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# Understanding the ECG. Part 8: Myocardial ischaemia & infarction (part A)

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## Introduction

Despite advances in medical treatment, and improvements in public health, myocardial ischaemia and infarction remain common causes of hospital admission worldwide (Nabel and Braunwald, 2012). The most common cause is coronary heart disease (CHD), a condition in which the coronary arteries supplying blood to the heart muscle are narrowed or blocked (Marshall, 2011). In the UK, CHD consumes a large part of the NHS budget, and is the cause of death in 15% of men, and 10% of women (British Heart Foundation, 2015). Although mortality rates have fallen over the past four decades, prevalence of the disease is rising, and increasing numbers of patients are seen in both primary and secondary care (Bhatnagar et al, 2015).

Given the scale of the problem, the diagnosis and management of CHD is a priority in all healthcare settings (National Institute of Health and Care Excellence (NICE), 2010). The 12-lead ECG plays an important role in this process, and is especially important in the diagnosis of acute ischaemic events (Hampton, 2013). Changes in the QRS complex, ST-segment, and T-wave occur during ischaemia, and are combined with patient history and blood results when making a diagnosis (Houghton and Gray, 2014). Of particular importance is the identification of persistent ST segment elevation, which has the strongest association with acute myocardial infarction (MI) (Thygesen et al, 2012).

In this eighth instalment of our ECG series, we examine the ECG changes that occur during myocardial ischaemia and infarction, and place them in the context of underlying pathophysiology and clinical management. Because of the size of the subject, we will address it in two parts. In this first part we examine the coronary circulation, the pathophysiology of CHD, and the diagnosis and treatment of ST elevation MI (STEMI). In the second part, to be published next month, we will complete our discussion of STEMI, before moving on to non-ST elevation MI (NSTEMI) and unstable angina.

## The coronary circulation

Every cell in the human body requires oxygen to maintain its normal function (Marieb and Hoehn, 2015). Although many cells can function for brief periods without oxygen, a sustained loss of blood supply results in cellular dysfunction and ultimately cell death (Pappano and Wier, 2014). The heart is especially vulnerable to ischaemia, having a high demand for oxygen, and a poor capacity for

anaerobic metabolism (Ramanathan and Skinner, 2005). To meet its high demand for blood flow, the heart has a dedicated supply in the form of the coronary circulation (Kim et al, 2006).

The coronary circulation consists of a branching network of arteries, veins and capillaries (Grech, 2011). The left and right coronary arteries arise from the base of the aorta, just above the aortic valve, and run across the epicardial surface of the heart in the first part of their course (Marieb and Hoehn, 2015) (*Figure 1*). Both arteries give off important branches that also run on the outer surface of the heart, with smaller sub-branches diving down into the myocardium to feed an extensive capillary network (Tortora and Nielsen, 2014). A network of cardiac veins returns blood to the right atrium, via the coronary sinus (Pappano and Wier, 2013).

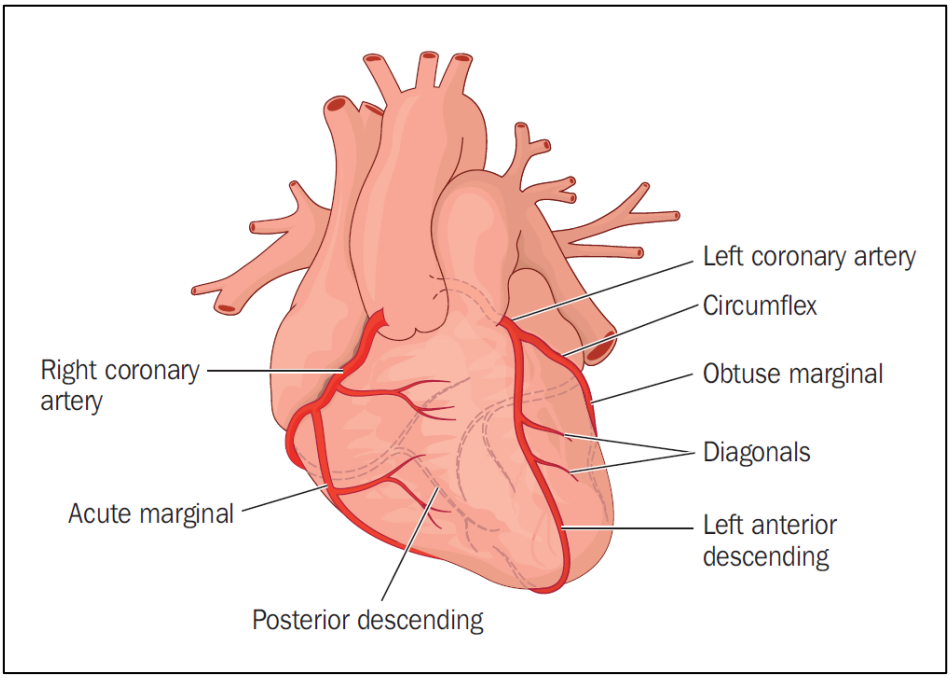


***Figure 1. Typical coronary artery anatomy***

The right coronary artery (RCA) runs in the groove between the right atrium (RA) and right ventricle (RV), before coursing down the right side of the heart towards the apex (Tortora and Nielsen, 2014). It gives off important branches including the sinus node, RV and acute marginal branches (Kim et al, 2006). In most people it also gives rise to the posterior descending artery (PDA), which supplies the inferior wall of the left ventricle (LV) (Cademartiri et al, 2008). Broadly speaking, the RCA supplies blood to the RA, RV, proximal conduction system, and inferior LV (Jowett and Thompson, 2007). Occlusion of the RCA is associated with inferior MI, often with RV involvement (Ondrus et al, 2013). Because the RCA supplies the proximal conduction system, AV block is common in this type of MI (Aehlert, 2011).

The left coronary artery (LCA) runs in the left atrioventricular groove (Marieb and Hoehn, 2015). Following a short initial section referred to as the left main stem (LMS), the artery divides to form the left anterior descending (LAD) and left circumflex (LCx) arteries (Cademartiri et al, 2008). The LAD runs down the front of the heart, giving off diagonal branches that supply the anterior wall of the LV, as well as perforators that penetrate and supply the interventricular septum (Tortora and

Nielsen, 2014). Of the three major coronary arteries, the LAD provides the greatest proportion of blood to the LV (Rinta-Kiikka et al, 2014). Blockage of this vessel therefore has the greatest effect on LV function, and is associated with increased morbidity and mortality (Lee et al, 1995). The LCx continues around the left side of the heart in the atrioventricular groove (Marieb and Hoehn, 2015). Its major branches are the obtuse marginals, which supply blood to the lateral wall of the LV (Cademartiri et al, 2008). It also supplies the left atrium (LA) and posterior wall of the LV (Kim et al, 2006). The relationship between coronary arteries and cardiac territory is summarised in *table 1*.

| **Coronary artery** | **Area of the heart supplied** |
|---|---|
| Right coronary artery (RCA) | RA, RV, sinus node, AV node, inferior LV. |
| Left anterior descending (LAD) | Anterior LV, apex, interventricular septum |
| Left circumflex (LCx) | LA, lateral LV, posterior LV |

***Table 1. Areas of the heart typically supplied by each coronary artery***

## Individual variation

Although the arrangement described above is common, there can be significant variation in the branching pattern of the coronary circulation (Marieb and Hoehn, 2015). In one third of people, the LCA trifurcates after the LMS to form three major branches instead of two (Rinta-Kiikka et al, 2014). The additional artery is referred to as the ramus intermedius, and supplies an area equivalent to the first diagonal branch (Jowett and Thompson, 2007). Other people are born with a single coronary artery supplying the entire heart, or have arteries with highly unusual origins or courses (Cademartiri et al, 2008).

A second area of variation is in collateral circulation. Collaterals are vessels that link coronary arteries together via anastomoses, helping to improve blood supply across the heart (Tortora and Nielsen, 2014). A growth in collateral circulation is seen in people who exercise regularly, and also in individuals with chronic CHD (Klabunde, 2012). Research suggests that one in four people with CHD has sufficient collateral circulation to prevent ischaemia during brief coronary artery occlusion (Seiler et al, 2013). This has a protective effect during acute ischaemic events, and is associated with decreased morbidity and increased survival (Regieli et al, 2009).

This variation in coronary anatomy means that there is not always a direct relationship between ECG findings and the location of ischaemic events in the heart (Rinta-Kiikka et al, 2014). One of the most important confounders to be aware of is the source of the PDA, and therefore the blood supply to the inferior LV (Jowett and Thompson, 2007). In 85% of people, the circulation is said to be right dominant, meaning that the PDA arises from the RCA (Kim et al, 2006). In these people, an inferior MI is the result of RCA occlusion (Hampton, 2013). In contrast, 7-8% of people have a left dominant circulation, in which the PDA arises from the LCx, making this the likely culprit vessel during inferior MI (Houghton and Gray, 2014). In the remaining 7-8% of the population, the coronary circulation is co-dominant; the inferior LV is perfused by branches arising from both the RCA and the LCx (Rinta-Kiikka et al, 2014).

## Pathophysiology of coronary heart disease

As with all arteries, those in the coronary circulation are composed of three layers; an outer 'tunica adventitia', a middle 'tunica media' and an inner 'tunica intima' (Marieb and Hoehn, 2015) (*figure 2*). The adventitia is composed of tough connective tissue, the media of smooth muscle cells, and the intima of endothelial cells (Tortora and Nielsen, 2014). When the demand for blood flow is high, for example during exertion, the smooth muscle in the media relaxes, dilating the vessel lumen and increasing blood flow (Marieb and Hoehn, 2015). This process is regulated by the release of vasoactive hormones from the endothelial cells of the intima (Ramanathan and Skinner, 2005). The endothelial lining also provides a smooth, non-stick surface to the inside of the artery, promoting blood flow and resisting the adhesion of white blood cells (Libby et al, 2011).

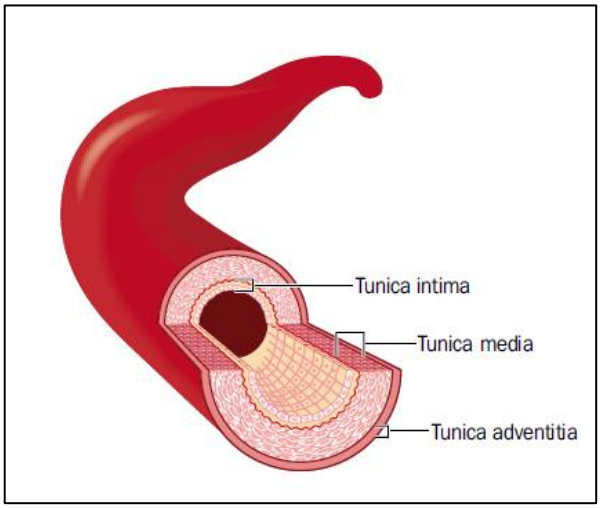


***Figure 2. Arterial wall layers***

CHD occurs when the endothelial lining of the coronary artery becomes damaged by oxidative, haemodynamic or biochemical processes (Nabel and Braunwald, 2012). Common causes of damage include cigarette smoking, hypertension and high levels of glucose or cholesterol in the blood (Yusef et al, 2004). Further risk factors for CHD development are detailed in *box 1*. Damage to the endothelium triggers a chronic inflammatory process in which white blood cells and lipids accumulate in the intimal layer of the artery wall (Libby et al, 2011). Over time, the lesion is swelled by the migration of smooth muscle cells from the media, and the formation of a fibrous cap (Stone et al, 2011).

| **Modifiable risk factors** | **Non-modifiable risk factors** |
|---|---|
| Smoking | Age |
| Alcohol intake | Male gender |
| Lack of physical exercise | Family history of premature CHD |
| Low consumption of fruits and vegetables | Ethnicity |
| Hypertension | |
| Hypercholesterolaemia | |
| Diabetes | |
| Abdominal obesity | |
| Psychosocial factors | |

***Box 1. Risk factors for coronary artery disease (Kennedy 2008)***

As the lesion grows, the lumen of the vessel becomes progressively narrower, although significant impairment of blood flow may not occur until 60 to 70% of the lumen is obstructed (Gould, 2009). At this point, ischaemia may occur during increased demand, resulting in predictable, exertional

symptoms described as stable angina (Grech, 2011). The classical symptom of stable angina is retrosternal chest pain that is provoked by exertion, and relieved by rest or short-acting nitrates (Nabel and Braunwald, 2012). The pain is often described in terms of pressure, tightness or weight on the chest, and may radiate to the shoulders, neck, jaw, back or arms (Fox et al, 2006). It is rarely severe, and usually does not last more than 15 minutes (NICE, 2010). If there is a well-developed collateral circulation, individuals may have few or no symptoms despite high grade stenosis or even complete vessel occlusion (Seiler et al, 2013).

In contrast, unpredictable or severe symptoms are usually related to sudden rupture of the cap covering a lesion (Marshall, 2009). This exposes the lipid rich contents to the circulating blood, triggering platelet aggregation, blood clot formation, and vasoconstriction (Libby et al, 2011) (*figure 3*). This may completely or partially occlude the vessel, resulting in severe ischaemia or infarction of the area supplied by the artery (Steg et al, 2012). These sudden and life threatening thrombotic events are described as acute coronary syndromes (ACS) (Arbab-Zadeh et al, 2012). Unlike stable angina, ACS is typified by chest pain that is persistent, unrelieved by rest or nitrates, and often associated with nausea, vomiting and sweating (NICE, 2010). ACS is a medical emergency, warranting emergency assessment and treatment (Marshal, 2009).

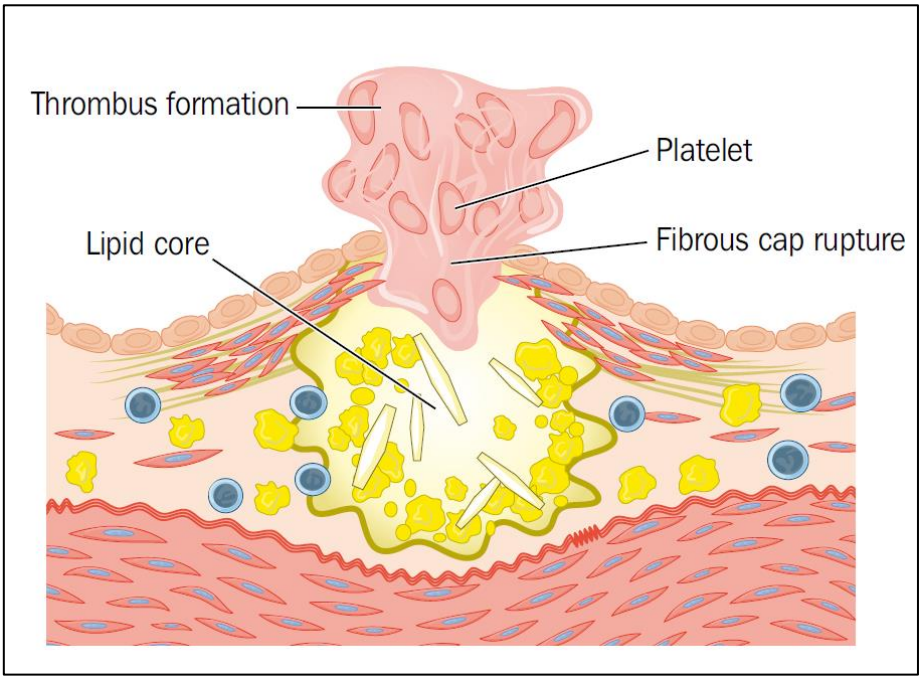


***Figure 3. Rupture of the cap covering an atherosclerotic lesion, resulting in platelet aggregation and blood clot formation (adapted from Libby et al, 2011).***

## The 12 lead ECG in ACS

During the early minutes of an ACS, the 12-lead ECG may be entirely normal despite severe symptoms (Morris and Brady, 2009). Serial ECGs should therefore be performed, ideally at 15-30 minute intervals, or using continuous computer-assisted recording (Thygesen et al, 2012). Serial ECGs will also capture dynamic changes, providing information about the timing of events, the affected artery, the amount of myocardium at risk, and potential treatment strategy (Steg et al, 2012). Where possible, comparison should be made with previous ECGs as prior ischaemic events, or chronic changes, may confound diagnosis (Houghton and Gray, 2014). ECG evaluation should also be made in the light of patient history, physical examination, and the level of biochemical markers, ideally troponin I or T (Hampton, 2013).

Depending on the findings of clinical evaluation, ACS can be classified as one of the following:

- ST elevation MI (STEMI)
- Non-ST elevation MI (NSTEMI)
- Unstable angina

At initial presentation, the focus is on confirming or excluding the presence of STEMI because current treatment guidelines recommend immediate reperfusion therapy in these patients (NICE, 2013; Steg et al, 2012). Troponin levels are not needed to initiate treatment in STEMI, although they are essential to the management of NSTEMI and unstable angina (Marshall, 2011).

## ST elevation MI

STEMI results from the acute occlusion of an epicardial coronary artery, and is associated with transmural ischaemia of a significant area of the heart (Wei et al, 2013). If ischaemia is not relieved, infarction of the affected area may ensue within an hour, with progressive tissue damage as further time elapses (Rinta-Kiikka et al, 2014). Early identification of STEMI, and reperfusion of the affected area, limits infarction size and decreases complications and mortality (McNamara et al, 2006).

STEMI can be identified by a patient history of typical symptoms, in association with ST elevation in at least two anatomically contiguous leads, in other words two ECG leads looking at the same area of the heart (Steg et al, 2012). For the purpose of contiguous leads, the ECG can be divided into three groups of leads, each of which corresponds approximately with an anatomical region, and the blood supply from a major coronary artery (Morris and Brady, 2009) (*table 2*).

| **ECG leads** | **Anatomical area** | **Probable culprit artery** |
|---|---|---|
| II, III, aVF | Inferior LV wall | Right coronary artery |
| V1-V4 | Anterior LV wall | Left anterior descending |
| I, aVL, V5, V6 | Lateral LV wall | Left circumflex |

***Table 2. Contiguous leads in STEMI diagnosis. Probable culprit artery is based on a typical right dominant circulation.***

Although ST elevation is the key diagnostic feature in STEMI, a number of associated ECG changes are commonly seen (Hampton, 2013). These appear in a sequential fashion, creating a dynamic ECG pattern that is characteristic of STEMI (Thygesen et al, 2012). These dynamic changes help to differentiate STEMI from other causes of chest pain, although not every feature is seen in every patient (Houghton and Gray, 2014). Typical features include (Garcia, 2015) (*figure 4*)

- Hyperacute T-waves
- ST segment elevation
- Q-wave formation
- Loss of R-wave height
- T-wave inversion
- Reciprocal ST segment depression

Let's examine each of these changes in turn.

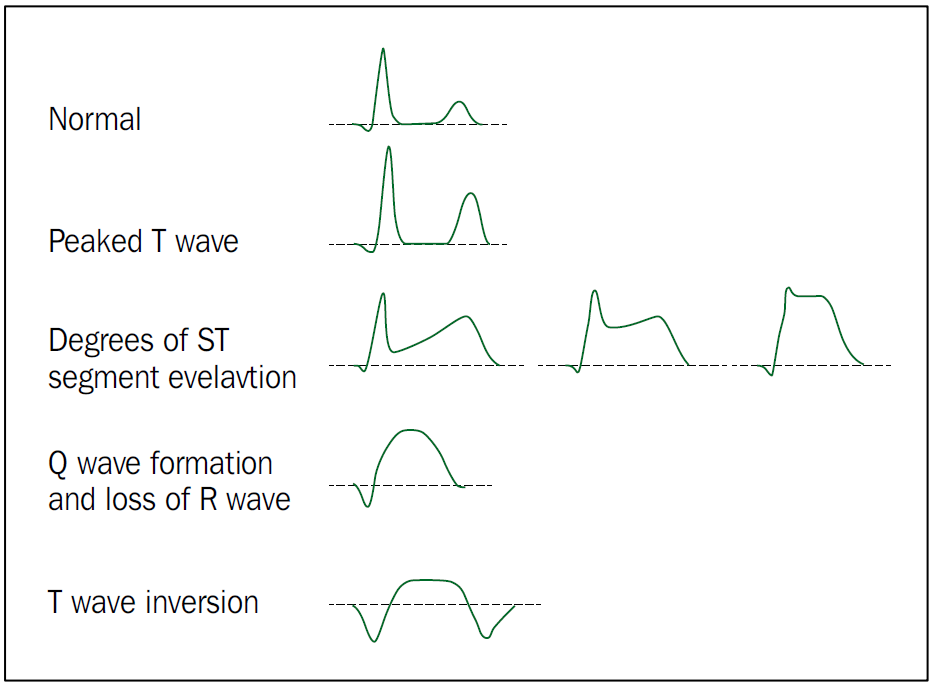


***Figure 4. Evolution of a STEMI***

## Hyperacute T-waves

Hyperacute T-waves are the first ECG sign of an evolving STEMI, and appear within five to thirty minutes of artery occlusion (Morris and Brady, 2009) (*figure 4*). Because of the time that usually elapses between symptom onset and the recording of an ECG, they may not be seen, making ST elevation the first detected ECG change (Houghton and Gray, 2014). Hyperacute T-waves are taller, more symmetrical, and more peaked than normal (Garcia, 2015). They appear in the ECG leads overlying the ischaemic area of the heart, and tend to be more prominent in the chest leads (Hampton, 2013). Comparison with previous ECGs may be needed to confirm their presence (Morris and Brady, 2009).

## ST segment elevation

The ST segment is the short section of baseline that joins the end of the QRS complex to the T-wave *(figure 5*). This segment is normally isoelectric, meaning that it is at the same level as the TP segment (Aehlert, 2011). Elevation of the ST segment follows hyperacute T-waves in the evolution of STEMI, and occurs within the first few hours of ischaemia (Houghton and Gray, 2014) (*figure 6*). The initial rise may be small, however a progressive increase in the height of the ST segment often occurs (Garcia, 2015). The ST segment may merge with the T-wave, with complete loss of the ST-T angle (Morris and Brady, 2009). If the ST segment reaches the top of the QRS as well, a single monophasic waveform results, commonly referred to as a 'tombstone' (Hampton, 2013). *Figure 7* illustrates different degrees of ST elevation.

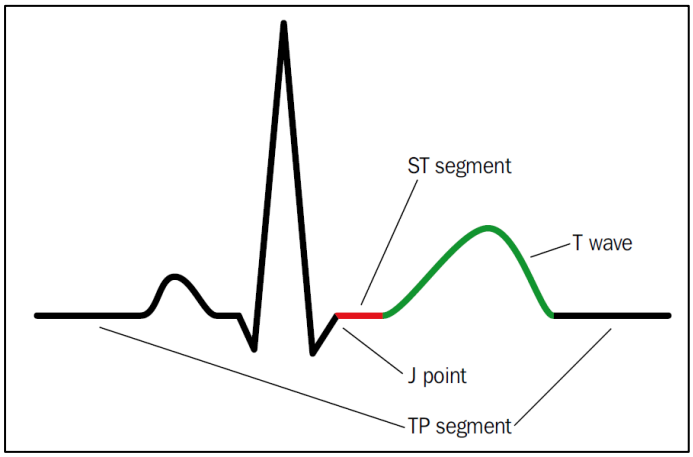


***Figure 5. The ST segment (in red) starts at the J point, and is isoelectric with the TP segment.***

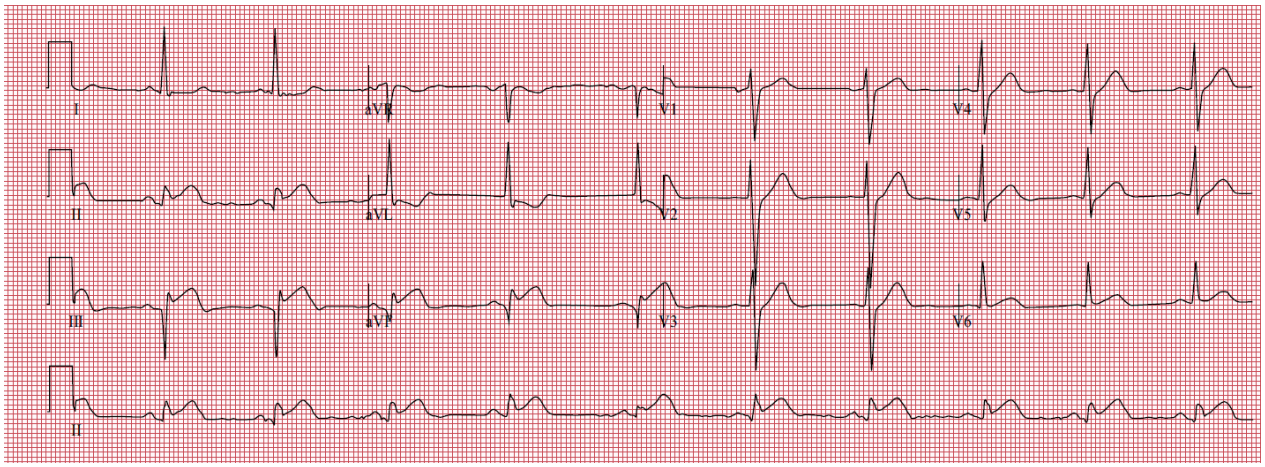


***Figure 6. Inferior STEMI. There is > 1mm of ST elevation in leads II, III and aVF. The ST depression in lead aVL is a reciprocal change.***

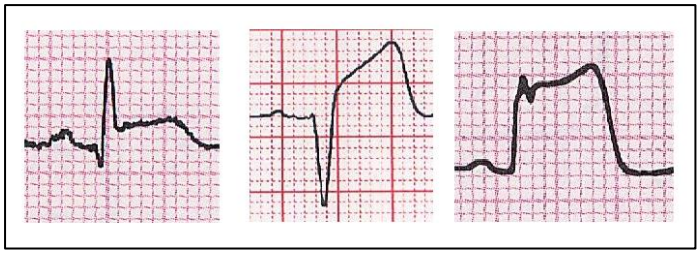

***Figure 7. Degrees of ST elevation. In the right hand example, the ST segment has almost entirely merged with the QRS and T-wave to form a single 'tombstone' complex.***

ST segment elevation is measured at the J-point (Houghton and Gray, 2014). This is the point where the QRS complex transitions into the ST segment (*figure 5*). Elevation of the J point is a normal variant in leads V2 and V3, especially in young men (Hampton, 2013). This phenomenon is often referred to as 'high take-off' or an 'early repolarisation pattern' (Garcia, 2015) (*figure 8*). The degree of ST elevation required to diagnose STEMI is therefore greater in these leads, and greater in men than women (Thygesen et al, 2012) (*table 3*).

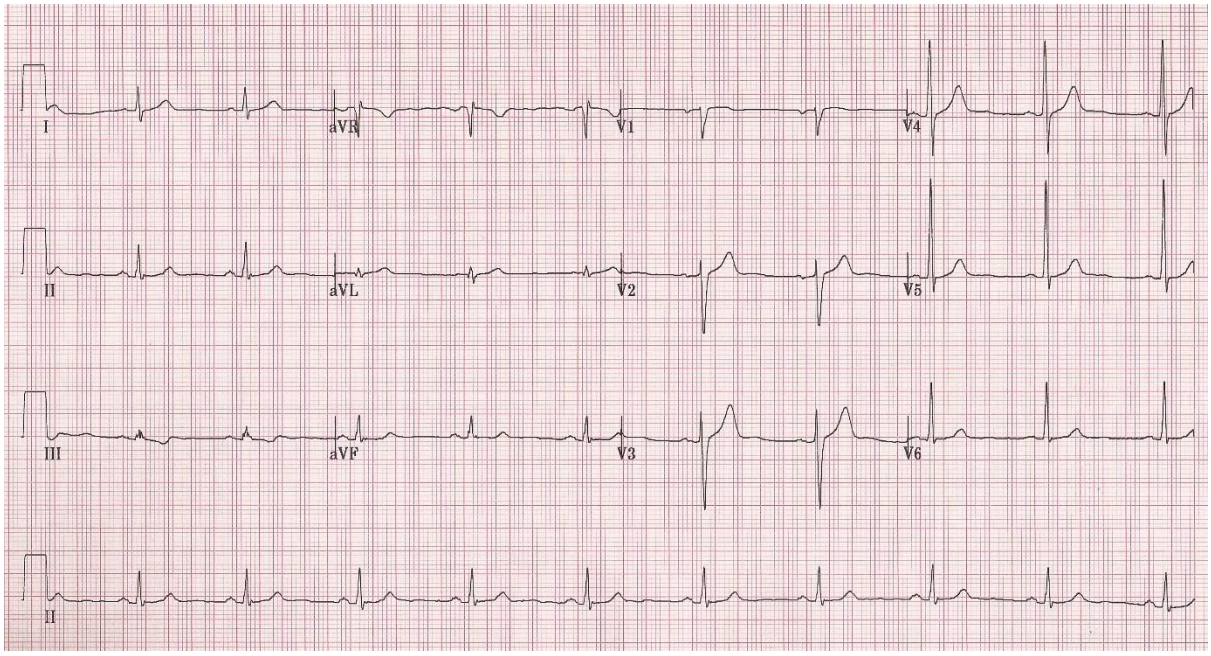


***Figure 8. High take off in leads V2 and V3. The patient was a fit and healthy young man attending for routine health screening. There is isolated T-wave inversion in lead III, which is normal variant.***

| ECG leads | Population | ST elevation considered diagnostic |
|---|---|---|
| All leads except V2/V3 | All patients | ≥ 1 mm |
| V2/V3 | Women | ≥ 1.5 mm |
| V2/V3 | Men ≥ 40 years old | ≥ 2 mm |
| V2/V3 | Men < 40 years old | ≥ 2.5 mm |

***Table 3. Degree of ST elevation needed to diagnose STEMI in the standard ECG leads. Assumes normal calibration of the ECG (10mm = 1mV), and the absence of LVH or LBBB.***

## Q-wave formation and loss of R-wave height

A Q-wave refers to any downward deflection preceding an R-wave (Aehlert, 2011). Small, narrow Q-waves are a normal finding in the leads facing the LV, and reflect left to right depolarisation of the interventricular septum (Hampton, 2013). These 'septal Q-waves' are normally seen in leads I, aVL, aVF, V4, V5 and V6 (Thygesen et al, 2012) (*figure 9*). A Q-wave is also a normal finding in lead aVR, and is a normal variant in leads V1 and III (Houghton and Gray, 2014).

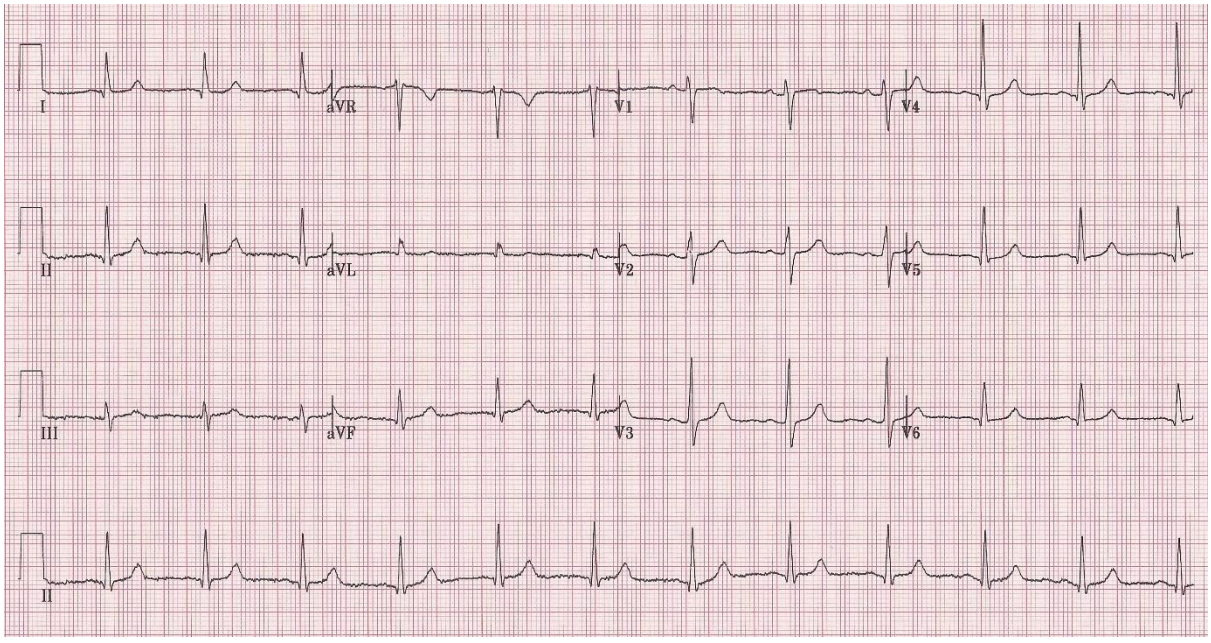


***Figure 9. Septal Q-waves. There are small, narrow Q-waves in leads I, II, aVL, aVF, V4-V6.***

Abnormal, or pathological, Q-waves may develop as a STEMI evolves, and usually appear while the ST segment is still elevated (Houghton and Gray, 2014). They are caused by a loss of viable myocardium below the recording electrode, and are therefore a marker of tissue necrosis (Garcia, 2015). They are often accompanied by a loss of R-wave height in the same leads (Aehlert, 2011) (*Figure 10*). The explanation for Q-wave appearance is simple. Infarcted myocardium is electrically inert – it does not produce electricity, and is therefore invisible to the ECG (Hampton, 2013). An ECG

electrode placed over an area of dead tissue therefore 'sees' through the infarcted area to the healthy wall on the opposite side of the heart. Because depolarisation starts at the endocardium and moves outwards through the heart wall, the electrical vector that the lead sees is moving away from it, and therefore a negative deflection is recorded (Jowett and Thompson, 2007).

The timing of pathological Q-wave formation is variable; it may occur within the first few hours, or not until 24 hours have elapsed (Morris and Brady, 2009). Because of this, Q-wave development does not accurately predict the timing of ischaemic events, nor does it indicate a completed MI (Marshall, 2009). Q-waves often persist as a permanent marker of MI, although in smaller infarcts contraction of scar tissue during healing can result in their disappearance (Rinta-Kiikka et al, 2014).

Pathological Q-waves are usually wide and/or deep, and may occur in leads where Q-waves are not normally seen (Marshall, 2009). Houghton and Gray (2014) suggest that the features of abnormal Q-waves are

- Width > 1 small square, and/or
- Depth >2 small squares / > 25% of the subsequent R-wave height

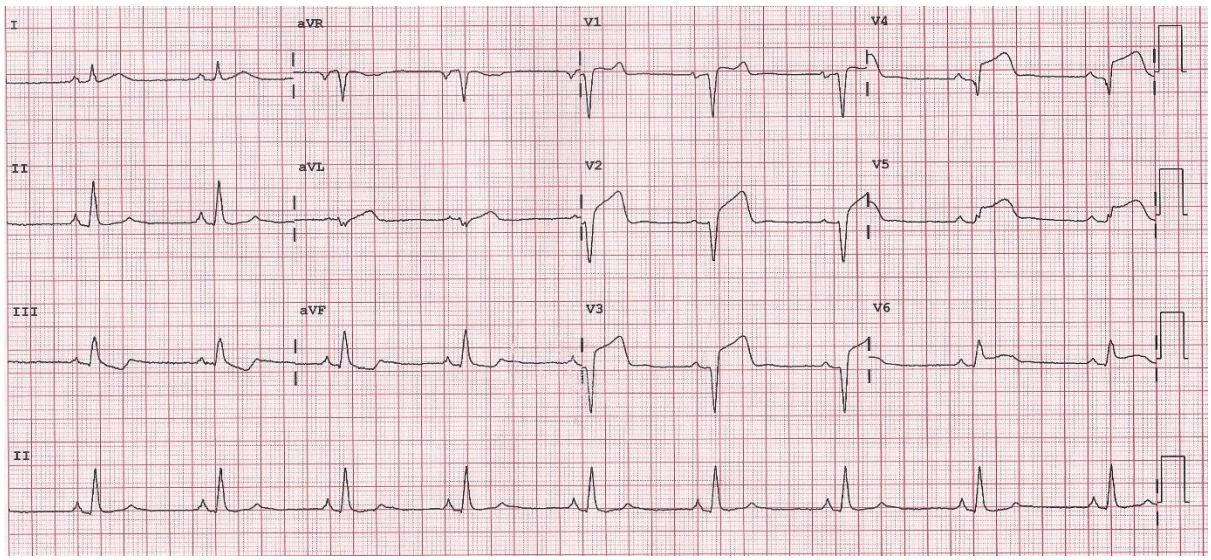


***Figure 10. Anterior STEMI. Note the ST elevation in leads V1-V6, and deep, wide Q-waves in leads V1-V4.***

## T-wave inversion

With further evolution of the STEMI, the ST segment starts to return towards baseline, and the T-waves begin to invert (Garcia, 2015) (figure 11). It may take several weeks for the ST segment to completely normalise; T-wave inversion may persist for months, or even remain as a permanent marker of infarction (Morris and Brady, 2009). Persistent ST elevation is suggestive of an LV aneurysm, although it is not a reliable indicator (Hampton, 2013) (*figure 12*). Houghton and Gray (2014) suggest that this late complication of MI occurs in around 10% of survivors.

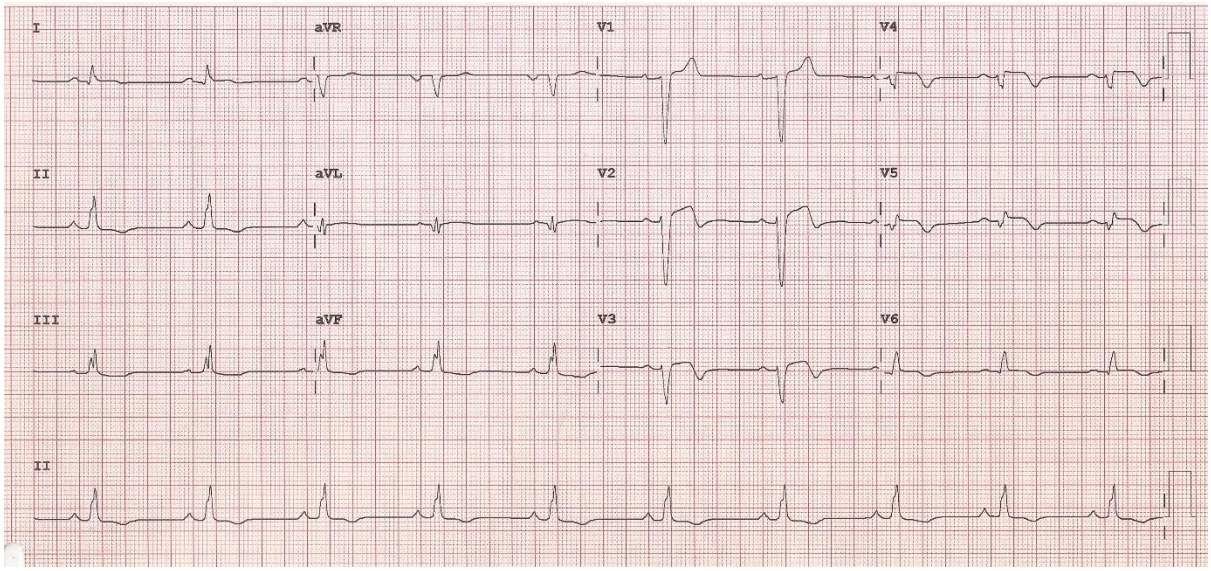


***Figure 11. The same patient as figure 10, following primary PCI. The ST segments are returning towards baseline and the T-waves are inverting.***

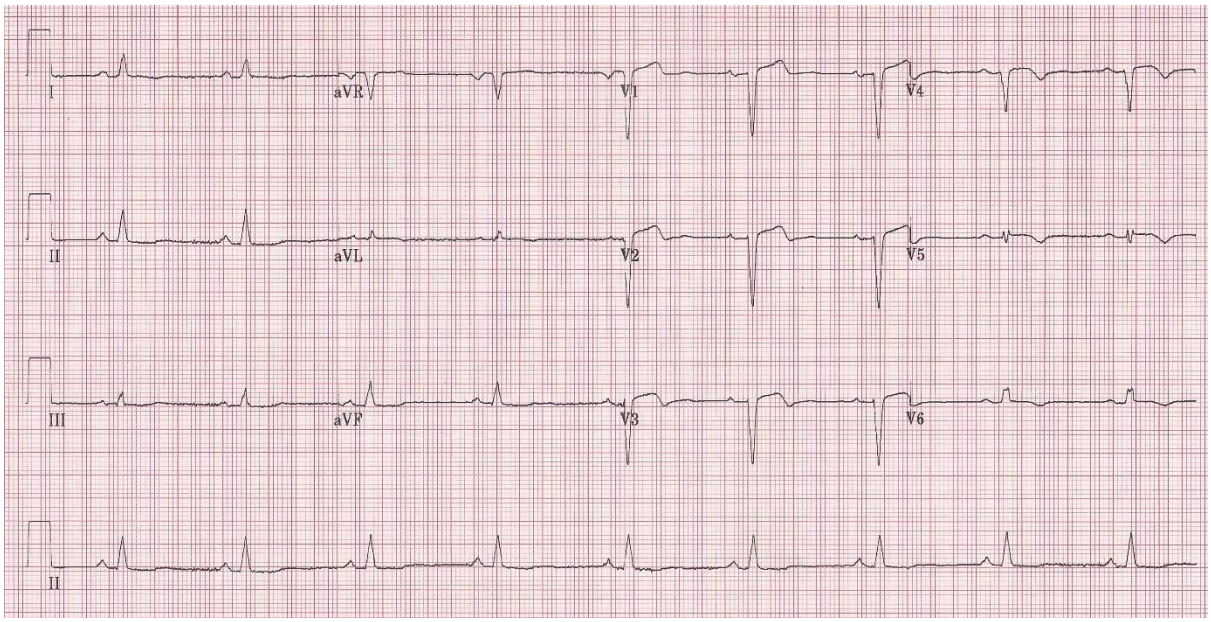


***Figure 12. The same patient, 12 months post MI. The ST segments are still slightly elevated. Echocardiography excluded LV aneurysm. Note the Q-waves, loss of R-wave height and inverted T-waves.***

## Reciprocal ST depression

Reciprocal ST depression refers to changes that are seen in leads remote from the region of the infarct (Houghton and Gray, 2014) *(figure 6*). It is often described as a 'mirror image' change, in other words the ST depression seen reflects ST elevation occurring on the other side of the heart (Garcia, 2015). Reciprocal ST depression is seen in around 70% of inferior STEMI, and 30% of anterior (Morris and Brady, 2009). It has a high positive predictive value for STEMI, and is therefore useful in supporting a diagnosis when ECG features or symptoms are atypical (Hampton, 2013).

The cause of reciprocal ST depression is uncertain; it may represent ischaemia remote from the site of the infarction, or be a benign electrical phenomenon (Morris and Brady, 2009). Recent research suggests that persistent reciprocal ST depression after STEMI is associated with increased infarct size, and a higher risk of adverse cardiovascular events in the future (Reinstadler et al, 2015).

## Treatment of STEMI

The treatment priority for patients presenting with STEMI is to re-open the occluded artery, and reperfuse the ischaemic area of the heart (Steg et al, 2012). One half of potentially salvageable tissue may be lost within the first hour of ischaemia, and two thirds by three hours (Reimer et al, 1977). Time is therefore of the essence (Lassen et al, 2013). Reperfusion can be achieved by one of two methods; thrombolysis or primary percutaneous coronary intervention (PCI) (Jowett and Thompson, 2007).

Thrombolysis uses intravenous drugs to dissolve the clot blocking the artery, and was the treatment of choice in the 1980s and early 1990s (Nabel and Braunwald, 2012). Although thrombolysis improves outcomes in most patients, it is unsuccessful in 20-30% of cases (Department of Health (DH), 2008). It is also associated with a risk of acute bleeding, and causes haemorrhagic stroke in 1% of recipients (NICE, 2013). It can, however, be administered rapidly in any healthcare setting, including local healthcare facilities or the back of an ambulance (Gershlick et al, 2013). It may be the only viable option in remote locations with poor access to a large hospital (Redberg, 2012).

In contrast, PCI requires admission to a hospital equipped and staffed to perform cardiac catheterisation (Nabel and Braunwald, 2012). This limits treatment to specialised centres, usually located in large, regional hospitals (DH, 2008). Coronary angiography is performed to identify the blocked artery, and balloon angioplasty to re-open it (Cooper, 2015). A stent is usually deployed to prevent re-occlusion (Stefanini and Holmes 2013). Other techniques such as clot aspiration may also be used to maximise vessel patency, and to minimise complications such as downstream embolization of micro-emboli (Lam, 2015). PCI is associated with fewer complications than thrombolysis, and improved outcomes provided it is delivered in a timely fashion (Asseburg et al, 2007).

The speed with which reperfusion can be delivered is the key discriminator in deciding between thrombolysis and PCI (Gershlick et al, 2013). Current national guidance recommends PCI if it can be delivered within 120 minutes of when thrombolysis could be given (NICE, 2013). If the delay is likely to be greater than 120 minutes, thrombolysis is the preferred option. There are several caveats to this guidance. Firstly, PCI is recommended in patients with cardiogenic shock, even if treatment delay is likely to exceed two hours. Secondly, the guidance only applies if the patient has presented within 12 hours of symptom onset. If presentation is later than this, PCI is not recommended unless there is evidence of ongoing ischaemia (NICE, 2013). This recommendation is based on research,

including the large Occluded Artery Trial, that failed to show a benefit of delayed PCI compared to medical treatment (Hochman et al, 2006). National audit data for England in 2013-14 shows that 98.5% of eligible STEMI patients received PCI, and only 1.5% thrombolysis (Myocardial Ischaemia National Audit Project, 2014). Other acute interventions for STEMI are listed in *table 4*, however their delivery should not delay reperfusion (NICE, 2010).

| **Intervention** | **Rationale / notes** |
|---|---|
| Aspirin 300mg orally | To inhibit further platelet aggregation |
| Sublingual or buccal nitrates (e.g. GTN spray) and intravenous opiates (usually morphine) | To relieve pain and reduce cardiac work; pain increases sympathetic tone, making the heart work harder and use more oxygen. |
| Intravenous anti-emetic | To relieve nausea and vomiting, which is a common side effect of opiates, as well as a common symptom of STEMI. |
| Supplemental oxygen, if indicated. | Only if SpO2 falls below 94%. Target SpO2 is<br>• 94-98% in most adults<br>• 88-92% in people with chronic obstructive pulmonary disease, when there is a risk of hypercapnic respiratory failure |
| Continuous monitoring of ECG and vital signs | To detect hypoxia, hypotension and arrhythmias. Hypotension may result from STEMI, or the administration of nitrates / opiates. Arrhythmias are common in acute STEMI, especially AV blocks and VT/VF, and may be life threatening. |
| Serial ECGs | To detect dynamic ECG changes |
| Emergency pacing / defibrillation | Should be available in the event of life threatening arrhythmia. |
| Information and reassurance | STEMI is a frightening experience for both patient and relatives. Simple information giving and appropriate reassurance can reduce anxiety. |

***Table 4. Interventions during acute STEMI (NICE, 2010; Steg et al, 2012)***

## Conclusion

Although ACS presents in a variety of ways, persistent ST elevation has the strongest association with acute myocardial infarction, and is an indication for emergency reperfusion (Steg et al, 2012). During a STEMI, various ECG changes may be seen, including hyperacute T-waves, ST segment elevation, pathological Q-waves, loss of R-wave height, and T-wave inversion (Houghton and Gray, 2014). These progressive changes are seen in contiguous leads, and provide information about the location of the infarct, its progress, and the area of myocardium at risk (Thygesen et al, 2012).

Although ECG changes during STEMI are characteristic, they must be correlated with patient history and physical examination in reaching a diagnosis (Hampton, 2013). Care should also be taken to

exclude normal variants such as early repolarisation or septal Q-waves (Garcia, 2015). Once diagnosis is confirmed, reperfusion by PCI is the preferred option, although thrombolysis is an alternative if a treatment delay of more than two hours is anticipated (NICE, 2013). Aspirin, analgesia and continuous monitoring are also important while reperfusion is arranged, but should not delay definitive treatment (NICE, 2010).

## Next month

Despite the usefulness of the ECG in the diagnosis of STEMI, it does have some weaknesses (Steg et al, 2012). Firstly, there are a number of non-ischaemic conditions that produce ST elevation on the ECG, some of which also cause chest pain (Houghton and Gray, 2014). This can result in diagnostic uncertainty, or misdiagnosis (Edhouse et al, 2009). Secondly, the right ventricle and posterior wall of the heart are not 'seen' by the standard ECG leads, making infarction of these regions harder to diagnose (Wei et al, 2013). Additional ECG leads are often necessary (Garcia, 2015). Thirdly, new left bundle branch block is a STEMI equivalent, and an indication for reperfusion (Thygesen et al, 2012). It may also be long standing problem with a non-ischaemic aetiology, making diagnosis problematic (Kumar et al, 2013).

Next month, we complete our examination of STEMI by considering these difficulties. We also turn our attention to the other two ACS presentations, NSTEMI and unstable angina, and consider the role of the ECG in their management.

## Key points

- Acute coronary syndromes (ACS) are life threatening events associated with coronary artery lesion rupture and thrombus formation. Symptoms include persistent chest pain that is not relieved by rest or nitrates, nausea and vomiting, and sweating.
- ACS can be divided into STEMI, NSTEMI and unstable angina, according to ECG appearance and the result of biochemical markers such as troponin. Persistent ST segment elevation has the strongest association with myocardial infarction, and is an indication for immediate thrombolysis or PCI. Confirmation of troponin rise is not needed to initiate reperfusion in STEMI.
- In the standard ECG leads, the diagnostic criteria for STEMI is ST elevation of 1mm or more in two contiguous ECG leads, although the cut-off is increased in leads V2 and V3. The diagnostic level in these two leads depends on age and gender.
- During STEMI, ST elevation is often accompanied by progressive change in the ECG that includes Q-wave formation, loss of R-wave height, and T-wave inversion. Hyperacute T-waves may precede ST elevation, but are often not seen. Dynamic change in the ECG strengthens the diagnostic certainty of STEMI, as does reciprocal ST depression.
- Delay in achieving reperfusion is associated with larger infarct size and increased mortality. Current NICE guidance recommends PCI if it can be performed without excessive delay. If the delay to treatment is likely to exceed two hours, thrombolysis should be considered instead. Additional treatment such as aspirin, analgesia and oxygen may be given, but should not delay reperfusion. Patients should be monitored for hypoxia, hypotension and arrhythmias.

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