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MEDICAL FACULTY
Department of Therapy № 2 Specialty General Medicine

I.S. Sabirov, K.A. Dzhailobaeva, Z.T. Radzhapova

ELECTROCARDIOGRAPHY

Handbook for Students

Approved by the Ministry of Education and Science
of the Kyrgyz Republic as a handbook
for students of higher educational institutions

Dedicated to the 30th years
of Medical Faculty of the Kyrgyz-Russian
Slavic University named after B.N. Yeltsin

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Reviewers:
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N.A. Toktogulova, MD, PhD, Associate Professor, Kyrgyz-Russian Slavic University named after B.N. Yeltsin,
A.S. Kerimkulova, MD, PhD, Associate Professor, Kyrgyz State Medical Academy named after I.K. Akhunbaev

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One of the most common and accessible methods of instrumental diagnostics of patients is electrocardiography (ECG), which, together with clinical data and the results of other diagnostic methods, makes it possible to make a timely and accurate diagnosis and, accordingly, prescribe treatment. In this connection, this method applies not only to functional diagnostic doctors but also to cardiologists and family doctors.

The basic knowledge acquired by students on ECG will allow them to be applied in clinical practice in the future.

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CONTENT

ABBREVIATIONS

AF – atrial flutter
AFib – atrial fibrillation
AMI – acute myocardial infarction
AV – atrioventricular (node)
BRs – Brugada syndrome
ECG – electrocardiogram
HR – heart rate
IHD – ischemic heart disease
IVS – interventricular septum
LA – left atrium
LAFB – left anterior fascicular block
LAH – left atrium hypertrophy
LBBB – left bundle branch block
LPFB – left posterior fascicular block
LV – left ventricle
LVH – left ventricular hypertrophy
PAC – premature atrial contraction
PAT – paroxysmal atrial tachycardia
PVC – premature ventricular contraction
RA – right atrium
RBBB – right bundle branch block
RAH – right atrium hypertrophy
RV – right ventricle
RV – right ventricular hypertrophy
SA – sinoatrial (node)
VT – ventricular tachycardia

INTRODUCTION

Electrocardiography is one of the oldest methods of cardiac examination, which has been used for more than a century and still takes a valuable place in the diagnosis of heart disease.

Despite the release of new alternative diagnostically relevant and sensitive methods, electrocardiography remains the most accessible tool for detecting heart pathology and, in particular, myocardial infarction, especially in the early stages of diagnosis. In addition, it is the main method for diagnosing arrhythmias and heart blocks.

Knowledge of the basics of an electrocardiogram interpretation is necessary for any specialty doctor to make a diagnosis timely and prescribe treatment. The textbook is intended for teaching medical students. The book also presents an algorithm for interpreting an electrocardiogram with a sequential assessment of the waves, segments, and intervals of a standard ECG. Algorithms are provided that allow for differential diagnosis of ECG changes in various conditions.

The purpose of this textbook is to teach students to quickly and competently read an electrocardiogram. The basic knowledge acquired by students on ECG will allow them to be applied in clinical practice in the future.

CHAPTER 1. NORMAL ECG

The electrocardiogram (ECG) is a graphical record of electric potentials generated by the heart muscle during each cardiac cycle. The signals are detected on the surface of the body using electrodes attached to the extremities and chest wall. These signals are then amplified by the electrocardiograph machine and displayed on special graph paper.

Normal conduction system of the heart

Conduction system of the heart: **sinoatrial node (SA)** is one of the major elements in the cardiac conduction system, the system that controls the heart rate (HR). This unique system generates electrical impulses and conducts them throughout the muscle of the heart, stimulating the heart to contract and pump the blood.

The SA (Fig. 1) node is the natural pacemaker of the heart. It consists of a cluster of cells that are located in the upper part of the wall of the right atrium (the right upper chamber of the heart) where electrical impulses are generated.

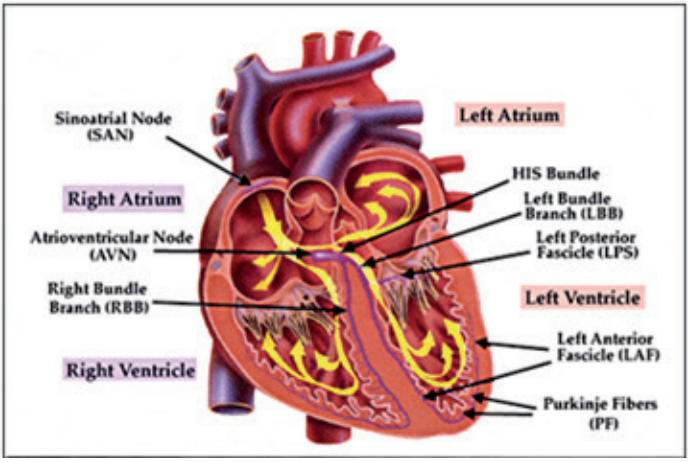


Figure 1. Normal conduction system of the heart

The electrical signal generated by the SA node moves from cell to cell down through the heart until it reaches the atrioventricular (AV) node, a cluster of cells located in the center of the heart between the atria and the ventricles. The AV node serves as a gate that slows the electrical impulse before the signal is permitted to go down through to the ventricles. This delay ensures that the atria have a chance to completely contract before the ventricles are stimulated. After passing the AV node, the electrical impulse goes to the ventricles along special fibers embedded in the walls of the lower part of the heart.

The autonomic nervous system controls the firing of the SA node to trigger cardiac cycle begining. The autonomic nervous system can send a message quickly to the SA node so it in turn can increase the heart rate to twice normal within only 3 to 5 seconds. This fast response is important during exercise when the heart has to increase its beating race to keep up with the body's increased oxygen demand.

AV node is an electrical relay station between the atria and the ventricles. Electrical signals from the atria must pass through the AV node to reach the ventricles.

How ECG works

When cell membranes in the heart depolarize, voltages change and currents flow. Because a human can be regarded as a bag of salt water (with bad attitude), in other words, a volume conductor, changes in potential are transmitted throughout the body, and can be measured. When the heart depolarizes, it's convenient (and fairly accurate) to represent the electrical activity as a dipole – a vector between two point charges (Fig. 2). Remember that a vector has both a size (magnitude), and a direction. By looking at how the potential varies around the volume conductor, one can get an idea of the direction of the vector. This applies to all intra-cardiac events, so we can talk about a vector (or axis) for P waves, the QRS complex, T waves, etc.

The normal cardiac cycle begins with spontaneous depolarization of the sinus node, an area of specialized tissue located in the right atrium (RA). A wave of electrical depolarization then spreads through the RA and across the inter-atrial septum into the left atrium (LA).

The atria are separated from the ventricles by an electrically inert fibrous ring, so that in the normal heart the only route of transmission of electrical depolarization from the atria to the ventricles is through the AV node. The AV node delays the electrical signal for a short time, and so the wave of depolarization spreads down the interventricular septum (IVS), via the bundle of His and the right and left bundle branches, into the right (RV) and left (LV) ventricles. Hence with normal conduction the two ventricles contract simultaneously to maximize cardiac efficiency.

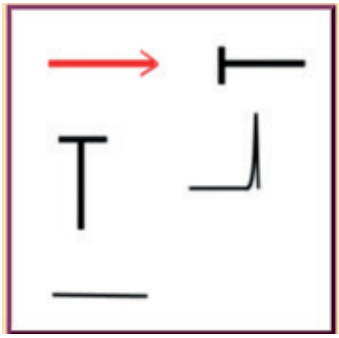


Figure 2. The schematic ECG lead on the right `sees' the vector moving towards it, shown as a positive deflection on the ECG; the lead at 90 degrees to this shows isoline

After complete depolarization of the heart, the myocardium must then depolarize, before it can be ready to depolarize again for the next cardiac cycle.

Function of the heart and ECG

The normal HR is between 60 and 100 beats per minute at rest, with some normal variations. For example, athletes at rest have slower HR than most people. This rate is set by a small collection of specialized heart cells called the SA node – "natural pacemaker." It has "automaticity," meaning it discharges all by itself without control from the brain. Two events occur with each discharge: both atria contract, and an electrical impulse travels through the atria to reach another area of the heart called the AV node, which lies in the wall between the two ventricles. The AV node serves as a relay point to further propagate

the electrical impulse. From the AV node, an electrical wave travels to both ventricles, causing them to contract and pump the blood. The normal delay between the contraction of the atria and of the ventricles is 0.12–0.20 seconds. This delay is perfectly timed to account for the blood flow from the atrium to the ventricle. Intervals shorter or longer than this range indicate possible problems.

The ECG records the electrical activity that results when the heart muscle cells in the atria and ventricles contract.

Atrial contractions (both right and left) reflect as the P wave.

Ventricular contractions (both right and left) reflect as a series of 3 waves, Q-R-S, known as the QRS complex.

The third and last common wave in an ECG is the T wave. This reflects the electrical activity produced when the ventricles are repolarizing for the next contraction.

ECG waves and intervals

ECG waves are labeled alphabetically starting with the P wave which represents atrial depolarization. The QRS complex represents ventricular depolarization and the ST-T-U complex (ST segment, T wave, and U wave) represents ventricular repolarization. The J point is the junction between the end of the QRS and the beginning of the ST segment. Atrial repolarization is usually too small in amplitude to be detected but may become apparent in some conditions such as acute pericarditis or atrial infarction.

The QRS-T waveforms on the ECG (Fig. 3) correspond in a general way to the different phases of simultaneously obtained ventricular action potentials, the intracellular recordings from single myocardial fibers:

- The rapid upstroke (phase 0) of the action potential corresponds to QRS onset.
- Phase 2 (plateau phase), during which myocardial fibers normally achieve the same potential, corresponds to the isoelectric ST segment.
- Phase 3 (active repolarization) corresponds to the T wave.
- Phase 4 (recovery) is seen as the isoelectric segment between the end of the T wave and the next depolarization.

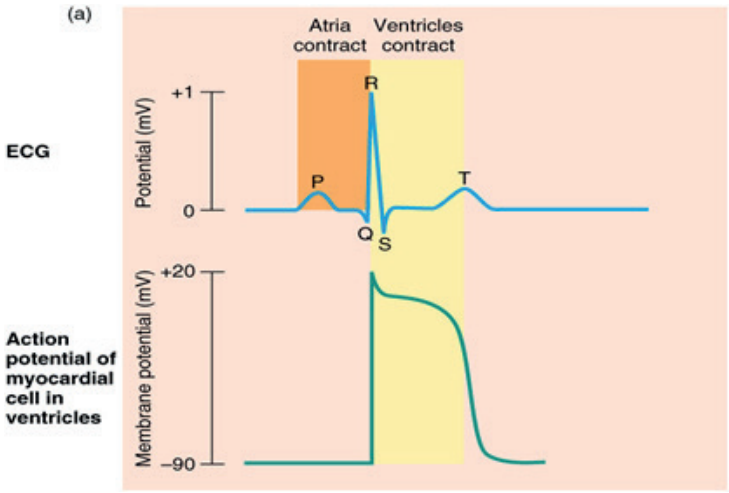


Figure 3. Ventricular action potential and QRS-T cycle

Some Important Points

Septum depolarizes from left to right side. On ECG LV has more influence than RV (hence, the normal cardiac axis is typically more left). Depolarization happens from the endocardium to the epicardium, and repolarization – from the epicardium to the endocardium.

In normal sinus rhythm, the atrium depolarization vector is directed from the right to the LA and downward towards the AV junction (arrow on Fig. 4). As result, the P wave will normally be upright in lead II and negative in lead avR.

Ventricular depolarization can be divided into two major phases, each represented by a vector (Fig. 5). Panel A: The first phase (arrow 1) denotes depolarization of the ventricular septum, beginning on the left side and spreading to the right. This process is represented by a small "septal" **r** wave in the anterior-posterior lead V1 and a small "septal" **q** wave in the left-right lead V6. Panel B: Simultaneous depolarization of the LV and RV constitutes the secondary phase. This secondary vector (arrow 2) is oriented to the left and posteriorly, reflecting the electrical predominance of the LV. The net effect is an S wave in lead V1 and an R wave in lead V6.

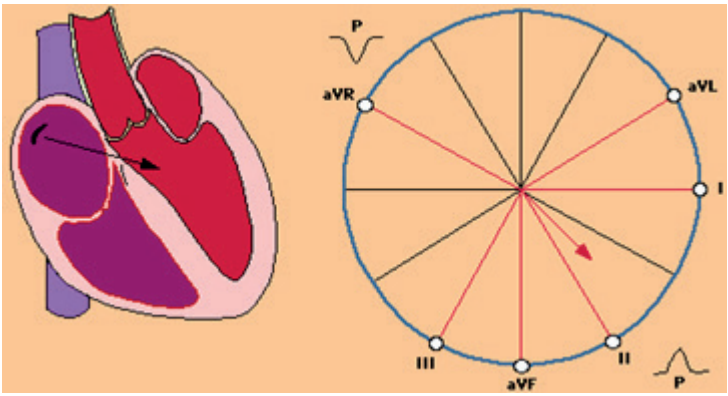


Figure4. Generation of the P-wave

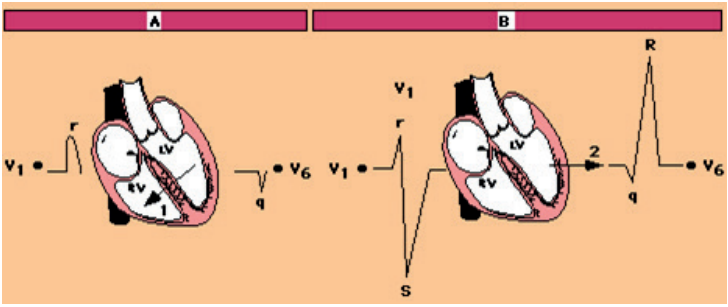


Figure 5. Generation of the QRS complex

THE CARDIAC ELECTRICAL CYCLE

The initiating event for cardiac contraction is the spread of depolarizing electrical currents through the heart. These currents are produced by three primary components: cardiac pacemaker cells; specialized conduction tissue; and the heart muscle itself. The surface ECG, however, only records the depolarization and repolarization potentials generated by the "working" atrial and ventricular myocardial fibers.

The depolarization stimulus for the heartbeat normally begins in the SA or sinus node, a collection of pacemaker cells with spontaneous automaticity. The initial phase of cardiac electrical activation consists

of the spread of the depolarization wave through the right and left atria, resulting in atrial contraction. The impulse then stimulates the pacemaker and specialized conduction tissues in the AV nodal and His-bundle areas. Together, these two regions constitute the AV junction.

The bundle of His splits into two main branches, the right and the left bundles, that rapidly transmit depolarization waves to the right and left ventricular myocardium, respectively, by way of Purkinje fibers. The main left bundle bifurcates into two primary subdivisions: left anterior and left posterior branches. The depolarization waves spread through the ventricular wall, from the endocardium (inner layer) to the epicardium (outer layer), triggering intracellular calcium release and myofilament contraction (electromechanical coupling).

Methodology of registration ECG

The ECG waves are recorded on special graph paper (Fig. 6) which is divided into 1 mm grid-like boxes. The ECG paper speed is ordinarily 25 mm/sec. As a result, each 1 mm horizontal box corresponds to 0.04 second (40 ms), with heavier lines at larger 0.20 sec (200 ms) intervals. Vertically, the ECG graph measures the height (amplitude) of a given wave or deflection, as 10 mm equals 1 mV with standard calibration.

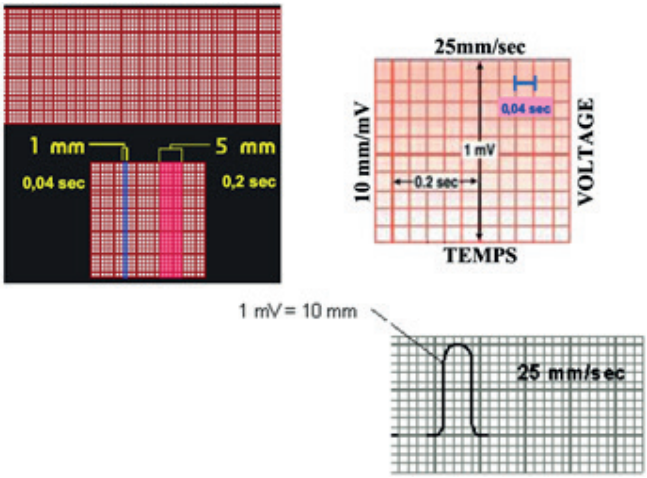


Figure 6. Voltage and speed on ECG

The Standard 12 Lead ECG

3 Standard Limb Leads
3 Augmented Limb Leads
6 Precordial Leads

12 conventional ECG leads record the difference in potential between electrodes at specific locations on the body surface. ECG leads (Fig. 7) are divided into two groups: six limb leads and six precordial leads.

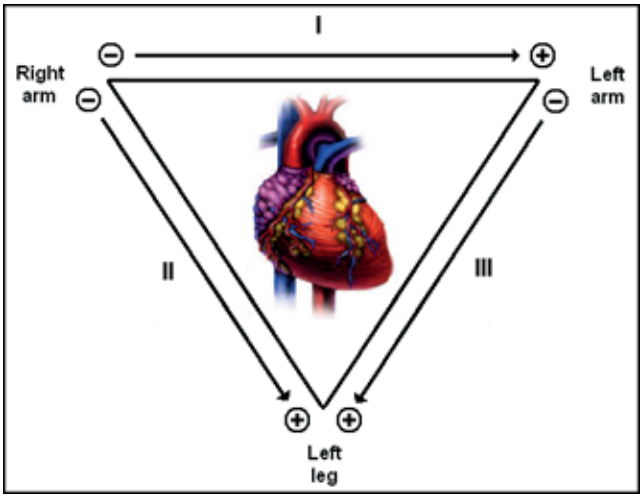


Figure 7. Standard Limb Leads

Limb leads record potentials transmitted onto the frontal plane and the chest leads record potentials transmitted onto the horizontal plane. The six limb leads are further subdivided into three bipolar leads (I, II, and III) and three unipolar leads (aVR, aVL, and aVF). Each bipolar lead (Fig. 8) measures the difference in potential between electrodes at two extremities:

- Lead I records the difference in electrical potentials between the left arm and the right arm
- Lead II records the difference in electrical potentials between the left leg and the right arm
- Lead III records the difference in electrical potentials between the left leg and the left arm

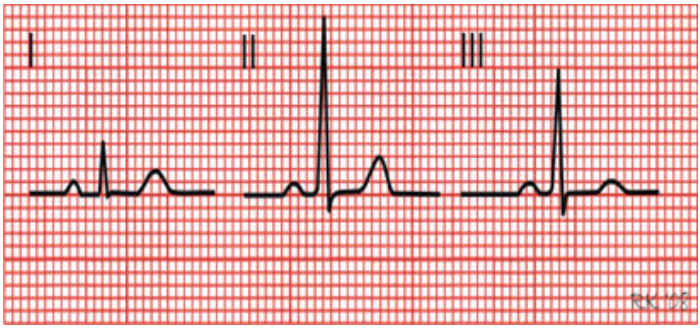


Figure 8 Standard Limb leads on normal ECG tracings

For a heart with a normal ECG and a mean electrical axis of +60º, the standard limb leads will appear as follows:

In addition to the three bipolar limb leads described above, there are three augmented unipolar limb leads (Fig. 9). These are termed unipolar leads because there is a single positive electrode that is referenced against a combination of the other limb electrodes.

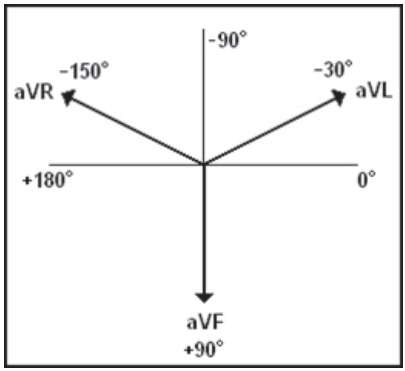


Figure 9. Augmented Limb Leads

The positive electrodes for these augmented leads are located on the left arm (aVL), the right arm (aVR), and the left leg (aVF). In practice, these are the same electrodes used for leads I, II, and III (Fig. 10).

Thus:

- Lead aVR records right arm potentials
- Lead aVL records left arm potentials

- Lead aVF records left leg (foot) potentials.

For a heart with a normal ECG and mean electrical axis of +60°, the augmented leads will appear as shown in Fig. 10.

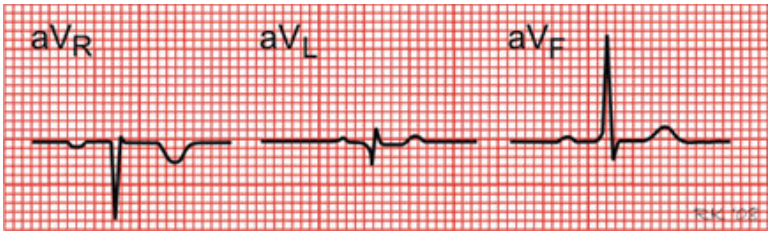


Figure 10. Augmented Limb leads on normal ECG

In addition to the three standard limb leads and the three augmented limb leads that view the electrical activity of the heart from the frontal plane, there are six precordial, unipolar chest leads. This configuration places six positive electrodes on the surface of the chest over different regions of the heart in order to record electrical activity in a plane perpendicular to the frontal plane. These six leads are named V_1 – V_6. The rules of interpretation are the same as for the limb leads.

They are obtained (Fig. 11) via electrodes placed in the following positions:

- Lead V_1 – 4th intercostal space (ICS), just to the right of the sternum.
- Lead V_2 – 4th ICS, just to the left of the sternum.
- Lead V_3 – between V_2 and V_4.
- Lead V_4 – 5th ICS in the mid-clavicular line.
- Lead V_5 – anterior axillary line, same level as V_4.
- Lead V_6 – mid-axillary line, same level as V_4 and V_5.

Because initial ventricular depolarization (Fig. 12) is from left to right across the septum, there is an initial R-wave in V_1 followed by an S-wave as the anterior and lateral walls of the left ventricle depolarize. Leads V_5 and V_6 show a large net positive QRS because these leads overlie the anterolateral wall of the left ventricle, which has a large muscle mass undergoing depolarization. Tracings from leads V_5 and V_6 are almost opposite in polarity from V_1 because they are viewing opposite sides of the heart. Leads V_2-V_4 are intermediate owing to their electrode placement.

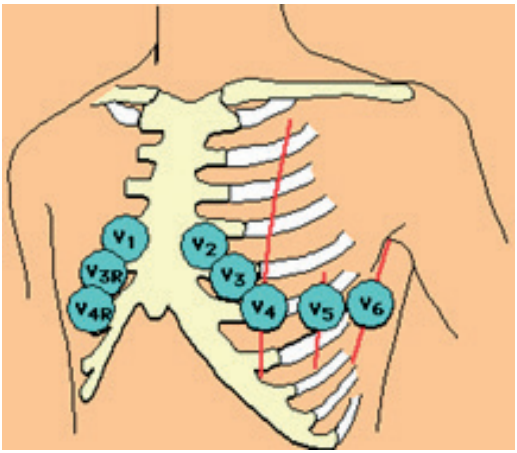


Figure 11. Precordial Leads

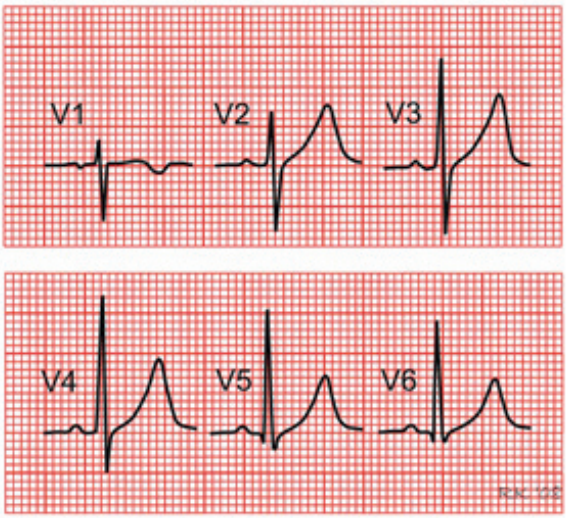


Figure 12. Chest leads on normal ECG tracings

In summary, the chest leads provide a different view of the electrical activity within the heart (Tabl. 1). So, the waveform recorded is different for each lead compared to the limb leads.

Table 1. The chest leads overlie the following ventricular regions

Leads	Ventricular Region
V_1–V_2	anteroseptal
V_3–V_4	anteroapical
V_5–V_6	anterolateral

In suspected myocardial infarction of the right ventricle or posterior wall it is also necessary to record additional leads RV_3–RV_4 and V_7–V_9 (Fig. 86, 87 in Chapter 6).

Determining Heart Rate from the Electrocardiogram

The term "heart rate" normally refers to the rate of ventricular contractions. However, because there are circumstances in which the atrial and ventricular rates differ (e.g., second and third-degree AV block), it is important to be able to determine both atrial and ventricular rates. This is easily done by examining an ECG rhythm strip, which is usually taken from Lead II. In the example below, there are four numbered R waves, each of which is preceded by a P wave. Therefore, the atrial and ventricular rates will be the same because there is a one-to-one correspondence.

The atrial rate (Fig. 14) can be determined by measuring the time intervals between P waves (P–P intervals). The ventricular rate can be determined by measuring the time intervals between the QRS complexes, which is done by looking at the R–R intervals.

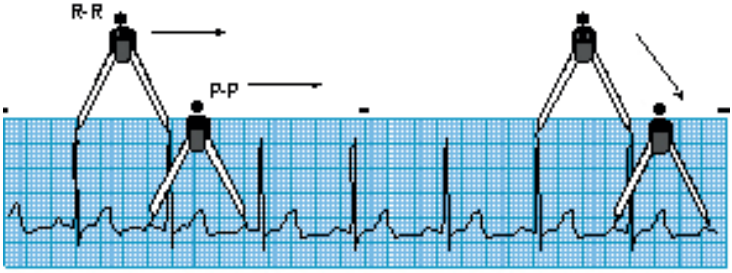


Figure 14. Measuring the time P–P intervals and the R-R intervals

There are different short-cut methods (Fig. 15) that can be used to calculate heart rate, all of which assume a recording speed of 25 mm/

sec. One method is to divide 1500 by the number of small squares between two R waves. For example, the rate between beats 1 and 2 in the above tracing is 1500/22, which equals 68 beats/min.

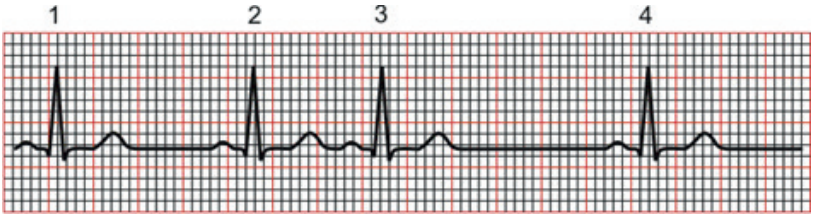


Figure 15. Method of calculation of heart rate

Alternatively, can be calculated by dividing 300 by the number of large squares, which is 300/4.4 (68 beats/min).

Another method (Fig. 16), which gives a rough approximation, is the "triplet method". Simply count the number of large squares between R waves with the following rates: 300 – 150 – 100 – 75 – 60 – 50 – 43 – 38. For example, if there are three large boxes between R waves, then the rate is 100 beats/min.

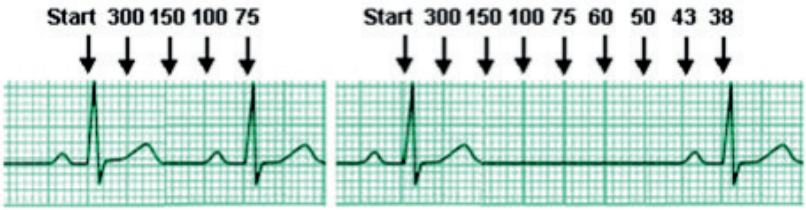


Figure 16. Triplet heart rate technique

The atrial rate can be determined like the ventricular rate, but using the P waves. Remember, if there is in sinus rhythm and one-to-one correspondence between P waves and QRS completes, then the atrial rate will be the same as the ventricular rate.

In the above examples, the ventricular rate was determined based on the interval between the first two beats. However, it is obvious that the rate would have been faster had it been calculated using beats 2 and 3 (104 beats/min) because of a premature atrial beat, and slower

if it had been calculated between beats 3 and 4 (52 beats/min). This illustrates an important point when calculating the rate between any given pair of beats. If the rhythm is not steady, it is important to determine a time-averaged rate over a longer interval (e. g., over ten seconds or longer). For example, because the recording time scale is 25 mm/sec, if there are 12.5 beats in 10 seconds, the rate will be 75 beats/min.

ECG waveform and intervals

ECG waves are labeled alphabetically starting with the P wave which represents atrial depolarization. The QRS complex represents ventricular depolarization and the ST–T–U complex (ST segment, T wave, and U wave) represents ventricular repolarization. The J point is the junction between the end of the QRS and the beginning of the ST segment. There are four major ECG intervals: R–R, PR, QRS, and QT (Fig. 17).

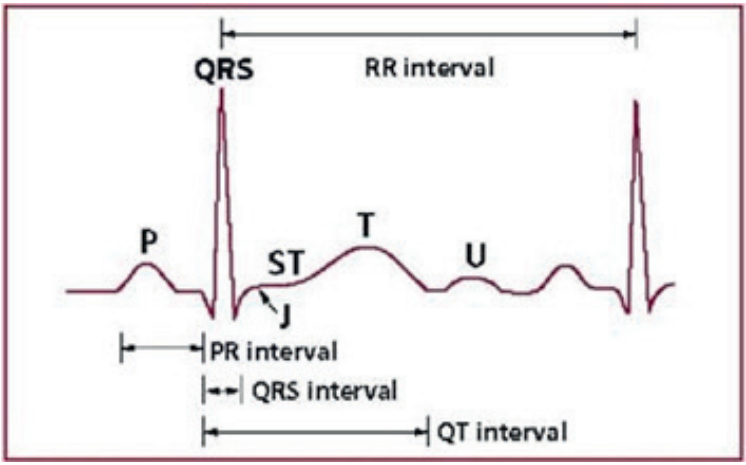


Figure 17. ECG Waves and intervals

Formation of the normal ECG

P wave

The P wave represents the wave of depolarization that spreads from the SA node throughout the atria.

- P duration < 0.12 sec
- P amplitude < 2.5 mm
- Frontal plane P wave axis: 0° to +75° (PII > PI > PIII)
- May be present notched P waves in the frontal plane

"Sinus" morphology: positive P wave in leads I and avF. In leads III, aVL **P** wave can be positive, biphase, negative. The normal P wave in lead V_1 may be biphasic with a positive component reflecting right atrial depolarization, followed by a small (<1 mm^2) negative component reflecting left atrial depolarization and both phases is equal.

P–R (or P–Q) interval

The period of time from the onset of the P wave to the beginning of the QRS complex is termed the P-R interval, which normally ranges from 0.12 to 0.20 seconds in duration. This interval represents the time between the onset of atrial depolarization and the onset of ventricular depolarization. PT constant, but PT interval changes when HR changes. At sinus tachycardia normal P–R interval is -0,12 -0,18sec., at sinus bradycardia normal P-R interval is to 0,22 sec.

QRS Complex

The QRS represents the *simultaneous* activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature (Fig. 18). **Q** wave: first downstroke of the QRS complex. This wave is not always present.

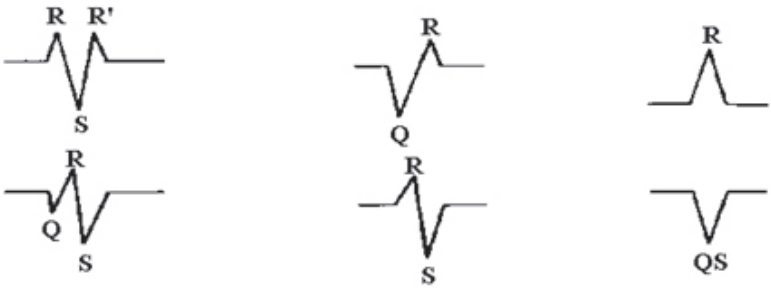


Figure 18. Multiple variations of the QRS complex

However, if there is an upward deflection (i. e. R wave) noted before a "Q wave" occurs, then that Q wave is actually S wave because Q waves must be before any R wave by definition. R wave: first upward deflection of the QRS complex. Upward deflections occurring after S wave are noted by a "prime mark" such as R'. S wave: the first downward deflection occurring after the R wave.

- QRS duration 0.08–0.12 seconds.

- QRS amplitude is quite variable from lead to lead and from person to person.

Two determinates of QRS voltages are:

1. Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)

2. The proximity of chest electrodes to the ventricular chamber (the closer, the larger the voltage)

Frontal plane leads:

The normal QRS axis range (+90 o to -30 o); this implies that the QRS be mostly positive (upright) in leads II and I.

Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They are often seen in leads I and aVL when the QRS axis is to the left of +60o, and in leads II, III, aVF when the QRS axis is to the right of +60o. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.

Precordial leads:

- Small r-waves begin in V_1 or V_2 and progress in size to V_5. The RV6 is usually smaller than RV_5.
- In reverse, the s-waves begin in V_6 or V_5 and progress in size to V_2. SV_1 is usually smaller than SV_2.
- The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V_3 or V_4.
- Small «septal» q-waves may be seen in leads V_5 and V_6.

Thus, a right precordial lead like V_1 will record this biphasic depolarization process with a small positive deflection (septal r wave)

followed by a larger negative deflection (S wave). In contrast, a left precordial lead such as V_6 will record the same sequence with a small negative deflection (septal q wave) followed by a relatively tall positive deflection (R wave). Intermediate precordial leads show a relative increase in R wave amplitude and a decrease in S wave amplitude progressing across the chest from right to left. This normal R wave progression in the precordial leads is a reflection of the progressive more leftward orientation of these leads. The precordial lead where the R wave and S wave are of about equal amplitude is referred to as the transition zone (usually lead V_3 or V_4).

QT interval

The QT interval represents the duration of activation and recovery of the ventricular muscle. This duration varies inversely with the heart rate. QT interval: ~400ms – the time between the beginning of QRS complex and end of T wave.

Bazett's Formula: $QTc = QT/\sqrt{RR}$.

Hodges formula: $QTc = QT + 1.75$ (ventricular rate – 60).

S wave

S wave ends at the beginning of ST segment. This point is sometimes called the J point. In the usual "textbook" ECG, the S wave ends on the isoelectric line. However, in many cases, the ST segment is not so flat. In Figure 19, notice that the ST segment starts below the isoline. The J point is the the junction between the termination of the QRS complex and the beginning of the ST segment.

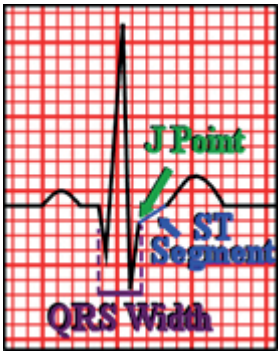


Figure 19. The width of the QRS complex

ST segment

The isoelectric period (ST segment) following the QRS is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential.

The ST segment is important in the diagnosis of ventricular ischemia or hypoxia because under those conditions, the ST segment can become either depressed or elevated (Fig. 20).

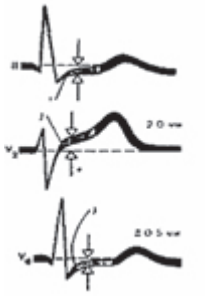


Figure 20. Multiple variations of ST segment

Normal ST segment elevation can be in leads with large S waves (e. g., V_1-V_3), and the normal configuration is ***concave upward***. ST-segment elevation with concave upward appearance may also be seen in other leads; this is often called ***early repolarization*** (Fig. 21).

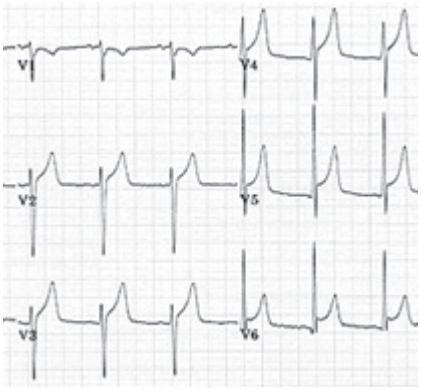


Figure 21. Early repolarization in leads V_4–V_6

T wave

The T wave represents ventricular repolarization and is longer in duration than depolarization (i.e., conduction of the repolarization wave is slower than the wave of depolarization).

In some normal individuals, particularly women, the T wave is symmetrical and a distinct, horizontal ST segment is present. The normal T wave is usually in the same direction as the QRS except in the right precordial leads. In the normal ECG the T wave is always upright in leads I, II, V3–V6, and always inverted in lead aVR.

U wave

Sometimes a small positive U wave may be seen following the T wave. This wave represents the last remnants of ventricular repolarization. Inverted or prominent U waves indicates underlying pathology or conditions affecting repolarization.

The normal U Wave: (the most neglected of the ECG waveforms):

- U wave amplitude is usually < 1/3 T wave amplitude in same lead.
- U wave direction is the same as T wave direction in that lead.
- U waves are more prominent at slow heart rates and are usually best seen in the right precordial leads.
- The origin of the U wave is thought to be related to **after depolarizations** which interrupt or follow repolarization.

How to Measure QRS Axis

The frontal plane QRS axis represents only **the average direction of ventricular activation** in the frontal plane. As such this measure can inform the ECG reader of changes in the sequence of ventricular activation (e. g., left anterior fascicular block), or it can be an indicator of myocardial damage (e. g., inferior myocardial infarction).

First find the **isoelectric** lead if there is one; i. e., the lead with equal forces in the positive and negative direction. Often this is the lead with the smallest QRS. The QRS axis is **perpendicular** to that lead's orientation. Since there are two perpendiculars to each isoelectric lead, chose the perpendicular that best fits the direction of the other ECG leads. If there is no isoelectric lead, there are usually two leads that are nearly isoelectric, and these are always 30^{o} apart (Fig. 22). Find

the perpendiculars for each lead and chose an approximate QRS axis within the 30o range. Occasionally each of the 6 frontal plane leads is small and/or isoelectric. The axis cannot be determined and is called **indeterminate**. This is a normal variant.

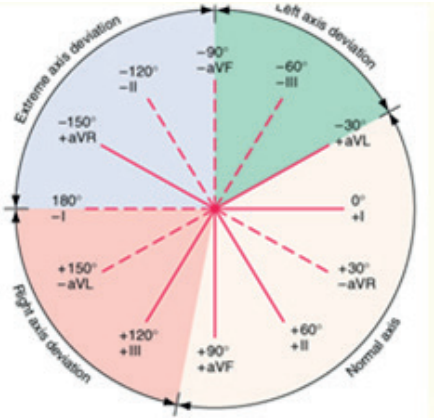


Figure 22. The range QRS axis

In the diagram below (Fig. 23):

1. The normal range is identified – angle α (-30° to +90°) or $R_{II}>R_I>R_{III}$.
2. Left axis deviation is defined from – angle α (-30° to -90°) or $R_I>R_{II}>R_{III}$ and $S_{III}>R_{III}$.
3. Right axis deviation is defined from – angle α (+90° to +150°) or $R_{III}>R_{II}>R_I$ and $S_I>R_I$.

	Normal Axis 0 to 90	Left Axis Physiological 0 to -30	Left Axis Pathological -30 to -90	Right Axis 90 to 180	Extreme Axis -90 to 180	Indeterminate Axis ?
Lead I						
Lead II						
Lead III						

Figure 23. Diagram showing how the polarity of the QRS complex in leads I, II, and III can be used to estimate the heart's electrical axis in the frontal plane

CHAPTER 2. HYPERTROPHY

Atrial hypertrophy
Left atrial hypertrophy

In left atrial hypertrophy (LAH) the electrical impulse due to the enlarged left atrium is strengthened. This electrical impulse is directed mainly along lead I or opposite to the direction of lead V_1. Because the atrial activation starts from the right atrium, the aforementioned left atrial activation is seen later, and therefore, the P-wave includes two phases. In lead I these phases have the same polarities and in lead V_1 the opposite polarities. This typical P-wave form is called the mitral P-wave. The specific diagnostic criterion for left atrial hypertrophy (Fig. 24) is the terminal portion of the P-wave in V_1, having a duration 0.04 s and a negative amplitude 0.1 mV.

ECG criteria of the left atrial hypertrophy

Two-humped P wave in I, II, aVL, V_5, V_6 leads.

1. The distance between two parts of P waves is greater 0,02 seconds.
2. The duration of P wave is 0,11 seconds and more.
3. P is negative in aVR lead.
4. Left or horizontal deviation of the electrical axis of P wave: $P_I > P_{II} > P_{III}$
5. In V_1 lead P is a dysphasic wave, with the prevalence of the second negative phase.

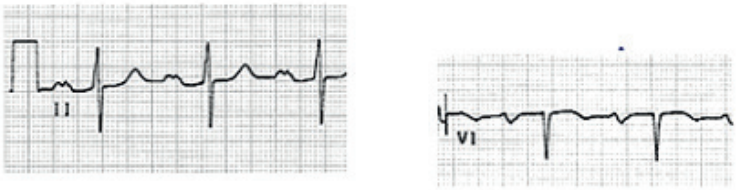


Figure 24. ECG of the left atrial hypertrophy

Right atrial hypertrophy

Right atrial hypertrophy (RAH) is a consequence of right atrial overload. This may be a result of tricuspid valve disease (stenosis or insufficiency), pulmonary valve disease, or pulmonary hypertension (increased pulmonary blood pressure). The latter is most commonly a consequence of chronic obstructive pulmonary disease or pulmonary embolism.

In RAH the electrical force due to the enlarged right atrium is larger. This electrical force is oriented mainly in the direction of lead II but also in leads aVF and III (Fig. 25). In all of these leads an unusually large (i. e., 0.25 mV) P-wave is seen.

ECG criteria of the RAH

1. Tall, peaked P wave, 2,5 mm in II, III, aVF leads, Electrical axis of the atrium – $P_{III}>P_{II}>P_{I}$.
2. P wave peaked and negative in aVR lead.
3. P wave peaked in V_1, tall or biphasic with prevalence of first positive phase.
4. Time of right atrium excitation increases (Norm = 0,04 sec.).

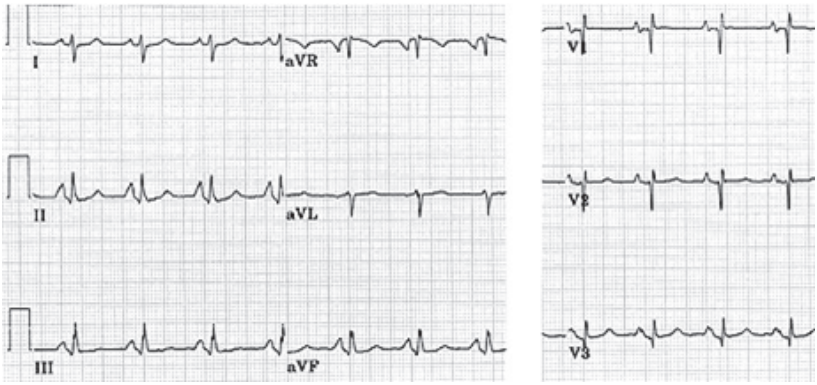


Figure 25. ECG of the RAH

Bi-Atrial Hypertrophy

1. Features of both RAH and LAH in same ECG.
2. P wave in lead II >2.5 mm tall and >0.12s in duration.

3. Initial positive component of P wave in V1 >1.5 mm tall and prominent P-terminal force (Fig. 26).

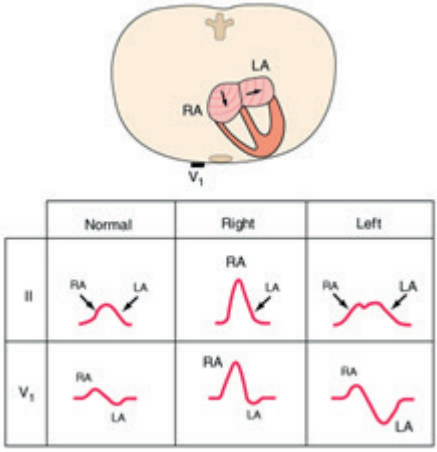


Figure 26. Change P wave at the LAH and RAH

Ventricular hypertrophy
Left ventricular hypertrophy

Left ventricular hypertrophy (LVH) is a consequence of left ventricular overload. It can be developed due to mitral valve disease, aortic valve disease, or systemic hypertension. LVH may also be a consequence of obstructive hypertrophic cardiomyopathy, which is a sickness of the cardiac muscle cells.

LVH increases (Fig. 27, 28) the ventricular electric forces directed to the left ventricle – that is, to the left and posteriorly. Evidence of this is seen in lead I as a tall R-wave and in lead III as a deep S-wave (2.5 mV). Also a deep S-wave is seen in precordial leads V_1 and V_2 and a tall R-wave in leads V_5 and V_6 (3.5 mV).

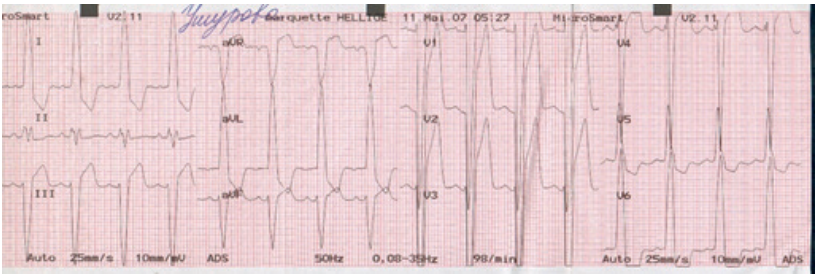


Figure 27. Combination LVH and left bundle-branch block

ECG criteria of the left ventricular hypertrophy:

1. Amplitude of R wave in V_5, V_6 > 25 mm.
2. Amplitude of S wave in V_1 > 12 mm.
3. Sokolow-Lyon Index: SV_1 + R (V_5 or V6) ≥ 35 mm.
4. The modified Sokolow-Lyon index (largest S-wave + largest R-wave > 35 mm).
5. Displacement of transition zone to the right.
6. Deviation of electrical axis of heart to the left (α angle – from 0° to 30°).
7. Amplitude of R wave in aVL > 11 mm.
8. Sum of amplitudes of R I and S_{III} ≥ 25 mm.
9. Cornel voltage index: (S V_3 + R aVL > 28 mm (for males) and > 20 mm (for females).
10. Cornell voltage QRS duration product (> 244 mV·ms)
11. In combination of LVH and left bundle branch block: SV_1 + RV_6 ≥ 45 mm.

Relative signs of left ventricular hypertrophy:

1. Amplitude of R wave in V_5, V_6 leads > 26 mm.
2. R avL > 7 mm.
3. R_I + S_{III} > 20 mm.
4. SV_1>12 mm.
5. QV4-V6 ≥ 2,5 mm and Q < 0,03 sec., Q/R < ¼ R in these leads.
6. Time lapse of left ventricle of heart excitation in V5,6 ≥ 0,05 sec.

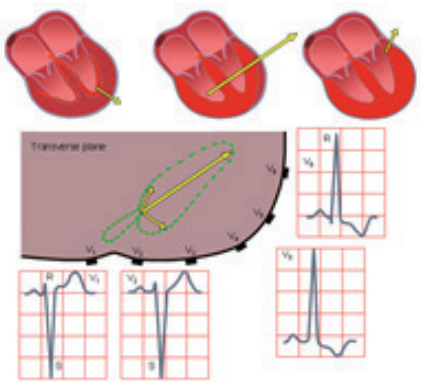


Figure 28. Left ventricular hypertrophy

Right ventricular hypertrophy

Right ventricular hypertrophy (RVH) is a consequence of right ventricular overload. This is caused by pulmonary valve stenosis, tricuspid insufficiency, or pulmonary hypertension. Also, many congenital heart defects, such as a ventricular septal defect, may cause right ventricular overload.

RVH (Fig. 29) increases the ventricular electrical forces directed to the right ventricle – that is, to the right and front. This is seen in lead V1 as a tall R-wave of 0.7 mV.

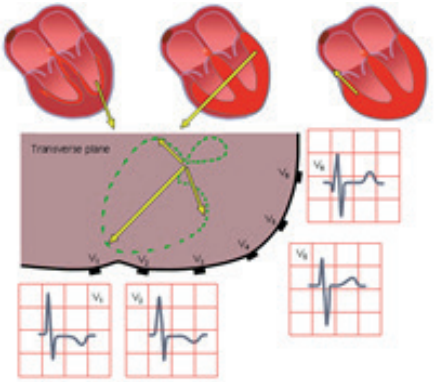


Figure 29. Right ventricular hypertrophy

ECG – right ventricular hypertrophy criteria (Tabl. 2) can be of R and S types (is estimated based on QRS complex form in V_1 lead).

1. R V_1 > 7 mm.
2. S V_1 < 2 mm.
3. S V_5 > 7 mm.
4. Decreased amplitude of R in V_6 < 5 mm.
5. Sum of amplitudes of RV_1 and S V6 > 10,5 mm.
6. R_{AVR} ≥5 mm.
8. Displacement of the electrical axis of the heart to the right (α angle is > +90°).
9. R_I: S_{V1} > 1.0.
10. R type of right ventricular hypertrophy on qR, rSR′ types (complete or incomplete right bundle-branch block). At incomplete right bundle-branch block, the height of R1 in V1 lead is ≥ 10 mm, at incomplete ≥ 15 mm.
11. S type right ventricular hypertrophy is characterized by deviation of electrical heart axis to the right (rS_I, qR_{III}) or deep S waves in I, II, III, leads and in V_1–V_6 leads, rS form of ventricular complex is observed.

Table 2. R and S types of right ventricular hypertrophy

Right ventricular hypertrophy		
R-type		S-type
qR	Rs	

qR type of RVH is characterized with presence of q wave (width and depth have no meaning) and tall R wave at QRS <0,10 sec (Fig. 30).

Rs-type of RVH (Fig. 31) is characterized by the presence of tall R wave in V1 > 7 mm, amplitude of S wave in V_1 < 2 mm. R_{V1} ratio to S_{V1} > 1.0.

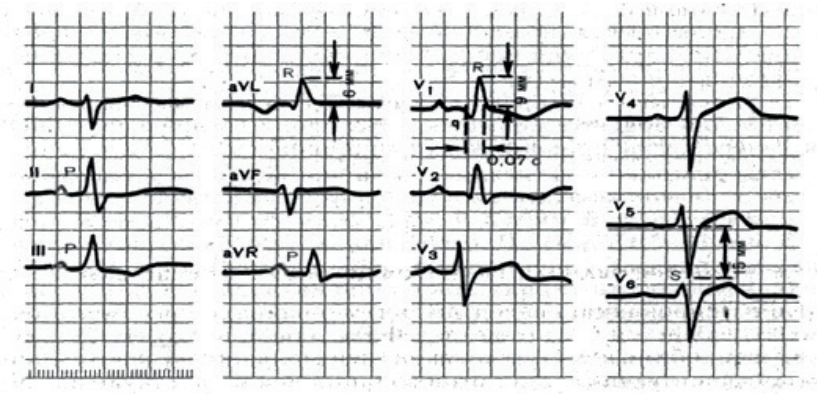


Figure 30. qR-type of the RVH

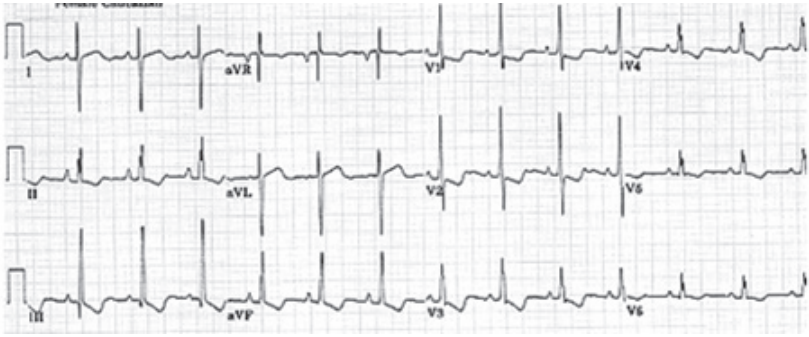


Figure 31. Rs-type of the RVH

qR and R types of RVH take place at mass prevalence of the right ventricle of the heart myocardium over the mass of myocardium of the left ventricle of the heart.

RVH is characterized by the presence tall R wave in V1 > 7 mm, amplitude of S wave in V_1 < 2 mm. Ratio R_{V1} to S_{V1} > 1.0.

Incomplete type of right bundle-branch block (rsR'), whereas QRS < 0, 12 sec. and R' ≥ 10 mm in V_1 lead.

Complete type of right bundle-branch block (rsR'), whereas QRS ≥ 0,12 sec. and R' ≥ 15 mm in V_1 lead.

S-type (Fig. 32) is characterized by deviation of the electrical axis of the heart to the right (rS_I, qR_{III}), or deep S waves in I, II, III leads and in V_1 to V_6 leads is observed rS form of ventricular complex.

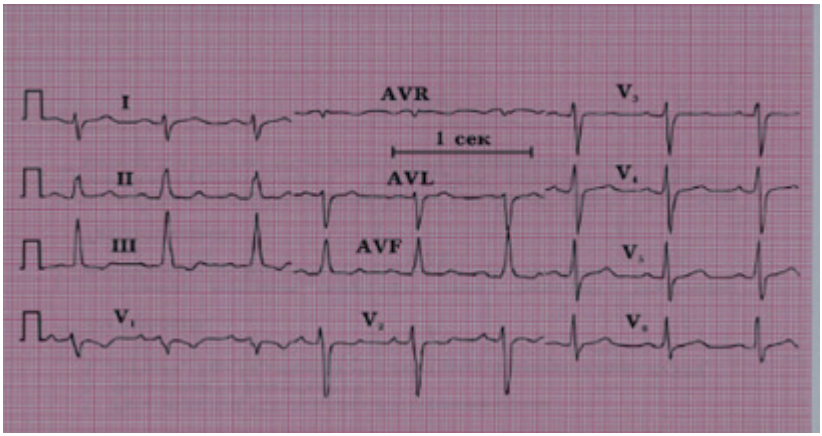


Figure 32. S-type of the RVH

Combined ventricular hypertrophy

Signs of right and left ventricular hypertrophy are identified on ECG at the same time:

1. In V_5, V_6 leads a tall R wave is registered (more often R V_5, V_6 > R V_4), is caused by LVH. In V_1, V_2 leads; R wave is also tall and exceeds 5–7 mm (Fig. 33).
2. Signs of LVH in V_5, V_6 leads and at the same time an image of complete or incomplete right bundle-branch block in V_1, V_2 leads.
3. A combination of signs of LVH and deviation of the electrical axis of the heart to the right.
4. A combination of signs of RVH and deviation of the electrical axis of the heart to the left.
5. Signs of RVH with tall R waves in V_1, V_2 leads, q wave is absent in V_5, V_6 leads.
6. Signs of LVH with tall R wave in V_5, V_6, leads, S wave in V_1, V_2 leads with small amplitude, quite often with simultaneously increased of R wave height in V_1, V_2 leads.
7. Criteria of LVH, deep S wave in V_5 lead or in V_6, or in these both leads.

8. Signs of RVH registered evident q wave in $V_{5,6}$ and tall R in $V_{5,6}$. q wave is due to hypertrophy of the left side the interventricular septum.
9. Signs of RVH and sum RV_5 or $RV_6 + SV_1$ or $SV_1 > 28$ mm in person > 30 years or 30 mm at person < 30 years.

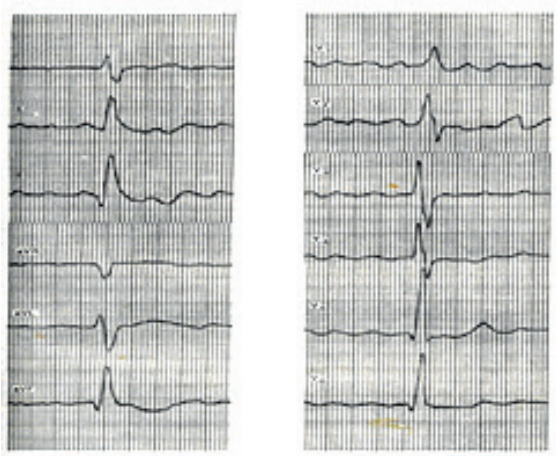


Figure 33. Combined ventricular hypertrophy

CHAPTER 3.
ECG IN EXCITABILITY DISTURBANCE

Classification of excitability disturbance

I. Impulse diffusion disturbance, more often circulation (re-entry) of excitability, for which it is necessary:

1. Presence of two ways of impulse conduction.
2. Development of unidirectional blockade of impulse on one of them.
3. Slow impulse conduction through another way and retrograde (reverse) return through the earlier blocked way, which leads to loop closing re-entry.
4. Multiple circulations of impulse lead to the formation of tachycardia, a single-premature beat.

II. Excitability formation disturbance can be caused by two reasons:

A. Increase of automatism due to:

1) increased entry of catecholamine into the bloodstream;
2) electrolytic disorders (hypokalemia, hypercalcemia);
3) hypoxia, ischemia;
4) mechanical effect;
5) digitalis.

B. Trigger activity in the atrium, ventricles, and in His-Purkinje system tissue, at local increase of catecholamine concentration, hypokalemia, hypocalcemia, and intoxication with digitalis medication.

In the core of the trigger mechanism is excessive accumulation of calcium ions in myocardium cells. This is accompanied by their depolarization. The amplitude of post-depolarization can reach to threshold level, which leads to the formation of premature contraction.

Classification of excitability disturbance

Nomotopic rhythm.

1. Sinus tachycardia.
2. Sinus bradycardia.
3. Sinus arrhythmia.
4. Sick sinus syndrome.

Heterotropic rhythm

A. Vicarious (substitutional) heterotopia

1. Rhythm from atrioventricular connection.
2. Ventricular (idioventricular) rhythm.
3. Migration of pacing lead.
4. Escape contraction from the atrium, AV bundle, and ventricular.

B. Active heterotopia

1. Premature beat (premature contraction, premature systole, or extrasystole):

1. Supraventricular.
2. Ventricular.

2. Paroxysmal tachycardia:

1. Supraventricular.
2. Ventricular.

3. Atrial fibrillation and atrial flutter.

4. Flutter, ventricular fibrillation.

Pathogenesis of excitability disturbance

Re-entry (Fig. 34) can take place within a small local region within the heart or it can occur, for example, between the atria and ventricles (global re-entry). For re-entry to occur, certain conditions must be met that are related to the following:

1) the presence of a unidirectional block within a conducting pathway;

2) critical timing;

3) the length of the effective refractory period of the normal tissue.

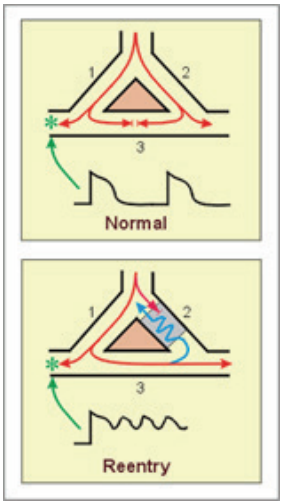


Figure 34. The mechanism of reentry

Nomotopic rhythm disturbances
Sinus tachycardia

Originates in the SA node (Fig. 35). Rapid rhythm which occurs with increased oxygen demand (exercise, infection, hypovolemia, hypoxia, myocardial infarction, and to stimulant drugs).

1. Heart rate > 100–180 / bpm.
2. R–R interval is shortened. Rhythm is regular.
3. Sinus P waves of usual configuration, their amplitude might be increased.
4. Upsloping ST depression on 1 mm.
5. T wave is tall.
6. PQ interval is approaching the bottom border of normal (0.12 sec.)
7. Every P has a QRS and every QRS has a P, QRS: normal

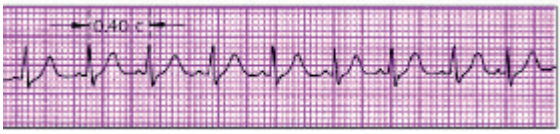


Figure 35. Sinus tachycardia

Sinus bradycardia

Originates in the SA note (Fig. 36). Rate is slower because of sympathetic input or excessive vagal tone. Seen most often with inferior MI, hypoxia, hypothermia, or drug reactions. Patient may be asymptomatic.

1. Heart rate < 60 min/bpm
2. Rhythm is regular, sinus arrhythmia is typical.
3. R–R interval is lengthened.
4. Sinus P waves, flattened.
5. Flattened T wave.
6. PQ interval is approaching the upper border or norm (0.20 sec.)
7. Every P has a QRS and every QRS has a P.

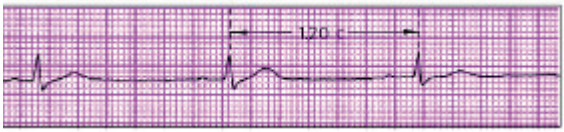


Figure 36. Sinus bradycardia

Sinus arrhythmia

Sinus arrhythmia is very common in all age groups. This arrhythmia is so common in young people that it is not considered a heart disease.

1. Sinus P waves are preserved in usual configuration.
2. Heart rate variation between minimal and maximal values > 15 %.
3. R–R interval is different, variations are >15 %.
4. P–Q intervals are different, of normal duration.

One origin for the sinus arrhythmia (Fig. 37) may be the vagus nerve which mediates respiration as well as heart rhythm. The nerve is active during respiration and, through its effect on the sinus node, causes an increase in heart rate during inspiration and a decrease during expiration. The effect is particularly pronounced in children. Changes of R–R interval depend on breathing phases: R-R interval shortens on inspiration, and it lengthens on exhalation.

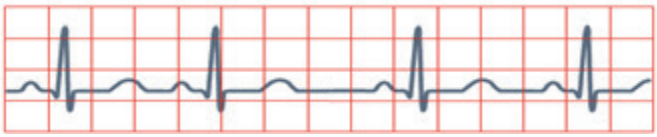


Figure 37. Sinus arrhythimia

Sick sinus syndrome – disturbance of SA nodal function that results in a markedly variable rhythm.

1. Steady sinus bradycardia.
2. Periodic appearance of ectopic, accelerated (non-sinus) rhythms.
3. Presence of sino-atrial blockade.
5. Syndrome of bradycardia-tachycardia.
6. Atrial fibrillation, bradysystolic form.

ECG-Criteria of vicarious heterotopia: rhythm from A–V connection, migration of pacing lead, escape beat, idioventricular rhythm.

AV Junctional Rhythm

I type – fastened retrograde excitation installation from AV node to the atria and slowed conduction to the ventricles (Fig. 38, a)

1. P wave is negative in II, III, aVF leads.
2. P wave is positive in aVR lead.
3. P–Q interval < 0,12 sec.
4. QRS complex is not changed.
5. Frequency of heartbeat from 30 to 50 bpm.
6. At heartbeat ≥ 60 to 100 bpm this disturbance is called accelerated ectopic atrioventricular nodal rhythm.

II type – simultaneous excitation impulse installation on atria and ventricles (Fig. 38)

1. Absence of P wave (it merges with QRS complex)
2. QRS complex is not changed.
3. The frequency of heartbeat is less than 60/bpm.

III – fastened excitation conduction to the ventricles and slowed retrograde conduction to the atria (Fig. 38, c)

1. P wave is registered after QRS complex.

2. R–P distance is equal to 0.10–0.20 seconds.
3. P wave is negative in all leads.
4. Frequency of heartbeat is less than 60/bpm.

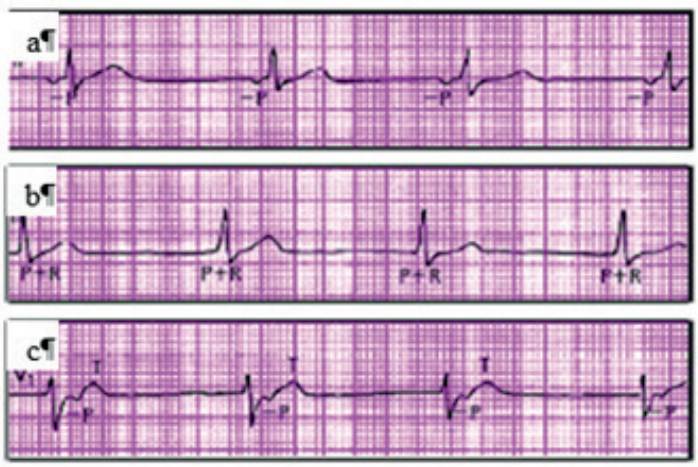


Figure 38. ECG of patients with slow escape rate

a – atrial rhythm;

b – rhythm from AV junction with simultaneous excitation of atria and ventricles;

c – rhythm from AV junction with excitation of ventricles, preceding the atria.

Idioventricular Rhythm

Escape rhythm (safety mechanism) to prevent ventricular arrest. His-Purkinje system takes over as the heart's pacemaker.

1. Heart rhythm is regular.
2. P wave can be absent or can be atrial fibrillation or complete AV block.
3. QRS wide, > 0,12 sec.
4. HR < 30 bpm.

Wandering atrial pacemaker

The origin of the atrial contraction may also vary or wander (Fig. 39). Shifting the natural cardiac pacemaker site between the SA node, the atria, and / or the AV node is called wandering atrial pacemaker.

ECG:

1. Morphological changes in the P-wave (flattened, notched, or biphasic).
2. P–Q interval persistently change (not always).
3. R–R interval can vary.
4. QRS complexes are not changed.

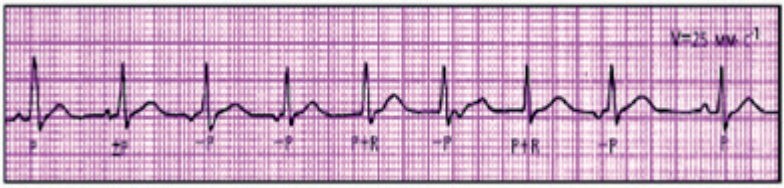


Figure 39. Wandering pacemaker

Escape beat

I type – Junctional escape beat

1. Rate 40–60 bpm (Fig. 40).
2. Rhythm: irregular in single junctional escape complex; regular in junctional escape rhythm.
3. P waves: Depends on the site of the **ectopic** focus. They will be inverted and may appear before or after the QRS complex, or they may be absent, hidden by the QRS.
4. P-R interval: If the P wave occurs before the QRS complex, the interval will be less than 0.12 seconds.
5. QRS Complex: Usually normal in duration and morphology, less than 0.12 seconds.

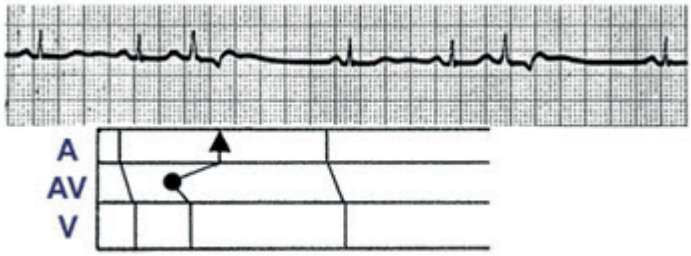


Figure 40. Junctional escape beat

II type escape beat from the branches of the bundle of His and Purkinje

1. Interval R– R before escape beat longer than normal interval (Fig. 41).
2. QRS complex wide, deformed.
3. P wave before complex QRS absent.

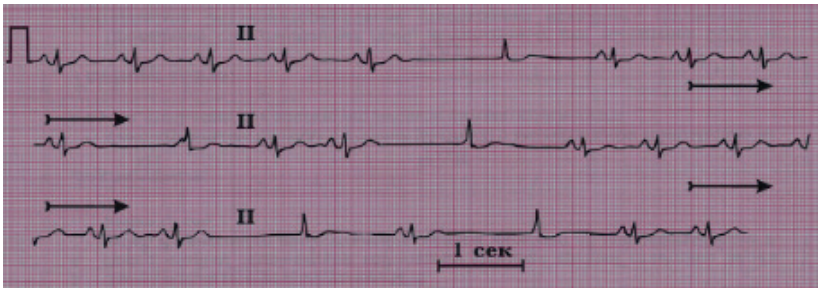


Figure 41. Escape beat from the branches of the bundle of His and Purkinje

Premature contractions, paroxysmal tachycardia, atrial fibrillation and flutter, ventricular fibrillation and flutter. Premature contractions

The terms "premature beat", "premature contraction", "premature systole", or "extrasystole" indicate that the atria, AV junction, or ventricle are stimulated prematurely. These premature beats are called "atrial premature beats" when they arise in some part of the atria. AV junctional premature beats arise in the AV junction. Ventricular premature beats arise in one of the branches of the bundle of His, Purkinje fibers, or the ventricular muscle.

Premature Atrial Contractions (PAC)

Originates in the atria. Occurs before the normal beat is expected. May occur in the healthy heart. Can be triggered by anxiety, fever, increased sympathetic input, caffeine and other stimulants, drug interactions, acute myocardial infarction, cardiac ischemia, valvular heart disease, and fever.

ECG signs PAC

1. A premature P wave is present (Fig. 42). It may be superimposed on the preceding T wave because it is premature.

2. The premature P wave is usually followed by QRS complex and T wave. Occasionally, it is not followed by QRS complex and T wave (blocked atrial premature beat).

3. The QRS and T waves that follow the premature P waves usually resemble the other QRS and T waves in the lead.

4. The P–R interval of the PAC is usually longer than the normal PR intervals.

5. The pause after PAC is usually incomplete; i. e., the PAC usually enters the sinus node and resets its timing, causing the next sinus P to appear earlier than expected.

6. The ventricular complex is usually normal but may be aberrant in from if the PAC coincides with the refractory phase of the previous ventricular beat. The aberrant QRS is called aberrant conduction.

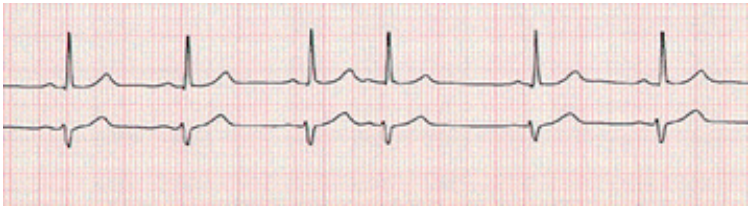


Figure 42. Premature atrial contractions

PACs can have **three different outcomes** depending on the degree of prematurity (i.e., coupling interval from the previous P wave), and the **preceding cycle length**. This is illustrated (Fig. 43) in the "ladder" diagram where normal sinus beats (P) are followed by three possible PACs; in the diagram, the refractory periods of the AV node and bundle branches are indicated by the width of the boxes.

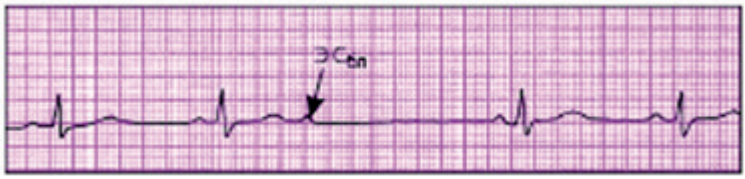


Figure 43. Blocked atrial premature beat

A "ladder" diagram (Fig. 44) is an easy way of conceptualizing the conduction of impulses through the heart, and the resulting complexes (i.e., P waves and QRS waves).

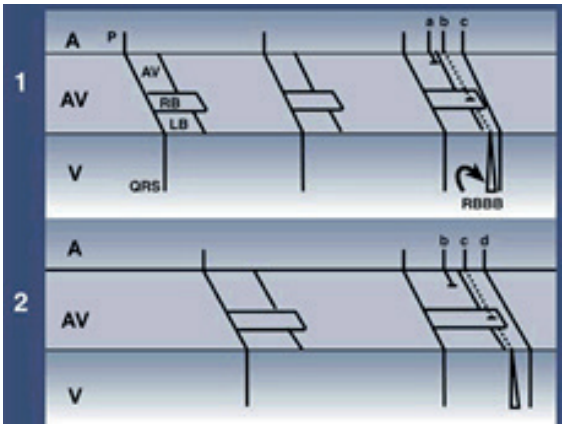


Figure 44. Three different outcomes of premature atrial contraction

1. **Nonconducted** (blocked); i. e., no QRS complex because the PAC finds AV node still refractory (see PAC labeled 'a' in the upper diagram 1).

2. **Conducted with aberration;** i. e., PAC makes it into the ventricles but finds one or more of the conducting fascicles or bundle branches refractory. The resulting QRS is usually wide, and is sometimes called an ***Ashman beat*** (see PAC 'b' in diagram 1).

3. **Normal conduction;** i.e., similar to other QRS complexes in the ECG. (See PAC 'c' in the diagram 1).

AV Junctional premature beats

1. In premature AV junction P wave is followed by a QRS and T wave (Fig. 45).
2. The AV junction P waves in aVR become upward. P waves in II, III, and aVF are downward. PR interval is usually less than 0.12 second if the P waves are before the QRS complexes. The P waves may appear after the QRS complexes or may be hidden within the QRS complex. An AV junctional premature beat is followed by a fully compensatory.

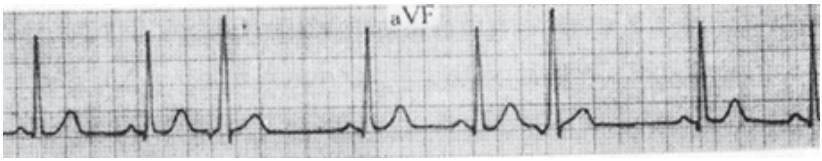


Figure 45. AV Junctional premature beats

Premature Ventricular Contraction (PVC)

1. Premature QRS complex is 0.12 second (Fig. 46, 47) or wider, and is aberrant, notched, or slurred. It is associated with a T wave that usually points in a direction opposite to the main deflection of the QRS complex.

2. The premature QRS complex is not preceded by a P wave.

3. PVC is often followed by a fully **compensatory pause** (the sum of the R-R intervals including the pre-premature beat and the post-premature beat interval equals the sum of two normal R–R intervals).

4. Multiply, PVC that arise from a single focus show a similar shape and usually a similar coupling interval (distance from the preceding normal QRS complex to the PVC) in any one lead.

5. Occasionally, at PVC will occur simultaneously with the apex of the preceding T wave. This is "R on T phenomenon". When this occurs, it may be a precursor of ventricular tachycardia.

6. In PVC from the left ventricular QRS reminds one of the right bundle-branch block in leads $V_{1,6}$.

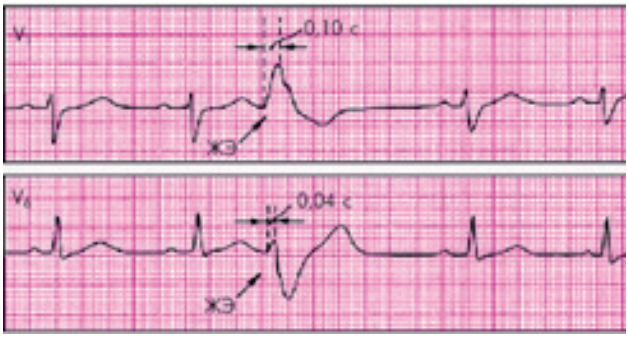


Figure 46. Premature ventricular contraction from left ventricular

In PVCs from the right ventricle, QRS reminds one of the left bundle-branch block in leads $V_{1,6}$.

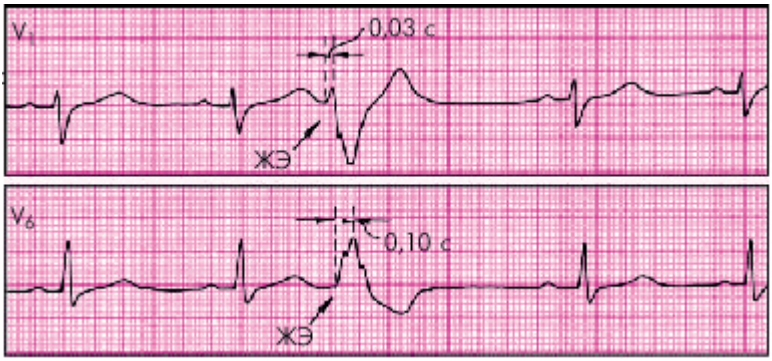


Figure 47. Premature ventricular contractions from the right ventricle

The Lown grading system of premature ventricular extrasystole represent in table 3.

Table 3. The Lown grading system of premature ventricular extrasystole

Lown Class	Description
I	Monoform (monofocal) VES < 30 / hr
II	Monoform (monofocal) VES ≥ 30 / hr
II	Multiform (multofocal) VES
IVa	2 consecutive (couplet)
IVb	≥ 3 consecutive (run of ventricular tachycardia)
V	"R on T" phenomenon

PVCs especially after myocardial infarction and Lown's class III–V are associated with increased risk for ventricular tachycardia, ventricular fibrillation, and sudden cardiac death.

PVCs may be monofocal (uniform, unifocal), means they arise from the same focus, on ECG they are identical in size, shape and direction (Fig. 48).

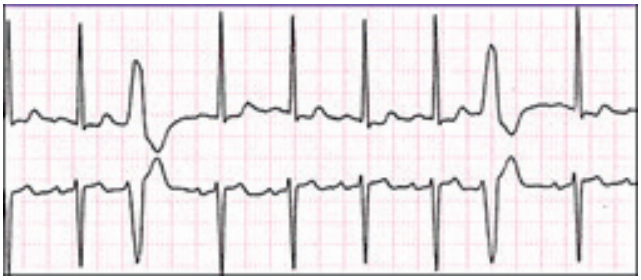


Figure 48. Monofocal premature ventricular contraction

Multifocal (multiform) PVCs differ in shape, size, and direction due to different focus sites (Fig. 49). Multiformed PVCs usually have the same coupling intervals (because they originate in the same ectopic site but their conduction through the ventricles differ. Multiformed PVCs are common in digitalis intoxication.

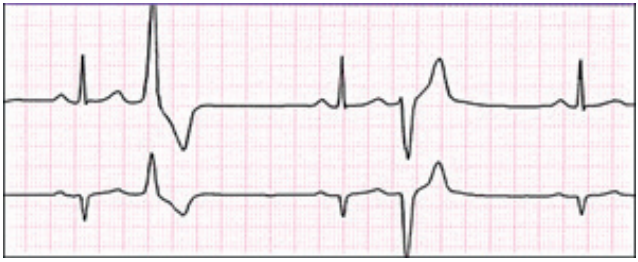


Figure 49. Multifocal premature ventricular contraction

PVCs may occur as isolated single events or as couplets (pairs) (Fig. 50), triplets, and salvos (3 and more PVCs in a row), also called ventricular tachycardias (Fig. 51).

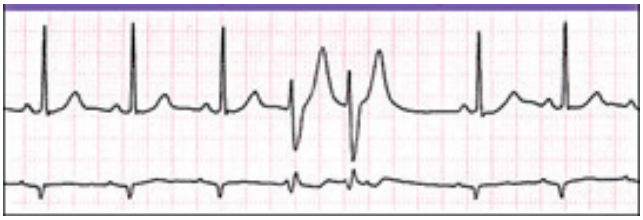


Figure 50. Coupled premature ventricular contraction

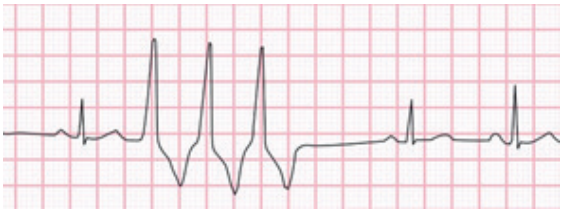


Figure 51. Runs of premature ventricular extrasystole (ventricular tachycardia)

PVCs may occur during the vulnerable period of ventricular repolarization (on or near the peak of the T wave) and called "R-on-T" phenomenon (Fig. 52). R-on-T PVCs may be especially life-threatening in an acute ischemic situation, because Stimulation of the ventricle at this time may initiate repetitive ventricular contractions, resulting in ventricular tachycardia or ventricular fibrillation.

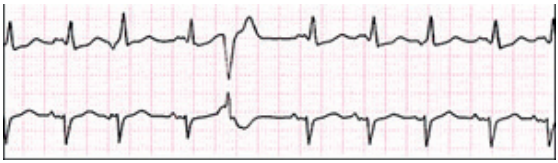


Figure 52. Premature ventricular contraction "R on T" phenomenon

PVCs can also be represented on ECG as **allorrythmias**: every other extrasystolic beat called bigeminal pattern, every third beat – trigeminal pattern, every fourth beat – quadrigeminal pattern (Fig. 53).

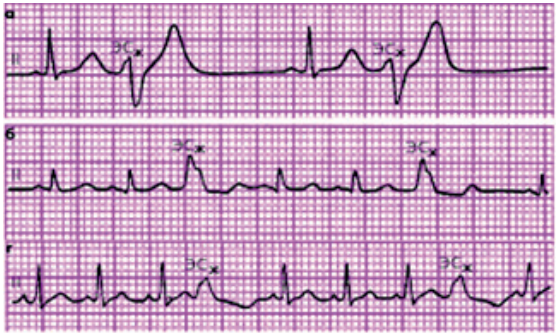


Figure 53. Different types of allorrythmias (bigemeny, trigemeny, and quadrigemeny)

Ectopic tachycardia

The paroxysmal tachycardia can be divided into two main groups:

1. Paroxysmal supraventricular tachycardia.
2. Paroxysmal ventricular tachycardia.

Paroxysmal atrial tachycardia

Paroxysmal atrial tachycardia (PAT) describes the condition when the P-waves are a result of a re-entry activation (circus movement) in the atria, usually involving the AV node. This leads to a high rate of activation, usually between 160 and 250 bpm/min. In the ECG the P-wave is regularly followed by the QRS-complex. The isoelectric baseline may be seen between the T-wave and the next P-wave.

ECG criteria:

1. Heart rate is regular with a rate of 160-250 bpm/minute (Fig. 54).
2. The QRS complex is usually normal and narrow.
3. P wave: may not always be different due to fast rate, frequently hidden in the preceding T wave.
4. Abrupt onset and abrupt ending.

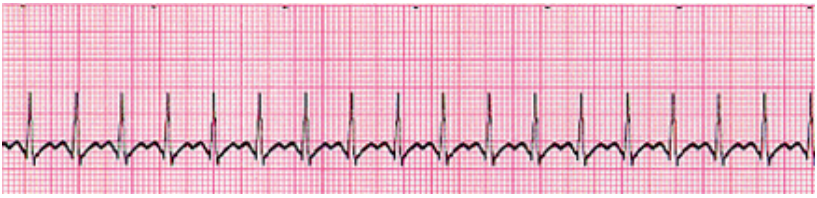


Figure 54. Paroxysmal Supraventricular Tachycardia on ECG tracings

Paroxysmal Junctional Tachycardia

Paroximal junctional tachycardia is caused by the sudden rapid firing a very irritable automaticity focus in the AV junction.

ECG criteria:

1. Heart rate is regular rhythm with a rate of 160–250 bpm / min (Fig. 55).
2. The QRS complex in form is usually normal.
3. The P wave is negative or absent.

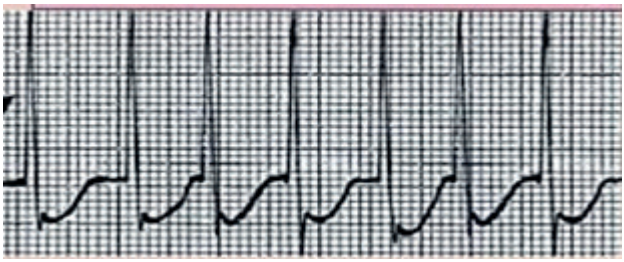


Fig. 55. Paroximal junctional tachycardia

Nonparoxysmal tachycardia: This usually begins as an accelerated rhythm but the heart rate gradually increases to 100–140 bpm. Heart rate is regular rhythm. There may be AV dissociation, or retrograde atrial capture may occur. The two most common causes are ischemia (usually from right coronary artery occlusion) and digitalis intoxication.

Paroxysmal ventricular tachycardia

Paroxysmal ventricular tachycardia (VT) is produced by a very irritable ventricular automaticity focus that suddenly paces in the 150–250 bpm.

ECG criteria:

1. The QRS complex ≥ 0.12 second, wide, aberrant, and are followed by aberrant ST segments and T waves (Fig. 56).
2. Ventricular rate regular rhythm or slightly irregular, and about 150–200 bpm.
3. P waves have no relation to the QRS complexes.
4. Fusion beats or ventricular capture are present.
5. PR interval: not measurable.

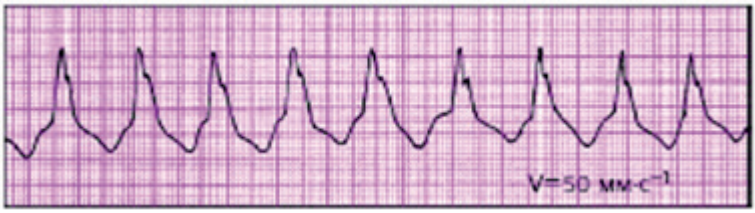


Figure 56. Ventricular paroxysmal tachycardia

Torsade de Pontes (pirouette-type tachycardia)

Torsade de Pontes (pirouette-type tachycardia) is a type of polymorphic VT characterized on electrocardiogram by oscillatory changes in amplitude of the QRS complexes around the isoelectric line. Torsades de Pointes is associated with QTc prolongation. The rhythm may terminate spontaneously or may be developed into ventricular fibrillation.

ECG is characterized by twisting of the QRS complexes around an isoelectric line (Fig. 57). Torsades de Pointes is triggered by a PVC occurring on a preceding T wave. The onset of Torsades de Pointes is often preceded by a run of short-long-short R–R intervals.

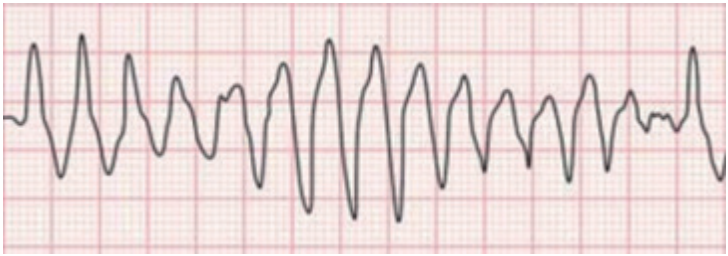


Figure 57. Torsade de Pontes (pirouette-type tachycardia)

Flutter and Fibrillation

The flutter and fibrillation arise from excitable ectopic focus in the atria and ventricle and with a rapid rate and appropriate conduction block. Thus, they are easily caused by a re-entry.

Atrial Flutter

Atrial flutter (AF) originates in an atrial automaticity focus that fires at a rate of 250–350 bpm. Mostly this type of arrhythmia can occur in mitral valve defects, hyperthyroidism, myocardial infarction, chronic obstructive lung disease, obstructive sleep apnea, diabetes, etc.

ECG criteria:

1. There are no P waves, the presence of saw-tooth flutter waves (F waves). F waves are always uniform in size, shape, and frequency (Fig. 58).

2. Regular atrial rhythm with a rate of 25–-350 or irregular depending upon the AV conduction properties and AV node slowing drugs on board (e.g., digoxin, beta-blockers).

3. Ventricular rhythm – regular if conduction is regular (AV node conducts impulses to the ventricles at a 2:1, 3:1, 4:1, or greater).

4. Absence of isoelectric line.

5. The QRS complex is normal.

6. PR interval: not measurable.

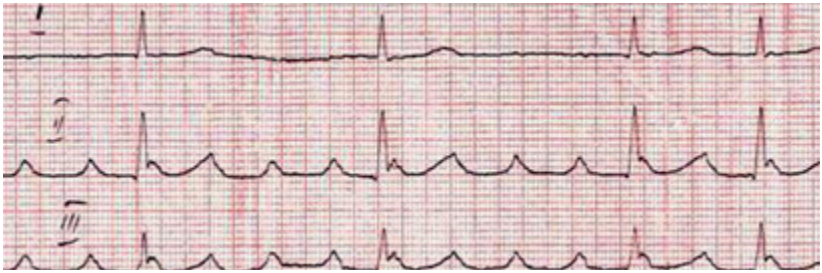


Figure 58. Atrial Flutter

Atrial Fibrillation

Atrial fibrillation (AFib) is caused by the continuous rapid-firing of multiple atrial automaticity foci (rate of 350-450 bpm). No single impulse depolarizes the atria completely, and only an occasional, random atrial depolarization reaches the AV node to become conducted to the ventricles; this produces an irregular ventricular rhythm. The causes are the same as for AF.

ECG-criteria:

1. Absence of P waves, P waves replaced by “f” waves which are irregular in size, shape, amplitude (Fig. 59).

2. Irregularly irregular rhythm. Atrial heart rate: 350–450 bpm, ventricular –depending on conduction.

3. QRS complex is normal.

4. PR interval: not measurable.

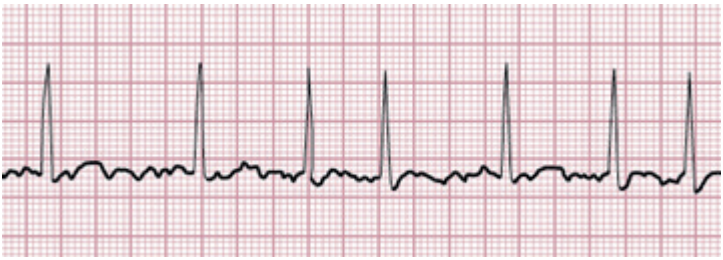


Figure 59. Atrial Fibrillation

In AFib, and in AF, the following are observed in the frequency of the ventricular rhythm during wakefulness: normosystolic variant (HR 60–100 bpm); tachysystolic variant (HR more than 100 bpm); and bradysystolic variant (HR less than 60 bpm).

Ventricular Flutter

Ventricular flutter is produced by singular ventricular automaticity focus fires at an exceptionally rapid rate of 250–350 pm. It produces a rapid series of smooth sine waves of similar amplitude.

Causes: organic diseases of the heart.

ECG:

1. No identifiable P waves, QRS complexes, or T waves.
2. Smooth sine ventricle waves (similar in size, shape, and amplitude).
3. Frequency from 250 to 300 bpm (Fig. 60).
4. Intervals are equal to each other.
5. There is no isoelectric interval.

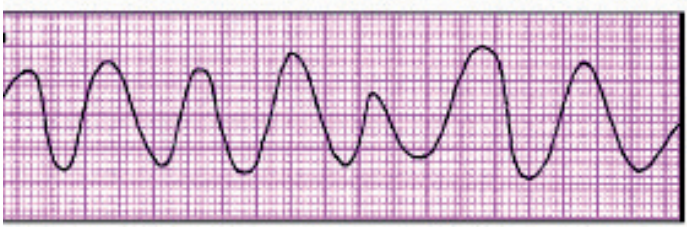


Figure 60. Ventricular Flutter

Ventricular Fibrillation

Ventricular fibrillation is caused by rapid-rate discharges from many irritable, parasystolic ventricular automaticity foci, producing an erratic, rapid twitching of the ventricles (350–450 pm). Causes: organic diseases, congenital, etc.

ECG-criteria:

1. Rhythm: irregular-coarse or fine, waveform varies in size and shape (Fig.61)
2. No discernible complexes
3. Absence of isoelectric line.

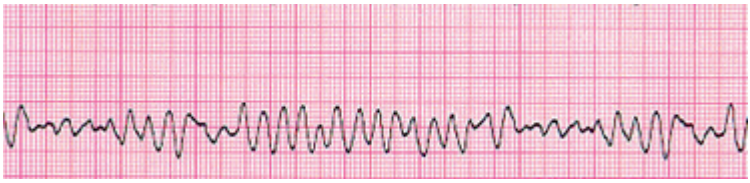


Figure 61. Ventricular Fibrillation

Ventricular asystole

Ventricular asystole – cessation of electrical and mechanical activity of the heart. Asystole typically results from decompensation of prolonged ventricular fibrillation arrest. Additionally, attempted defibrillation of ventricular tachycardia or ventricular fibrillation can precipitate asystole. However, any cause of cardiac arrest can eventually result in asystole if not promptly treated.

ECG: lack of P-waves, QRS complexes, and T-waves. Presense of isoelectric “flat” line (Fig. 62).

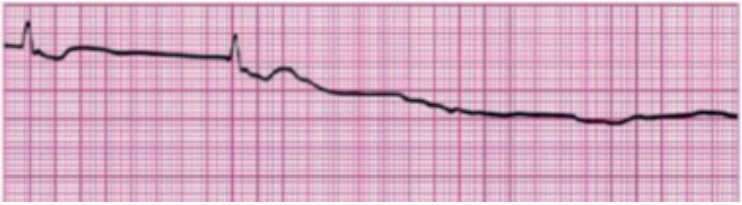


Figure 62. Ventricular asystole

Electromechanical dissociation

Electromechanical dissociation (pulseless electrical activity) - the absence of mechanical activity of the heart with stored electrical activity (Fig. 63).

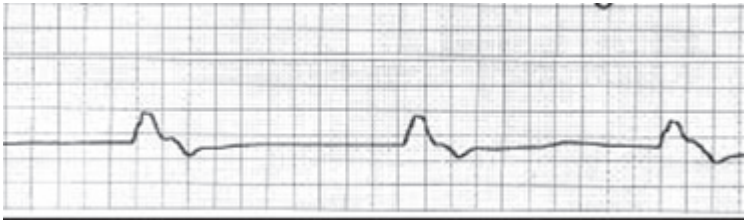


Figure 63. Electromechanical dissociation

CHAPTER 4.
ECG IN CONDUCTION ABNORMALITIES

Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and their fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction **(1st degree)**, intermittent conduction failure **(2nd degree)**, or complete conduction failure **(3rd degree)**. In addition, 2nd degree heart block occurs in two varieties: **Mobitz Type I (Wenckebach)** and **Mobitz Type II**. The term **exit block** is used to identify conduction delay or failure immediately distal to a pacemaker site. Sino-atrial (SA) block is an exit block.

Classification of Conduction disorder

1. Sino-atrial (SA) block
1st degree SA block is impossible detect on ECG
2nd degree SA block
Mobitz Type I (Wenckebach)
Mobitz Type II
3rd degree (Complete) SA block is impossible determine on ECG
2. Interatrial block (bundle Bachman)
1st degree Interatrial block
2nd degree Interatrial block
Mobitz Type I (Wenckebach)
Mobitz Type II
3rd degree (Complete) Interatrial block
3. Atrio-ventricular block (AV)
1st degree AV block
2nd degree AV block
Mobitz Type I (Wenckebach)
Mobitz Type II

High grade AV block: 2:1, 3:1; 4:1.

3rd degree or Complete (proximal and distal type) AV block.

4. Bundle branch blocks

Right bundle branch block (complete and incomplete).

Left bundle brunch block (complete: anterior and posterior hemiblock; and incomplete).

Siniatrial block

2nd degree SA block – the only SA block that can be recognized on ECG (i.e., intermittent conduction failure between the sinus node and the right atrium). There are two types, although because of sinus arrhythmia, they may be hard to differentiate. Furthermore, the differentiation on ECG is interesting but not clinically important.

2nd degree SA block Mobitz type I (Wenckebach): the following 3 rules represent the classic rules of Wenckebach, which were originally described for **Type I SA block** (Fig. 64, 65). The rules are the result of decremental conduction where the increment in conduction delay for each subsequent impulse gets smaller until conduction failure finally occurs. This declining increment results in the following findings:

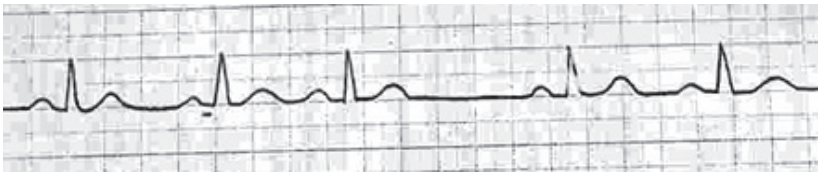


Figure 64. Second degree sinoatrial block

1. PP intervals gradually shorten until a pause occurs (i. e., the blocked sinus impulse fails to reach the atria).
2. The pause duration is less than the two preceding PP intervals.
3. The PP interval following the pause is greater than the PP interval just before the pause.

2nd degree SA block Type II:

PP intervals fairly constant (unless sinus arrhythmia present) until

1. Conduction failure occurs (Fig. 66).
2. The pause is approximately twice the basic PP interval.

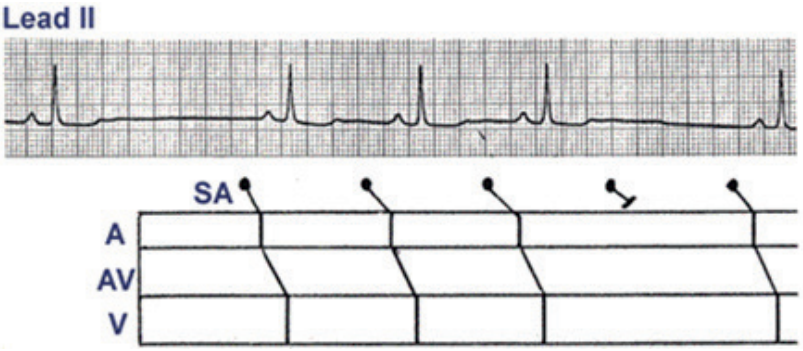


Figure 65. Sinoatrial block type I

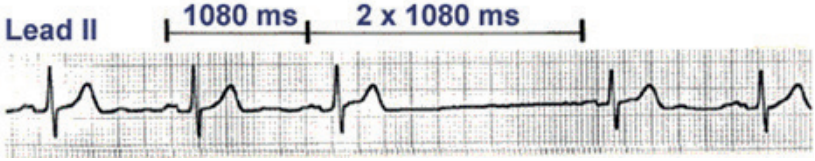


Figure 66. Sinoatrial block type II

Atrioventricular conduction abnormalities
2nd degree atrioventricular (AV) block

1. PR interval > 0.20 sec (Fig. 67).
2. All P waves conduct to the ventricles.
3. QRS complex is a normal.

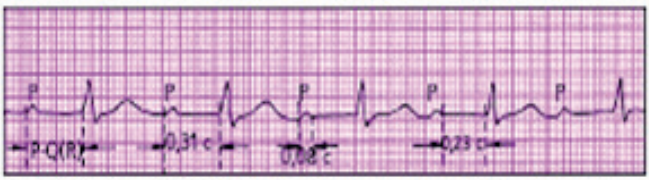


Figure 67. First-degree AV block

Second degree AV blocks

This is prolongation of the P–R interval with intermittent failure of conduction of atrial impulses to the ventricles, causing “dropped” beats. The condition is usually due to impaired AV nodal conduction.

There are two types:

AV block 2nd degree Mobitz type I (Wenckebach's phenomenon):

1. There is progressive lengthening of the P–R interval (Fig. 68) following each atrial impulse, until an atrial impulse fails to be conducted to the ventricles.

2. Complex QRS is a normal.

3. After long pause occur minimal interval PQ.

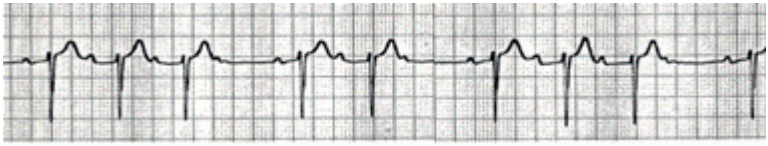


Figure 68. AV block 2nd degree Mobitz type I

AV block 2nd degree Mobitz type II

In AV block 2nd degree Mobitz type II characterized intermittent failure of atrial impulses conduction to the ventricles without progressive lengthening of the P-R interval, so on ECG will be dropped QRS complex with constant PR or PQ (Fig. 69).

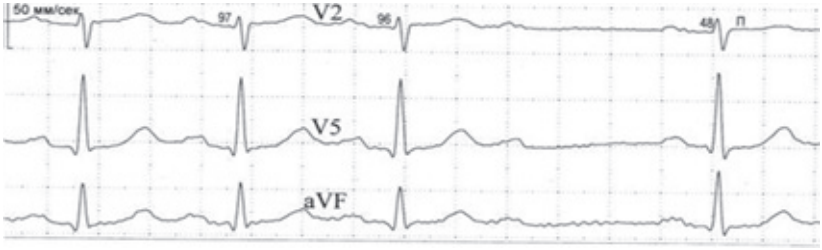


Figure 69. AV block 2nd degree Mobitz type II

High grade AV block

High grade AV block is intermediate between second and third degree.

ECG criteria:

1. The number of P wave is more than quantity of complexes QRS.

2. There are two or more “blocked P” after which complex QRS does not follow.

3. Complex QRS is not changed (Fig. 70).

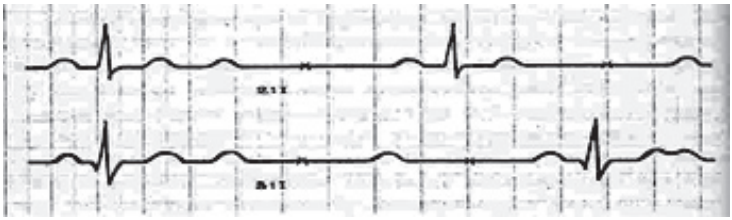


Figure 70. High grade AV block

Third-degree or complete heart block

Third-degree AV block can occur at the AV node or infra-nodally in the His-Purkinje system. In nodal block, the new, subsidiary distal pacemaker will arise above, or in the bundle of His.

Proximal type of complete heart block

1. QRS complexes are narrow (Fig. 71).
2. R-R interval equal to each other.
3. Complete AV dissociation (atrial and ventricular rate and rhythm are independent of one another).
4. Ventricular HR > 40 bpm (the pacemaker focus usually in the AV node).

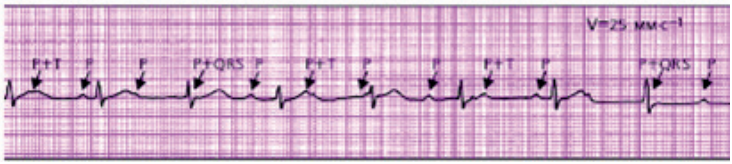


Figure 71. Complete AV block proximal type

Distal type of complete heart block

1. QRS complexes will be wide.
2. R-R interval equal to each other (Fig. 72).
3. HR < 40 bpm (AV blocks occurring in or below the His bundle usually have a subsidiary pacemaker in the right or left bundle branches).

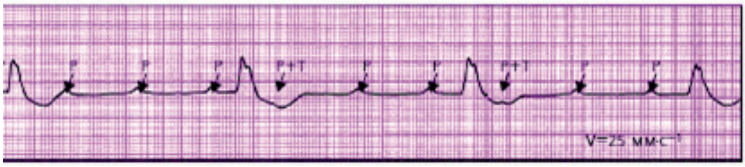


Figure 72. Complete AV block distal type

Atrial fibrillation and complete heart block (Frederick's syndrome)

Frederick's syndrome is a rare syndrome characterized by atrial fibrillation or atrial flutter and complete heart block occurring simultaneously.

ECG criteria of Frederick's syndrome (Fig. 73):

1. Absent P waves.
2. Presence of atrial fibrillation f waves or atrial flutter waves.
3. Regular ventricular rhythm 40–60 bpm.
4. Third degree AV block characterized by AV dissociation.

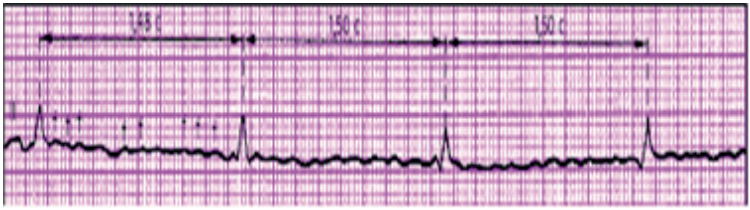


Figure 73. Frederick's syndrome

Wolff-Parkinson-White Syndrome

Wolff–Parkinson–White (WPW) syndrome is a ventricular pre-excitation syndrome due to an accessory conduction pathway (Fig. 74). The presence of WPW syndrome increases the likelihood of paroxysms of AFib and AF. In WPW syndrome during AFib and AF, the conduction of impulses to the ventricles occurs primarily along additional pathways (bundle of Kent or Mahaim fibers) and only partially through the normal conduction system of the heart.

ECG criteria of WPW:

1. Short PR interval (<0.12s).
2. Slurred upstroke at the beginning of the QRS complex (delta wave).
3. Wide QRS complex greater than 120 ms.
4. Secondary ST-T changes due to the altered ventricular activation sequence.

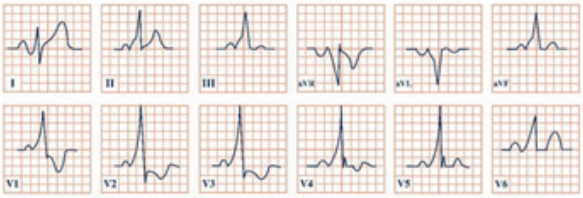


Figure 74. Wolff-Parkinson-White syndrome

The QRS complex can take different forms depending on the location of the accessory pathway, diving WPW into two types.

Type A is characterized by a positive delta wave in V1; positive QRS complex in leads V1, II, III, and aVF; and negative or isoelectric Q waves in the lateral leads.

Type B is characterized by negative delta waves and positive upright QRS complexes in leads II, III, and aVF.

Lown-Ganong-Levine syndrome

Lown-Ganong-Levine syndrome (LGL) is a syndrome (Fig. 75) of pre-excitation of the ventricles due to an accessory pathway providing an abnormal electrical communication from the atria to the ventricles.

1. Interval P–Q is short.
2. Complex QRS is a normal.

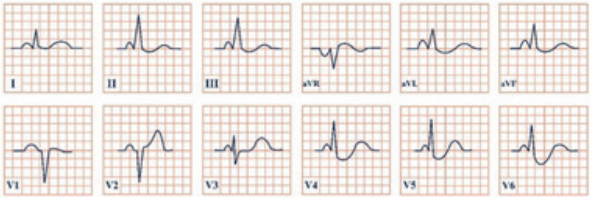


Figure 75. Lown–Ganong–Levine syndrome

CHAPTER 5.
INTERVENTRICULAR BLOCKS

The ventricular conduction system is composed of two major divisions: right bundle branch block and left bundle branch block.

Complete Left Bundle Branch Block (LBBB)

1. QRS 0.12 sec or more (Fig. 76).
2. Absent q waves in I, V_5, and V_6.
3. Wide, notched, or slurred R waves in V_{5-6} with depressed ST segments, and downward T waves.
4. Wide QS or rS patterns with elevated ST segments and upward T waves in $V_{1–2}$.

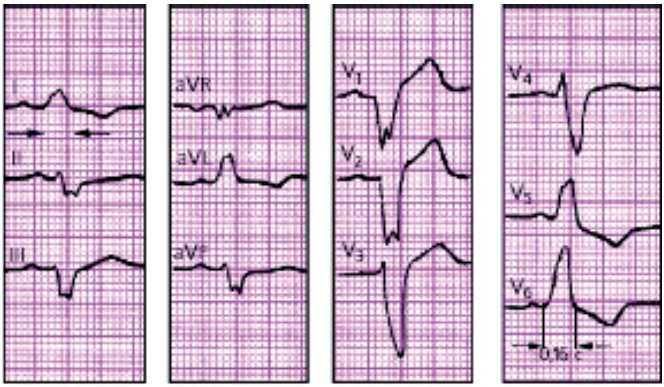


Figure 76. Complete left bundle branch block

Incomplete left bundle branch block

In incomplete LBBB the pattern is similar, but the QRS width is less than 0.12 second (Fig. 77). R wave notched and absent q wave in V_{5-6}.

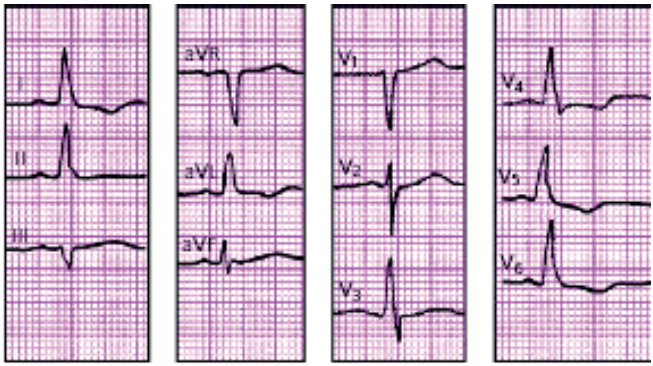


Figure 77. Incomplete left bundle branch block

Left Anterior Fascicular Block

1. Left axis deviation in the frontal plane, usually -45 to -90 degrees (Fig. 78).
2. rS complexes in leads II, III, aVF.
3. Small q-wave in leads I and/or aVL.
4. R-peak time in lead aVL >0.04s, often with slurred R wave downstroke.
5. QRS duration usually <0.12s unless coexisting right bundle brunch block (RBBB).

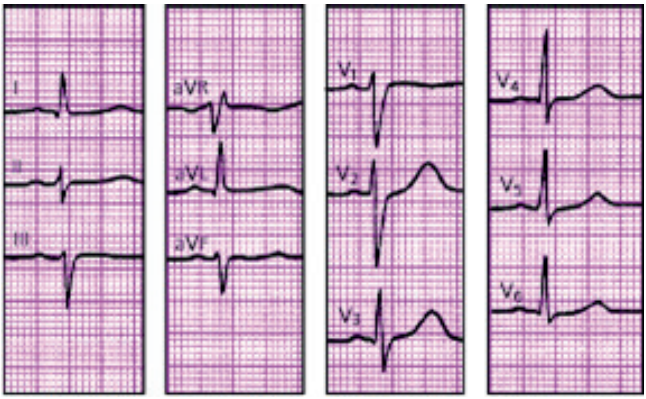


Figure 78. Left Anterior Fascicular Block

6. Usually see poor R progression in leads V_1-V_3 and deeper S waves in leads V_5 and V_6.
7. May mimic LVH voltage in lead aVL, and mask LVH voltage in leads V_5 and V_6.
8. $R_I > R_{II} > R_{III}$.
9. $S_{III} > R_{III}$.
10. $S_{II} > R_{II}$.
11. $S_{III} > S_{II}$.
12. $S_{aVF} > R_{aVF}$.
13. $R_{aVR} > Q(S)_{aVR}$.

Left Posterior Fascicular Block

1. Right axis deviation in the frontal plane (usually > +120 degrees).
2. rS complex in lead I (Fig. 79).
3. qR complexes in leads II, III, aVF, with R in lead III > R in lead II.
4. QRS duration usually <0.12s unless coexisting RBBB.
5. $R_{III} > R_{II} > R_I$.
6. $S_I > R_I$.
7. $R_{aVR} > Q\ (S)_{aVR}$.
8. deep S wave in V_5, V_6.

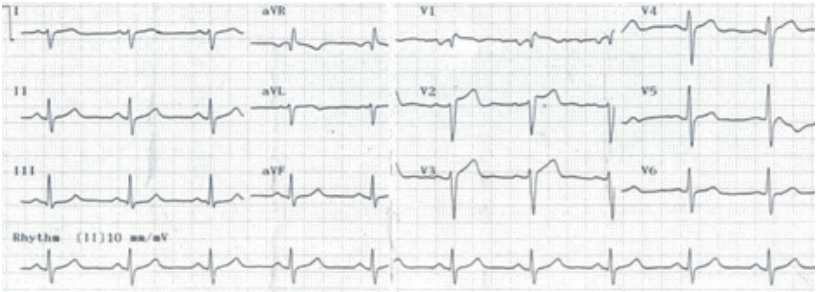


Figure 79. Left Posterior Fascicular Block

Complete right bundle branch block

1. QRS duration >0.12 ms (Fig. 80).
2. Close examination of QRS complex in various leads reveals that the terminal forces (i. e., 2nd half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized after the left ventricle.
3. Terminal R' wave in lead V_1 (usually see rSR' complex) indicating late anterior forces.
4. Terminal S waves in leads I, aVL, V_6 indicating late rightward forces.
5. Terminal R wave in lead aVR indicating late rightward forces.
6. The frontal plane QRS axis in RBBB should be in the normal range (i.e., -30 to +90 degrees). If left axis deviation is present, think about LAFB, and if right axis deviation is present, think about LPFB in addition to the RBBB.

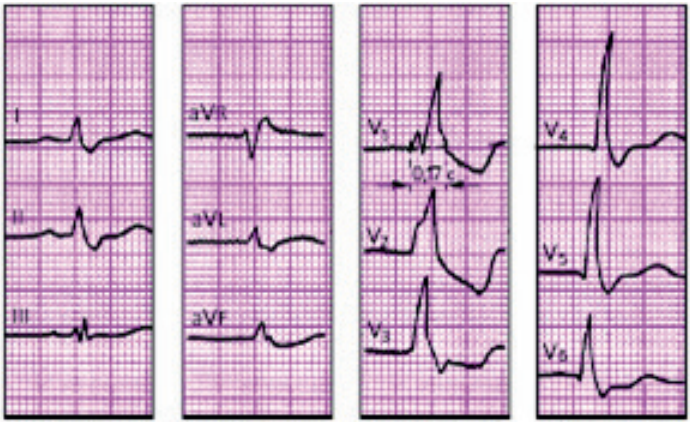


Figure 80. Complete right bundle branch block

Incomplete right bundle branch block

1. Incomplete RBBB has a QRS duration of 0.10-0.12s with the same terminal QRS features. This is often a normal variant (Fig. 81).

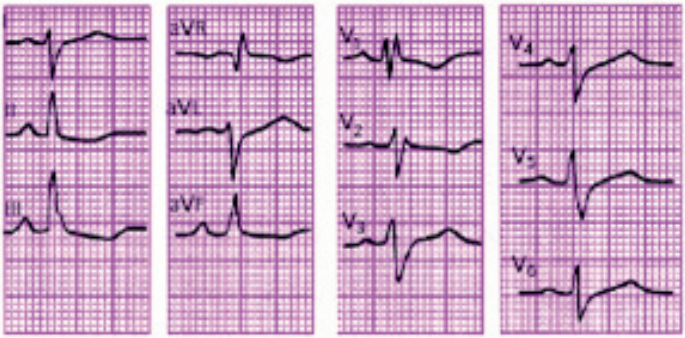


Figure 81. Incomplete right bundle branch block

2. The "normal" ST–T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i. e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards. If the ST–T waves are in the same direction as the terminal QRS forces, they should be labeled primary ST–T wave abnormalities.

Bifascicular Blocks

1. RBBB plus (Fig. 82) either LAFB (common) or LPFB (uncommon).
2. Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.).

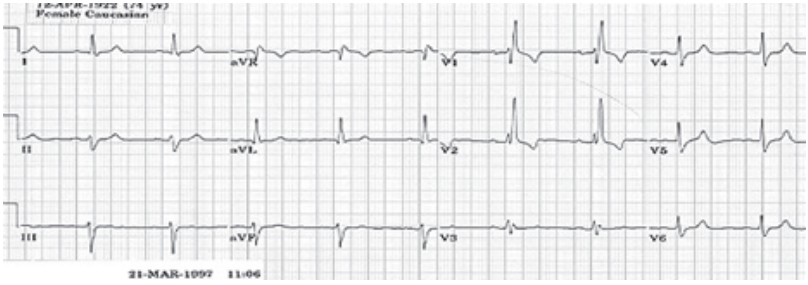


Figure 82. Right bundle branch block and left anterior fascicular block

Anterior Fascicular Block

1. RBBB plus LAFB occur sign RBBB and left axis deviation in frontal plane.
2. QRS duration >0.12s
3. In leads V_1, V_2 complex QRS deform (rsR', rSR', rSr') with wide R wave'.
4. $\angle\alpha$<-60°, $R_I > R_{II} > R_{III}$, $S_{III} > R_{III}$, $S_{avF} > R_{avF}$, $S_{II} > R_{II}$, $S_{III} > S_{II}$, $R_{avR} > Q_{avR}$.

Posterior Fascicular Block

1. RBBB plus LPFB occur sign RBBB and right axis deviation in frontal plane deeply appear.
2. QRS duration >0.12 s.
3. In leads V_1, V_2 complex QRS deform (rsR', rSR', rSr') with wide R wave', in leads V_5, V_6 wide S wave.
4. $\angle\alpha \geq +120°$, $R_{III} > R_{II} > R_I$, $S_I > R_I$, $R_{avR} > Q_{avR}$.

CHAPTER 6.
ECG IN ISCHEMIC HEART DISEASE

In ischemic heart disease (IHD) ischemia, injury, and necrosis can be developed in cardiac muscle depending on expressiveness and character of coronary arteries damage. Each process in a myocardium has ECG signs.

Ischemia is a transitory disturbance of coronary arteries. T wave and ST segment on ECG reflect changes.

In ischemia of subepicardial zone (Fig. 83) transitory negative symmetrical T wave appear on ECG.

Figure 83. Subendocardial ischemia (transitory negative symmetrical T wave)

Horizontal or downsloping (Fig. 84) ST segment depression (below the isoline) on 1 mm and duration ≥ 0,08 sec or ST segment elevation above the isoline > 1mm ischemia of a myocardium.

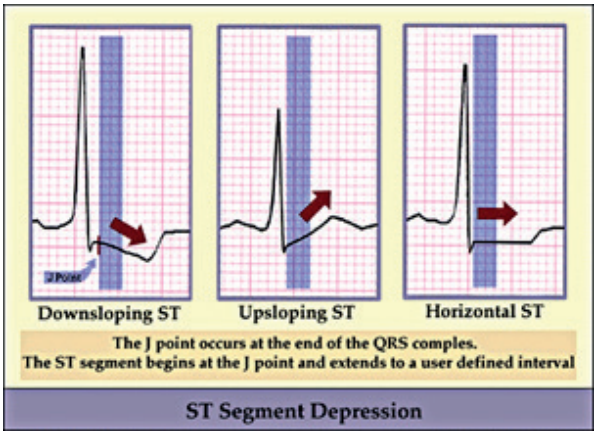


Figure 84. Different types of ST depression

ECG changes in acute myocardial infarction

Injury

In prolonged ischemia > 30 minutes, it leads to more changes in a myocardium, accompanied by infringements of biochemical processes in a cardiac muscle that leads to infringements of processes repolarization longer period. On an ECG this process finds reflection in change of ST segment and T wave.

In subepicardial injury (transmural injury or infarction) ST elevation appear on ECG (Fig. 85).

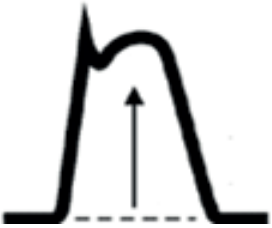


Figure 85. Subepicardial injury on ECG

If acute coronary syndrome is suspected, a 12-lead ECG should be recorded within 10 minutes of initial contact with medical personnel

and repeated within the first hours, after 24 hours, and during recurrence of symptoms. STEMI is characterized by the occurrence of ST segment elevation in at least two consecutive (conjugate) leads, which is assessed relative to the isoline at the level of J point. In leads V_2–V_3, ST segment elevation ≥ 2 mm in men over 40 years of age is diagnostically significant, ≥ 2.5 mm in men under 40 years of age, ≥ 1.5 mm in women, regardless of age. In all other chest and standard leads, ST segment elevation ≥ 1 mm is considered diagnostically significant (Table 4).

These criteria do not apply to cases where the ECG shows complete LBBB or severe LVH, in which ST segment elevation in the right precordial leads is secondary and not related to ischemia.

Table 4. ECG signs of acute ischemia (in the absence of LVH or LBBB)

Signs of acute ischemia	**ECG criteria**
ST elevation	New ST elevation at the J point (the junction between the termination of the QRS complex and the beginning of the ST segment) in two contiguous leads ≥ 0.25мV in men < 40 years (V2-V3); ≥ 0.2 мV in men ≥40 years (V2-V3); ≥ 0.15 мV in women (V2-V3) and / or ≥ 0.1 мV in other leads.
ST depression and T wave inversion	New horizontal or downslope depression ST ≥ 0.05 мV in two contiguous leads; and / or T wave inversion ≥ 0.1 мV in two contiguous leads with marked R wave or with R/S ratio >1.
Pathological Q wave	Any Q wave in V2-V3. Q wave ≥0.03 sec and ≥0.1мV or QS in leads I, II, aVL, aVF, V4-V6, in two contiguous leads. R wave ≥0.04 sec in V1-V2 and R/S ratio ≥ 1 with concordant positive T wave in the absence of conduction disorders.

With the development of transmural ischemia in the posterior wall area, conventional leads do not reveal ST segment elevation; a high R and T wave may appear in leads V_1–V_3 with ST segment depression below the isoline by 0.5 mm or more. To detect ST segment elevation, it is necessary to record additional leads V_7–V_9, for which chest electrodes are installed at the level of leads V_4–V_6, respectively, along the posterior axillary, scapular and paravertebral lines (Fig. 86). Diagnostically significant ST segment elevation in these leads is ≥ 0.5 mm (≥ 1 mm in men under 40 years of age).

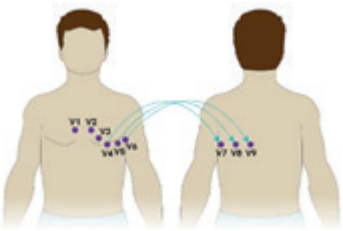


Figure 86. Registration of ECG in V_7–V_9 leads for the diagnosis of focal changes in the posterior wall of the left ventricle

If the right ventricle MI is suspected (usually with infarction of the inferior wall, less often in isolation), it is necessary to register the right chest leads RV_3 and RV_4, while the chest electrodes are installed as leads V_3 and V_4, but on the right half of the chest (Fig. 87). ST segment elevation ≥ 1 mm is diagnostical.

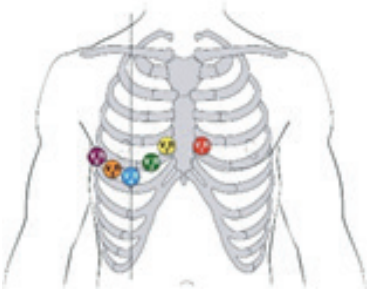


Figure 87. Registration of ECG in RV_3–RV_4 leads for the diagnosis of focal changes of the right ventricle

One of the most dangerous lesions is occlusion of the main trunk of the left coronary artery, manifested mainly by depression of the ST segment, which is recorded in 8 or more chest and standard leads, and elevation ≥ 1 mm is detected only in lead aVR (sometimes in V_1) (Fig. 88).

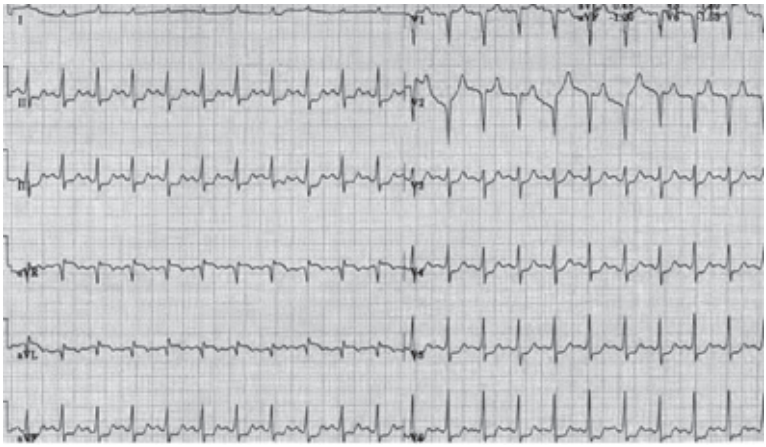


Figure 88. Occlusion of the left main coronary artery

Registration of the first (or presumably for the first time) detected complete LBBB **(new LBBB)** in a patient with symptoms of ischemia is the basis to regard it as a manifestation of acute coronary syndrome with ST elevation (Fig. 89).

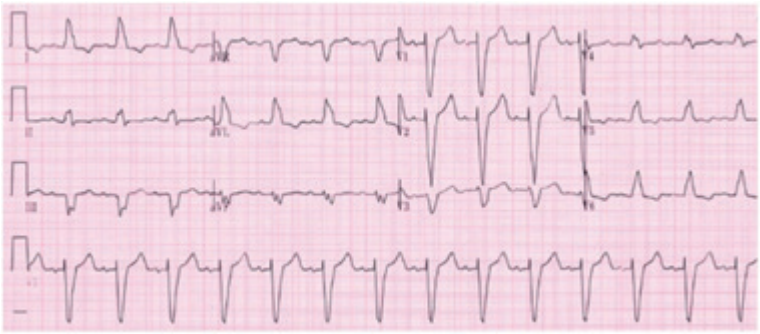


Figure 89. Complete left bundle branch block

It is often difficult to identify MI in patients with existing complete left bundle branch block, so the Sgarbossa criteria have been proposed.

Sgarbossa's criteria are used to diagnose acute myocardial infarction in complete LBBB or ventricular pacing.

Sgarbossa criteria include 3 criteria (Fig. 90):

1. ST segment elevation more than 1 mm, concordant with the QRS complex – 5 points.

2. ST segment depression greater than 1 mm in V_1, V_2 or V_3 – 3 points.

3. ST segment elevation more than 5 mm, discordant to the QRS complex – 2 points.

If ≥3 points = 90% specificity for MI (sensitivity 36 %).

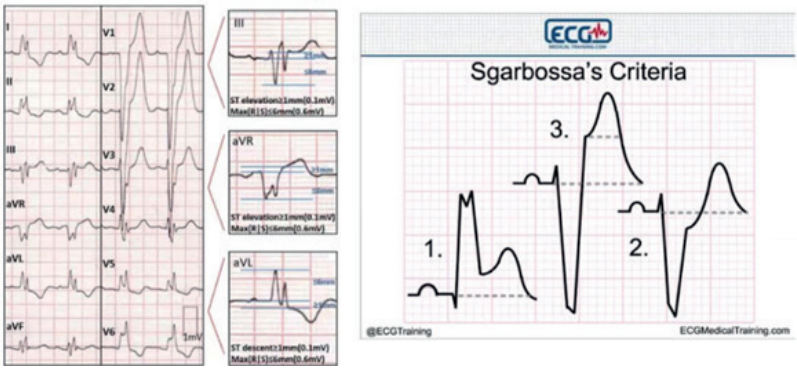


Figure 90. Sgarbossa's criteria for MI in left budle branch block
(Image: ECGMedicalTraining.com)

According to the last data much more specific and sensitive are **Smith-Modified Sgarbossa Criteria** (Fig. 91):

1. Concordant ST elevation ≥ 1 mm in ≥ 1 lead.

2. Concordant ST depression ≥ 1 mm in ≥ 1 lead of V_1–V_3.

3. Proportionally excessive discordant STE in ≥ 1 lead anywhere with ≥ 1 mm ST elevation, as defined by **≥ 25%** of the depth of the preceding S-wave.

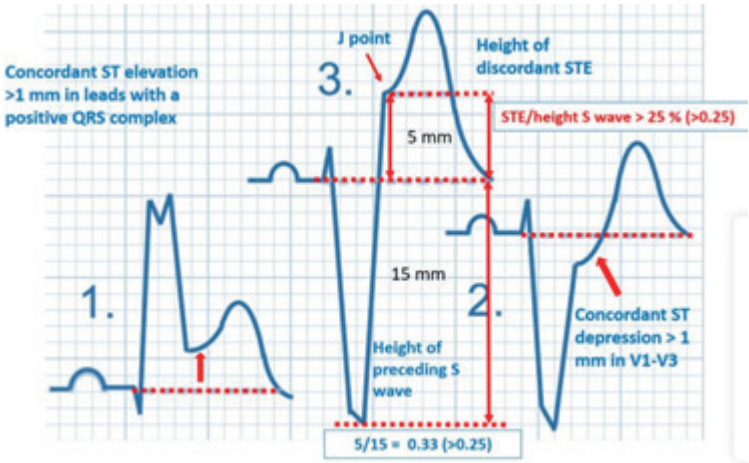


Figure 91. Smith-Modified Sgarbossa's criteria for MI in left budle branch block

(Image: https://clincasequest.hospital)

With persistent ischemia and injury caused by the cessation of coronary circulation, morphological changes in the myocardium develop in the form of necrosis of the heart muscle – the development of acute myocardial infarction (AMI). Changes of QRS and the appearance of pathological Q waves on ECG reflect necrosis of cardiac muscle.

MI Stages of myocardial infarction

In the development of MI, there are **4 main stages: hyperacute, acute, subacute and old stage.** In an uncomplicated course, the duration of the acute stage does not exceed 7–10 days and in its development passes through the phases of ischemia (from several minutes to 1–2 hours), ischemic damage (from 1 hour to 1 day, in the presence of residual blood flow – up to 3 days) and necrosis.

In the acute stage of non-ST-elevation acute coronary syndrome, a tall, pointed T wave (ischemia) and ST segment elevation (damage) are formed. The ST segment has a horizontal, concave, convex or oblique shape and can merge with the T wave, forming a monophasic curve. In leads characterizing the myocardial zones opposite to the infarction, reciprocal depression of the ST segment can be recorded.

Acute stage. A pathological Q wave or QS complex appears. A Q wave with a duration of more than 0.03 s and an amplitude of more than 1/4 of the amplitude of the R wave in leads I, aVL, V_1–V_6 or more than 1/2 of the amplitude of the R wave in leads II, III and aVF is considered pathological. The R wave may decrease or disappear, and in the opposite leads it may increase.

Subacute stage. The ST segment returns to the isoline, the presence of a pathological Q or QS wave, and a negative T wave is formed.

Stage of scarring (post-infarction cardiosclerosis). The amplitude of the negative T wave decreases, and over time it becomes isoelectric and positive. ST segment on isoline. The Q wave is usually preserved, but in some cases it may decrease or disappear (regress) due to compensatory hypertrophy of healthy myocardium. These ECG changes are characteristic of myocardial infarction with a Q wave (large focal, transmural). MI without a Q wave (fine-focal, intramural, subendocardial) is diagnosed based on dynamic changes in the ST segment and T wave.

Topical diagnostics of myocardial infarction

Typical changes are pathological Q wave ($\geq$ 0.04 s and > 1/4 subsequent R wave) or QS complex, RS-T elevation, negative (coronal) T wave and reciprocal mirror changes in leads opposite the localization of MI.

Localization MI CHANGE ECG (THE EXISTENCE OF QR or QS)

Different types of MI depending on localization is represented below (Fig. 92–95).

Table 5. Topical diagnostics of myocardial infarction

Leads	Localization of MI	Involved coronary artery
Septal	V_1–V_2	LAD
Anteroseptal	V_1–V_4	LAD
Anterior	V_3–V_4	LAD
Extensive anterolateral	V_1–V_6, I, aVL	LAD
Anterolateral	V_3–V_6, I, aVL	LCX or LAD diagonal branch
Lateral	V_6, I, aVL	LCX or LAD diagonal branch
High lateral	I, aVL	LCX or LAD diagonal branch
Inferior lateral	II, III, aVF, V_4–V_6	LCX
Inferior	II, III, aVF	RCA or LCX
Posterior	V_7-V_9	RCA or LCX
Right ventricle	RV_3–RV_6	RCA

Note: LAD – left anterior descending artery, LCX – left circumflex artery, RCA – right coronary artery.

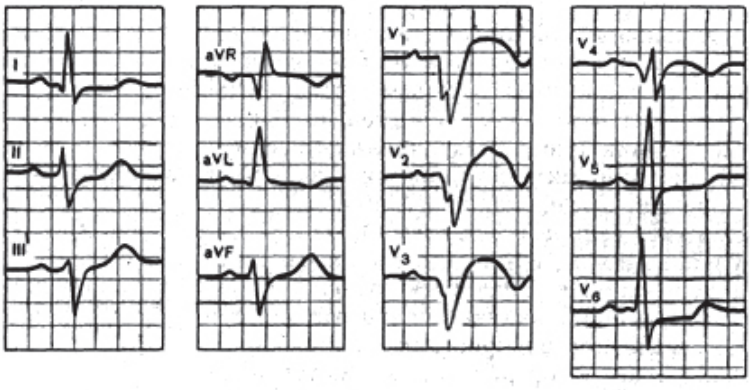


Figure 92. Anterior-septal MI

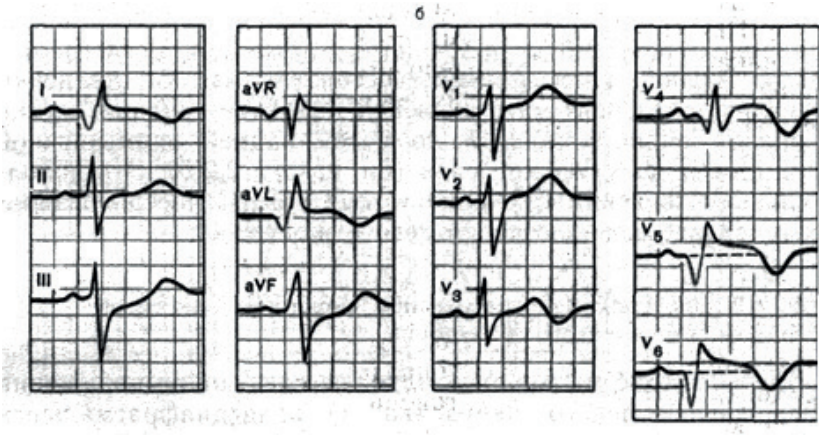


Figure 93. Lateral wall MI

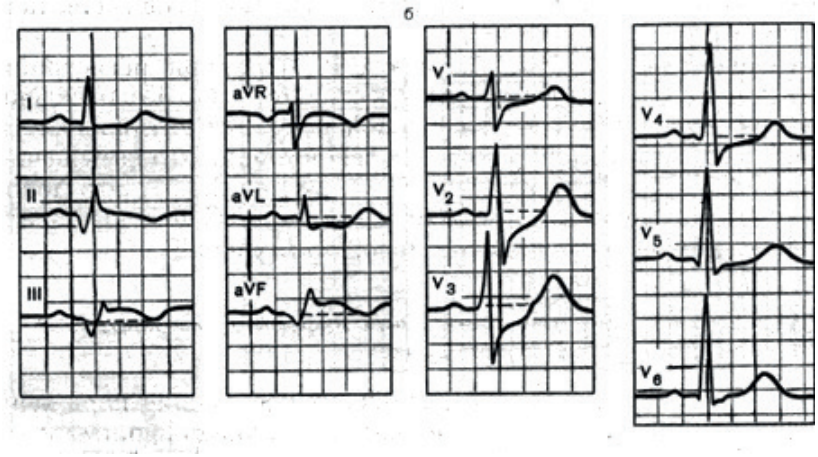


Figure 94. Inferior wall MI

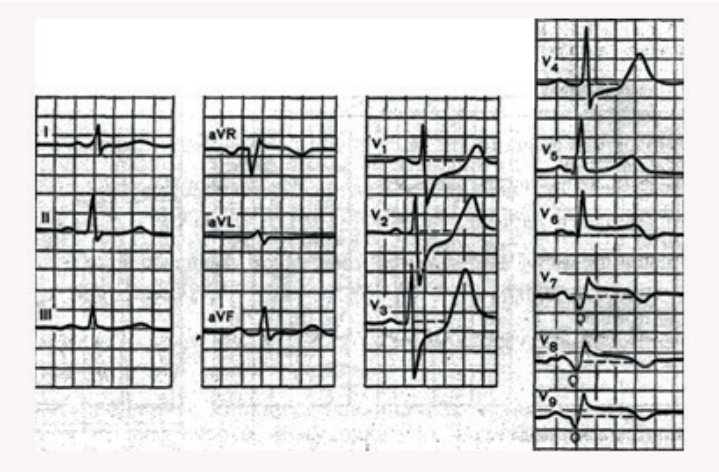


Figure 95. Posterior wall MI

MI and Bundle Branch Block
MI and Right Bundle Branch Block

1. Elevation ST segment in V_1 и V_2 leads in the first 2–5 days of MI (> 2 mm concordantly QRS complex) (Fig. 96).

2. Pathological Q wave in leads V_1–V_4 at anterior-septal MI; at lateral MI and RBBB appears pathological Q wave in V_5 and V_6 leads.

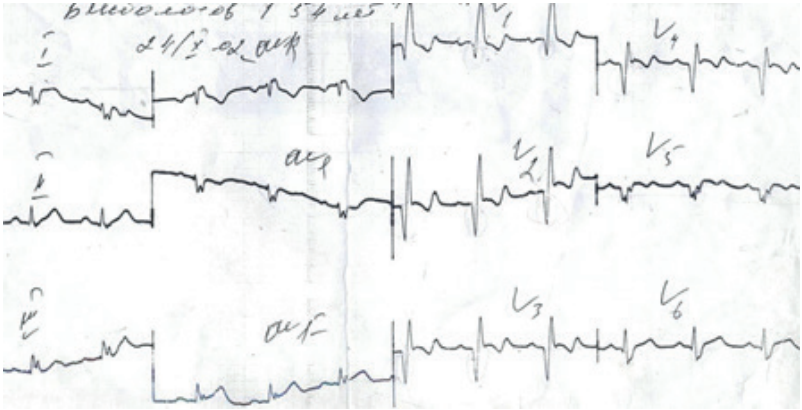


Figure 96. ECG MI and RBBB

MI and Left Bundle Branch Block

Often a difficult ECG diagnosis because in LBBB the right ventricle is activated first and left ventricular Q-wave MI may not appear at the beginning of the QRS complex (unless the septum is involved).

1. Suggested ECG features, not all of which are specific for MI include:

a) Q waves of any size in **two** or more of leads I, aVL, V_5, or V_6 (See below: one of the most reliable signs and probably indicates **septal** infarction, because the septum is activated early from the right ventricular side in LBBB).

b) Reversal of the usual R wave progression in precordial leads (see above).

c) Notching of the downstroke of the S wave in precordial leads to the right of the transition zone (i. e., before QRS changes from a predominate S wave complex to a predominate R wave complex); this may be a Q-wave equivalent.

d) Notching of the upstroke of the S wave in precordial leads to the right of the transition zone (another Q-wave equivalent).

e) rSR' complex in I, V_5 or V_6 leads (S wave is a Q-wave equivalent occurring in the middle of the QRS complex).

f) RS complex in V_{5-6} rather than the usual monophasic R waves seen in uncomplicated LBBB (S wave is a Q-wave equivalent).

g) "Primary" ST–T wave changes (i. e., ST–T changes in the same direction as the QRS complex rather than the usual "secondary" ST–T changes seen in uncomplicated LBBB); these changes may reflect an acute, evolving MI.

Non-Q-wave MI

1. Recognized by evolving ST-T changes over time without the formation of pathologic Q waves (in a patient with typical chest pain symptoms and/or elevation in cardiac-specific enzymes) (Fig. 97).

2. Although it is tempting to localize the non-Q-wave MI by the particular leads showing ST-T changes, this is probably only valid for ST elevation pattern.

3. Evolving ST-T changes may include any of the following patterns:

a) Convex downward ST segment depression only (common) > 48 h;

b) Convex upwards or straight ST segment elevation only (uncommon);

c) Symmetrical T wave inversion only (common) > 10 days;

d) Combinations of above changes.

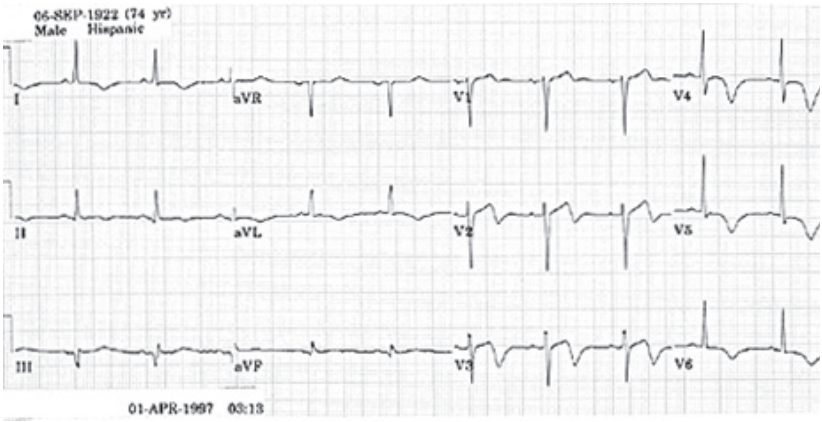


Figure 97. ECG in anterolateral MI

CHAPTER 7.
ECG IN ACUTE PERICARDITIS

Acute pericarditis – the ST segment elevation may mimic acute transmural injury (Fig. 98).

- Concave upwards ST elevation in most leads except aVR.
- No reciprocal ST segment depression (except in aVR).
- Unlike "early repolarization", T waves are usually low amplitude, and the heart rate is usually increased.
- PR (PQ) segment depression **(Spodick's sign)**, a manifestation of atrial injury.

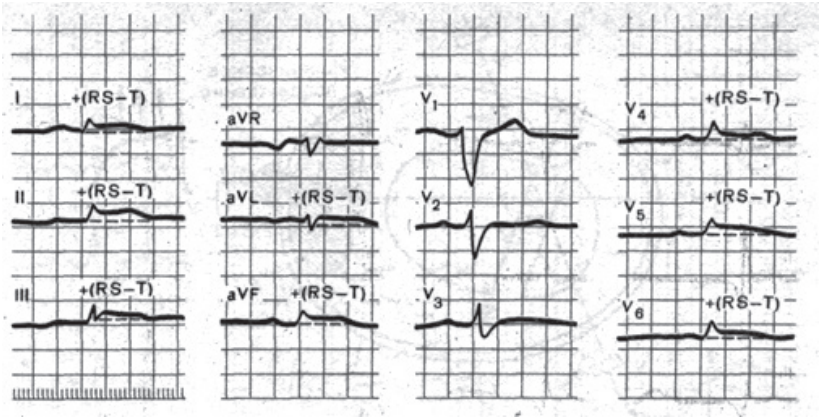


Figure 98. ECG in acute pericarditis

The ECG criteria of **cardiac tamponade** include (Fig. 99):

1. Low QRS voltage in the limb leads alone, or in the precordial leads alone or, in all leads (**Low QRS voltage** is defined as maximum QRS amplitude in precordial lead < 1 mV and < 0.5 mV in the limb leads due to insulating properties of fluid).
2. PR (PQ) segment depression.
3. Electrical alternans.
4. Sinus tachycardia.

ECG cannot be used as a screening tool for diagnosing cardiac tamponade due to its low sensitivity. However, with clinical correlation, electrocardiography is a valuable adjuvant test to ‘rule in’ cardiac tamponade because of its high specificity.

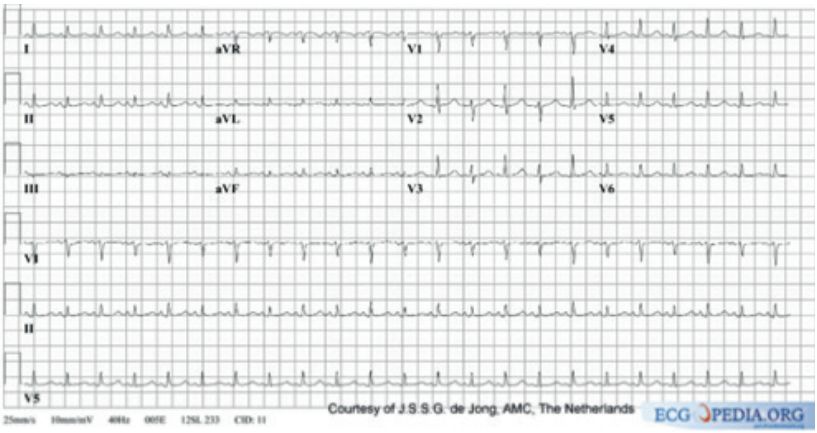


Figure 99. ECG in cardiac tamponade

CHAPTER 8.
BRUGADA SYNDROME

Brugada syndrome (BrS) is a clinical and electrocardiographic syndrome that combines cases of syncope and sudden cardiac death due to fatal ventricular arrhythmias (polymorphic ventricular tachycardia, and ventricular fibrillation), in patients without organic changes of the cardiovascular system and electrolyte disorders, transmitted in an autosomal dominant manner.

On ECG in BrSs can be signs of complete or incomplete RBBB in combination with characteristic ST segment elevation in leads V1 to V3, including during the transient course of the syndrome. In accordance with the ECG in the right chest leads, three types of SB are distinguished.

Type I BrS is characterized by coved ST elevation ≥ 2 mm ("bull terrier" type) accompanied with an inverted T wave in the right precordial leads occurring either spontaneously or after provocative drug test with IV administration of Class I antiarrhythmic drugs (Fig. 100). High-lead positions usage increases the diagnostic yield by ~ 1.5 times compared with standard lead positions.

Type II BrS is characterized by a saddle-shaped ST elevation (saddle-back type) ≥ 2 mm accompanied with a positive or biphasic T wave (Fig. 100).

Type III BrS is characterized by saddle-shape ST elevation ≤1 mm (Fig. 100). Currently, in accordance with the consensus document, a diagnosis of BrS without additional studies is possible in the presence of type I ECG pattern whereas in type II and III patterns patients should undergo sodium channel blocker provocation testing (IV administration of Class I antiarrhythmic drugs induces a type I ECG morphology).

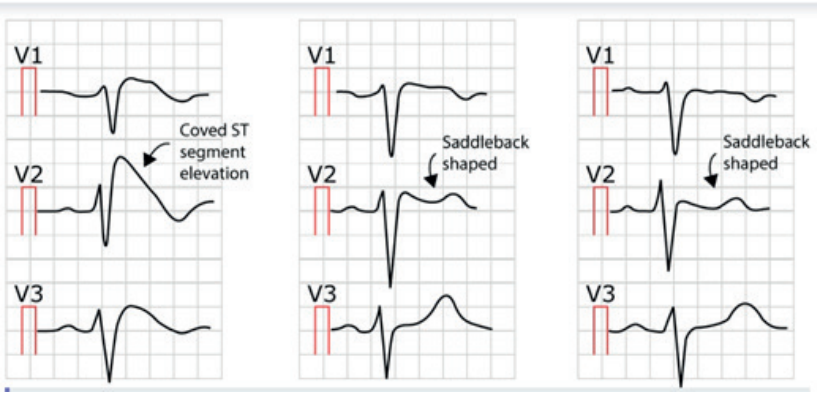


Figure 100. Three types of Brugada syndrome

CHAPTER 9.
ECG IN HYPERKALEMIA

Hyperkalemia is accompanied by progressive ECG changes (Fig. 101) that can lead to ventricular fibrillation and sudden cardiac death. As potassium increases, T waves begin to increase in all 12 leads. This effect can be easily confused with peaked T waves in AMI. One difference is that changes in T waves in AMI are limited to those leads that lie above the infarction area, whereas in hyperkalemia, the changes are diffuse.

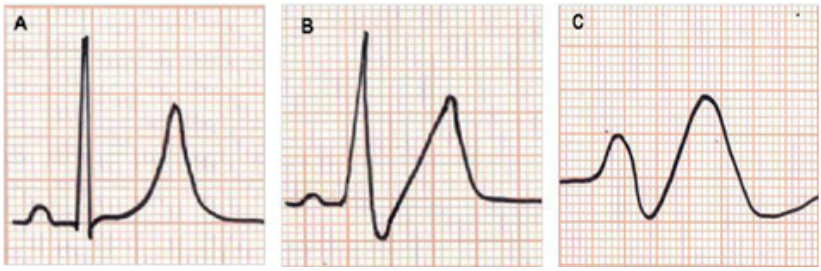


Figure 101. Typical changes in ECG in the development of hyperkalemia. Serum potassium ion, 6.5–7.5 mmol/L (A); 7–8 mmol/L (B); > 8 mmol/L (C)

Subsequently, the QRS complex widens, then it merges with the T wave, forming sinusoidal complexes. Eventually ventricular fibrillation develops.

It is important to note that these changes do not always correspond to blood potassium levels. Progression from hyperkalemia to ventricular fibrillation can occur very quickly. Any ECG change due to hyperkalemia requires close clinical attention. So, ECG changes are not sensitive and specific enough to identify the magnitude of hyperkalemia.

CHAPTER 10.
ECG IN HYPOKALEMIA

Hypokalemia can develop due to vomiting, aggressive gastric suctioning, diarrhea (secondary to infectious diseases), or abuse of potassium-wasting diuretics such as furosemide. T wave flattening, ST-segment depression, and / or U wave development can be seen in 12-lead ECG in hypokalemia (Fig. 102).

Although U waves are the most characteristic feature of hypokalemia, they are not an accurate diagnostic sign. U waves can sometimes be noticeable in normal conditions and when potassium levels are normal.

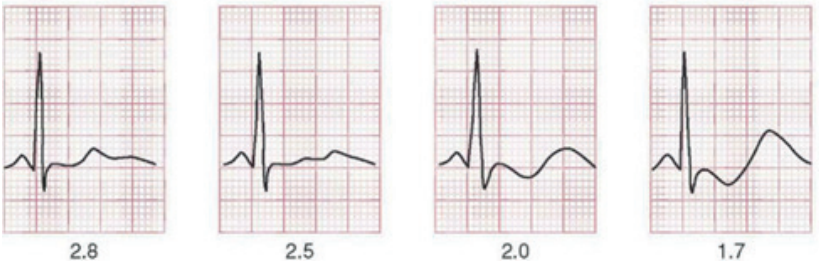


Figure 102. Typical changes in ECG in the development of hypokalemia. Serum potassium ion, 2.8 mmol/L (A); 2.5 mmol/L (B); 2.0 mmol/L (C); 1.7 mmol|L (D).

CHAPTER 11.
DIGITALIS INTOXICATION

Digoxin toxicity ECG features include: shortening of QT interval, “Scooped” or “sagging” ST depressions, J point depression, flattened / inverted / biphasic T waves (Fig. 103). Classic arrhythmias thought to be associated combine some supraventricular tachycardia with decreased AV conduction.

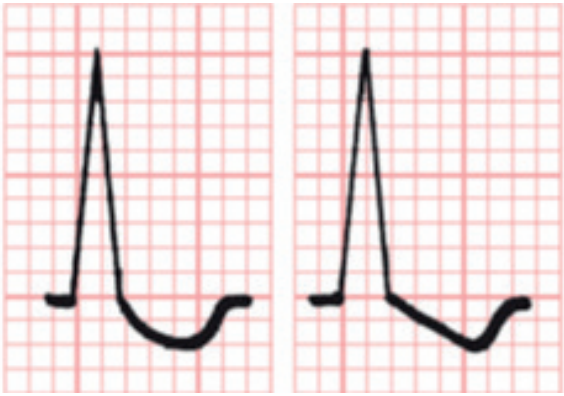


Figure 103. ECG changes in digoxin toxicity

TESTS

1. What is the normal duration of P-Q(R) interval?
a. 0.08 – 0.12 sec
b. 0.1 – 0.2 sec
c. 0.12 – 0.18 sec
d. 0.15 – 0.22 sec
e. more than 0.22 sec

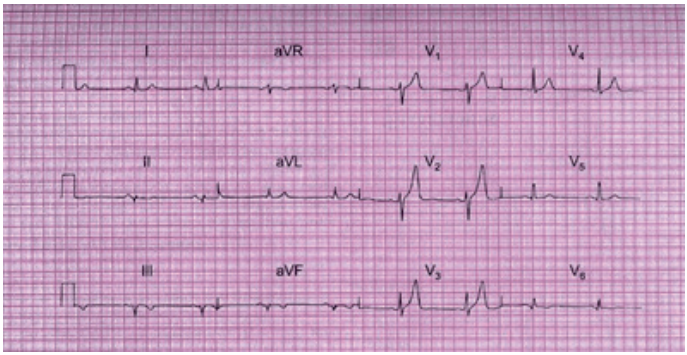
2. What is the normal pacemaker of the heart?
a. AB – connection
b. SA node
c. branches of the His bundle
d. bundle branches
e. Purkinje fibers

3. Which process reflects the P wave on the ECG?
a. ventricular depolarization
b. atrial depolarization
c. ventricular repolarization
d. repolarization of the atria
e. repolarization of the atria and ventricles

4. What is the axis of the heart if the highest QRS (RII>RIII>RI) in standard lead II?
a. horizontal axis deviation
b. vertical axis deviation
c. normal axis of the heart
d. left axis deviation
e. right axis deviation

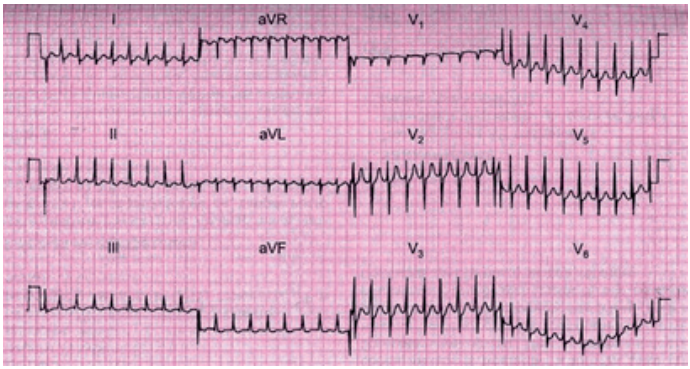
5. What is the heart rate in bradycardia?
a. > 100 beats/min
b. 90–99 beats/min
c. 70–80 beats/min
d. 80–89 beats/min
e. < 60 beats/min

6. A 60-year-old man complained of mild chest pain during physical activity, which arose for the first time. Assess changes on the ECG.



a. MI of anterior wall, hyperacute stage
b. Inferior wall MI, old stage
c. Normal ECG
d. Lateral MI of anterior wall, hyperacute stage
e. MI of anterior wall, acute stage

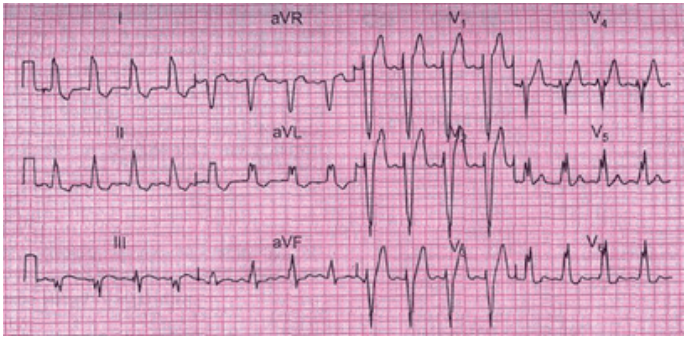
7. A 26-year-old woman complains of palpitations. Similar attacks have occurred many times in the past. Assess changes on the ECG.



a. Supraventricular tachycardia
b. Ventricular tachycardia
c. Atrial flutter

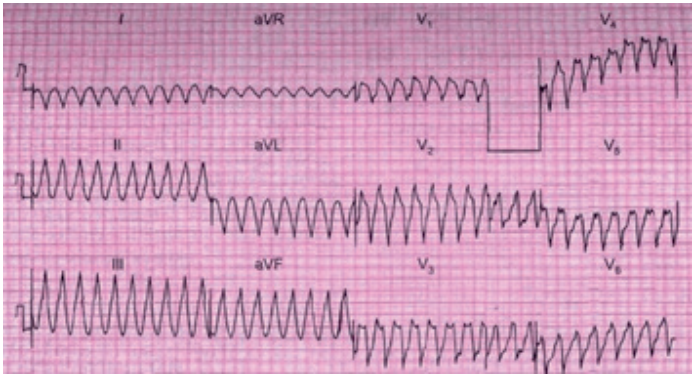
d. Atrial fibrillation
e. WPW syndrome

8. A 75-year-old woman complains of a feeling of heaviness in the chest and dizziness during physical activity. Assess changes on the ECG.



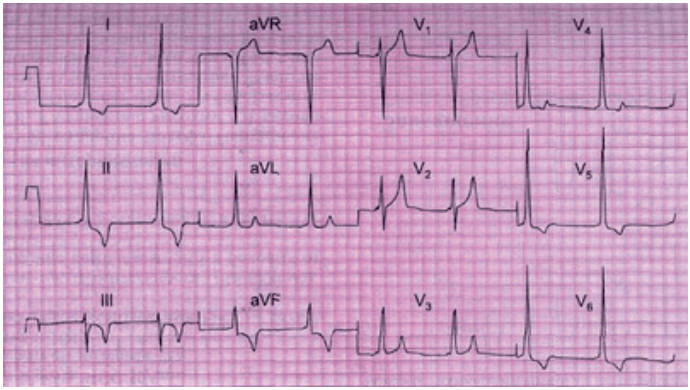
a. Right bundle branch block
b. Sinus rhythm with left bundle branch block
c. Left bundle branch block
d. AV block of the first-degree
e. Atrial fibrillation

9. A patient with acute anterior myocardial infarction is in the cardiology department. The skin is cold, and covered with sweat, blood pressure cannot be determined. Assess changes on the ECG.



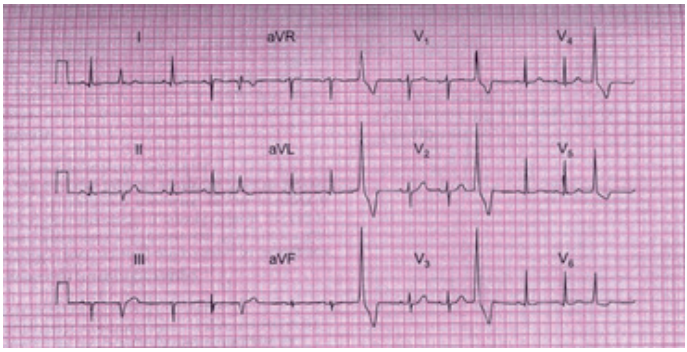
a. Supraventricular tachycardia
b. Ventricular tachycardia
c. Atrial flutter
d. Atrial fibrillation
e. Ventricular flutter

10. An ECG was taken from a 35-year-old man during a preventive appointment. An increase in blood pressure to 180/105 mm Hg was detected, and he had no complaints. Assess changes on the ECG.



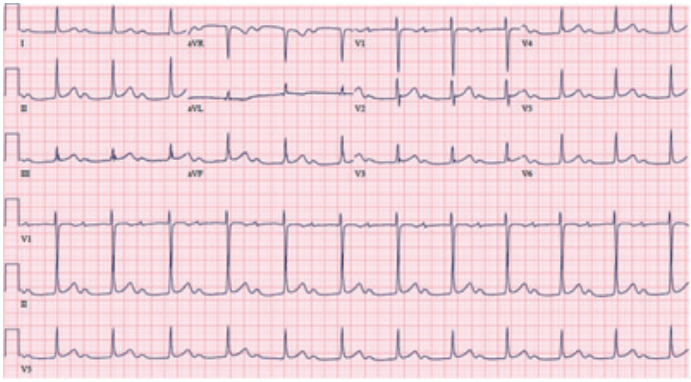
a. AV – first-degree blockade
b. Wolff-Parkinson-White syndrome
c. Digoxin toxicity
d. AV – second-degree block
e. Frederick syndrome

11. An ECG was recorded in a 35-year-old woman with complaints of palpitations, especially when lying down at night. Assess changes on the ECG.



a. Normal ECG
b. Ventricular extrasystole
c. Atrial fibrillation
d. Left bundle branch block.
e. Supraventricular extrasystole

12. An ECG was recorded in a 45-year-old man who had no complaints during a routine examination. Assess changes on the ECG.



a. AV block 1st degree
b. AV block II degree (2:1)
c. AV block third degree
d. Left bundle branch block
e. Supraventricular extrasystole

13. Which ECG changes occur in acute pericarditis?

a. ST-segment elevation
b. T wave inversion
c. Right bundle branch block
d. AV block
e. Pathological Q-wave

14. What is the presence of q wave in leads V_1-V_3 and ST-segment elevation

in these leads characterized for?
a. acute MI of the anterior wall
b. acute MI of the posterior wall
c. acute MI of the lower wall
d. old MI of inferior wall
e. old MI of lateral wall

15. What is the criteria of ischemia in V_2-V_3 leads on ECG for men ≥40 years old?

a. new ST elevation ≥ 0.1мV
b. new ST elevation ≥ 0.15 мV
c. new ST elevation ≥ 0.2 мV
d. new ST elevation ≥ 0.25 мV
e. new ST elevation ≥ 0.30 мV

16. What is the more specific and sensitive criteria for patients with existing complete left bundle branch block and acute MI?

a. Sgarbossa criteria
b. Smith-Modified Sgarbossa criteria
c. Sokolow-Lyon criteria
d. Cornell criteria
e. Spodick's sign

17. What is saddle-shape ST elevation ≤1 mm characterized for?

a. Frederick syndrome
b. Wolff-Parkinson-White syndrome
c. Brugada syndrome
d. Lown-Ganong-Levine syndrome
e. Sick sinus syndrome

18. What is the difference between tall, peaked T waves in acute MI and hyperkalemia on ECG?

a. in hyperkalemia the changes are diffuse
b. in MI changes are in all leads
c. in hyperkalemia are in precordial leads
d. in MI changes are concordant changes
e. in hyperkalemia is also pathological Q wave

19. In which pathology can be seen U wave in ECG?

a. acute MI
b. hypokalemia
c. hyperkalemia
d. Brugada syndrome
e. WPW syndrome

20. In which pathology can be seen "scooped" or ST-depression?

a. hypokalemia
b. hyperkalemia
c. hypercalcemia
d. digoxin toxicity
e. hypomagnesemia

ANSWERS

1. c	6. b	11. b	16. b
2. b	7. a	12. a	17. c
3. b	8. c	13. a	18. a
4. c	9. e	14. a	19. b
5. e	10. b	15. c	20. d

CONTROL QUESTIONS

1. Normal electrocardiography (Normal conduction system of the heart. Methodology of registration ECG. Normal ECG, vector analysis of ECG).

2. Hypertrophy (left and right atrial hypertrophy, left and right ventricular hypertrophy, combined ventricular hypertrophy).

3. Excitability disturbances (Sinus tachycardia. Sinus bradycardia. Sinus Arrhythmia. Sick sinus syndrome. Idioventricular rhythm. Wandering atrial pacemaker. Escape beat. Premature atrial, AV junctional, and ventricular contraction. Paroxismal atrial, junctional, and ventricular tachycardia. Torsade de Pontes. Atrial and ventricular flutter. Atrial and ventricular fibrillation. Ventricular asystole. Electromechanical dissociation.).

4. Conduction abnormalities (Sino-atrial block. Interatrial block. Atrioventricular block. Bundle branch blocks.).

5. Ischemic heart disease (Acute myocardial infarction. MI and bundle branch block.).

6. ECG changes in pericarditis, Brugada syndrome, hyper-, and hypokalemia, digitalis intoxication).

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I.S. Sabirov, K.A. Dzhailobaeva, Z.T. Radzhapova

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