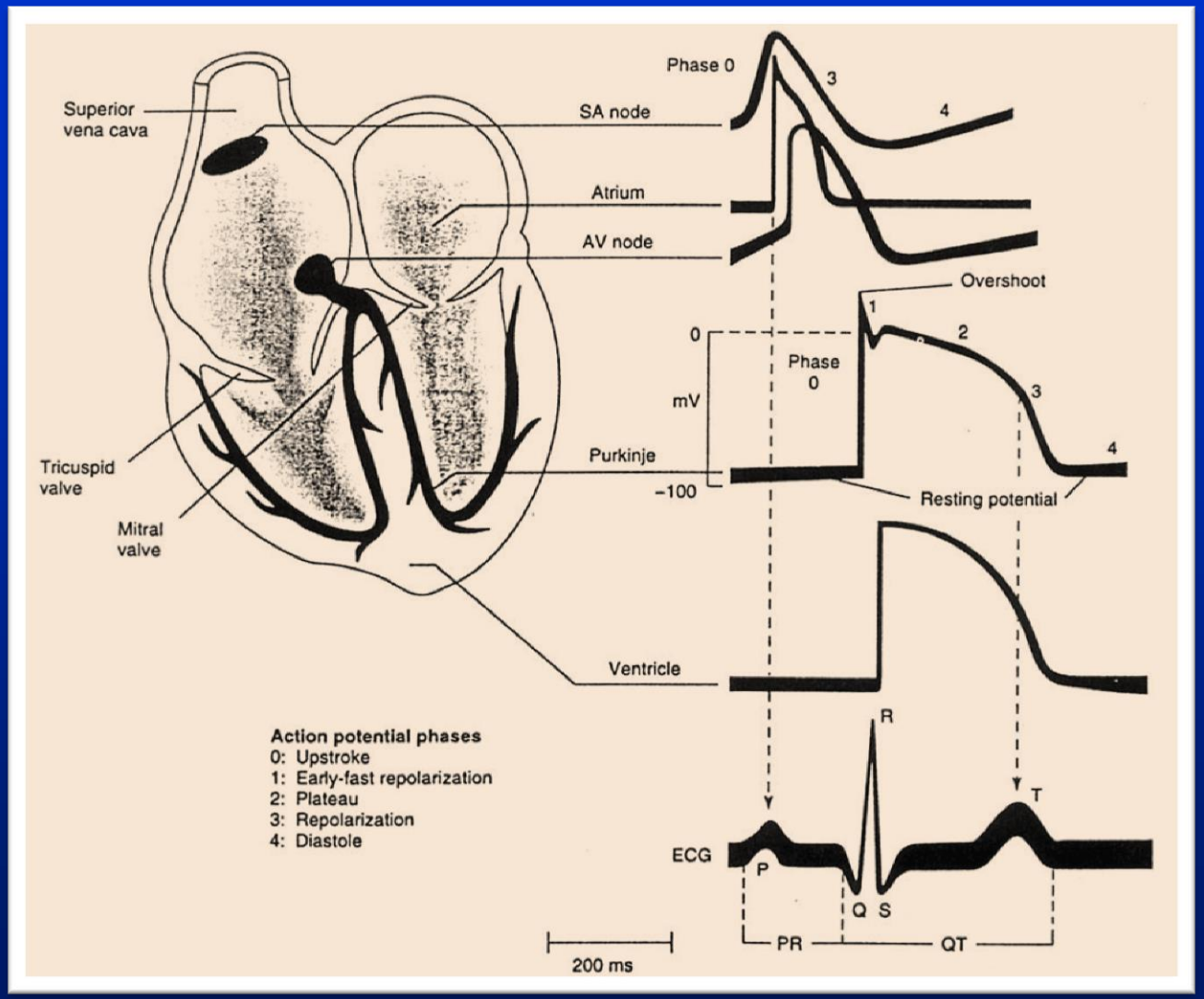
Heart Physiology

P - atria depolarization
QRS - ventricle depolarization
PR - conduction A-V
T - ventricle repolarization
QT - duration ventricle of repolarization





Heart Physiology



Closed system
Pressure driven
Supply nutrients/O_2
Remove metabolites

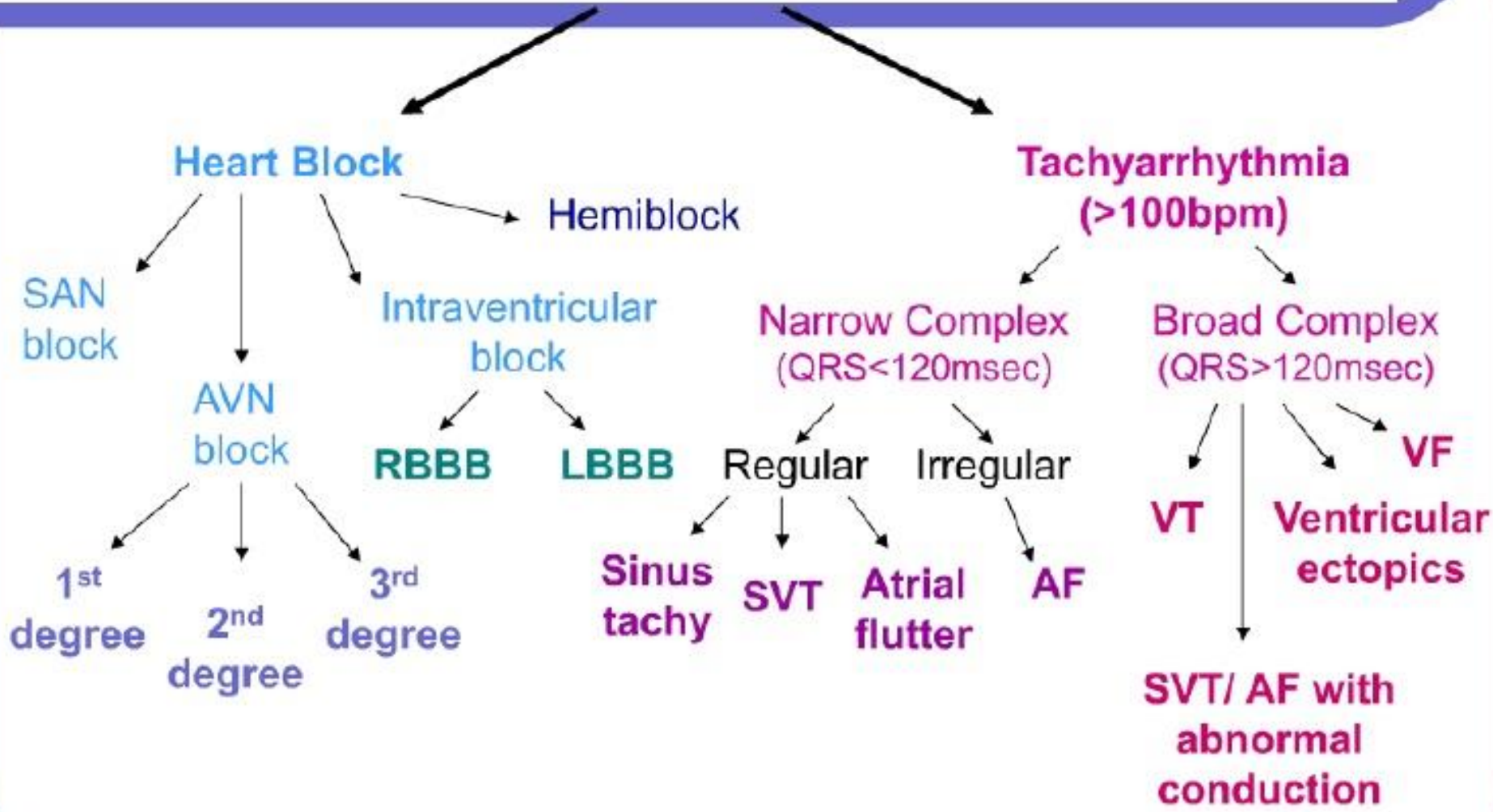
P - atria depol.
QRS - ventricle depol.
PR - conduction A-V
T - ventricle repol.
QT - duration ventricle repolarization

Clasification

Supraventricular tachycardias (SVTs)

Ventricular tachycardias (VTs)

Arrhythmias



Supraventricular Tachycardias

- Rhytms Originating in the Atria
- Rhytms Originating in the AV Junction

Essentials of Diagnosis

- Heart rate greater than 100 bpm.
- Narrow QRS complex

Pathophysiology & Etiology

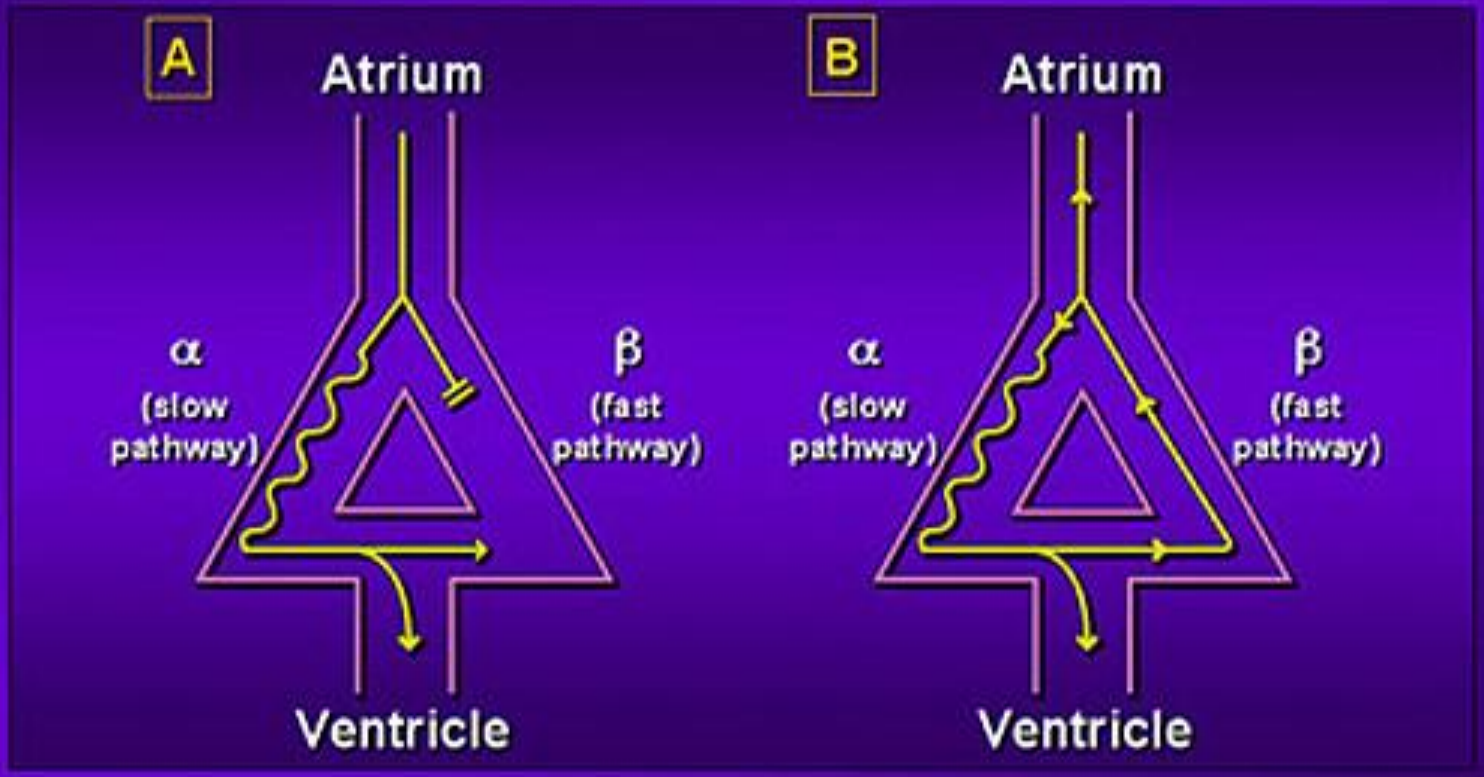
Three main mechanisms

- reentry - is most common
- enhanced or abnormal automaticity
- triggered activity

Reentrant arrhythmias

sustain themselves by repetitively following a revolving pathway comprising two limbs, one that takes the impulse away from, and one that carries it back to the site of origin

Electrophysiological Mechanism of AVNRT



Automaticity

refers to spontaneous and, often, repetitive firing from a single focus, which may either be ectopic or may originate in the sinus node

It should be noted that automaticity is an intrinsic property of all myocardial cells

Triggered arrhythmias

- depends on oscillations in the membrane potential that closely follow an action potential
- These arrhythmias can be produced by early or late after-depolarization, depending on the timing of the first after-depolarization relative to the preceding action potential

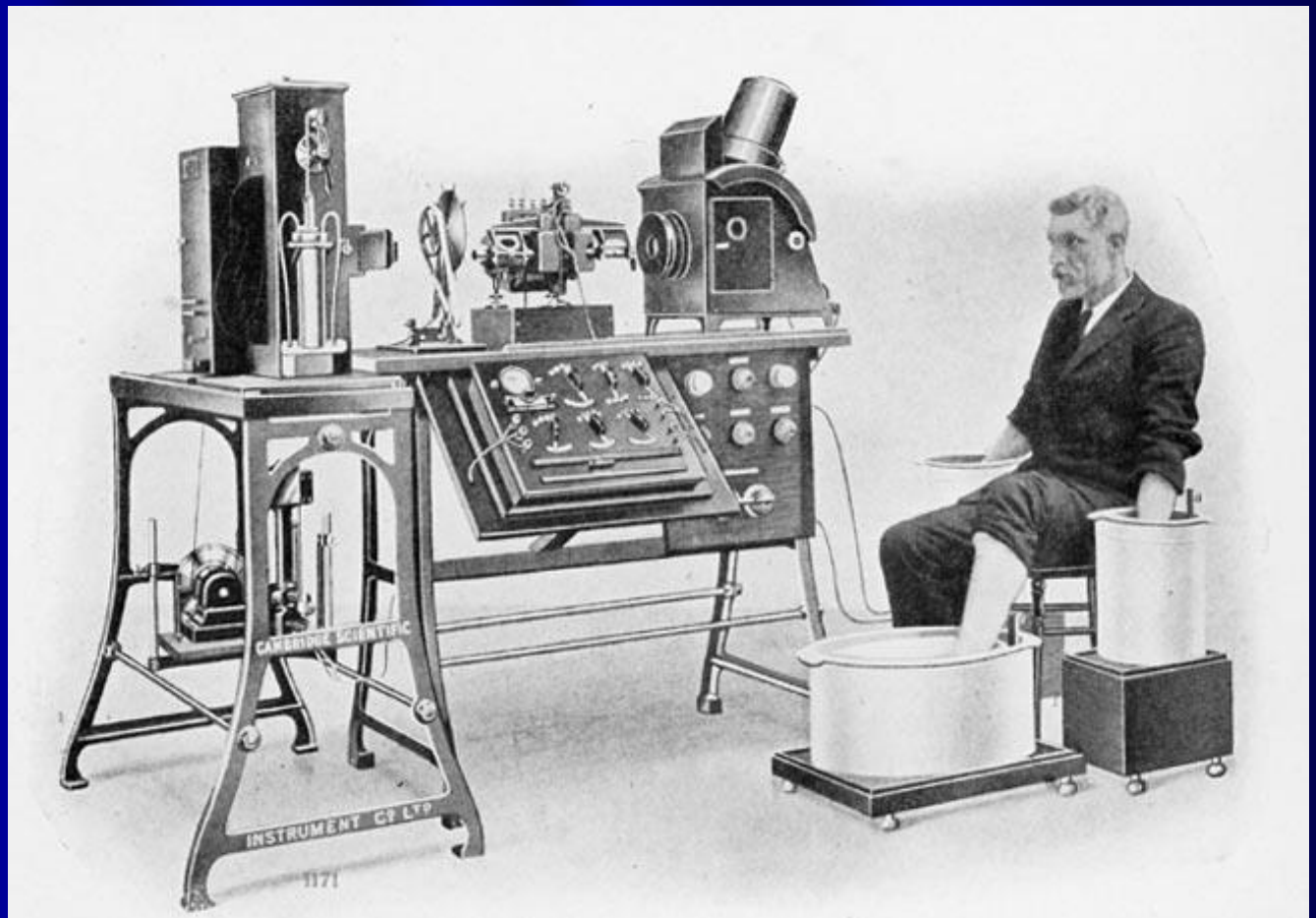
Common causes

- exercise
- hypotension
- hypoxemia
- heart failure
- infection
- fever
- hyperthyroidism
- fluid depletion
- blood loss

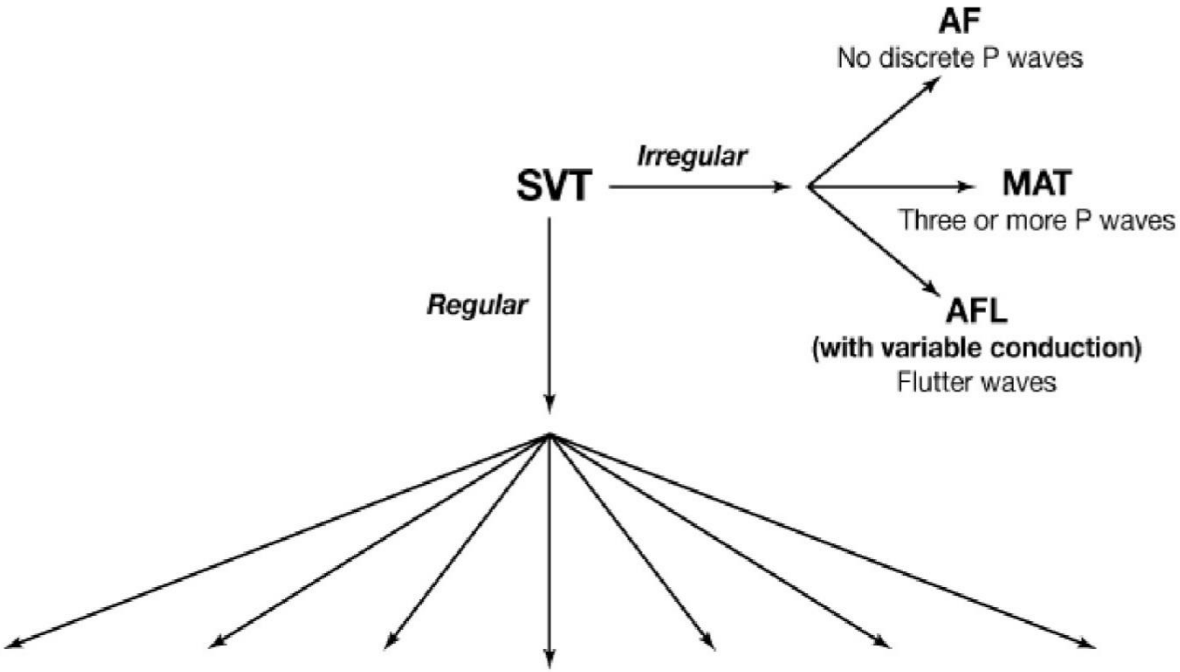
Clinical Findings

- Dizziness
- syncope
- Palpitations
- Angina-type chest pain
- Dyspnea
- Weakness
- Fatigue heart failure may ensue

ECG Diagnostic



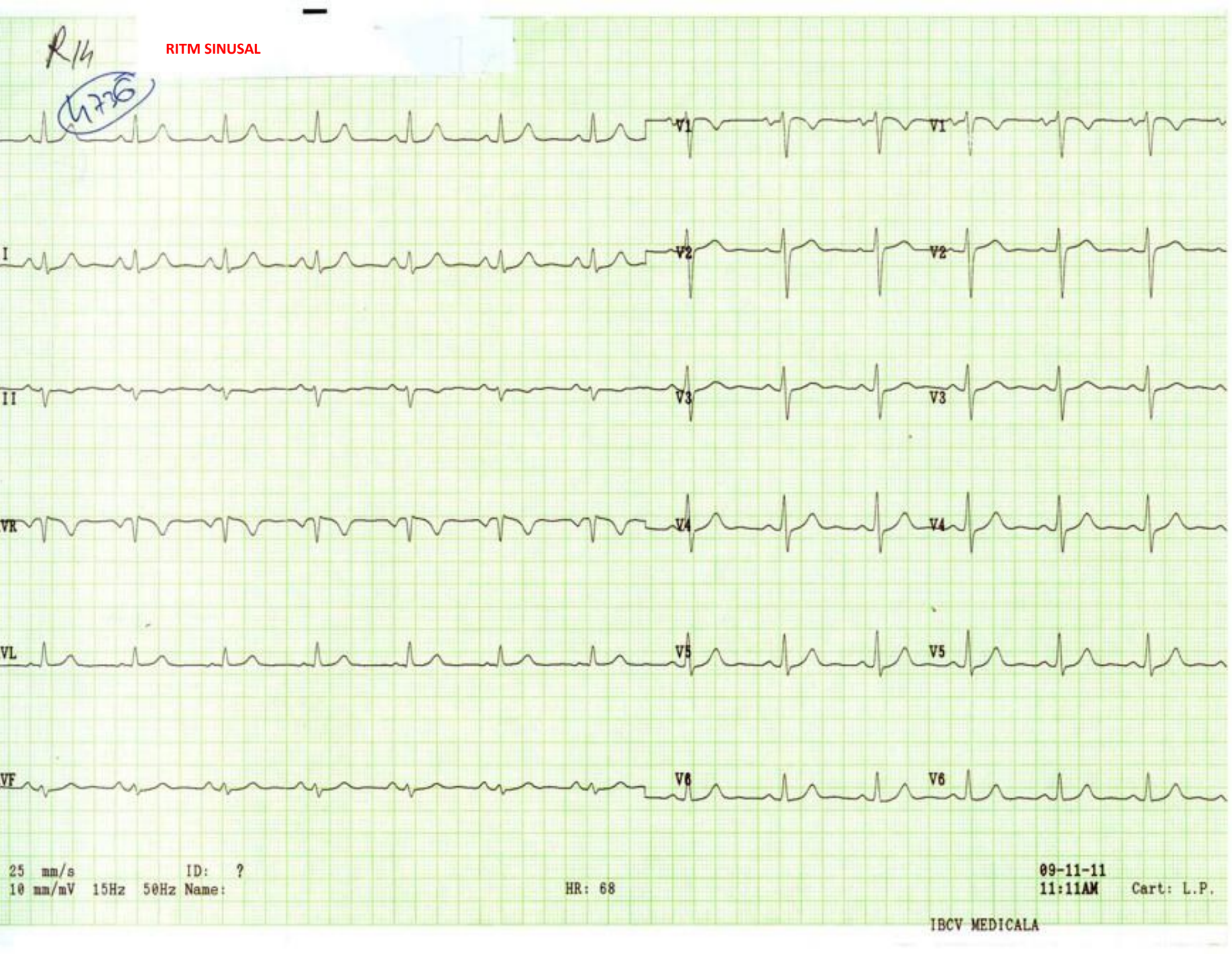
PHOTOGRAPH OF A COMPLETE ELECTROCARDIOGRAPH, SHOWING THE MANNER IN WHICH THE ELECTRODES ARE ATTACHED TO THE PATIENT, IN THIS CASE THE HANDS AND ONE FOOT BEING IMMERSED IN JARS OF SALT SOLUTION



	ST	SN reentry	AFL	AT	AVNRT	JT (Accelerated AV junction rhythm)	AVRT
Onset and termination	Gradual	Sudden	Sudden	Sudden	Sudden	Gradual	Sudden
Heart rate (bpm)	100–(220 – age)	100–160	Multiple of 300	100–180	120–200	70–120	140–250
P wave	Sinus Ps	Sinus Ps	Flutter waves	Ectopic Ps	Retrograde Ps	Retrograde Ps	Ectopic Ps
R-P relationship	Long R-P	Long R-P	Undefined	Long R-P	Short R-P or no P	Short R-P or no P	Short R-P

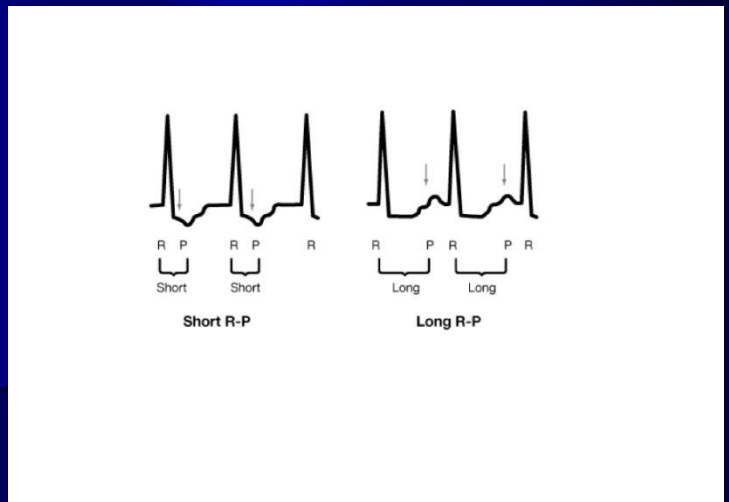
12-lead ECG

- The first step is to determine whether the rhythm is regular or irregular
- If it is irregular, the rhythm is likely either atrial fibrillation, atrial flutter with variable conduction, or multifocal atrial tachycardia (MAT)
- The appearance of the P waves will usually distinguish between these three entities
- In atrial fibrillation, there is chaotic atrial activity
- In atrial flutter, P waves are seen at rate of 240–320 bpm.
- In MAT, there are P waves preceding each QRS complex, and there are at least three different P wave morphologies

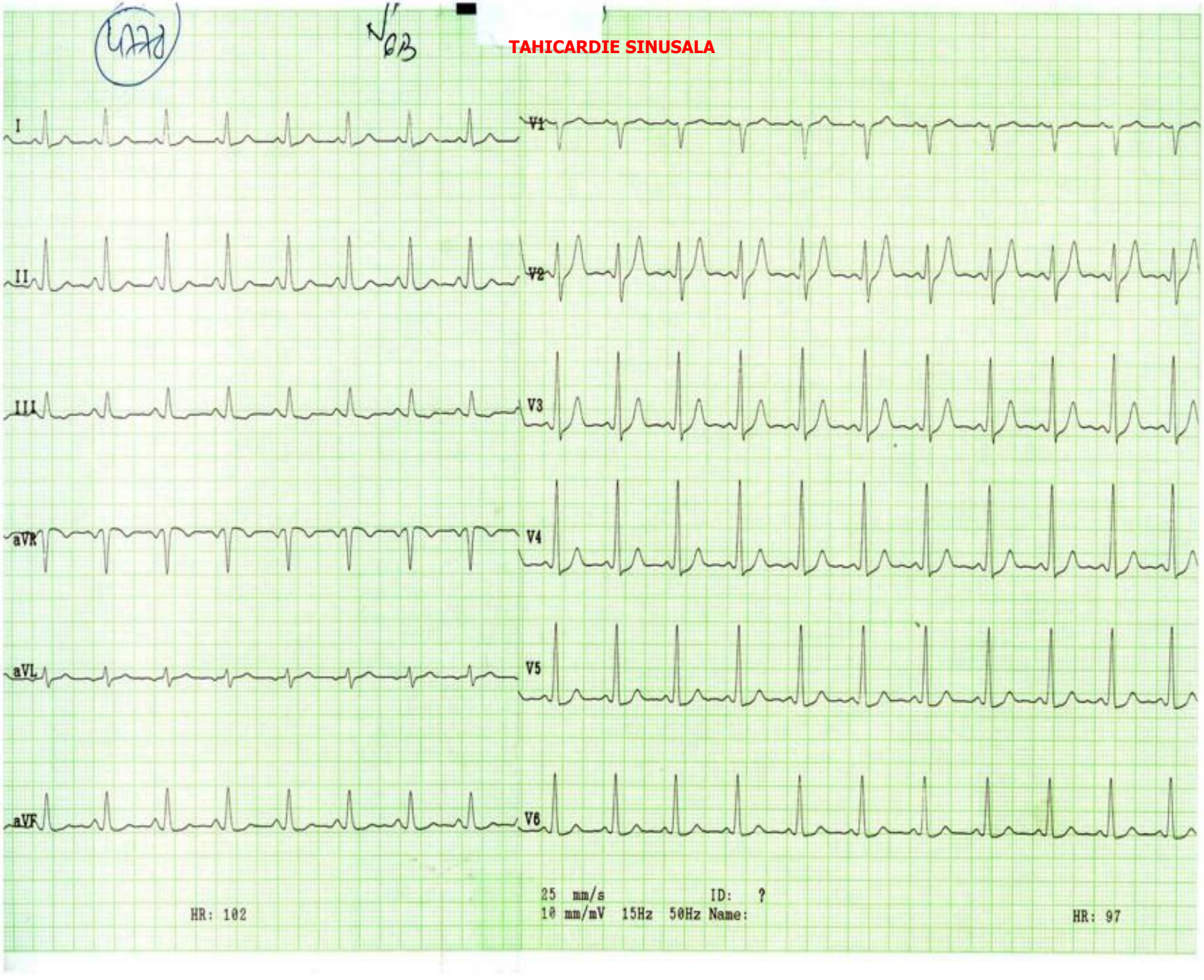

RITM SINUSAL
I
II
VR
VL
VF
V1
V2
V3
V4
V5
V6
25 mm/s
10 mm/mV 15Hz 50Hz
ID: ?
Name:
HR: 68
09-11-11
11:11AM
Cart: L.P.
IBCV MEDICALA

SINUS TACHYCARDIA & SINUS NODE REENTRY

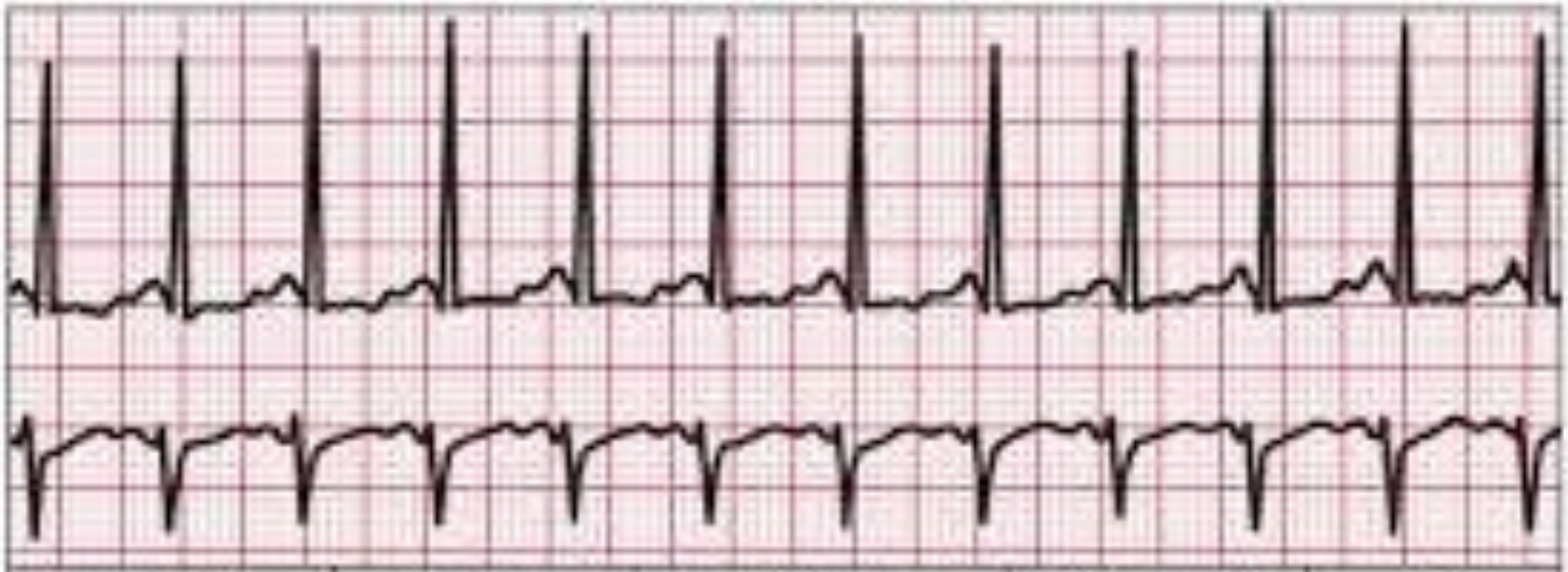
- Onset and termination: Gradual
- Heart rate: 100 to (220 – age) bpm
- P wave: Identical to normal sinus rhythm P wave
- R-P relationship: Long



TAHICARDIE SINUSALA



SINUS TACHYCARDIA



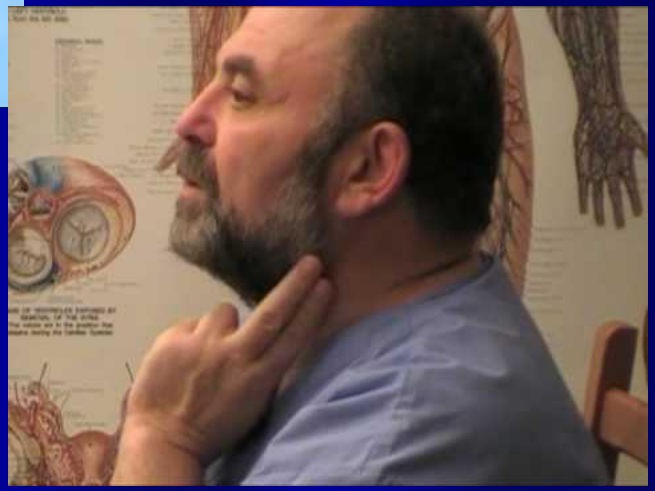
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
> 100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

Treatment

- Vagal maneuvers slow the tachycardia gradually but when the vagal stimulus is removed the heart rate gradually returns to where it started
- Management is usually focused on treating the underlying cause of the sinus tachycardia

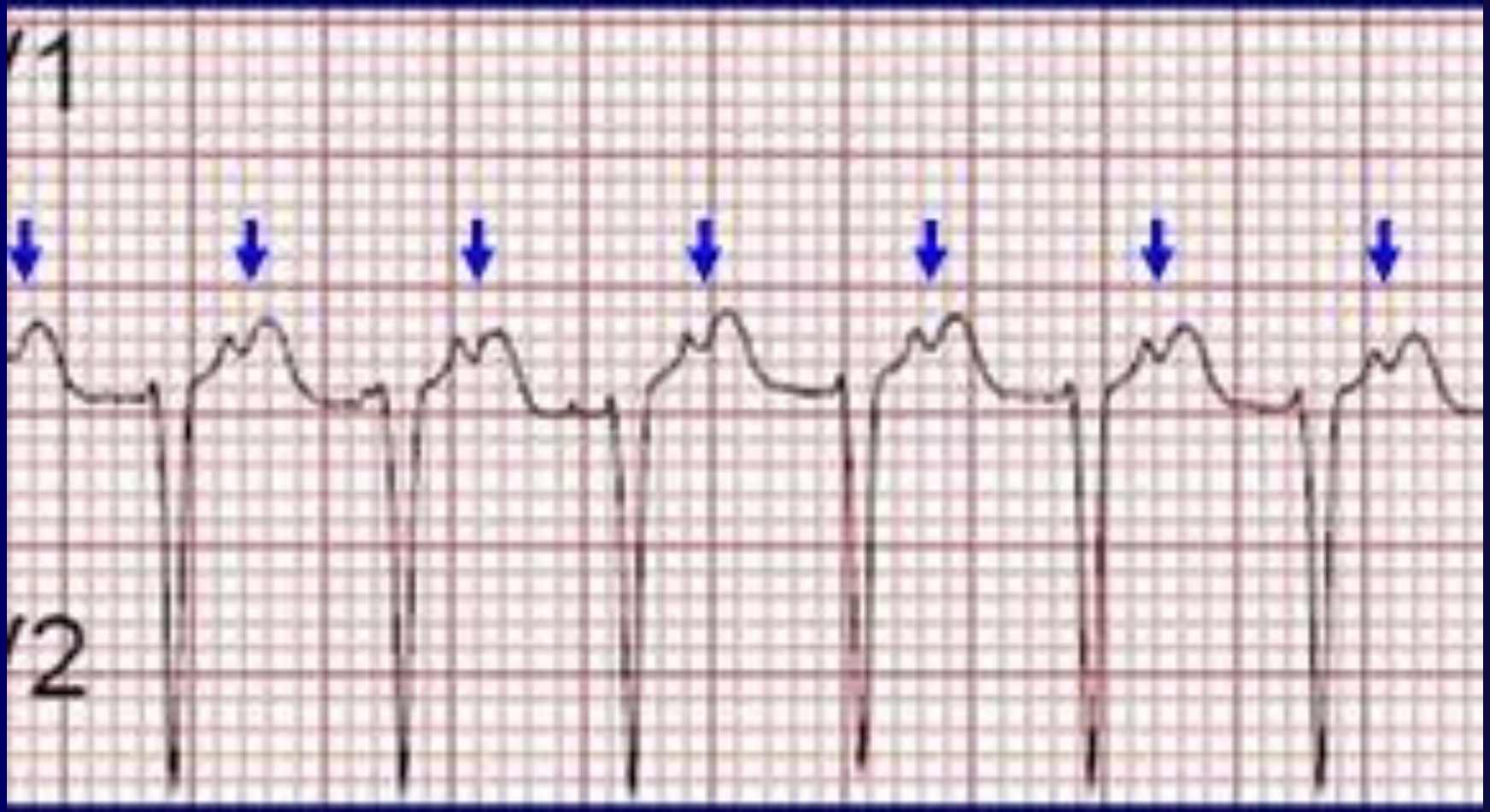
Vagal maneuvers

- USED TO SLOW FAST HEART RATES
- Gagging.
- Holding your breath and bearing down (Valsalva maneuver).
- Immersing your face in ice-cold water (diving reflex).
- Coughing.



Sinus Node Reentry

- Onset and termination: Sudden
- Heart rate: 100–160 bpm
- P wave: Identical to normal sinus rhythm P wave
- R-P relationship: Long


V1
V2

Treatment

- The arrhythmia can be terminated quickly with intravenous adenosine, verapamil, or diltiazem, or via carotid massage
- Long-term treatment uses β-blockers and calcium channel blockers
- catheter ablation described success

ATRIAL FLUTTER

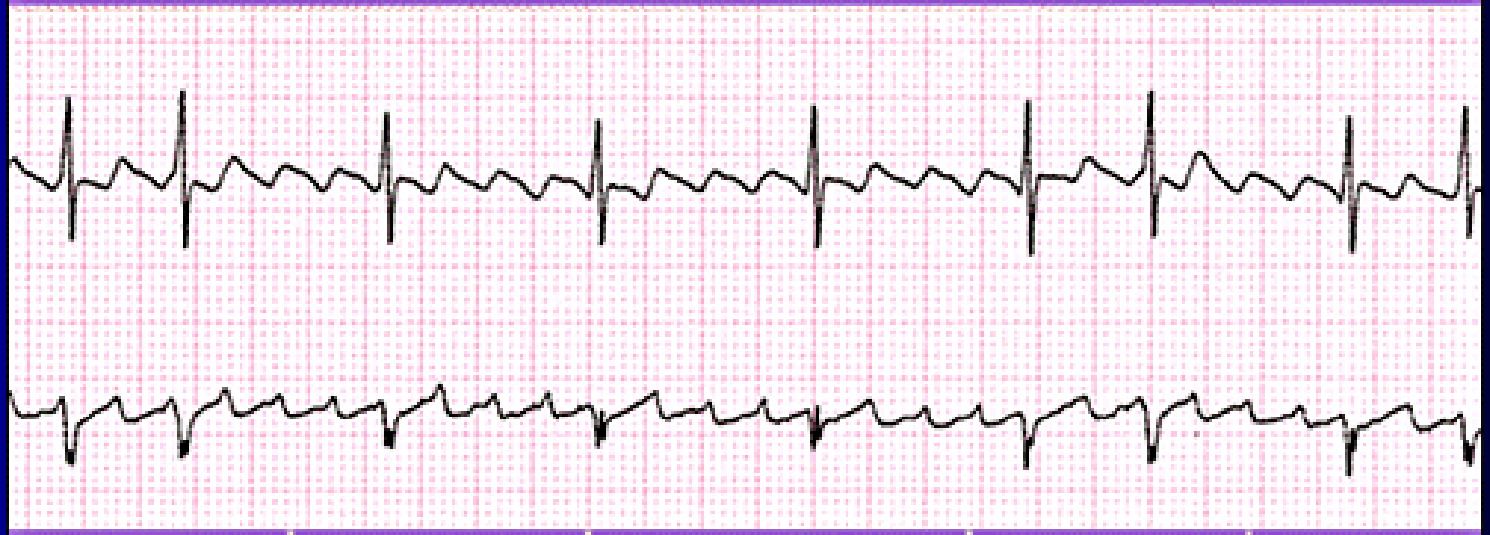
- Onset and termination: Sudden.
- Heart rate: Usually a multiple of 300.
- P waves: Flutter waves at 250–340 bpm.
- R-P relationship: Undefined due to flutter waves.
- Prominent neck vein pulsations of about 300/min.

Atrial flutter

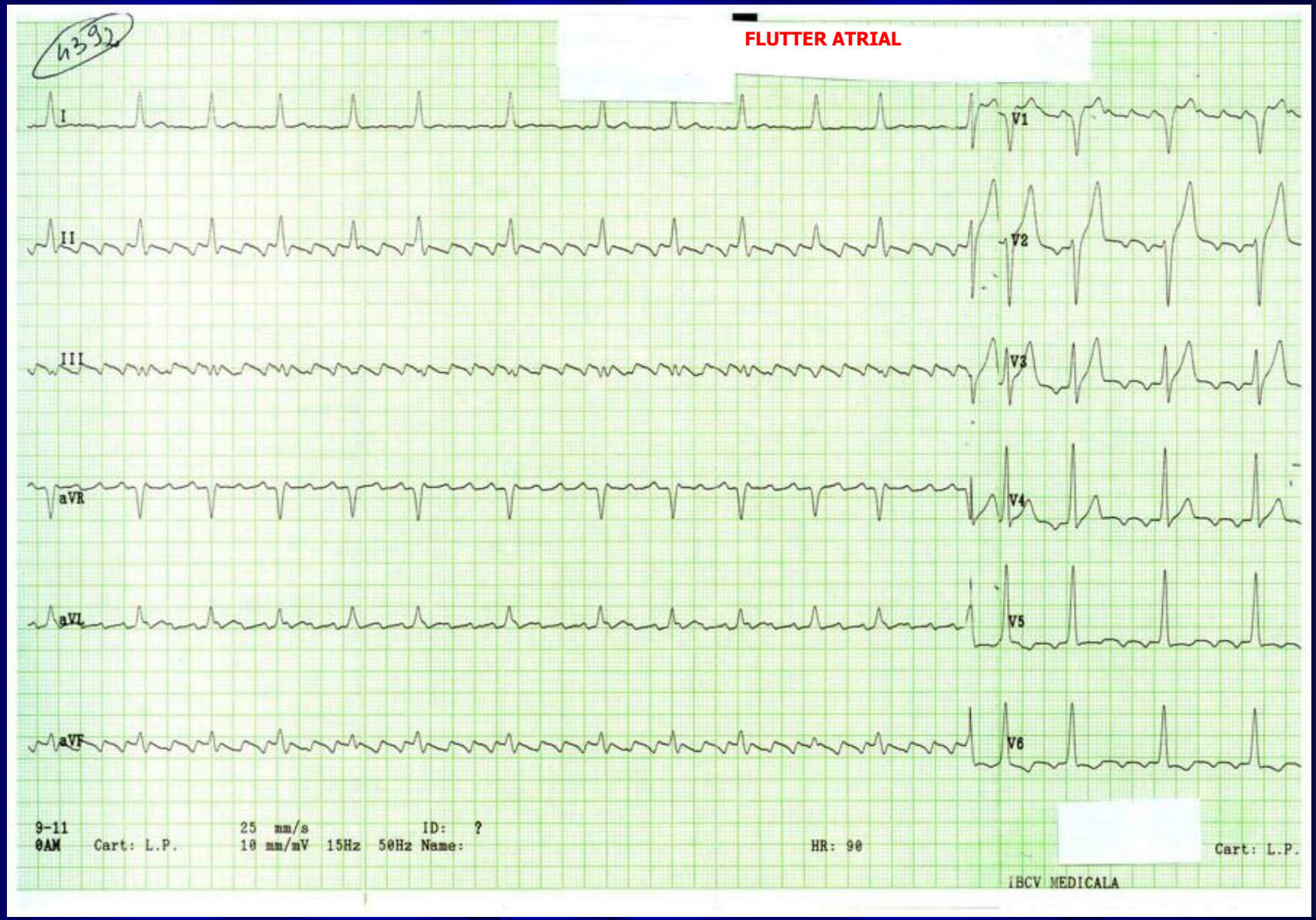
- is usually associated with organic heart disease
- produces a "sawtooth" appearance (F waves)
- the ventricular rate depends on conduction through the AV node
- 1:1 conduction may occur in rare circumstances
- 2:1 or 4:1 block is the usual scenario
- If flutter is suspected but F waves are not clearly visible, vagal maneuvers or pharmacologic agents, such as adenosine, can help unmask the flutter waves by enhancing the degree of AV block

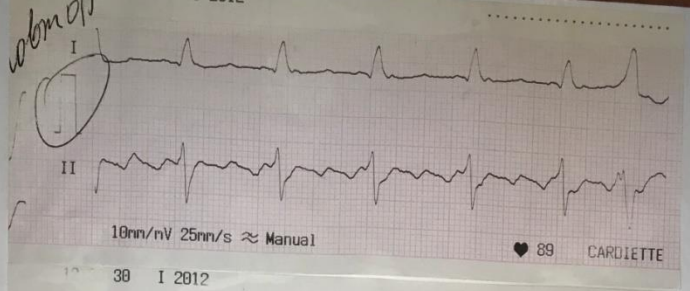
ECG

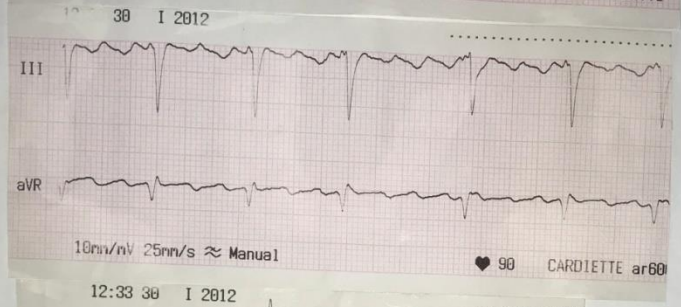
Atrial Flutter

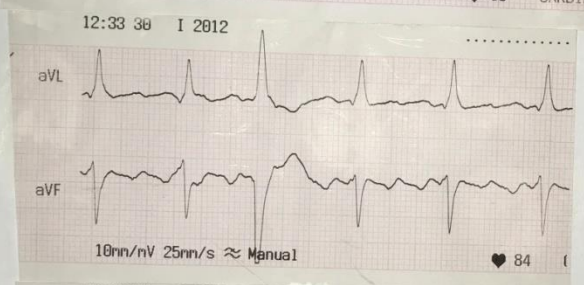


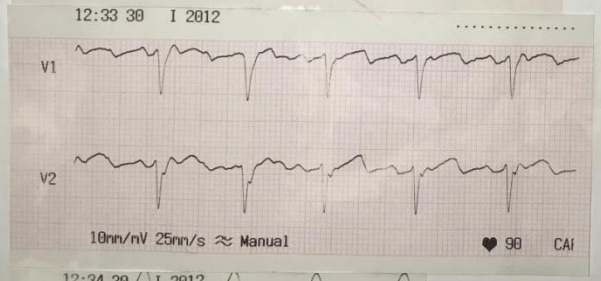
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

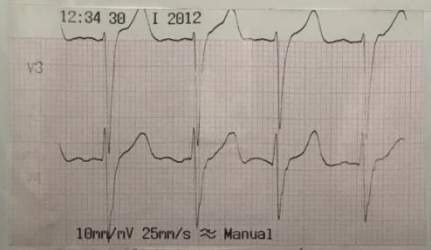

4392
FLUTTER ATRIAL
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
9-11
0AM
Cart: L.P.
25 mm/s
10 mm/mV 15Hz 50Hz
ID: ?
Name:
HR: 90
Cart: L.P.
iBCV MEDICALA

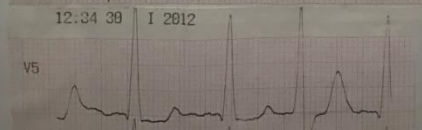

I
II
10mm/mV 25mm/s Manual
89 CARDIETTE

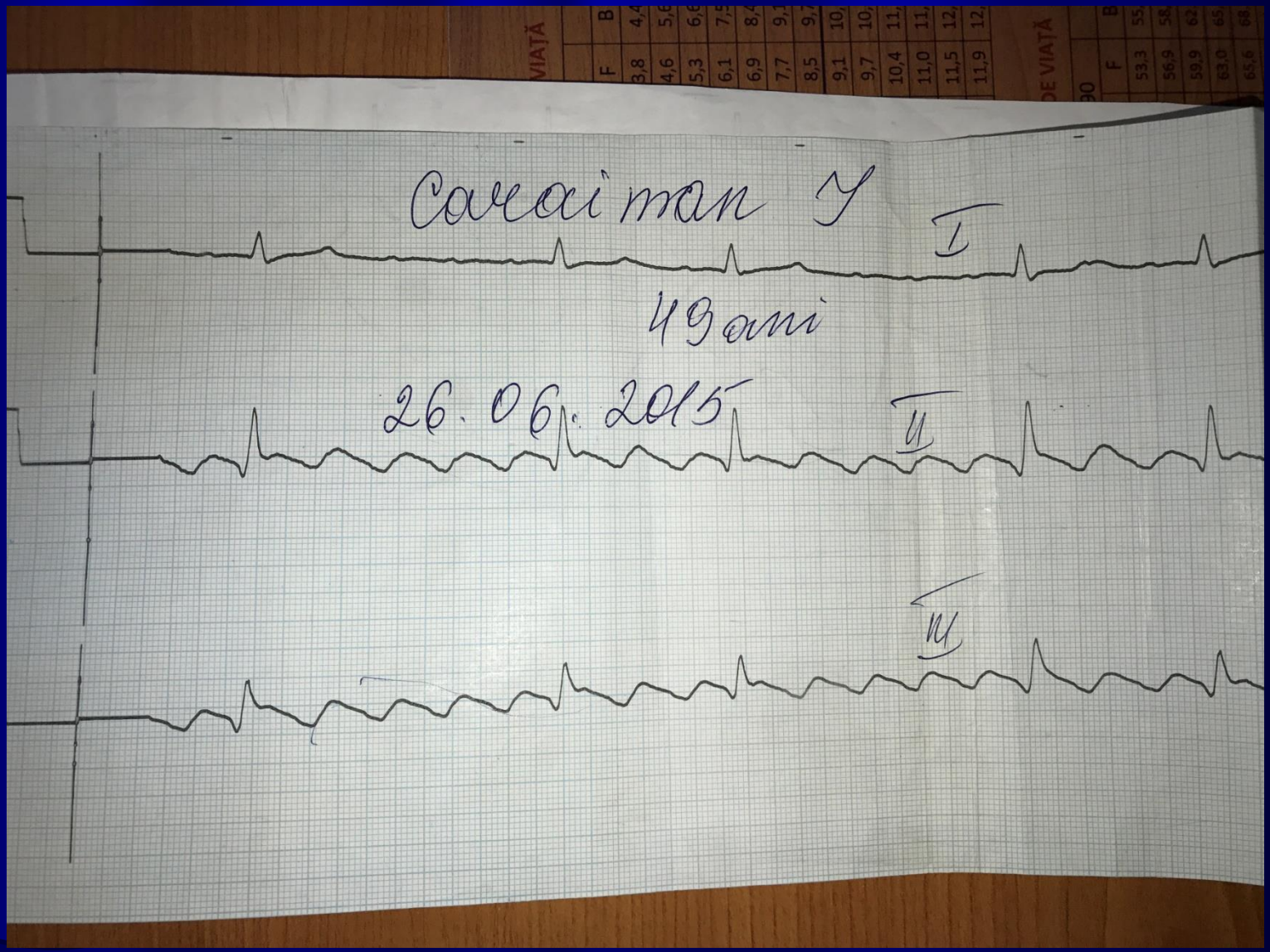

30 I 2012
III
aVR
10mm/mV 25mm/s Manual
90 CARDIETTE


12:33 30 I 2012
aVL
aVF
10mm/mV 25mm/s Manual
84


12:33 30 I 2012
V1
V2
10mm/mV 25mm/s Manual
90


12:34 30 I 2012
V3
10mm/mV 25mm/s Manual

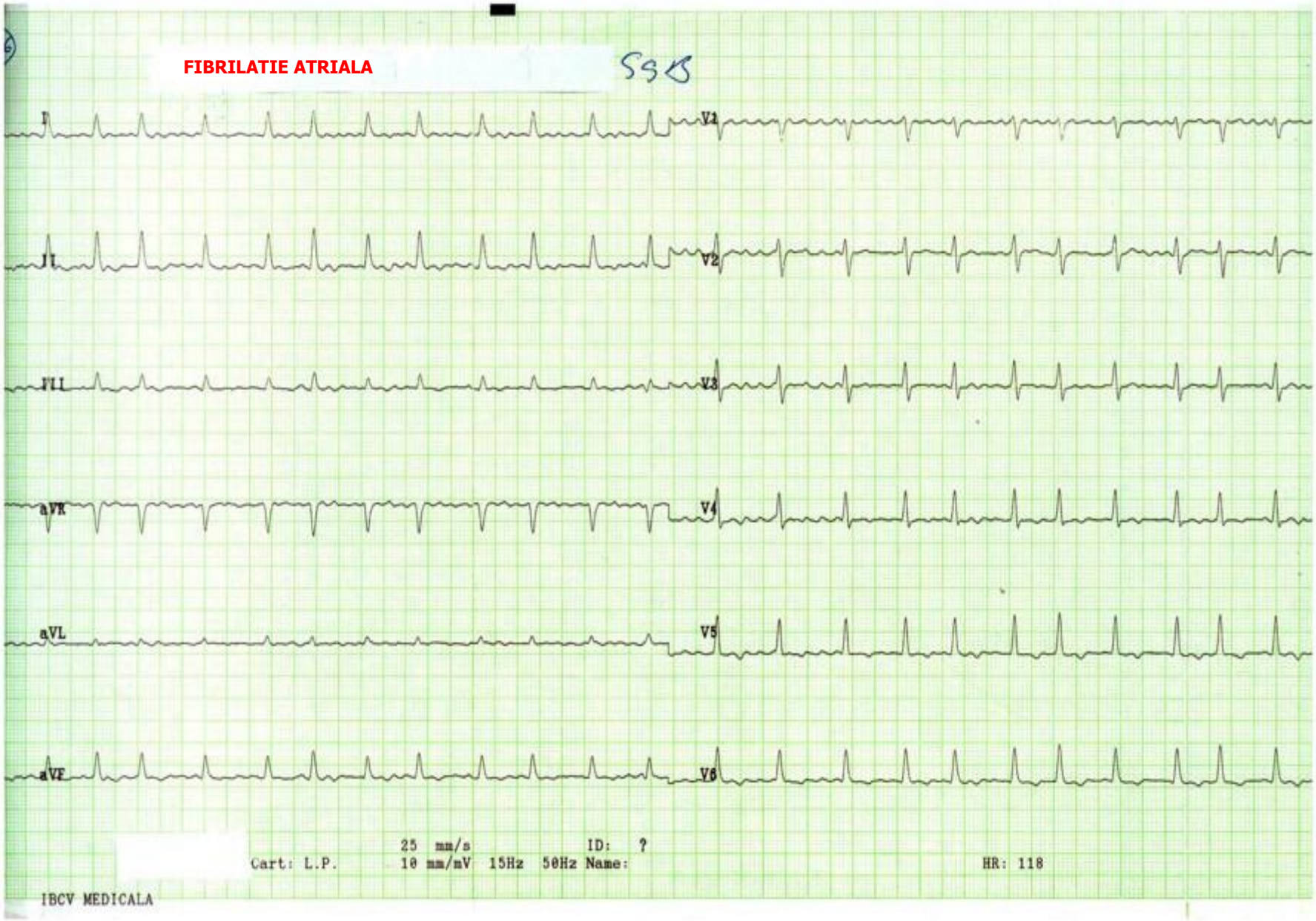

12:34 30 I 2012
V5

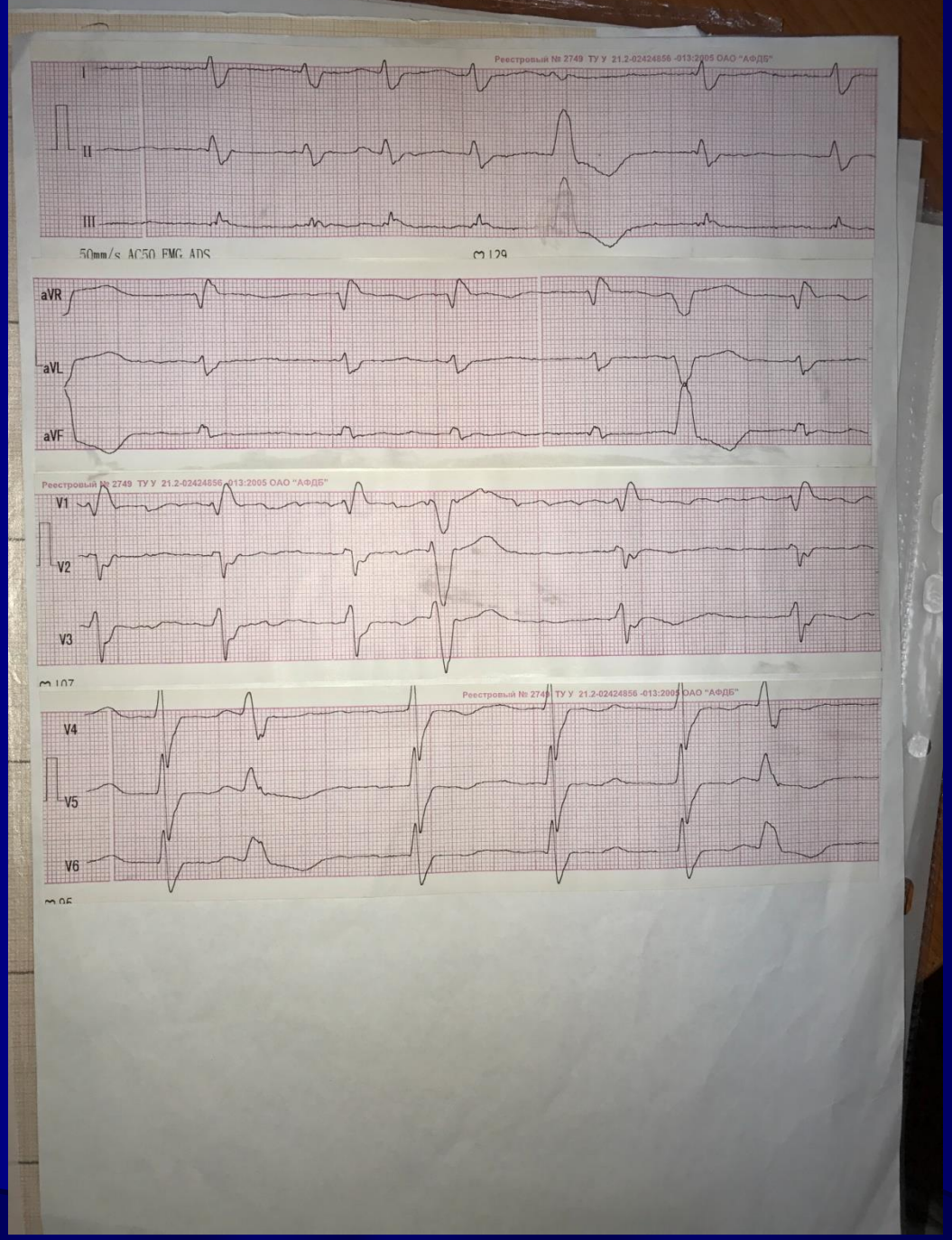

Cavaleiman Y
49 ani
26.06.2015
I
II
III

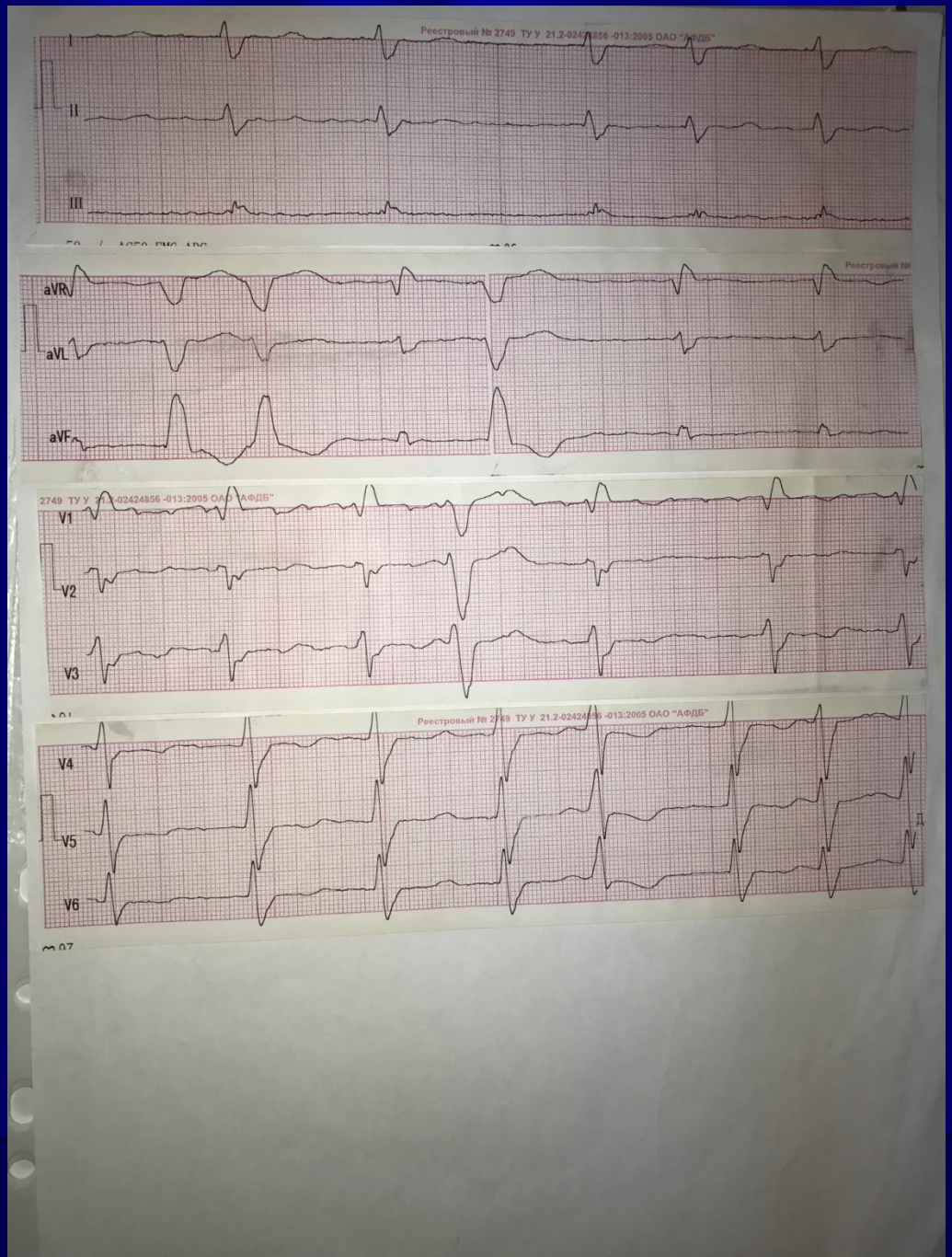
R
C
F

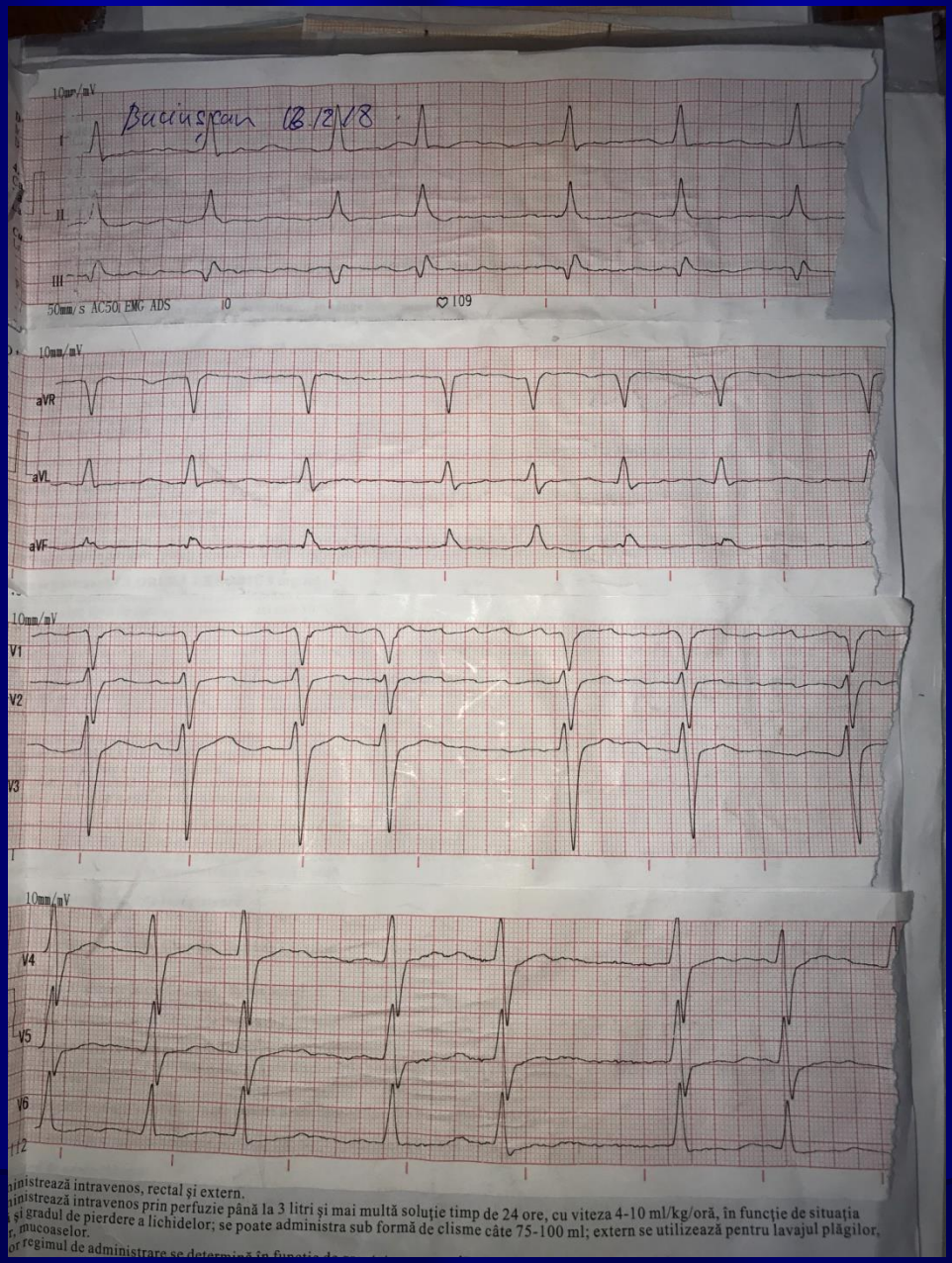
Atrial fibrillation

- Rhythm – ventricular rhythm usualy irregularly
- Rate – atrial rate usualy 400 to 600 beats/min, ventricular rate variable
- P waves prezent; erratic, wavy baseline
- RR interval – not measurable
- QRS duration 0.11 sec or less unless abnormal conductued


FIBRILATIE ATRIALA
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
25 mm/s
10 mm/mV 15Hz 50Hz
ID: ?
Name:
HR: 118
Cart: L.P.
IBCV MEDICALA


Реестровый № 2749 ТУ У 21.2-02424856 -013:2005 ОАО "АФДБ"
I
II
III
50mm/s AC50 EMG ADS
aVR
aVL
aVF
Реестровый № 2749 ТУ У 21.2-02424856 -013:2005 ОАО "АФДБ"
V1
V2
V3
Реестровый № 2749 ТУ У 21.2-02424856 -013:2005 ОАО "АФДБ"
V4
V5
V6


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6



inistrează intravenos, rectal şi extern.
inistrează intravenos prin perfuzie până la 3 litri şi mai multă soluţie timp de 24 ore, cu viteza 4-10 ml/kg/oră, în funcţie de situaţia
şi gradul de pierdere a lichidelor; se poate administra sub formă de clisme câte 75-100 ml; extern se utilizează pentru lavajul plăgilor,
r, mucoaselor.
or regimul de administrare se determină în funcţie de

Vaughan-Williams Classification of Antiarrhythmic Drugs

- Class I sodium-channel blockade Reduce phase 0 slope and peak of action potential.\
- IA - moderate Moderate reduction in phase 0 slope; increase APD; increase ERP
- IB - weak Small reduction in phase 0 slope; reduce APD; decrease ERP
- IC - strong Pronounced reduction in phase 0 slope; no effect on APD or ERP
- II beta-blockade Block sympathetic activity; reduce rate and conduction
- III potassium-channel blockade Delay repolarization (phase 3) and thereby increase action potential duration and effective refractory period
- IV calcium-channel blockade Block L-type calcium-channels; most effective at SA and AV nodes; reduce rate and conduction.

Prevent recurrences

- class Ia and Ic agents are effective
- Class III agents, such as sotalol and amiodarone, can also work very well
- Dofetilide, a newer class III agent, which blocks the rapid form of the delayed rectifier current
- Drugs that are contraindicated with its use include verapamil, ketoconazole, cimetidine, trimethoprim, prochlorperazine, megestrol, and hydrochlorothiazide

Conversion

- patient's status will dictate whether to perform cardioversion immediately (48 hours)
- Immediate cardioversion can be accomplished with synchronized cardioversion
- rapid atrial pacing to interrupt the macroreentrant circuit, or intravenous infusion of an antiarrhythmic agent
- For DC cardioversion, as little as 25 J may be all that is required; however, at least 50 J is recommended to avoid extra shocks, and 100 J will terminate almost all episodes of atrial flutter

Pharmacologic cardioversion Rithm control

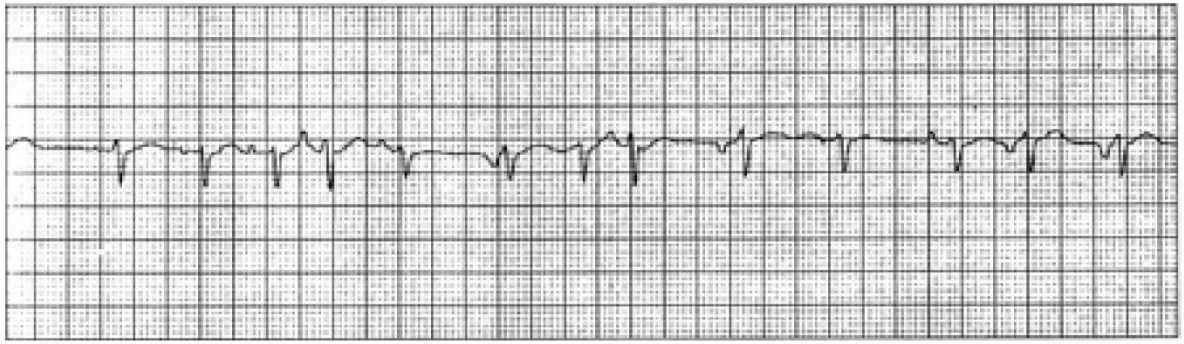
- Ibutilide is a unique class III antiarrhythmic agent with a rate of conversion of approximately 60% in patients with atrial flutter of less than 45 days duration
- Cardioversion can be expected within 30 minutes of administration. The major complication with this agent is the development of torsades de pointes
- Procainamide is another intravenous agent that can be given to pharmacologically convert atrial flutter.

Rate Control

- controlling the ventricular rate in atrial flutter is more difficult than in atrial fibrillation
- β-Blockers and calcium channel blockers are moderately effective in controlling the rate
- Digoxin is less helpful since it only weakly blocks the AV node conduction
- Intravenous amiodarone has been shown to be at least as efficacious as digoxin

MULTIFOCAL ATRIAL TACHYCARDIA

- Heart rate: Up to 150 bpm.
- P waves: Three or more distinct P waves in a single lead.
- Variable P-P, P-R, and R-R intervals



- Three ECG criteria must be met to diagnose MAT
- (1) The presence of at least three distinct P wave morphologies recorded in the same lead
- (2) The absence of one dominant atrial pacemaker
- (3) Varying P-P, P-R, and R-R intervals

Treatment

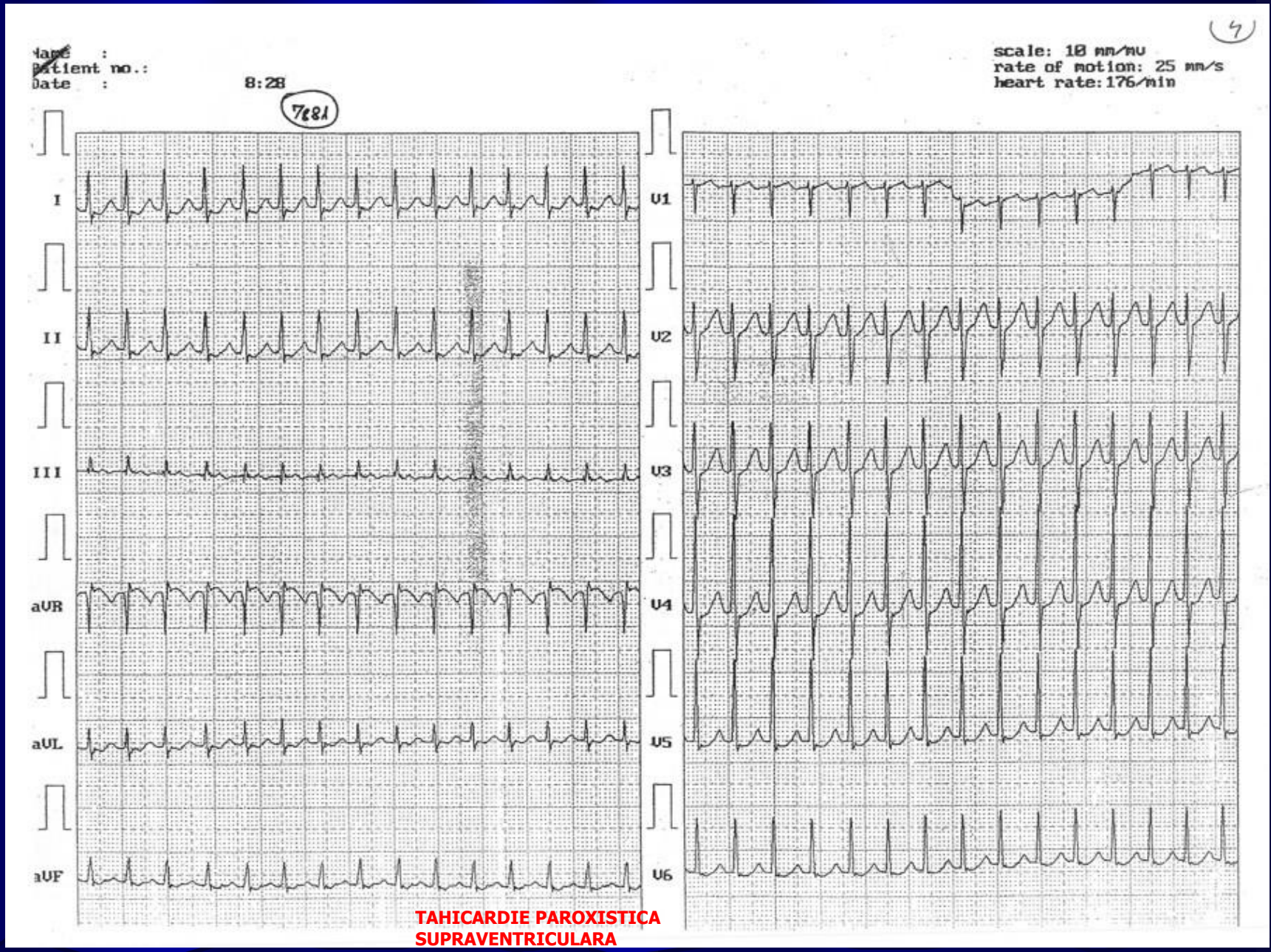
Oral and intravenous verapamil and several formulations of intravenous β-blockers have been effective to varying degrees in either slowing the heart rate (without terminating the rhythm) or in converting the arrhythmia to sinus rhythm

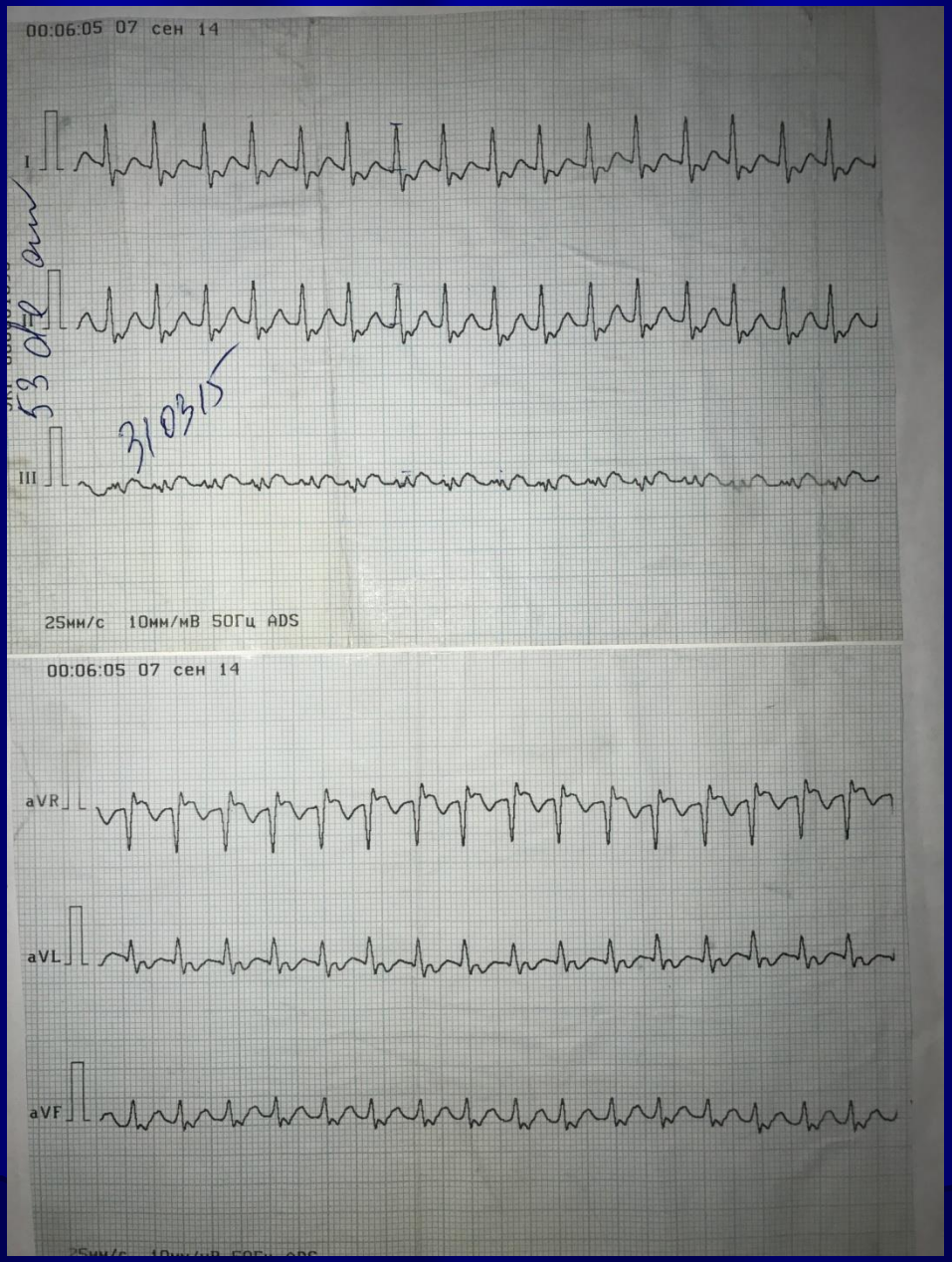
Intravenous magnesium and potassium, even in patients with serum levels of these electrolytes within the normal range, convert a significant percentage of these patients to sinus rhythm.

Digoxin is not effective in treating this condition. Moreover, treatment with digoxin may precipitate digitalis intoxication

ATRIAL TACHYCARDIA

- Onset and termination: Sudden.
- Heart rate: 100–180 bpm.
- P wave: Distinct P waves that differ from sinus P waves
- R-P relationship: Long


Name :
Patient no.:
Date :
8:28
scale: 10 mm/mV
rate of motion: 25 mm/s
heart rate: 176/min
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
TAHICARDIE PAROXISTICA
SUPRAVENTRICULARA


00:06:05 07 сен 14
I
II
III
53 Oct an
310315
25мм/с 10мм/мВ 50Гц ADS
00:06:05 07 сен 14
aVR
aVL
aVF

Pharmacologic Therapy

- β-blockers and calcium channel blockers are at least partially effective, particularly if the underlying mechanism of the tachycardia is abnormal automaticity or triggered activity. Because of their safety profile, these drugs are usually first-line medical therapy

The use of class IC antiarrhythmic drugs may be somewhat successful

Flecainide and propafenone are often well tolerated in patients without structural heart disease and thus can be considered a reasonable first-line antiarrhythmic therapy

there may be a small subset of lidocaine-sensitive atrial tachycardias in which mexilitine may be effective. Sotalol may also be effective, in part because of its inherent β-blocker (class II) properties

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

- Onset and termination: Sudden.
- Heart rate: Usually 120–200 bpm but can be faster; neck pulsations correspond to heart rate.
- P waves: Retrograde P waves; P waves not visible in 90% of cases.
- R-P relationship: Short, if P waves visible.

JUNCTIONAL TACHYCARDIA (ACCELERATED AV JUNCTIONAL RHYTHM)

- Onset and termination: Gradual.
- Heart rate: 70–120 bpm.
- P waves: Retrograde.
- R-P relationship: Short, if P waves visible.

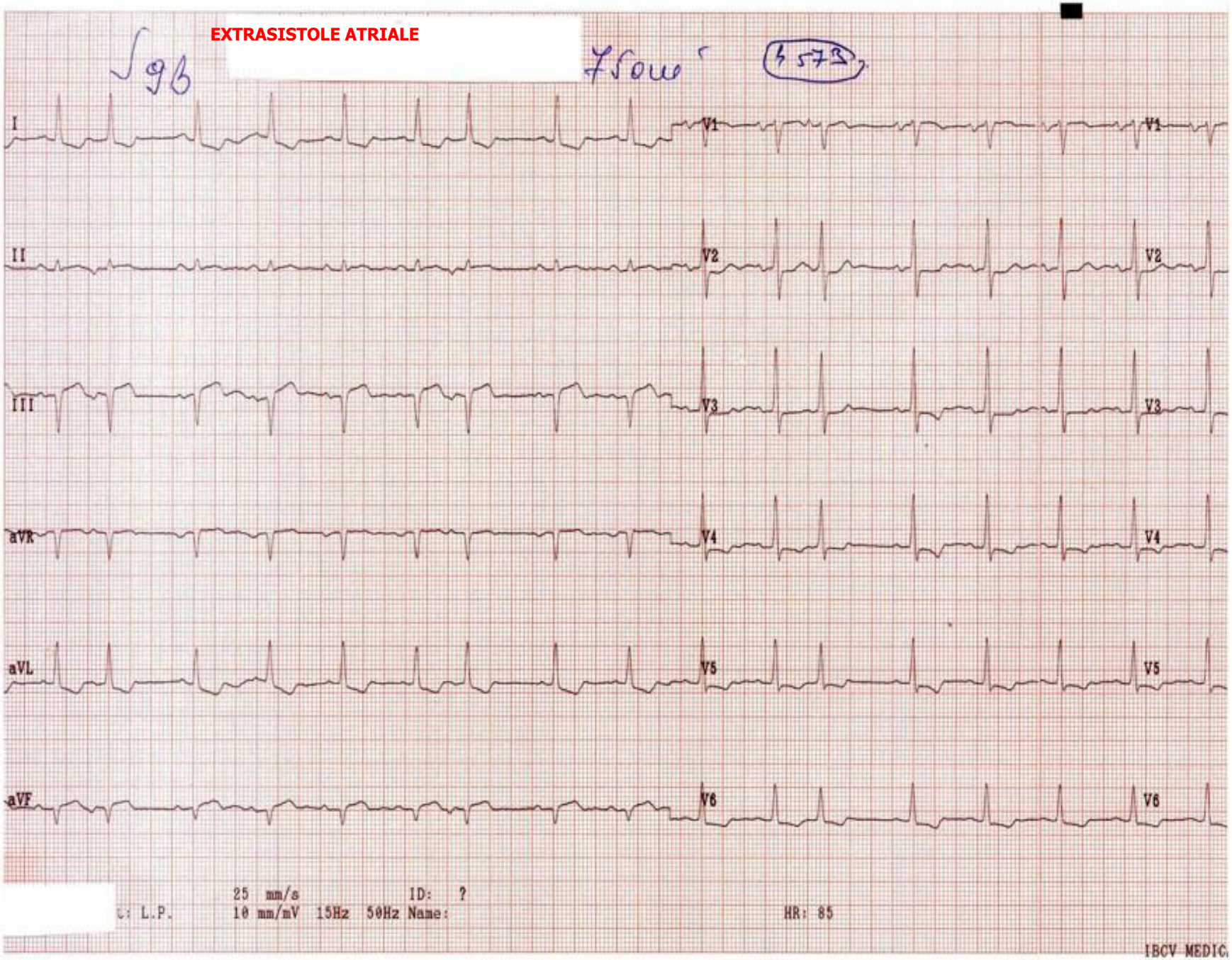
ATRIOVENTRICULAR RECIPROCATING TACHYCARDIA

- Onset and termination: Sudden.
- Heart rate: 140–250 bpm.
- P wave: Ectopic.
- R-P relationship: Short.
- Delta wave on baseline ECG if bypass tract conducts antegrade.

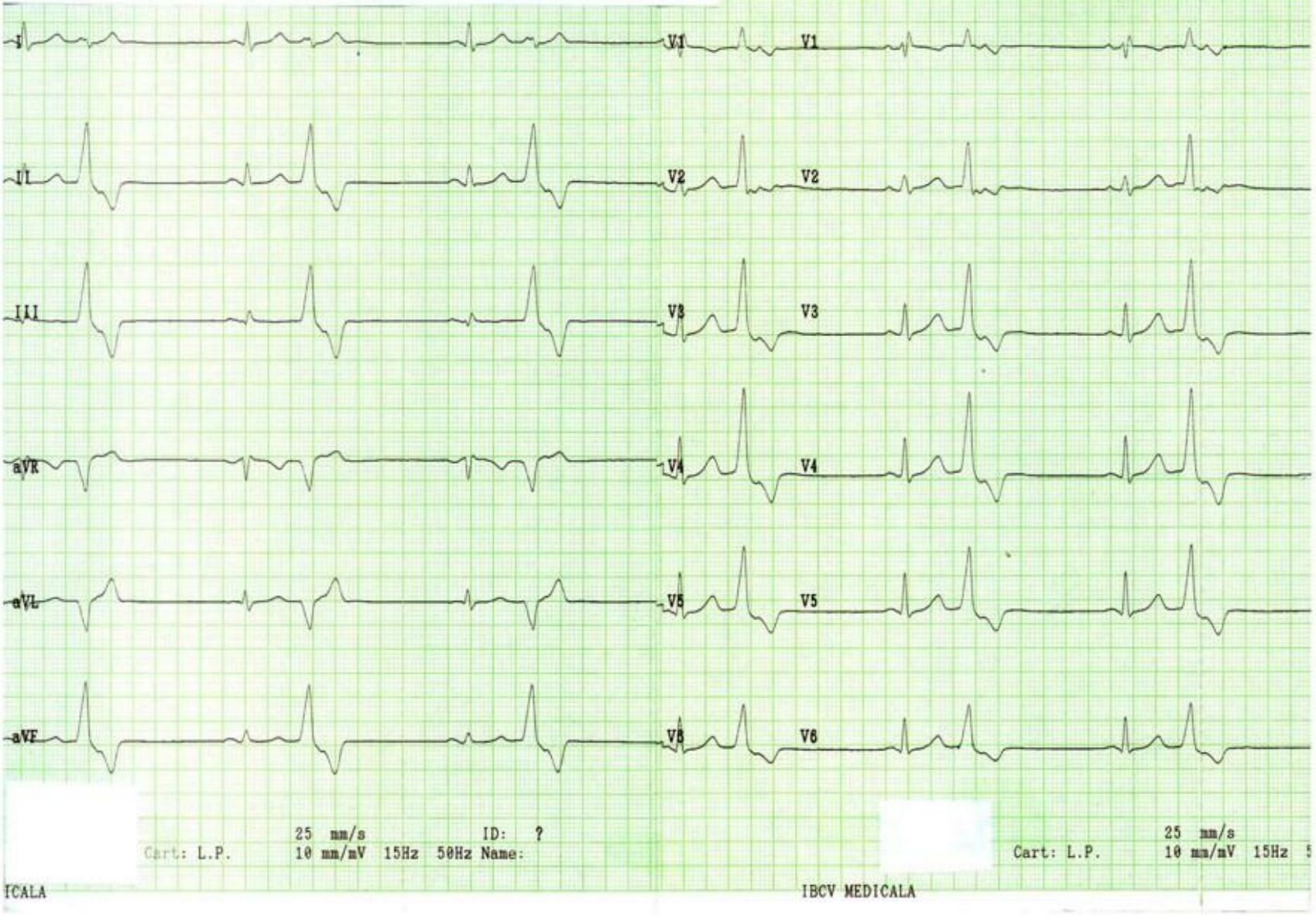
Ventricular tachycardia

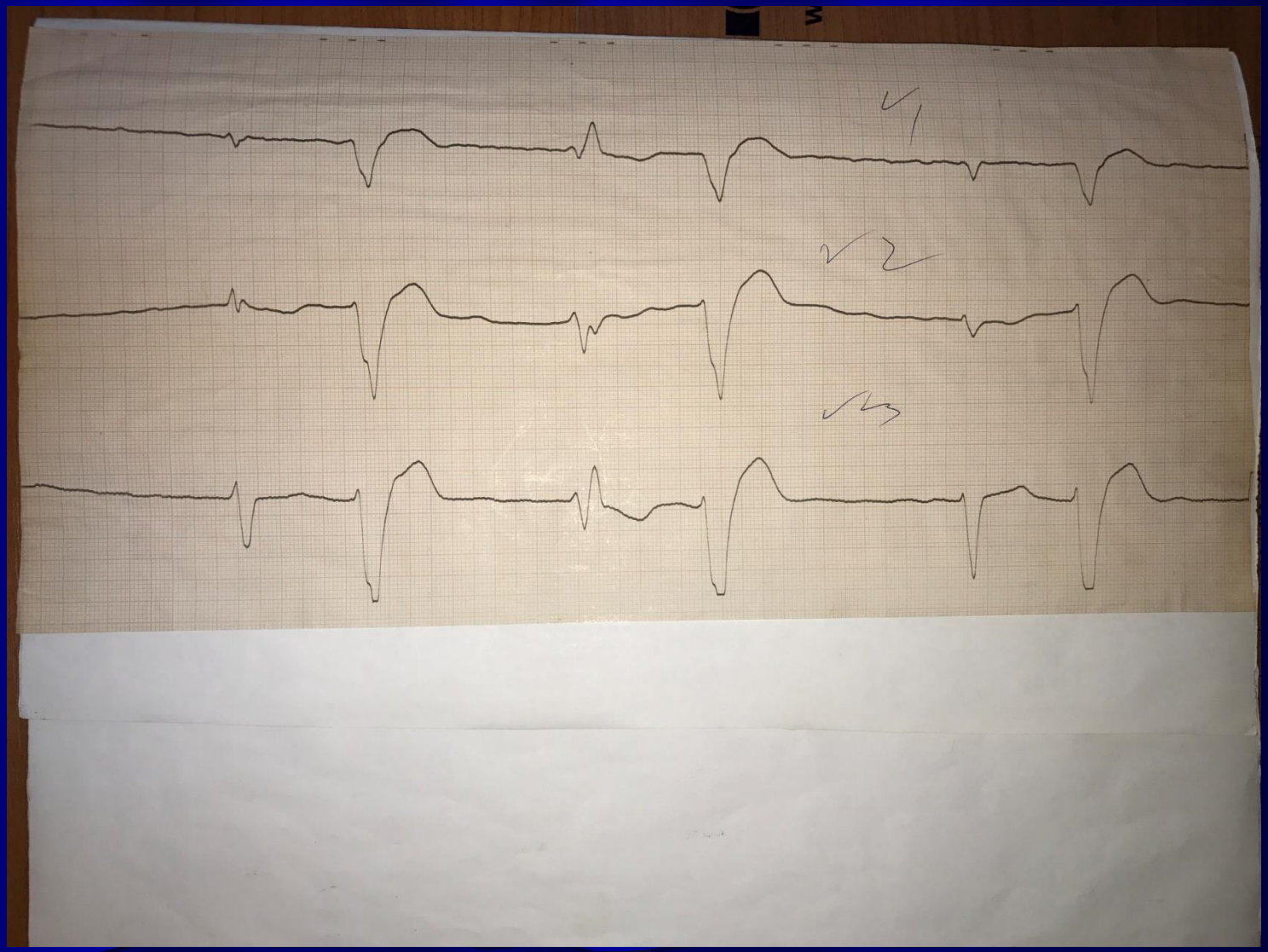
- VT is one of the most common health problems encountered in clinical practice, can best be appreciated in terms of its various clinical manifestations, which include ventricular fibrillation (sudden cardiac death, syncope or near syncope, and wide QRS tachycardia

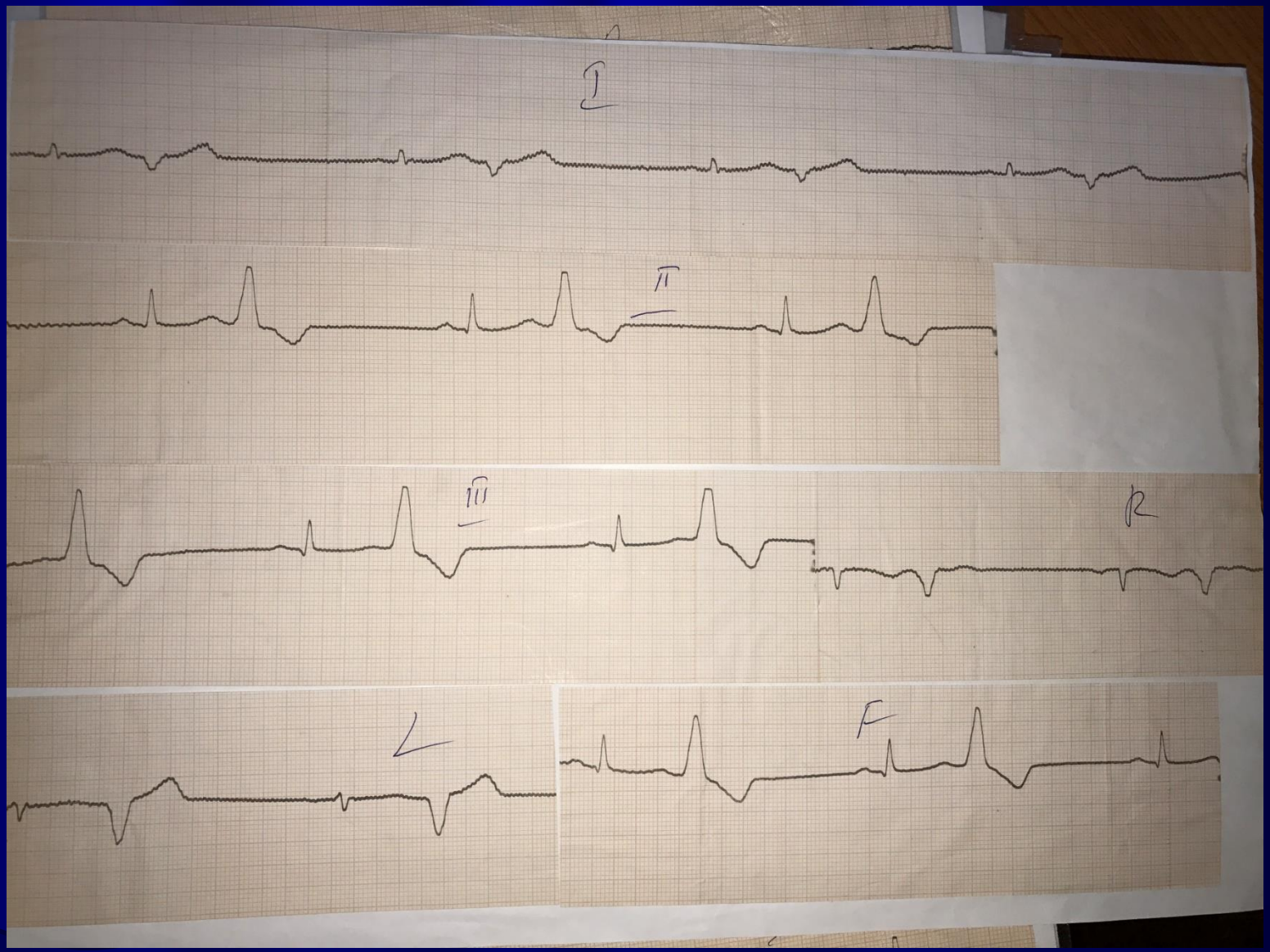
EXTRASISTOLE ATRIALE

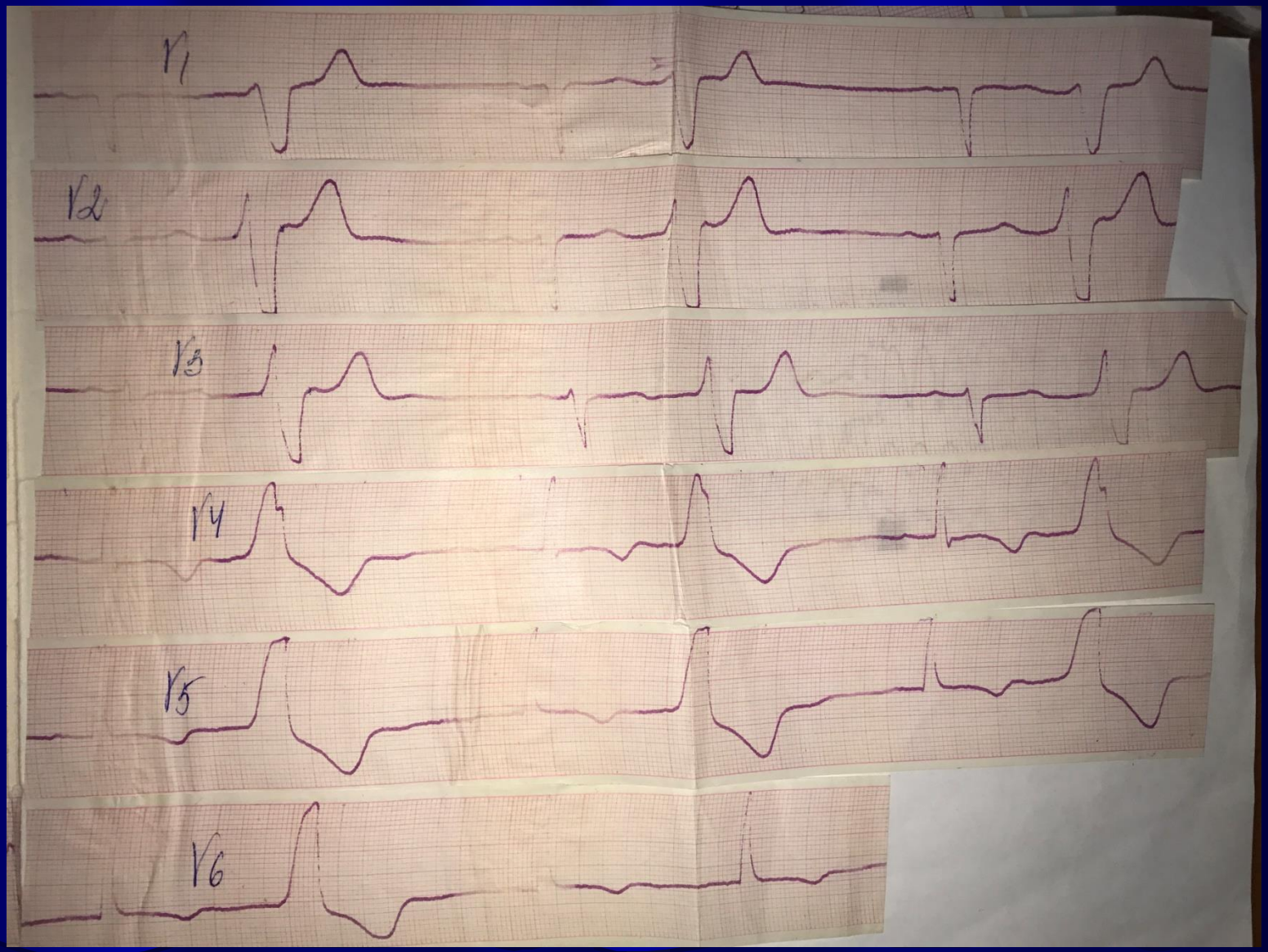


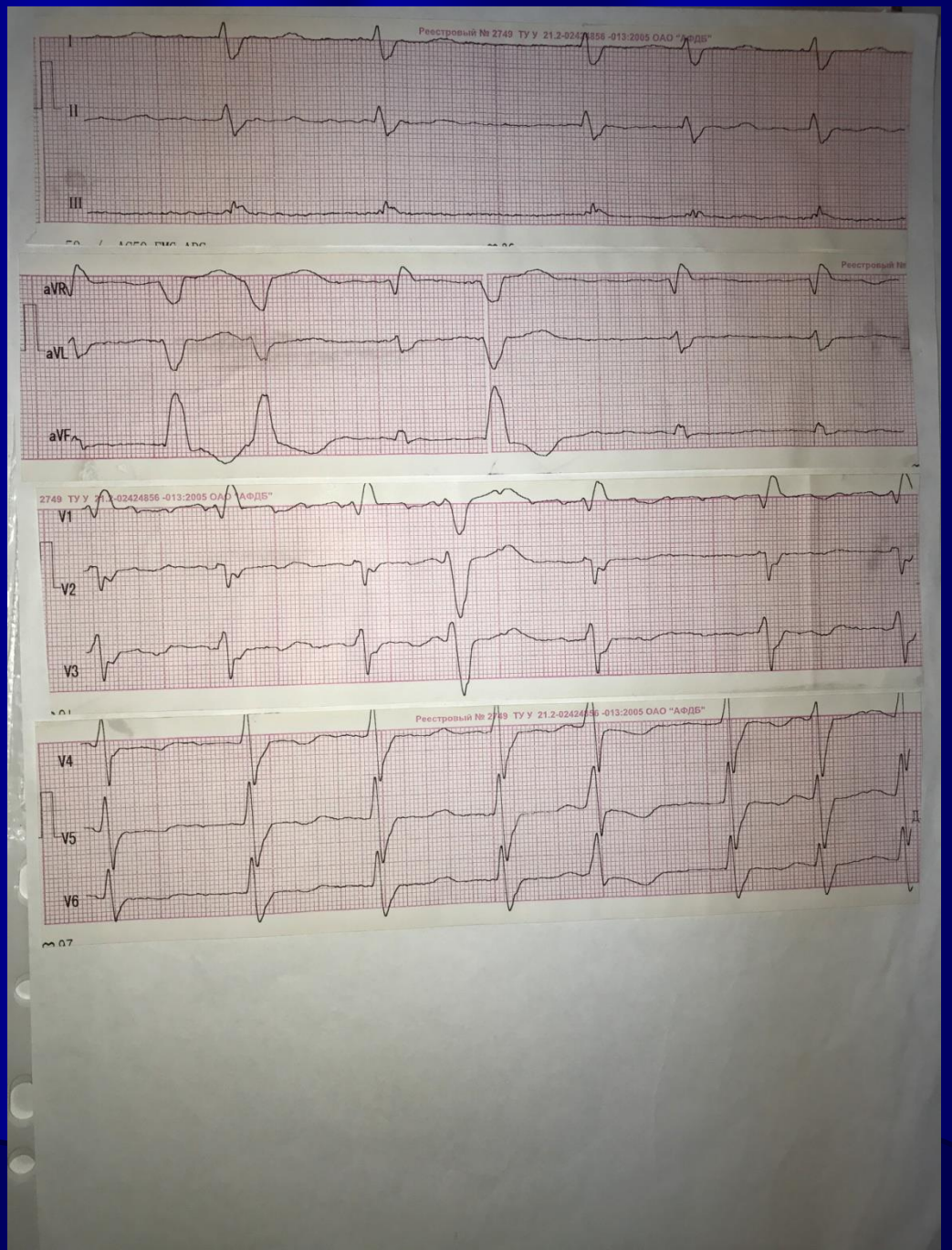
EXTRASISTOLE VENTRICULARE MONOMORFE BIGEMINATE




V1
V2
V3

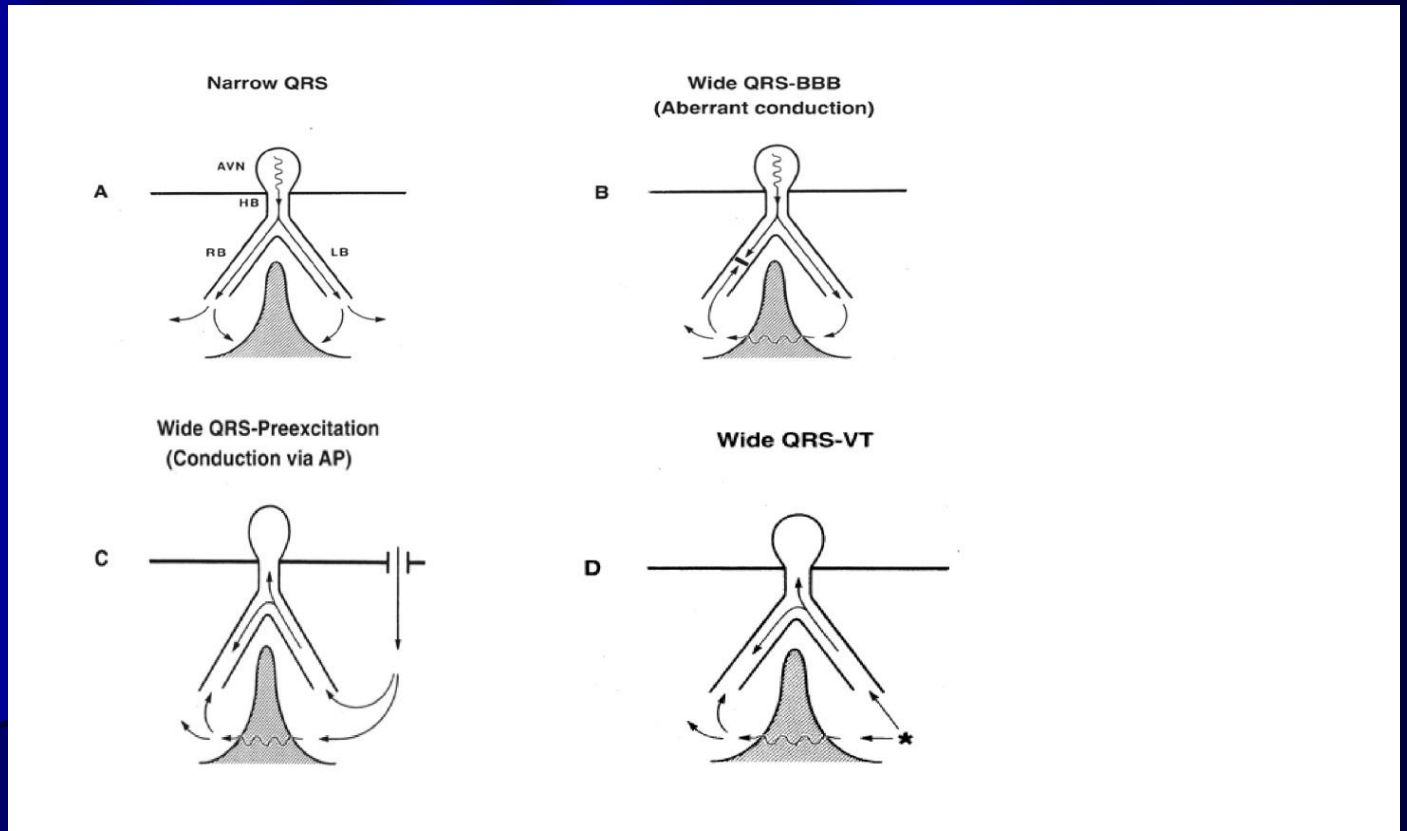

I
II
III
R
L
F


V1
V2
V3
V4
V5
V6


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Mechanism Ventricular tachycardia

- A: Narrow QRS from simultaneous activation of the right and left ventricles
- Wide QRS shown in B–D, there is sequential rather than simultaneous activation of the left and right ventricle and a variable amount of muscle-to-muscle conduction.



Classification and Causes of Common Ventricular Tachycardias

Monomorphic ventricular tachycardia

Chronic coronary artery disease

Idiopathic dilated cardiomyopathy

Right ventricular dysplasia

No structural heart disease

ECG configuration

Right bundle branch block configuration

Left bundle branch block configuration

Repetitive monomorphic ventricular tachycardia

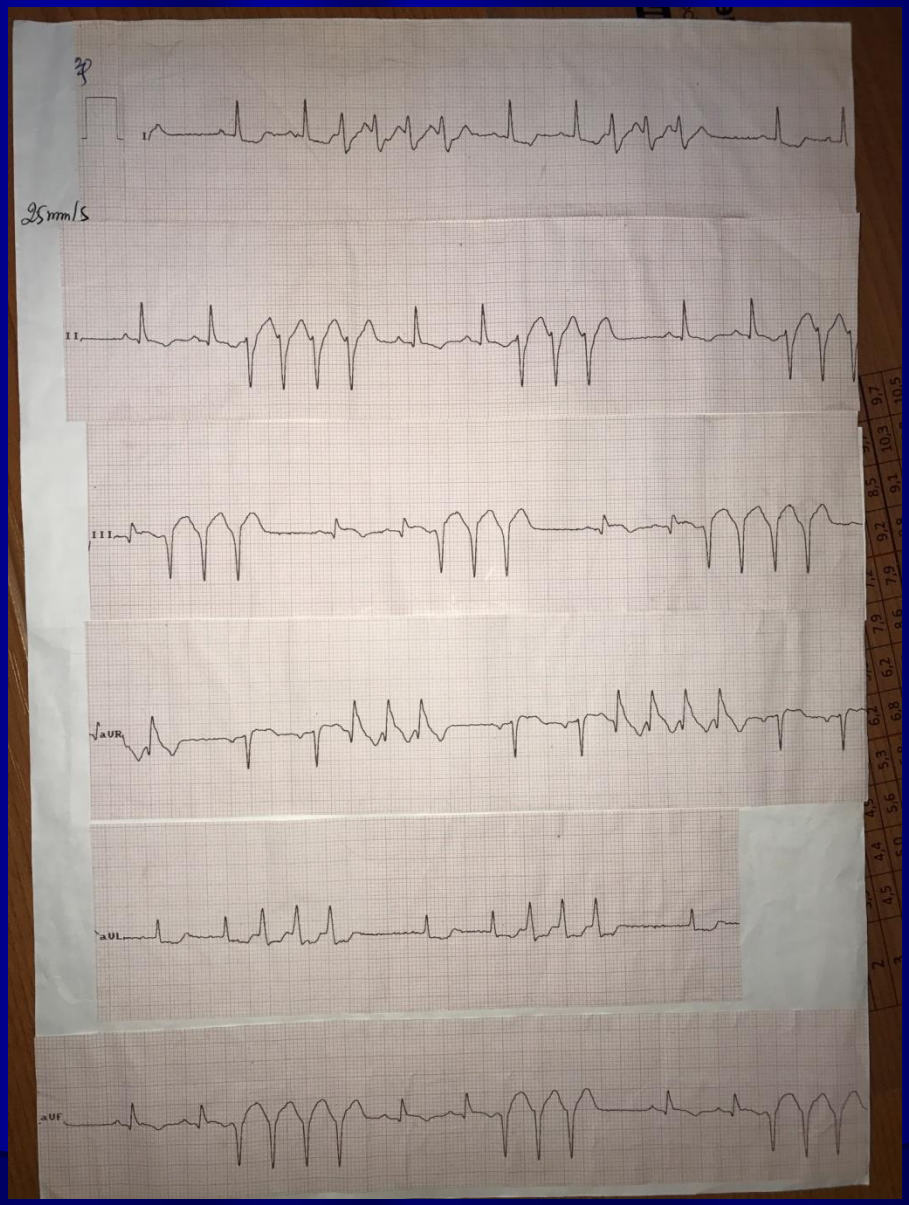
Other forms

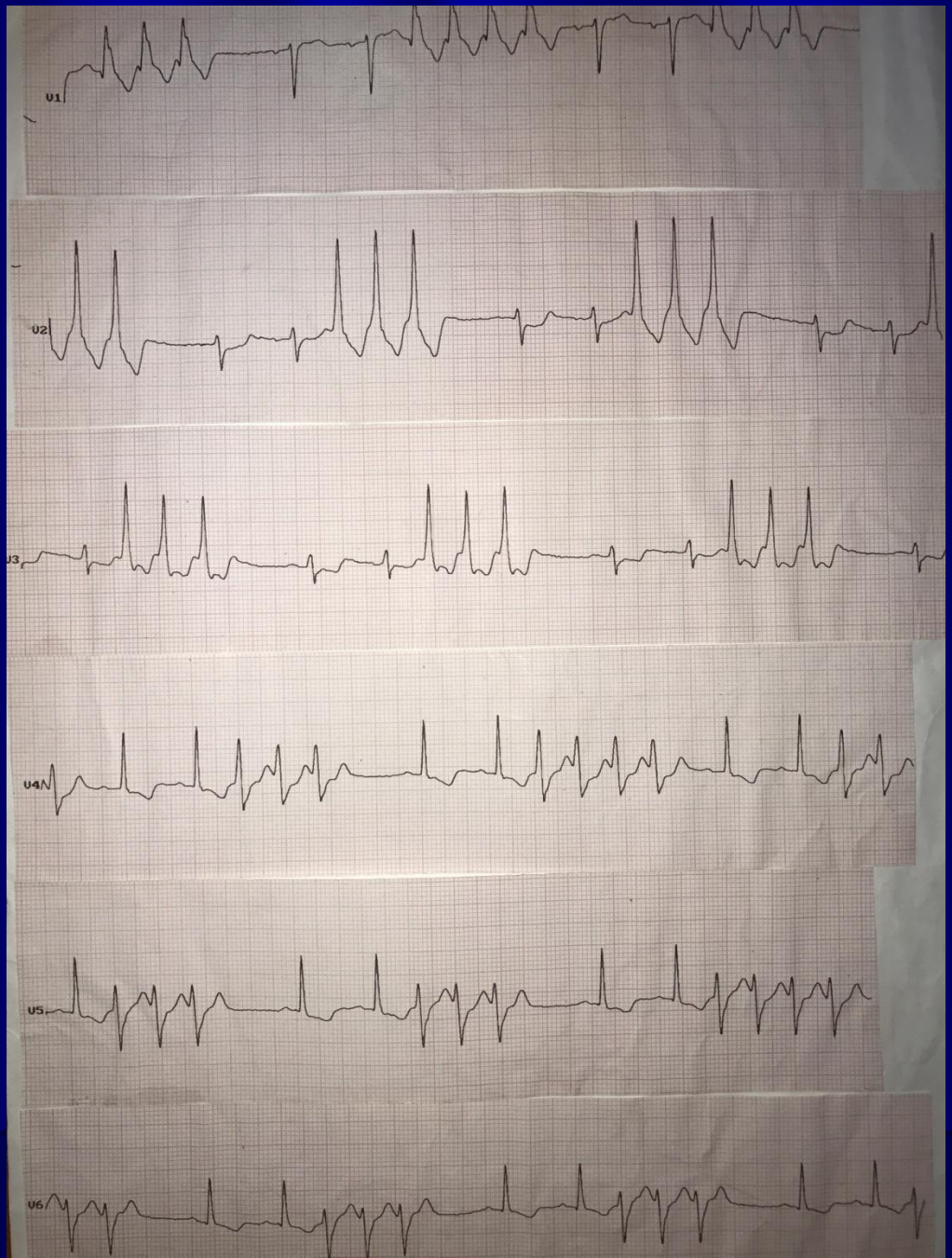
Polymorphic ventricular tachycardia

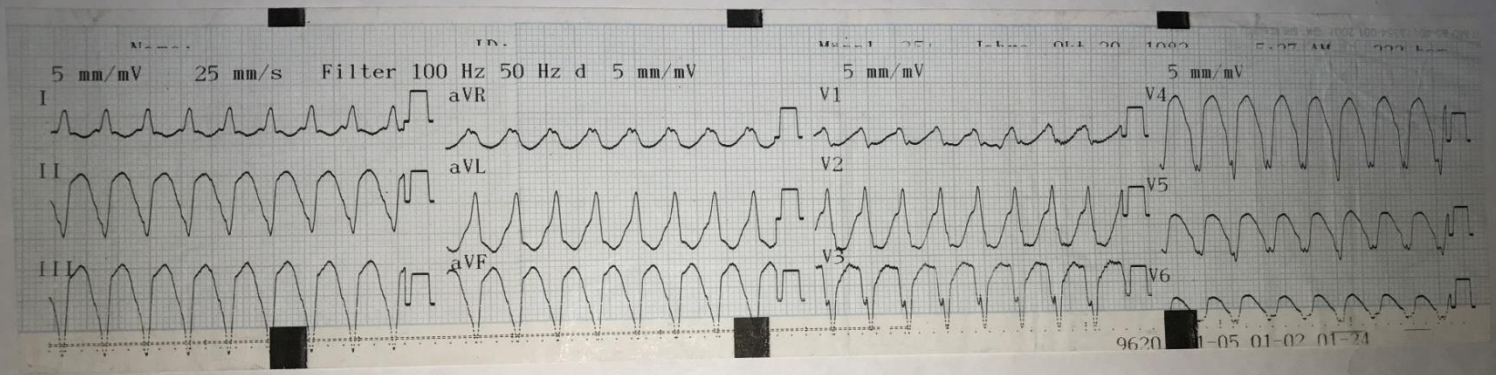
- Prolonged QT interval (torsades de pointes)
- Congenital
- Acquired
- *ECG configuration*
- Normal QT interval
- Acute ischemia

Classifcation

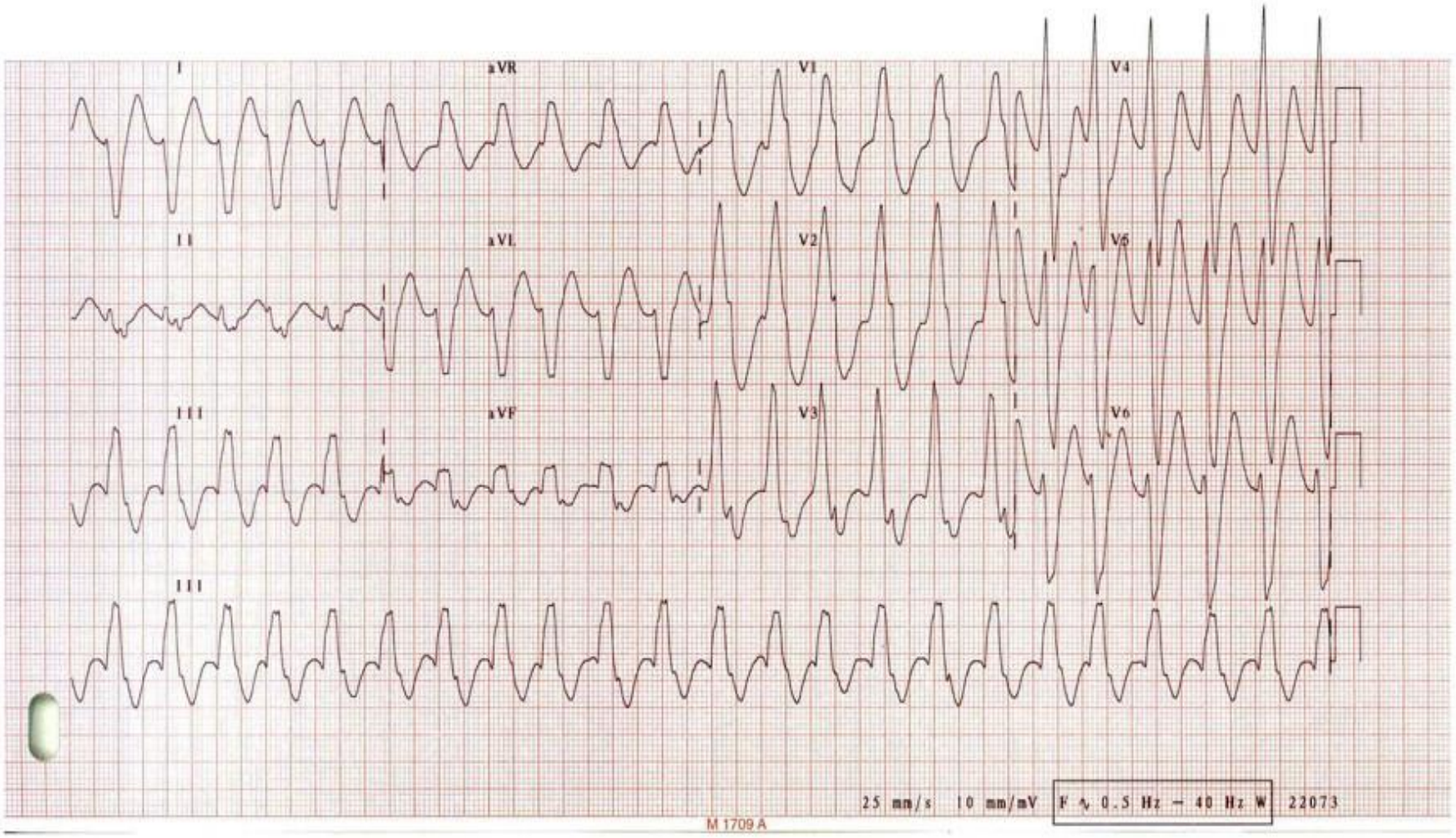
- **Nonsustained**: Three or more consecutive QRS complexes of uniform configuration of ventricular origin at a rate of more than 100 bpm.
- **Sustained**: Lasts more than 30 seconds; requires intervention for termination.
- Monomorphic ventricular tachycardia.
- Polymorphic ventricular tachycardia: Beat-to-beat variation in QRS configuration

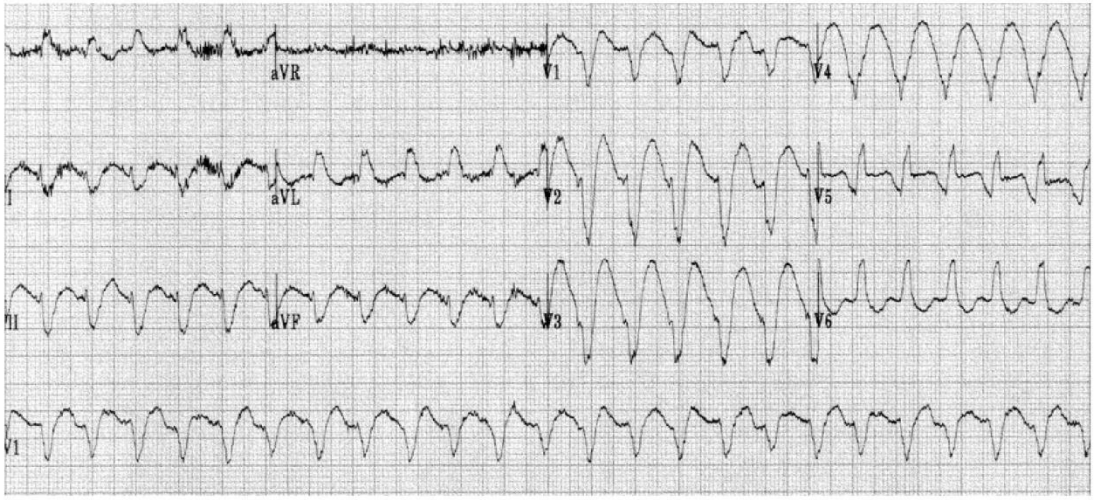

25mm/s
I
II
III
aVR
aVL
aVF


V1
V2
V3
V4
V5
V6

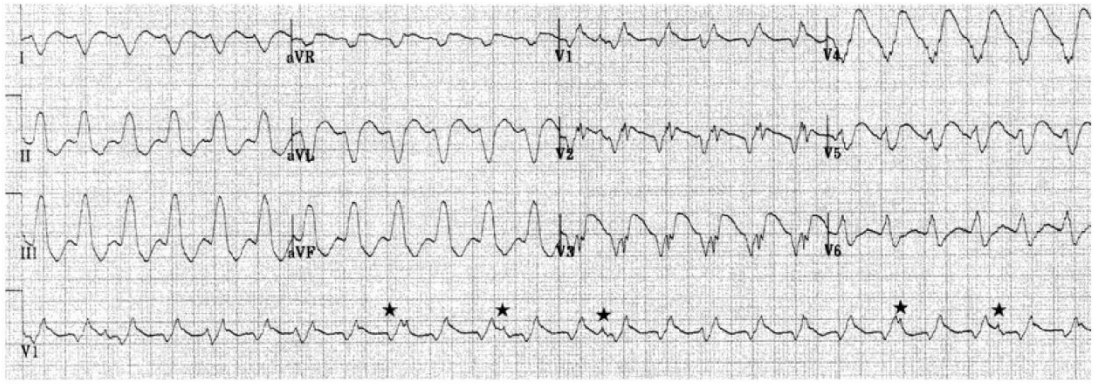

5 mm/mV 25 mm/s Filter 100 Hz 50 Hz d 5 mm/mV
I
II
III
aVR
aVL
aVF
5 mm/mV
V1
V2
V3
5 mm/mV
V4
V5
V6

TAHICARDIE PAROXISTICA VENTRICULARA



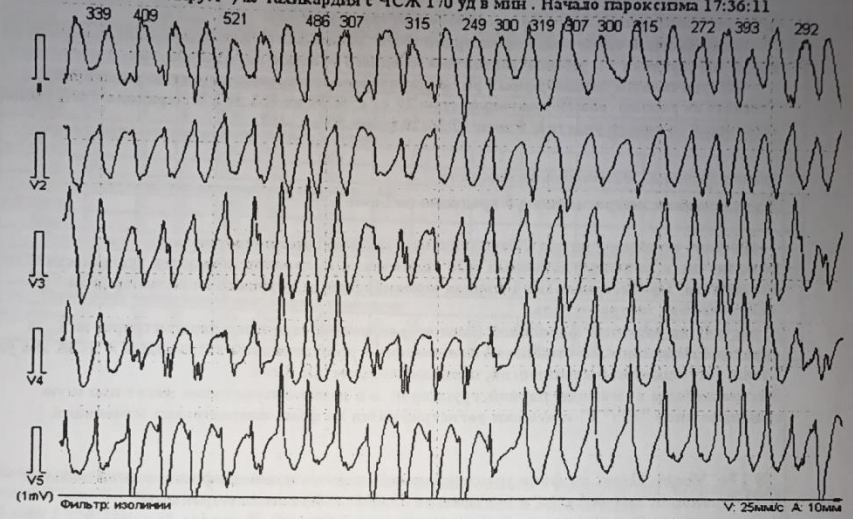

aVR
V1
V4
I
aVL
V2
V5
III
aVF
V3
V6
V1

A

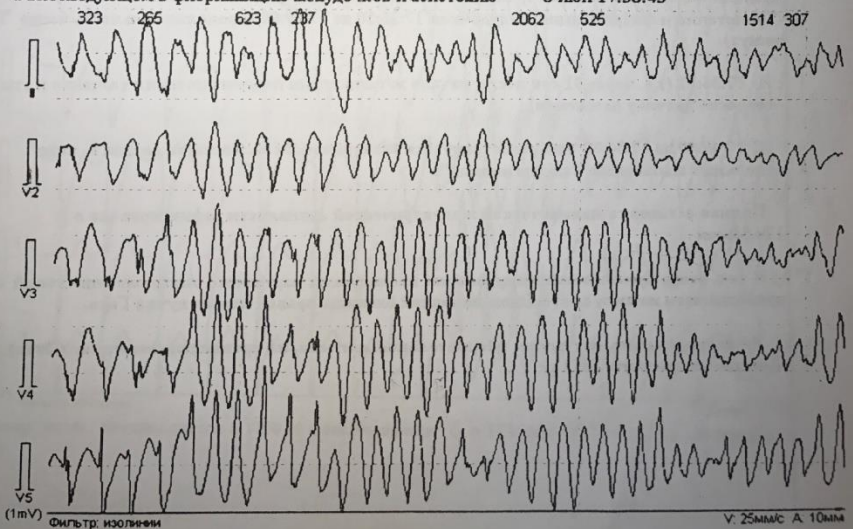

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

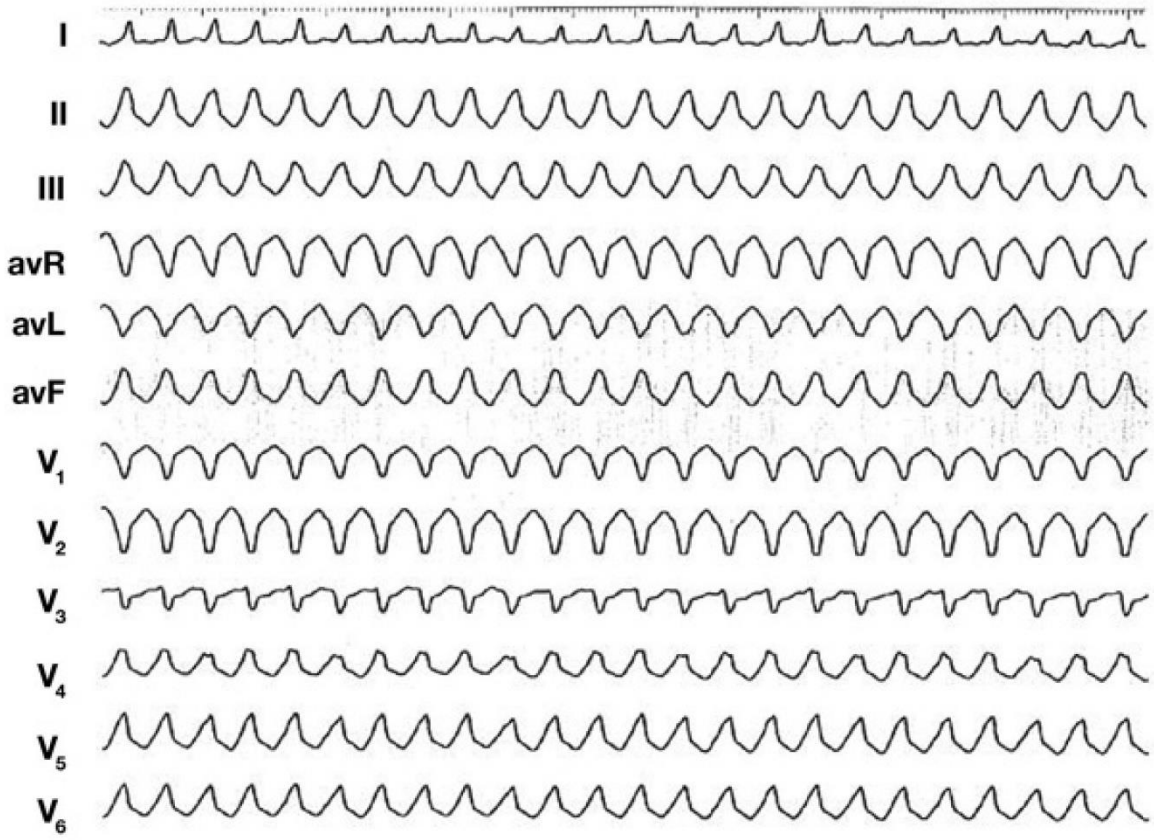
B

Максимальная ЧСЖ. Регистрируется неустойчивая полиморфная (двунаправленная, "веретено-образная", типа "пируэт") ж/ тахикардия с ЧСЖ 170 уд в мин . Начало пароксизма 17:36:11

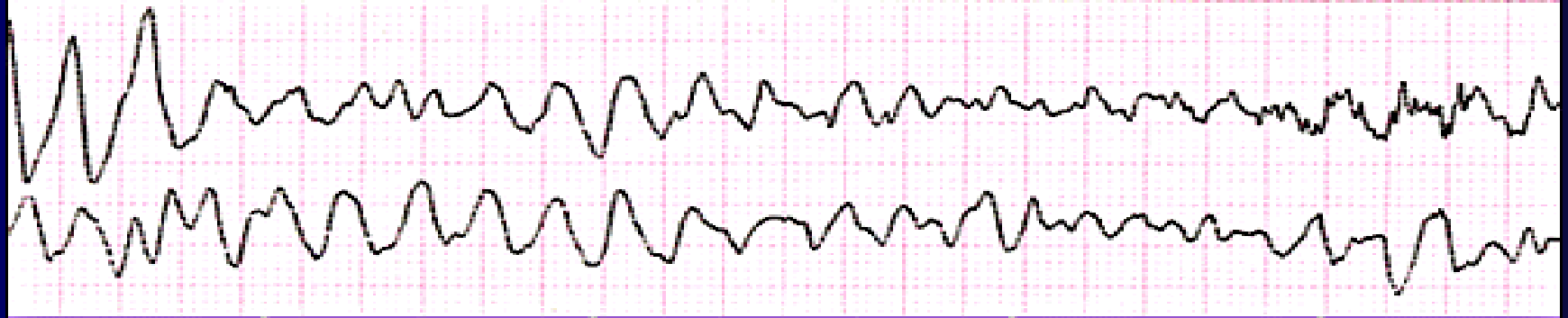


Пример ЭКГс фатальным НРС: переход двунаправленной желудоковой тахикардии в трепетание, а впоследующем в фибрилляцию желудочков и асистолию б июн 17:36:43



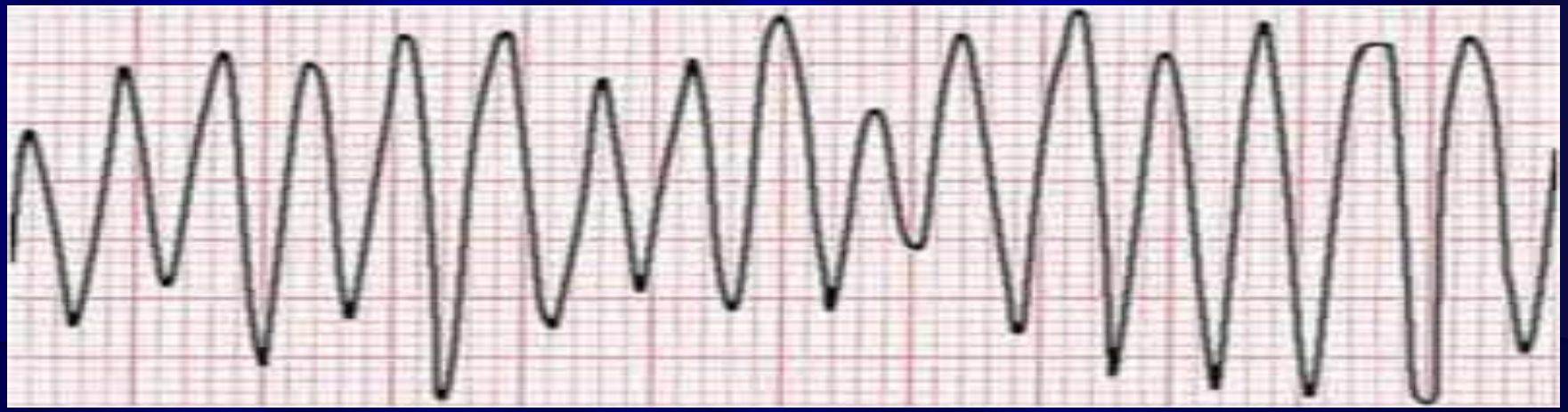

I
II
III
avR
avL
avF
V1
V2
V3
V4
V5
V6

Ventricular Fibrillation

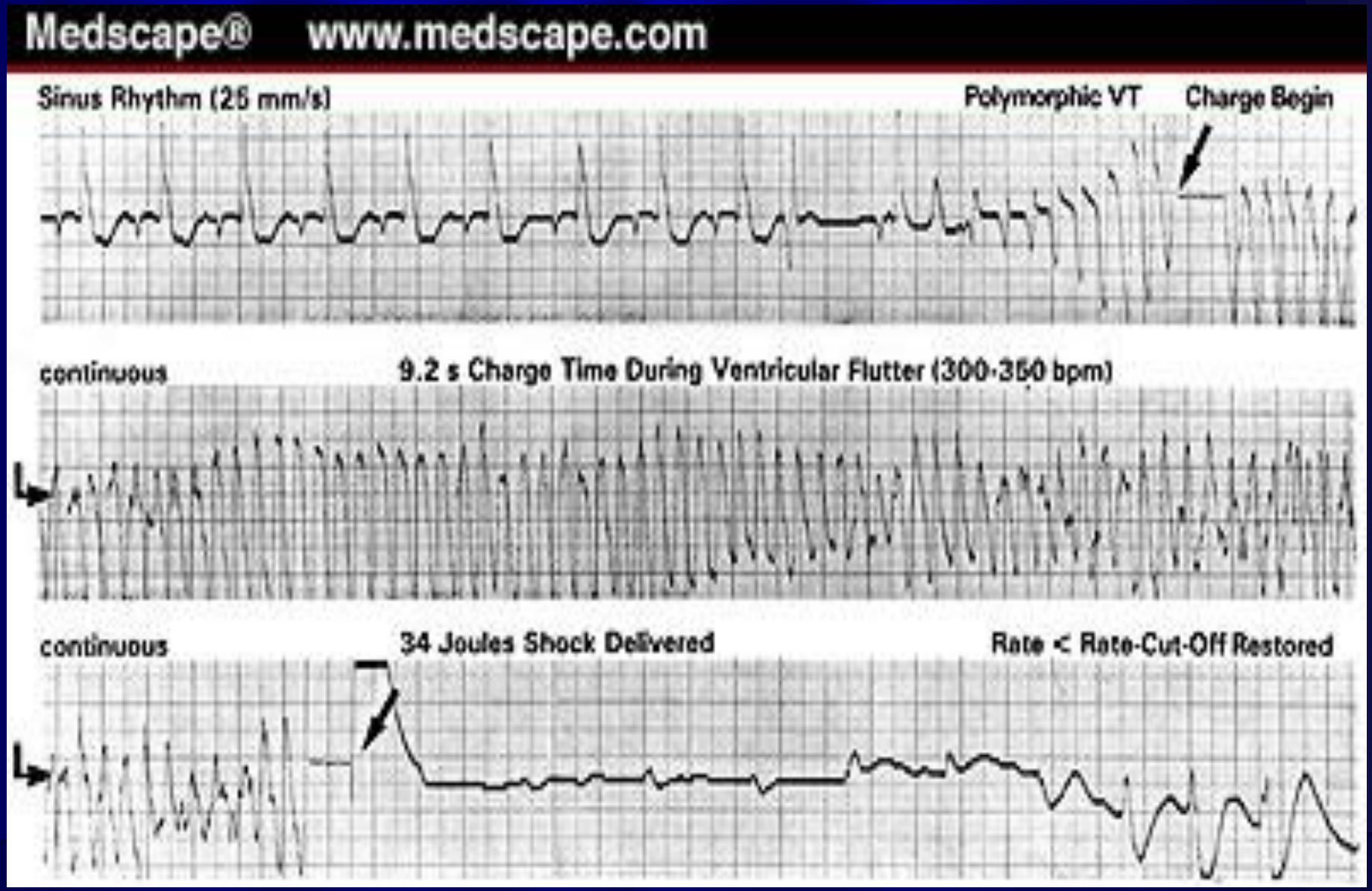


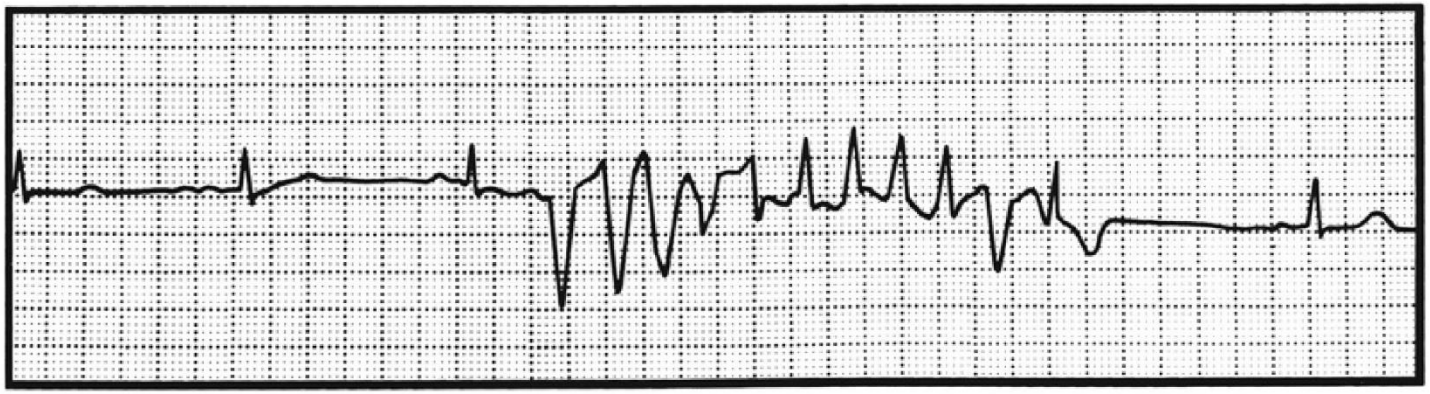
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
300-600	Extremely irregular	Absent	N/A	Fibrillatory baseline

Ventricular Flutter

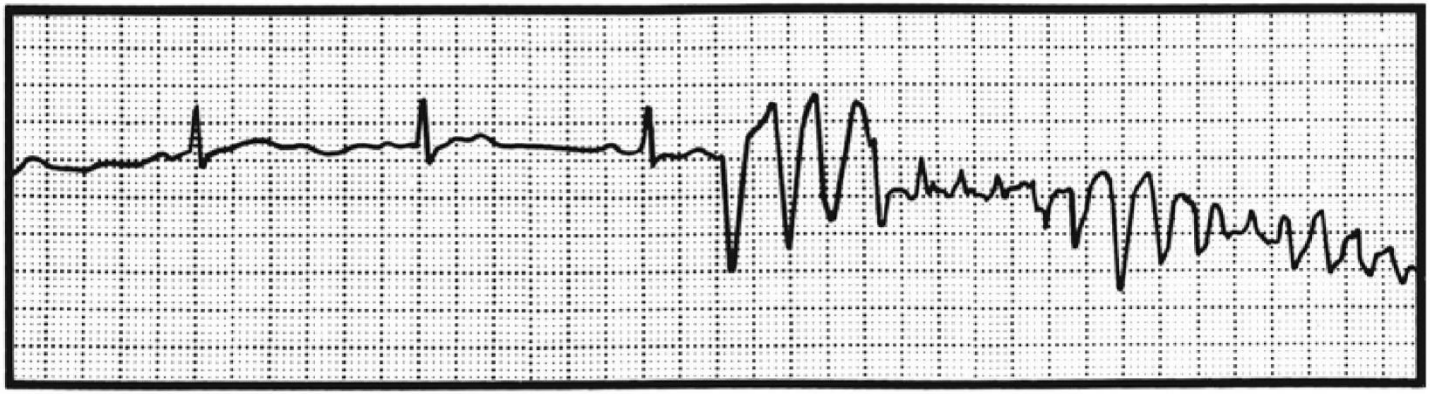


Ventricular Flutter

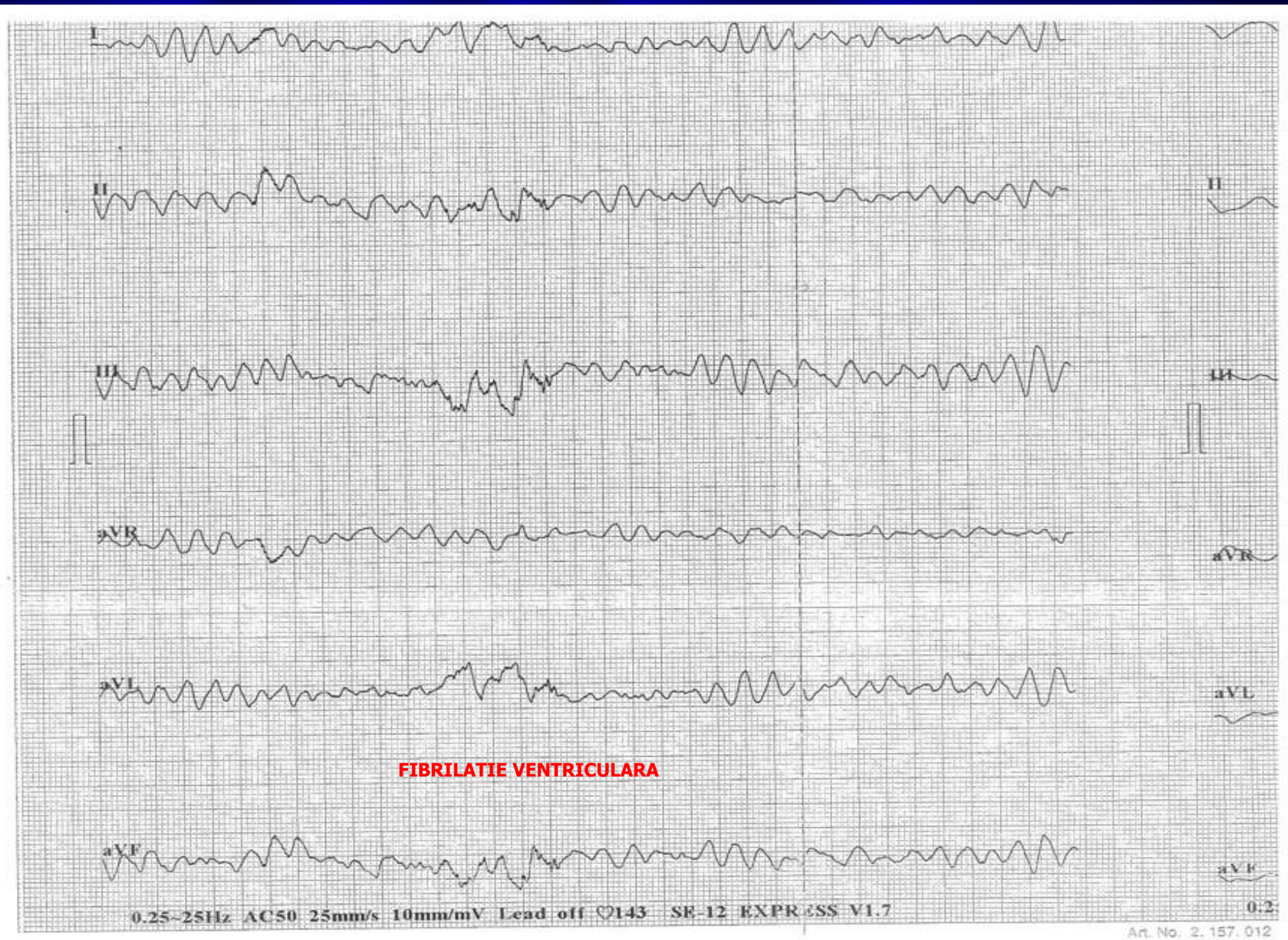




A



B


I
II
III
aVR
aVL
aVF
II
III
aVR
aVL
FIBRILATIE VENTRICULARA
0.25~25Hz AC50 25mm/s 10mm/mV Lead off ♡143 SE-12 EXPRESS V1.7
Art. No. 2.157.012

Treatment depends on the hemodynamic tolerance of the tachycardia

- If patient loses consciousness or has severe hypotension, a synchronized DC cardioversion should be attempted, sedation prior to cardioversion is advised in patients who are awake
- With hemodynamically well-tolerated VT, there is ample time to gather complete information, including a 12-lead ECG, before initiating therapy
- Intravenous amiodarone is the drug of choice for treating VT
- VT in association with acute myocardial infarction may respond to lidocaine (2–3 mg/kg)
- Intravenous procainamide (10–15 mg/kg) at a rate of 50–100 mg/min is also effective, but it can lead to hypotension

VT Treatment

- When VT is triggered or aggravated by exercise, intravenous β-blockers may be tried as the initial therapy
- If there is any uncertainty regarding the safety of β-blockers in patients with VT, it is somewhat safer to use agents with a short half-life, such as esmolol

Class III agents
(amiodarone, sotalol, and azimilide)

- for control of VT and ventricular fibrillation (VF)
- After a loading dose of 1200–1800 mg/day for 1–2 weeks, amiodarone can be used in a maintenance dose of 200–400 mg/day

Long-term side effectsof amiodarone

- It can effect the thyroid, lungs, gastrointestinal tract, eyes, skin, genitourinary system, and central nervous system
- Baseline thyroid function test, liver function test, pulmonary function test, and ophthalmologic examination should be done in patients when amiodarone therapy is started, with repeat tests every 6 months

Prevention

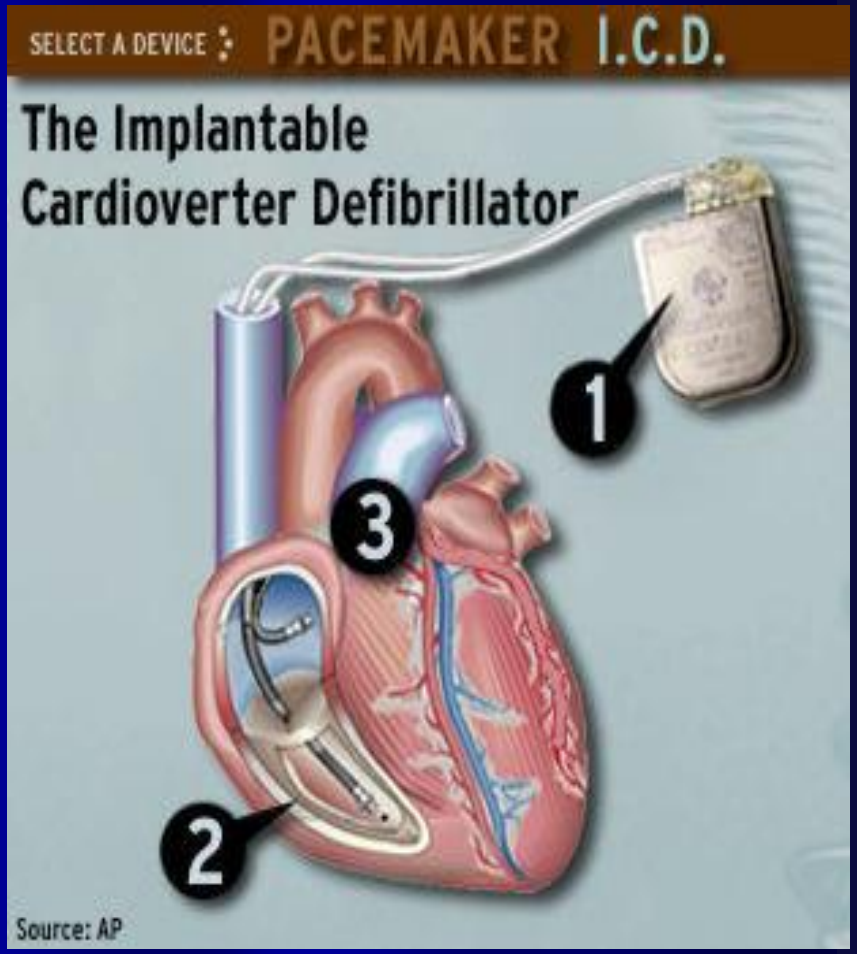
- Sotalol is 120–240 mg/day with monitoring of the ECG for QT prolongation
- It should be pointed out that in high-risk patients with coronary artery disease and reduced left ventricular function
- None of the antiarrhythmics, including amiodarone, have been shown to decrease mortality compared with placebo

Class Drug

- Ia Quinidine, procainamide, disopyramide
- Ib Mexiletine, lidocaine
- Ic Flecainide, propafenone, others (ethmozine)
- II β–Blockers
- III Amiodarone, sotalol, bepridil
- IV Calcium channel blockers

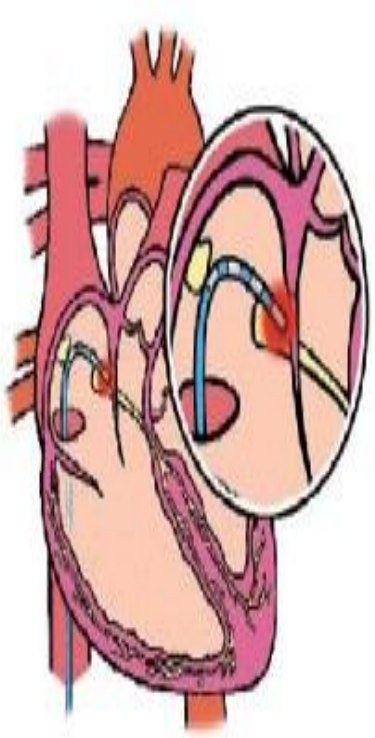
Nonpharmacologic Therapy

- **Implantable Cardioverter-Defibrillators** – These devices clearly provide the most effective form of therapy for preventing SCD in patients with VT

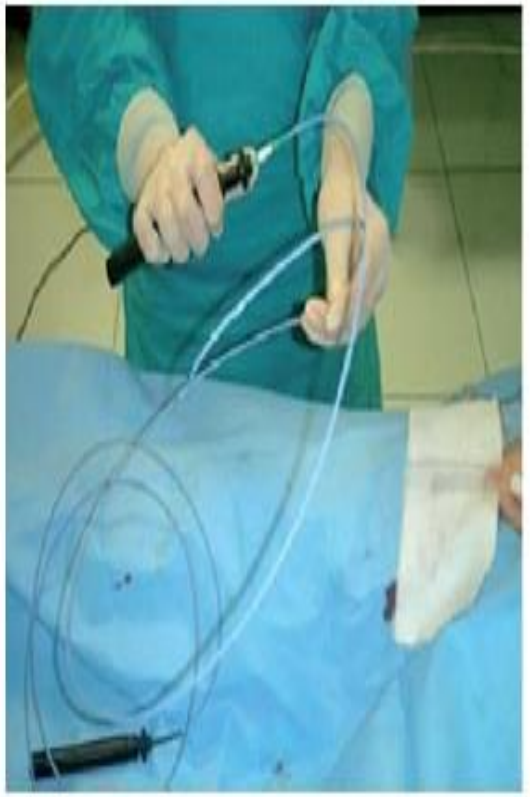


Catheter-Based Ablation

- The development of radiofrequency catheter ablation as a therapeutic option for the treatment of arrhythmias has dramatically altered the approach to the tachycardic patient



Exemplu de stimul energetic



Electrocateter ablative

Surgical Ablation

- In patients with coronary artery disease and prior myocardial infarction, the VT often originates close to the infarct, thus providing the opportunity for surgical destruction of the VT site of origin

Success!