ECG diagnostics. Conduction and heart rhythm disturbances

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Conduction defect

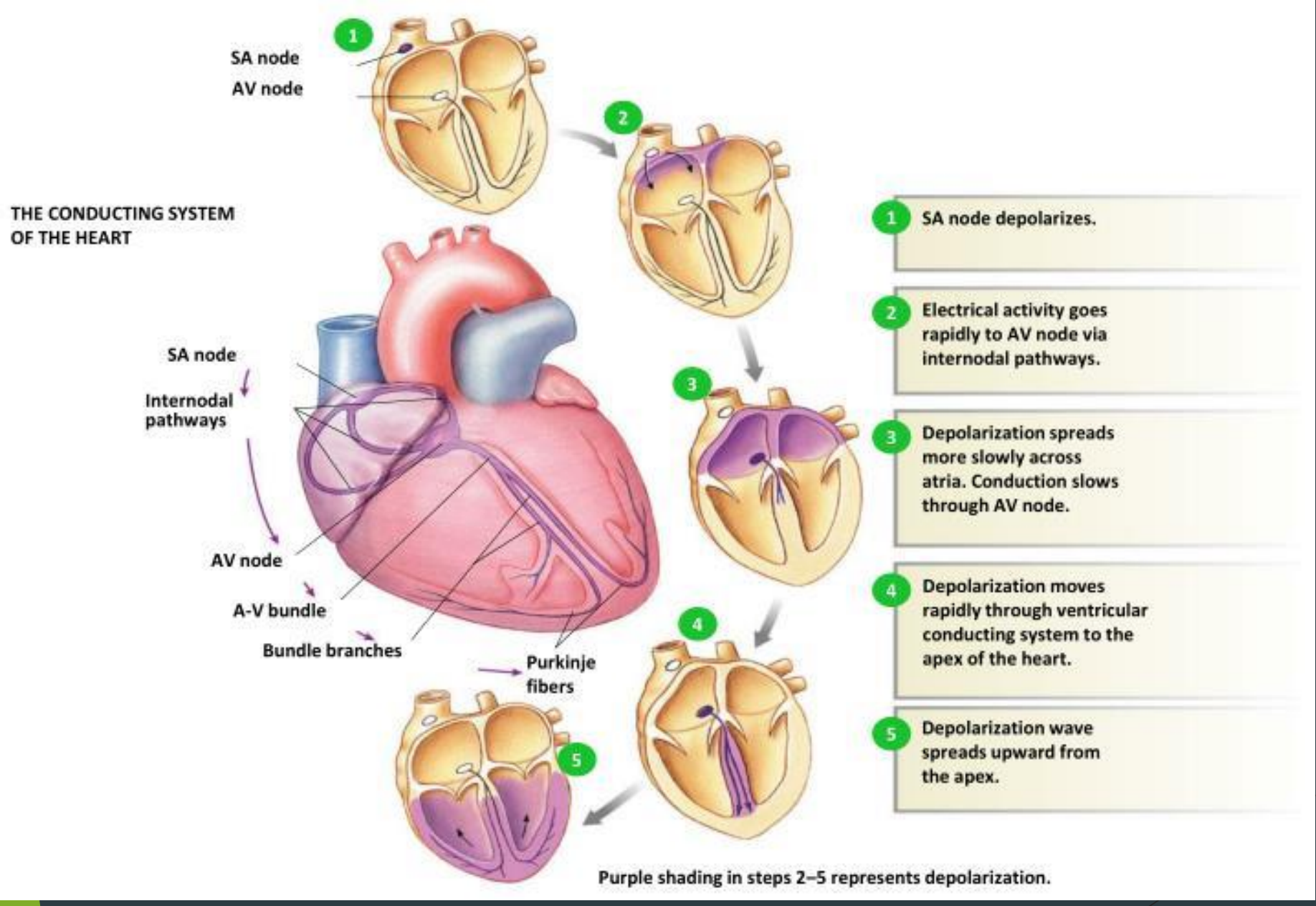
a failure of the normal passage of controlling electrical impulses through the specialized conduction muscle fibres of the heart.

Conduction defects lead to various forms of heart block.

Collins Dictionary of Medicine © Robert M. Youngson 2004, 2005

Normal electrical conduction through the heart muscle takes a predicted pathway. It travels from the Sinoatrial node (SA node) to the Atrioventricular node (AV node) to the Bundle of His and then onto the left and right bundle branches (usually in a left to right pattern), ultimately ending up in the Purkinje fibers. Additionally, the Left Bundle has an anterior and posterior component called a fascicle. In normal cardiac electrophysiology, the electrical conduction occurs from left to right, essentially stimulating the left bundle, and left ventricle first. We will discuss the syndromes that result in abnormal conduction at every step of the electrical pathway, the aberrations that are manifested in the ECG as a result and the relevance of each abnormality in a clinical setting.

Electrical Conduction in Heart



https://www.slideserve.com/wolfe/electrical-conduction-in-myocardial-cells

International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10)-WHO Version 2019

Chapter IX

Diseases of the circulatory system (I00-I99):

Other forms of heart disease (I30-I52):

I44 Atrioventricular and left bundle-branch block

I45 Other conduction disorders

I46 Cardiac arrest

I47 Paroxysmal tachycardia

I48 Atrial fibrillation and flutter

I49 Other cardiac arrhythmias

Chapter XVIII
Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (R00-R99)

Symptoms and signs involving the circulatory and respiratory systems (R00-R09):

R00 Abnormalities of heart beat

Abnormal findings on diagnostic imaging and in function studies, without diagnosis (R90-R94):

R94.3 Abnormal results of cardiovascular function studies:

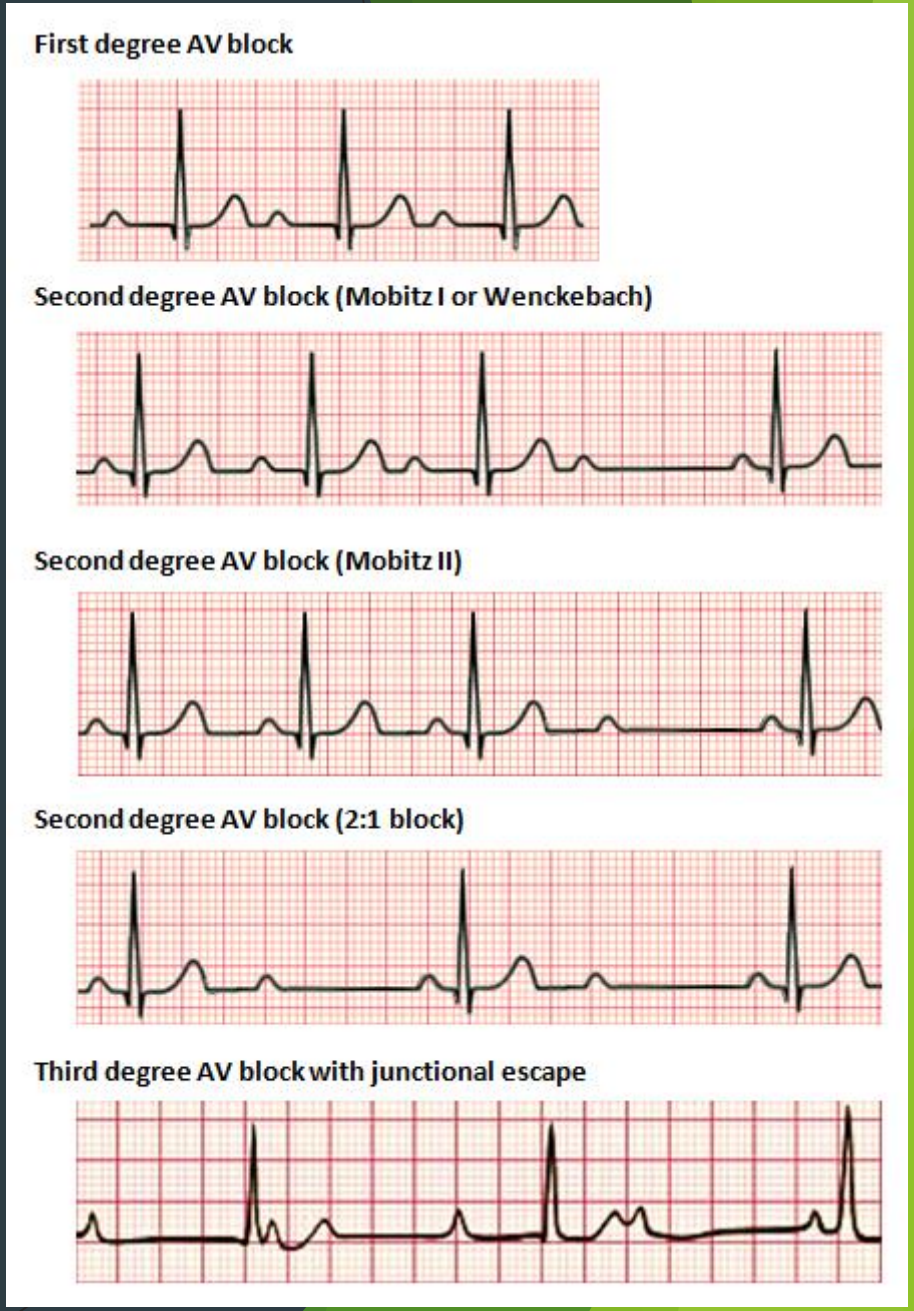
Abnormal:

- electrocardiogram [ECG]
- electrophysiological intracardiac studies
- phonocardiogram
- vectorcardiogram

Atrioventricular block (AV block)

is a type of heart block that occurs when the electrical signal traveling from the atria to ventricles is impaired.

- I44.0 Atrioventricular block, first degree
- I44.1 Atrioventricular block, second degree
- I44.2 Atrioventricular block, complete
- I44.3 Other and unspecified atrioventricular block



I44.0 Atrioventricular block, first degree

A first degree AV node block occurs when ***conduction through the AV node is slowed***, thus delaying the time it takes for the action potential to travel from the SA node, through the AV node, and to the ventricles.

A first degree AV block is indicated on the ECG by a prolonged PR interval.

The PR interval is normally 0.12-0.20 seconds or 120 to 200 milliseconds. ***A PR interval consistently longer than 0.20 seconds (greater than 5 small boxes) indicates a 1st degree AV block.***

Etiology:

· Increased vagus tonus.

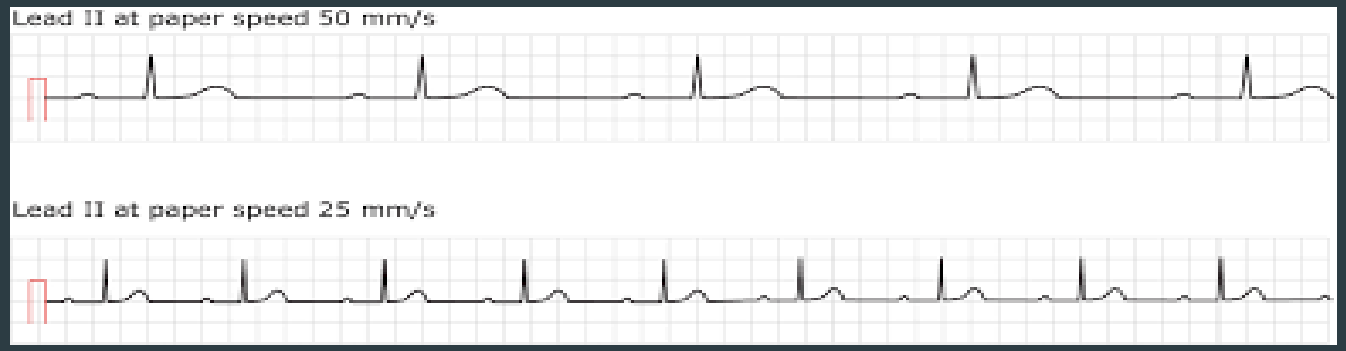
· Acute myocardial infarction, especially in the area of the lower wall.

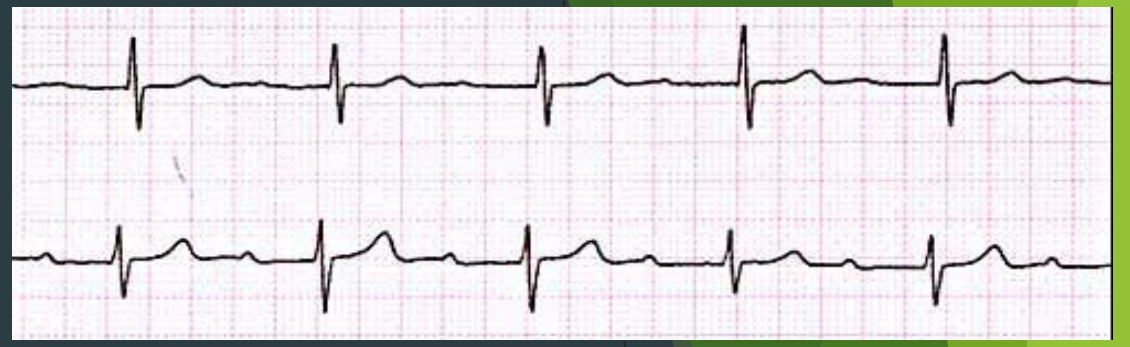
· Myocarditis

· Electrolyte disturbances (hypokalemia, hypomagnesemia).

· Accepted drugs: class Ia antiarrhythmics (quinidine, procainamide), class Ic (propafenone, etacizin, flecainide), class II (beta-blockers), class III (amiodarone, sotalol), class IV (non-dihydropyridine calcium antagonists - verapamil, diltiazem) , digoxin, magnesia. Especially often: an overdose of cardiac glycosides.

In general, a 1st degree AV block is a benign finding that does not require any treatment, however it may be an indicator of higher degree AV block in the future. Higher doses of AV blocking medications should be avoided.





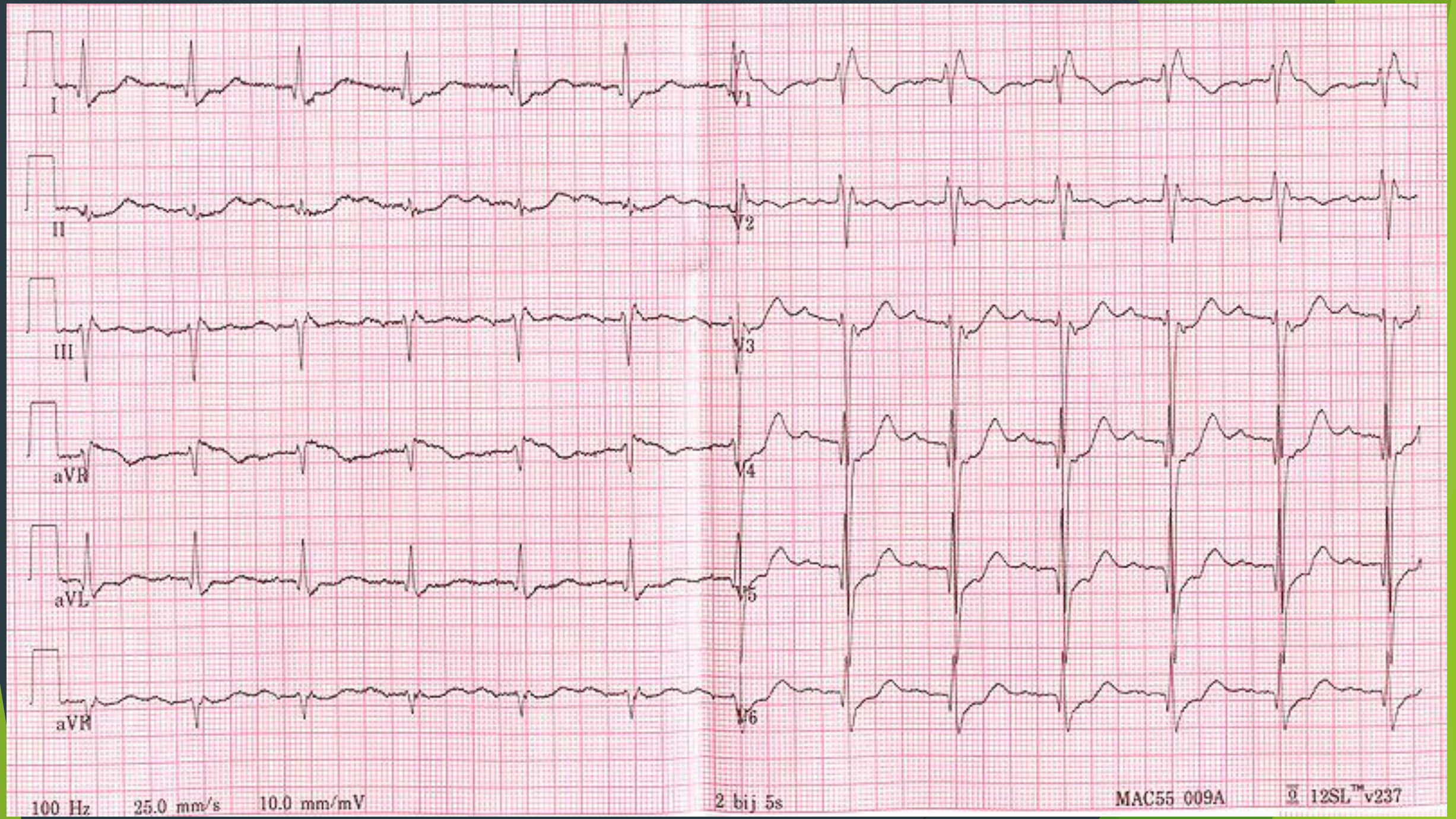
The rhythm is regular, normocardia.

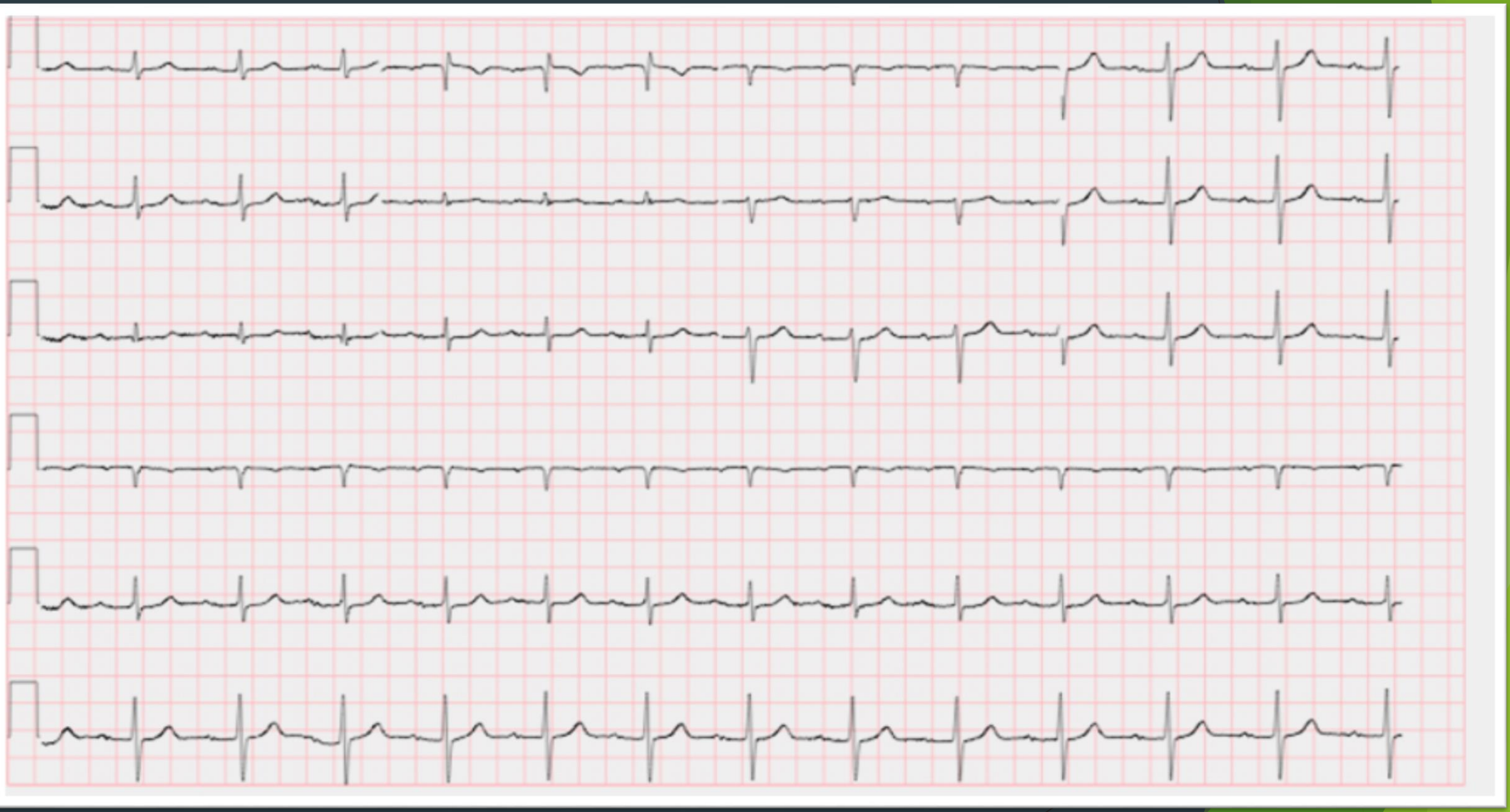
A PR interval more than 0.20-0.22 seconds and is constant in all complexes.

There is a 1:1 ratio between P waves and QRS complexes (each P followed by QRS).

Usually P wave and QRS complex are not changed.

The interval P - P (R - R) are the same if there is no sinus arrhythmia.


I
II
III
aVR
aVL
aVR
V1
V2
V3
V4
V5
V6
100 Hz
25.0 mm/s
10.0 mm/mV
2 bij 5s
MAC55 009A
12SL™v237



I44.1 Atrioventricular block, second degree

AV block II degree - a violation of conduction, in which not all atrial impulses are conducted from the atria to the ventricles.

On the ECG, this is ***manifested by the absence of QRS complexes after some P waves***.

There are 4 types of AV blockade of the II degree:

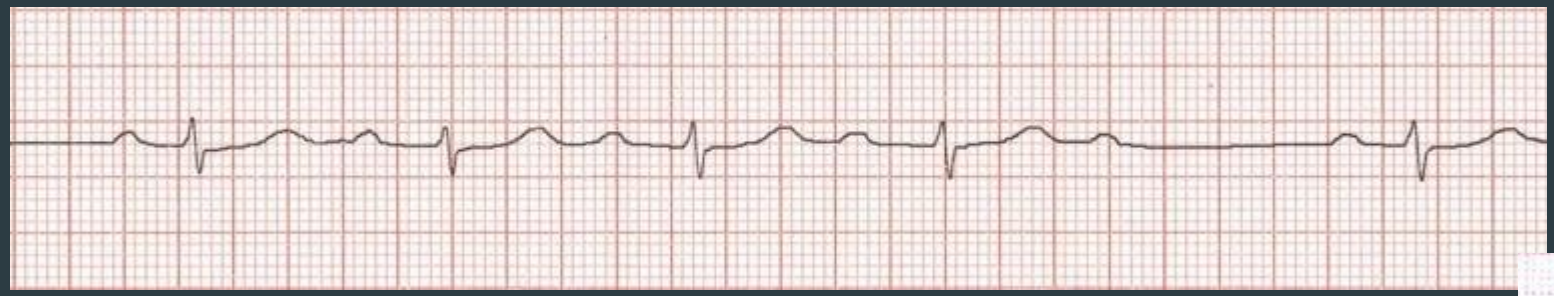
1. Partial AV block of the II degree with the periods of Wenckebach (Mobitz type 1).
2. Partial AV block of the II degree, type 2 (Mobitz type 2).
3. Partial AV block of the II degree 2:1.
4. Progressive AV block/ “high-grade” AV block.

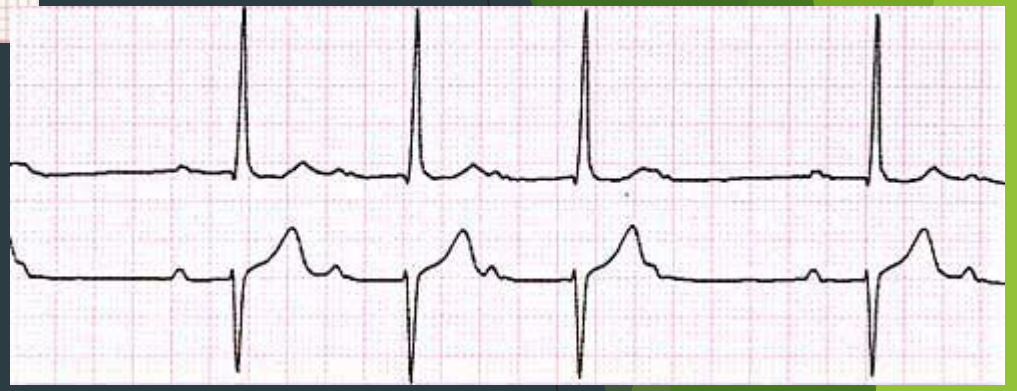
AV block Mobitz type 1

It is associated with the lengthening of the absolute and relative refractory period in the AV compound. With this blockade, the conductivity in the AV node progressively worsens from contraction to contraction until the AV connection is unable to conduct another impulse to the ventricles ("dropped beat") . This leads to periodic loss of ventricular contractions. During a long pause, the conduction in the site is restored, after which the entire cycle is repeated.

On the ECG, this is manifested by ***a progressive lengthening of the PQ interval from complex to complex, then only P wave is recorded, and the ventricular QRS complex falls out***. In the first complex, after falling out, the PQ interval is the smallest, but then the cycle repeats (***Wenckebach period***). Since the loss of ventricular complexes is natural, then there is an AV block with a ratio of 3: 2, 4: 3, etc.

Often such blockages occurs with an overdose of cardiac glycosides, antiarrhythmic drugs, and myocardial infarction.



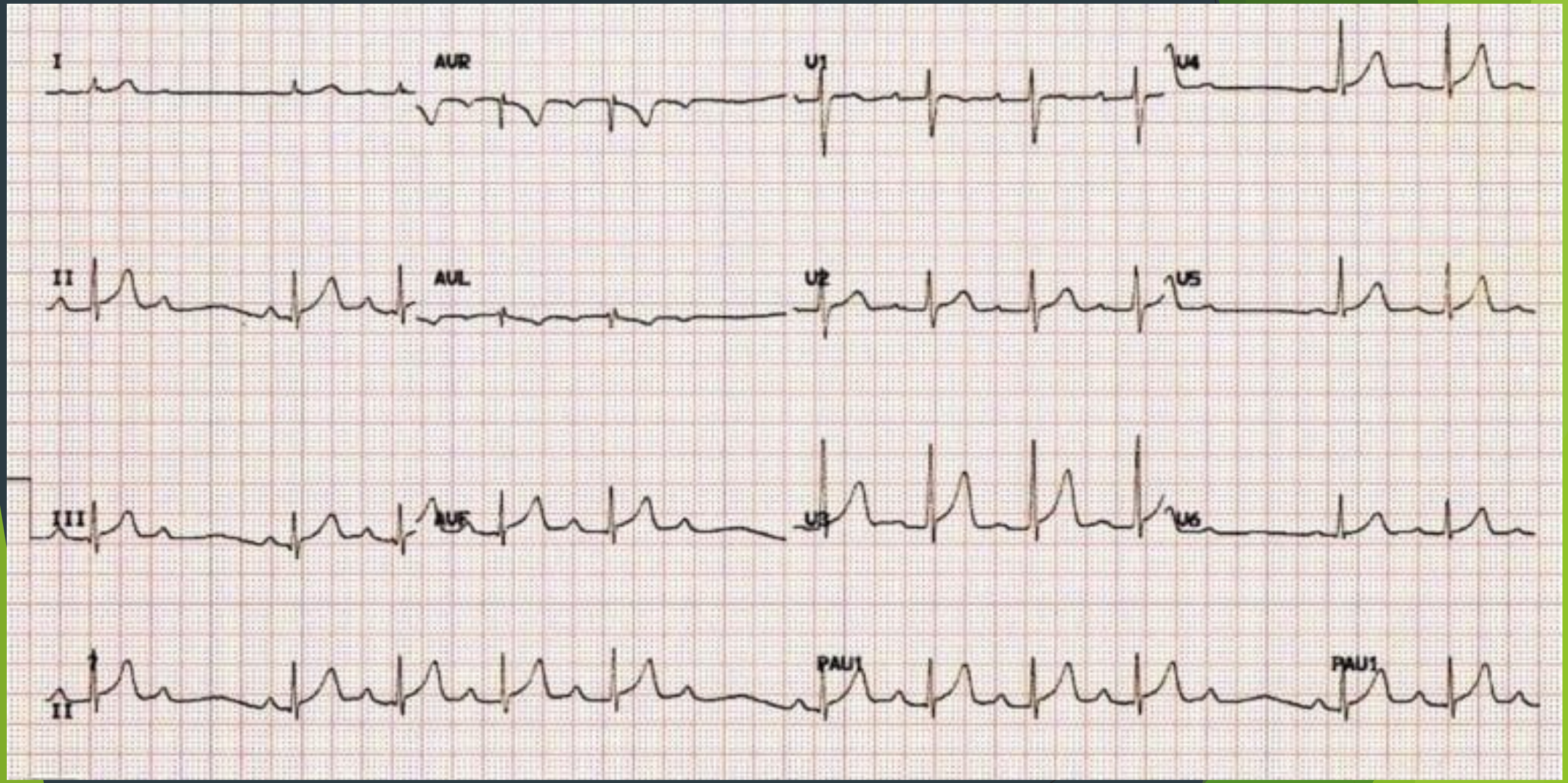


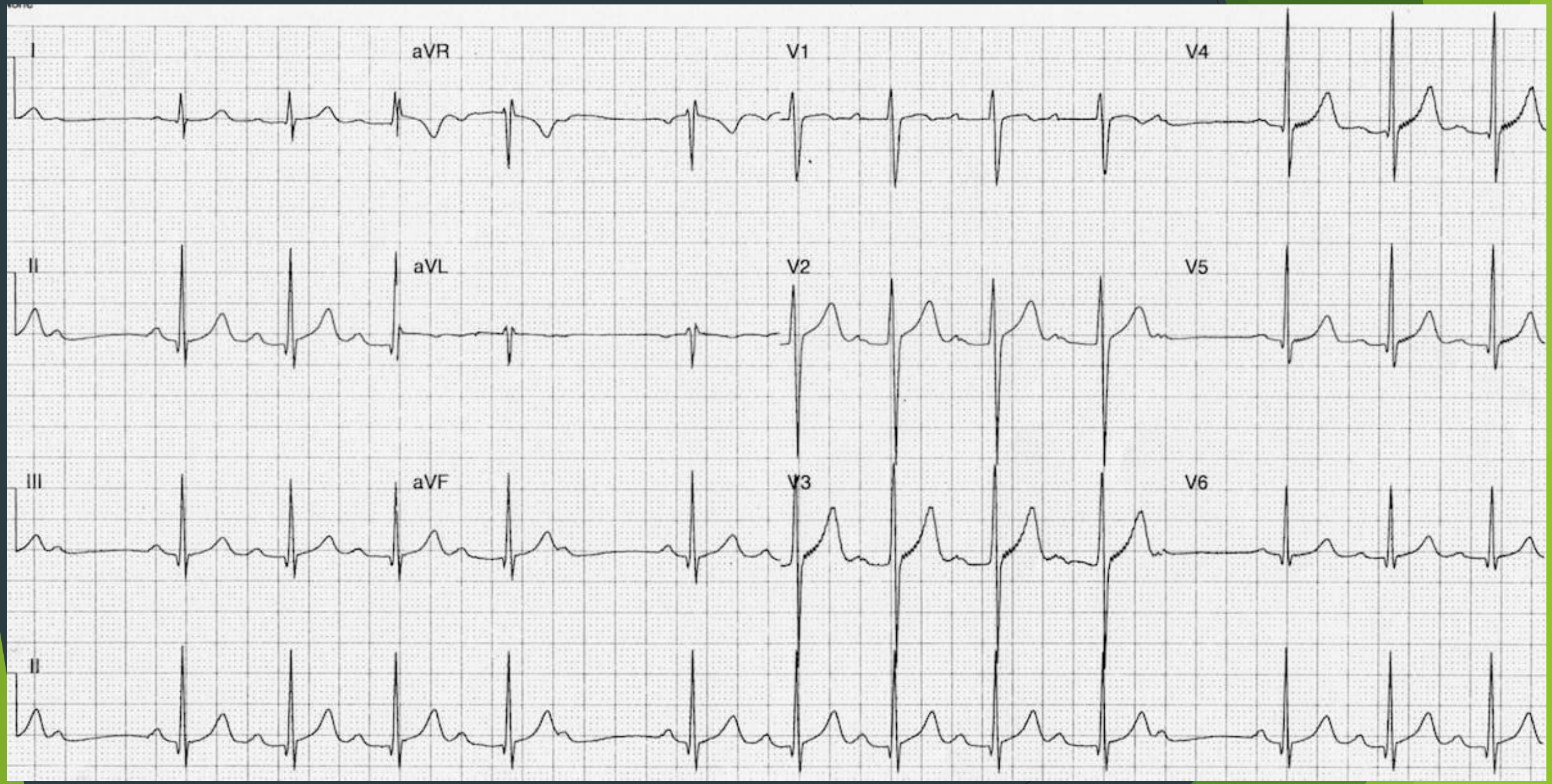
The rhythm is irregular, brady-/normocardia.

The P-P interval remains relatively constant.

The Wenckebach pattern tends to repeat in P:QRS groups with ratios of 3:2, 4:3 or 5:4.

The QRS complex generally remains within the normal range, because the blockage is localized above the His bundle.

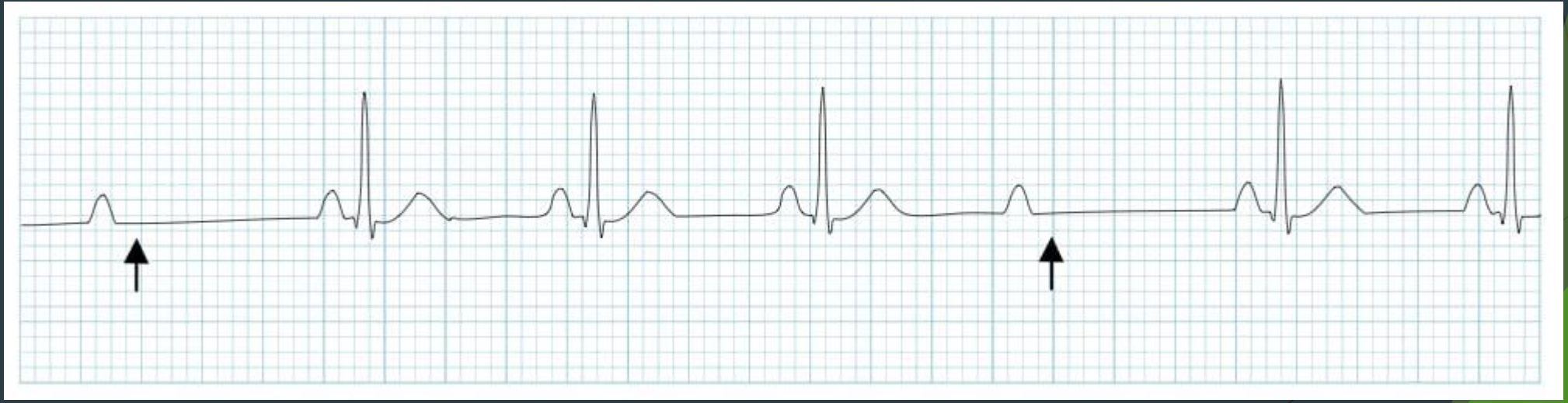

I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6
II
PAU1
PAU1

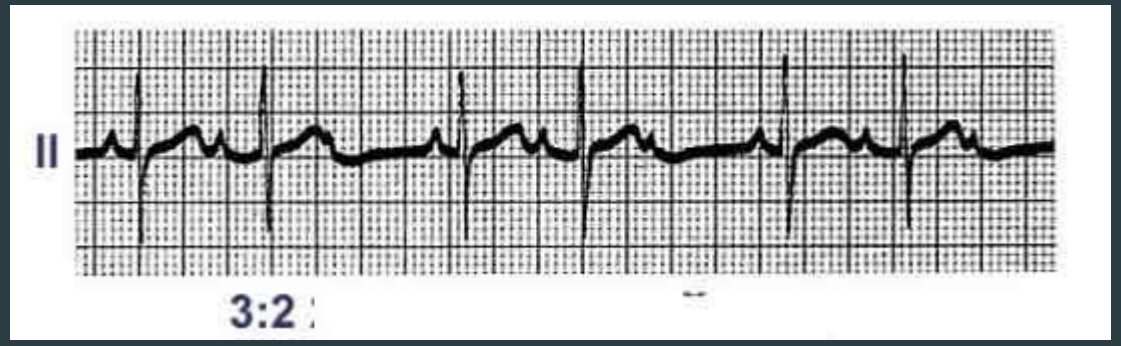

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

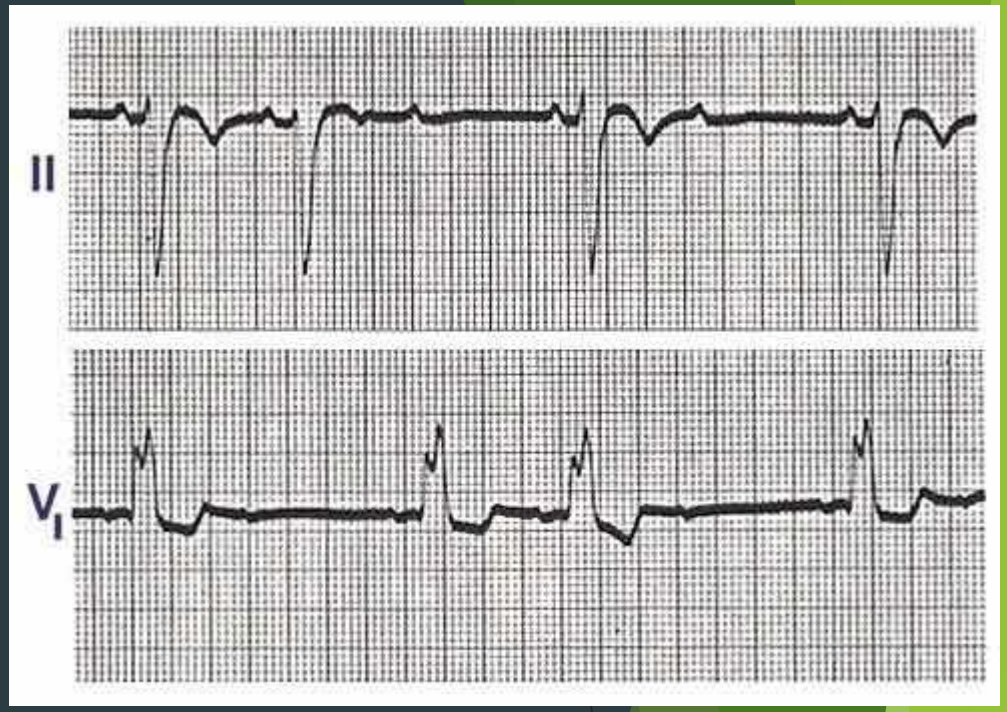
AV block Mobitz type 2

It is characterized by ***periodic loss of ventricular contractions*** without a cycle of changes in the ***PQ interval***, which ***can be elongated or normal***. The loss of ventricular complexes can be regular (every 3, or 4, or 5) or irregular, chaotic. The diagnosis of such cases is sometimes complicated by the layering of extrasystoles.

The risks and possible effects of Mobitz II are much more severe than Mobitz I in that it can lead to severe heart attack.





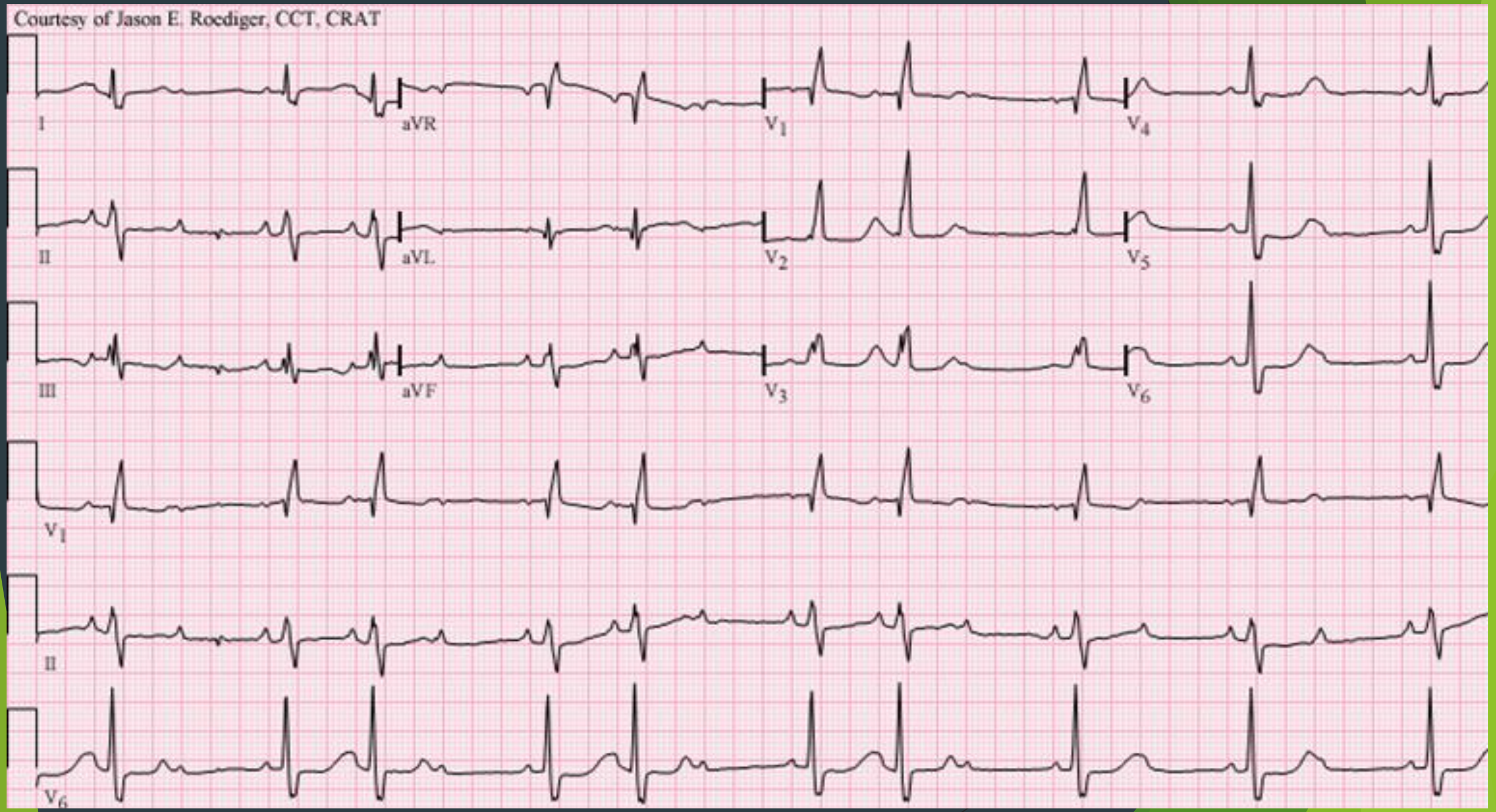


The rhythm is irregular, brady-/normocardia.

The PQ interval is normal or increased.

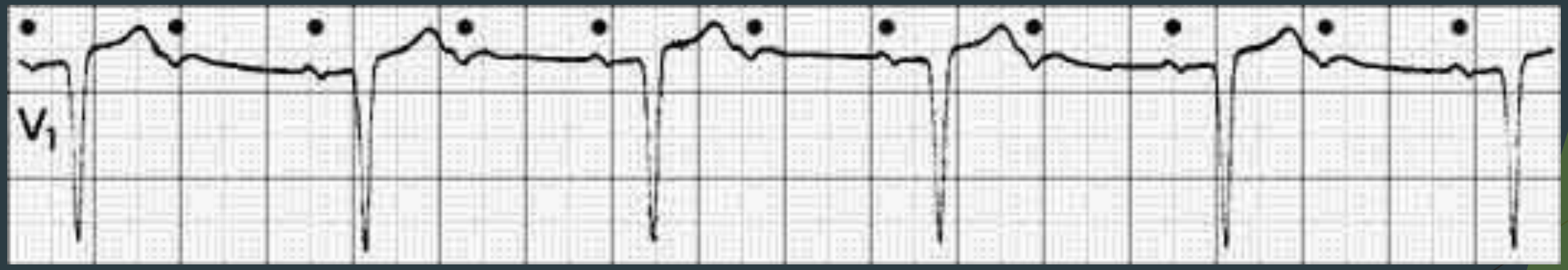
There can be no constant ratio between P waves and QRS complexes.

QRS could be increased.


Courtesy of Jason E. Roediger, CCT, CRAT
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V6

Partial AV block of the II degree 2: 1

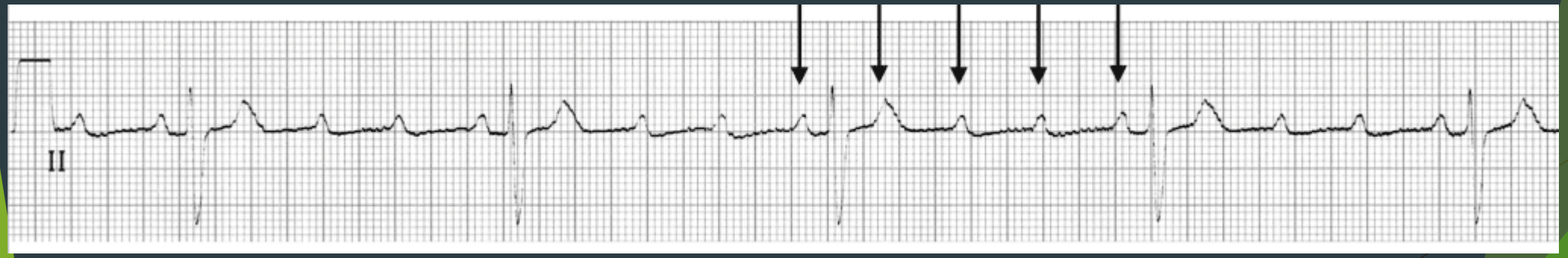
With this type, every second impulse is blocked and ***every second ventricular contraction regularly falls out***. In the absence of sinus arrhythmia, the ***P - P distance is the same*** and the ***QRS distances are the same, but twice as large***. Bradycardia develops. Such a blockade usually occurs with severe heart damage.

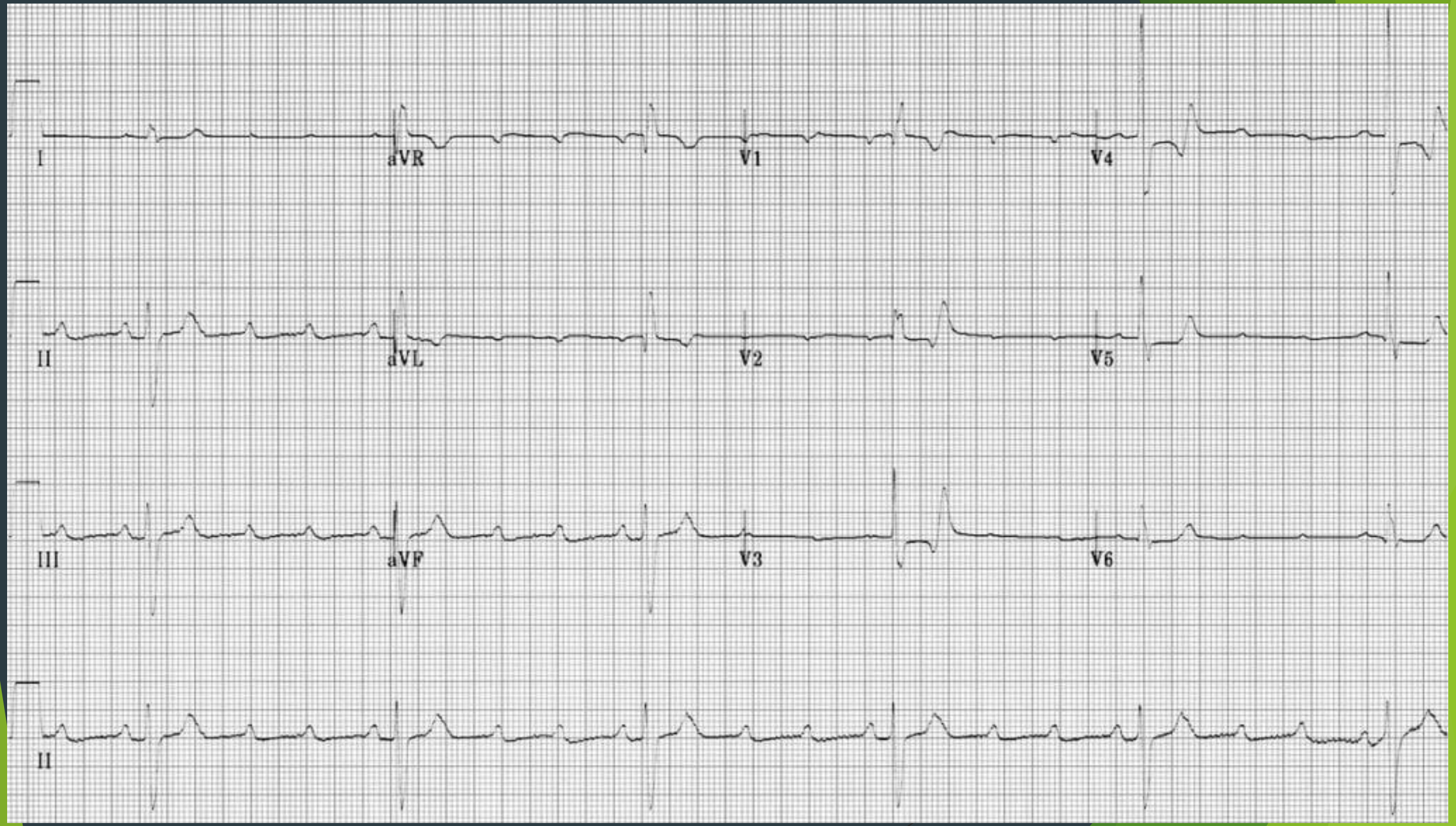


“High-grade” AV block.

With such AV blockade, conduction is disturbed so sharply that ***2 or more ventricular contractions in a row are blocked*** (3: 1, 4: 1, 5: 1), and such blocking can follow rhythmically and irregularly.

The patient may have Stokes–Adams syndrome.



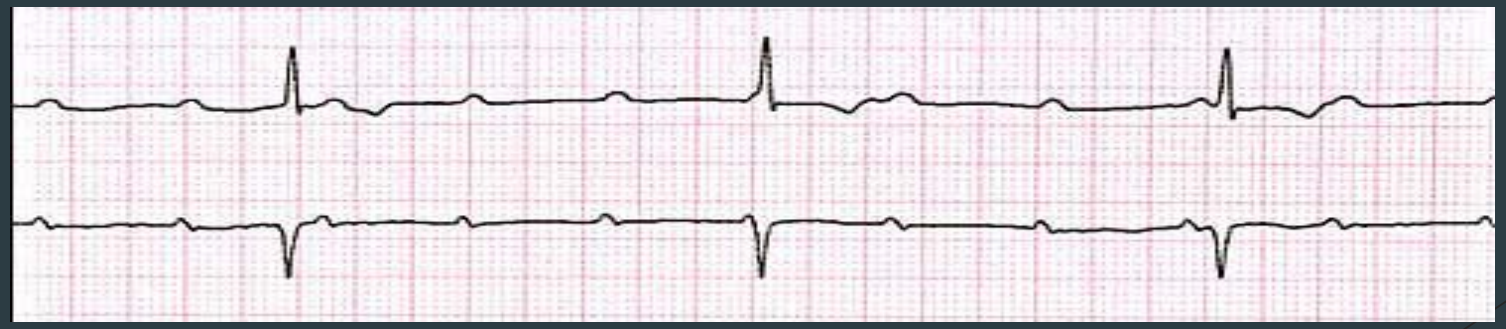

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

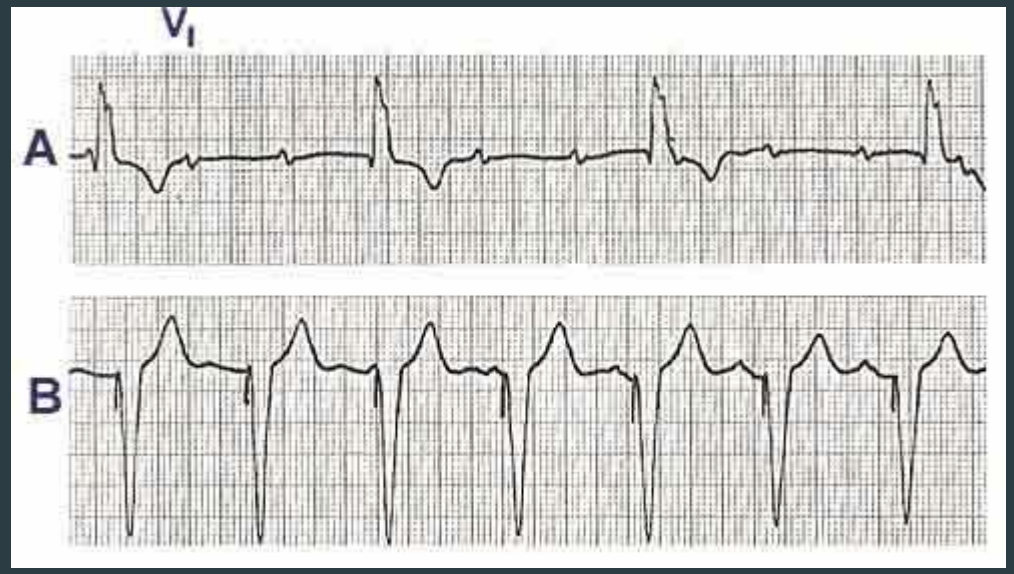
I44.2 Atrioventricular block, complete

In third degree heart block, the electrical ***depolarization of the atria and the ventricles occur independently from each other***. The atria are excited from the sinus node, and the ventricles from the atrioventricular node or ectopic foci.

This is recognized when the ***P waves and the QRS complexes*** "march through" the rhythm strip, ***each at their own independent rate***, without regard to each other.

The P wave rate is 60 beats per minute and the QRS rate is 37 beats per minute. If you examine only a few beats on a 12 lead ECG, there may seem to be a relationship between the P waves and QRS complexes. However, when observed over a longer strip, it is readily apparent that the atrial and ventricular rhythms are not associated with one another, but instead are firing ("marching through") at separate rates, independently of one another.

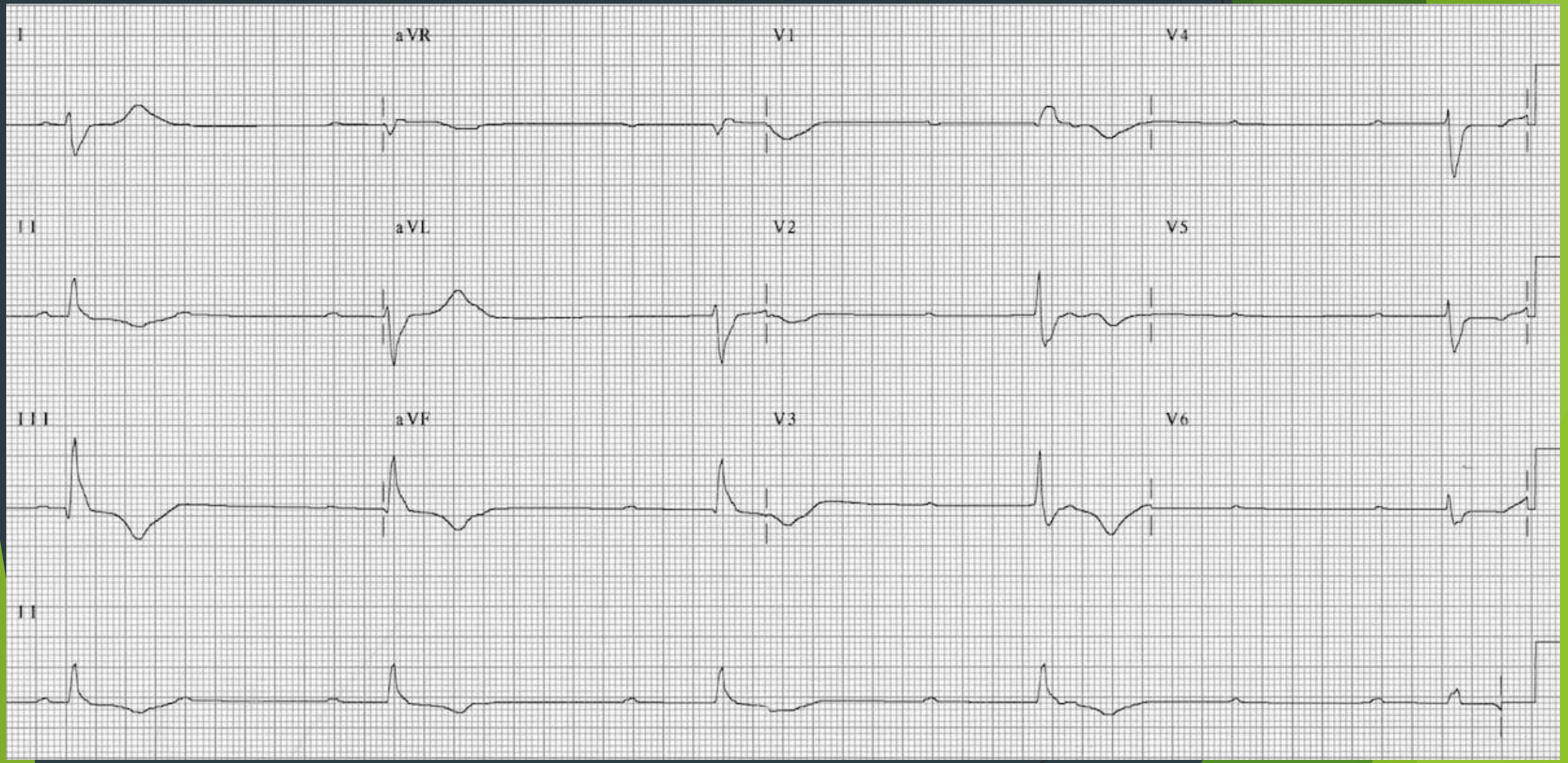


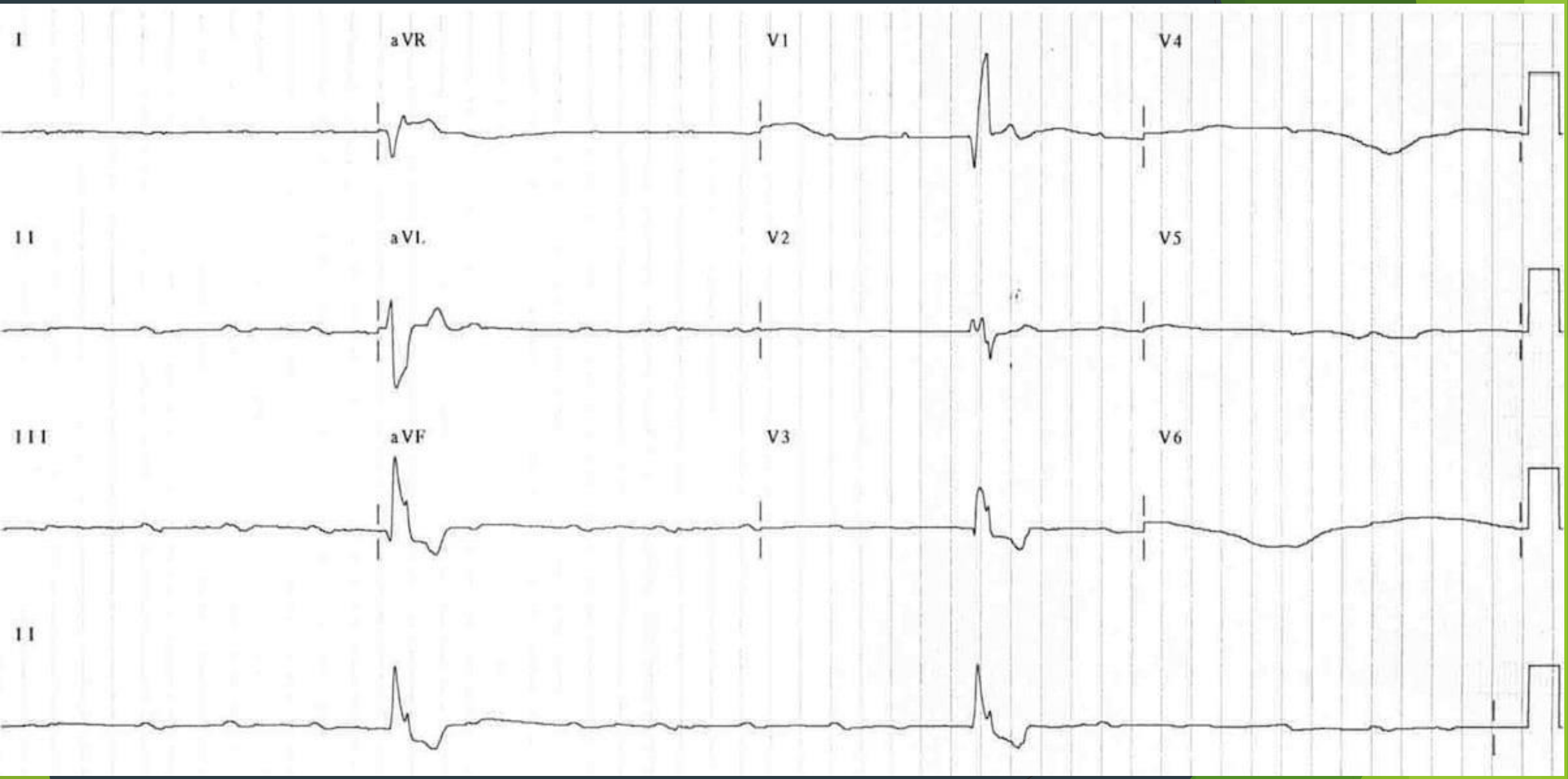


Bradycardia.

The PR interval is variable. The PP interval represent the first rhythm. The QRS complexes with a regular RR interval represent the second rhythm. So the hallmark of complete heart block is lack of any apparent relationship between P and QRS.

Ps more then QRSs.

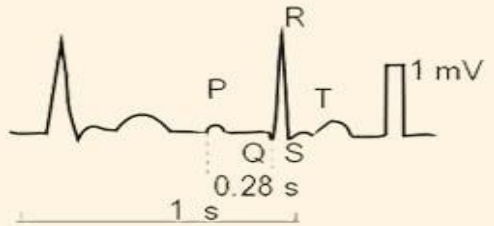

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


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

The Heart Block Poem

by the Princeton Surgical Group & nurseslabs

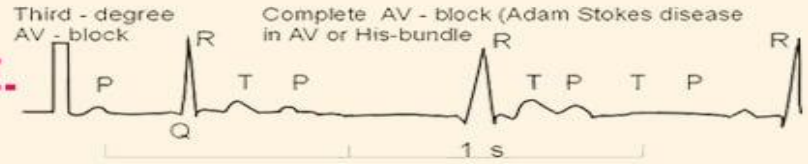
If the **R** is far from **P**,
then you have a **FIRST DEGREE**.



Longer, longer, longer, drop!
Then you have a **WENKEBACH**.

If some **P**s don't get through,
then you have **MOBITZ II.**

If **P**s and **Q**s don't agree,
then you have a **THIRD DEGREE.**



https://nurseslabs.tumblr.com/post/91133209935/the-heart-block-poem-if-you-liked-this-check-out

Bundle Branch Blocks

Certain pathologies (e.g., MI, pulmonary hypertension, digoxin intoxication) can result in an interruption of any part of the electrical conducting system.

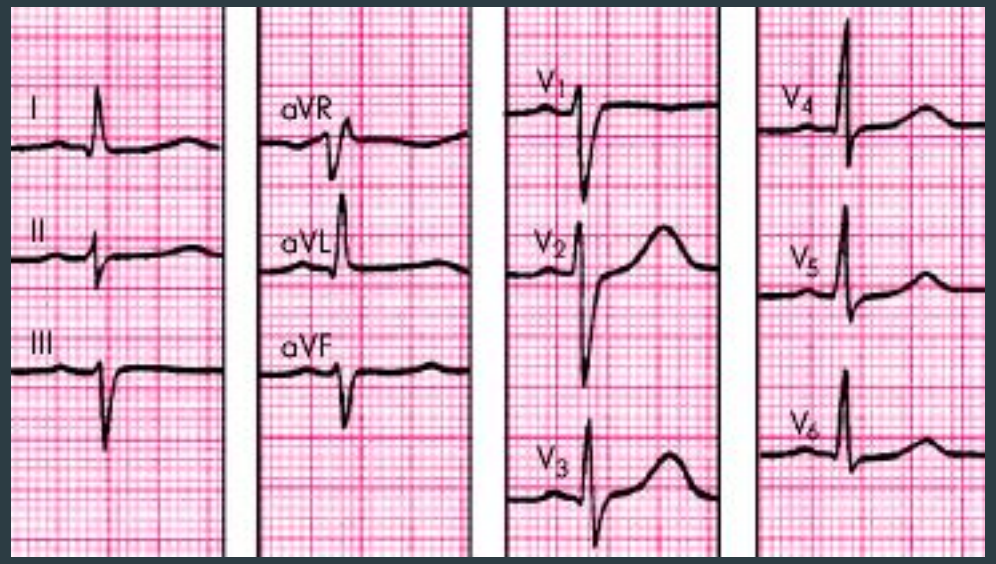
Clinically, bundle branch blocks delay in the conduction pathway and they can be helpful in determining the underlying etiology to a patients' presenting complaint.

The blockade may be:

complete/incomplete,

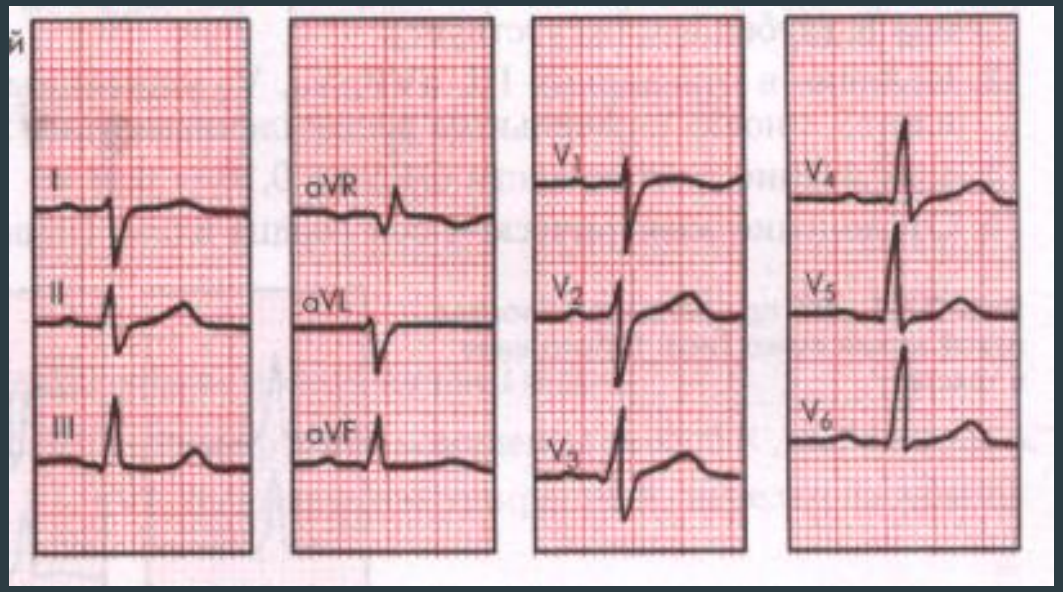
permanent/transient,

one-sided/two-sided.



I44.4 Left anterior fascicular block

1. A sharp deviation of the electrical axis of the heart to the left. RI> RII> RIII; SIII> RIII; SaVR> RaVR. The most important tests are SII> RII; RaVR> SaVR
2. The QRS complex is not widened or slightly widened (0.10-0.11).
3. There may be no change in chest leads.
4. In II, III, AVF leads - rS (deep S).
5. In I, AVL, V5-V6 - qR (high R).
6. In V1-V2 - qrS or QS.



I44.5 Left posterior fascicular block

1.A sharp deviation of the electrical axis of the heart to the right, with the absence of hypertrophy of the right heart, the absence of emphysema. RIII>RII>RI; SI>RI; RaVR>=(Q)SaVR.

2. The QRS complex can be normal (0.10-0.11 sec.).

3. The QRS is type qR in II, III, AVF (with a small Q wave, high R).

4.The QRS is type rS in leads I, AVL (with a small R and deep S).

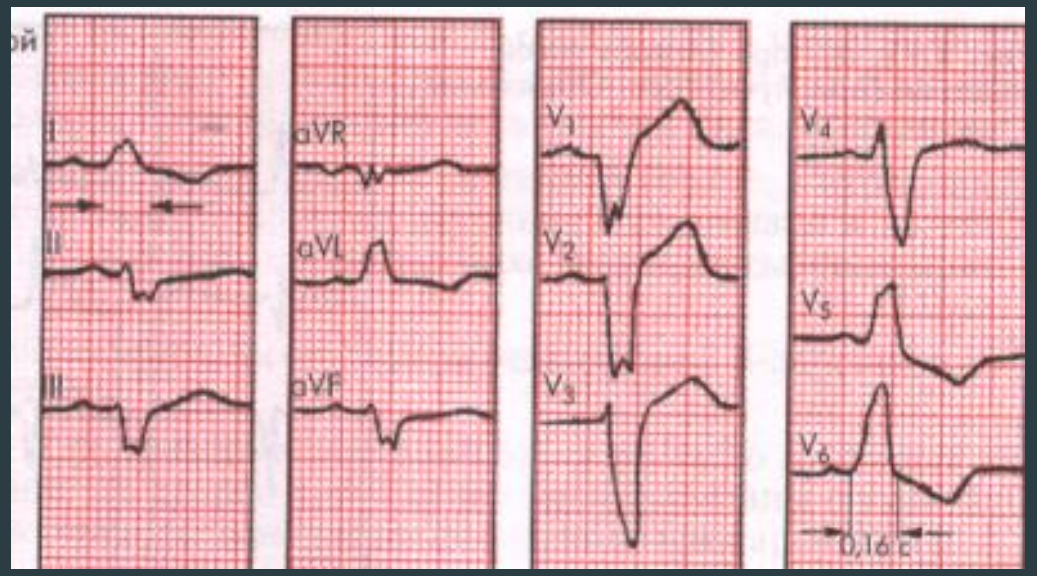
Summation

With blockade of the fasciculars of the left branche His bundle, ***the ventricular complex does not expand.***

Blockade of the branches of the left bound should be excluded in in I and II standard leads. If the S is larger than the R in these leads, then this is the LBBB.

- If in I standard lead, the S is larger than the R, and confirmed in the aVL, this is a block of the posterior branch.

- If in II standard lead, the S is larger than the R, this is a block of the anterior branch.



I44.7 Left bundle-branch block

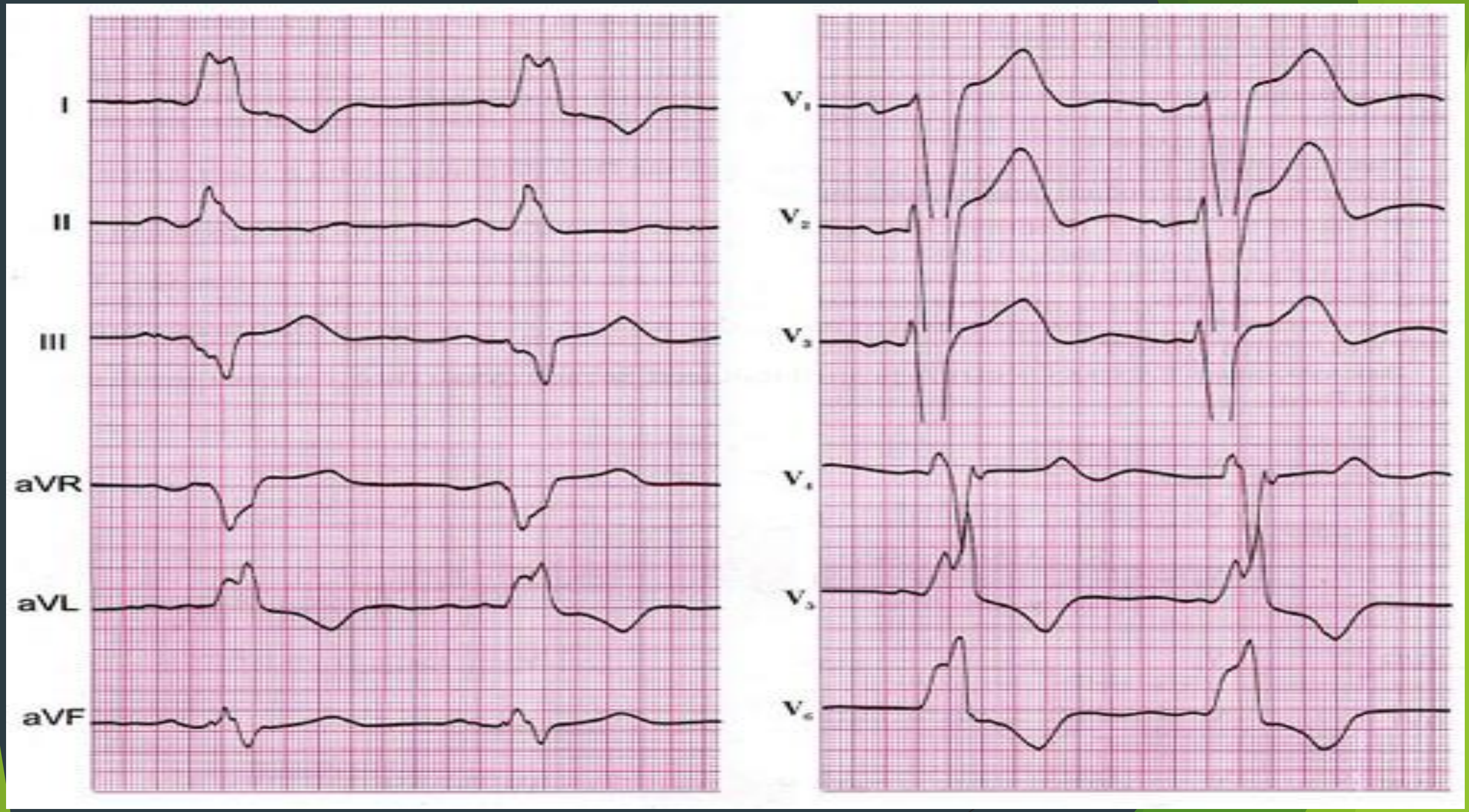
- QRS complex more than 0.12 sec .

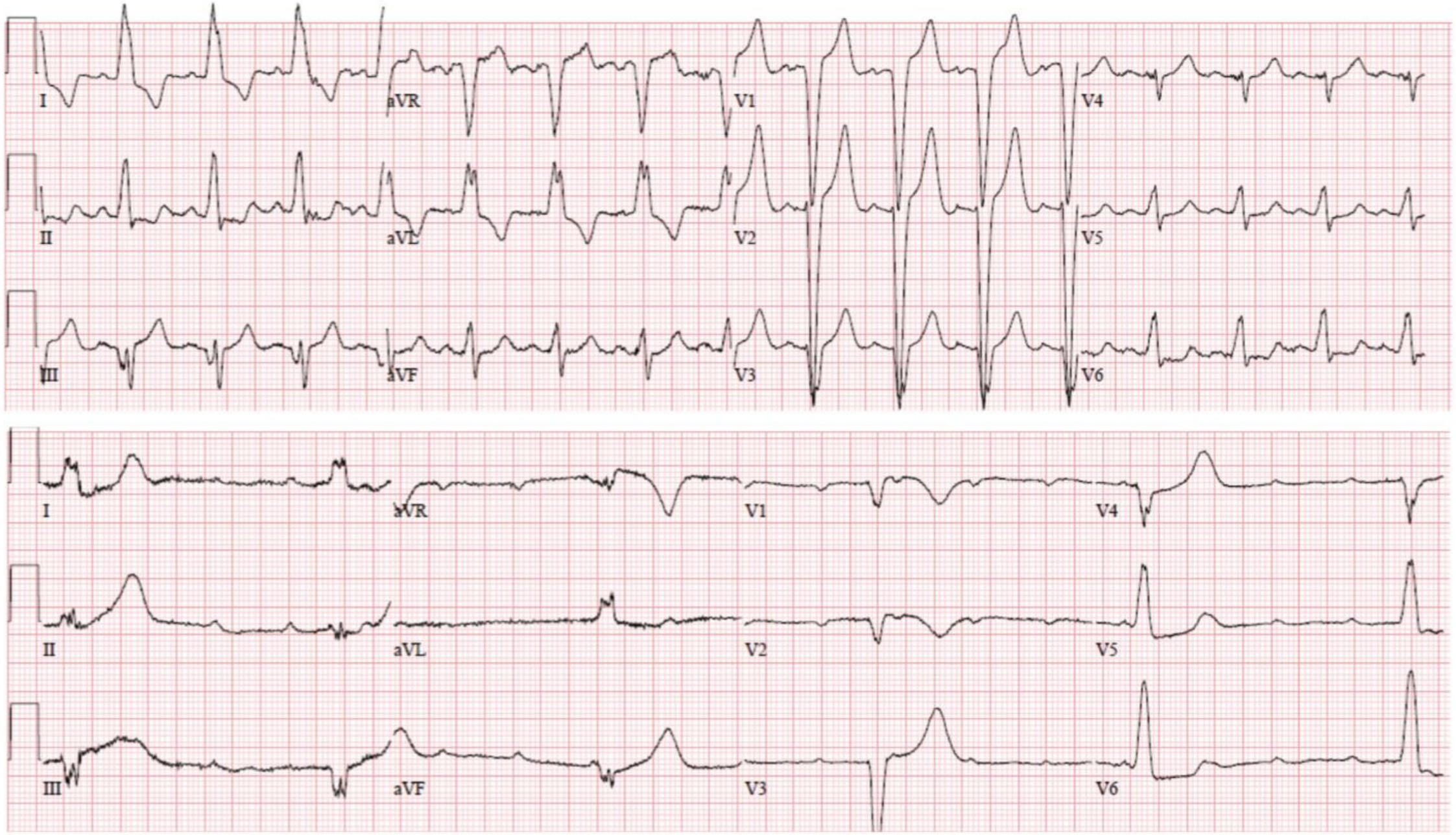
- wide split R with an internal deviation time of 0.08 sec. in the leads corresponding to the left ventricle (V5, V6, I, AVL);

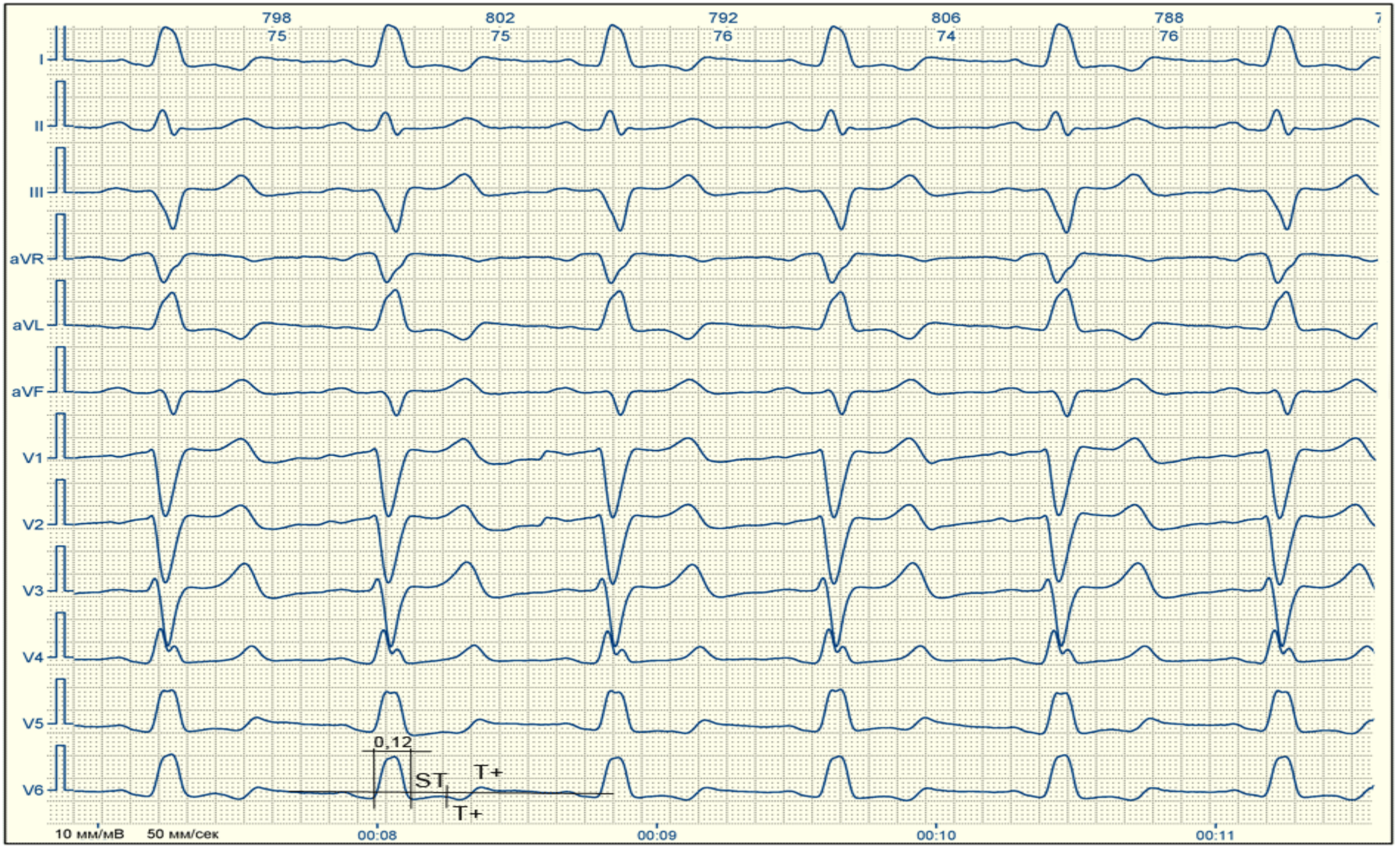
- complete absence of Q wave in I, aVL, V5, V6 leads.

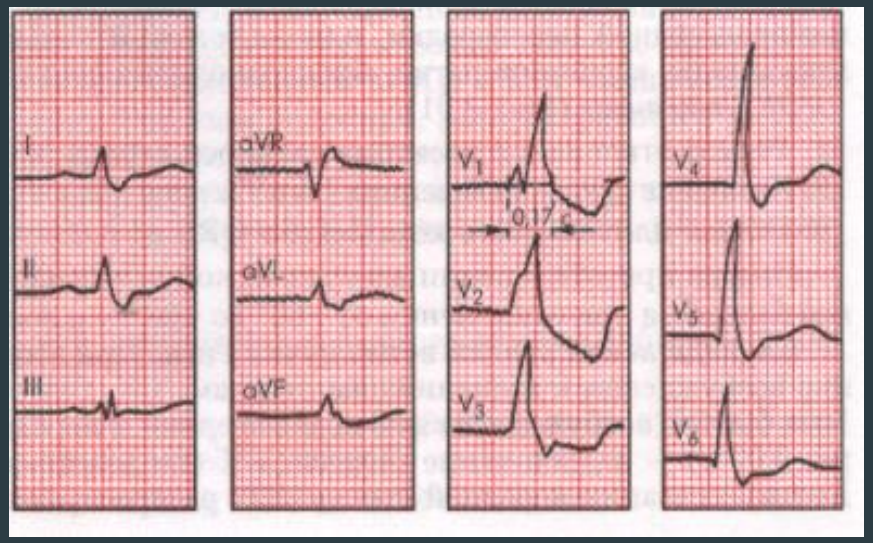
- extended serrated tooth S, QS in opposite leads V1, V2, III, AVF;

- ST segment displaced downward and negative T (-) in the leads corresponding to the left ventricle (V5-V6, I, AVL), ST upward shift and positive T (+) in the leads from the right ventricle (V1-V2, III, AVF).


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

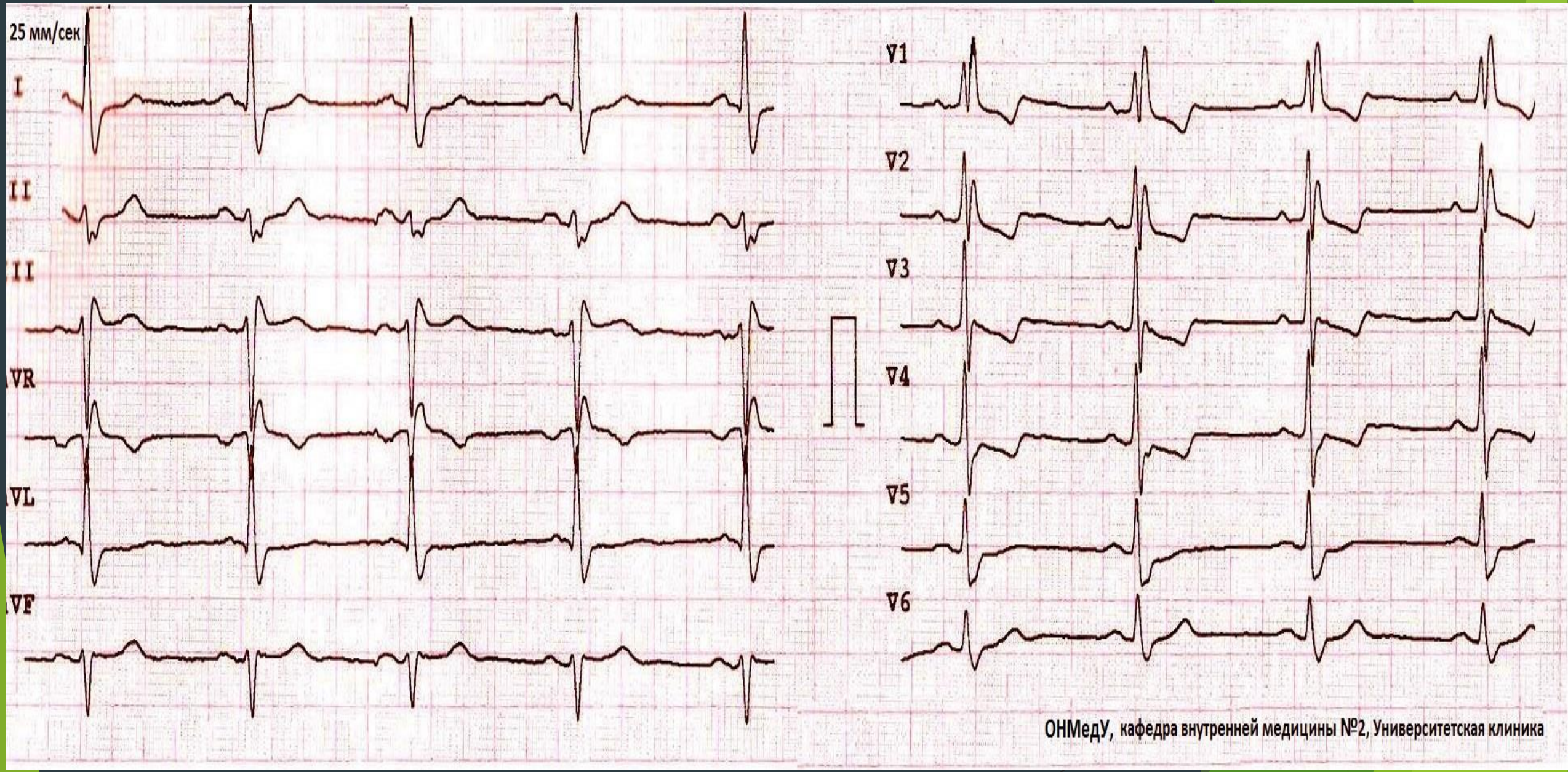

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

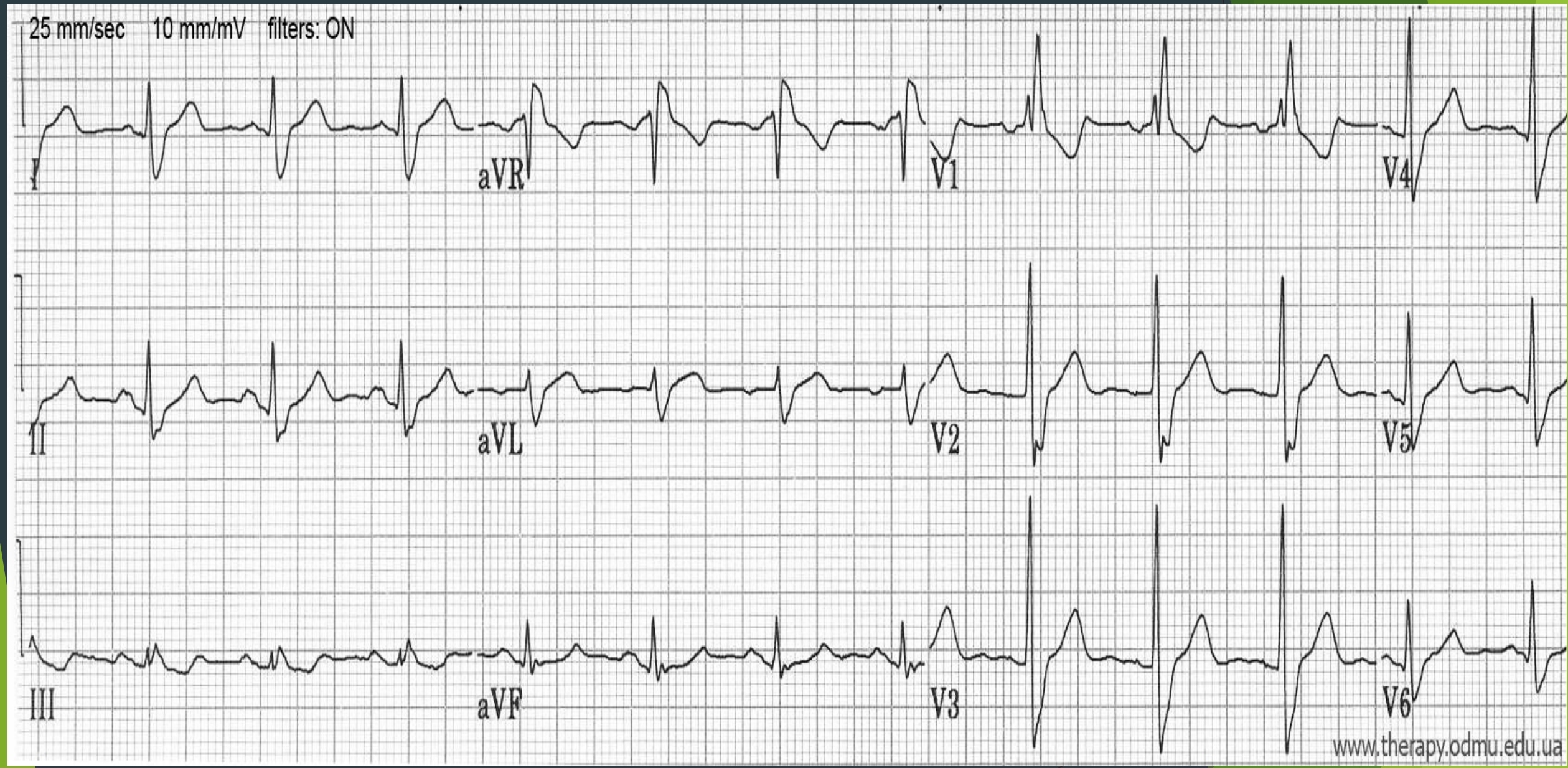

798
75
802
75
792
76
806
74
788
76
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
0,12
ST
T+
T+
10 мм/мВ
50 мм/сек
00:08
00:09
00:10
00:11

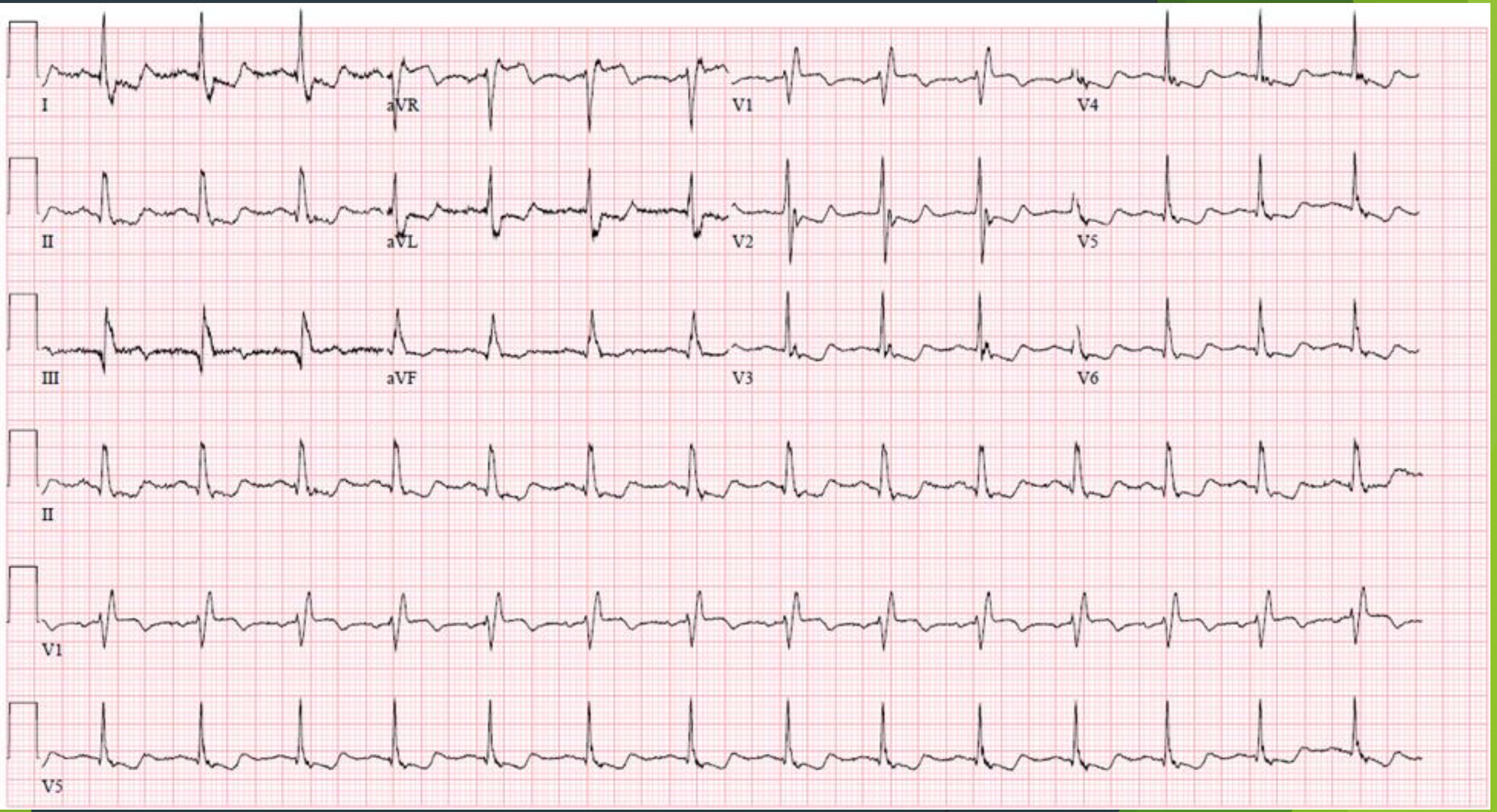


I45.1 Right bundle-branch block

- QRS broadening greater than 0.12 sec.
- in leads V1-V2 the QRS is in the form of the letter «M», rSR, RSR or rR
- time of internal deviation of QRS more than 0.06 sec. in assignments of V1, V2, V3, aVR;
- deep serrated S - duration of more than 0.04 sec. in V5-V6, I, aVL, in I -II leads R>S;
- ST segment displaced downward and negative T (-) in V1-V3,
- III, aVF - discordance of rR up and ST segment, T (-) down.


25 мм/сек
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
ОНМедУ, кафедра внутренней медицины №2, Университетская клиника


25 mm/sec 10 mm/mV filters: ON
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
www.therapy.odmu.edu.ua


I
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aVL
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V5
III
aVF
V3
V6
II
V1
V5

I45.4 Nonspecific intraventricular block

Non-specific defects of intraventricular conduction are diagnosed by ECG with a QRS complex duration > 12.0 sec, but it's shape is not typical for either LBBB or RBBB.

The slowdown can be outside the Purkinje system and be a manifestation of the slow conduction between cardiomyocytes.

No specific treatment is prescribed.

I45.5 Other specified heart block. Sinoatrial block

violation conduction of impulse from the sinus node to the atria.

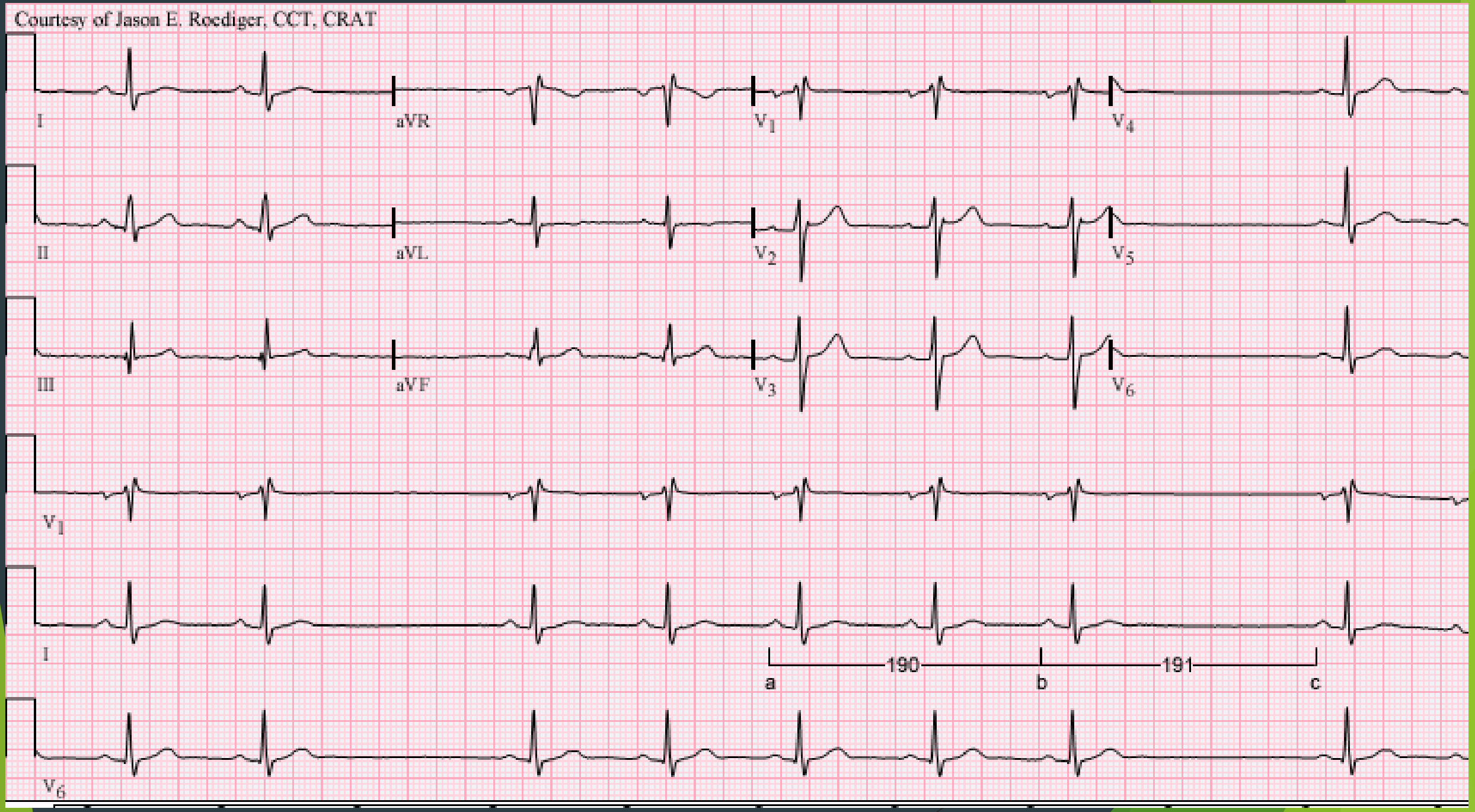
There are 3 degrees of SA block:

- I degree - is not detected on the routine ECG;
- II degree:

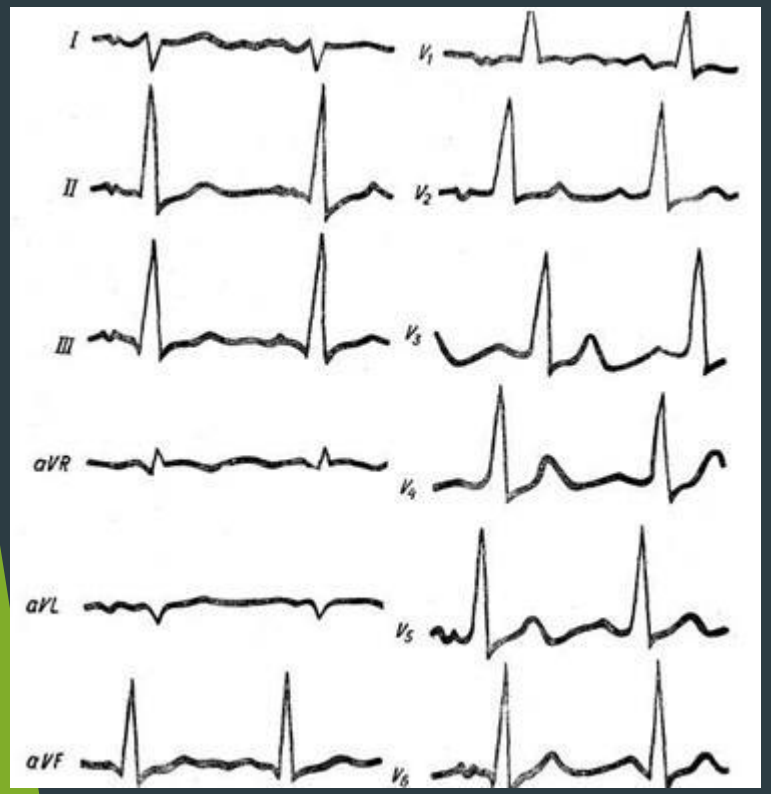
- the rhythm is wrong;
- periodic loss of the heart cycle P-QRST;
- during long pauses, complexes and rhythms slip out.

- III degree - stop of the sinus node:

- ectopic rhythms - atrial, nodular, idioventricular etc.


Courtesy of Jason E. Roediger, CCT, CRAT
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
I
190
191
a
b
c
V6

I45.5 Other specified heart block.
Sinoauricular block



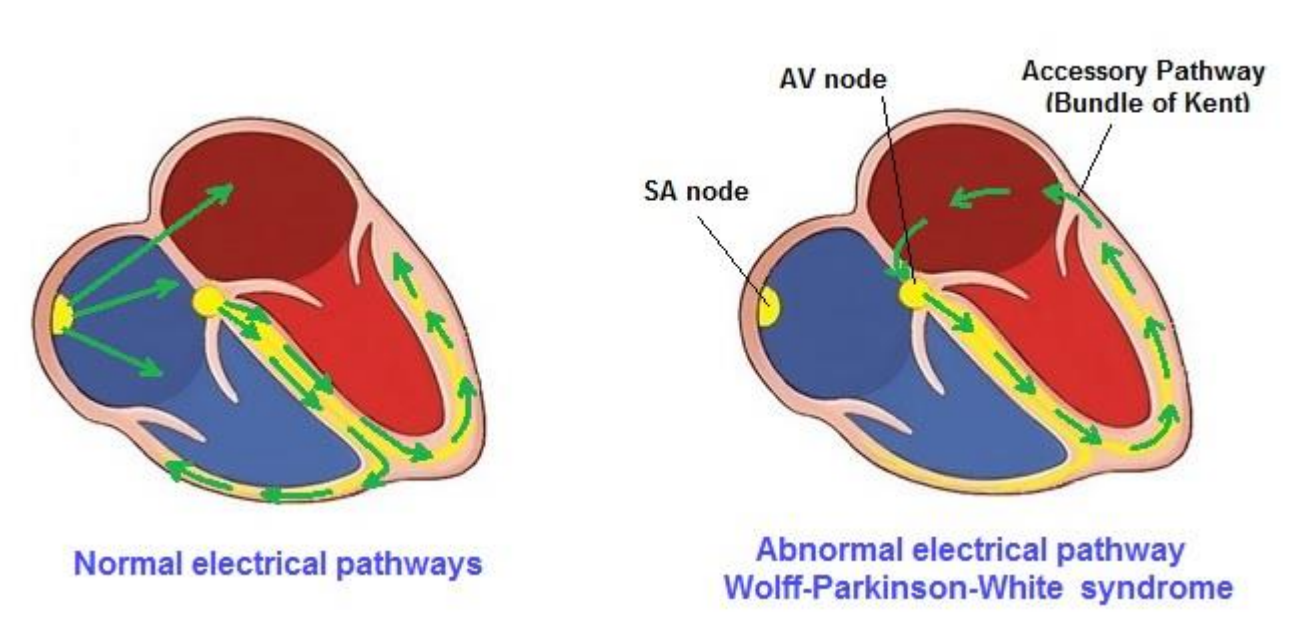
Atrial block is a violation of the passage of excitation in the atrias. The mechanism of intra-atrial blockade can be explained by the defeat of the intra-atrial bundles of the cardiac conduction system, mainly the Bachmann bundle.

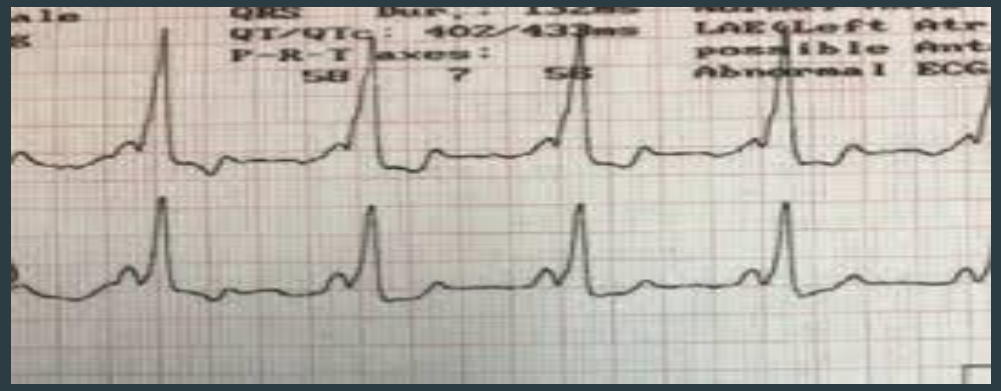
In the clinic, atrial block is observed with large morphological changes in the atrial myocardial contractility.

On ECG the P wave broadens (more than 0.12 s), splits or bifurcates, and sometimes becomes biphasic. Often, atrial block is combined with intraventricular block. In case of complete intra-atrial block, two different forms of the P with an independent rhythm are noted. At the same time, ventricular contractions can be associated with one of these sources of rhythm, or alternately with both.

I45.6 Pre-excitation syndrome.
WPW Syndrome.

Wolff-Parkinson-White syndrome is the most common syndrome of premature ventricular excitation that occurs when there is an additional Kent bundle. Most people do not have signs of heart disease.



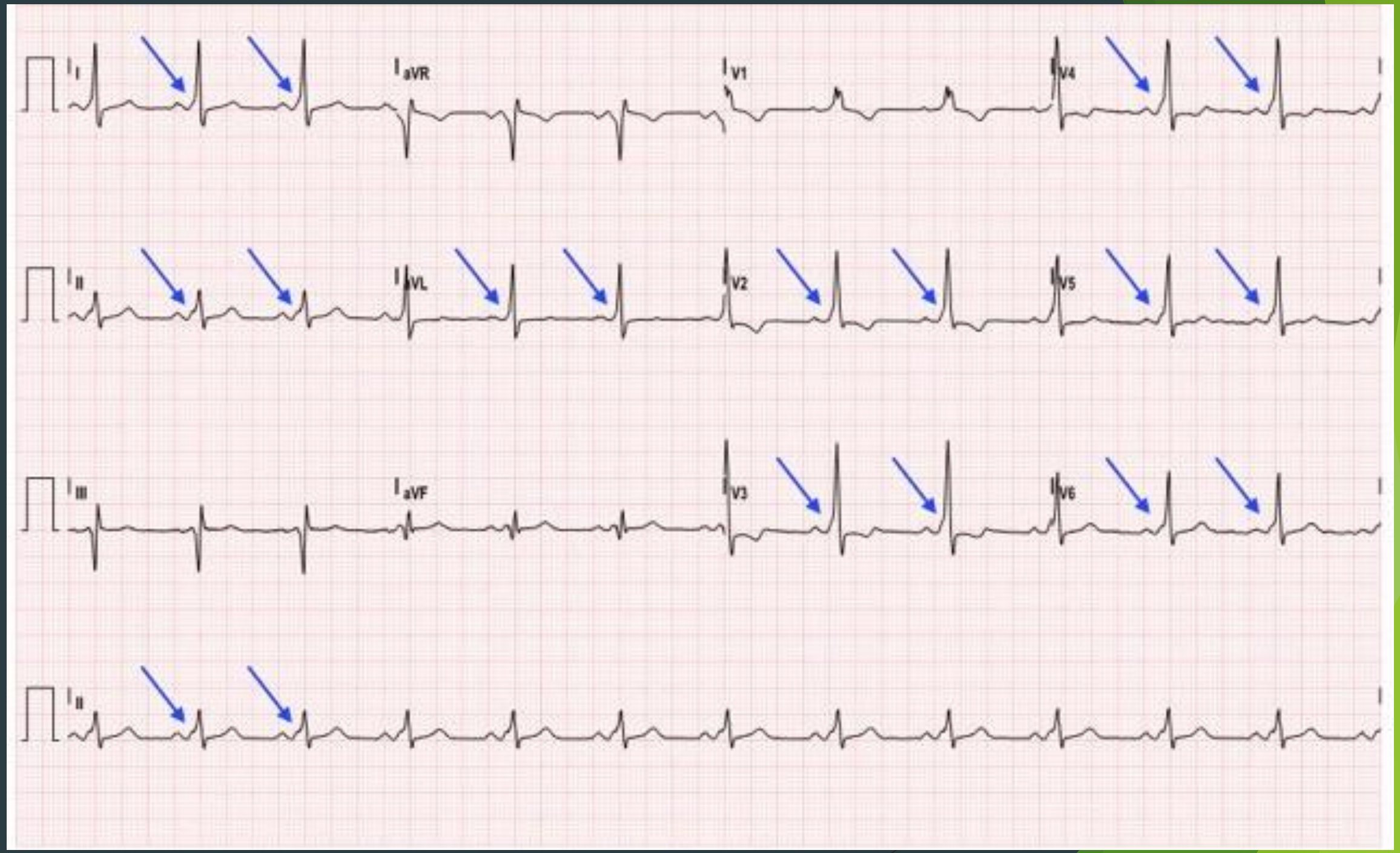


The ***short interval P- Q (P-R), less than 0.12 sec.***

The wave Δ. Its appearance is associated with a "confluent" contraction of the ventricles (first, the excitation of the ventricles through the additional conduction path, and then through the AV connection).

The expansion of the ***QRS complex is more than 0.1 sec*** due to the wave Δ.

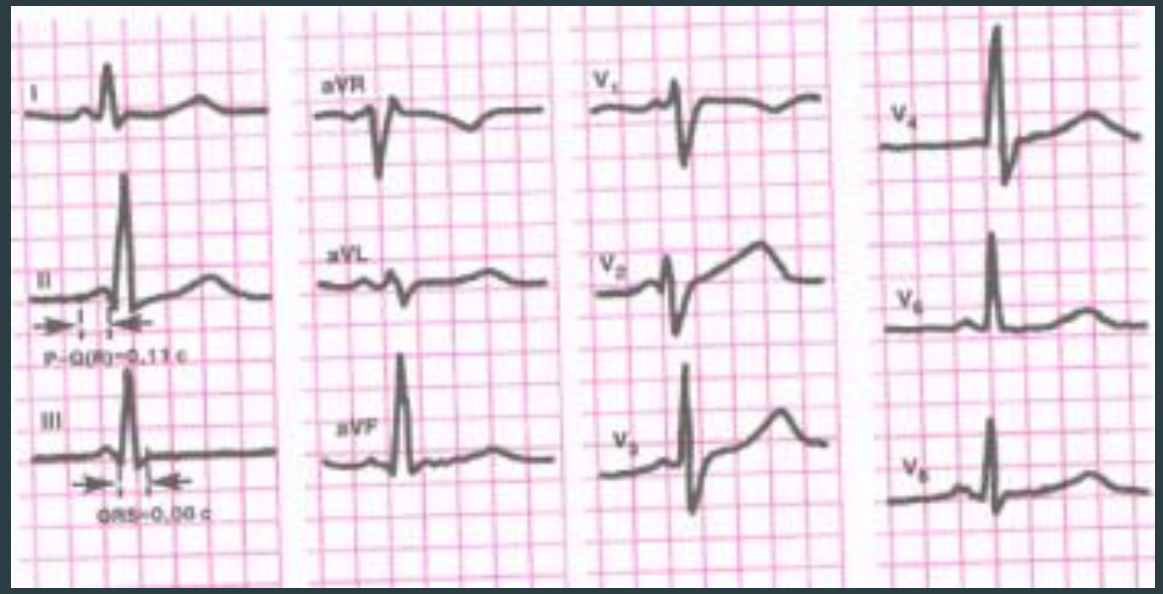
Tachyarrhythmias: orthodromic and antidromic supraventricular tachycardia, atrial fibrillation and flutter. Tachyarrhythmias usually occur after supraventricular extrasystoles.


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

I45.6 Pre-excitation syndrome.
LGL Syndrome.

The Lown-Ganong-Levine (LGL) syndrome occurs when an accessory pathway is congenitally present that directly connects the atria to the ventricles, bypassing the AV node similar to the WPW syndrome.

The main distinguishing feature between LGL and WPW syndromes is that the accessory pathway in LGL syndrome connects distally to the normal conduction pathway (bundle of His), and in WPW the accessory pathway connects to the ventricular myocardium.



The ***short interval P- Q (P-R), less than 0.12 sec.***

Normal (unchanged) QRS complex.

Tachyarrhythmias: orthodromic and antidromic supraventricular tachycardia, atrial fibrillation and flutter.

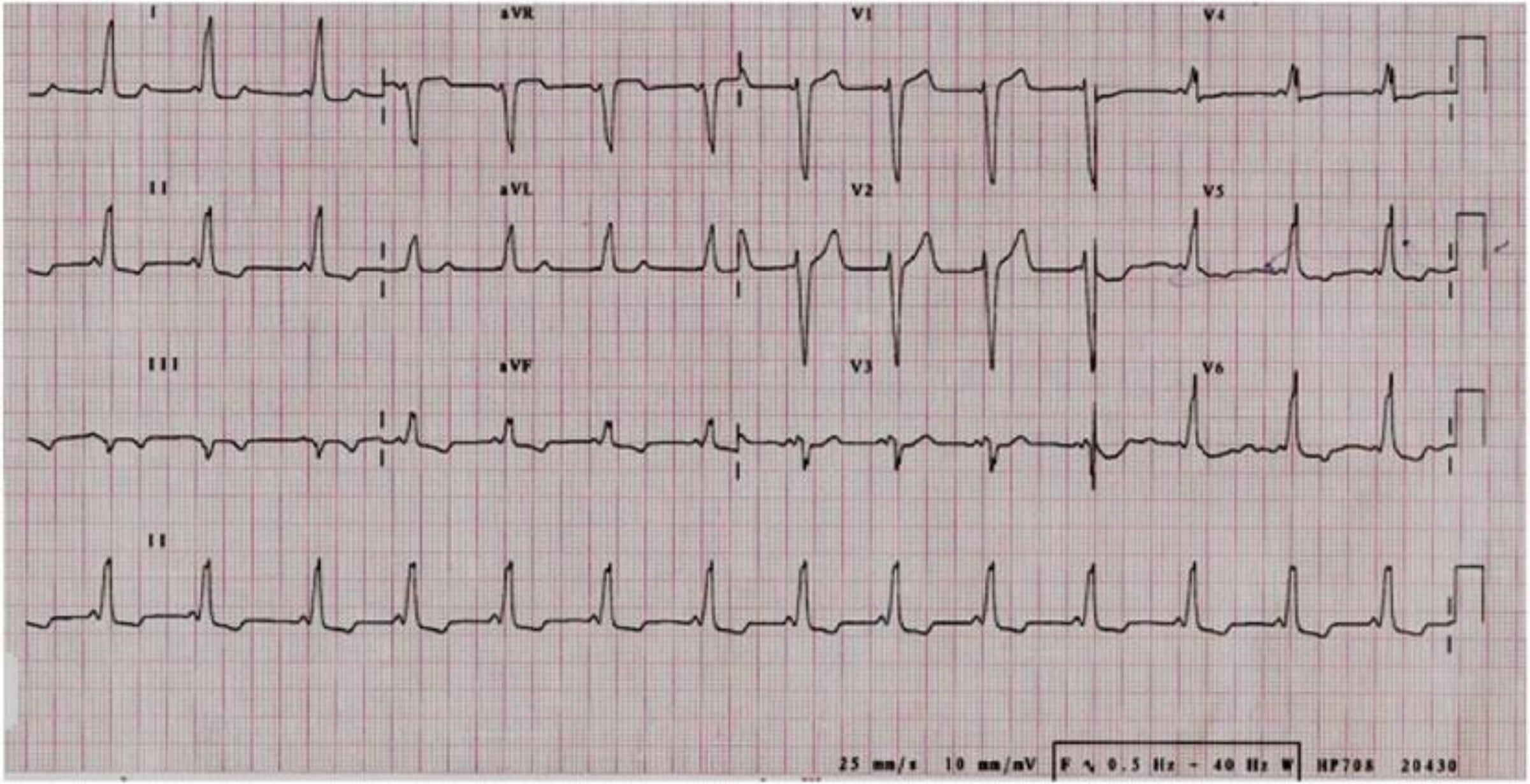


Figure 1. Twelve-lead ECG of the patient.

I45.6 Pre-excitation syndrome.

The essence of the syndrome (phenomenon) of premature ventricular excitation consists in the anomalous propagation of excitation from the atria to the ventricles along the so-called additional pathways, which in most cases partially or completely "bypass" the AV node.

- Maheima fibers connecting the AV node to the right side of the interventricular septum or the branches of the right bundle branch, less commonly the bundle of the bundle with the right ventricle. In this situation on ECG:

the normal (rarely elongated) ***PQ interval***,

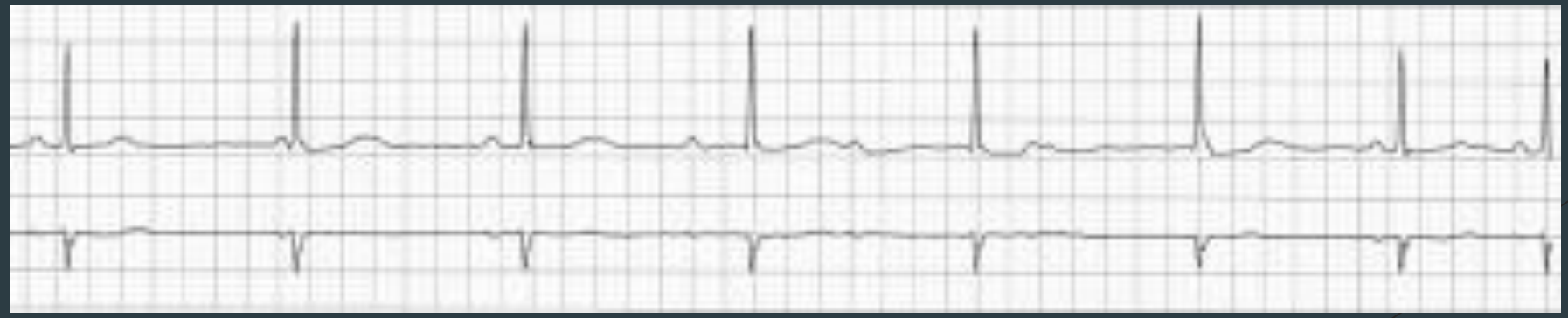
the delta wave,

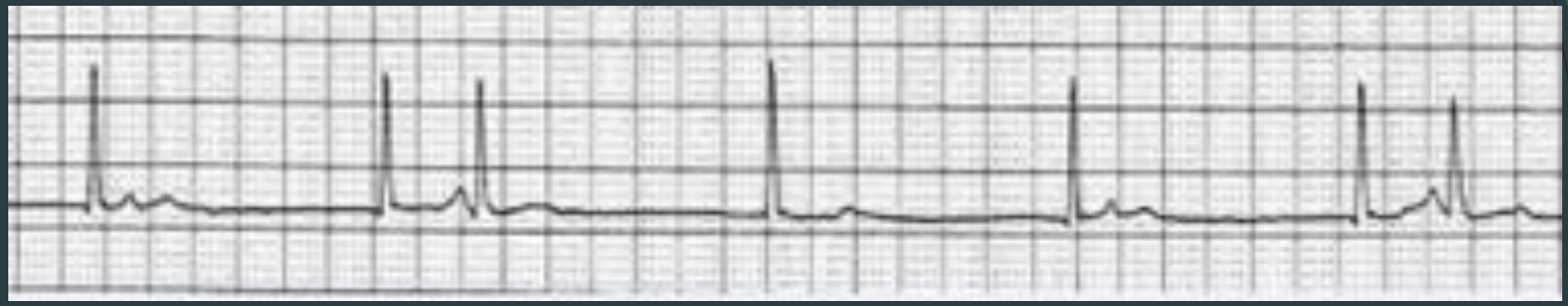
and ***the extended QRS complex*** are recorded.

I45.8 Other specified conduction disorders
Atrioventricular [AV] dissociation

AV dissociation is present when atrial and ventricular activation are independent of each other. It can result from complete heart block or from physiologic refractoriness of conduction tissue. AV dissociation can also occur in a situation when the atrial/sinus rate is slower than the ventricular rate (eg, with accelerated junctional tachycardia and ventricular tachycardia).

Occasionally, the atrial and ventricular rates are so close that the tracing would suggest normal AV conduction; only careful examination of the long rhythm strip may reveal a variation in the PR interval.





Atrial-ventricular dissociation is characterized by the presence of independent pacemakers of the atria and ventricles in the absence of retrograde conduction of ventricular impulses. It can occur in combination with AV blockade or in the absence of the latter.

A prerequisite for the development of atrial-ventricular dissociation and the main criterion for its diagnosis is a ***higher frequency of ventricular rhythm compared with the frequency of atrial excitation*** caused by a sinus or ectopic atrial pacemaker. Often this difference is very small.

I45.8 Other specified conduction disorders
Interference dissociation

Contradirectional interference results when 2 stimuli arising in different foci in any part of the heart spread in opposite directions toward each other. Interference dissociation is defined as that type of dissociation which is due to repetitive contradirectional interference.

60years
Female Caucasian

Vent. rate 59 bpm
PR interval ms
QRS duration 84 ms
QT/QTc 426/421 ms
P-R-T axes 80 85 79

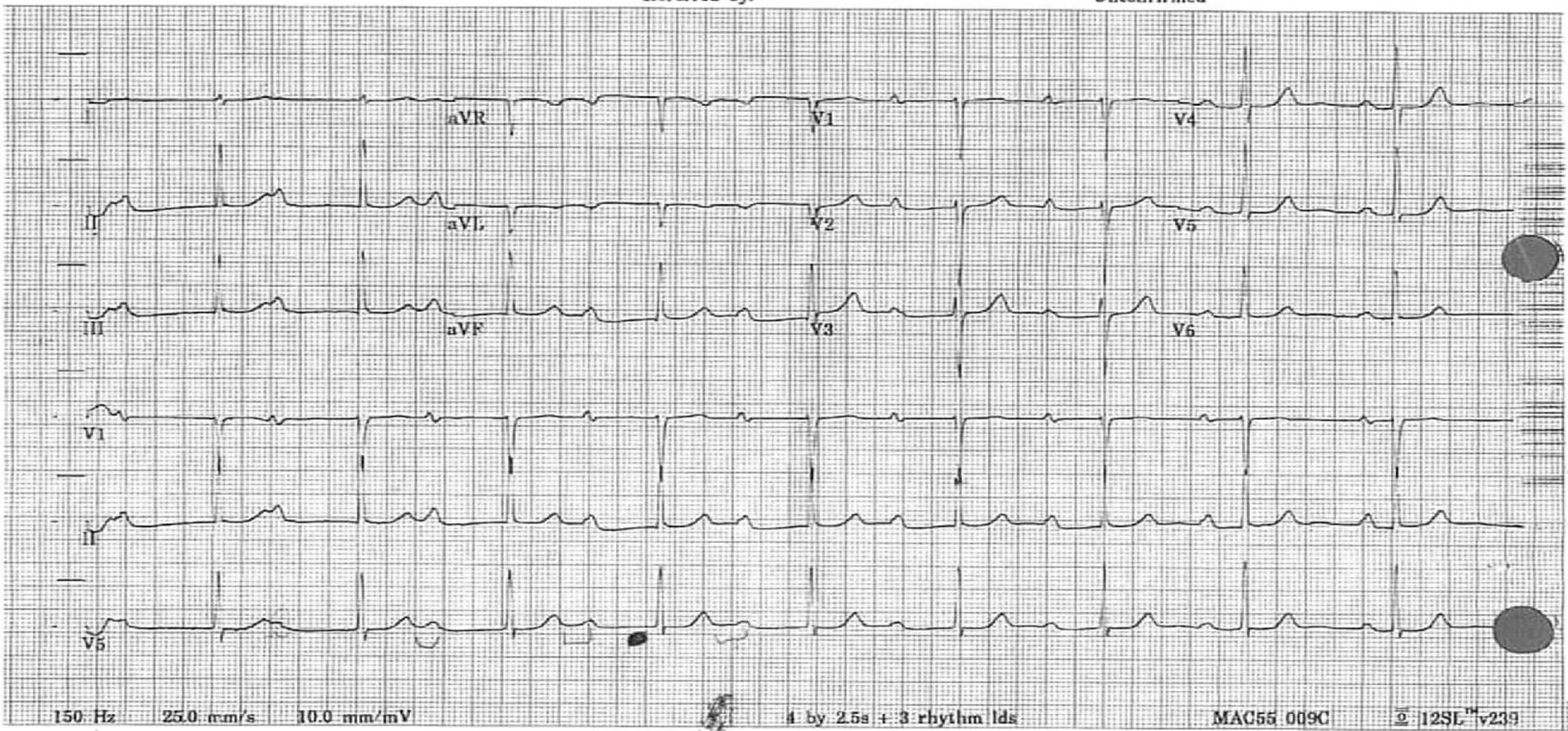
Sinus bradycardia with AV dissociation and Junctional rhythm with sinus/atrial capture
Abnormal ECG

3° blk

Technician:
Test ind:

Referred by:

Unconfirmed



150 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MAC55 009C 12SL v239

Gerath J. Evans, PA
LT, MSC, USN
Emergency Medicine
NPI 1720237845

01:01:18 p.m. 08-18-2015

https://www.heartrhythmcasereports.com/article/S2214-0271(16)30104-X/fulltext

Sinus node dysfunction (Sick sinus syndrome)

a collective term. It includes several rhythm and conduction disorders associated with malfunctioning of the sinus node and leading to the development of bradycardia:

- Severe sinus bradycardia that does not correspond to the patient's activity (or <40 beats / min during the day or <30 beats / min during sleep on a holter monitoring).

- CA block II.

- Substitutive rhythms (atrial, AV-nodular, idioventricular)

- Pauses for 2 seconds and more (due to sinus arrest or high-grade CA blockade).

- Tachycardia-bradycardia syndrome (alternation of supraventricular tachycardia - sinus, AV-nodular, atrial fibrillation - and bradycardia - sinus, atrial, AV-nodal, etc.).

- Slowing the restoration of the sinus node after episodes of tachycardia (a pause, normally not exceeding 1.5 seconds, can last 4-5 seconds and be accompanied by loss of consciousness).

Adams–Stokes syndrome

A syncope caused by a sharp decrease in cardiac output and cerebral ischemia due to acute cardiac arrhythmias (sinoatrial block 2 degrees or complete atrioventricular block, paroxysmal tachycardia, ventricular fibrillation, weakness and dullness of the sinus-atrial node, etc.).

This is clinical and functional syndrome!

I 46 Cardiac arrest

I46.0 Cardiac arrest with successful resuscitation

I46.1 Sudden cardiac death, so described

I46.9 Cardiac arrest, unspecified

Cardiac arrest is the abrupt loss of heart function in a person who may or may not have been diagnosed with heart disease. Cardiac arrest is often fatal, if appropriate steps aren't taken immediately.

Cardiac arrest and heart attack are not synonyms!

Cardiac arrest is when the ***heart malfunctions and suddenly stops beating*** unexpectedly, is an "electrical" problem.

Heart attack is when ***blood flow to the heart is blocked***, is a "circulation" problem.

I47 Paroxysmal tachycardia

Paroxysmal tachycardia - a type of arrhythmia, which is manifested by a ***sudden increase in heart rate (paroxysms) with heart rate from 140 to 250 beats per minute***, occurring under the influence of ectopic impulses atrioventricular junction or ventricles. The duration of an attack can range from a few seconds to several hours, and only rarely it lasts longer.

In the genesis of paroxysmal tachycardia, two mechanisms of its occurrence have greatest importance - the pathological circulation of an ordinary electrical impulse and the activation of a pathological focus of high automatism.

I47.0 Re-entry ventricular arrhythmia

I47.1 Supraventricular tachycardia

Tachycardia (paroxysmal):

- atrial
- atrioventricular
- junctional
- nodal

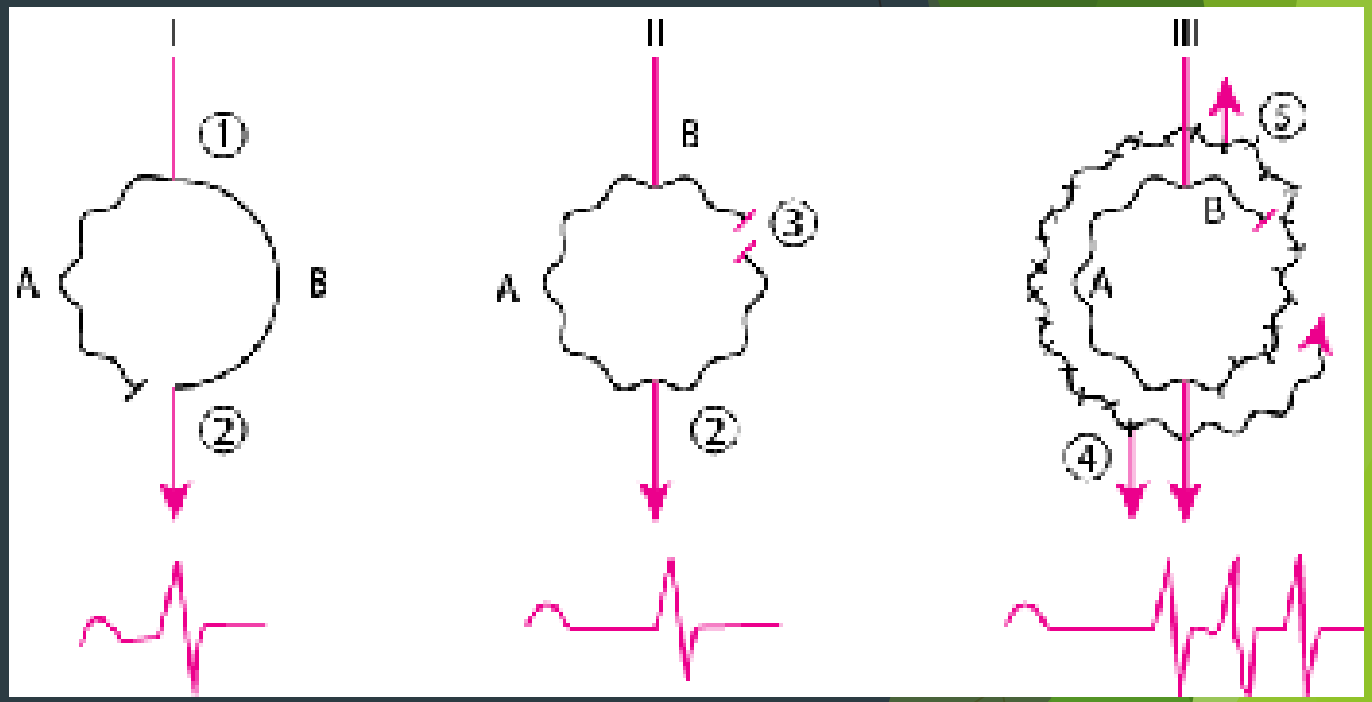
I47.2 Ventricular tachycardia

I47.9 Paroxysmal tachycardia, unspecified

Bouveret(-Hoffmann) syndrome

I47.0 Re-entry ventricular arrhythmia

Reentry describes a self-sustaining cardiac rhythm abnormality. In reentry, the action potential propagates in a circus-like closed loop manner. It is a disorder of impulse conduction and thus describes one kind of arrhythmogenesis and is differentiated from disorders of impulse generation such as automaticity and triggered activity. Dysrhythmias based on the reentry mechanism include Atrial tachyarrhythmias such as Atrial flutter, Atrioventricular nodal reentry, Atrioventricular reentry like Wolff-Parkinson-White syndrome and ventricular reentry such as Bundle branch reentry.



I47.1 Supraventricular tachycardia

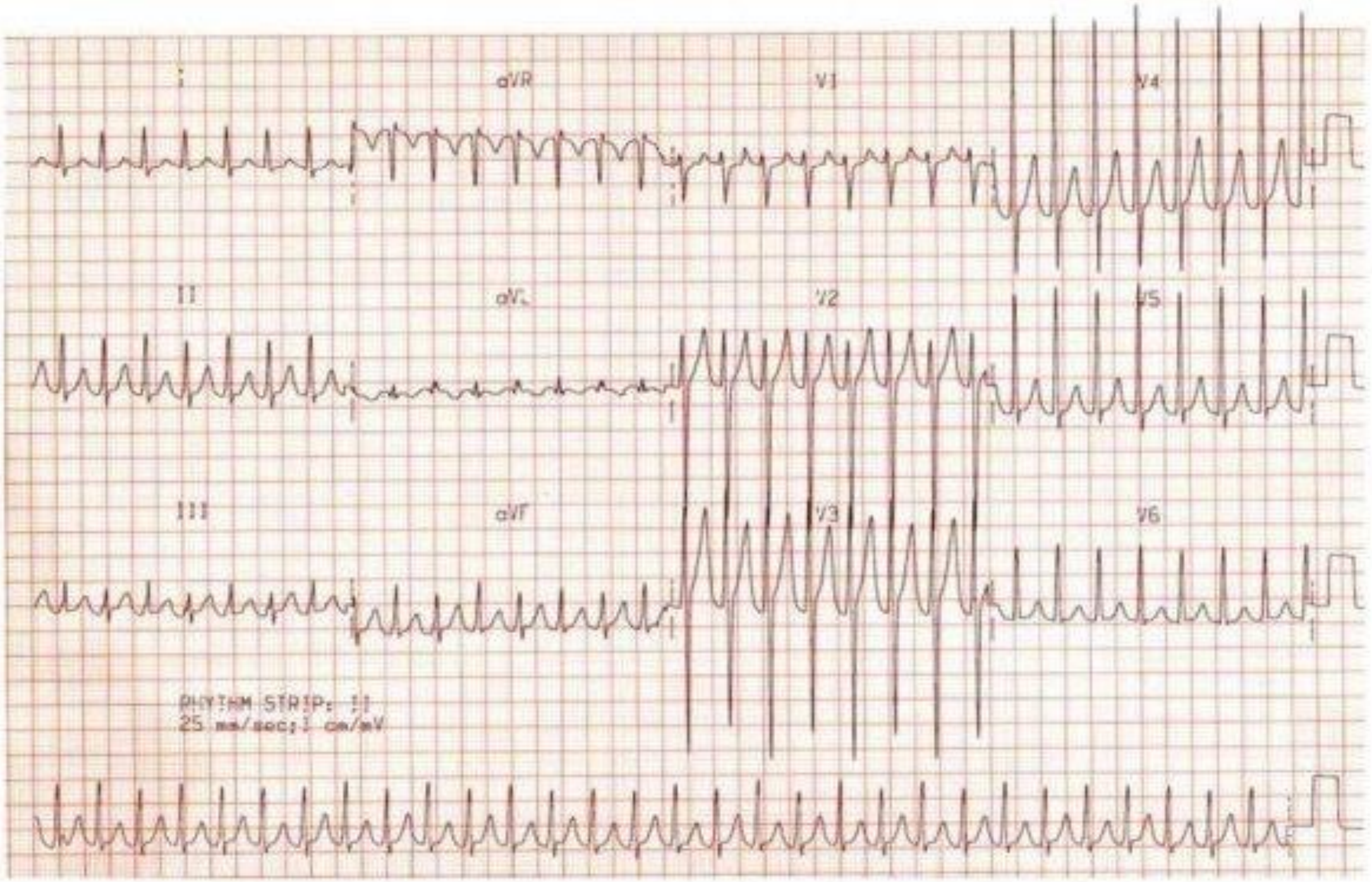
Sudden onset and sudden end of paroxysm.

Heart rate ***from 140 beats per minute***.

Correct (regular) rhythm.

Usually the QRS complexes are normal.

P-waves can be different. In case of atrial PT, P waves are before the QRS complexes, but are reduced or deformed. If PT from the AV node, the P waves are located after the QRS complexes or are layered on them.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

I47.2 Ventricular tachycardia

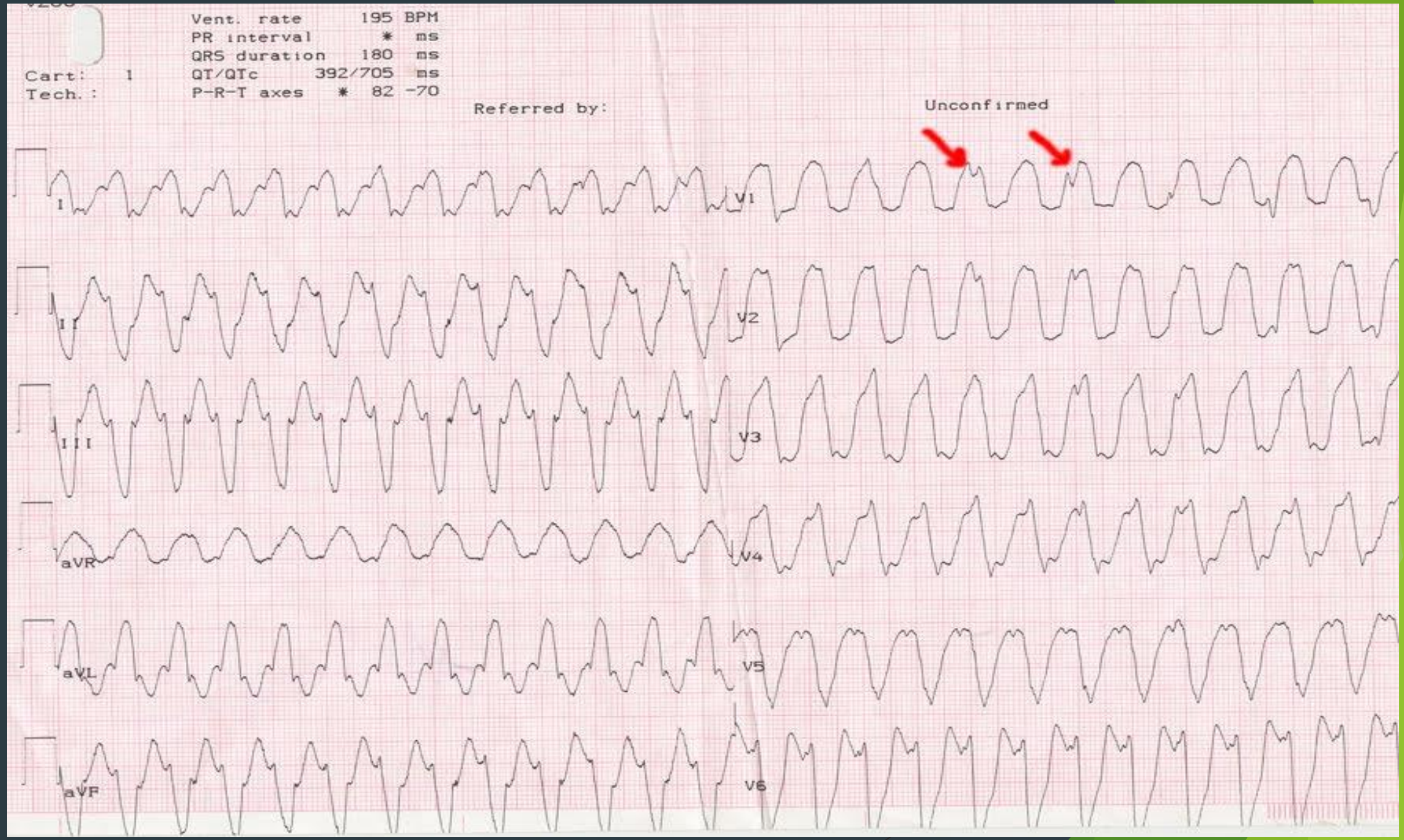
Heart rate ***from 140 beats per minute***.

Deformation of the QRS complex, reminiscent of the form of a bundle branch blockade (QRS more then 0.12 s).

Dissociation in the activity of the atria and ventricles.

The R — R are significantly shortened, constant.

If 5 or more ventricular extrasystoles follow in a row, we can talk about ventricular paroxysmal tachycardia.


Vent. rate 195 BPM
PR interval * ms
QRS duration 180 ms
QT/QTc 392/705 ms
P-R-T axes * 82 -70
Cart: 1
Tech. :
Referred by:
Unconfirmed
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

I48 Atrial fibrillation and flutter

I48.0 Paroxysmal atrial fibrillation

I48.1 Persistent atrial fibrillation

I48.2 Chronic atrial fibrillation

I48.3 Typical atrial flutter

Type I atrial flutter

I48.4 Atypical atrial flutter

Type II atrial flutter

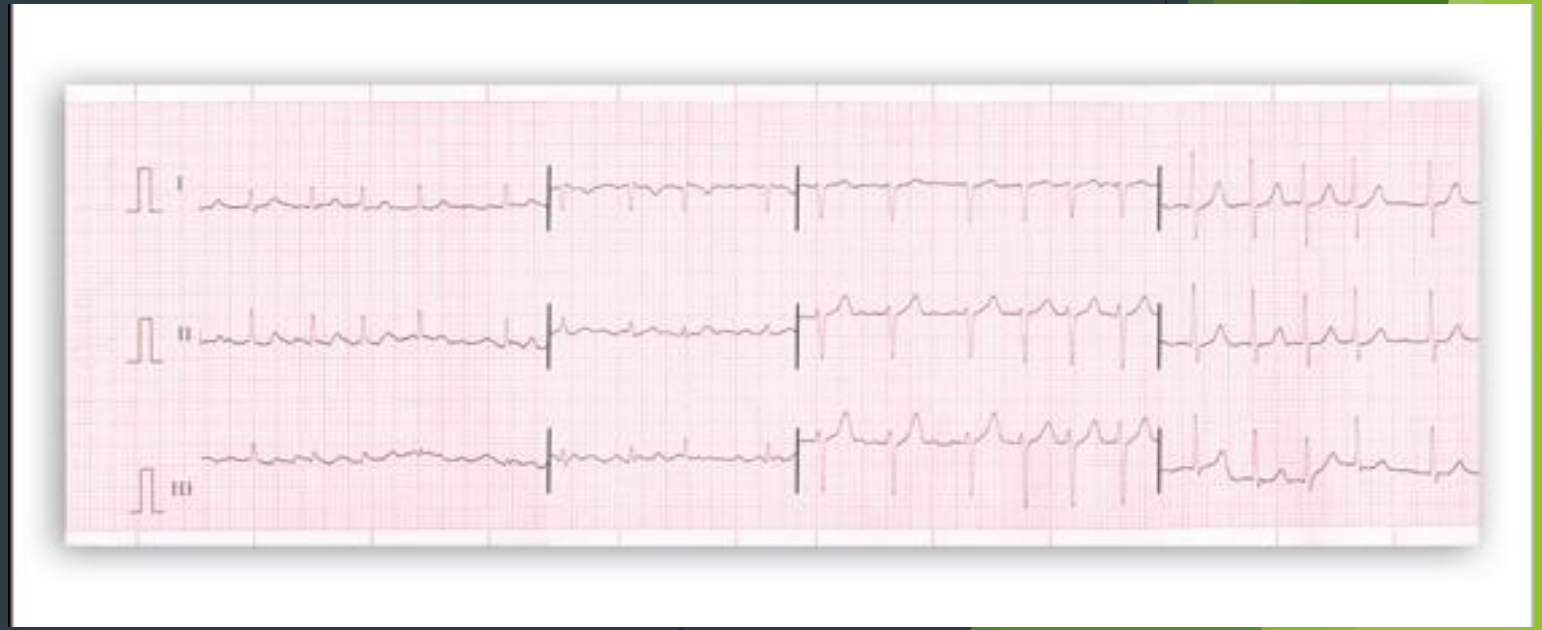
I48.9 Atrial fibrillation and atrial flutter, unspecified

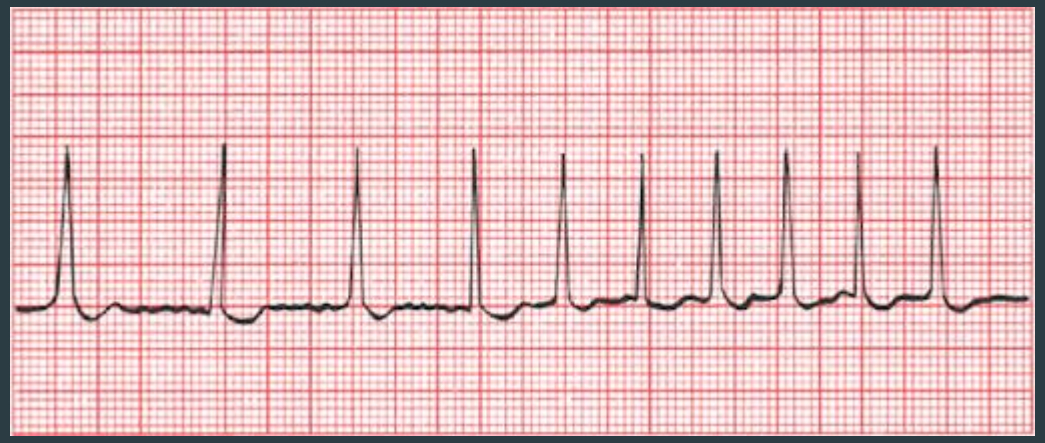
I49 Other cardiac arrhythmias

Atrial fibrillation

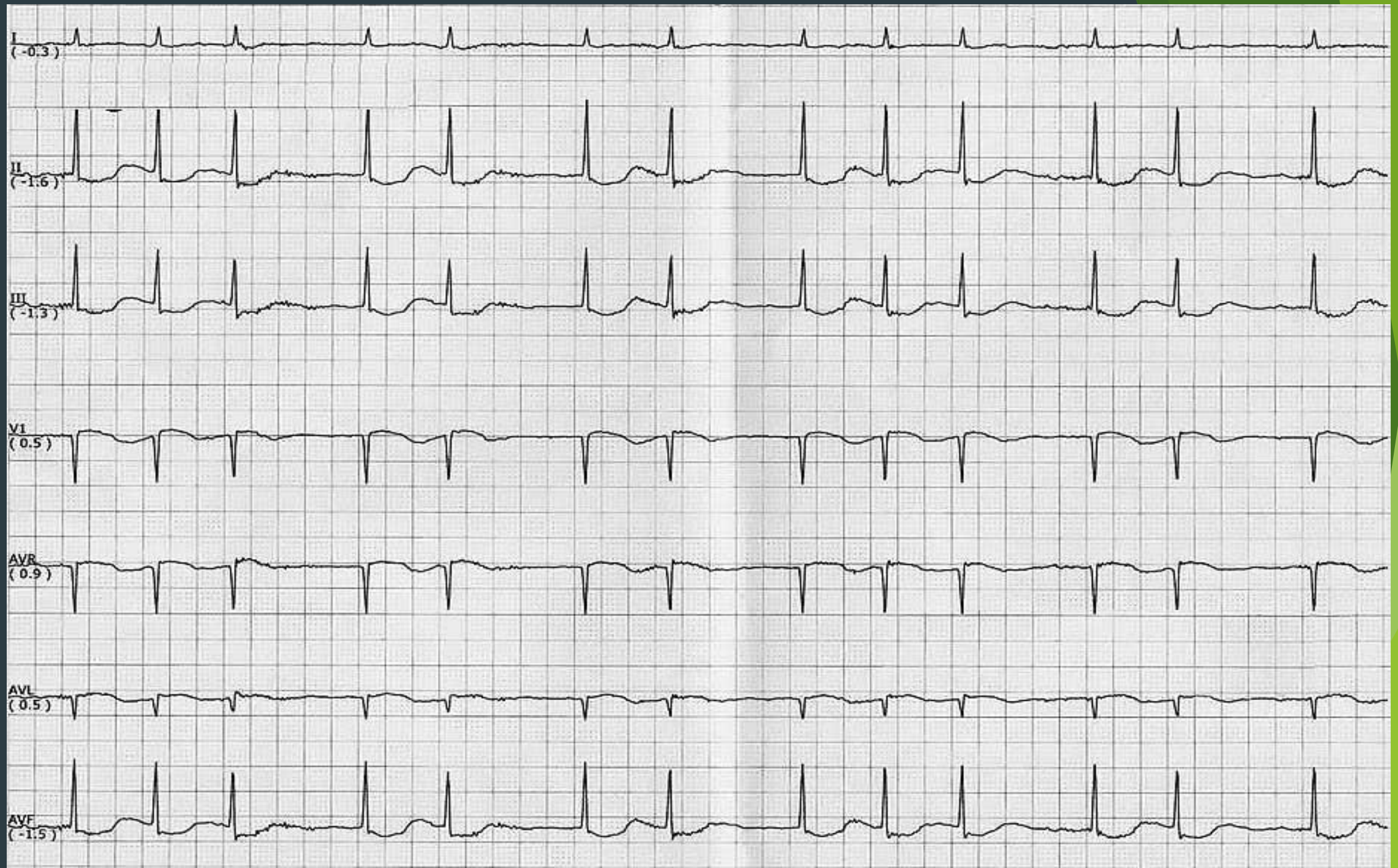
is a very frequent chaotic ***excitation of individual muscle fibers of the atria*** with a ***frequency of 350-700 per minute***. There is no excitation of the atria as a whole. The atrioventricular node receives a different number of impulses of different strength per unit of time. Some of them, sufficient in strength, pass to the ventricles, causing their excitation and contraction.

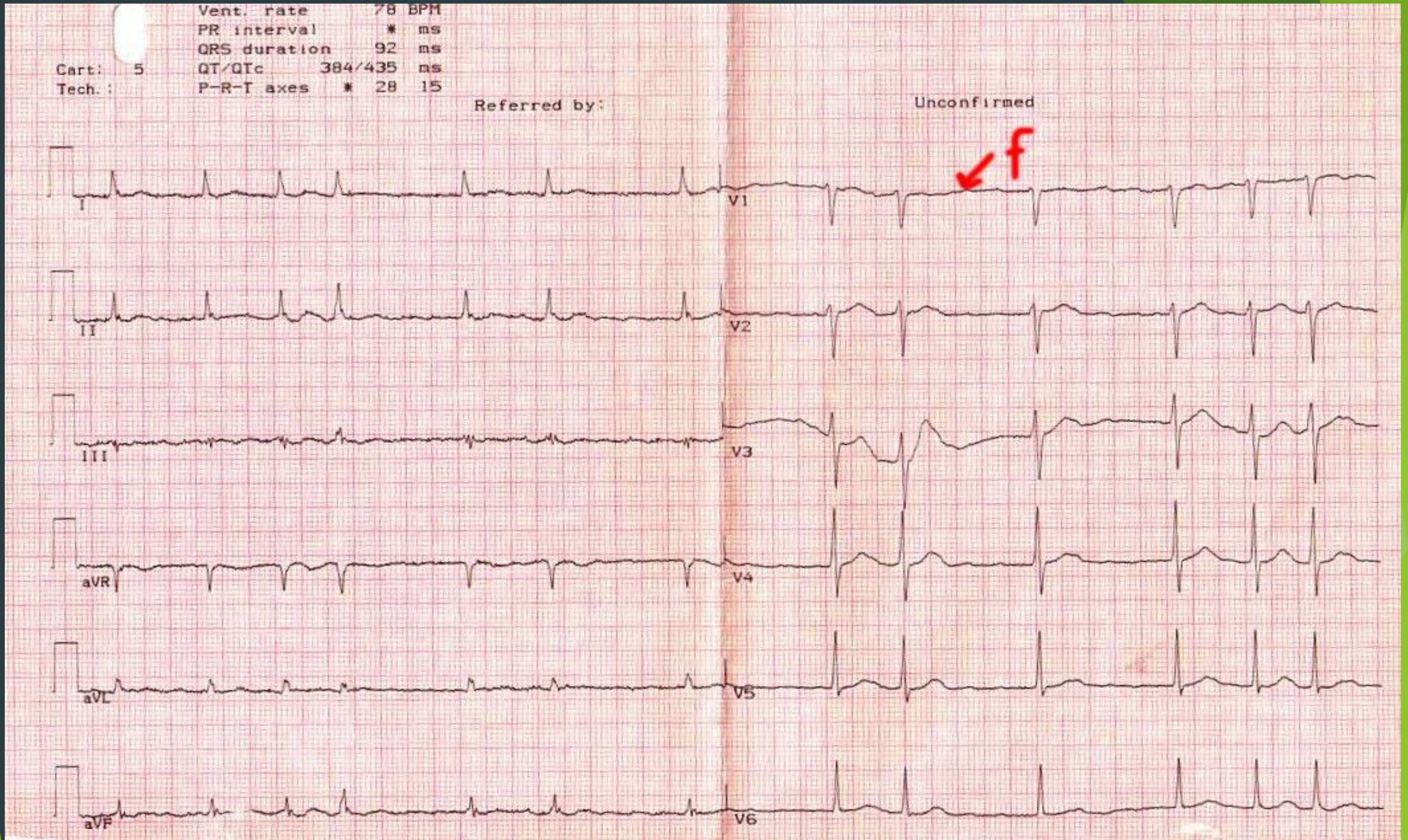
There are bradystolic (the number of ventricular contractions is less than 60 per 1 minute), normosystolic (the number of ventricular contractions 60-90 per 1 minute), tachysystolic (the number of ventricular contractions above 90 per 1 minute) forms of atrial fibrillation.





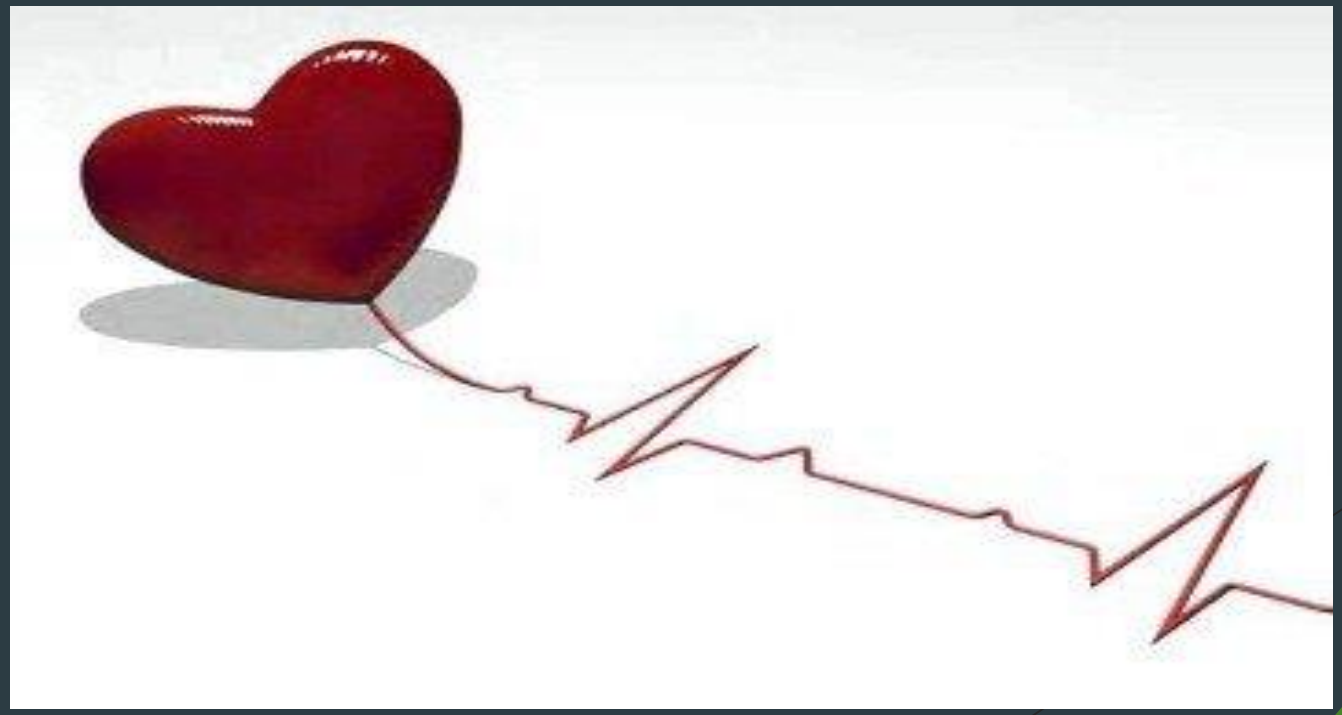
Absence of P waves in all leads (rhythm is not sinus);
The presence of ***small waves "f"*** with a frequency of over 350 per minute, irregular, with different amplitude and shape. Depending on the size of the "f" large-wave, small-wave atrial fibrillation is distinguished.
QRS complexes are not changed.
R-R intervals are different (the ventricular rhythm is irregular).

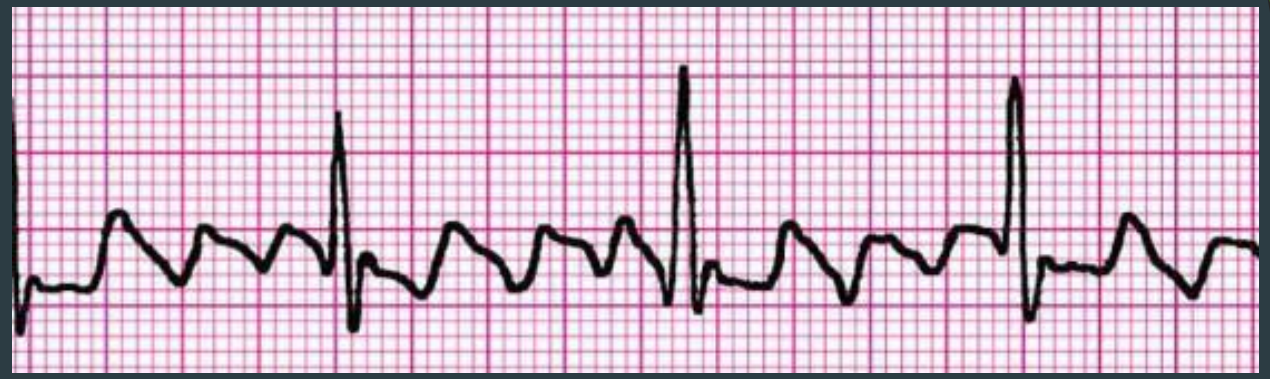

I
(-0.3)
II
(-1.6)
III
(-1.3)
V1
(0.5)
AVR
(0.9)
AVL
(0.5)
AVF
(-1.5)


Vent. rate 78 BPM
PR interval * ms
QRS duration 92 ms
QT/QTc 384/435 ms
P-R-T axes * 28 15
Cart: 5
Tech. :
Referred by:
Unconfirmed
f
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Atrial flutter

excitation of individual groups of muscle fibers of the atria with a frequency of 200-350 beats/min. The atrioventricular node receives a constant number of impulses from the atria per unit time, but not all of them reach the ventricles (every second or less often).



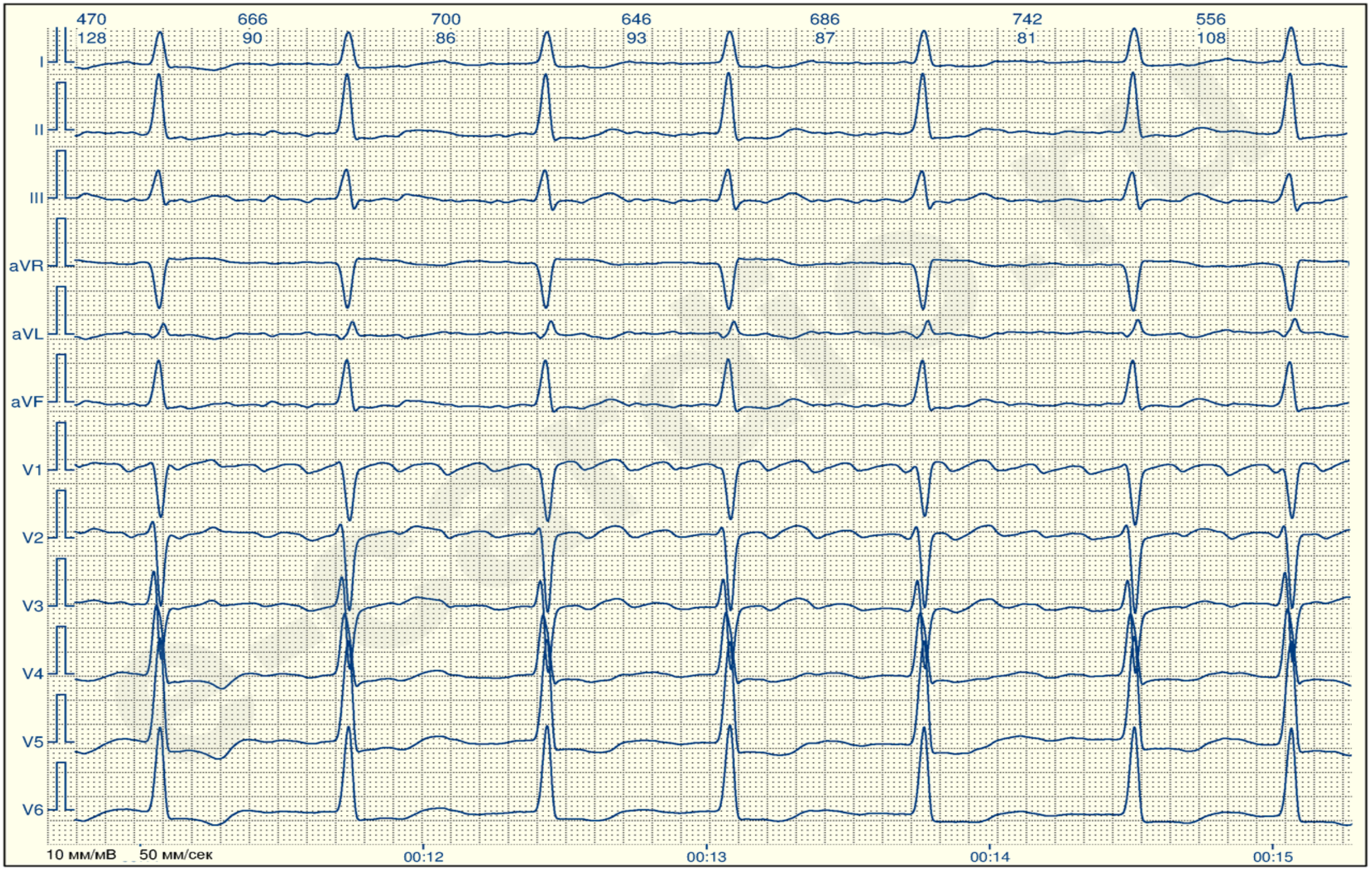


Absence of P waves in all leads (not sinus rhythm).

The appearance of frequent (200-350 per minute), regular, sawtooth-shaped "F" teeth (more often in II, III, aVF, V1, V2).

More often the correct, regular, ventricular rhythm (***equal R-R intervals***).

Unchanged ventricular QRS complexes, each of which is preceded, more often by a constant number of ***atrial waves "F"*** (2: 1, 3: 1).

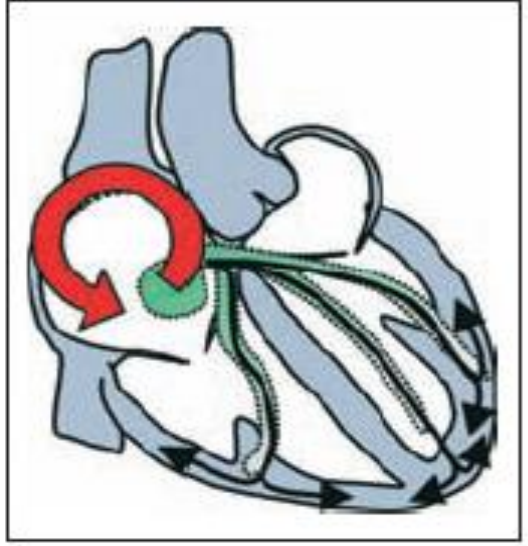

470
128
666
90
700
86
646
93
686
87
742
81
556
108
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
10 мм/мВ
50 мм/сек
00:12
00:13
00:14
00:15

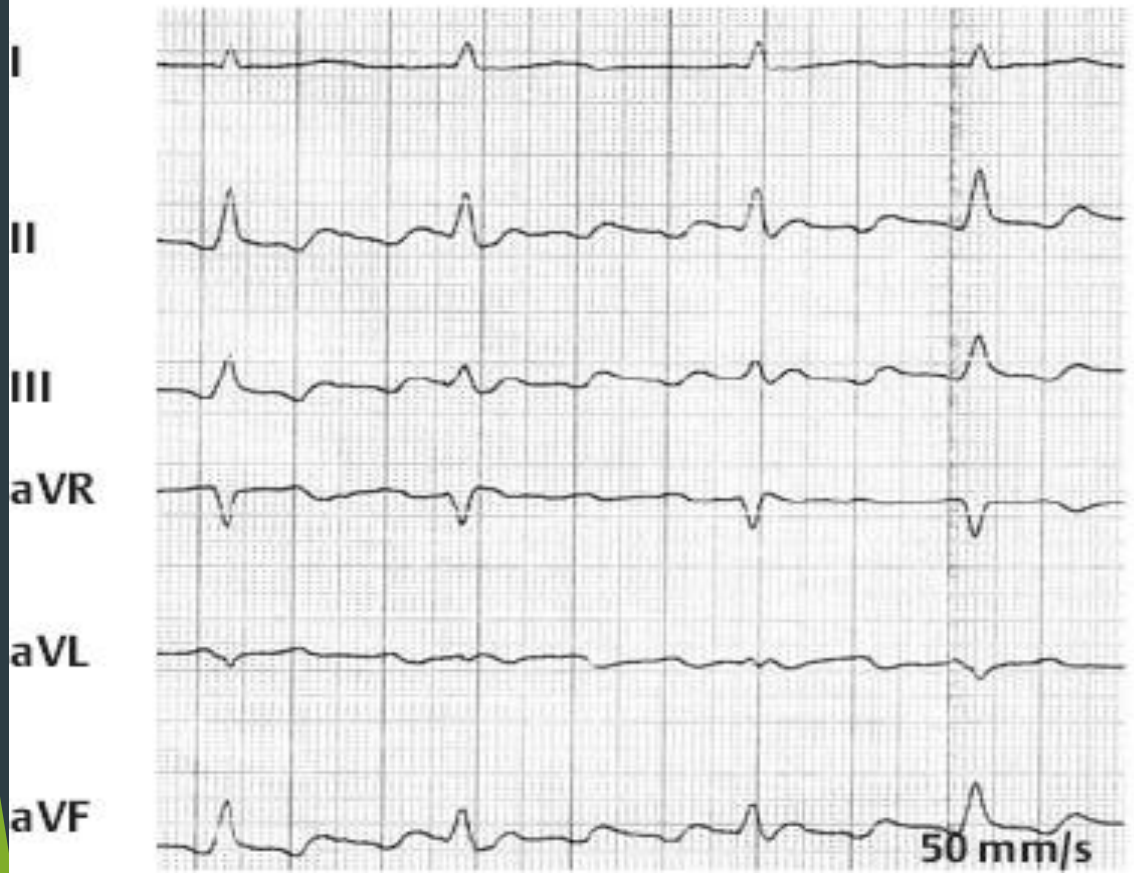
Type I atrial flutter

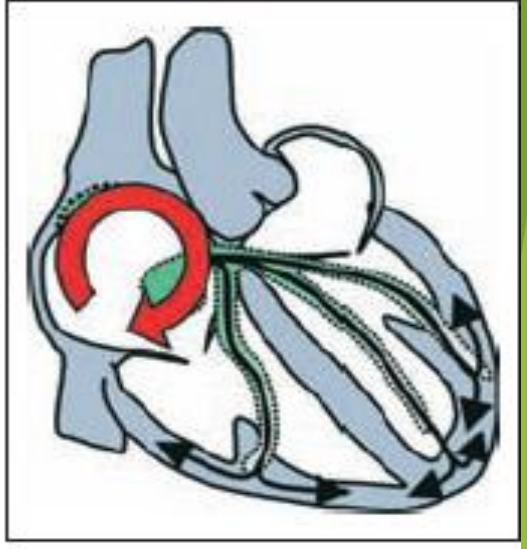
also known as common atrial flutter or typical atrial flutter, has an atrial rate of 240 to 340 beats/minute. The reentrant loop circles the right atrium, passing through the cavo-tricuspid isthmus – a body of fibrous tissue in the lower atrium between the inferior vena cava, and the tricuspid valve. Type I flutter is further divided into two subtypes, known as counterclockwise atrial flutter and clockwise atrial flutter depending on the direction of current passing through the loop.

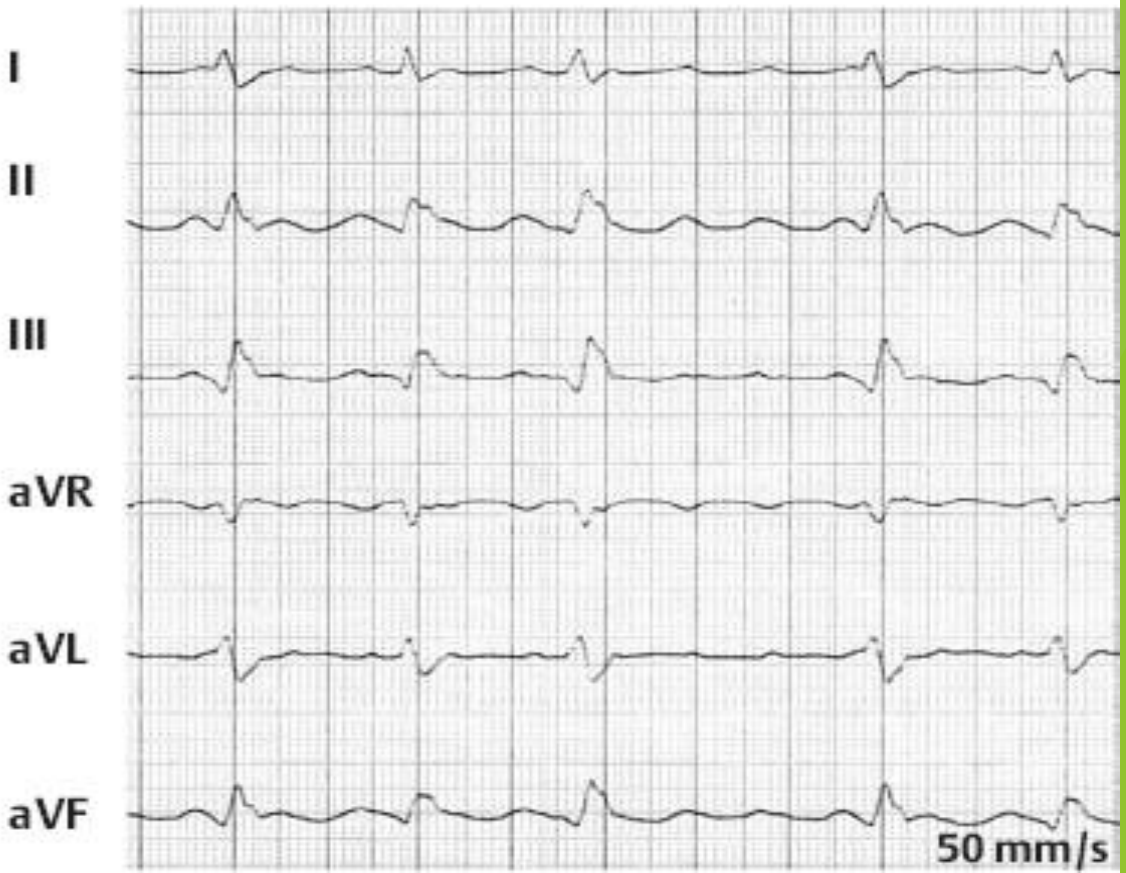
Counterclockwise atrial flutter (known as cephalad-directed atrial flutter) is more commonly seen. ***The flutter waves*** in this rhythm ***are inverted in ECG leads II, III, and aVF***.

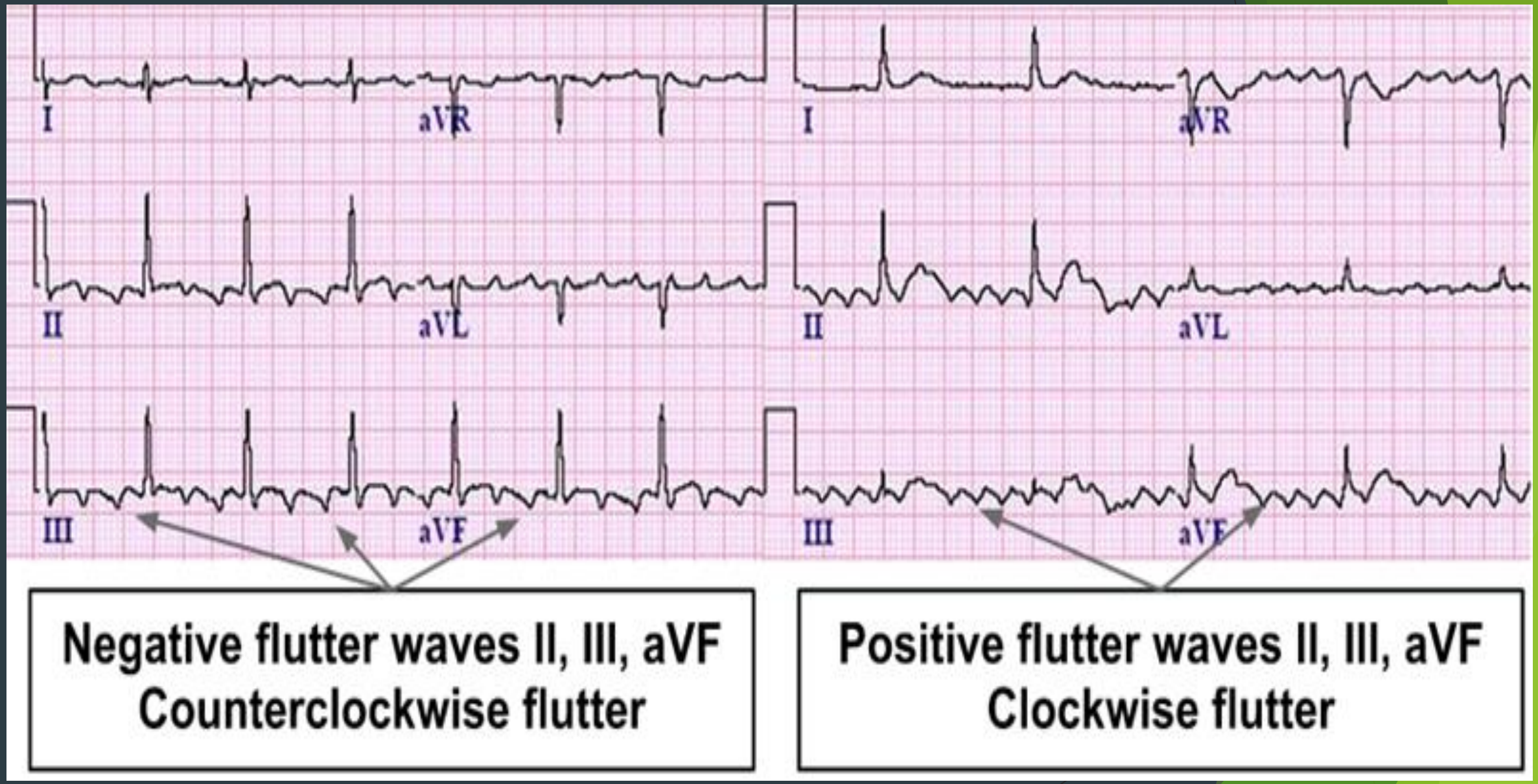
The re-entry loop cycles in the opposite direction in ***clockwise atrial flutter***, thus the ***flutter waves are upright in II, III, and aVF.***




I
II
III
aVR
aVL
aVF
50 mm/s




I
II
III
aVR
aVL
aVF
50 mm/s


I
aVR
II
aVL
III
aVF
I
aVR
II
aVL
III
aVF
Negative flutter waves II, III, aVF
Counterclockwise flutter
Positive flutter waves II, III, aVF
Clockwise flutter

Type II atrial flutter

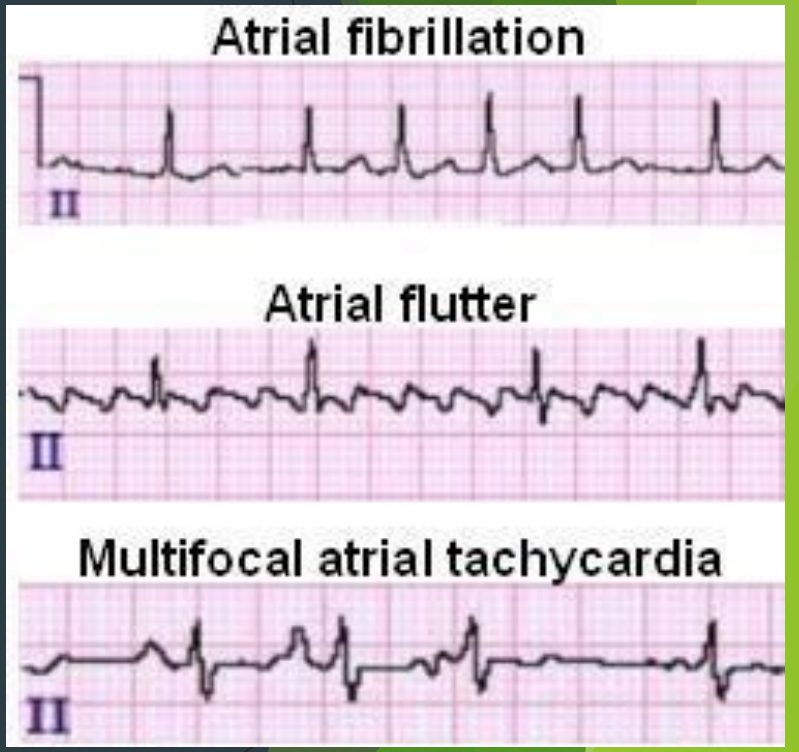
Does not fulfill criteria for typical atrial flutter.

Often associated with higher atrial rates and rhythm instability.

Less amenable to treatment with ablation.

This classification is based on the anatomical location and direction of the re-entry circuit.

Determination of the type of atrial flutter (its dependence on the cavo-tricuspid isthmus) is a prognostic factor, an important step in catheter ablation, but this does not change the primary approaches to treatment and the clinical picture.



I49 Other cardiac arrhythmias

I49.0 Ventricular fibrillation and flutter

I49.1 Atrial premature depolarization

Atrial premature beats

I49.2 Junctional premature depolarization

I49.3 Ventricular premature depolarization

I49.4 Other and unspecified premature depolarization

Ectopic beats

Extrasystoles

Extrasystolic arrhythmias

Premature

I49.0 Ventricular fibrillation and flutter

Ventricular flutter appears on the ECG as a sine wave pattern that is characterized by ***regular, large oscillations***. The rhythm may be difficult to distinguish from rapid VT and can progress to ventricular fibrillation.

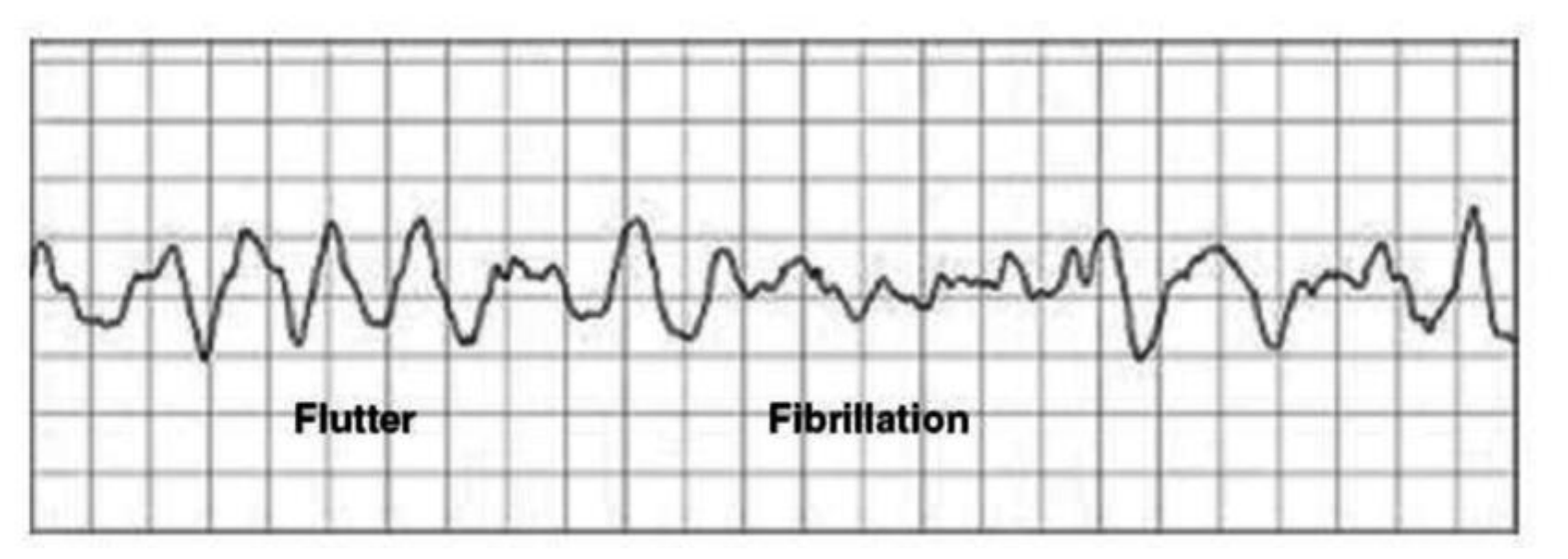
Ventricular fibrillation is a terminal arrhythmia in which ventricular contractions are uncoordinated and too weak to eject blood. The ECG shows ***irregular, chaotic deflections of varying amplitude and shape***.

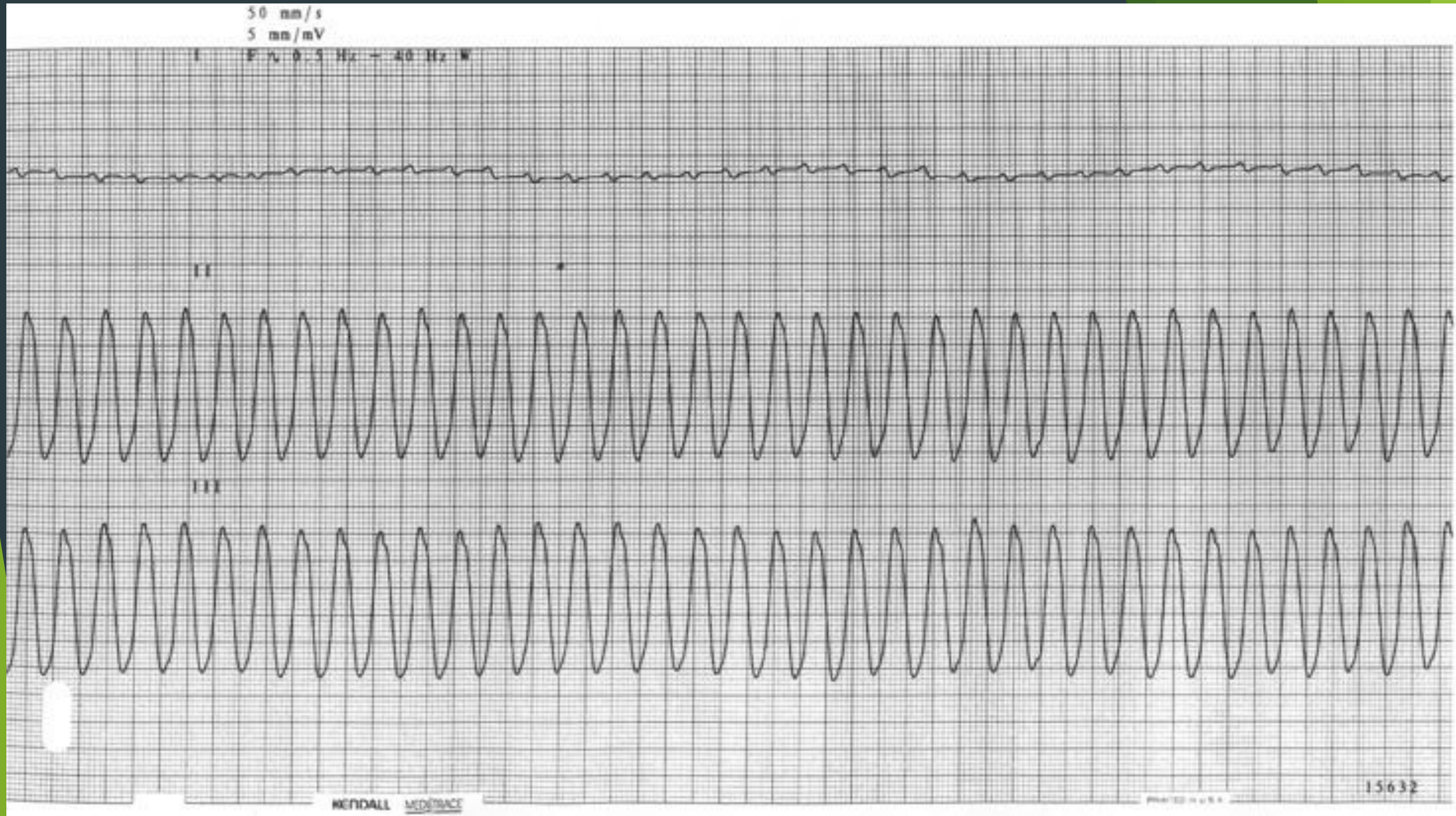
Immediate defibrillation and cardiopulmonary resuscitation are necessary. Both ventricular flutter and ventricular fibrillation generally result in loss of consciousness and usually fatal within minutes unless intervention is successful.

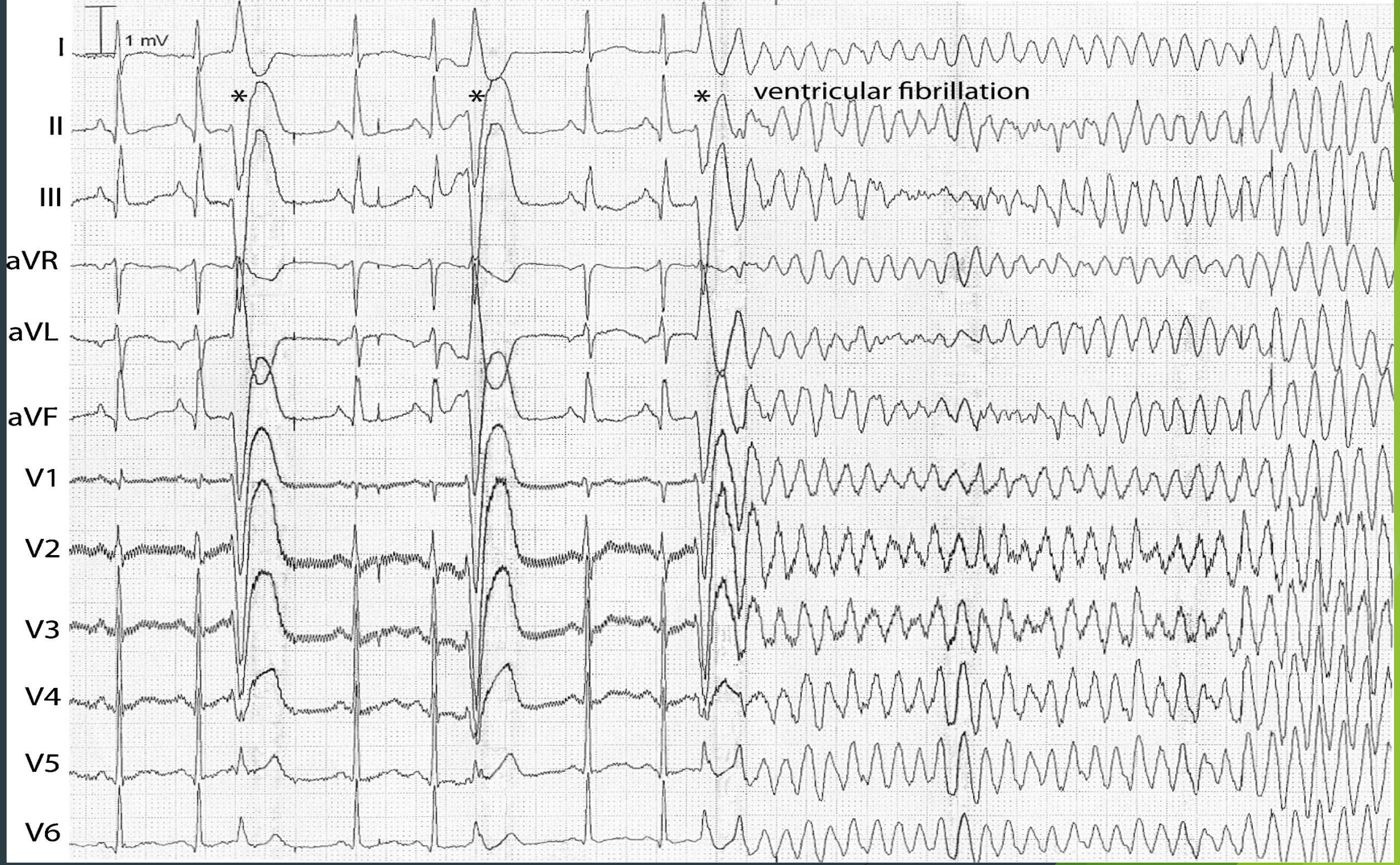
Absence of normal ventricular QRST complexes.

With ***ventricular flutter***, frequent (***200-300 per minute***) waves of the same shape and amplitude are recorded, resembling a sinusoidal curve.

With ***ventricular fibrillation***, frequent (***300-500 per minute***) irregular waves are recorded, varying in shape and amplitude.




50 mm/s
5 mm/mV
I
F ∿ 0.5 Hz – 40 Hz W
II
III
15632
KENDALL MEDITRACE


1 mV
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
ventricular fibrillation

I49.5 Sick sinus syndrome

Tachycardia-bradycardia syndrome

I49.8 Other specified cardiac arrhythmias

Brugada syndrome

Long QT syndrome

Rhythm disorder:

- coronary sinus
- ectopic
- nodal

I49.9 Cardiac arrhythmia, unspecified

Arrhythmia (cardiac) NOS

I49.5 Sick sinus syndrome (SSS)

Tachycardia-bradycardia syndrome (Short's syndrome) - inability of the sinus-atrial node (SAS) to adequately perform the function of automatism center; partial or complete loss of the central pacemaker role in the heart rhythm regulation by the SAS, that leads to the development of brady- and tachyarrhythmias: sinus bradycardia, alternation of pronounced sinus bradycardia with paroxysms of atrial tachycardia, atrial fibrillation or migration of supraventricular pacemaker. The severity of the SSS clinical course depends on the severity of the sinoatrial blockade.

Long-term recording of an ECG by the Holter method, the use of an event recorder, and sometimes electrophysiological study (usually by the transesophageal method) helps.

Persistent or episodic severe ***sinus bradycardia < 40 /min.***

Failure of the sinus-atrial node.

Second-degree sinus block (pauses > 3 s).

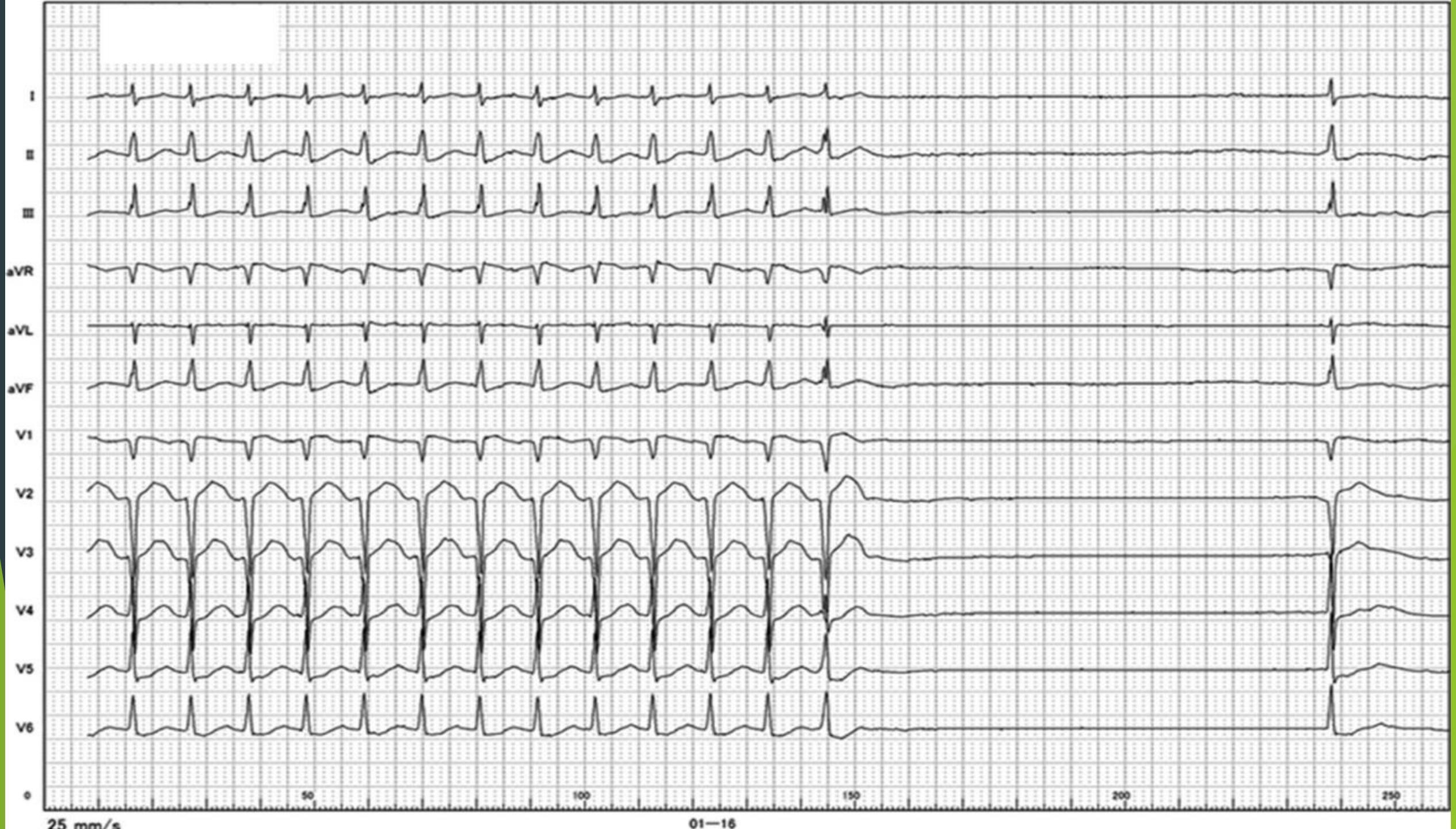
Alternating sinus bradycardia with flutter or atrial fibrillation.

Posttachycardic depression - slow recovery of sinus rhythm after cardioversion or cessation of supraventricular tachycardia (recovery time of sinus node function > 1400 ms).

Chronotropic insufficiency - inconsistency of the sinus rhythm frequency with the body needs.

The pause in the sinus rhythm is lengthened at the time of the supraventricular tachyarrhythmia termination.

Notice: AF and AT may mask sinus node dysfunction!

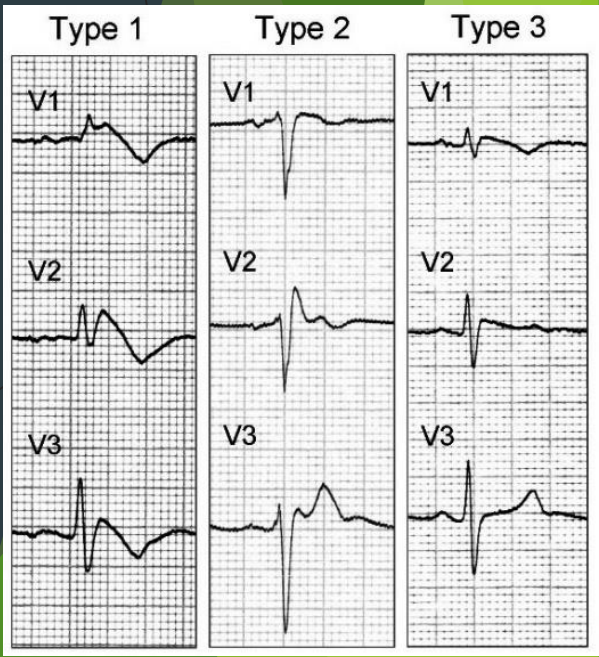

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
0
50
100
150
200
250
25 mm/s
01—16

Brugada syndrome

- hereditary disease caused by mutation of the gene, which encodes the biosynthesis of protein subunits of the sodium channel of cardiomyocytes.

The pattern seen on the ECG includes ST elevation in leads V_1-V_3 with a right bundle branch block appearance. Three forms of the Brugada ECG pattern have been described:

- Type 1 has a coved type ST elevation with at least 2 mm (0.2 mV) J-point elevation and a gradually descending ST segment followed by a negative T-wave.
- Type 2 has a saddle-back pattern with at least 2 mm J-point elevation and at least 1 mm ST elevation with a positive or biphasic T-wave. Type 2 pattern can occasionally be seen in healthy subjects.
- Type 3 has either a coved (type 1 like) or a saddle-back (type 2 like) pattern, with less than 2 mm J-point elevation and less than 1 mm ST elevation. Type 3 pattern is not rare in healthy subjects.



Long QT syndrome

is a heart rhythm condition that can potentially cause fast, chaotic heartbeats. These rapid heartbeats might trigger to suddenly faint, in some severe cases - sudden death.

Some people are born with a genetic mutation that causes long QT syndrome (congenital long QT syndrome). Long QT syndrome may be caused by certain medications, mineral imbalances or medical conditions (acquired long QT syndrome).

ECG criteria:

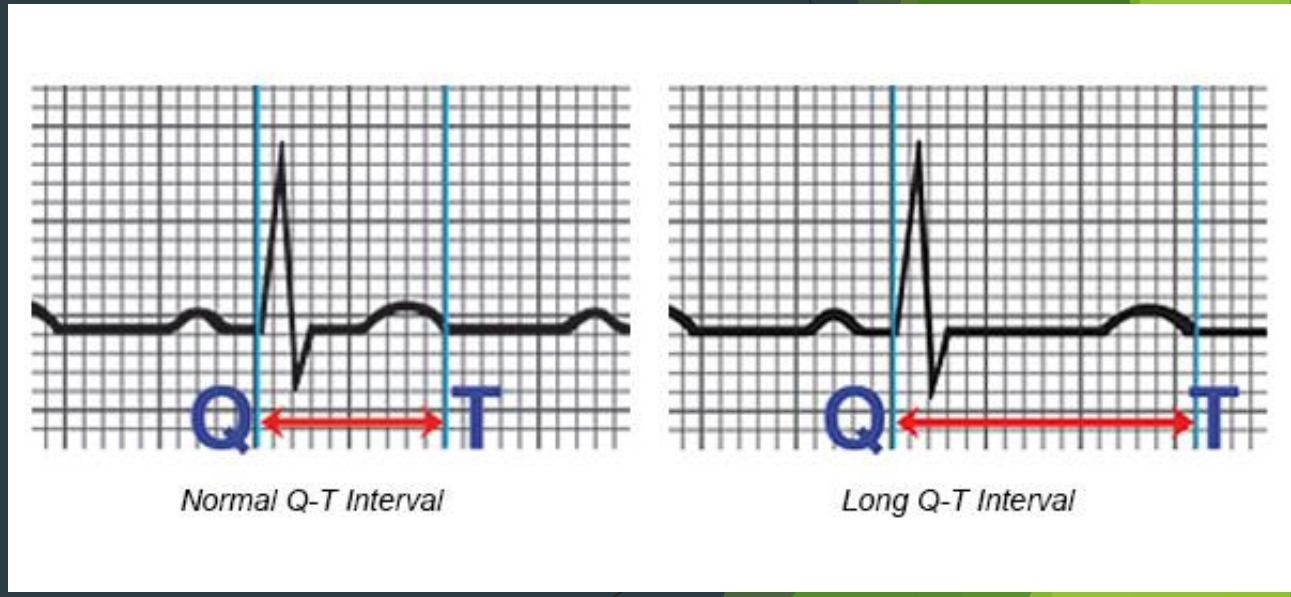
for females QTc ≥ 460 ms,

for males ≥ 450 ms

To correct the QT interval in relation to heart rate

the Bazett or Friderici formulas is applied, in case of atrium fibrillation – Sagie formula (online calculators

are available in the internet.



Thank you for attention!