Electrocardiographic Abnormalities in Normal and Apparently Normal Individuals*

DR. RUSTOM JAL VAKIL**

Despite the fact that a massive literature has grown within the past few decades on the subject of electro-cardiography, considerable confusion still exists in the minds of many as to what exactly constitutes "normal" and what is "abnormal" or "pathological" in electrocardiographic interpretation.

Time and again, one encounters difficulty in deciding whether a given deviation from the accepted normal of an electrocardiographic"wave" or "segment" should be considered a normal variant or a pathological manifestation indicative of disease. Many of the so called atypical electrocardiographic features enumerated here are common to both normal and diseased, thus making at times their true interpretation and significance extremely difficult. To facilitate these distinctions between "normal" and "abnormal" certain criteria of normality have to be remembered in the case of each wave and segment of the electrocardiogram.

For convenience, the various waves and segments of the electrocardiogram are considered in serial order.

The P wave

The 'P' is normally upright in the electrocardiographic leads, but it may be negative in lead III and aVL without being pathological. Of course, in aVR it is always negative, this being a mirror-image lead.

In normals, the voltage of P is less than 2.5 mm. and the duration (the width of its contour on the base line) is less than 0.11 second. Slight slurring or notching of the upstroke of 'P' is not significant. The initial part of 'P' is due to right atrial depolarization and the terminal part due to left atrial depolarization.

In dextrocardia and levocardia, when the SA node is on the left side of the heart the 'P' in lead I is normally inverted. Inversion of the P wave in leads II, III, aVF, V5 and V6, with normal upright 'Ps' in leads I, aVR and aVL and diphasic 'P_S' in V1 and V2 suggests a "nodal rhythm" which may exist in the absence of organic heart disease.

A large, prominent, broad and notched, slurred or double-peaked 'P' wave in lead

* Lecture delivered on 8th December 1972, at the Institute of Aviation Medicine IAF, Bangalore.

** Honorary Consulting Physician, K.E.M Hospital Bombay, and Visiting Cardiologist, Nanavaty Hospital, Bombay.

I and aVL (with a duration of over 0.11 second),with small or diphasic 'P' waves in leads II, III and aVF and large and inverted 'P'waves in leads V1 and V2, is pathological and suggests a left atrial hypertrophy, as in "mitral stenosis" (mitral 'P' or 'P' mitrale).

Tall and peaked'P'waves(with an amplitude of over 2.5 mm, but with a normal duration) in leads II, III and aVF, with low or flat 'P waves in leads I and aVL and tall upright'P'waves in the right chest leads(V1 and V3R) or in a space higher than V1 are suggestive of right atrial hypertrophy as in "cor pulmonale" or "congenital heart disease" ('P' pulmonale or 'P' congenitale) and are always pathological.

In atrial infarction, certain changes in 'P' wave contour or in the atrial T wave (Ta) with depression of P–R interval or some atrial arrythmia or conduction disturbance may be observed.

The P–R Interval

The interval from the onset of the 'P' wave to the onset of the QRS is called the P–R interval.It represents the time taken by an impluse leaving the sinus node to reach the ventricles. The normal duration of P-R ranges from 0.12 to 0.21 second,-a duration that must be recorded in all the leads. The lead that shows the longest duration of P-R should be accepted for measurement. A depression of P-R or PQ of upto 1 mm. may be normal and due to the imposition of the Ta or auricular T wave on the P-R segment. The maximal normal duration of PR is 0.21 second for adults, 0.18 for children and 0.16 for infants.

A P–R prolongation or increased duration of over 0.21 second may be due to heart disease(with first degree A- V block) or non-cardiac causes (digitalis, quinidine or infections). At times, a prolonged P–R interval of over 0.21 second is encountered in perfectly normal healthy individuals. When neutralized or abolished by an injection of atropine, such a P–R prolongation is probably due to increased vagal tone. If not abolished, it is likely to be due to some unrecognised or incipient diphtheretic or rheumatic infection (fresh or even old) causing a local partial A-V block. Transitory prolongation of the P-R may be due to some infection, like rheumatic fever.

A shortened P-R interval of less than 0.10 second is usually due to nodal rhythm the presence of an ectopic pace-maker (shift of the SA pacemaker to the AV node or auricles), a coronary sinus rhythm, or in accelerated conduction or the WPW syndrome.

The QRS Complex

In normal individuals, a low voltage of the QRS below 5 mm. in the standard leads may be observed as a normal variant but in the absence of other clinical and electrocardiographic abnormalities. Also a notching or slurring of QRS complexes may be observed normally in small-sized QRS deflections and the QRS deflections of V leads over transitional zone.

Abnormally low voltage of QRS (below 5 mm.) in all standard extremity leads, in conjunction with normal sized QRS in the precordial leads, may be seen in myocardial infarction, bundle branch block or abnormal electrical position of the heart.

Small QRS deflections, usually with other clinical or electrocardiographic abnormalities, are seen in congestive cardiac failure generalized oedema, pericardial effusion, constrictive pericarditis, pneumopericardium, pleural effusion, ascites and myxoedema (due to short circuiting of cardiac currents by the abnormal conduction through fluid or air.)

Notching or slurring of QRS in precordial leads may be due to AV block, intraventricular block, bundle branch block partial block or periinfarction block.

The Q Wave

Q in lead III. This is frequently observed in perfectly normal healthy individuals with a high Diaphragm(broad chested individuals with excess of abdominal fluid or gas or liver enlargement). In such cases, to distinguish a significant or infarctional from a harmless or insignificant Q wave of a high diaphragm, the following features are of value : (i) A deep inspiration (IIIR) eliminates the Q wave of a high Diaphragm in lead III. (ii) Pardee's criterion. The Q waves when significant is deep (over 25 per cent of the highest R voltage.) (iii) The Q wave in infarction is wide, broad or notched. (iv) There is the presence of a Q wave in leads II and aVF also.

Deep Q in III (and occasionally in aVF). Besides inferior wall myocardial damage, it may be at times observed in acute cor pulmonale, emphysema or right bundle branch block and rarely in left ventricular hypertrophy.

Q in aVF. This may occur normally with a vertical heart, but then, the Q is usually small and not wide or notched.

Deep Q wave in aVF. This may occasionally occur in normal obesity with the patient recumbent. To distinguish it from a pathological Q wave, the adoption of a sitting up or upright posture helps by eliminating the Q wave, in normal individuals. The rest of the electrocardiogram must also be evaluated to rule out myocardial disease.

A deep Q wave in aVF is usually observed pathologically in inferior or diaphragmatic infarction, and occasionally also in left bundle branch block, left ventricular hypertrophy (with QS in V1 and V2), acute cor pulmonale (due to extreme clockwise rotation with forward displacement of apex) and in right atrial or high right ventricular disease (where Q wave is also present in the form of a Qr wave in V1 and V3R).

Q in aVF. To be significant and suggestive of an inferior wall myocardial damage, (i) the aVF should have atleast one deflection of over 5 mm. in amplitude; (ii) Q must constitute at least 25 per cent of that deflection; (iii) the larger and wider the Q wave, the more is it likely to be infarctional in origin; (iv) associated ST or T wave changes are present with myocardial damage.

Inferior wall myocardial infarction without Q wave in III and aVF. It has been shown recently that even with a proved diaphragmatic myocardial damage, the Q or qs may be replaced in III and aVF by an "rs" or "rsR'" complexes.

qR in aVF. This may be due to a vertical heart (since the left ventricle faces the left hip). There is qR present in the

left ventricular leads also. The aVL shows an rS pattern.

qR or QR in aVL. This is seen in forward rotation of the apex. With moderate rotation, the LV potential (qR) is recorded in aVF. With marked forward rotation of the apex, the potential of the back of the heart (QR) is recorded with an inverted T wave in aVF.

Q wave in aVF. This may occure in: (a) Perfectly normal individuals with the socalled S_1 S_2 S_3 syndrome. All the three standard limb leads show prominent S waves with a usually normal QRS duration and a minute 'r' wave in lead VI. It can occure in perfectly normal healthy young adults with no evidence of heart disease (due to clockwise rotation with backward displacement of the apex). (b) A prominent Q wave (QS) may be normal in aVL in the case of the vertical heart. (c) There may be a QR wave in aVL with backward rotation of the apex (as in the S_1 S_2 S_3 syndrome) (d) In pregnant women. The S_1 S_2 S_3 syndrome can also occure abnormally in cases of (i) congenital heart disease with right ventricular hypertrophy, (ii) cor pulmonale, (iii) acute myocardial infarction and (iv) rarely in mitral stenosis with right ventricular hypertrophy. When the Q wave in aVL is within normal limits, the Q is less than 0.04 second in duration and is neither notched nor slurred.

A prominent Q wave (QS or Qs) in aVL, with an upright P wave and usually associated with ST-T changes suggests a high anterolateral myocardial damage.

qR complex in lead aVL. This can be due to a horizontal heart, with the LV facing the left shoulder (similar qR waves occur in the left ventricular leads, with an rS in aVF).

A QR complex with inverted or diphasic P and an inverted T in aVL (and aVR). This is seen in backward rotation of the apex (the back of the heart facing the right and left shoulders). The aVF shows an rS pattern with a positive or negative T wave (due to the right ventricular pattern.

QR pattern in aVR. This occurs with a clockwise rotation of the heart (since the back of the heart faces the right shoulder). Also, prominent S waves are present in the left ventricular leads. In marked rotation, there is also present a QR pattern in lead V1.

Presence of Q wave in V1 or in V1 and V2. Although this normally suggests a localized septal myocardial damage, it may occur on rare occasions in normal individual. In the case of the former, there is an initial R wave in the QRS of right sided leads (V3 R and V4 R) and ST-T abnormalities are present. In the absence of these changes, the Q wave in V1 (and rarely in V2) can be regarded as a normal variant.

Deep QS in right precordial leads V1, V2 and V3. This is most frequently observed in anteroseptal infarction, but also occurs in left bundle branch block and left ventricular hypertrophy. Occasionally, Q waves are observed in V1 (and perhaps V2) in normal individuals. They may also be observed in acute cor pulmonale with ST segment elevation.

Q wave in left ventricular leads. A Q wave may occur normally over the left

ventricular leads (V5, V6). However, a broad and deep Q wave in V5, V6, with ST or T waves changes or a small R wave should suggest some myocardial disease or damage.

Q wave in normal high chest leads. An initial q wave may be observed on rare occasions in normal individuals in the higher chest leads (V′ and V″)

A qR pattern beginning from V3 (instead of the usual V4 to V5 position) suggests a moderate counter-clockwice rotation. When beginning from V2 (with rS in V1), it suggests a marked counterclockwise rotation.

QS in I, III, aVL (or aVF) and chest leads. Although such a tracing is usually suggestive of an extensive myocardial infarct, it may arise in chronic cor pulmonale due to a low diaphragm or position of the heart with backward displacement of the apex, giving rise to intracavity potentials.

Q wave in I and aVL or/and V6. Though this picture is usual with anterolateral myocardial damage , it may occur in left ventricular diastolic overloading.

Tall prominent R in lead VI. This is usually indicative of a strict posterior myocardial damage, or right ventricular hypertrophy or a preexcitation syndrome.

Prominent R wave in V1 and V3R. The main causes are strictly posterior myocardial infarction, right ventricular hypertrophy, right bundle branch block and the preexcitation syndrome. In strictly posterior infarction, there is a tall, slurred, wide R wave ("the reciprocal sign") or an RSR¹ complex with an R/S ratio of 1 or more in V1 and V3R. No Q waves are observed anywhere except in the oesophageal leads. In high posterior or high posterolateral infarction, Sodi-Pallares reports tall R waves in V1, V2 and perhaps V3, with septal or left ventricular patterns in V4, V5, V6.

ST-T Complexes

The ST segment and T wave should be considered as a "single unit" to help unravel abnormalities of the last part of ventricular systole. The ST segment must be correlated with the succeeding T wave in order to enhance the evaluationing of the cardiogram.

In evaluation the ST-T complex, attention may be focussed also on the QRS complex, P wave and PR interval.

A mild elevation of the ST segment with a tall T wave is within normal limits, but the same deviation with a low T wave is abnormal.

The P wave affects the ST-T complex because a certain amount of gradient is present between the P wave and the Ta, the Ta frequently falling into the first part of the ST-segment, causing a deviation of the ST segment or depression of ST segment. The beginning of QRS complexes can be used as a reference level for the S-T junction in order to discount the effect of an unusually large Ta wave.

S-T junction abnormalities. The beginning of the QRS complexes is the reference level (Katz) for the S-T junction. (i) An ST junction elevation of more than 2 mm. is abnormal in the limb leads. (ii)

An S-T junction depression of more than 0.5 mm. in I and II or of more than 1 mm. in III is abnormal. Even smaller depressions are abnormal if the T wave is tall or large (in the limb leads).

S-T segement abnormalities. It is advisable to relate the ST segment slope to the ST junction. The "slope" and the "curvature" of the ST must be studied in each case.

The S-T segment may be horizontal or may slope upwards or dowards. The normal S-T segment slopes upward with a gradient, increasing at the end to meet the T wave. With an inverted T wave, the sloping is downwards.

An upward or downward "bowing" is significant, particularly if the T wave is in a direction opposite to the bowing of the S-T segments. An universally "short" or notched S-T segment is also considered abnormal.

For determining S-T segment deviation, the reference level recommended by the American Heart Association is the junction of PR interval with QRS complexes. This is not satisfactory when the Ta (repolarization of T wave of atrial beat) extends beyond the QRS causing depression or deviating S-T segment. Even the T-P may slope. In such cases, the T-P interval following it may be used as the reference level rather than the P-R interval. Also, if the heart rate is rapid, the T-P is better as a reference level.

Specific ST-T complex patterns. These are observed in : (1) Heart strain and intraventricular block. (2) Coronary insufficiency. (3) Digitalis effect. (4) Hyperthyroidism.

T Wave

This is usually positive in I, II, aVL, aVF. It is inverted normally in aVR and often in III and in aVL in vertical hearts. In the chest leads, it is positive except in V1 and occasionally in V2 and rarely in V3. In limb leads, T is normally over 1 mm. in amplitude. It may be as tall as 8 mm. in unipolar extremity leads and even 12 mm. in chest leads. A T wave less than 10 per cent of the amplitude of R wave in left ventricular leads is suspicious of disease.

T wave abnormalities are of two main types: (1) Primary (due to disturbed myocardial cellular metabolism) (2) Secondary (due to depolarization conduction disturbance, as in bundle branch block and ventricular extrasystoles).

Contour of the T waves. Primary T wave changes show (1) symmetrically inverted and peaked T waves. (2) Isoelectric or slightly elevated J point or ST junction. Secondary T wave changes show (1) asymmetric T waves (2) depression of ST segments with upward convexity and (3) depressed J points.

Primary T wave abnormalities, due to (a)normal or physiological conditions, such as head-up tilting, Valsalva manoeuvre, use of automomic drugs and neurocirculatory asthenia (b) Abnormal conditions e.g., (i) haemodynamic factors (e.g. increased intraventricular pressure or increase of thickness of ventricular wall). (ii) metabolic factors (normal or abnormal), e.g., in sleep, exercise and eating and in fever, myxoedema, malnutrition, anoxia.

electrolyte distubances, drugs, thyrotoxicosis, infections, shock and hepatitis. (iii) ischaemic factors. The T is inverted in the precordial leads in subepicardial ischaemia, normal in intramyocardial ischaemia and normal or tall and peaked (with pro longed QT) in subendocardial ischaemia.

The T wave amplitude, duration and direction are susceptible to a host of factors. The T wave is the "most labile and least specific feature" of the cardiogram.

"Isolated T negativity syndrome" Negative T waves in leads V3 or V4 may occur in healthy young adult males The mechanism is obscure. The condition is variable and may come and go. It is unaffected by an overdose of potassium with resultant hyperpotassemia (which normalizes most functional T wave abnormalities,) but is affected by exercise.

U wave

(1) U wave is normally present, but is very small and therefore missed in the limb leads but is better seen in the V leads (particularly in V3 to the right of the transitional zone).

(2) It is difficult to decipher when the T wave is notched or fused with the U wave.

(3) The cause of the U wave is obscure. It may be due to (a) late repolarization forces during the supernormal period of recovery or (b) to late repolarization forces in the septum or (c) due to negative afterpotentials (due to K ions being slowly reabsorbed during diastole.)

(4) Prominent U waves occur in hypopotassemia, bradycardia and many metabolic disturbances.

(5) U waves are absent in hyperpotassemia.

(6) Negative U waves in left ventricular leads suggest antero lateral ischaemia or left ventricular hypertrophy with strain. In right-sided chest leads, inversion of U wave suggests anteroseptal ischaemia or right ventricular hypertrophy or strain.

(7) U wave alterations may occur after exercise, digitalis or quindine.

Exercise Test

A case of ischaemic heart disease or angina pectoris may show a normal cardiogram at rest By putting the patient under a standardized stress by exercise (such as Master's two-step or double two step test), it is possible to evoke evidence of ischaemic heart disease. The patient is made to go up and down two 9 inch high steps within 1½ minutes over a certain number of times (trips) (depending on the age, sex and weight). The electrocardiogram is recorded immediately after, 2 minutes later and 6 minutes later. Further 10 minutes interval cardiograms may be taken if the cardiogram remains abnormal. In the "double test", the trips are doubled over a period of 3 minutes. The electrocardiogram must be compared with the "rest" cardiogram.

Criteria for a positive test

(i) ST segment depression or elevation of 1 mm. or more in I, of 1.5 mm, or more in II and III. of 2 mm. or more in chest leads. An all-round depression is a positive test, downward sloping of the ST segment is a "possible positive", while depression of J alone is not positive. The duration of the ST interval (and QT) is

also important according to Master. (2) An appearance of negative or diphasic T waves in I, II or left ventricular leads. (3) Bundle branch block. (4) Bigeminy (ventricular extrasystoles). (5) Inverted U waves. The most important and commonest of the findings is the ST change.

Electrolyte or metabolic derangments of the cardiogram.

These may occur in cardiac, renal or metabolic diseases.

Hyperkalemia or hyperpotassemia (normal serum K – 3.5 to 5.5 mEq/L). The serum K is 5.5 to 7.8 mEq in hyperkalemia. The main electrocardiographic abnormalities are:

(1) Tall symmetrically peaked or "tented" T waves (slightly similar to those of posterior wall myocardial damage) (2) Later, ST depressions. (3) Prolonged P–R interval. (4) P waves broad and flattened (followed dy atrial arrest or fibrillation). (5) Widening of the QRS. (6) Absence of U waves. (7) S waves wide and deep with R small.

Causes of hyperkalemia are uraemia, Addison's disease, shock, dehydration, excessive potassium by mouth, acute nephritis, leukaemic crisis and after heavy exercise.

Hypokalemia (hypopotassemia)

1. Flattening and later inversion of the T waves.

2. Prominent U waves (blending with T waves).

3. P prominent and peaked.

4. Later, A-V conduction disturbances.

5. Ectopic rhythms.

Causes—Diuretic therapy, corticosteriod therapy in excess, diabetic acidosis and coma, starvation, chronic diarrhoea and vomiting, meningitis, encephalitis and familial periodic paralysis.

Hypercalcemia A high serum calcium produces (1) shortening of the QT interval. (2) shortening of the ST segment, and (3) ectopic rhythms. The causes include hyperparathyroidism, large doses of vitamin D, renal damage, excess of calcium and alkali, and bone diseases.

Hypocalcemia—(1) Prolonged QT interval, (2) Prolonged ST segment, (3) T waves normal or narrowed. The causes are sprue, hypoparathyroidism, alkalosis and servere diarrhoea.

The Hyperventilation Syndrome. Also present in anxiety neurosis, the main changes are flattening or inversion of the T waves and depression of the ST segments in some or all of the leads. The cause is not clear. It may be due to alkalosis, sympatheticotonia or lowering of the Diaphragm with a change of heart position and prolongation of P-R in some leads. To distinguish organic heart disease changes from those of hyperventilation, ergotamine tartrate corrects the abnormalities due to hyperventilation.

Effect of Food. Occasionally, electrocardiograms taken half to one and half hours after a meal may show low or inverted T waves.

Influence of Age

Juvenile type of electrocardiogram. During the first year of life, the P-R interval is about 0.12 second (upto 0.14), the S1

and Q3 are prominent, the amplitude of R is about 2/3rd amplitude of S in lead I and the Q amplitude in lead III may be about two-thirds the amplitude of the R wave. R is highest in lead II. The T waves in standard leads are usually positive (but T3 may be diphasic or negative.) In the chest leads, R < S in V1 but from V2 (where R = S) to V 6, the R and S are about equally prominent, until V5 or V6 where R > S T is markedly negative in V1 to V2 or V1 to V3 and at times upto V4. The S1 Q3 type of picture with negative T waves in V1 to V3 (juvenile pattern)—due to rotation of the heart, may persist upto adult life—causing confusion. T wave negativity in V2 (and V3) may be persistent, but with changes from time to time.

Senile electrocadiogram. The amplitude of waves may become higher than normal in individuals over 70 years without any pathology, while T wave abnormalities are observed in 25 per cent of cases due partly to age and partly to vascular degeneration.

Physiological Variations of the Normal Electrocardiogram by Respiratory, Postural and Autonomic Influences.

(1) Respiratory Effects on Normal Electrocardiogram. These are due to alterations in the level of the diaphragm through respiratory movements. They are more marked in Lead III in cases which show a postural left axis deviation.

During deep inspiration (IIIR) a positive Q wave may disappear completely even if large, a negative P wave may become positive or a markedly negative T wave may become diphasic or even positive. Notches when present change in degree or position with respiration.

During inspiration, depression of the heart with rotation on the longitudal axis may even affect the V leads. In V2, a pronounced change of form may be noted in the R/S relationship, with changes in the notches of QRS and sizes of the T waves. The R tends to become smaller in inspiration and the T wave higher or lower. A negative T wave may, however, become positive over V2.

(2) Postural Effects. The electrocardiogram is a good index of the "constitutional type" of the individual in relation to size, shape and weight. An asthenic or thin man has a "vertical" type of heart while a broad-chested or pyknic individual possesses horizontal type of heart with left axis deviation. The relationship of the cardiogram to the habitus is not strictly constant, being dependant also on the degree of rotation of the heart on its long axis.

Striking changes in the electrodardiogram may be obvious at times on changing the position of the body from the left to the right side (or vice versa) during recumbency or on adopting the sitting up or standing posture, the maximal changes being observed at times in lead III (and at times in lead I, especially in juvenile subjects). On standing (or even sitting up), the T wave may show flattening or even inversion rarely, maximally in leads III and II and in left vetricular leads with depressions of ST segments – the electrocardiogram becoming "abnormal" on rare occasions. Transient orthostatic or "rising" reactions.

(3) Autonomic influences. (i) Increased sympathetic tone increases the rate of

the heart, the size of the P waves (especially in leads II and III), shortens the P-R interval and the QRS duration with relative prolongation of the Q-T duration. The majority of observers have reported an increase in the size of the T (this is controversial).

(ii) Increased vagal tone (vagotonia or vagal overaction) causes sinus bradycardia, low P waves (especially in leads II and III), a prolongation of the P-R interval, slight flattening of the T waves with ST elevations and at times mild increase in sinoauricular or auriculoventricular conduction time.

(iii) With autonomic imbalance or instablity, considerable diurnal variations in the amplitude of T waves are observed, particularly in lead II, being tallest in the evening and shortest in the forenoon.

(4) The Electrocardiogram of "training" or "atheletic" heart or habitas. A vagotonic influence is observed, with a sinus bradycardia (even 40 to 50 p. m.) prolonged P-R interval (even over 0.2 second) prolonged QRS at times, an A-V dissociation.

Abnormal Electrocardiogram due to Artifacts or Technical Faults.

Deformation of tracings may be due to: (1) A muscular or Parkinsonian tremor (small or medium sized peaks of irregular frequency and amplitude). (2) An alternating current from the electric light mains or a short-wave apparatus. (3) Sudden large changes of level due to bodily movements. (4) A poor contact of the electrodes with small changes of level. (5) Respiratory (wave-like) changes of level through a bad application of the electrodes. (6) Overshoot waves or displacement of the ST-T segments in leads V1 to V4 due to a bad application of the chest elecrode. (7) Circumscribed tremors or oscillations due to vibrations on the electrocardiographic machine. (8) Amplifier trouble or "damping" effect. (9) Wrongly connected limb leads (e. g. right arm lead to left arm and vice versa, giving a mirror image in lead I and reversal of tracings of II and III as in cases of dextrocardia or situs inversus.