

## Editorial

# The ECG: What is 'normal'?

Many errors of clinical interpretation have arisen (and no doubt may continue to arise) from a failure to recognise the variability that is characteristic of man in all his measurable features. Such variability between one person and another is clearly a necessary and distinctive attribute of life. How then in this situation can we define a "normal" value? On what point of the scale should we begin to regard them as abnormal, i.e. pathological?

Sir Austin Bradford Hill, 1977<sup>1</sup>

The above quote identifies a problem common to medicine and life in general. 'Normality' is partly a statistical concept, but also has philosophical and moral connotations. The ECG occupies a special place in cardiac assessment. A 'normal' ECG is very reassuring (often falsely), whereas an abnormality may engender considerable anxiety both for the doctor and the patient. This is particularly so when a well person has an ECG as part of a 'check-up'.

The human electrocardiogram was first recorded by Augustus Waller in 1887,<sup>2</sup> but the ECG as we know it was revealed to the world by William Einthoven in 1901–1904.<sup>3</sup> While recording methods differ, tracings recorded by him are recognisable and analysable today. What has changed over the century since then has been the huge increase in the usefulness of ECG recording, as a result of continuous refinement. ECG analysis is continually being informed by accumulated data of clinical syndromes, pathology, biochemical and genetic information. For example, a new clinical/ECG syndrome has been recognised as recently as 1992 (Brugada syndrome<sup>4</sup>) in which young survivors of ventricular fibrillation cardiac arrest manifest characteristic ST segment and QRS changes in V1 and V2. This syndrome has now been pinned down to a single gene coding for part of the myocardial sodium channel.

## ECG analysis

The ECG can be analysed in terms of measurement and patterns. Normal values for measurements of conduction, QRS voltage and repolarisation are well established. Normal patterns for P waves, QRS complexes, ST segment and T waves are well-characterised. Deviations from these norms have varying degrees of sensitivity and specificity for different cardiac pathologies. Difficulties may arise with deviations from normal values or patterns in people with no other evidence of cardiac abnormality.

## Why do we do ECGs?

The ECG is a widely available, cheap, non-invasive and highly useful investigation, almost without parallel in modern medicine. It is frequently performed for a variety of indications:

- to confirm suspected pathologies, e.g. acute myocardial infarction
- to decide on the mode of treatment, e.g. ST elevation versus ST depression in acute coronary syndromes
- to assess the rhythm
- to predict future morbidity and mortality
  - clinically
  - for insurance
- as part of a check-up to reassure the patient and ourselves of 'normality'.

The interpretation of ECG patterns and measurements may differ, depending on the context and the reason the ECG was performed. If an abnormality is detected in someone with no symptoms or signs of cardiovascular disease, what does it mean? Wrong interpretation may have serious consequences, from missing serious and treatable pathology, to engendering a cardiac neurosis in a healthy individual.

**TABLE I. FACTORS INFLUENCING THE NORMAL ECG**

**Intrinsic cardiac factors**

- electrical axis
- individual variation in conduction
- heart position
  - vertical
  - horizontal
  - rotation
- heart size

**Extrinsic factors**

- age
- gender
- body size
- body habitus
- chest wall thickness
- posture
- the lungs
  - hyperinflation
  - mediastinal shift
- autonomic
  - vagal/sympathetic balance
- electrolytes
  - potassium
  - pH
  - calcium
- drugs
  - digoxin
  - beta-blockers
  - QT prolonging agents

**Technical/recording**

- electrode position
- recorder quality
- artefacts
  - electrode contact
  - movement
  - muscle noise, tremor (e.g. Parkinson's)
  - AC interference
  - digitising artefacts e.g. pacemaker spikes
- labelling

**TABLE II. ECG ABNORMALITIES IN ATHLETES**

**Rhythm**

- sinus bradycardia
- sinus arrest with junctional escape
- sinus arrhythmia
- wandering atrial pacemaker
- first-degree AV block
- Wenckebach second-degree AV block

**QRS complex**

- increased left ventricular voltage
- increased right ventricular voltage
- rightward QRS axis
- incomplete right bundle branch block

**Repolarisation changes**

- ST segment elevation
- tall peaked T-waves
- biphasic T-waves
- T-wave inversion

cardiomyopathy, bundle branch block, or a prolonged QT interval all have prognostic implications, even in the absence of symptoms.

Elite athletes pose special problems. The incidence of ECG abnormalities is high<sup>5,6,7</sup> and may suggest pathologies known to be associated with an increased risk of sudden death, e.g. hypertrophic cardiomyopathy (HCM) or arrhythmogenic right ventricular cardiomyopathy (ARVC). Any practitioner confronted with such a problem needs to be aware of the range of patterns and changes in the ECG known to be associated with the athlete's heart. (See article by Maznev *et al.* on page 12 of this issue.)

Abnormalities of both measurements and patterns are common. The main causes of the ECG changes are the high vagal tone associated with a high level of physical fitness, and increases in left ventricular volume and wall thickness, particularly in endurance athletes. Table II lists some of the abnormalities that have been described. Is it correct to call these changes 'abnormal'? In the sense that they are outside of the range of statistical normality, the answer is yes. One can hardly consider an elite Olympic athlete 'normal'. He or she is at the extreme of the distribution curve of ability and physical capacity. 'Supernormal' might be an appropriate description. In the sense of pathology, the answer is no. Very few individuals with significant cardiac pathology attain the heights of physical prowess required for competitive athletics of the kind associated commonly with ECG changes. An athlete's 'abnormal' ECG may be considered 'normal' in so far as it does not predict present or future cardiac disease or death. Studies have shown that the abnormalities tend to disappear with prolonged cessation of training, confirming their functional nature.

Despite the above, it is important to be aware that the occasional athlete will have a potentially lethal cardiac abnormality, manifesting initially only as an abnormal ECG. Examples include HCM (said to be the leading cause of sudden death in young sportspeople in the USA<sup>8</sup>) and ARVC, which in Italy accounts for the highest number of sudden deaths during exercise.<sup>9</sup> The ECG changes in both of these conditions may be similar to those in normal highly trained

## Normal variation

The above problem is compounded if the practitioner is ignorant of the huge variation in the normal ECG. Table I lists some of the many factors which influence the electrical activity of the heart as recorded from the body surface by conventional ECG recording.

## Assessment of an abnormal ECG in an otherwise normal person

A number of abnormalities may be found in otherwise normal people, e.g. when an ECG is done for insurance purposes. For example, a short PR interval and wide QRS due to a delta wave (Wolff-Parkinson-White pattern) indicate the presence of an accessory AV connection, even in the absence of a history of tachycardias. While considered benign from an insurance point of view, its significance to the individual depends on the functional properties of the accessory pathway and the person's circumstances. Pathological Q waves of silent myocardial infarction, patterns of left ventricular hypertrophy with repolarisation changes due to hypertrophic

people. However, a combination of markedly increased left ventricular voltage and repolarisation changes should prompt careful echocardiographic examination for HCM as well as asking about a family history of sudden death. The echocardiographic differentiation of HCM from physiologic hypertrophy may not be easy.<sup>10</sup> A combination of T-wave inversion in the right chest leads and ventricular arrhythmias should raise the suspicion of ARVC and prompt further investigation.

Conduction abnormalities, including Wenckebach second-degree AV block and, rarely, transient complete heart block, occur much more frequently in highly trained individuals than in the general population. High vagal tone is thought to be responsible. The block will therefore only be evident at rest, particularly while asleep, and will be associated with a slow sinus rate and sinus arrhythmia. In some individuals, however, AV nodal conduction remains abnormal after total autonomic blockade with atropine and propranolol. It may be that the increased vagal tone engendered by the intense training may have unmasked the abnormality. If the athlete can be persuaded to stop training (for up to several months), the ECG changes related to training and high vagal tone will return to normal. The same is true of the structural changes in left and right ventricular dimensions.

The abnormal patterns of repolarisation, including ST-segment elevation and T-wave inversion in the chest leads are not confined to athletes. They may occur in otherwise normal young people, particularly black Africans and African-Americans.<sup>11</sup> Several patterns were described by Grusin in 1954 in black patients without heart disease at Baragwanath Hospital.<sup>12</sup>

## Conclusions

The range of normality of the ECG is very wide. The patterns considered to be abnormal occasionally occur in individuals without other evidence of heart disease, particularly in elite athletes and young black men. Awareness of the pos-

sibility will help to prevent undue anxiety and over-investigation. Nevertheless, the ECG is occasionally the only clue to a serious condition in an individual without symptoms.

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