

Ministry of Health of Ukraine
Kharkiv National Medical University

**ELECTROCARDIOGRAPHIC EXAMINATION OF
PATIENTS WITH AUTOMATICITY AND
EXCITABILITY FUNCTION ALTERATIONS**

Methodical instructions for students

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Electrocardiographic examination of patients with automaticity and excitability function alterations

Cardiac arrhythmias are diagnosed by noting changes in the regularity and the rate of the heart beats (Tab. 4.50). The three electrophysiologic properties of the heart are most intimately related to cardiac arrhythmias are automaticity, excitability, and conductivity.

Tab. 4.50. ECG signs of normal sinus rhythm and cardiac arrhythmias.

Normal Sinus Rhythm	Cardiac Arrhythmias
1. The SA node is pacemaker. Heart rate: 60-80 beats per minute.	1. 60>Heart rate>90 beats per 140minute.
2. Regular cardiac rhythm: the R-R intervals are approximately equal ($\pm 10\%$).	2. Irregular cardiac rhythm: the R-R intervals vary significantly ($>10\%$).
3. The P waves originate from SA node: most prominent upright R waves in leads II, III, aVF, may be inverted in aVR, or biphasic in leads V ₁ , V ₂ .	3. The P waves originate from a site outside SA node: changes of the P waves configuration (inverted, biphasic \pm).
4. The relationship of the P wave and the QRS complex: all P waves are followed by QRS complex.	4. Changes of the relationship of the P wave and QRS complex.
5. The conduction to the ventricles is 1:1 (equal amount of P waves and QRS complexes).	5. The P waves amount are larger than QRS complexes.
6. Constant and normal duration of P wave, P-Q interval, QRS complex.	6. Changed the P wave, P-Q interval, and QRS complex duration.

Cardiac Arrhythmias

- I. Abnormalities of the Impulse Formation
 - A. *Altered Automaticity of the Sinoatrial Node (Nomotopic Arrhythmias).*
 1. Sinus Tachycardia
 2. Sinus Bradycardia
 3. Sinus Arrhythmia

4. Sick Sinus Syndrome
- B. *Increased Automaticity of an Ectopic Pacemaker*
 1. Atrial Rhythm
 2. Junctional (AV) Rhythm
 3. Ventricular or Idioventricular Rhythm
 4. Wandering pacemaker
- C. *Ectopic (heterotopic) Arrhythmias caused by increased excitability of the myocardium*
 1. Premature heart beat (contractions)
 2. Paroxysmal Tachycardias
 3. Atrial and Ventricular Flutter and Fibrillation.
- II. Abnormalities of Conduction
 1. Sinoatrial Block
 2. Atrioventricular Block
 3. Intraventricular Blocks (His Bundle-Branch Blocks)
 4. Ventricular Preexcitation Syndromes:
 - Wolff-Parkinson-White syndrome (WPW syndrome)
 - Shorted P-Q interval syndrome or Clerk-Levy-Critesco syndrome (CLC syndrome)

Abnormalities of the impulse formation

Altered automaticity of the sinoatrial node (nomotopic arrhythmias).

Sinus Tachycardia

In the sinus tachycardia in the adults, impulses are initiated in the SA node at a rate from 90 to 180 beats per minute (Fig. 4.76). Sinus tachycardia even at a very high rates is gradual, not sudden, in onset; if followed over a sufficient time, it will exhibit changes in rate of more than 10 beats per minute. It may be seen in physical and emotional exertion, during meals, in alcohol, smoking, coffee, and tee abuse. Pathological conditions such as fever, sepsis, electrolyte disturbances, hemorrhage, shock, hyperthyroidism, cardiovascular diseases (myocarditis, heart valvular diseases, heart failure), and chronic pulmonary diseases as well as drugs such as atropine, epinephrine, and isoproterenol can produce sinus tachycardia. Clinical symptoms of sinus tachycardia are heart

palpitation and accelerated pulse.

Diagnostic ECG signs of sinus tachycardia

1. Decreased duration of the R-R intervals, increased heart rate to 90-180 per minute.
2. Regular sinus rhythm: the P waves are easily seen, upright in the leads I, II, aVF, V₄, V₅, V₆. The conduction to the ventricles is 1:1, the P-P and R-R intervals are constant.
3. The P-Q interval is 0.12 second or more.



4. The QRS complexes are normal.

Fig. 4.76. Sinus tachycardia with rate of 116 beats per minute.

Sinus Bradycardia

Sinus bradycardia is rhythm in which the ventricular rate customarily is less than 60 beats per minute (Fig. 4.77).

Sinus bradycardia is commonly seen in athletes, particularly marathon runners (HR accelerates during exercise), during night sleep, in stoppage of the breathing, and often in the aged. It may also occur with acute myocardial infarction, hypothyroidism, increased intracranial pressure, toxicosis, and with administration of drugs (digitalis, cardiac glycosides, β -adrenoblockers). Extreme sinus bradycardia may be caused by increased parasympathetic tone of any cause. Clinical signs of significant bradycardia (less than 40 beats per minute) are nausea, dizziness, syncope due to cerebral ischemia, and slow pulse rate (p. rarus, p. bradus).

Diagnostic ECG signs of sinus bradycardia

The impulse is initiated in the sinus node and spreads in normal sequence through the conducting system of the ventricles.

1. Increased duration of the R-R intervals, slow heart rate less than 60 beats per minute.
2. Regular sinus rhythm: the P waves are easily seen, upright in the leads I, II, aVF, V₄, V₅, V₆. The conduction to the ventricles is 1:1, the P-P and R-R intervals are constant.
3. The P-Q interval is between 0.12 and 0.20 second, although at very slow rates it may be slightly longer.
4. The QRS complexes are normal.

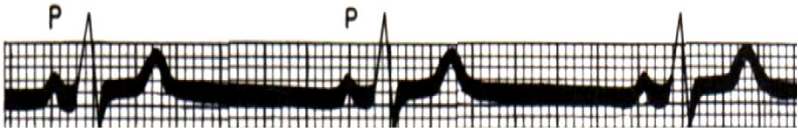


Fig. 4.77. Sinus bradycardia with a rate of 43 beats per minute.

Sinus Arrhythmia

In sinus arrhythmia, the impulse is initiated in the sinus node, but the rate varies with respiration. During inspiration the rate increases; during expiration the rate slows (Fig. 4 78).

Sinus (respiratory or juvenile) arrhythmia may be seen in children and adolescents. It may be caused by irregular impulse discharge from SA node and (or) changes of heart filling by blood during respiration.

Pathological conditions such as neurocirculatory dystonia by cardiac type, rheumocarditis as well as some infectious diseases can produce sinus arrhythmia. There are no clinical signs of sinus arrhythmia.

Diagnostic ECG signs of sinus arrhythmia

1. The P-P and R-R intervals vary significantly and rhythmically in such manner that the R-R intervals gradually shorten and lengthen with the respiratory cycles. The R-R intervals length may vary by 0.16 second or more.
2. Regular sinus rhythm: the P waves are upright in the leads I, II, aVF, V₄, V₅, V₆. The conduction to the ventricles is 1:1.
3. The P-Q intervals are constant.
2. The QRS complexes are normal.

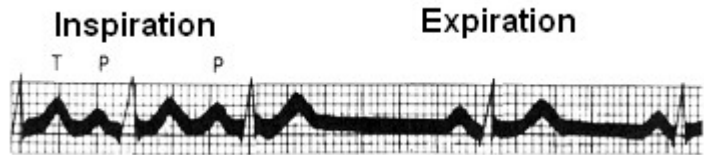


Fig. 4.78. Sinus arrhythmia, showing acceleration of heart rate with inspiration, and slowing with expiration.

Sick Sinus Syndrome

The term “Sick sinus syndrome” described in 1960 by American cardiologist Town, attributes the symptoms to the slow rate resulting from failure of impulse formation in the SA node or its conduction to the AV node.

Sick sinus syndrome includes a number of arrhythmias such as SA block with or without an ectopic or “escape” rhythms, marked bradycardia, atrial fibrillation, and attacks of tachycardia (bradycardia-tachycardia syndrome) (Fig. 4.79).

Such diseases as coronary heart disease, atherosclerosis, myocarditis, cardiomyopathy, cardiac or renal amyloidosis, diabetes mellitus, and toxicosis can caused sick sinus syndrome.

Clinical symptoms may be due to a very slow (dizziness, syncope) or a very rapid heartbeat (heart palpitation).

Diagnostic ECG signs of sick sinus syndrome

1. Constant bradycardia.
2. Periodic ectopic rhythms.
3. SA block.
4. Bradycardia-tachycardia syndrome.

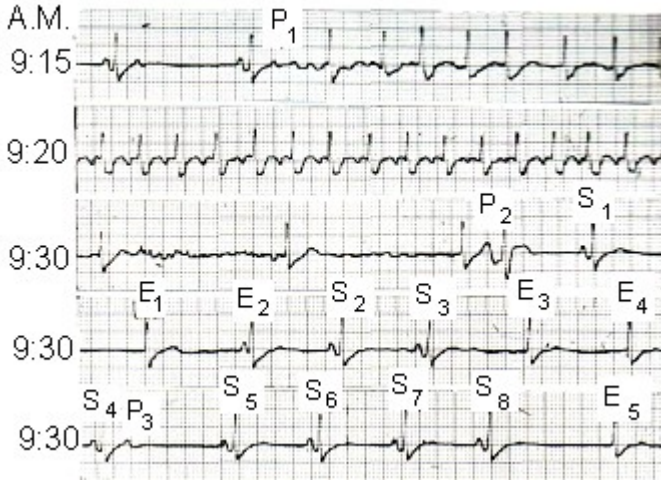


Fig. 4.79. Sick sinus syndrome (monitor leads).

Note particularly, the alternating tachycardia and bradycardia, which occurred spontaneously. In the top strip, taken at 9:15, sudden onset of atrial fibrillation occurred when the ectopic atrial beat (P_1) appeared. In the second strip, taken at 9:20, the rhythm is atrial flutter with 2:1 block. In the following three continuous strips, taken at 9:30, the rhythm is initially atrial fibrillation with very slow escape rhythm, probably lower junctional in origin; succeeded by a conducted atrial premature contraction (P_2), a sinus beat (S_1), then two escape beats (E_1 and E_2);

two sinus beats (E_3 , E_4) followed by a sinus beat (S_4), a blocked atrial premature contraction (P_3), since the expected escape beat does not occur on time; the last sinus beats (S_5 through S_8) with an escape beat (E_5) that occurred at the expected time.

Increased Automaticity of an Ectopic Pacemaker

Arrhythmias occurring from an ectopic pacemaker may be active or passive.

Active arrhythmias develop because of increased automaticity of an ectopic pacemaker.

Passive arrhythmias occur because an established pacemaker fails to discharge at its usual rate, allowing a slow subsidiary pacemaker to emerge. Passive rhythms protect the heart from a long asystole period.

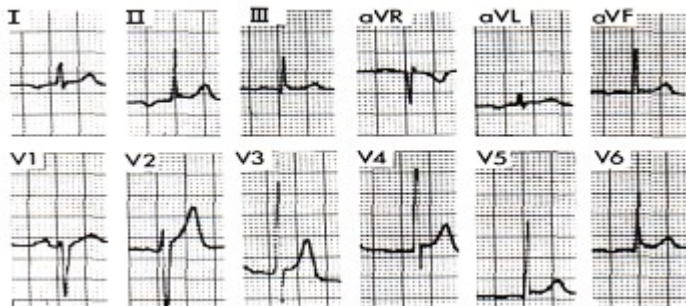
Atrial, junctional and ventricular rhythms are distinguished.

Atrial Rhythm

In atrial rhythm the pacemaker lies within the atria resulting in abnormal direction of their depolarization. The direction of ventricular activation is normal (Fig. 4.80).

Diagnostic ECG signs of the atrial rhythm

1. Heart rate is from 60 to 90 beats per minute.
2. Inverted P wave in leads II, III, aVF within an upright P wave in lead aVR or biphasic P wave depend on the site of the pacemaker.
3. The P-Q interval is normal.



4.

The QRS configuration is normal.

Fig. 4.80. Left atrial rhythm.

Note the negative P waves in leads I and V₆ and the “dome and dart” P waves in V₁. The dome-and-dart P wave is characterized by an initial smooth, slowly rising positive wave punctuated by high amplitude, spiked, terminal positive component.

Junctional (AV) Rhythm

A junctional rhythm is found in acute rheumatic carditis, acute myocardial infarction, and with digitalis intoxication.

If the junctional pacemaker depolarized the atria in retrograde fashion, an inverted P wave may be seen. The QRS complex occurs after a longer pause than normal. The QRS complex is usually normal, although it may show slight aberration (Fig. 4.81).

Diagnostic ECG signs of junctional rhythm

1. Heart rate is 40-60 beats per minute.
2. An inverted P wave in leads II, III, and aVF may be seen just preceding or following the QRS complexes; sometimes no P wave is seen.
3. The P-Q interval duration is more than 0.2 second.
4. The QRS complex is usually normal.



Fig. 4.81. Junctional rhythm.

The rhythm is regular at 58 beats per minute. No P waves are visible.

Ventricular or Idioventricular Rhythm

Occasionally, the sinus pacemaker is suppressed, and the junctional pacemaker fails to respond. Under these circumstances, an idioventricular pacemaker may discharge to maintain cardiac function. When such beats occur in succession, an idioventricular rhythm is initiated.

Idioventricular rhythm most commonly observes in complete AV

block.

The course of ventricular activation is abnormal, and QRS complex is therefore wide and of abnormal configuration. The sinus P wave may be seen, but it has no relationship to the QRS complex.

Diagnostic ECG signs of ventricular rhythm

1. Heart rate is less than 40 beats per minute.
2. The sinus P wave or its absence. There is no relationship of T wave to the QRS complex.
3. Wide and deformed QRS complex.

Wandering Pacemaker

The term wandering pacemaker refers to conditions where in the primary pacemaker of the heart is presumed to move from site to site in the atrium. This may vary from areas near the SA node to areas near AV node. The rhythm is only slightly irregular and the rate is essentially unchanged. The P wave morphology changes from beat to beat, varying from peaked to flat to slightly inverted (Fig. 4.82).

Diagnostic ECG signs of the wandering pacemaker

1. The P wave configuration changes from cycle to cycle.
2. Changes of the P-Q interval duration depend on the site of pacemaker.
3. The cardiac rhythm is slightly irregular, different R-R intervals.
4. The QRS complex morphology is normal.



Fig. 4.82. Wandering atrial pacemaker.

A, note the different contour of the P waves (arrows) with constant P-Q intervals;

B, note the varying P-Q intervals with changing contour of the P waves. The 5th arrow points to a P wave that is not visible because the QRS complex is superimposed on it.

Ectopic Arrhythmias Caused by Increased Excitability of the Myocardium.

Ectopic arrhythmias may be due to increased automaticity of an ectopic pacemaker or reentry mechanism. Reentry or circus mechanisms occur when an impulse returns to its site of origin and reenters the conduction pathway in its nonrefractory state (Fig. 4.83). For reentry to occur, an impulse must enter an area of myocardium and divide into two branches. One branch must be in its refractory state. The impulse cannot pass through this refractory branch; however, through circuitous paths, the impulse returns to its point of origin and reenters the same nonrefractory pathway. Reentry requires a slight prolongation of the refractory period in some area of the heart, so the impulse is blocked in one direction but allowed to gain access from another direction.

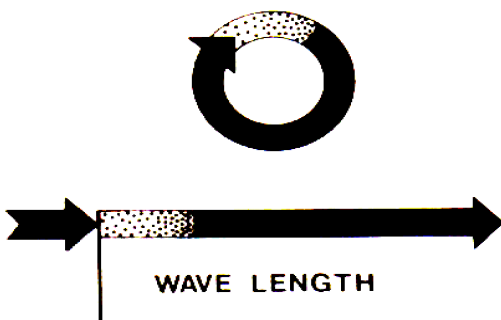


Fig. 4.83. Schematic drawing of a reentrant circuit.

The black area represents tissue, which is in its unexcitable phase, whereas the dotted area indicates relatively refractory tissue. No excitable gap exists between head and tail of the excitation wave.

An ectopic impulse may arise in the atria, junctional tissue, or ventricles. Junctional premature contractions are the least common; atrial and ventricular premature beats are quite common.

Premature heart contractions can observe in normal individuals: emotional exertion, in heavy smokers, in coffee, strong tee, and alcohol abuse. Coronary heart disease (most commonly acute myocardial infarction), hypertension, rheumatic heart valvular diseases, myocarditis, congestive heart failure cause premature contractions as well as by reflex in the diseases of the abdominal organs premature betas can arise. Premature heartbeats can also occur in hyperthyroidism, menopause, and digitalis intoxication.

Clinical signs: Patients with premature cardiac contractions can feel their heart missing a beat (escape beat) and subsequent strong stroke. In auscultation premature beat with a specific loud first sound is heard. Study of the arterial pulse reveals premature weak pulse wave following by long pause. If premature beat is close to the normal one the pulse wave does not reach radial artery causing pulse deficit.

Premature Atrial Contraction

Atrial premature contractions (APCs) may arise from either atrium. A P wave is always present and occurs earlier than the normal sinus P wave. The premature P wave varies in configuration depending on its site of origin or reentrant mechanism. If the impulse originates in or near the SA node and depolarization follows the usual course, the P wave will resemble the normal P wave. If the ectopic atrial site is near the AV node, atrial activation will occur retrogradely and will be directed toward the SA node, giving rise to an inverted P wave (Fig. 4.84).



Fig. 4.84. Atrial premature contraction (A).

The P wave (A) occurs early, it is of different configuration; the QRS-T wave following it is normal appearance, and the compensatory pause is incomplete.

After an APC, there is a pause before the next sinus beat. This is usually not a full compensatory pause (Fig. 4.85).

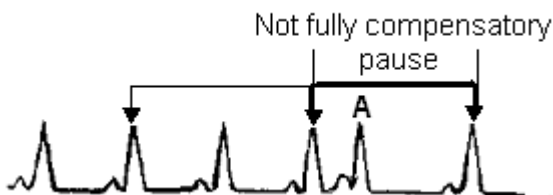


Fig. 4.85. Mechanism of compensatory pause following atrial extrasystole.

The atrial premature contraction not only activates the ventricles but also is able to spread retrograde direction and to discharge the sinus node prematurely. The premature discharge of the sinus pacemaker causes transient depression of its rhythmicity so that the sinus cycle immediately following the APC is slightly longer than the usual sinus cycle. However, the lengthening of the postectopic sinus cycle is usually less in amount than the preceding cycle is shortened by the atrial premature beat. Consequently, the interval between the last R wave preceding the APC and the first R wave following the extrasystole is less

than two cycle lengths of the sinus rhythm – in other words, the postectopic pause is not fully compensatory.

ECG signs of atrial premature contractions

1. Premature appearance of the P wave following by the QRS complex.
2. Changes of the P wave polarity depend on the site of the ectopic focus.
3. Normal configuration of the QRST complex.
4. Presence of the not fully compensatory pause after the atrial premature contraction.

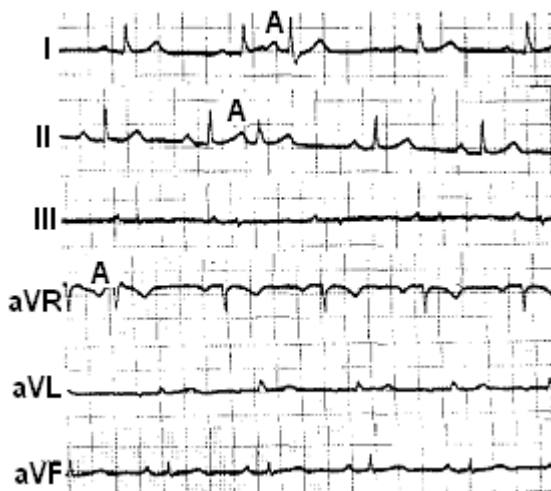


Fig. 4.86. Premature atrial contractions (A).

Junctional Premature Contractions.

In junctional premature contraction the pacemaker is in the AV node. The P wave may or not may be identified. The QRS complex has the same morphology as the QRS complex of the normal sinus complex in the same lead because ventricles are activated by usual pathways. When there is retrograde conduction into the atria, the inverted P waves are present, which occur before, after, or within the QRS complex. If it occurs simultaneously with the QRS complex, it may be buried in the QRS complex and not visible, although it may deform the complex somewhat. The atrial impulse is normally delayed in the junctional tissues, a delay, which accounts for most of the duration of the P-Q interval. If no delay in retrograde or antegrade conduction occurs, then the ectopic focus in the junctional tissue is located above the site of delay, and the inverted P waves will precede the QRS complex (formerly called an upper nodal extrasystole). If the ectopic focus is below the site of junctional delay, retrograde atrial activation will be delayed and the P wave will coincide with the QRS complex (formerly called a midnodal extrasystole). The P wave may follow the QRS, (formerly called a lower nodal extrasystole) (Fig. 4.87).

ECG signs of the junctional premature contractions.

1. Premature appearance of the normal QRS complex.
2. Inverted P wave before, after, or within the QRS complex.
3. Presence of not fully compensatory pause.

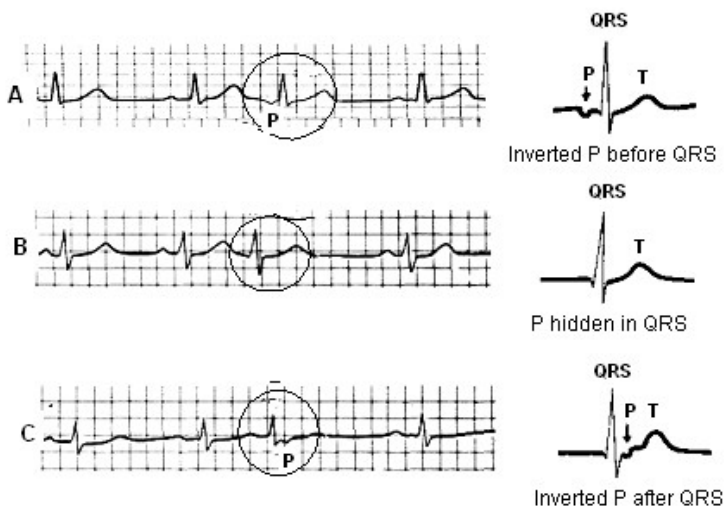


Fig. 4.87. Junctional premature contractions.

A – an upper nodal extrasystole; the P wave is inverted before QRS complex, the QRS complex resembles the others, and the compensatory pause is incomplete.

B – a midnodal extrasystole; the P wave cannot be identified, the QRS complex resembles the others, and the compensatory pause is incomplete.

C – a lower nodal extrasystole; the P wave is inverted after QRS complex, the QRS complex resembles the others, and the compensatory pause is incomplete.

Ventricular Premature Contractions.

As the name denotes, premature ventricular contractions (PVCs) originate in the ventricles below the AV node. Because the PVCs do not follow the normal conduction path in the ventricles, they show a bizarre QRS configuration on the ECG. It is characterized by being wide, slurred, and notched. An ectopic impulse arising near the bundle of His may produce a relatively normal-appearing QRS. The QRS complexes of PVCs are not preceded by P wave because impulse cannot pass retrogradely through the AV node to cause atrial activation. The T wave

is usually opposite in direction to the main deflection of the QRS. Usually the sinus node maintains control of the atria and sinus-initiated P wave may occur in the T wave of the premature ventricular beat. Since the ventricle is refractory at this time, there is no conduction to the ventricle from this sinus impulse, and a compensatory pause occurs. Since there has been no premature discharge of the sinus node and the sinus rhythm is undisturbed, the interval between the normally conducted beats that surround the premature ventricular beat is exactly twice the normal R-R interval. This is termed a fully compensatory pause (Fig. 4.88).

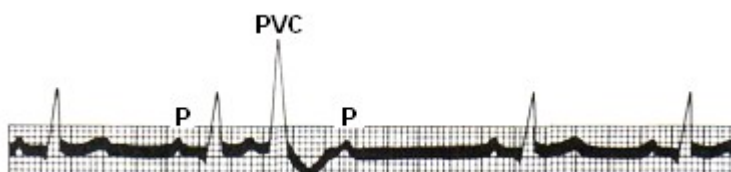


Fig. 4.88. Ventricular premature contraction (PVC).

The QRS-T complex has a bizarre shape, being wide and slurred; the P wave following occurs at the proper interval after the preceding P wave; the compensatory pause is complete.

Occasionally the atrial depolarization occurring during the ventricular premature contraction does not succeed in traversing the AV node and succeeds in the depolarizing the ventricle. Under these circumstances, there is no compensatory pause. The PVC does not interrupt the sinus rhythm. This type of PVC is called interpolated PVC (Fig. 4.89).

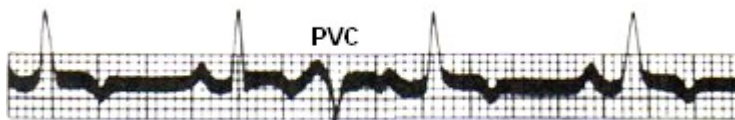


Fig. 4.89. Interpolated premature ventricular contraction (PVC),
occurring between two normal beats without compensatory pause.

If the ectopic impulse originates in the right ventricle, the impulse spreads slowly from the right ventricle to the left, producing high amplitude R wave in leads I, aVL, V₄, V₅, V₆ and deep S wave in leads III, aVF, V₁, V₂, V₃. An impulse originating in the left ventricle spreads slowly to the right ventricle to produce high amplitude R wave in leads III, aVF, V₁, V₂, V₃ and deep S wave in leads I, aVL, V₄, V₅, V₆.

ECG signs of PVCs.

1. Premature appearance of significantly wide and distorted QRS complex.
2. The S-T segment and T wave of PVC opposite in direction to the main deflection of the QRS complex.
3. The QRS complexes of PVCs are not preceded by a P wave.
4. Presence of the full compensatory pause after PVCs.

PVCs from the same site have the same configuration in the same lead, and is called – **unifocal PVCs** (Fig. 4.90).

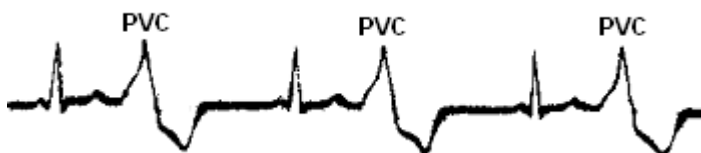


Fig. 4.90. Unifocal premature ventricular contractions, bigeminy is present. Note the same configuration of PVCs.

PVCs that originate from the different site have different

configuration when recorded in the same lead, and is called – ***multifocal PVCs*** (Fig. 4.91).



Fig. 4.91. Multifocal premature ventricular contractions (V).

Continuous strip of lead II show premature beats from multiple foci. Note also multifocal atrial beats (A).

Ventricular ***bigeminy*** occurs when PVCs alternate with normal contractions (Fig. 4.90).

Trigeminy is a term applied to a grouping of the PVCs in runs of three, such as two normal beats followed by a PVC (Fig. 4.92), or a normal beat is followed by two PVCs (Fig. 4.93). I

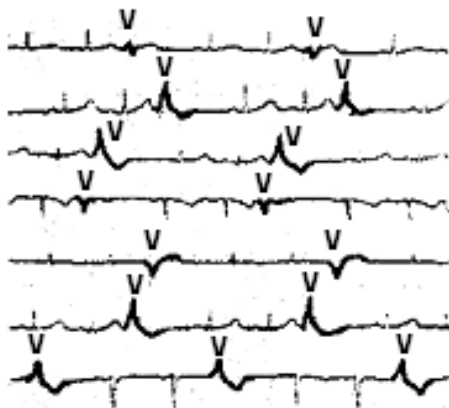


Fig. 4.92. Ventricular trigeminy.

Premature ventricular contractions (V) from a single focus occurring regularly after two normal beats.

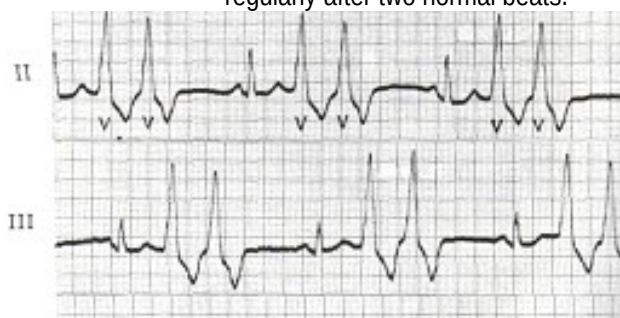


Fig. 4.93. Ventricular trigeminy.

Sinus rhythm with frequent ventricular premature contractions, producing a trigeminal rhythm consisting of one sinus beat followed by two consecutive ventricular premature beats.

Group PVCs occur in groups of two or three.

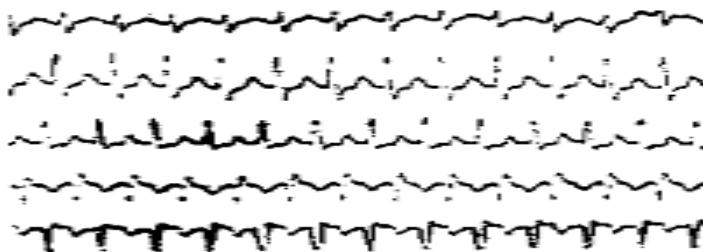
Premature ventricular contractions are especially dangerous when they:

1. Occur more frequently than one in 10 beats.
2. Occur in groups of two or more.
3. Are multifocal: Several ventricular sites are irritable.
4. Occur on or near the T wave. At this time in the cycle, the conduction tissue is partially repolarized and may respond in an erratic manner. Some cells respond immediately and other later, causing intermittent depolarization and triggering ventricular fibrillation.

Paroxysmal Tachycardias

Depend on ectopic site location atrial, junctional, and ventricular paroxysmal tachycardias are distinguished. Conventionally, however, paroxysmal atrial and junctional tachycardias refer to supraventricular paroxysmal tachycardias.

Supraventricular paroxysmal tachycardia



Supraventricular paroxysmal tachycardia is an arrhythmia characterized by abrupt onset and cessation. The ectopic impulse is initiated in either atrium or junctional tissue, and the rate of discharge is usually 140 to 220 beats per minute. The ventricular response is 1:1, with normal-appearing QRS complex. When the impulse arises in the atria, the QRS complex is preceded by abnormal P wave and the rhythm is termed paroxysmal atrial tachycardia. When the ectopic focus or reentry site lies in the junctional tissue, the QRS complex may precede, follow, or occur simultaneously with the P waves. The P waves, when seen, are produced by retrograde conduction, and are usually inverted. This rhythm is called paroxysmal AV or junctional tachycardia. In many cases of paroxysmal atrial or junctional tachycardia, the P waves are not well seen, and the general term supraventricular is used (Fig 4.94).

Fig. 4.94. Supraventricular tachycardia with a rate of 190 beats per minute. The P and T waves are superimposed.

ECG signs of paroxysmal supraventricular tachycardia.

1. Sudden acceleration of heart rate 140 to 220 beats per minute.
2. The QRS complexes configuration is normal.
3. Inverted P wave before, after, or simultaneously with QRS complex.

Paroxysmal Ventricular Tachycardia

Ventricular tachycardia is a cardiac arrhythmia originating in the ventricles at a rate of 100 beats per minute or faster. A minimum of three consecutive beats is required to establish this diagnosis. Paroxysmal ventricular tachycardia starts and stops abruptly; it is usually regular rhythm, but may show slight irregularities of ventricular response (Fig. 4.95).

ECG signs of paroxysmal ventricular tachycardia.

1. Abrupt starts and stops of the heart rate acceleration 100 to 220 beats per minute in regular rhythm.
2. Deformation and broadening of the QRS complex with opposite to the main QRS deflection S-T segment and T wave.
3. AV dissociation (independent atrial and ventricular rhythms).



Fig. 4.95. Paroxysmal ventricular tachycardia, ventricular rate 107 beats per minute.

Ventricular Flutter and Fibrillation.

Ventricular flutter is characterized by fairly regular, oscillating waves of large amplitude with no isoelectric interval, and a rate between 200 – 300 cycles per minute. In **ventricular fibrillation**, electrical and mechanical activity of the heart is totally disorganized. There is no cardiac output; clinical cardiac arrest is present. The ECG is bizarre, with complete absence of the characteristic P, QRS, and T waves; it shows irregular waves of varying amplitude and shape occurring 250 to 500 times per minute (Fig. 4.96). Both rhythms usually result in death unless they are quickly converted by drugs or electrical defibrillation.

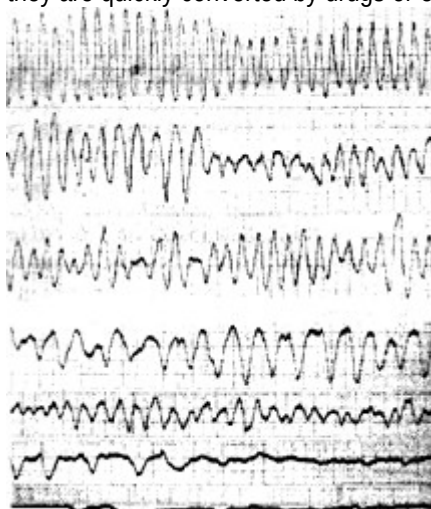


Fig. 4.96. Dying heart.

Continuous strip of lead II. Note onset in top lead episode of ventricular flutter with regular oscillatory waves (no isoelectric baseline or T waves identified) changing to irregular undulating waves characteristic of ventricular fibrillation, ending with a straight line.

Atrial Flutter

Atrial flutter may result from a rapid series of impulses arising from a single ectopic focus, a series of impulses from multiple ectopic atrial foci, or a circus movement. The atrial rate is usually between 240 and 350 beats per minute.

The ECG in atrial flutter is usually characterized by a uniformly appearing series of flutter waves (F waves), often described as resembling a picket fence, or as having a saw-toothed appearance. These waves are usually best seen in leads II, III, aVF, and V₁. There is a sharp upstroke with a more gradual down-stroke, and no isoelectric interval exists between the waves (Fig. 4.97).

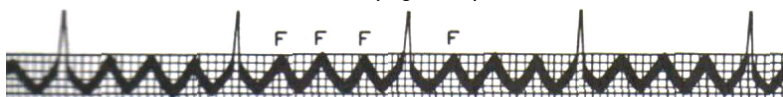


Fig. 4.97. Atrial flutter with 3:1 conduction.

The atrial rate is 300 beats per minute and the ventricular rate is 75 beats per minute. Note the saw-toothed baseline due to the flutter waves.

At rest, the AV node will not allow more than 150-180 impulses per minute to pass. Therefore, when atrial flutter is present in its untreated state, the conduction to the ventricles is blocked (2:1, 3:1, 4:1 etc).

ECG signs of atrial flutter.

1. Presence of rapid (150-180 per minute) regular, uniform atrial F waves, which are best seen in leads II, III, aVF, V₁.

2. Regular ventricular rhythm with equal R-R intervals.
3. The QRS complex is usually normal in configuration.
4. The conduction to the ventricles is 2:1 or 3:1, or 4:1 etc.

Atrial Fibrillation

In atrial fibrillation the atria are beating very rapidly and irregularly. The rate is between 350 and 700 beats per minute. Most of these impulses are blocked in the AV node, so the ventricular rate is much slower, but totally irregular. Many impulses reach the junctional tissue while it is still refractory from preceding impulses. Actually, some impulses may not reach the junctional tissue at all, and the impulses that do arrive and conduct to the ventricle do so quite irregularly, giving rise to rapid and very irregular ventricular response. Usually the ventricular rate is between 110 and 160. There are no definite P waves; there are, instead, continuous irregular undulations of the baseline of varying amplitude, spacing, and contour. These are known as fibrillation waves (f waves). The QRS complexes have relatively normal configuration (Fig. 4.98).



Fig. 4.98. Atrial fibrillation with an irregular ventricular beat. Note that the normal contractions of the atria replaced by irregular oscillations.

ECG signs of atrial fibrillation.

1. Instead P wave irregular undulations of varying shape and amplitude (f waves), which are best seen in leads II, III, aVF, V₁.
2. Irregular ventricular rhythm with different R-R intervals.
3. The QRS complexes have relatively normal configuration.

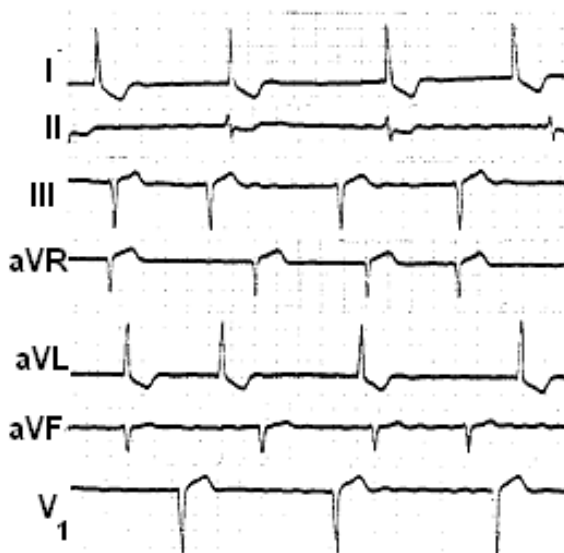


Fig. 4.99. Atrial fibrillation with a slow, irregular ventricular rate of 42 to 70 beats per minute. Note the fairly continuous undulating fibrillatory waves, varying in size, shape, and spacing.

TESTS

1. Identify ECG - sign of paroxysmal tachycardia?
 - A. Disappearance of the P wave;
 - B. Appearance of "sawtooth" f-waves with a frequency of 250-400 per minute;
 - C. Maintaining of regular sinus rhythm.
 - D. Sudden onset and end.
 - E. No differentiation of waves
2. What is the ECG sign of alorhythm?
 - A. Maintaining of regular sinus rhythm.
 - B. Maintaining of sinus rhythm.
 - C. There is no relationships P wave and QRS complex;
 - D. Tachycardia-bradycardia syndrome
 - E. Pattern of extrasystole appearance
3. What is ECG sign of ventricular extrasystole?
 - A. There are no relationships of P wave and QRS complex;
 - B. Premature excitation (QRS complex).
 - C. Deformed and enwidened QRS complex ($> 0,12$ c).
 - D. Shortening of P-P interval '.
 - E. The compensatory pause is absent or incomplete
4. Identify ECG sign of bigeminy?
 - A. The appearance of each second extrasystole;
 - B. The appearance of two monotopic extrasystole.
 - C. The appearance of two paired extrasystoles;
 - D. Interchange of different extrasystoles;
 - E. The pattern of extrasystole occurrence
5. What is ECG sign of atrial fibrillation?
 - A. P wave absence;
 - B. Appearance of "sawtooth" f-waves with a frequency of 250-400 per minute;
 - C. Maintaining of regular sinus rhythm.
 - D. Sudden onset and end.
 - E. No differentiation of waves
6. What is ECG sign of idioventricular rhythm?
 - A. Alternation of different P wave shapes, amplitudes and polarity.
 - B. There is no relation of P wave and QRS complex;
 - C. Tachycardia-bradycardia syndrome

- D. Change of P wave polarity and positivity.
 - E. Different QRS complexes amplitude
7. What is ECG sign of wandering pacemaker?
- A. Different QRS complexes amplitude
 - B. Alternation of different P wave shapes, amplitudes and polarity.
 - C. There is no relation of P wave and QRS complex;
 - D. Tachycardia-bradycardia syndrome
 - E. Change of P wave polarity and positivity
8. What is ECG sign of sick sinus syndrome?
- A. Tachycardia-bradycardia syndrome
 - B. Alternation of different P wave shapes, amplitudes and polarity
 - C. There is no relation of P wave and QRS complex;
 - D. Disappearance of the P wave;
 - E. ECG has the form sinusoids;
9. What is ECG sign of ventricular fibrillation?
- A. Tachycardia-bradycardia syndrome
 - B. Alternation of different P wave shapes, amplitudes and polarity.
 - C. There is no relation of P wave and QRS complex;
 - D. Disappearance of the P wave;
 - E. ECG has the form sinusoids
10. The negative P wave is recorded on ECG. Your opinion?
- A. Sinus rhythm;
 - B. Atrial rhythm;
 - C. Idioventricular rhythm;
 - D. Nodal rhythm;
 - E. Artificial pacemaker of the heart
11. In which rhythm P wave is absent or isoelectric?
- A. Sinus arrhythmia;
 - B. Wandering pacemaker;
 - C. Idioventricular rhythm;
 - D. Atrial premature beats;
 - E. Upper nodal rhythm
12. In which rhythm P wave is absent?
- A. Sinus arrhythmia;
 - B. Wandering pacemaker;

- C. Atrial premature beats;
- D. Ventricular fibrillation
- E. Upper nodal rhythm

13. In which case there is no waves differentiation on ECG?

- A. Atrial fibrillation;
- B. Atrial flutter;
- C. Ventricular fibrillation;
- D. Ventricular flutter;
- E. Artificial pacemaker of the heart

14 In patients with coronary artery disease ECG is recorded as small- and large waves line. Naming possible arrhythmias.

- A. Atrial fibrillation;
- B. Ventricular flutter
- C. Atrial flutter;
- D. Ventricular fibrillation
- E. Artificial pacemaker of the heart

15. In which case periodic missing of P wave on ECG is recorded?

- A. Sinus arrhythmia
- B. Atrial fibrillation;
- C. Artificial pacemaker of the heart
- D. Extrasystole
- E. Nodal rhythm;

16. The ECG recorded instead of the missing P wave tooth f-wave. Provide ECG conclusion.

- A. Atrial fibrillation;
- B. Atrial flutter;
- C. Idioventricular rhythm;
- D. Ventricular fibrillation;
- E. Ventricular flutter;

Standards of answers: 1D. 2E. 3C. 4A. 5A., 6B. 7B., 8A. 9E. 10B. 11C. 12D., 13D. 14B. 15D., 16A.

Methodical instructions

**ELECTROCARDIOGRAPHIC EXAMINATION OF
PATIENTS WITH AUTOMATICITY AND
EXCITABILITY FUNCTION ALTERATIONS**

Methodical instructions for students

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