ECG

Patrick Davey

Abstract
This contribution details the features that should be looked at on an ECG to determine whether or not it is normal. It describes the abnormalities found in certain conditions including atrial enlargement, ventricular hypertrophy, acute chest pain, ST elevation myocardial infarction, and syncope with many illustrations.

Keywords atrial enlargement; Brugada syndrome; cardiovascular disorders; diagnosis; ECG; ventricular hypertrophy; ST elevation myocardial infarction; syncope

What's new?

- Renewed importance of the ECG in determining the management of acute coronary syndromes
- Discovery of new pro-arrhythmic genetic conditions, e.g. Brugada syndrome
- New ambulatory and implantable ECG monitoring devices

The normal ECG

It is important to confidently determine whether an ECG is normal, according to the following parameters (Figure 1).

P wave is 110 ms or less and 0.25 mV or less; the first two-thirds are from right atrial depolarization and the last two-thirds from left atrial depolarization. In health, the P wave is upright in lead II and biphasic in lead V1; the first, positive deflection is larger than the second, negative one.

PR interval is 140–210 ms. A short PR interval may indicate an accessory pathway, which allows early ventricular depolarization (pre-excitation). A long PR interval reflects slow conduction through the AV node and bundle of His, and may indicate disease of the conducting tissue predisposing to bradyarrhythmia through high-grade AV block.

QRS axis is normal at $-30°$ to $+90°$ (Figure 2). The QRS complexes in leads I and II are 'positive' (R wave > S wave); the QRS in lead III is 'negative' (S wave > R wave). Axis deviation implies either ventricular hypertrophy or disease of one of the left bundle hemifasicles (left axis deviation indicates left anterior hemifasicular block, right axis deviation indicates posterior hemifasicular block).

QRS height, when too great, reflects thin stature or ventricular hypertrophy; too little reflects obesity, hypothyroidism, pericardial effusion, or diffuse loss of myocytes as a result of ischaemic heart disease or cardiomyopathy.

QRS duration is 120 ms or less. A broad QRS usually reflects disease of the right, the left or both bundle branches.

R wave and S wave

The R wave exhibits normal progression in the chest leads. Its height initially increases from V1 to about V5, then declines. The S wave increases (becomes deeper) from V1 to V2 (sometimes V3), and then declines. The 'transition point' (where the R wave equals the S wave) is usually V3, and reflects the position of the interventricular septum. The transition point can be moved towards V2 in health (clockwise rotation) or towards V4 in obesity (anticlockwise rotation). 'Poor R wave progression' (little/no increase in R wave height until lead V4) may indicate an old anterior wall myocardial infarction.

No pathological Q waves

Small Q waves (two small squares or less) are seen in lead III (varying with respiration) and laterally in the chest leads, reflecting left-to-right septal depolarization. A large Q wave is seen in lead aVR (as aVR examines the endocardial surface of the heart, it registers the endocardial to epicardial spread of depolarization as a Q wave).

ST segment is isoelectric. 'Physiological' ST elevation is usually restricted to leads V1–V3/4.

T wave polarity reflects whether the R or S wave is dominant; if R is greater than S, the T wave is upright, and vice versa. Thus, the T wave is normally inverted in leads III, aVF, aVR and V1, and upright elsewhere.

QT interval is normal after correction for heart rate. Fridericia's correction ($QTc = 3\sqrt{(QT/RR\ interval)}$) is the 'best' heart rate correction formula. (All such formulae are scientifically unsound, because they fail to allow for individual QT–heart rate variation with day/night, health/disease and many drugs.)

Right and left atrial enlargement

The ECG is unreliable in the diagnosis of atrial enlargement. The classic signs are as follows (Figure 3).

In right atrial enlargement, the P wave height in lead II increases to two and a half small squares or more.

In left atrial enlargement, the P wave is dominated by the late, long left atrial depolarization, leading to a late second peak in lead II, and a late negative deflection in lead V1 greater than the early positive deflection. V1 is more sensitive to left atrial enlargement than lead II.

Right and left ventricular hypertrophy

Right ventricular hypertrophy (RVH)

The ECG is poor at diagnosing RVH/pulmonary hypertension (Figure 4).

Patrick Davey *DM FRCP is a Cardiologist at Northampton General Hospital, Northampton, UK and the John Radcliffe Hospital, Oxford, UK. His interests include heart failure, interventional and nuclear cardiology. He is the editor of 'Medicine at a glance' and the author of 'The ECG in clinical decision making' and 'ECG at a glance'. Competing interests: none declared.*

A normal ECG

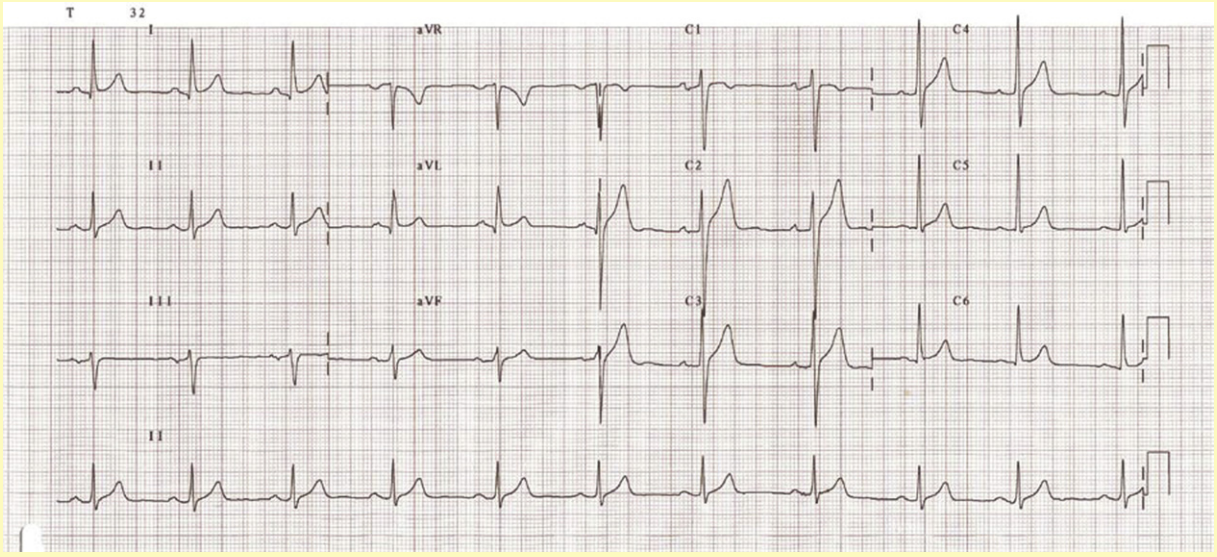


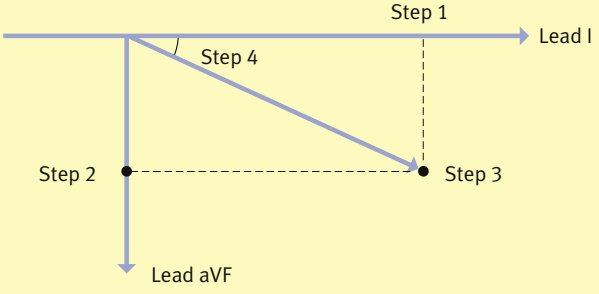
Figure 1

- The earliest sign is a rightwards shift in the QRS axis, followed by an increase in the size of the V1 R wave.
- Subsequently, the V1 R wave becomes larger than the S wave (i.e. dominant) (Table 1).
- The T waves in leads V1–V3 invert ('RV strain').
- Finally, right bundle branch block may occur, particularly in acute right heart strain associated with pulmonary embolism.

Left ventricular hypertrophy (LVH)

- Initially, the R wave height increases in the left-sided standard and chest leads (I, II, aVL, and V4–V6), often with commensurate deepening of the S wave in leads V2 and 3.
- The small 'septal' Q waves in lead V5/6 increase in size with increasing hypertrophy.
- As LVH increases, the QRS axis shifts to the left, and the lateral-lead T waves flatten then invert ('strain', associated with a worse prognosis in hypertension).

Determining the QRS axis

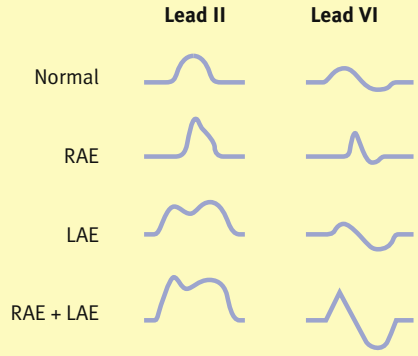


To determine the overall QRS vector, determine whether the R or S wave is the larger in leads I and aVF, then obtain the overall vector (step 3) that can be measured (step 4) to give the QRS axis. Other leads can be used if these complexes are too small – it is best to choose leads at right angles to each other (e.g. lead aVL and lead II).

Source: Davey P. The ECG in clinical decision making. London: RSM Press, 2004.

Figure 2

P wave changes in atrial enlargement



In right atrial enlargement, the P wave is dominated by the wave of depolarization passing through the enlarged right atrium; this passes towards both lead I and V1 and, as normal right atrial depolarization occupies the first two-thirds of the P wave, changes in RAE are predominantly in the first two-thirds – three-quarters of the P wave. In left atrial enlargement, the P wave is dominated by the wave of depolarization passing through the enlarged left atrium; this is towards lead I but away from lead V1. As the normal left atrium depolarization occupies the latter two-thirds of the P wave, abnormalities are found in this phase of the P wave. (RAE = right atrial enlargement, LAE = left atrial enlargement). (Source: Marriot's practical electrocardiography, 10th ed. Philadelphia: Lippincott Williams and Wilkins, 2001.)

Figure 3