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ECG – A Technologists Guide to Interpretation

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ABSTRACT

Cardiac stress testing, gated cardiac imaging and monitoring critical patients expose the nuclear medicine technologist to the electrocardiogram (ECG). Basic ECG interpretation skills are essential for the nuclear medicine technologist to enhance patient care and to recognise key arrhythmias. This article provides an insight into the anatomy of an ECG trace, basic ECG interpretation and a case example typical in the nuclear medicine environment.

Introduction

Historically in nuclear medicine, the goal of monitoring the ECG by technical staff was to either detect changes in heart rate and rhythm, or to employ it as a physiological trigger for imaging (generate a prominent R wave). Changes in heart rate and rhythm are evaluated on the horizontal axis of the ECG and, like the R trigger for imaging, generally required only a single lead (*1*). Evaluation of changes in amplitude on the vertical axis and, indeed, interpretation of a 12-lead ECG was not a priority. Nonetheless, the emergence of more widespread use of ECG monitoring associated with cardiac imaging, cardiac monitoring in critical care inpatients for imaging, and the greater emphasis on advanced practice and the extended role of the nuclear medicine technologist have driven an increased demand for more detailed understanding of ECG interpretation. This article provides foundation understanding in the science of ECG and interpretation basics.

The Heart

The heart is a critical organ that is essentially a pump driven by electric impulses that are generated and conducted by an internal conduction system. The cardiac cycle begins with the contraction of both atria, which actively fills the main pumping chamber known as the ventricles (*2*,*3*). Once ventricular filling is completed, the ventricles contract and drive the blood into the arterial system. The ventricles then relax to their baseline state, allowing the passive filling of the chambers (*2*,*3*). This is followed by the next cardiac cycle.

Internally, the cardiac myocyte has a negative charge which causes a voltage difference across the cell membrane (transmembrane potential) (*2-4*). Since the cardiac myocyte is excitable, it can be stimulated to allow ions to move through open channels in the cell membrane (*2-4*). The resultant cardiac action potential has three phases; depolarisation, repolarisation and a resting phase (*2-4*). Depolarisation and repolarisation are electrical activities that cause muscle activity with depolarisation generates an action potential and myocardial contraction via an electrical impulse while repolarisation returns to a resting state corresponding to myocardial relaxation. During depolarisation, sodium channels open allowing the positively charged sodium ions to enter the cell (*2-4*). The cardiac membrane potential returns to normal during repolarisation via interactions involving sodium, potassium, and calcium making contraction not possible (*2-4*). The resting phase is that period where there is no net movement of ions across the myocyte cell membrane (*2-*

4). The duration of the action potential of the myocyte is longer than nerve or skeletal muscle due to slow calcium channels and this ensures a regular cardiac rhythm (*4*).

The conduction system generates electric impulse as the cells spontaneously depolarise due to their specific membrane and ion channel properties (*2,3*). Contraction of the myocardium is activated by these impulses. The myocardium relaxes as the cells repolarises in preparation for the next impulse.

The conduction system begins with the sinoatrial (SA) node at the top of the right atrium which acts as a "pulse generator" because of the relatively high rate of spontaneous depolarisation (*2,3*). The impulse spreads through the atrium and causes both atria to contract simultaneously which fills the ventricles with expelled blood. The impulse then enters the ventricles via the atrioventricular (AV) node, which serves as the protective gateway to prevent the ventricles from being stimulated by excessive impulses (*2,3*). The AV node is located inferior to the right atrium near the inter-ventricular septum and here the impulses are slowed before progressing to the ventricles (*2,3*). The impulses progress from the AV node, through the Bundle of His to the left and right bundle branches within the left and right ventricles respectively (*2,3*). The Purkinje fibres carry impulses from the bundle branches to myocardial cells to ensure contraction of both ventricles in a coordinated manner (*2,3*).

The Electrocardiogram (ECG)

The 12-lead electrocardiogram (ECG) is a quick non-invasive method to convey important information on cardiac activity by recording the variations in membrane potentials across the cardiac cycle (*2,3,5*). The standard 12-leads ECG utilises 10 electrodes, which includes 3 limb leads (I, II and III), 3 augmented limb leads (aVR, aVL and aVF) and 6 precordial leads (V1-V6). This arrangement records the cardiac activities from different directions and allows mapping of the electrical axis. The impulse that travels towards an electrode will be recorded as an upright signal in that lead while the impulse travelling away from the electrode will be recorded as an inverted signal. The standard "paper speed" is 25mm/sec, thus 1 small square equals 40 milliseconds (ms) while 1 large square (5 small squares) equals 200ms (Figure 1). The PR interval represents the period from onset of atrial depolarisation until the beginning of ventricular

depolarisation, the ST segment represents the period when the ventricles are depolarised, the T wave is the subsequent repolarisation of the ventricles, the QRD complex corresponds to ventricular depolarisation, a the R-R interval represents the cycle from ventricular contraction to the next ventricular contraction (Figure 1). A signal frequency of 1 large square is equal to a rate of 300/minute. Calibration of the ECG equipment and accurate (consistent) electrode positioning are crucial to ECG interpretation (*1*). A detailed discussion of the lead theory is beyond the scope of this article, however, deeper insight was detailed by Horacek et. Al. (*6*).

The electrical activities of the normal cardiac cycle consist of a P wave, followed by the QRS complex and then the T wave. The P wave is generated by atrial depolarization. As the muscle mass of the atrium is small, the P wave is a relatively small signal. The QRS complex represents ventricular depolarization. The QRS complex is the dominant signal of the ECG as the ventricular mass is large. The T wave is associated with the return of the ventricular activity back to its resting state, i.e. repolarization. The duration from the beginning of the P wave to the beginning of the QRS complex is known as the PR interval. The normal length ranges between 120-200ms. The normal width of the QRS complex is <100ms. The duration from the start of the QRS complex to the end of the T wave is the QT interval, which should be <440ms when corrected to the heart rate (QTc). The normal heart rhythm is the sinus rhythm (Figure 1). The normal heart rate is between 60-100 beats per minute (bpm) (Figure 2). This is characterised by the presence of a P wave before the QRS complex with the P wave being inverted in aVR and upright in aVF/aVL. Tachycardia refers to a heart rate >100bpm (Figure 3) and bradycardia refers to a heart rate <60bpm.

Other than pathology, the ECG is influenced by a number of physiological and technical factors (*1*). Consequently, there is a need for technical staff in the medical radiation sciences to have a basic understanding of both the interpretation of pathophysiological manifestations on an ECG and non-pathologic aberrations. While the ECG can vary with gender, age, ethnicity, height, weight, and other factors, ECG interpretation is an individual patient analysis rather than compared between patients (*1*). From a technical perspective, there are a number of common issues that impact on the ECG including poor skin preparation or inadequate contact with the skin can decrease cardiac signal and increase skin impedance (*1*). Noise in an ECG is associated with

aberrant signal or artefact arising from a number of factors, including without being limited to, muscle tremor, movement (including shivering), electrical interference (*1*).

Dysfunction of SA Node and AV Conduction

Dysfunction of the SA node resulting in the slowing of impulse generation or even sinus node arrest is known as sick sinus syndrome. This can be due to degeneration or damage to the SA node. In the event of SA node arrest, the generation of the cardiac impulse may be taken over by other parts of the conduction system but at a slower rate. Bradycardia-tachycardia syndrome is a variant of sick sinus syndrome in which slow arrhythmias and fast arrhythmias alternate. It is often associated with ischaemic heart disease.

Atrioventricular block (AV block) is a type of heart block in which the conduction between the atria and ventricles of the heart is impaired. First degree AV block is defined as a PR interval of >200ms with every P wave being conducted (Figure 4). Second degree AV block (Figure 5) is divided into Mobitz type I, also called Wenckeback block, and Mobitz type II block. In Mobitz type I block, the PR interval is progressively prolonged, eventually resulting in a dropped beat. In Mobitz type II block, the PR interval remains constant and a P wave suddenly fails to conduct. Third degree AV block is known as complete heart block where the P wave fails to conduct into the ventricle, thus resulting in AV dissociation (figure 6). The ventricular rhythm is usually taken over by a slower junctional rhythm. A conduction defect in the bundle branches such as right bundle branch block (RBBB) or left bundle branch block (LBBB) cause the QRS complex to widen. LBBB is important because the widened QRS is also associated with ST changes which could be mistaken for ischaemia.

Supraventricular Arrhythmia

There are 3 main types of supraventricular tachyarrhythmias:

1. Sinoatrial origin
    - Sinoatrial node re-entrant tachycardia (SANRT)
2. Atrial origin
    - Atrial tachycardia (unifocal or multifocal)
    - Atrial fibrillation (AF)

- Atrial flutter

3. Atrioventricular junctional origin
    - AV junctional re-entrant tachycardia (AVJRT)
    - Accessory AV re-entrant tachycardia (AVRT) including Wolf Parkinson White Syndrome (WPW)

AF is characterised by the chaotic electrical activity of the atrium with the loss of synchronised atrial contraction. The typical ECG shows an irregular and rapid (>100 BPM) ventricular rate with no discrete P wave (Figure 7). A ventricular rate of <100bpm during AF is considered well controlled.

Atrial flutter is caused by a large re-entrant pathway within the atrium typically at a frequency of 300/min. The ECG may show the “saw-tooth” flutter wave with the QRS conduction ratio varying from 4:1 to 2:1 (Figure 8). Atrial tachycardia is a relatively uncommon form of atrial arrhythmia. The impulse comes from within the atrium but not from the SA node. Atrial tachycardia may be associated with drug toxicity such as digoxin overdose. More rarely, atrial tachycardia can be due to a re-entrant mechanism.

Atrial ectopic beats are associated with premature atrial contraction and manifests quite differently to premature ventricular contraction. Atrial ectopic beats results in an early QRS complex and a normal T wave which obscures the P wave. Atrial ectopic beats result from a premature depolarisation that produces a normal QRS because AV node conduction is normal.

Ventricular Arrhythmias

Ventricular tachycardia (VT) is a wide complex (QRS>100ms) tachycardia (Figure 3) recognised by the wide QRS complex. There may be AV dissociation during tachycardia where the P wave bears no relation to the QRS complex. VT is usually due to re-entrant within the ventricle associated with ischaemic heart disease or some types of cardiomyopathy. There are also hereditary forms of VT with normal cardiac structures, but these are relatively uncommon. VT is a life threatening arrhythmia that leads to haemodynamic compromise and it may degenerates into

ventricular fibrillation (VF). VF refers to the chaotic ventricular electrical activities (Figure 9) leading to an acute loss of pump function and death. This is the commonest form of cardiac arrest.

Ventricular ectopic beats are associated with premature ventricular contraction (PVC). Typically the ECG has a wider QRS complex that occurs earlier than expected with a higher voltage (amplitude) and has an inverted T wave that obscures the P wave (Figure 10). The depolarisation of the ventricles occurs prematurely outside the usual conduction pathway and, consequently, the depolarisation is slower, producing the wider and unusual shaped QRS. This also results in less efficient ventricular contraction.

Junctional Re-entrant Tachycardia

Junctional tachycardia may be caused by re-entrant mechanism from a pathway close to the AV node known as AV junctional Re-entrant Tachycardia (AVJRT). Re-entrant through an additional pathway outside the AV node, known as an accessory pathway, may also be the cause of junctional tachycardia. Junctional tachycardia is typically a narrow complex tachycardia with a QRS width of <100ms. The ventricular rate during junctional re-entrant tachycardia is usually rapid at >150bpm. The P wave may not be visible or it may follow closely behind the QRS complex. Resting ECG during sinus rhythm may be completely normal but delta waves at the beginning of the QRS complex can occasionally be seen. The presence of the delta wave is indicative of an atrial to ventricular conducting accessory pathway.

A Basic Approach to ECG Interpretation

The first step in ECG interpretation is to check the heart rate for bradycardia or tachycardia. Then determine whether the rhythm is sinus by looking for the P wave. The presence of P wave does not always mean sinus rhythm. The P wave vector must be checked in leads aVR, aVL and aVF. A P wave arising from the SA node will be inverted in aVR and upright in aVL and aVF. In contrast, a P wave originating from the low atrium will be upright in aVR and inverted in aVF. If the P is upright in aVR and inverted in aVL, that indicates the P wave is travelling from left to right, which is the opposite to the normal P vector. The limb lead placement should then be checked as misplacing of the leads is the commonest cause of this pattern. Check the relationship between

the P and the QRS should the rhythm be sinus. AV block is present if the P wave is not all conducted.

If the rhythm is not sinus and the P wave is absent, check if the QRS rate is irregular, e.g. AF. Also look for the "saw-tooth" appearance of atrial flutter. Check the width of the QRS complex and look for the RBBB/LBBB morphology. In case of widened QRS complex with a normal heart rate, look for ventricular pacing spikes as ventricular pacing causes wide QRS complex with LBBB morphology (Figure 11).

When the ECG shows a non-sinus tachyarrhythmia with narrow and regular QRS complex, the main differential diagnosis is junctional re-entrant tachycardia or atrial flutter. Wide QRS complex tachycardia is much more serious as that can be VT, which is life threatening. Junctional re-entrant tachycardia with aberrant conduction leading to wide complex tachycardia may masquerade as VT. Distinguishing between VT and junctional re-entrant tachycardia with aberrant conduction requires considerable experience. If in doubt, consider the tachyarrhythmia VT until proven otherwise.

Myocardial Ischaemia

Other than cardiac arrhythmia, the ECG is a key diagnostic test for myocardial ischaemia and acute myocardial infarction. Indeed, patients undergoing cardiac imaging in nuclear medicine, often present with known or suspect ischaemia / infarction. On the ECG, myocardial ischaemia typically manifests as horizontal or down-slopping ST segment depression (Figure 12). Patients presenting for cardiac imaging generally undergo stress testing in conjunction with imaging. These ischaemic changes may be noted from baseline and the increased heart rate should also be noted as stress induced rather than tachycardia. An acute transmural myocardial infarction appears as $\geq$1mm ST elevation (Figure 13). The group of leads that show the ST elevation indicate the location of the infarct. An ECG showing ST elevation during a prolonged episode of chest pain is a key indication for urgent coronary angiography with a view to revascularisation. On the other hand, ST elevation may be seen in the relatively benign condition known as pericarditis. The peri-carditic ST elevation has a different morphology to an acute myocardial infarct and the ST changes tend to be wide spread.

Case Example

A patient presents with a history of ischemic heart disease for myocardial perfusion stress/rest SPECT evaluation. The pre-stress ECG (figure 14) provides a baseline. The stress test demonstrates up-sloping ST depression in leads associated with the infero-lateral wall (figure 15). Since the ST depression is up-sloping, it is not specifically indicative of myocardial ischaemia. Post stress, however, the ST depression becomes horizontal and as such is diagnostic of myocardial ischaemia (figure 16).

Conclusion

Monitoring an ECG is an essential part of the role of the nuclear medicine technologist. ECG monitoring is especially important in patients undergoing exercise or pharmacologic stress, and in critical care patients. While interpretation is generally beyond the scope of the nuclear medicine technologist, basic interpretive skills will allow recognition of potentially problematic or fatal arrhythmias; potentially life-saving advanced skills.

About the authors

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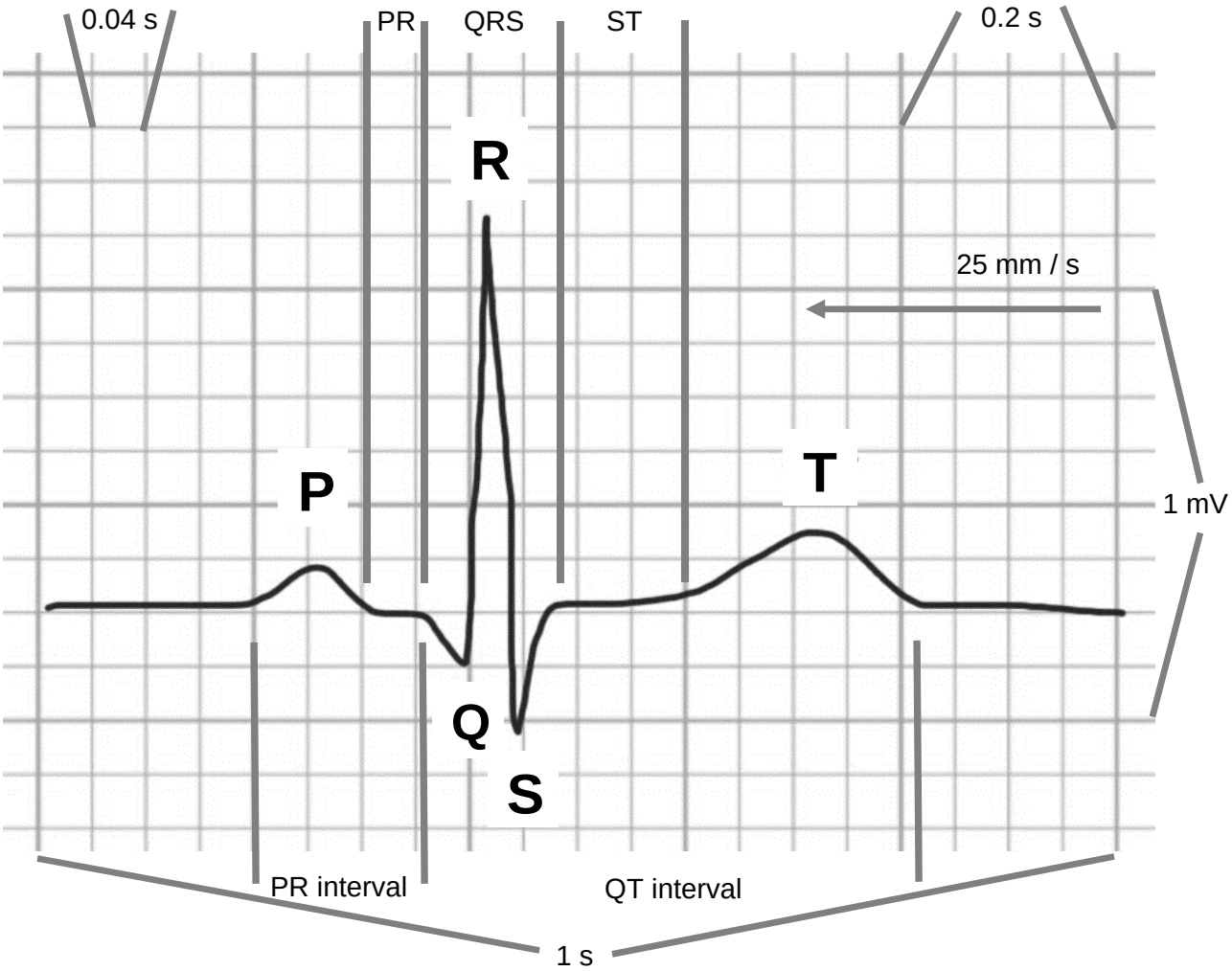

Figure 2: Normal sinus rhythm with the R-R interval approximately 1 second (60 BPM).

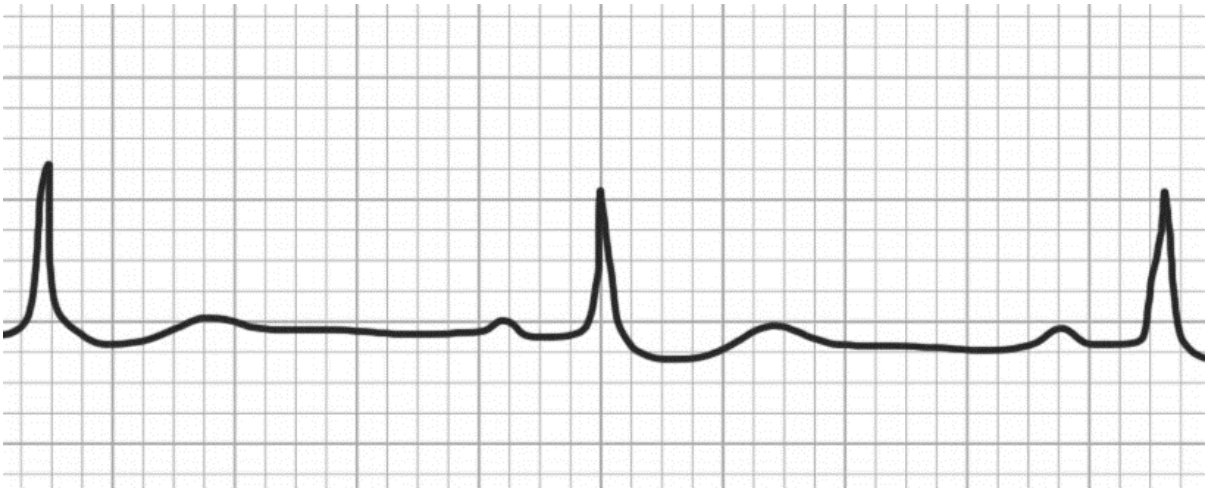

Figure 3: Sinus tachycardia with the R-R interval approximately 2 seconds (>120 BPM). Approximately 2.2 large squares indicates that the heart rate is 136 BPM (300/2.2).

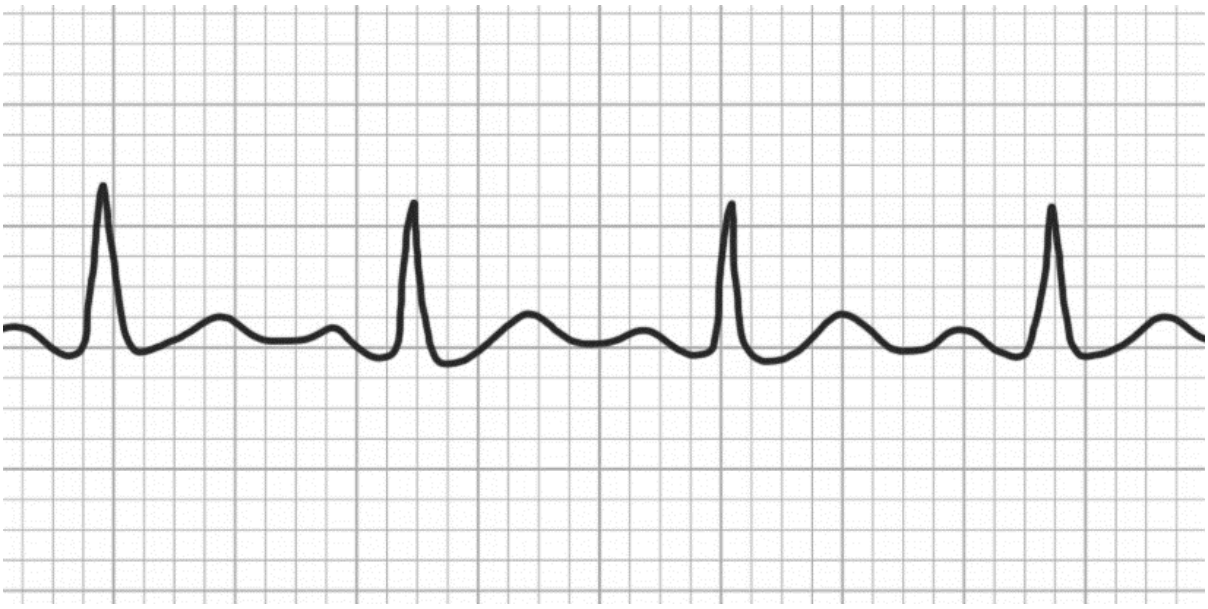

Figure 4: Rhythm strip demonstrating first degree AV block with the PR interval >200ms.

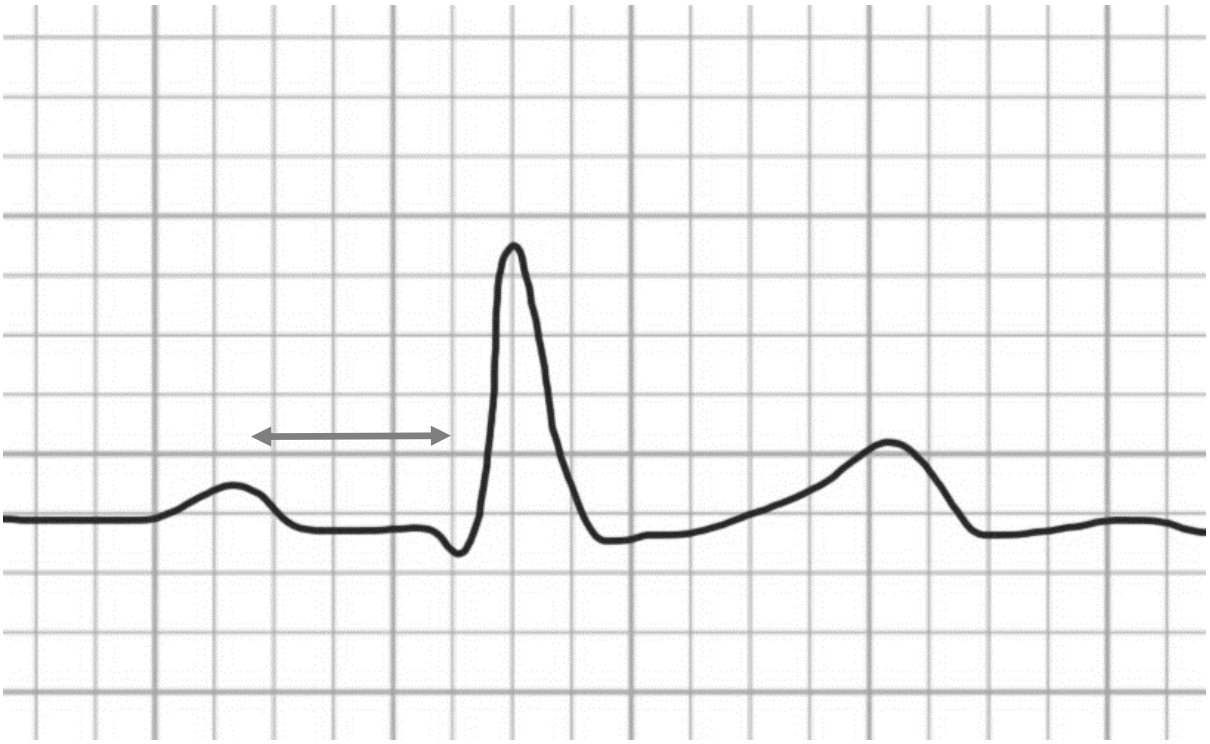

Figure 5: Rhythm strip demonstrating second degree AV block with progressively lengthening of the PR interval demonstrated here with 2 P waves per R-R interval (arrows).

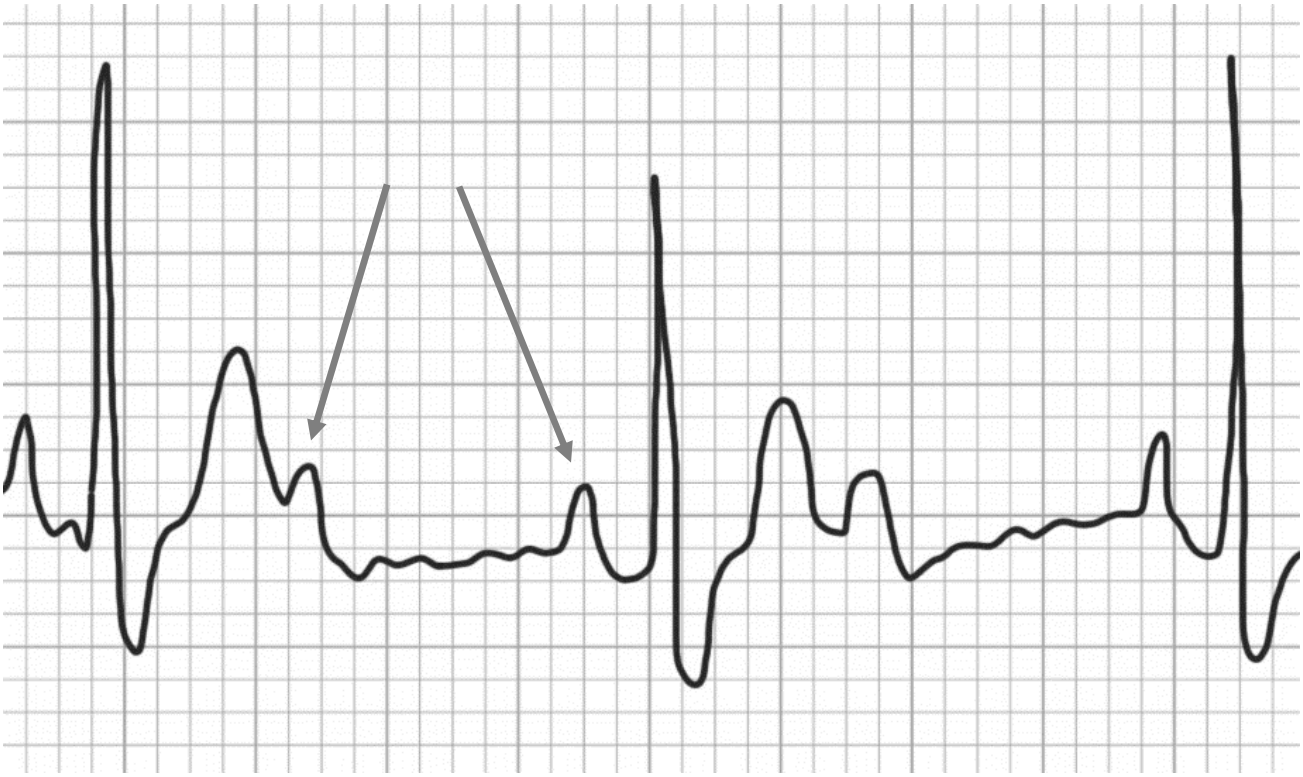

Figure 6: Rhythm strip demonstrating third degree AV block. AV dissociation results in no QRS but a prominent P wave. The absence of the Q wave allows visualisation of the repolarisation of atria (arrow).

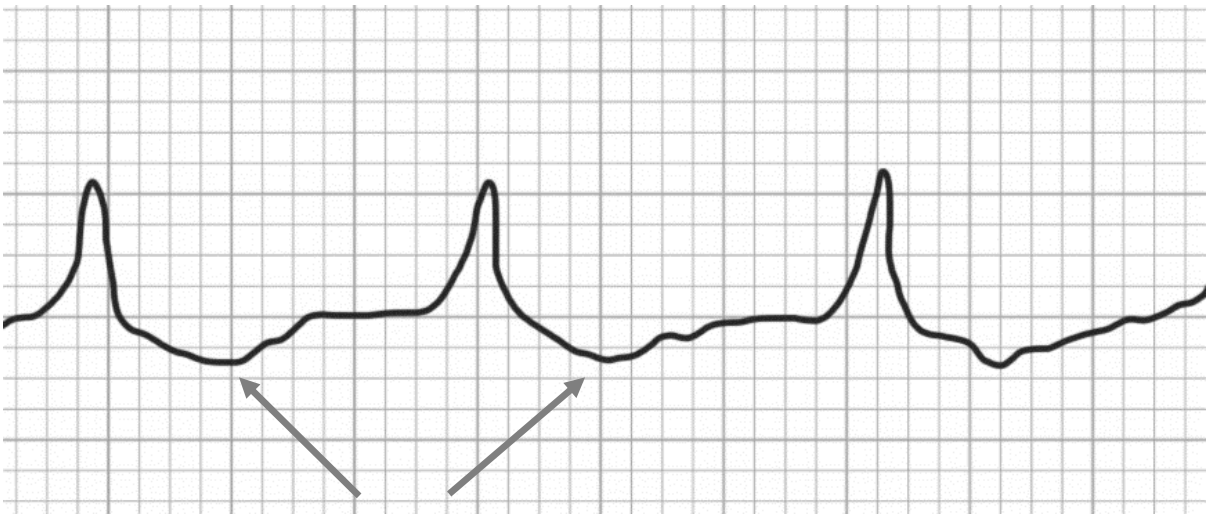

Figure 7: AF with rapid ventricular response. No P waves are seen and the ventricular rate is irregular and rapid. The QRS complex is narrow.

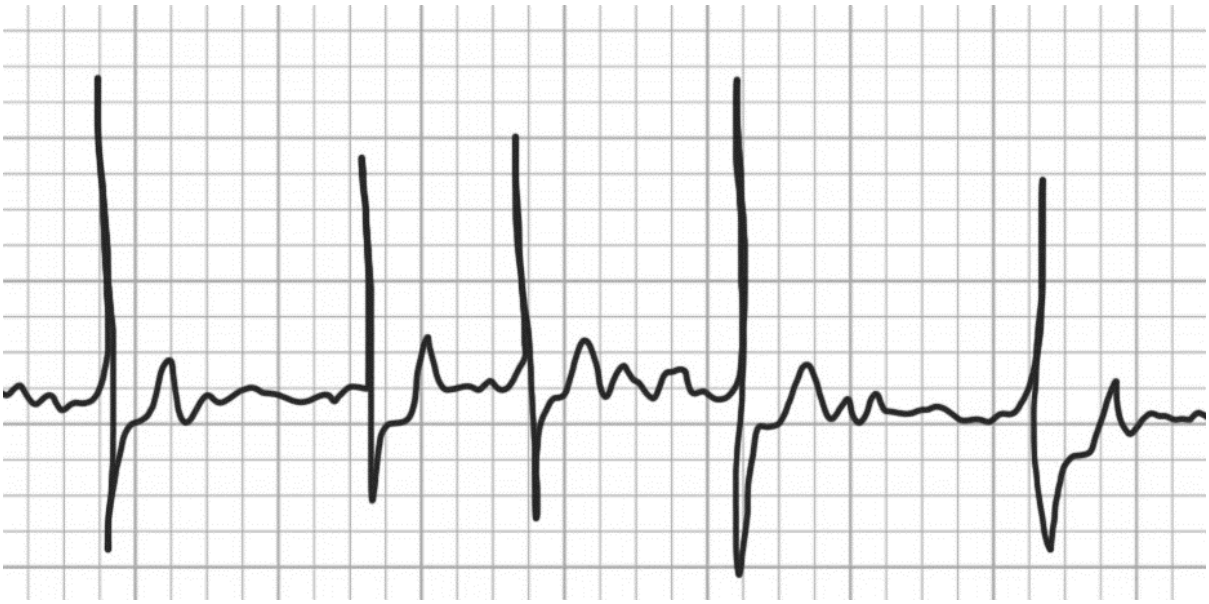

Figure 8: Atrial flutter with the “saw-tooth” flutter wave (arrows).

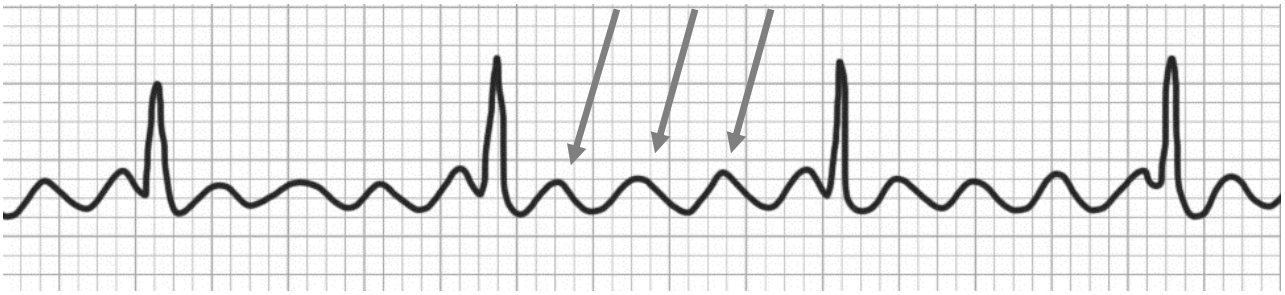

Figure 9: VF with no organised QRS complex.

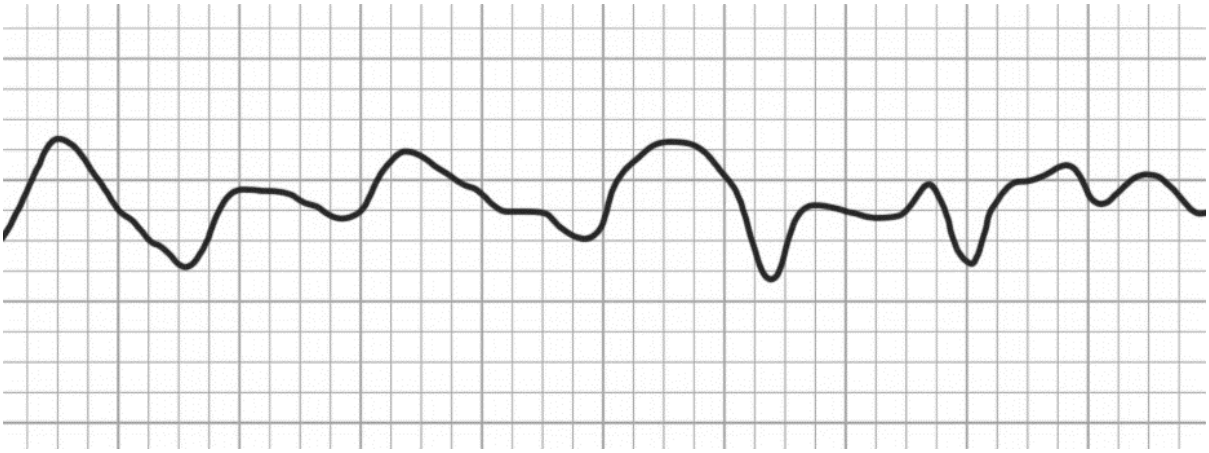

Figure 10: Ventricular ectopics

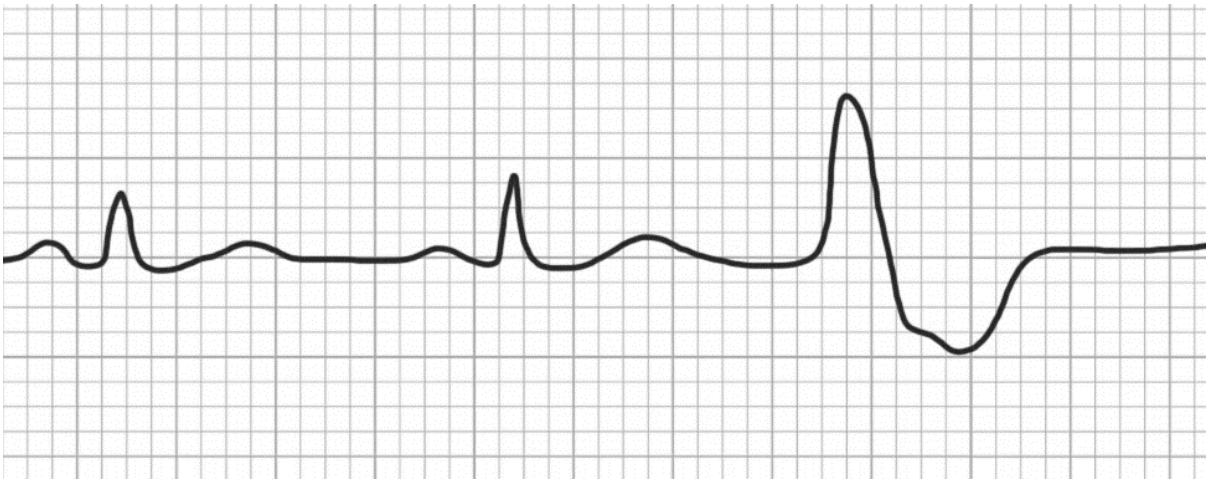

Figure 11: Ventricular pacing spikes (arrow) and the QRS morphology associated with LBBB.

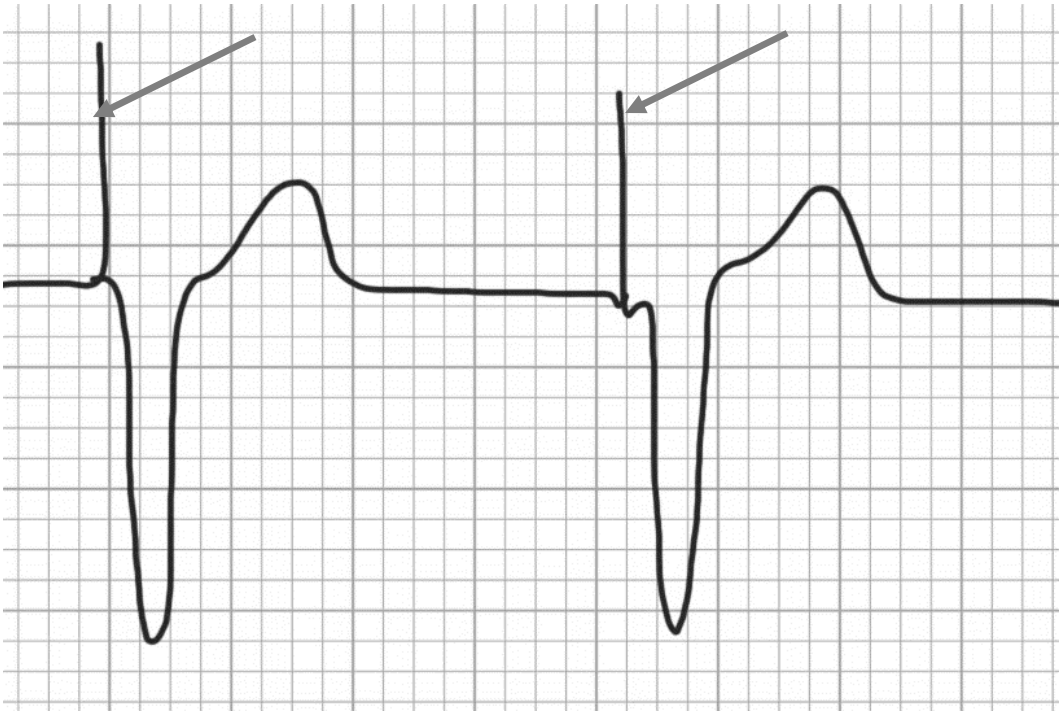

Figure 12: ECG appearance of myocardial ischaemia spectrum clockwise from normal (P QRS T), up-sloping ST depression (>1.5mm), horizontal ST depression (>1mm), down-sloping ST depression (>1mm) and ST elevation.

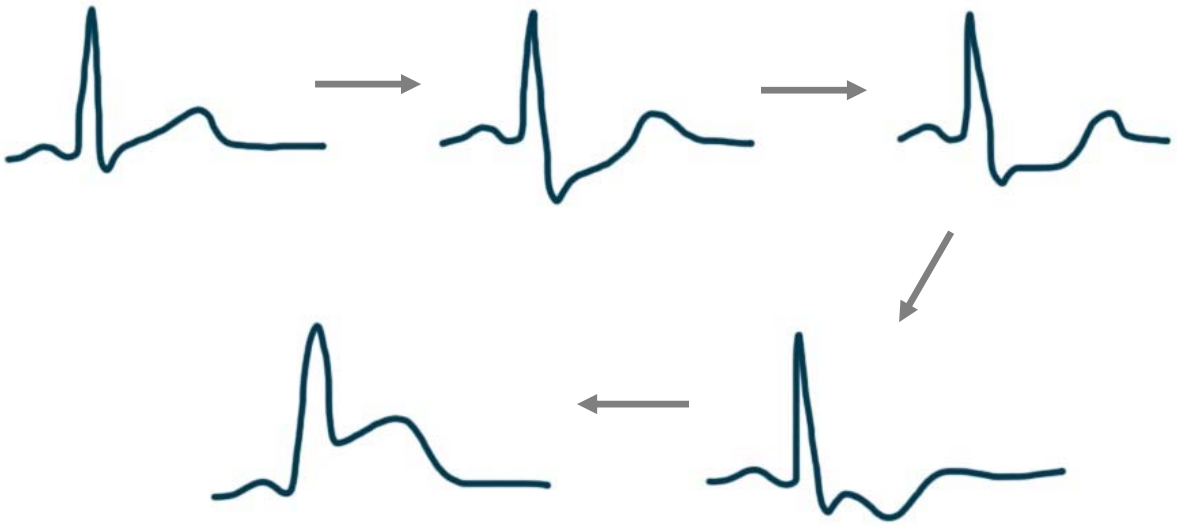

Figure 13: ECG appearance of myocardial infarction spectrum clockwise from normal, hyperacute MI (T wave), transmural (ST elevation), necrosis (ST elevation, Q waves and T wave inversion), necrosis / fibrosis (Q waves and T wave inversion) and fibrosis (Q wave and upright T wave).

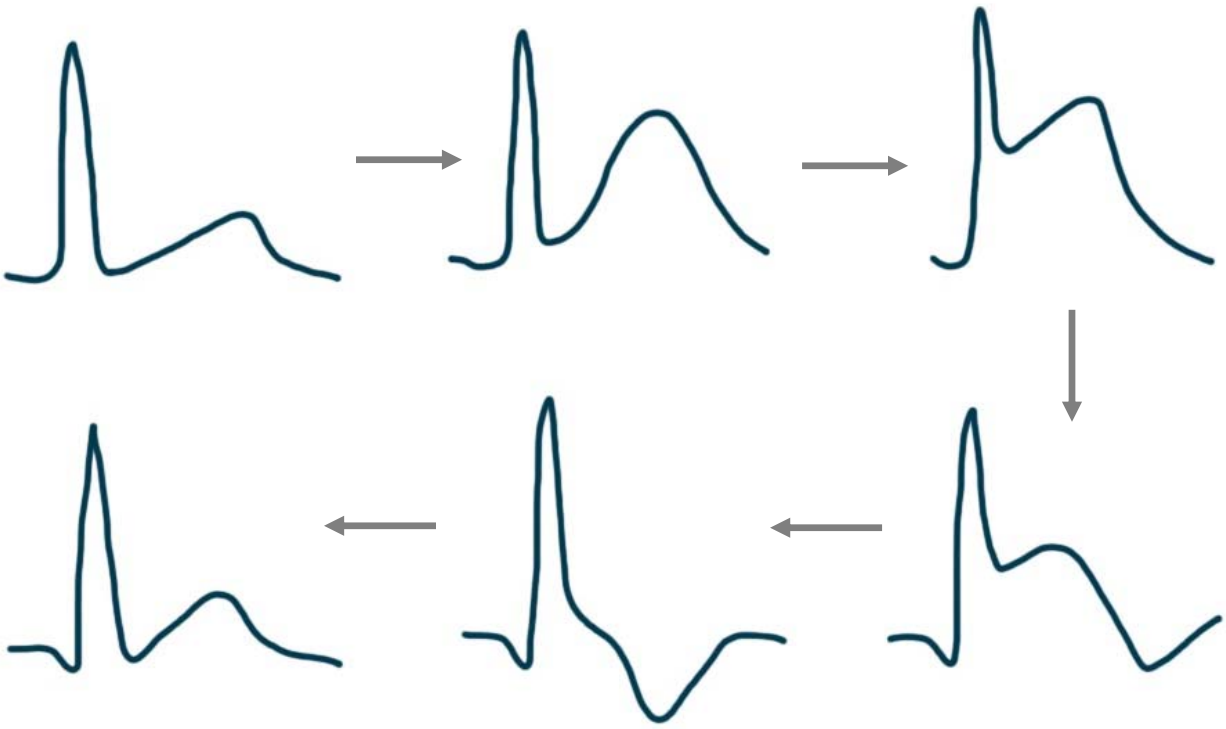

Figure 14: ECG appearance at baseline for a patient with IHD.

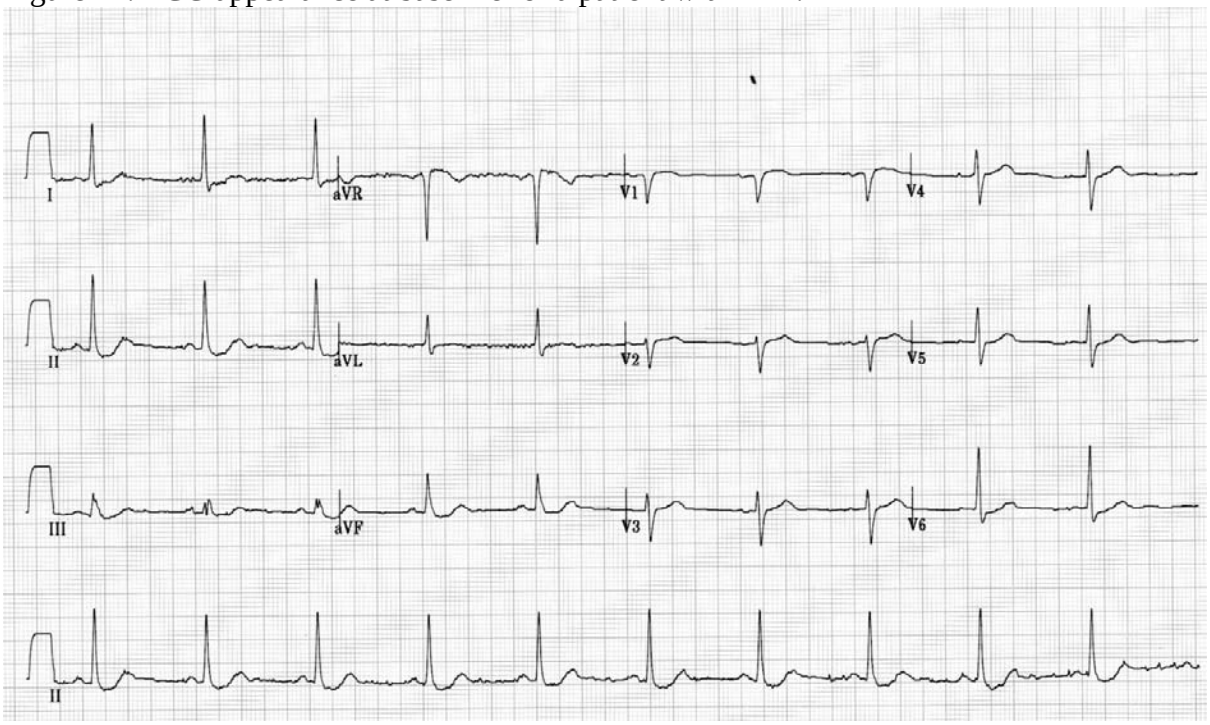

Figure 15: The stress ECG demonstrating infero-lateral up-sloping ST depression.

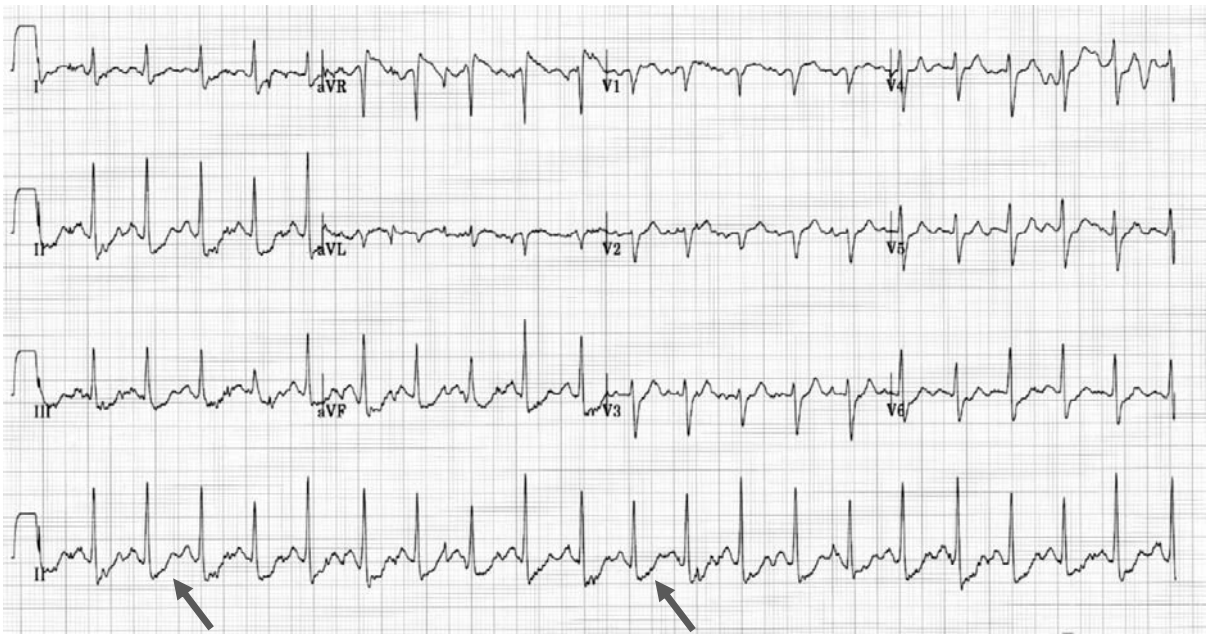

Figure 16: The post-stress ECG demonstrating infero-lateral horizontal ST depression which is indicative of myocardial ischaemia.