UNIT 5 BASIC OF ELECTROCARDIOGRAPHY

Structure

5.0 OBJECTIVES

After reading this unit, you should able to:

- understand the electrical conduction system of heart;
- know the basic principle of generating various waves and interval of ECG;
- know how to record ECG;
- enumerate the various leads of ECG; and
- know how to calculate the rate of ECG.

5.1 INTRODUCTION

An ECG is the oldest heart test, i.e., still in routine use today. It is a method of recording the electrical activity of the heart. Each heartbeat is caused by a section of the heart generating an electrical signal, which then conducts through specialized path ways to all parts of the heart. These electrical signals also get transmitted through the chest to the skin where they can be recorded.

The ECG is essential for the diagnosis, and management of various cardiovascular abnormalities.

5.2 ELECTRICAL CONDUCTION AND THE HEART

The electrical conduction system of the heart, which produces the electrical activity measured by the electrocardiogram, is composed of the sinoatrial (SA) node, the internodal and interatrial conduction tracts, the atrioventricular (AV) junction (consisting of the atrioventricular node and the bundle of HIS), the right and left bundle branches, and the Purkinje fibers.

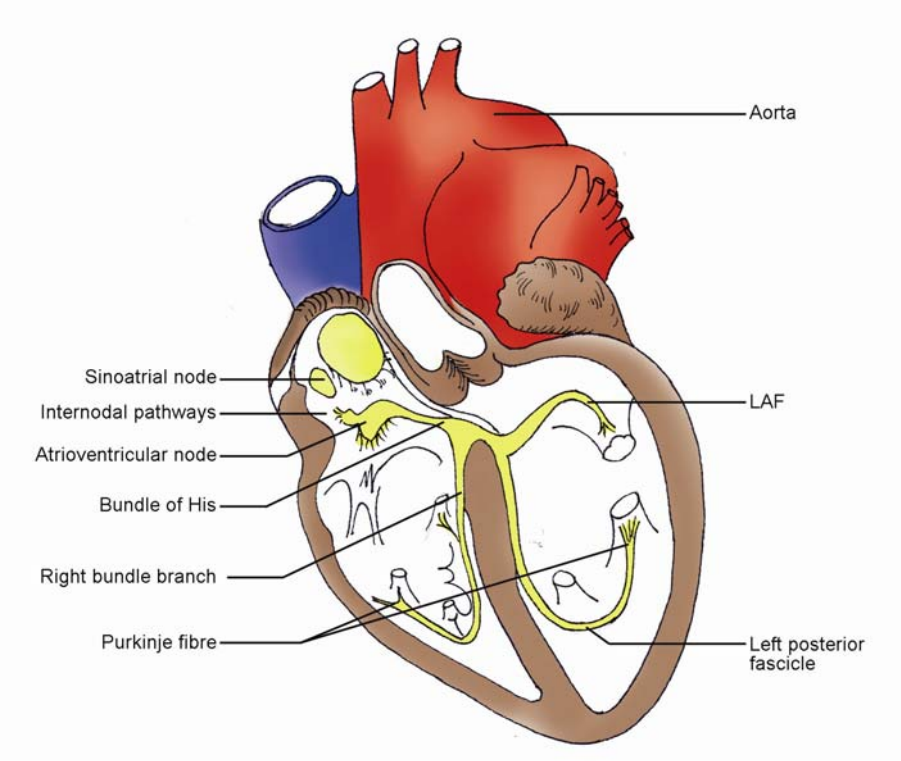

Fig. 5.1: Conduction system of heart

The sinoatrial node, located at the junction of the superior vena cava and the right atrium, is the area of the heart, which, under normal conditions, depolarizes most rapidly. The specialized tissue in the sinoatrial node consists of cells that generate electrical current automatically and regularly. Thus, the sinoatrial node is often referred to as the pacemaker of the heart. Each electrical impulse travels from the sinoatrial (SA) node through the right atrium via three internodal atrial conduction tracts to the atrioventricular (AV) node, located at the proximal part of the atrioventricular junction.

The interatrial conduction tract, also referred to as Bachmann's bundle, branches off one of the internodal atrial tracts and extends across the atria, conducting the impulse from the right to left atrium.

Once the wave of depolarization reaches the AV node, there is a delay that allows the atria to contract, thus emptying blood into the ventricles before the ventricles are stimulated to contract. During this time, electrical activity moves very slowly from the atrium into and through the AV node and into the proximal portions of the ventricular conduction system, the bundle of His, and the bundle branches. Depolarization spreads along the ventricular conduction system (the distal bundle branches and Purkinje fibers) first through the septum, then through the apex and into the bulk of the left and right ventricular walls. The muscle cells of the heart form a syncytium, i.e., they are joined in a way that enables electrical activity to move quickly and easily from one cell to the next. The depolarization of each cardiac cell acts as an electrical impulse on adjacent cells, causing them to depolarize. It is this propagation of electrical impulse from cell to cell that produces waves of depolarization that can be measured as an electric current flowing in the direction of depolarization.

As the cells repolarize, another electric current is produced, similar to the first but moving in the opposite direction. The magnitude and direction of the electrical activity occurring during depolarization and repolarization can be detected by electrodes attached to the skin; this information is then amplified and displayed on the ECG as waves and complexes.

Although the heart has four chambers, it can be considered to be having only two, from the electrical point of view. The two atria as one unit and the two ventricles as another electrical unit, separated by a non-conducting fibrous body through which the HIS bundle penetrates forming the only electrical connection between the two sets of contractile masses.

5.3 NOMENCLATURE OF ECG DEFLECTIONS AND INTERVALS

Each heart beat results in three "waves" or deflections on an ECG. In a normal cardiac cycle, the P-wave occurs first, followed by the QRS complex and the T-wave.

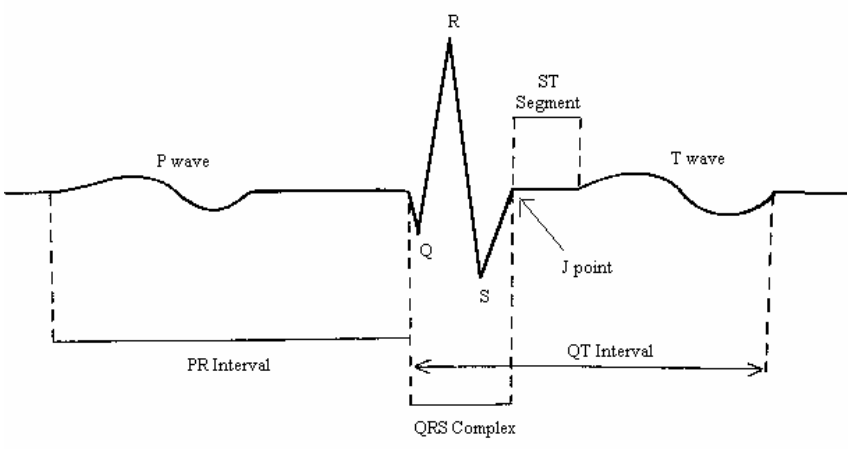

Fig. 5.2: Normal ECG

Check Your Progress 1

1) What are the parts of the conduction system of the heart?

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2) What happens to the electrical impulse when it reaches the AV Node (atrio ventricular node)?

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3) What are the three waves on the ECG?

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5.3.1 P-Wave

Atrial depolarization begins at the SA node and travels through the right atrium, across the intra-atrial septum to the left atrium. The electrocardiographic representation of atrial depolarization is the P-wave. Right atrial depolarization forms the initial portion of the P-wave. The left atrial depolarization forms the terminal portion of the P-wave. The last part of P-wave is also contributed by the lower part of right atrium. The normal P-wave axis is falls between $+45^{o}$ and $+60^{o}$. There is no distinctly visible wave representing atrial repolarization in the ECG because it occurs during ventricular depolarization. The wave of atrial repolarization is relatively small in amplitude (i.e., has low voltage), and is masked by the much larger ventricular-generated QRS complex.

5.3.2 PR Interval

When the impulse leaves the atria and reaches the ventricle the AV node and rest of the conduction system at the AV node it encounters a slight delay. The tissues of the node do not conduct impulses as fast as the other cardiac electrical tissues. This means that the wave of depolarization will take a longer time to get through the AV node. On the ECG, the time taken for the impulse to reach ventricular muscle from atria constitutes the PR interval (PRI).

The PR interval extends from the beginning of the P-wave (the beginning of atrial depolarization) to the onset of the QRS complex (the beginning of ventricular depolarization). It should not exceed 0.20 seconds as measured on ECG graph paper, where each small square represents 0.04 seconds. In other words, the PR interval should not exceed five little boxes in width. Changes in conduction through the AV node are the most common cause of changes in the PR interval. The P

to R interval is important in identification of heart blocks. PR interval prolongation can also occur due to disease in the bundle branches.

The electrical activation (depolarization) of the upper chambers of the heart (the atria) results in the low amplitude P-wave. The subsequent electrical activation (depolarization) of the lower chambers of the heart (the ventricles) results in the high amplitude QRS complex. Repolarization of the atria is a low amplitude signal that occurs during the time of the high amplitude QRS and consequently, is not seen on a standard ECG. Replolarization of the ventricles results in the T-wave. A small positive deflection towards the end of T-wave is called the U-wave. The U-wave, which represents the final phase of repolarization, is often superimposed on the T-wave. Horizontal line between the P and QRS deflections and after the T-wave are said to be at the baseline of that ECG tracing. The line connecting the QRS complex to the T-wave is called the ST-segment and is normally quite close to the baseline. The junction between the end of QRS complex and ST-segment is called J point.

5.3.3 QRS Complex

The ventricular depolarization is shown on the ECG by a large complex of three waves the **Q,** the **R**, and the **S**-waves. Together, these three waves are called the **QRS complex**. The nomenclature is somewhat arcane; small deflections are reflected using lower case, and larger deflections capital letter. An initial downward deflection is a Q (or q), any negative deflection after this is an S. An upward deflection is an R. Note that we refer to a second deflection in the same direction by adding a prime, so we have R', R', S' and so on. Each QRS complex can look a bit different. In fact, some QRS complexes are lacking a Q-wave or others may lack the S-wave. Regardless of the appearance, they are always generically called the "QRS" and still indicate depolarization of the ventricles. The duration of normal QRS complex is 0.06-0.12 seconds. The upper limit of normal duration of the QRS complex is less than 0.12 seconds or three small boxes.

This relatively short duration indicates that ventricular depolarisation normally occurs very rapidly. Place one leg of the caliper on the beginning of the Q-wave and place the other leg of the caliper on the S-wave where it meets the ST-segment. A wide QRS complex (more than 0.12 seconds) may signify delayed conduction in one or more of the bundle branches. The following picture shows the method to measure QRS complex duration on ECG.

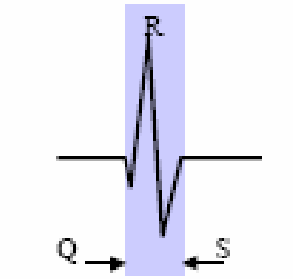

Fig. 5.3: QRS Complex

The QRS complex represents the electrical depolarization of the ventricles. Normally the QRS voltage or amplitude is much higher than the height of the P-wave. This is because ventricular depolarization involves a greater muscle mass and creates a larger complex. Normally, the septum depolarises before other parts of the left ventricle. This is seen as a small initial vector, which in the 'septal leads' (V1 and V2) is a positive deflection, and in lateral leads (e.g. V6) is

seen as a small q. This observation is of relevance, as in conditions such as left bundle branch block, where the septum cannot depolarise normally, the lateral (septal) q is conspicuously missing.

Ventricular activation time is the time it takes for the ventricle to depolarise. This can be estimated from the surface ECG by looking at the time from the onset of the QRS to the sudden downstroke of the QRS. This sudden downstroke is the 'intrinsicoid deflection'. In right orientated leads, a normal VAT is 0.02s, and on the left (e.g. V6) the duration should not exceed 0.04s.

Following picture shows different morphologies of QRS complexes.

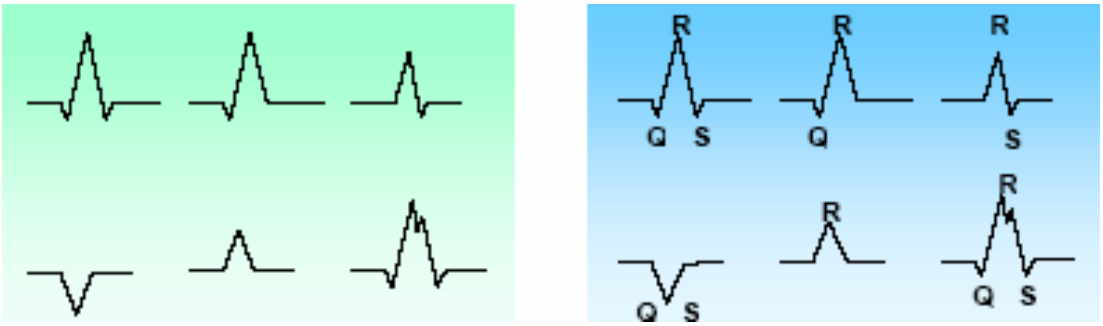

Fig. 5.4 (a) and (b): Different Morphologies of QRS Complexes

A QRS complex can have positive (upwards) or negative (downwards) deflections. If it starts with an initial negative deflection, that deflection is called a Q-wave. The first upward deflection is called an R-wave. A negative deflection following an R-wave is called an S-wave. A single negative deflection without any R-wave is called a QS complex. A second R-wave following an S-wave is called an R' ("R-primed") wave. Time is represented on the horizontal, x-axis on ECGs. The distance between 2 vertical lines is 1 millimeter representing 0.04 seconds with a recorder sweep speed of 25 millimeters per second. The vertical, y-axis represents the amplitude or strength of the electrical signal in millivolts. Each horizontal line represents 0.1 millivolts.

The sections of the ECG between the waves and complexes are called segments and intervals. Intervals include waves and complexes, while segments do not. For example, we speak of the PR segment, the ST-segment, the PR interval, the QT interval, and the R-R interval. When electrical activity of the heart is not being detected, the ECG is a straight, flat line, called the isoelectric line, or baseline.

The time it takes for electricity to be conducted from the atria to the ventricles is represented by the PR interval. This is measured from the beginning of the P-wave to the beginning of the QRS complex. The time it takes for the ventricles to become electrically activated is represented by the QRS duration. This is measured from the beginning of the QRS to the end of the QRS. The duration between two consecutive R-waves is called RR interval and represents the time between two ventricular activation. This interval is used to calculate the ventricular rate. Similarly, the distance between two consecutive P-waves reflects the duration between two atrial activations. The total amount of time the ventricles are electrically active (from onset of depolarization to completion of repolarization) is represented by the QT interval. This is measured from the onset of the QRS to the end of the T-wave.

The voltage of the P-wave and QRS complex is proportional to the total amount of muscle being depolarized. A higher than normal voltage implies overgrowth of the muscle of that chamber.

Since the left ventricle has a lot more muscle than the right ventricle, the QRS complex primarily represents electrical events of the left ventricle.

5.3.4 ST Segment

The ST segment begins at the J point ends with the onset of the T-wave. The J point is the point at which the QRS complex ends and the ST segment begins. Measure the ST segment duration from the J point up to the beginning of the T-wave. The ST segment represents the early part of repolarization of the entricles. The ST segment normally sits on the baseline or isoelectric line. It is also normal if the ST segment is slightly elevated or below the isoelectric line (no greater than one millimeter in either direction). Greater than 1 mm ST segment elevation or depression can be indicative of myocardial ischaemia or injury. The ST segment is important because it can show whether ischaemia or infarct is present. In general, an ST segment depression indicates ischaemia while elevation generally indicates infarction. When examining the ST segment, evaluate elevations or depressions 0.06 seconds after the J point (since the ST segment can at times be sloping). The location of the ST elevations on the ECG can help to identify a location of the infarct.

Causes of ST Segment Elevation

1) Normal Variant “Early Repolarization” (usually concave upwards, ending with symmetrical, large, upright T-waves).

2) Ischaemic Heart Disease-acute ischaemia or aneurysm (usually convex upwards, or straightened),

3) Acute Pericarditis,

4) Left ventricular hypertrophy (in right precordial leads with large S-waves)

5) Left bundle branch block (in right precordial leads with large S-waves)

6) Advanced hyperkalemia

7) Hypothermia (prominent J-waves or Osborne waves)

Common Causes of ST Segment Depression

Ischaemic Heart Disease

- Subendocardial ischaemia (exercise induced or during angina attack)
- Non Q-wave MI
- Reciprocal changes in acute Q-wave MI

Non-ischaemic Causes of ST Depression

- RVH (right chest leads) or LVH (left chest leads, I, aVL) Digoxin effect on ECG
- Hypokalemia

- Mitral valve prolapse
- CNS disease
- Secondary ST segment changes with bundle branch blocks.

5.3.5 T-Wave

Ventricular repolarization is represented on the ECG by a T-wave. The beginning of the T-wave is identified at the point where the slope of the ST segment appears to become abruptly or gradually steeper. The T-wave ends when it returns to the isoelectric baseline. The period from the beginning of the T-wave to nearly the end is called the "relative refractory period". At this time, the ventricles are vulnerable. A stronger than normal stimulus could trigger depolarization. If an R-wave (ventricular depolarization) should occur during this time, a potentially fatal arrhythmia could result.

The normal T-wave is usually in the same direction as the QRS except in the right precordial leads. Also, the normal T-wave is *asymmetric* with the first half moving more slowly than the second half. In the normal ECG, the T-wave is always upright in leads I, II, V3-6 and always inverted in lead aVR. In the other leads T-waves are variable, depending on the direction of the QRS and the age of the patient.

The T-wave is the most labile wave in the ECG. T-wave changes including may be the result of many cardiac and non-cardiac conditions.

Common Causes of T-Wave Inversion

1) Q-wave and non-Q-wave MI
2) Myocardial ischaemia
3) Pericarditis
4) Myocarditis
5) CNS disease causing long QT interval (subarrachnoid hemorrhage)
6) Idiopathic apical hypertrophy (a rare form of hypertrophic cardiomyopathy)
7) Mitral valve prolapse,
8) Digoxin effect and
9) RVH and LVH with "strain" (see below: T-wave inversion in leads aVL, V4-6 in LVH)

Common Cause of Tall T -Waves

1) Hyperkalemia;
2) Early stage of ST elevation myocardial infarction; and
3) Early repolarization abnormality in young individuals.

A Note About ST-T-Wave Changes

The cause of ST-T-wave abnormalities is provided more often by the clinical circumstances in which the ECG changes are recorded than by the changes themselves. Thus the term, nonspecific

ST-T-wave abnormalities, is frequently used when the clinical data is not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant and can be ignored. It is the onerous responsibility of the treating physician to ascertain the importance of the ECG findings. The ST-T-wave changes can be broadly of two types:

1) Primary ST-T-wave abnormalities-result from pathologic processes affecting the ventricular myocardial repolarization and are independent of changes in ventricular activation.

2) Secondary ST-T-wave abnormalities-result from alterations in the sequence of ventricular activation.

Common Causes of Primary ST-T-Wave Abnormalities are:

1) Intrinsic myocardial disease (e.g., myocarditis, ischaemia, infarction, infiltrative disorders)

2) Drugs (e.g., digoxin, quinidine, tricyclics, etc.)

3) Electrolyte abnormalities

4) Intracranial pathology (e.g., stroke, trauma, tumor, etc.)

5) Metabolic abnormalities (e.g., hypoglycemia, hyperventilation)

6) Ventricular conduction abnormalities and rhythms originating in the ventricles

Common Causes of Secondary ST-T Wave Abnormalities are:

1) ST-T changes seen in bundle branch blocks,

2) ST-T changes seen in fascicular block,

3) ST-T changes seen in nonspecific IVCD,

4) ST-T changes seen in WPW preexcitation and

5) ST-T changes in ventricular rhythms (VPCs, VT, etc.)

5.3.6 U-Wave

The U-wave is a small rounded deflection seen towards the end of T-wave. Sometimes it is absent. This deflection is best seen in leads V2 and V3. Normally the amplitude is about 10 per cent (not more than 1/3rd) of that of a normal T-wave. The direction of U-wave is usually the same as T-wave. The normal U-wave is asymmetric with the ascending limb moving more rapidly than the descending limb (just the opposite of the normal T-wave). This wave is separated from the T-wave by T-U junction along the baseline. The U-wave is prominent in hypokalemia. If there is a fusion between T and U-waves then it becomes difficult to measure QT interval. The origin of U-wave is uncertain. The commonly offered explanations for genesis of U-waves are:

1) Repolarization of papillary muscle or the Purkinje network or

2) After depolarization of ventricular myocardium.

5.3.7 Q-T Interval

The Q-T interval represents the time for both ventricular depolarization and repolarization to occur, and therefore estimates the duration of an average ventricular action potential. This interval can range from 0.2 to 0.4 seconds depending upon heart rate. At high heart rates, ventricular action potentials shorten in duration, which decreases the Q-T interval. Because

prolonged Q-T intervals can be diagnostic for susceptibility to certain types of tachyarrhythmias, it is important to determine if a given Q-T interval is excessively long. In practice, the Q-T interval is expressed as a "corrected Q-T (**Q-Tc**)" by taking the Q-T interval and dividing it by the square root of the R-R interval (interval between ventricular depolarizations). This allows an assessment of the Q-T interval, i.e., independent of heart rate. Normal corrected Q-Tc intervals are less than 0.44 seconds.

Systematic Analysis of ECG

It is necessary to analyse ECG systematically to make an accurate and complete interpretation. The following scheme may be helpful:

1) Check the patient details–is the ECG correctly labelled?
2) What is the rate?
3) Identify the rhythm.
4) How are the P-waves (Good places to look are II and VI)?
5) What is the PR interval?
6) What is the mean frontal plane QRS axis?
7) Identify the QRS complex abnormalities. Specifically, look for:
   - significant Q-waves
   - voltage criteria for LV hpertrophy
   - widened QRS complexes
   - any other abnormality
8) Identify the ST segment abnormalities. Are the ST segments normal, depressed or elevated? Quantify abnormalities.
9) Are the T-waves normal?
10) What is the QT interval?
11) Are there abnormal U-waves?

Approach to Wide QRS Complex Tachycardias

Tachycardias with QRS duration exceeding 120 msecs are termed wide QRS Tachycardia. This may arise from either a supraventricular or ventricular focus. Correct diagnosis of the origin of these tachycardias is important for several reasons:

- Drugs used for the treatment of supraventricular tachycardia (SVT) (verapamil Drugs, adenosine, or beta blockers) can cause severe hemodynamic deterioration in patients with ventricular tachycardia (VT), often worsen the hypotension, ischemia, and make it difficult to revert the arrhythmia. Prognosis is poor if patients with heart disease have VT as compared to SVT.

- Definitive therapy in the form of catheter ablation requires localization of the origin of the tachycardia.

History, clinical findings and ECG features which help to differentiate supraventricular and ventricular arrhythmias. Wide QRS complex tachycardia (WQRST) of ventricular origin is still commonly misdiagnosed as an SVT, especially if the arrhythmia is well tolerated hemodynamically.

Physiological Basis

Normal activation of the ventricles through the HIS Purkinje System (HPS) is rapid and is completed within 80 msecs. This is because of the rapid conduction thru the HPS. However conduction thru the myocardial tissue is much slower. A reentrant impulse within the scar of the infarct is slow to activate rest of the myocardium resuting in prolongation of the QRS duration during VT.

In patients with Bundle branch block initial activation of the ventricle is thru the HPS. Subsequent ventricular activation on the side of the bundle branch block occurs solely through the myocardial tissue causing prongation of the QRS duration.

Widening or aberrancy of the QRS complex can occur in any type of supraventricular rhythm including sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation (AF), AV nodal reentrant tachycardia (AVNRT), and AV reentrant tachycardia (AVRT) (associated with an accessory pathway). SVTs with a wide QRS complex can result from conduction over either an accessory pathway or the normal His-Purkinje conduction system.

Aberrantly Conducted SVT Without an Accessory Pathway

In the absence of conduction over an accessory pathway, widening of the QRS complex during any SVT can occur when conduction over the HIS conduction system is blocked in either the right or the left bundle or the distal Purkinje system (intraventricular conduction delay).

Supraventricular tachycardia occurring in a patient with preexistent Bundle branch block will present as WQRST. When intraventricular conduction is i.e., QRS duration is normal during sinus rhythm but aberrant during tachycardia, several of the following mechanisms have to be considered:

Functional (Rate-related) or Physiologic Phase three Aberration

Rate-related aberration can occur in normal fibers if a very premature impulse reaches the cell before its membrane has fully repolarized. This form of aberration is commonly seen at the early onset of paroxysmal SVT. It most frequently results in a RBBB QRS morphology, although a LBBB pattern can also occur. Bundle branch block aberrancy may be sustained if this mechanism continues.

Concealed Retrograde Impulse Conduction

Sustained QRS aberration more commonly results from concealed retrograde penetration A ventricular impulse can penetrate retrogradely in either the right or the left bundle. If retrograde penetration occurs in the right bundle, this structure is refractory when the next sinus impulse passes through the AV node; in this setting, intraventricular conduction only occurs over the left bundle, resulting in a RBBB. This mechanism called linking results in sustained aberration.

Use-dependent Effects of Antiarrhythmic Drugs

Patients receiving antiarrhythmic drugs, especially the class IC antiarrhythmic agents, may have a rate-related aberration as a result of the depressant effect of the drug on intraventricular conduction.

Preexcitation Syndrome

In the preexcitation syndromes, AV conduction occurs over two pathways the normal AV nodal–HIS pathway; and an accessory AV pathway. In this condition, the QRS complex represents a fusion beat.

Tachycardia can have a narrow or a wide QRS complex:

- If antegrade conduction to the ventricles occurs over the AV node and retrograde conduction back to the atria is over the accessory pathway, the QRS complex will be narrow unless intraventricular conduction demonstrates a functional, rate-related block. This narrow complex AVRT is known as an orthodromic AVRT .

- If antegrade conduction occurs over an accessory pathway and retrograde conduction occurs over the AV node or another accessory pathway, a wide QRS complex tachycardia ensues, known as an antidromic AVRT This arrhythmia is very difficult to differentiate from VT, because ventricular activation starts outside the normal intraventricular conduction system in both types of tachycardia.

Ventricular Tachycardia

Tachycardias which originate in ventricular tissue almost invariably result in a widened QRS complex. Rarely, tachycardias which arise in the HIS Purkinje system can produce narrow QRS complexes. These are termed bundle of HIS or fascicular tachycardias.

Artifact Mimicking VT

ECG artifact, particularly when observed on a rhythm strip, may be misdiagnosed as VT and can result in unnecessary invasive procedures

Clinical Approach to Diagnosis

The misdiagnosis of a wide complex tachycardia is very common. Among 122 patients with VT verified by electrophysiologic study, for example, only 39 were correctly diagnosed at the initial evaluation.

History, physical examination, response to certain maneuvers, and careful analysis of the ECG will help in correct diagnosis. Comparison of the ECG during the tachycardia with sinus rhythm also is very useful.

History

Age

Supra Ventricular tachycardia with aberrancy is more likely in a younger patient. But this is not always useful, due to significant overlap.

Presence of underlying heart disease

The presence of structural heart disease, especially a previous myocardial infarction (MI), strongly suggests that WQRS tachycardia is probably VT. In one report, for example, over 98 per

cent of patients with a previous MI had VT as the etiology of a wide complex tachycardia, while only 7 per cent of those with SVT had a MI .

Duration of the Tachycardia

SVT is more likely if the tachycardia has recurred over a period of greater than three years. The occurrence of a WQRS tachycardia for the first time after an MI strongly suggests it is VT.

Symptoms

Normal Blood pressure during a WQRS Tachycardia is often considered as a diagnostic pointer for SVT with aberrancy. This is incorrect. *Patients with VT often present with normal arterial Blood pressure.* Symptoms associated with a tachycardia can be mild, such as palpitations, lightheadedness, weakness, diaphoresis, a chest sensation, and polyuria. However, hemodynamic instability can occur, resulting in dizziness, angina, presyncope or syncope, cardiogenic shock, or seizures. Symptoms are not related to the underlying mechanism of the arrhythmia; they are primarily due to the elevated heart rate, any associated heart disease, and left ventricular function .

SVT is more likely to occur in a younger patient with normal left ventricular function and no heart disease. In this setting, the tachycardia often results in no symptoms suggestive of hemodynamic compromise. Hemodynamic instability and hypotension may result, however, if the rate is rapid (as may be seen when an accessory pathway is present) or if there is underlying heart disease (especially valvular or congenital abnormalities). Supra Ventricular tachycardia can result in serious hemodynamic compromise in patients with valvular heart disease , Hypertrophic cardiomyopathy and certain congenital heart disease.

On the other hand, VT does not necessarily result in hypotension . Whether hemodynamic compromise occurs in response to VT is based upon several factors like Ventricular rate, Presence and extent of underlying heart disease,Function of the left ventricle, Presence of AV asynchrony, Location of the VT focus (which results in a particular, often abnormal, sequence pattern of left ventricular activation).

Clinical Examination

Although the blood pressure and heart rate are not particularly helpful in distinguishing between SVT and VT, other elements of the physical examination can provide important diagnostic clues.

Evidence of AV dissociation is important in the diagnosis of a wide complex tachycardia. AV dissociation is present (although not always evident) in approximately 60 to 75 percent of patients with VT, but it is rarely seen in SVT. Thus, AV dissociation usually establishes a tachycardia as VT, although its absence is less helpful.

The presence of AV dissociation may be determined from the ECG or physical examination.

Cannon “A” waves can be observed upon examination of the jugular pulsation in the neck. Cannon waves are intermittent and irregular pulsations of greater amplitude than normal waves. They reflect simultaneous atrial and ventricular contraction; contraction of the atria against a closed AV valve produces a transient increase in atrial and venous pressure.

- Highly inconsistent fluctuations in the blood pressure may occur because of the variability in the degree of atrial contribution to LV filling, stroke volume, and cardiac output.

- Variation in the in pulse volume from beat to beat is also indicative of VT.

- Variability in the occurrence and intensity of heart sounds (especially S1) is also present.

Response to Interventions

The response to carotid sinus massage (which enhances vagal tone and therefore depresses sinus and AV nodal activity) and to different pharmacologic interventions can be helpful.

The heart rate during sinus tachycardia will gradually slow with carotid sinus pressure and then accelerate upon release.

- The ventricular rate of atrial tachycardia and atrial flutter will transiently slow with pressure (due to increased AV nodal blockade). The arrhythmia itself, which occurs within the atria, is unaffected.

- An SVT (either AVNRT or AVRT) will either terminate or remain unaltered with carotid sinus pressure.

- VT is generally unaffected by carotid sinus pressure, although this maneuver will slow the atrial rate and in some cases expose AV dissociation

- Termination of the arrhythmia with lidocaine suggests, but does not prove, that VT is the mechanism.

- Termination of the arrhythmia with digoxin, verapamil, diltiazem, or adenosine strongly implies SVT. However, VT may also rarely terminate after the administration of these drugs.

Unless the etiology for the wide complex tachycardia is established, drugs like verapamil, diltiazem, and even adenosine should not be administered, since they have been reported to cause hemodynamic collapse in patients with VT.

Electrophysiologic Study

Electrophysiologic testing has a role for the evaluation of a wide complex tachycardia when the etiology remains unclear and a correct diagnosis is important for therapy.

ECG Criteria for Differential Diagnosis

Diagnostic criteria in current use were defined from analysis of the 12-lead ECGs recorded during tachycardia in patients in whom the site of origin and the mechanism of the arrhythmia were determined during electrophysiologic investigation. A number of ECG findings are suggestive, if not diagnostic, for either VT or SVT:

- AV dissociation, fusion, or capture beats

- QRS morphology pattern in the precordial leads

- Duration of the QRS complex

- Axis of the QRS complex in the frontal plane.

AV Dissociation

The ECG should be carefully examined for evidence of AV dissociation which, if present, makes VT very likely. AV dissociation results from the absence of retrograde atrial activation (i.e., no ventriculoatrial conduction) due to retrograde AV nodal block. Atrial activity or the P wave is therefore independent of the ventricular activity or QRS complex during VT.

The atrial rate is usually slower than the ventricular rate. The diagnosis of AV dissociation is obviously impossible if AF is the underlying supraventricular rhythm.

While the presence of AV dissociation establishes VT as the etiology, its absence is not as helpful for two reasons. First, AV dissociation may be present but not obvious on the ECG.

The following ECG findings are helpful in establishing the presence of AV dissociation:

Dissociated P waves

Dissociated P waves may be clearly seen on the ECG or rhythm strip, or may be superimposed upon the ST segment or T wave (resulting in altered morphology). However, identifying independent P waves is sometimes difficult because T waves and initial or terminal QRS portions can resemble atrial activity. Furthermore, artifacts can be mistaken for P waves.

If P waves are not obvious or suggested on the ECG, the following alternative leads or modalities may help in their identification–a modified chest lead placement (Lewis leads), an esophageal lead (using an electrode wire or nasogastric tube), a right atrial recording (obtained by an electrode catheter in the right atrium or from a central venous line), carotid sinus pressure, or invasive electrophysiologic studies.

Fusion beat

The presence of a fusion beat (which has a morphology intermediate between the sinus beat and ventricular complex) results from the simultaneous activation of the ventricular myocardium via the atrium-AV node-His Purkinje system and the ventricular focus .

Dressler beat

An intermittent captured or a Dressler beat is a QRS complex that is normalized and is identical to the sinus QRS complex. It results from ventricular activation which is entirely the result of impulse conduction from the atrium via the AV node and His Purkinje system.

Fusion and Dressler beats are more commonly seen when the tachycardia rate is slower. These beats do not alter the rate of the VT, although a change in RR intervals can result.

Although a capture or fusion beat (which produces a QRS with a narrower morphology than the QRS of ventricular origin) may be observed during VT, the presence of a narrower beat during a wide QRS tachycardia is not always a marker for VT. Such beats can also occur in SVT with a bundle branch block when a ventricular premature beat arises in the ventricle having the bundle branch block and is conducted retrogradely through this bundle, fusing with the impulse coming antegradely via the other bundle. Furthermore, AF occasionally can conduct over the AV nodal-His Purkinje axis in Wolff-Parkinson-White (WPW) syndrome, resulting in narrower beats. In this circumstance, however, the rhythm is irregularly irregular and can be distinguished from a tachycardia with regular RR intervals.

Other electrocardiographic findings suggestive of VT include:

- A significant shift in axis, especially an extreme left axis is suggestive of VT. In RBBB morphology tachycardia, left axis deviation is suggestive of VT. In LBBB morphology tachycardia Right axis deviation is suggestive of VT.

- Slight or marked changes in QRS morphology which are not rate-related. In contrast, similarity in QRS form suggests, but does not prove, that SVT is the underlying etiology

QRS Morphology Pattern in the Precordial Leads

A diagnosis based upon the precordial QRS morphology pattern requires that the QRS polarity in lead V1 and V2 in a wide complex tachycardia must be able to be defined as either positive (RBBB-like pattern) or negative (LBBB-like pattern). QRS morphology in lead V6 offers additional diagnostic clues in both V1-positive and V1-negative tachycardias. Unfortunately, most of the association between the QRS morphology and tachycardia origin is based upon a statistical correlation with substantial overlap. In addition, most of the morphologic criteria favoring ventricular tachycardia are present in a substantial number of patients with an intraventricular conduction delay during sinus rhythm, limiting their applicability in these cases.

V1 Positive wide QRS Tachycardia

RBBB aberrancy can be identified in lead V1 from a triphasic rSR' pattern and in lead V6 from a triphasic qRS pattern with the R:S ratio greater than one. The small initial waves (r in V1 and q in V6) reflect normal septal activation, which is preserved in RBBB; the tall terminal forces (R in V1 and S in V6) indicate late activation of the right ventricle, which is due to right bundle conduction delay.

Since intraventricular conduction is bizarre in VT, such arrhythmias do not follow the rules that apply to functional or structural interruption of the normal conduction system. As a result, VT is suggested by a monophasic R or a biphasic qR pattern in lead V1 and a deep S wave (R to S ratio of less than one) in lead V6, morphologies rarely found in SVT.

A ventricular origin is also likely when a double-peaked R wave is recorded in V1, with the left peak taller than the right (the so-called rabbit ear sign) .[A taller right rabbit ear, however, does not help in distinguishing a ventricular from a supraventricular site of origin.

- V1 negative wide QRS tachycardia — A ventricular origin of a V1-negative (LBBB-like) wide QRS tachycardia is highly suspected in the presence of the following ECG findings

- A broad initial R wave of 0.03 sec (30 msec) or more in lead V1 or V2; this initial R wave is often taller during tachycardia than during sinus rhythm

- A duration of > 0.07 sec from the onset of the ventricular complex to the nadir of the QS or S wave in lead V1 and V2; and the presence of any Q wave in lead V6.

Features of SVT with LBBB

- The unopposed but small right septal vector causes a small narrow R wave in V2 and the frequent absence of any initial positive deflection in V1.

- The right to left septal vector is directed towards V6, causing initial positivity and absence of any Q wave in this lead.

- The downstroke of the S wave is clean, without any slurring or notch, and has a swift inscription.

QRS Width and QRS Axis

The width of the QRS complex and the QRS axis in the frontal plane are other useful ECG criteria. VT is more likely than SVT if there is:

- A QRS duration of > 0.14 second and a superior axis, especially in the "northwest quadrant" (-90 to -180 or an indeterminant axis).

A marked rightward or leftward shift in axis, primarily of the initial portion of the QRS complex.

Stepwise Approach to Wide QRS Tachycardias

This algorithm can be used sequentially.

Differential Diagnosis between VT and SVT with Aberrant Conduction

The first algorithm is based upon observations that prolongation of the RS interval in LBBB-like wide QRS complex tachycardias strongly favors the diagnosis of VT. As a result, prolongation of the intrinsic deflection in any precordial lead showing a clear RS complex is a marker of VT, independent of the morphology pattern in V1 (RBBB or LBBB pattern).

In a Stepwise Manner ECG is Analysed

- Negative Concordance: l If an RS complex cannot be identified in any precordial lead, the diagnosis of VT can be made with 100 percent specificity and further analysis is not needed.

- If an RS complex is clearly distinguished in one or more precordial leads, the interval between the onset of the R-wave and the deepest part of the S-wave (RS interval) is measured. The onset of ventricular activation can be more accurately measured if all precordial leads are recorded simultaneously. The longest RS interval is considered if RS complexes are present in multiple precordial leads. If the RS interval is >100 msec, the diagnosis of VT can be made with a specificity of 98 percent and further analysis is unnecessary.

- Evidence of AV dissociation is 100 percent specific for the diagnosis of VT, but this finding has a low sensitivity.

- If the RS interval is <100 msec and AV dissociation cannot clearly be demonstrated, the classic morphology criteria for V1-positive and V1-negative wide QRS complex tachycardias are considered and must be present in leads V1 or V2 and in lead V6.

In practice WQRS tachycardias are often due to VT especially in patients with prior MI. Unless the diagnosis of Supra Ventricular tachycardia with aberrancy is certain it is better to avoid calcium antagonists. When in doubt it is advisable to treat the tachycardia as VT.

5.4 RECORDING AN ECG

The contraction of any muscle is associated with electrical changes called 'depolarization', and these changes can be detected by electrodes attached to the surface of the body. Since all

muscular contraction will be detected, the electrical changes associated with contraction of the heart muscle will only be clear if the patient is fully relaxed and no skeletal muscles are contracting. The principle behind the way the ECG records the electrical impulse is quite simple. The electrical signal that starts in the atria and travels down to the ventricle is of course moving through three dimensions. Each lead inscribes a positive deflection for that component of the net electrical vector i.e. travelling towards its positive electrode and a negative deflection for that component of the net electrical vector i.e. travelling towards its negative electrode. Thus, by knowing the position of each lead, one can determine the direction the electrical signal is travelling. When the electrical vector and direction of the lead are perpendicular, then the lead records a horizontal line, namely isoelectric line.

An actual ECG is recorded by placing electrodes on each limb and 6 electrodes on the chest. This allows the recording of 12 ECG leads. A routine ECG is recorded by connecting 12 leads. Looking at the heart with 12 leads is like looking at a sculpture, building or car from multiple angles. The more points of view one has, the more one learns about it. In clinical practice, twelve leads are usually used in the diagnostic ECG, although there is no limitation to the number of leads one may select for special purposes.

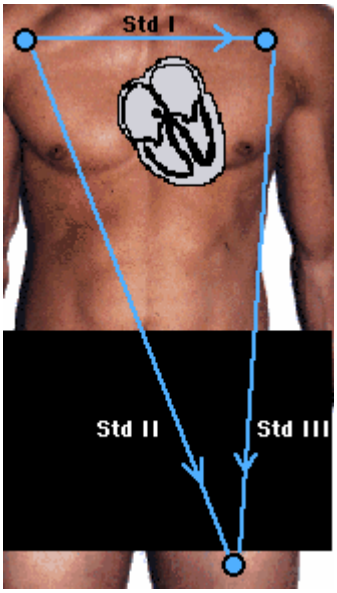

Fig. 5.5: Standard Leads

There are three of these leads which are usually designated as I, II and III. They are all bipolar leads (i.e., they detect a change in electric potential between two points) and detect an electrical potential change in the frontal plane. Lead I is between the right arm and left arm electrodes, the left arm being positive. Lead II is between the right arm and left leg electrodes, the left leg being positive. Lead III is between the left arm and left leg electrodes, the left leg again being positive. A diagrammatic representation of these three leads is termed Einthoven's triangle (shown below), after the Dutch doctor who first described the relationship. The central source of electrical potential in the triangle is the heart.

The same three leads that form the standard leads also form the three unipolar leads known as the augmented leads. These three leads are referred to as aVR (right arm), aVL (left arm) and aVF (left leg) and also record a change in electric potential in the frontal plane.

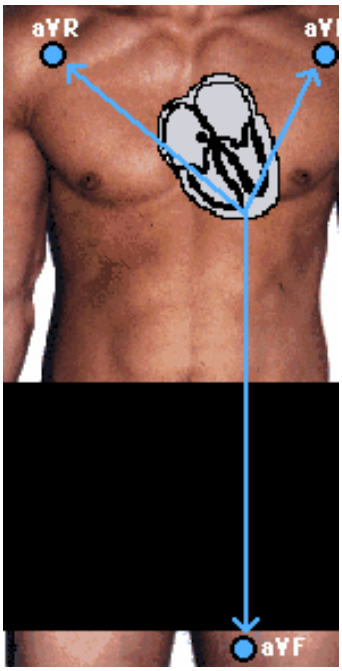

Fig. 5.6: Unipolar Leads

These leads are unipolar in that they measure the electric potential at one point with respect to a null point (one which doesn't register any significant variation in electric potential during contraction of the heart). This null point is obtained for each lead by adding the potential from the other two leads. For example, in lead aVR, the electric potential of the right arm is compared to a null point which is obtained by adding together the potential of lead aVL and lead aVF.

Precordial Leads

These six unipolar leads, each in a different position on the chest, record the electric potential changes in the heart in a cross sectional plane. Each lead records the electrical variations that occur directly under the electrode. The first chest lead is called V1 and is placed just to the right of the sternum in the fourth intercostals space. Chest lead V2 is placed just to the left of the sternum in the same space. Chest leads V3 through V6 are sequentially further to the left. These six unipolar leads, record the electrical variations that occur directly under the electrode. The first chest lead is called V1 and is placed just to the right of the sternum in the fourth intercostal space. Chest lead V2 is placed just to the left of the sternum in the same space. Chest leads V3 is placed between V2 and V4. Lead V4 is placed in the 5th intercostal space in the mid clavicular line.Leads V5 and V6 are placed horizontal to V4 ,in the anterior and mid axillary line.

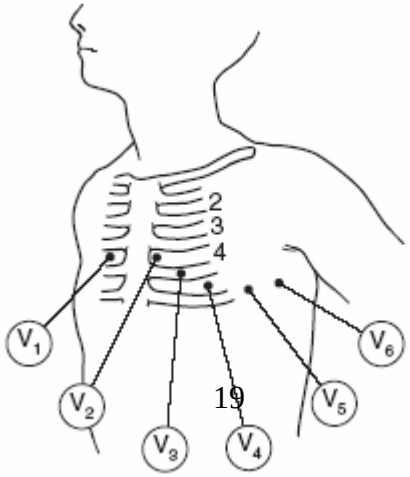

Fig. 5.7: Positioning of the Chest V Leads

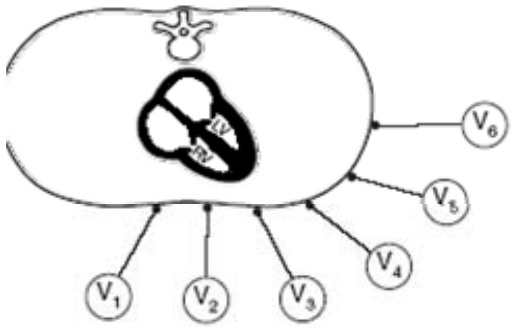

Fig. 5.8: The relationship between the six chest leads and the heart

Each of the 6 chest leads is a positive lead. The patient's back is considered the negative electrode for each.

A standard ECG machine records leads I, II and III simultaneously, then aVR, aVL, and aVF simultaneously, then V1, V2, and V3 simultaneously and finally V4, V5, and V6 simultaneously:

Standards Conventions when Reading an ECG

The rate of paper (i.e. recording of the ECG) is 25 mm/s which results in:

- 1 mm = 0.04 second (or each individual block)
- 5 mm = 0.2 second (or between 2 dark vertical lines)

The voltage recorded from the leads is also standardized on the paper where 1 mm = 0.1 mV (or between each individual block vertically) This results in:

- 5 mm = 0.5 mV (or between 2 dark horizontal lines)
- 10 mm = 1.0 mV (this is how it is usually marked on the ECG's)

The following picture shows ECG paper with conventional measurements.

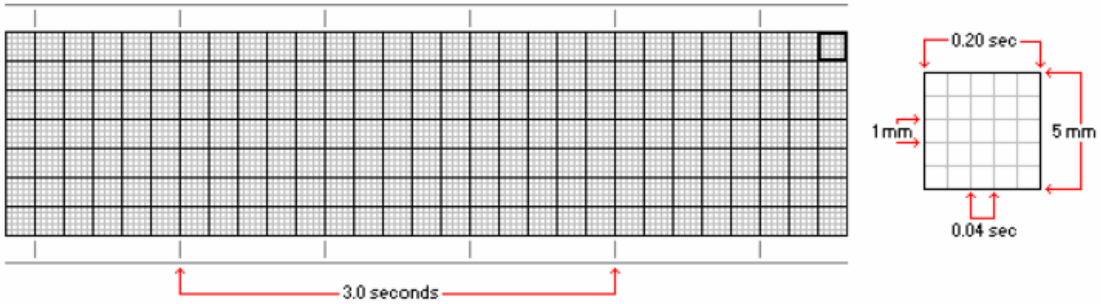

Fig. 5.9: ECG paper with its measurement

Check Your Progress 2

1) What are the three components that make up the ventricular depolarization wave?

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2) What is the QT interval and how is it measured?

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3) What does the ST segment represent and how is it measured?

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4) From .which part of the chest are the 6 precordial leads recorded?

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5.5 AXIS OF ECG

The average direction of spread of the depolarization wave through the ventricles as seen from the front is called the 'cardiac axis'. The mean electrical axis of the QRS complex is the average of the total depolarization of the ventricles in the frontal plane. There is a correlation between the mean electrical axis of the QRS complex and the electrical activity of the heart. It is useful to decide whether this axis is in a normal direction or not. Therefore, in pathologic hypertrophy of either ventricle (i.e. when there is more muscle being depolarized), the axis tends to shift in the direction of the hypertrophied ventricle.

Direction of Depolarization (Vector) of the QRS Complex

1) The left ventricle is thicker so the mean QRS vector is down and to the left. (The origin of the vector is the AV node with the left ventricle being down and to the left of this).

2) The vector will point toward hypertrophy (thickened wall) and away from the infarct (electrically dead area).

The QRS complex morphologies in frontal plane leads are variable. The mean QRS axes more positive than + 90 degrees represent right axis deviation. The axis more negative than – 30 degrees represents left axis deviation. Mean axes lying between – 90 and 180 degrees (or equivalently between + 180 and + 270 degrees) are referred to as extreme axis deviations. The

designation indeterminate axis is applied when all six extremity leads show biphasic (QR or RS) patterns; this finding can occur as a normal variant or may be seen in a variety of pathological conditions. Usually if the axis is abnormal aVF is predominantly negative.

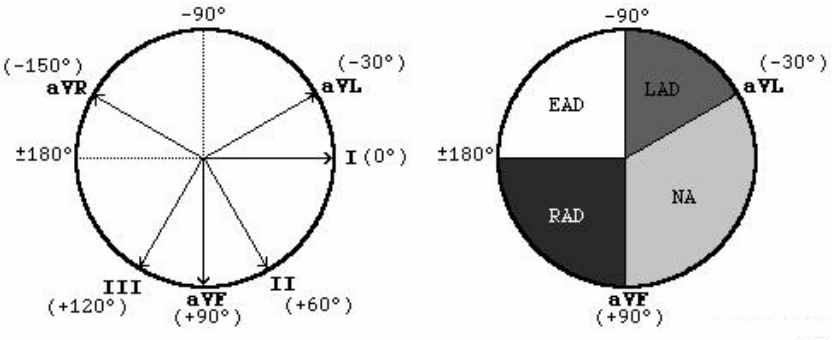

Fig. 5.10 (a) and (b): Axis of ECG

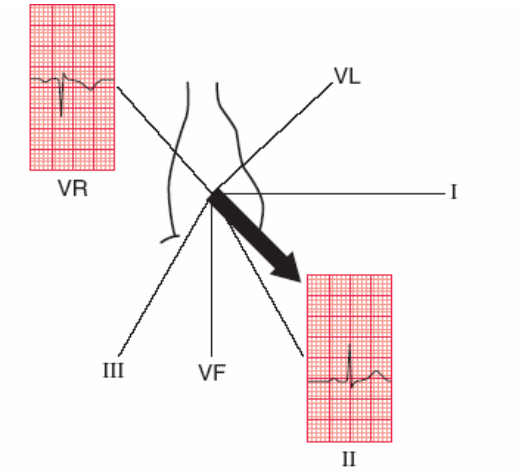

Fig. 5.11: Negative axis of ECG

Leads VR and II look at the heart from opposite directions. Seen from the front, the depolarization wave normally spreads through the ventricles from 11 o'clock to 5 o'clock, so the deflections in lead VR are normally mainly downward (negative) and in lead II mainly upward.

How to Determine Frontal Plane QRS Axis?

Lead I and aVF

Since lead I and aVF are perpendicular to each other, one can use those two leads to quickly determine the quadrant in which the QRS axis falls. Lead I runs from right to left across a patient's body, positive at the left hand. If the QRS in lead I is positive (mainly above the baseline), the direction of depolarization will be in the positive half (right half) of the circle above. Lead aVF runs from top to bottom across a patient's body, positive at the feet. If the QRS in lead aVF is positive (mainly above the baseline), the direction of depolarization will be in the positive half (lower half) of the circle above. If one realizes that there are two leads to consider and a positive (+) or (-) orientation for each lead, there would be four possible combinations. Memorize the following axis guidelines.

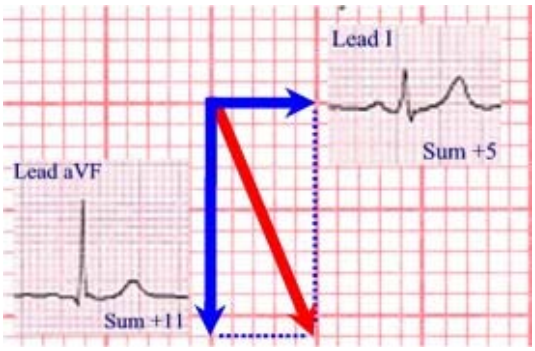

Fig. 5.12: Axis deviation of ECG

Subtract the number of little squares below the baseline in lead I from the number above the line (+ 5 mm in the example) and plot horizontally towards the lead. Repeat for lead AVF (+11 in the example) and plot vertically towards the lead. If the sum is negative then plot the number of squares away from the lead. The bottom line is, *if the axis is shifted out of the normal quadrant*, evaluate the reasons for this.

Lead I, II and III for Axis Determination

The direction of the axis can be derived easily from the QRS complex in leads I, II and III. A normal 11 o'clock–5 o'clock axis means that the depolarizing wave is spreading towards leads I, II and III and is therefore associated with a predominantly upward deflection in all these leads; the deflection will be greater in lead II than in I or III If the right ventricle becomes hypertrophied, the axis will swing towards the right the deflection in lead I becomes negative (predominantly downward) and the deflection in lead III will become more positive (predominantly upward). This is called 'right axis deviation'. It is associated mainly with pulmonary conditions that put a strain on the right side of the heart, and with congenital heart disorders.

Axis in Degrees-using Six Limb Leads

The cardiac axis is at right angles (90°) to the lead in which the R and S-waves are of equal size.The axis points towards any lead where the R-wave is larger than the S-wave. It points away from any lead where the S-wave is larger than the R-wave.

The cardiac axis is sometimes measured in degrees though this is not clinically particularly useful. Lead I looks at the heart 0°; lead II from +60°; lead aVF from +90°; and lead III from +120°. Leads aVL and aVR are said to look from –30° and –50°, respectively. The normal cardiac axis is in the range –30° to +90°. For example, if in lead II the size of the R-wave equals that of the S-wave, i.e, QRS is equiphasic, the axis is at right angles to lead II. In theory, the axis could be at either –30° or +150°. If lead I shows an R-wave greater than the S-wave, the axis must point towards lead I rather than lead III. Therefore, the true axis is at –30° – this is the limit of normality towards what is called the 'left'. If in lead II the S-wave is greater than the R-wave, the axis is at an angle of greater than –30°, and left axis deviation is present. Similarly, if the size of the R-wave equals that of the S-wave in lead I, the axis is at right angles to lead I or at +90°. This is the limit of normality towards the 'right'. If the S-wave is greater than the R-wave in lead I, the axis is at an angle of greater than +90°, and right axis deviation is present.

Summary

Using leads I and aVF the axis can be calculated to within one of the four quadrants at a glance.

If the axis is in the “left” quadrant take your second glance at lead II.

- both I and aVF +ve = normal axis
- both I and aVF -ve = axis in the northwest territory
- lead I - e and aVF +ve = right axis deviation
- lead I +ve and aVF -ve
- lead II +ve = normal axis
- lead II -ve = left axis deviation

Causes of Northwest Axis (No Man’s Land)

- emphysema
- hyperkalaemia
- lead transposition
- artificial cardiac pacing
- ventricular tachycardia

Causes of Right Axis Deviation

- normal finding in children and tall thin adults
- right ventricular hypertrophy

Chronic Lung Disease Even Without Pulmonary Hypertension

- anterolateral myocardial infarction
- left posterior hemiblock
- pulmonary embolus
- Wolff-Parkinson-White syndrome-left sided accessory pathway
- atrial septal defect
- ventricular septal defect

Causes of Left Axis Deviation

- left anterior hemiblock

- Q-waves of inferior myocardial infarction
- artificial cardiac pacing
- emphysema
- hyperkalaemia
  - — Wolff-Parkinson-White syndrome-right sided accessory pathway
  - — tricuspid atresia
  - — ostium primum ASD
  - — injection of contrast into left coronary artery

Check Your Progress 3

What is meant by right axis and left axis deviation?

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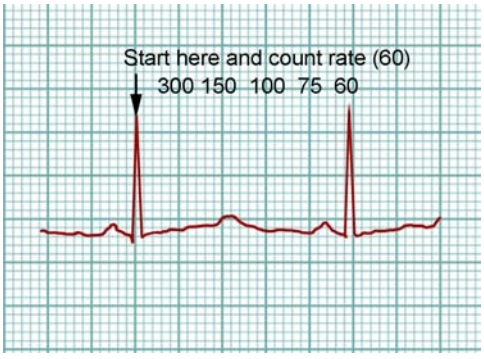
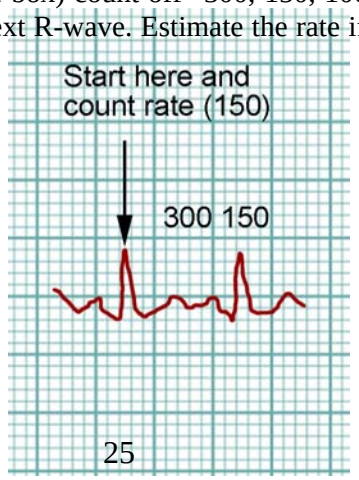
5.6 CALCULATION OF RATE OF ECG

Rate is cycles or beats per minute. Sinoatrial rates are normally between 60-100/minute. Rates less than 60 constitute bradycardia while the rates above 100 is called tachycardia in a normal adult. SA node is the usual pacemaker, other potential pacemakers (if SA node fails) are atrial pacemakers with inherent rates of 60-80, AV node (rate 40-60), or ventricular pacer (rate 20-40). In certain pathologic conditions ectopic pacemakers can go much faster at rates 150-250 cycles/minute and usurp the control of the SA node. There are three methods of calculating rate:

Most Common Method (Most Rates can be Calculated this Way)

Find an R-wave on a heavy line (large box) count off "300, 150, 100, 75, 60, 50" for each large box you land on until you reach the next R-wave. Estimate the rate if the second R-wave doesn't fall on a heavy black line.





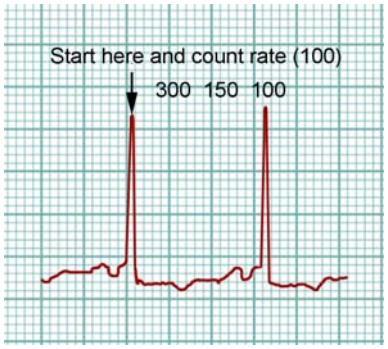


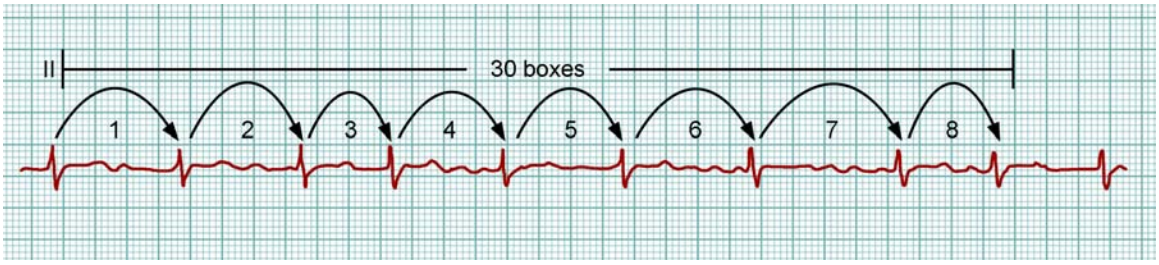
Fig. 5.13 (a), (b) and (c): Common Methods of Calculation of Heart Rate of ECG

Mathematical Method

This method is recommended if there is regular bradycardia, i.e. - rate < 50. Divide 300 with the total number of large boxes between two R-waves. This gives the ventricular rate.

Six-second Method

Count off 30 large boxes = 6 seconds (remember 1 large box = 0.2 seconds, so 30 large boxes = 6 seconds). Then, count the number of R-R intervals in six seconds and multiply by 10. This is the number of ventricular beats per minute. This is most useful if there is an irregular rhythm (like atrial fibrillation) when one wants to know an average rate. All these methods assume the speed of ECG recording at 25mm/second.



5.14: Six second method of calculation of heart rate

Check Your Progress 4

What is the PR interval and how is it measured?

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5.7 LET US SUM UP

In this unit, you have learnt the basic concept of electrocardiography. Electrical activity generated from sinoatrial node and each electrical impulse travel through internodal atrial conduction tracts to atrioventricular node and then further through its conduction system. Various types of waves has been form i.e. P-wave, QRS complex and T-wave generated during this process.

You have also learnt that how to record ECG, its various leads, cardiac axis and causes of deviation of different axis. Towards end of the unit you have leant how to calculate the rate of cardiac cycle.

5.8 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) The electrical conduction system of the heart, which produces the electrical activity measured by the electrocardiogram, is composed of the sinoatrial (SA) node, the internodal and interatrial conduction tracts, the atrioventricular (AV) junction (consisting of the atrioventricular node and the bundle of His), the right and left bundle branches, and the Purkinje fibers.

2) Once the wave of depolarization reaches the AV node, there is a delay in the AV node before the impulse passes down to the ventricle.This allows the atria to contract, thus emptying blood into the ventricles before the ventricles are stimulated to contract.

3) Each heart beat results in 3 "waves" or deflections on an ECG. In a normal cardiac cycle, the P wave occurs first, followed by the QRS complex and the T wave.

Check Your Progress 2

1) The ventricular depolarization is shown on the ECG by a large complex of three waves: the **Q,** the **R**, and the **S** waves. Together, these three waves are called the **QRS complex**.

2) The total amount of time the ventricles are electrically active (from onset of depolarization to completion of repolarization) is represented by the QT interval. This is measured from the onset of the QRS to the end of the T wave.

3) The ST segment represents the early part of repolarization of the ventricles.

   The ST segment begins at the J point ends with the onset of the T wave. The J point is the point at which the QRS complex ends and the ST segment begins.

4) Precordial Leads: These six unipolar leads, record the electrical variations that occur directly under the electrode. The first chest lead is called V1 and is placed just to the right of the sternum in the fourth intercostal space. Chest lead V2 is placed just to the left of the sternum in the same space. Chest leads V3 is placed between V2 and V4. Lead V4 is placed in the 5th intercostal space in the mid clavicular line.Leads V5 and V6 are placed horizontal to V4 ,in the anterior and mid axillary line.

Check Your Progress 3

The QRS complex morphologies in frontal plane leads are variable. The mean QRS axes more positive than +90 degrees represent right axis deviation. The axis more negative than –30 degrees represents left axis deviation.

Check Your Progress 4

The PR interval extends from the beginning of the P wave (the beginning of atrial depolarization) to the onset of the QRS complex (the beginning of ventricular depolarization). It should not exceed 0.20 seconds as measured on ECG graph paper, where each small square represents 0.04 seconds. In other words, the PR interval should not exceed five little boxes in width.