Electrocardiogram and arrhythmias

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Abstract

Introduced by Einthoven, electrocardiography remains the most common diagnostic procedure readily available to the physician in primary and secondary care. It is a graphical display of the electrical potential difference as it spreads through the heart and is recorded at the body surface. The electrocardiogram (ECG) is an indispensable tool to screen and monitor cardiac patients. Exercise ECG is used to diagnose coronary artery disease and ambulatory ECG to assess arrhythmias.

Keywords Atrial fibrillation; atrial flutter; atrioventricular block; bradycardia; ECG waves and their variations; electrical impulse of the heart; tachycardia; ventricular tachycardia

Royal College of Anaesthetists CPD matrix: 1A01

Learning objectives

After reading this article, you should be able to:

- recognize two common variations of each of the normal P, QRS and T waves
- distinguish the three atrioventricular blocks
- describe three common tachyarrhythmias.

ECG lead system

Heart muscle generates an electrical current that can be recorded from the surface of the body by the electrocardiogram (ECG). The 12 conventional ECG leads record the difference in potential between the electrodes on the body surface.

ECG leads are divided into two groups: six limb leads and six chest leads. Of the six limb leads, three are bipolar leads and have been in use for more than a century (lead I, lead II and lead III). These leads measure the difference in potential between electrodes at two extremities: lead I, left arm—right arm; lead II, left leg—right arm; and lead III, left leg—left arm.

All leads introduced later are unipolar leads and are termed 'V' leads. These measure the voltage (V) at one locus relative to a common central terminal (indifferent electrode) that has approximately zero potential. These comprise the three limb leads aVR, aVL and aVF, where aVR is the right arm, aVL is the left arm and aVF is the left leg (foot). These three limb leads detect only a small deflection of current, which is augmented 50% by the machine. This augmentation of potential is designated by the prefix 'a'. There are six unipolar chest (precordial) leads that are designated V1—V6; these are placed directly over the chest wall. Each chest lead records the impulse immediately beneath the electrode because the heart is close to the chest wall. (For detailed discussion about lead position and cardiac depolarization please refer to *Anaesthesia and Intensive Care Medicine* 2006; **7**: 264—6.)

ECG waves

P wave

P waves result from atrial activation by the sinoatrial (SA) node. Because the SA node is situated in the right atrium, right atrial activation begins first and is reflected by the proximal (ascending) limb of the P wave. Left atrial activation begins 0.03 seconds later and is represented by the descending limb of the P wave. Because of the orientation of the leads, this is best seen in standard lead II and lead V1. The P wave in these leads is usually positive, pyramidal and with a slightly rounded apex. It is normally inverted in aVR, and upright in aVF plus the left chest leads (V4—6). Amplitude does not usually exceed 2—3 mm in any lead.

Variations of the P wave

Inversion: in leads where it is normally upright (or upright in aVR), an inverted P wave would indicate that the impulse is travelling in an unusual path (e.g. atrial ectopic, atrioventricular (AV) junctional rhythm).

Increased amplitude: this is due to right atrial hypertrophy and is seen in cor pulmonale and congenital heart disease.

Biphasic (descending limb more negative than the ascending limb): P waves in leads III and V1 are a sign of left atrial enlargement.

P mitrale: notched P waves (distance between two peaks more than 0.04 seconds) owing to left atrial involvement in mitral disease. It is usually notched and taller in lead I than in lead III.

QRS complex

The QRS complex reflects rapid ventricular depolarization. An initial downward deflection is termed the Q wave and ensuing deflections are labelled in alphabetical order. The first positive deflection is designated R, whereas S is the first negative deflection that follows the R wave. This represents the terminal part of the ventricular activation.

The complex ventricular depolarization can be divided into two sequential phases. The first phase is the activation of the ventricular septum from left to right. The second phase is the simultaneous activation of the right and left ventricles, usually dominated by the bulky left ventricle.

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In the chest leads, as a consequence of this normal depolarization process, the right-oriented leads (V1 and V2) show a small upward deflection (septal R wave), followed by a deep S wave. The same sequence in the left-sided chest leads (V6, aVL, lead I) causes a small downward deflection (physiological Q wave) followed by a tall R wave. In the intermediate chest leads, there is a relative increase in the R wave and a reduction in the S wave amplitude (normal R wave progression) from left to right. R and S waves are approximately equal in the mid-chest leads (V3 and V4); this is called the transition zone.

The QRS pattern in the limb leads varies and depends on the mean QRS axis. Lead aVR, which records from the right shoulder, effectively 'looks' from the cavity of the heart with all the vectors directed away and thus has all negative deflections. The normal duration of the QRS complex is 0.05–0.10 seconds.

Variations of the QRS complex

Prolonged QRS duration: a duration of 0.12 seconds or more signifies conduction delay, such as in bundle branch block, but can also be present with pre-excitation of the ventricles via an accessory pathway, such as in Wolff–Parkinson–White syndrome.

Right bundle branch block (RBBB): this occurs more commonly than left bundle branch block, especially in people with structurally normal hearts. RBBB can also occur in acquired (valvular, ischaemic) and congenital heart disease, especially atrial septal defects.

Left bundle branch block (LBBB): this is usually due to ischaemic heart disease, hypertension, severe aortic stenosis and cardiomyopathy. Bundle branch blocks (QRS duration of 0.12 seconds or more) can be chronic or intermittent and they can be rate related as well. Therefore, some patients with supraventricular tachycardia (SVT) have broad complexes (aberrant conduction).

T wave

T wave: this is a marker of the ventricular recovery period (repolarization) and is normally inscribed in the same direction as the QRS complex. It is, therefore, normally upright in leads I and II and in the left-sided chest leads and is inverted in the aVR lead. It is variable in all other leads.

Variations of the T wave

Tall positive T waves can be a normal variant, but are also seen in hyperkalaemia, hyperacute myocardial ischaemia, cerebrovascular injury and left ventricular volume overload.

T wave inversion can be seen with cardiomyopathy, ischaemia, ventricular hypertrophy, myocarditis and intracranial bleeds.

U wave

U waves usually follow the T wave as a small rounded deflection ($\leq$ 1 mm). An abnormal increase in the amplitude is seen with hypokalaemia, and with drugs such as quinidine and procainamide. This could be a sign of increased susceptibility to torsades de pointes.

Arrhythmias

It is beyond the scope of this article to discuss arrhythmias in detail, but a brief overview is provided. The heart normally beats approximately 70 times per minute at rest. A rate less than 60 beats per minute is termed bradycardia, whereas the term tachycardia is reserved for rates in excess of 100 beats per minute.

Bradycardia

This is normally present during sleep and in fit athletes (athletes' heart syndrome). Pressure receptors (baroceptors) in the aorta and carotid arteries respond to arterial pressure, altering vagal tone through acetylcholine release. In carotid sinus syndrome, there is increased sensitivity of the baroceptors located in the carotid sinus region of the carotid artery. Therefore, pressure on the neck can result in extreme bradycardia, dizziness and syncope. Sometimes, it can trigger asystole for up to 10 seconds. Heart rate also varies with the phase of respiration; this is called 'sinus arrhythmia' (For further description, please refer to *Anaesthesia and Intensive Care Medicine* 2006; **7**: 264–6.)

Atrioventricular (AV) block

First-degree block: this is characterized by a prolonged PR interval ($>$ 0.20 seconds), and is due to constant delay rather than a block in conduction of the impulse from the atrium to the ventricle. First-degree block is often present in athletes, but can be associated with ischaemic heart disease, acute rheumatic carditis and drugs such as digitalis and β-blockers. It causes no symptoms and requires no treatment other than observation.

Second-degree block: type 1 second-degree AV block (Wenckebach, Mobitz type I); there is progressive prolongation of the PR interval, before a QRS complex fails to appear after a P wave. The block is usually in the AV node and the QRS is of normal duration. Causes are inferior wall myocardial infarction, drug intoxication with beta blockers, digoxin and calcium channel blockers. Type 1 block can be present in normal individuals with increased vagal tone, generally at night. If the ventricular rate is adequate and the patient is asymptomatic, observation is sufficient.

Type 2 second-degree AV block (Mobitz type II) (Figure 1 shows the 2:1 AV block): the PR interval remains constant before a sudden and unexpected failure of the P wave to conduct. It is usually due to disease of the His–Purkinje system and is often associated with an abnormal QRS wave. When two or more successive P waves are blocked, this is called high-grade AV block. Type 2 block can occur in the setting of anteroseptal myocardial infarction or sclerodegenerative disease of the fibrous skeleton of the heart. This is very likely to progress to symptomatic third-degree block or ventricular standstill, so permanent pacing is indicated. Physiological second-degree block is seen with fast supraventricular rhythms such as atrial tachycardia and atrial flutter.

Third-degree block (complete heart block): this is characterized by complete cessation of the electrical impulses from the atrium to the ventricle. P waves are dissociated from the ventricular complex and the two are asynchronously controlled by independent pacemakers. This is the most advanced form of AV block. It is mostly due to chronic degenerative changes in the bundle branches due to Lev's and Lenegre's disease. It can also occur with cardiomyopathy and inferior myocardial infarction. Complete heart block can be congenital owing to maternal

