CARDIOLOGY

FOR THE HOUSE OFFICER

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Williams & Wilkins

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Cardiology for the House Officer

Preface

This book grew out of the need for a concise reference resource covering common problems in cardiology encountered by the house staff at Harbor-UCLA Medical Center. The book began in 1978 as a looseleaf notebook which was field tested by the medical housestaff and cardiology fellows at Harbor and Huntington Memorial Hospitals. Each section was extensively re-written in response to new developments as well as suggestions by the book's users and illustrations were chosen from our case files.

In its present form it was an attempt to provide the house officer or generalist with core material in clinical and laboratory cardiology as well as tables for ready reference and a bibliography for more in-depth information.

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Chapter 1

Basic Electrocardiography

The following chapter reviews the causes of abnormalities of atrial and ventricular depolarization and repolarization which may be recognized on the electrocardiogram (ECG). Such abnormalities are manifested as abnormalities in P wave, QRS complex, ST segment, and/or T wave morphology or duration. Arrhythmias may also produce changes in the morphology and/or duration of the ECG deflections and will be discussed in a separate chapter (Chapter 15).

It is beyond the scope of this text to provide a comprehensive presentation of basic electrocardiography and vectorcardiography. Discussion of electrophysiologic mechanisms underlying changes in the surface ECG are minimized in the following discussion and it is assumed that the house officer has a basic understanding of ECG vector analysis (calculating mean axes). Emphasis has been placed upon the differential diagnosis of QRS, ST, and T wave changes and providing the house officer with readily available ECG criteria for the diagnosis of commonly encountered ECG abnormalities. Comprehensive discussions of basic electrocardiography may be found in a number of texts(1-3).

THE NORMAL ECG--WAVES, INTERVALS, AND SEGMENTS

The ECG is the surface representation of cardiac electrical activity. During myocardial depolarization and repolarization, deflections or waves are inscribed on the ECG. By convention, positive forces (electrical forces directed toward the ECG lead) produce upright deflections and negative forces (forces directed away from the ECG lead) are represented by downward deflections. The distance between deflections and waves are called segments and intervals, respectively.

THE P WAVE

- represents atrial depolarization
- P wave duration (width) is a measure of the time required for depolarization to spread through the atria to the atrioventricular (AV) node. In the normal

adult, P wave duration is < 0.10 sec
- mean frontal plane P wave axis (vector) is normally directed inferiorly and leftward (15-75°) and an upright deflection should be recorded in ECG leads I, II, and aVF. A negative deflection is seen in aVR. The P wave may be upright, isoelectric, (flat), or inverted in III and aVL

THE PR INTERVAL

- represents the time required for a supraventricular impulse to depolarize the atria, traverse the AV node, and enter the ventricular conduction system
- measured from the beginning of the P wave to the initial deflection of the QRS complex (Q or R wave) in a frontal plane lead
- normal PR interval is 0.12-0.20 sec in adults in sinus rhythm. The PR interval normally shortens as heart rate increases and lengthens at slower heart rates. First degree AV block is said to be present if the PR interval is > 0.20 sec. A PR interval of < 0.12 sec may be seen as a normal variant, in hypocalcemia, with ventricular pre-excitation, and in ectopic rhythms

THE QRS COMPLEX

- represents ventricular depolarization
- Q wave--the first downward deflection after the P wave and/or preceding the first upright deflection
- R wave--the first positive deflection
- S wave--a negative deflection following an R wave
- QS wave--a single downward deflection not preceded or followed by an upright deflection
- R' wave--a second positive deflection after the R wave
- upper and lower case letters are frequently used to signify approximate voltages or amplitudes
- QRS interval (duration)--an indication of intraventricular conduction time. In normal adults, QRS duration is < 0.10 sec measured in the frontal plane leads
- the mean frontal plane QRS vector is -30 to +90°
- the amplitude of R and S waves and the QRS axis are indicators of myocardial muscle mass and chamber size

THE ST SEGMENT

- an isoelectric segment following ventricular depolarization and preceding ventricular repolarization
- measured from the end of the QRS complex to the beginning of the T wave
- in contrast to the PR and QRS intervals, changes in the length of the ST segment are not as important

as its deviation from baseline or the isoelectric point. The interval from the end of the T wave to beginning of the P wave (TP interval) is usually taken as the isoelectric reference point. Deviation of ±1 mm from the isoelectric baseline is considered "abnormal" but not necessarily pathologic

THE T WAVE

- ECG representation of ventricular repolarization
- T wave vector normally directed inferiorly and leftward
- the T wave vector normally "tracks" with the QRS vector. If the QRS is predominantly negative in a standard lead, an inverted T wave is usually seen and is not necessarily abnormal
- an inverted (negative) T wave in V1 is considered normal. Inverted T waves in V2 and V3 may be normal in patients 30 years of age and in patients with "funnel chest" or "straight back" body habitus

THE QT INTERVAL

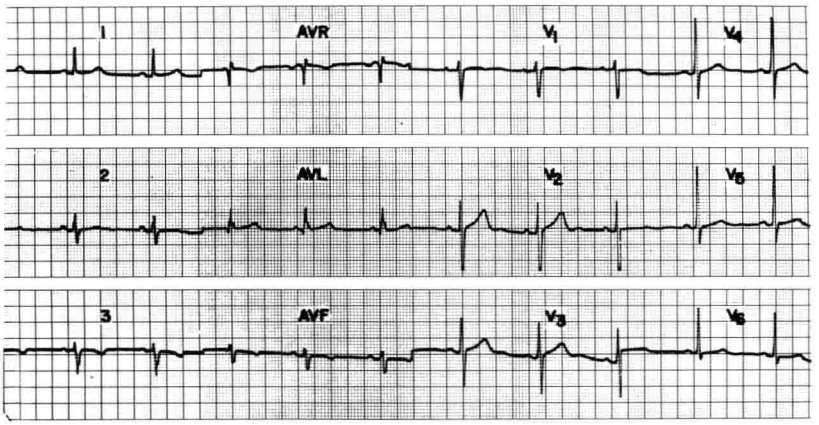
- measured from the beginning of the QRS complex to the end of the T wave and represents duration of electrical systole. Mechanical systole usually begins between the QRS complex and the T wave
- this interval varies with heart rate. The QT interval, corrected for heart rate, is usually $<$ .47 sec and is calculated by dividing the measured QT interval (in sec) by the square root of the R-R interval (in sec)

THE U WAVE

- a deflection following the T wave; electrophysiologic origin uncertain
- U wave vector tracks with the T wave vector, i.e., polarity or direction similar
- amplitude usually $>$ 10% of the amplitude of the preceding T wave; the larger the T wave, the more prominent the U wave

Figure 1.1 is an example of a normal ECG. The PR interval is 0.16 sec, the QRS duration 0.08 sec, and QT interval (corrected for heart rate) 0.42 sec. The P wave axis is normal (upright P waves in leads I, II, aVF). The mean frontal plane QRS axis is -30^{o} and the T wave axis is -10^{o}. Negative T waves are noted in III and aVF but are normal (angle between QRS axis and T wave axis is $< 45^{o}$).

Figure 1.1

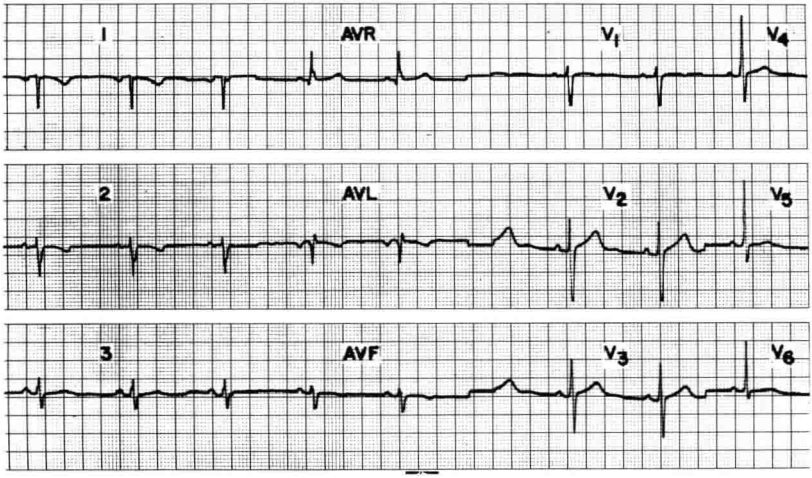


COMMONLY ENCOUNTERED ECG ABNORMALITIES

MISPLACED ECG LEADS

Figure 1.2 is an ECG tracing obtained from the same patient as in 1.1. Note the difference in the axis of the P wave, QRS complex, and T wave recorded in the standard limb leads. This is a common technician error caused by reversing the right and left arm ECG leads. In switching the right and left arm leads, the frontal plane P, QRS, and T wave axes have been shifted rightward. Such frontal plane vector changes may also be seen dextrocardia. However, the presence of a normal precordial (V1-V6) ECG eliminates dextrocardia as a differential possibility. With dextrocardia horizontal plane QRS forces should be directed anteriorly and to the right, with decreasing R wave amplitude over the left precordium.

Figure 1.2



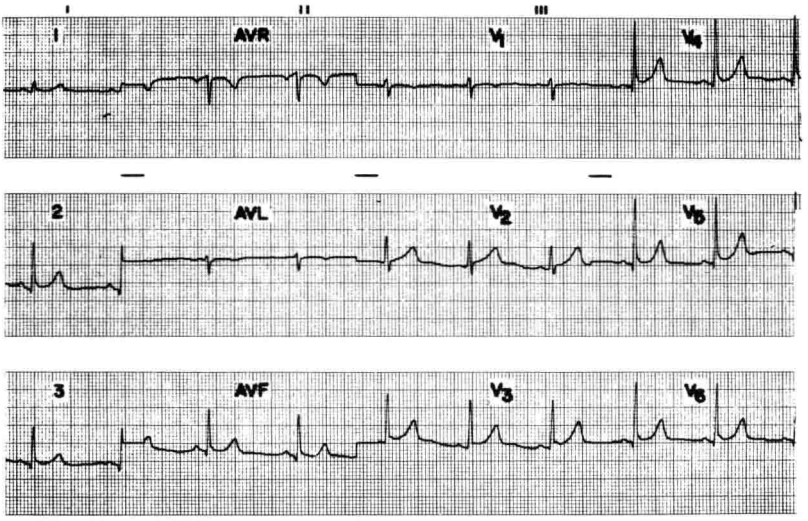
EARLY REPOLARIZATION

Normal variant ST segment elevation may be noted in the precordial and limb leads. "Early repolarization" is the descriptive term applied to this ECG pattern. Whether accelerated subepicardial repolarization is actually responsible for the ECG variant is uncertain. ST segment elevation is most prominent in the lateral precordial leads (V4-V6). The T wave in these leads are characteristically broad-based, tall, (usually $>$5mm) and upright. The limb leads may also show some degree of ST elevation, but rarely greater than 2mm. The early repolarization variant has been reported in all age groups, is more common in males, and is more prevalent in the black than the white population.

This variant may be confused with the ST segment changes noted during acute pericarditis or acute transmural myocardial infarction. The ratio of the ST segment amplitude to the T wave amplitude in lead V6 helps to distinguish the benign variant. In early repolarization, the ST/T ratio in V6 is

generally less than 0.25. A ratio greater than 0.25 is is usually seen in acute infarction and pericarditis. In the example (Figure 1.3), the ST/T ratio is 0.14.

Figure 1.3



PERICARDITIS

The ECG may be of considerable value in the diagnosis of pericarditis, especially if serial tracings are obtained. Evolutionary changes (stages) may be noted over several days.

Stage 1 (acute phase) (Figure 1.4)
- ST segment elevation in the precordial leads, especially V5 and V6, and in leads I and II
- ST/T wave ratio in V6 > .25 (ST/T ratio 0.6 in example-figure 1.4)
- An isoelectric or depressed ST segment is commonly seen in V1
- PR segment depression may be noted in leads II, aVF, V4-V6

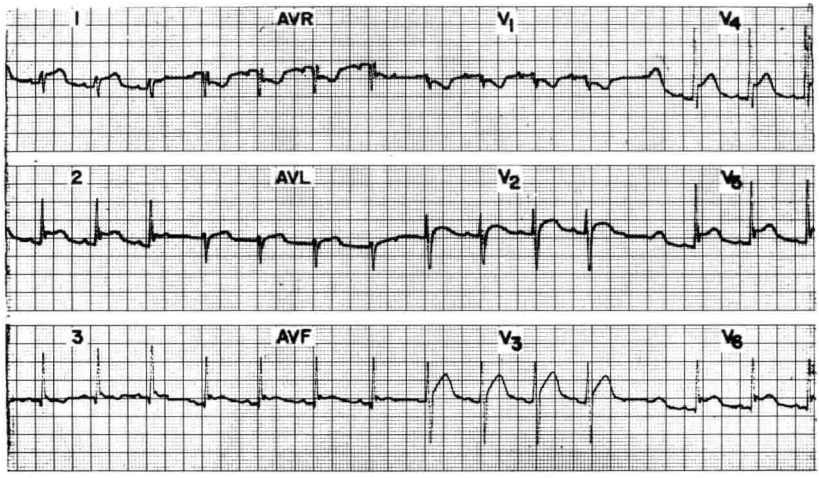
Stage 2
- ST segment begins returning to baseline (isoelectric line)
- T wave amplitude decreases

Stage 3
- ST segment isoelectric
- T waves inverted in those leads previously showing ST segment elevation

Stage 4
- resolution of T wave changes

Additionally ECG abnormalities may be noted during pericarditis (see Chapter 14) and include arrhythmias, low voltage QRS complexes (<7mm from depth of Q wave to peak of R wave in extremity leads), and electrical alternans (beat-to-beat variation in R wave amplitude).

Figure 1.4 - Acute Pericarditis



THE ECG IN CHAMBER ENLARGEMENT

ATRIAL ENLARGEMENT

Atrial enlargement may result from:

- valvular heart disease (e.g., mitral stenosis)
- pulmonary hypertension

- congenital heart disease (e.g., tricuspis atresia)
- ventricular hypertrophy (e.g., systemic hypertension)

The initial portion of the P wave results from right atrial depolarization and the terminal portion from left atrial depolarization. The normal mean P vector is the sum of the vectors generated by both atria and is directed leftward and inferiorly. Atrial enlargement may alter the P wave magnitude, duration, or vector orientation.

An accurate ECG diagnosis of atrial enlargement is not always possible. There is considerable normal variation in P wave amplitude, duration, and morphology. Tachycardia alone may increase P wave amplitude. Although it is not always possible to differentiate between left and right atrial enlargement electrocardiographically, the following criteria may be helpful:

RIGHT ATRIAL ENLARGEMENT (Figure 1.6)

- P wave $\geq$ 2.5 mm in lead II, III, or AVF
- Frontal plane P wave vector shifted rightward ($>+ 75^{o}$)

LEFT ATRIAL ENLARGEMENT (Figure 1.6)

- P wave duration $\geq$ .11 sec and P wave usually notched in lead II
- Frontal plane P wave vector shifted leftward $\leq + 30^{o}$ (terminal part of P wave may be negative in lead III and AVF)
- Biphasic P wave in lead V1 with wide, deep terminal component ($\geq$ .04 sec in duration and 1 mm in depth).

LEFT VENTRICULAR HYPERTROPHY (LVH)

The term left ventricular hypertrophy (LVH) refers to both hypertrophy and/or dilatation of the ventricle. In most instances in which LVH is diagnosed electrocardiographically, both components will be present. LVH manifests itself primarily as an increase in voltage (height of R wave) in those ECG leads which reflect left ventricular potentials. The increase in voltage is due to an increase in muscle mass and surface area and/or the proximity of the dilated heart to the sensing ECG electrode (heart closer to chest wall). The mean QRS vector tends to be rotated leftward and posteriorly. LVH does not change the sequence of ventricular depolarization but may delay it (delayed onset of intrinsicoid deflection). Repolarization may be altered--the left precordial leads may show depressed ST segments and inverted T waves--resulting in a left ventricular strain pattern. The ECG diagnosis of LVH is based on voltage changes, ST-T wave alterations, axis deviations and conduction delay (QRS dura-

tion > 0.08 sec but < 0.12 sec). A number of ECG criteria have been advanced, all of which have a relatively low sensitivity but a high degree of specificity.

Common ECG Voltage Criteria for LVH in the Adult

- sum of S wave in V1 and R wave in V5 or V6 ≥ 35 mm, or
- sum of maximum R and deepest S waves in precordial leads ≥45 mm
- R wave in V6 ≥18 mm, or
- R wave in aVL ≥11 mm, or
- sum of R wave in I and S wave in III ≥25 mm.
- QRS duration > .10

Limb lead criteria are less sensitive but highly specific.

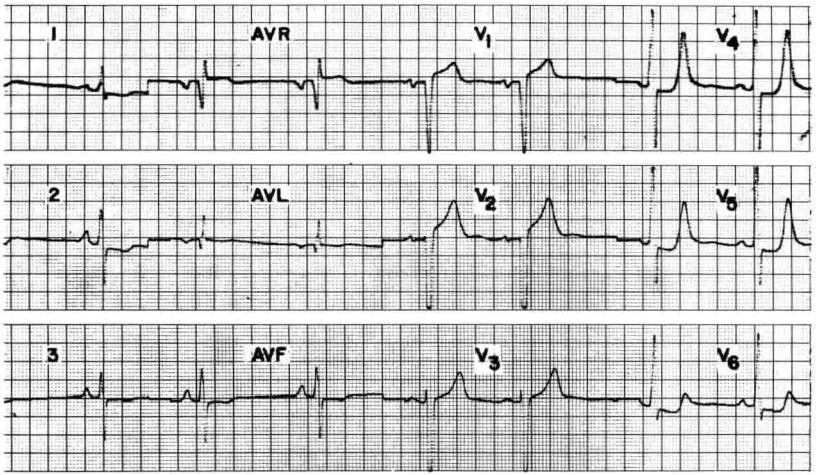
ST-T Wave Changes (strain pattern)

- downsloping ST segment depression and asymmetric T wave inversion in V4-V6 with
- ST-T wave changes in II, III, aVF if mean QRS axis vertical or
- ST-T wave changes in I and AVL if axis horizontal
- ST segment elevation may be seen V1-V3.

Strain pattern alone without voltage changes is not sufficient for diagnosis of LVE. Similar ST-T wave changes may be caused by subendocardial ischemia, digoxin, hypokalemia, phenothiazines and myocarditis.

Probable LVH - voltage criteria alone
Definite LVH (Figure 1.5) - voltage changes and ST-T changes with left atrial enlargement and/or left axis deviation(Figure 1.6).

Figure 1.5 - LVH



RIGHT VENTRICULAR HYPERTROPHY (RVH)

The ECG diagnosis of RVH is less accurate than that of left ventricular hypertrophy, and the ECG is frequently normal in the presence of RVH. The following ECG abnormalities are suggestive of RVH:

- right axis deviation (>+ 110° in an adult) in the absence of right bundle branch block, left posterior inferior fascicular block, or anterolateral or inferior myocardial infarction
- dominant R wave (>5mm) in lead V1 (a dominant R wave may also be seen in RBBB, posterior myocardial infarction, WPW syndrome, and as a normal variant.)
- R/S ratio in lead V1 greater than 1.0
- R/S ratio in lead V6 less than 1.0
- R' pattern in lead V1 with a QRS duration of less than .12 sec

The diagnosis of RVH is supported by the presence of RAE and/or right ventricular strain pattern (ST segment depression and T wave inversion in V1-V3). The ECG diagnosis of RVH may be obscured if a RBBB is also present.