

## Learning outcomes

To understand:

- ▶ **The disease process which gives rise to acute coronary syndromes**
- ▶ **How to differentiate between the acute coronary syndromes**
- ▶ **The immediate treatment of acute coronary syndromes**
- ▶ **Management of patients after recovery from an acute coronary syndrome**

## Introduction

Whilst rapid resuscitation offers the best chance of recovery from cardiac arrest, it is clearly better to prevent cardiac arrest whenever possible. Many cardiac arrests are caused by underlying coronary artery disease and occur in the context of an acute coronary syndrome (ACS). It is therefore important that the ALS provider understands how to recognise an ACS, how to assess a patient with an ACS, and what treatments may reduce the risk of cardiac arrest and death.

## Definitions and pathogenesis

The acute coronary syndromes (ACS) comprise:

- Unstable angina
- Non-ST-segment-elevation myocardial infarction
- ST-segment-elevation myocardial infarction

These clinical syndromes form parts of a spectrum of the same disease process. In the vast majority of cases this process is initiated by the fissuring of an atheromatous plaque in a coronary artery causing:

- haemorrhage into the plaque causing it to swell and restrict the lumen of the artery;
- contraction of smooth muscle within the artery wall, causing further constriction of the lumen;
- thrombus formation on the surface of the plaque, which may cause partial or complete obstruction of the lumen of the artery, or distal embolism.

The extent to which these events reduce the flow of blood

to the myocardium largely determines the nature of the clinical ACS that ensues.

## Angina (stable and unstable)

Angina is pain or discomfort caused by myocardial ischaemia and is felt usually in or across the centre of the chest as tightness or an indigestion-like ache. As with acute myocardial infarction (AMI), the pain/discomfort often radiates into the throat, into one or both arms, and into the back or into the epigastrium. Some patients experience angina predominantly in one or more of these areas rather than in the chest. Many patients perceive it as discomfort rather than pain. As with AMI, angina is sometimes accompanied by belching and this may be misinterpreted as evidence of indigestion as the cause of the discomfort. Pain of this nature, which is provoked only by exercise and which settles promptly when exercise ceases, is referred to as stable angina and is not an ACS.

In contrast, **unstable angina** is defined by one or more of:

1. Angina on exertion, occurring over a few days with increasing frequency, provoked by progressively less exertion. This is referred to as 'crescendo angina'.
2. Episodes of angina occurring recurrently and unpredictably, without specific provocation by exercise. These episodes may be relatively short-lived (e.g. a few minutes) and may settle spontaneously or be relieved temporarily by sublingual glyceryl trinitrate, before recurring within a few hours.
3. An unprovoked and prolonged episode of chest pain, raising suspicion of AMI, but without definite ECG or laboratory evidence of AMI (see below).

In unstable angina, the ECG may:

- a) be normal;
- b) show evidence of acute myocardial ischaemia (usually ST segment depression);
- c) show non-specific abnormalities (e.g. T wave inversion).

In unstable angina, cardiac enzymes are usually normal (but remember that there are causes other than myocardial infarction for elevated muscle enzymes such as creatine kinase [CK]), and troponin release is absent. ECG abnormality, especially ST segment depression, is a marker of increased risk of further coronary events in patients with unstable angina. However, a normal ECG

and absent troponin release does not necessarily mean that a patient with unstable angina is not at high risk of early further life-threatening coronary events. Only if the ECG and troponin concentration are normal, and further risk assessment (e.g. by exercise testing or non-invasive imaging) does not indicate evidence of reversible myocardial ischaemia, should other possible causes of acute chest pain be considered if the initial history suggested unstable angina.

### **Non-ST-segment-elevation myocardial infarction (NSTEMI)**

Acute myocardial infarction typically presents with chest pain that is felt as a heaviness or tightness or indigestion-like discomfort in the chest or upper abdomen, usually sustained for at least 20 - 30 min, often longer. The chest pain/discomfort often radiates into the throat, into one or both arms, into the back or into the epigastrium. Some patients experience the discomfort predominantly in one or more of these other areas rather than in the chest. Sometimes it may be accompanied by belching and this may be misinterpreted as evidence of indigestion as the cause of the discomfort.

When patients present with chest pain suggestive of AMI, non-specific ECG abnormalities such as ST segment depression or T wave inversion (Figures 4.1 and 4.2) or occasionally a normal ECG, and laboratory tests showing release of troponin (with or without elevated plasma concentrations of cardiac enzymes) this indicates that myocardial damage has occurred. This is referred to as NSTEMI. In this situation it is less likely that there has been abrupt complete occlusion of the culprit artery than in ST-segment-elevation MI (STEMI).

The amount of troponin or cardiac enzyme released reflects the extent of myocardial damage. Some of these patients will be at high risk of progression to coronary occlusion, more extensive myocardial damage, and sudden arrhythmic death. The risk of this is highest in the first few hours, days and months after the index event and diminishes progressively with time.

NSTEMI and unstable angina are classified together as 'non-ST-segment-elevation ACS' because the treatment of the two is essentially the same and differs in some respects from the treatment of STEMI. Treatment is dictated largely by assessment of risk.

### **ST-segment-elevation myocardial infarction (STEMI)**

A history of sustained acute chest pain typical of AMI, accompanied by acute ST segment elevation or new left bundle branch block (LBBB) on a 12-lead ECG is the basis for diagnosis of STEMI.

These findings almost always indicate on-going myocardial damage caused by acute complete occlusion of the 'culprit' coronary artery (after initial plaque

fissuring). Left untreated there is likely to be further myocardial damage in the territory of the occluded artery, usually reflected in the development of Q waves on the ECG. During the acute phase of STEMI there is a substantial risk of ventricular tachycardia (VT) and ventricular fibrillation (VF) and sudden death (Figure 4.3).

## **Diagnosis of acute coronary syndromes**

### **History**

An accurate history is a crucial first step in establishing a diagnosis, but there are potential sources of confusion. Some patients (e.g. the elderly, diabetics, patients during the peri-operative period) may develop an ACS with little or no chest discomfort. The pain of angina or myocardial infarction is often mistaken for indigestion both by patients and healthcare professionals. Symptoms such as belching, nausea or vomiting are not helpful in distinguishing cardiac pain from indigestion; all may accompany angina and myocardial infarction.

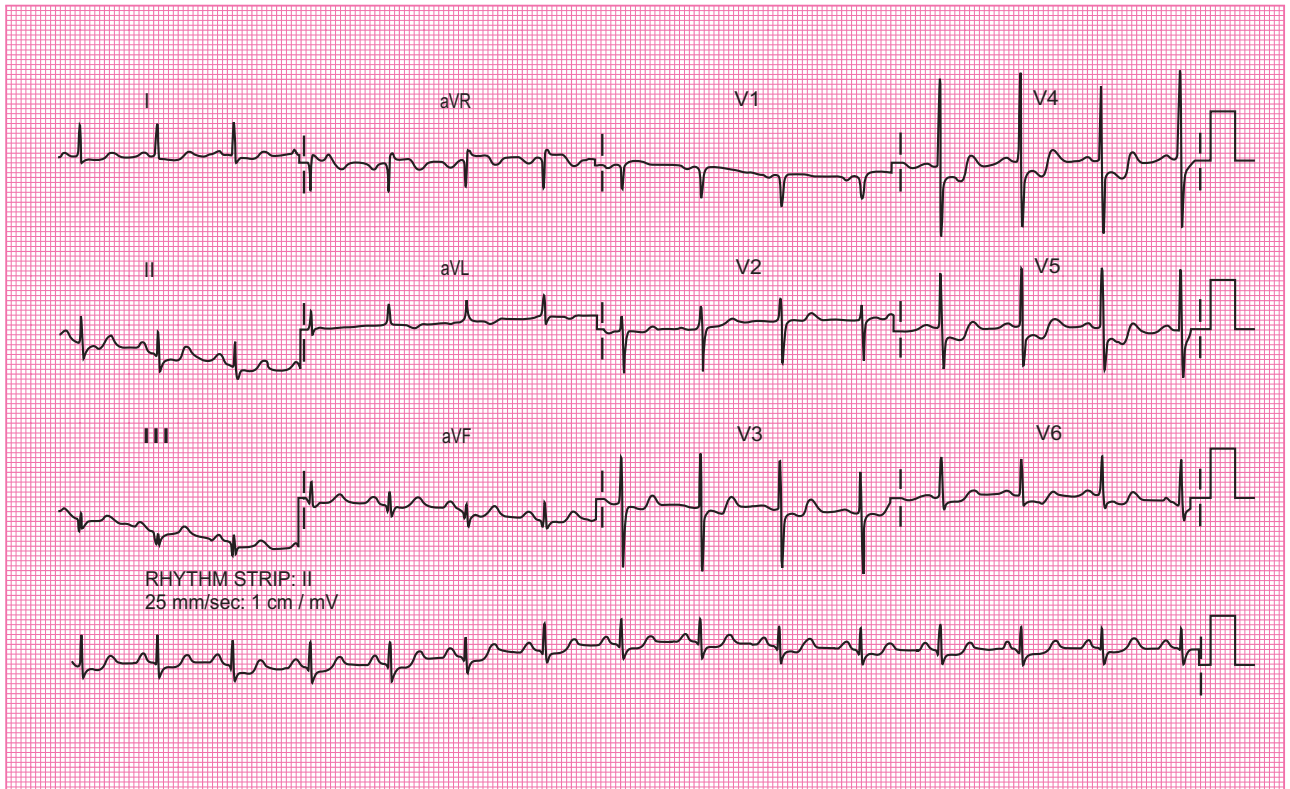
### **Clinical examination**

Clinical examination is of limited benefit in the diagnosis of ACS. Severe pain of any source may provoke some of the clinical signs, such as sweating, pallor and tachycardia, which commonly accompany ACS. History and examination are essential in order to recognise alternative, obvious causes for chest pain (e.g. localised severe chest wall tenderness), or identify other life-threatening diagnoses (e.g. aortic dissection, pulmonary embolism).

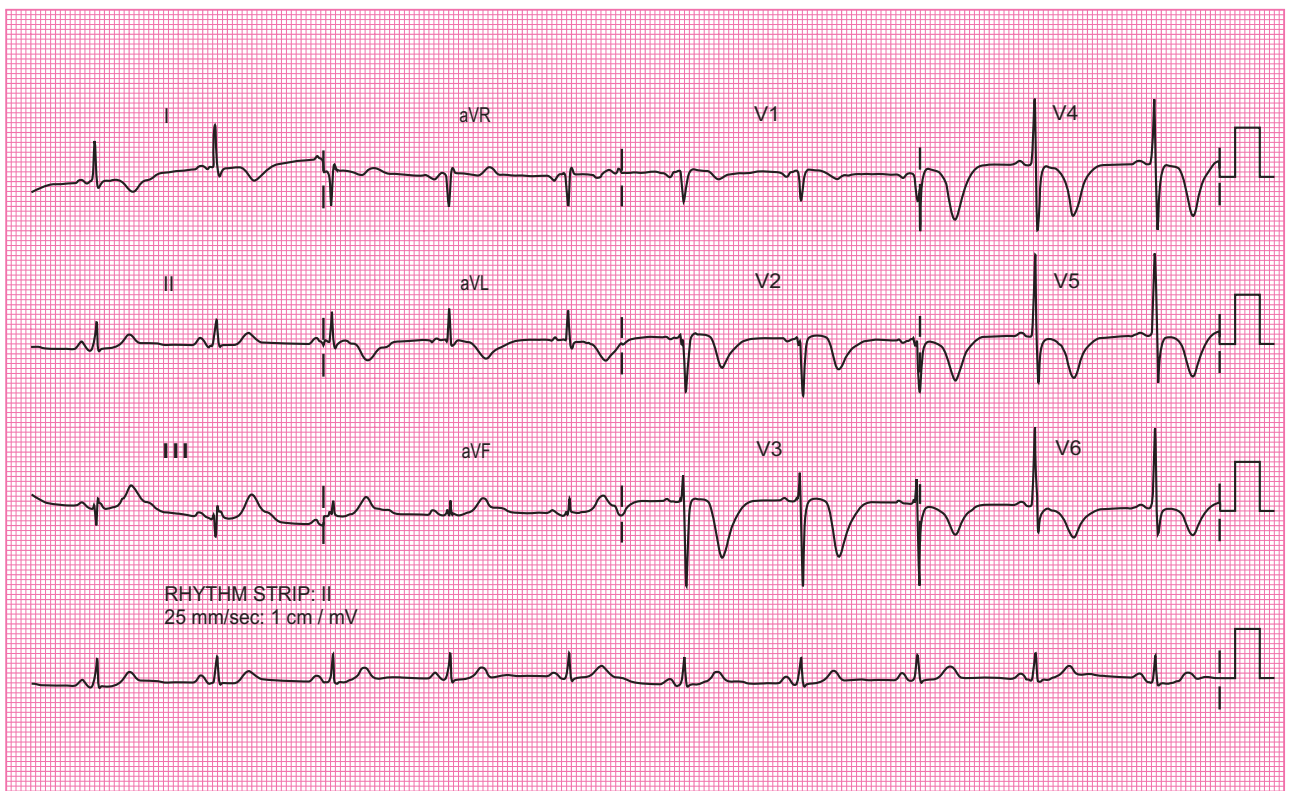
Examination may identify other important abnormalities (e.g. a cardiac murmur or signs of heart failure) that will influence choices of investigation and treatment. In patients with acute chest pain remember also to check for evidence of aortic dissection, especially if fibrinolytic therapy is intended. The presence of aortic dissection may be suggested by clinical signs such as loss of a pulse or asymmetry of the pulses in the upper limbs, acute aortic regurgitation, or signs of stroke from carotid artery involvement. Suspect aortic dissection in any patient whose acute chest pain is accompanied by marked hypotension but no obvious ECG evidence of AMI. However, in a patient with a good history and typical ECG evidence of STEMI do not delay reperfusion therapy without strong clinical evidence to justify prior investigation of possible aortic dissection.

Initial examination also serves as an important baseline so that changes, due either to progression of the underlying condition or in response to treatment, may be detected.

Also suspect extensive right ventricular (RV) infarction in patients with inferior or posterior STEMI who have elevated jugular venous pressure but no evidence of pulmonary oedema. Kussmaul's sign may be positive (JVP increases on inspiration). These patients are often hypotensive.



**Figure 4.1** 12-lead ECG showing acute ST-segment depression caused by myocardial ischaemia in a patient with a non-ST-segment ACS



**Figure 4.2** 12-lead ECG showing T wave inversion in a patient with NSTEMI



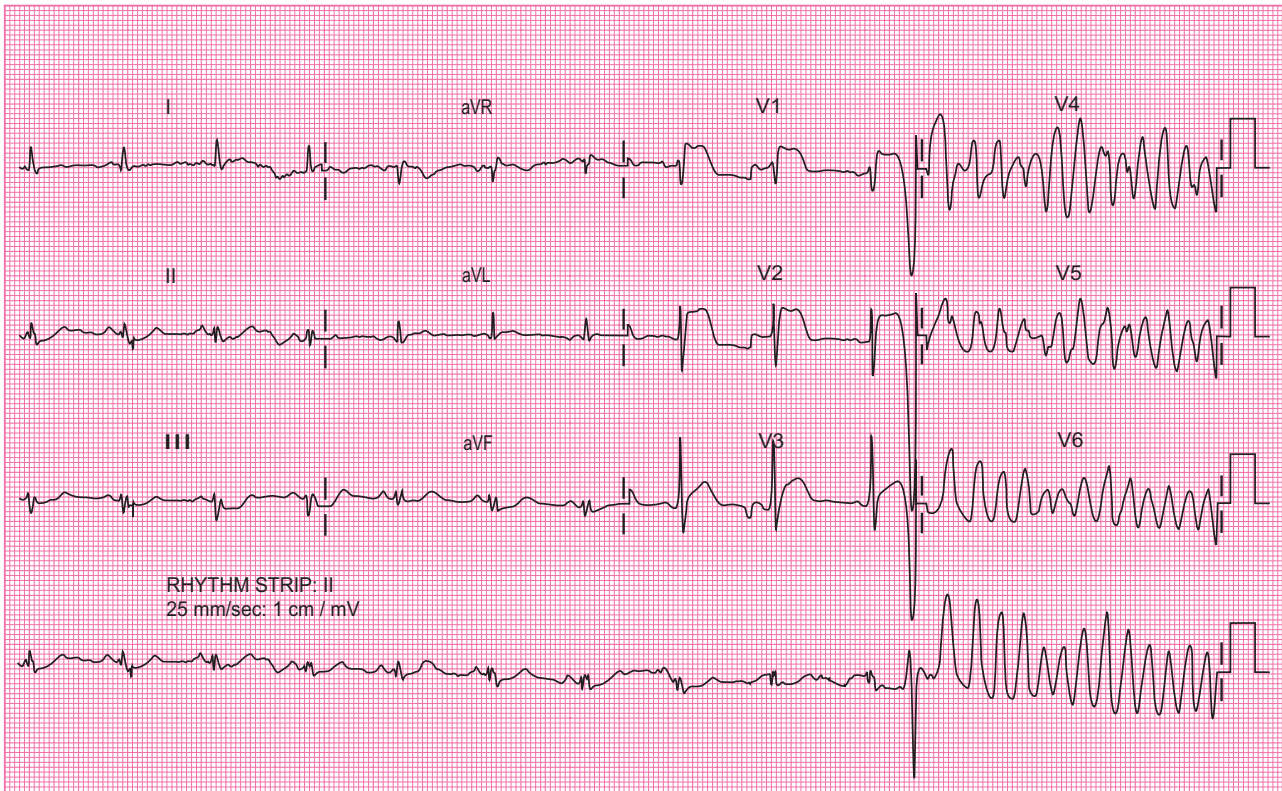


Figure 4.3 12-lead ECG showing onset of VF in a patient with an acute anteroseptal STEMI

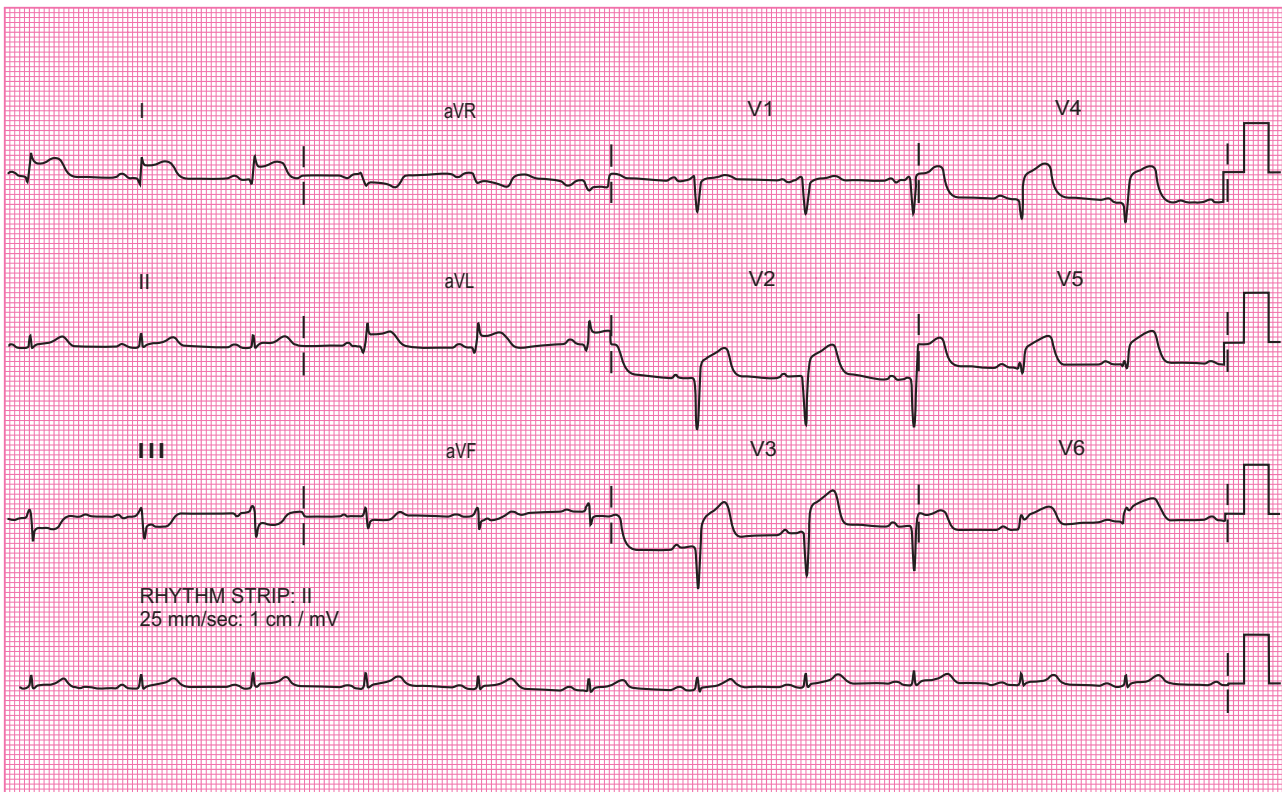


Figure 4.4 12-lead ECG showing an anterolateral STEMI

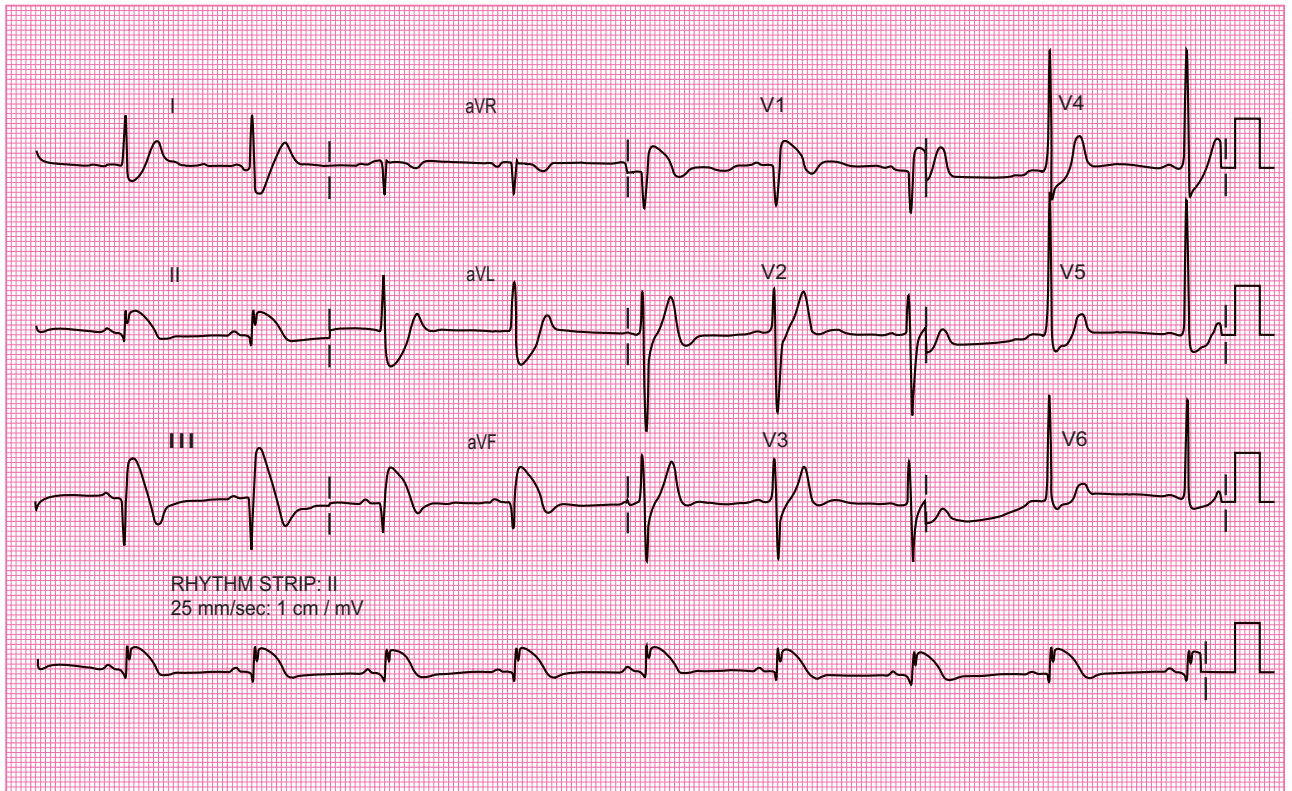


Figure 4.5 12-lead ECG showing an inferior STEMI

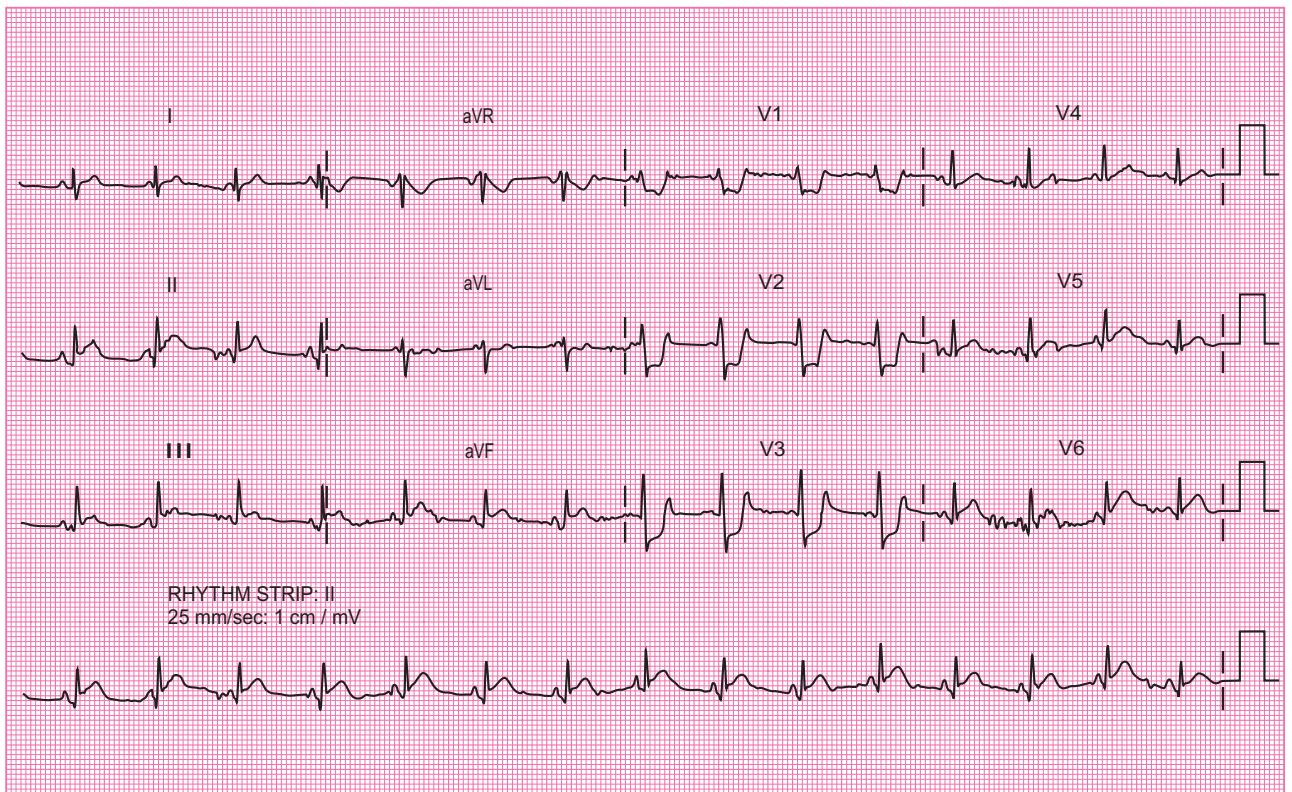


Figure 4.6 12-lead ECG showing a posterior STEMI

## Investigations

### The 12-lead ECG

Record a 12-lead ECG as soon as possible during the initial assessment and subsequently to assess progression of the ACS and the response to treatment. The presence of ECG abnormalities on the initial recording may support the clinical suspicion of ACS and indicate the appropriate treatment. A single normal 12-lead ECG does not exclude an ACS.

The ECG is a crucial component of risk assessment and planning of treatment. Acute ST segment elevation or new LBBB in a patient with a typical history of AMI is an indication for treatment to try to re-open an occluded coronary artery (reperfusion therapy), either by emergency percutaneous coronary intervention (PCI) or with fibrinolytic therapy. In contrast, ST segment depression suggests a low probability of benefit from fibrinolytic therapy, regardless of whether the ultimate diagnostic label is unstable angina or NSTEMI. In unstable angina, the presence of ST segment depression indicates a higher risk of further coronary events than if ST segment depression is absent. These higher-risk patients require immediate medical treatment (e.g. low-molecular-weight heparin, aspirin, clopidogrel, beta blockade, glycoprotein IIb/IIIa inhibitor), prompt investigation by coronary angiography, and often revascularisation by PCI, or coronary artery bypass surgery.

The ECG provides some information about the site and extent of myocardial damage in AMI particularly in STEMI. This is important since the site and extent of myocardial ischaemia or damage influences prognosis and, in some cases, the appropriate choice of treatment:

1. Anterior or anteroseptal infarction is seen usually in leads V1 - V4 and is almost always caused by a lesion in the left anterior descending (LAD) coronary artery. Extension to involve leads V5 - V6, I and aVL indicates an anterolateral infarct (Figure 4.4). An anterior MI has a worse prognosis and is more likely to cause substantial impairment of left ventricular function. Therefore these patients benefit more from immediate reperfusion therapy and early treatment with an angiotensin converting enzyme inhibitor (ACEI).
2. Inferior infarction is seen usually in leads II, III, and aVF (Figure 4.5), and is caused often by a lesion in the right coronary artery or, less commonly, the circumflex artery.
3. Lateral infarction is seen usually in leads V5 - V6 and/or leads I and aVL (sometimes aVL alone) and is caused often by a lesion in the circumflex artery or diagonal branch of the LAD artery.
4. Posterior myocardial infarction is usually recognised when there is a reciprocal change in the anterior chest leads (Figure 4.6). ST segment depression in

these leads reflects posterior ST elevation, and development of a dominant R wave reflects posterior Q wave development. This is also most commonly due to a right coronary artery lesion but may be caused by a circumflex artery lesion in those people in whom this artery provides the main blood supply to the posterior part of the left ventricle and septum. Suspicion of a posterior infarction can be confirmed by repeating the ECG with posterior leads. These leads (V8, V9 and V10) are placed in a horizontal line around the chest, continuing from V6 (mid-axillary line) and V7 (posterior axillary line). V9 is placed to the left of the spine, V8 half way between V7 and V9 and V10 to the right of the spine.

Right ventricular (RV) infarction may be present in up to one third of patients with inferior and posterior STEMI. Extensive RV infarction may be seen on a conventional 12-lead ECG when ST segment elevation in lead V1 accompanies an inferior or posterior STEMI; use of right-sided precordial leads, especially V4R can also be useful in detecting RV infarction. In this case right-sided precordial leads, particularly V4R, may reveal RV infarction. Two-dimensional echocardiography is also very useful. A diagnosis of extensive RV infarction is suggested by fluid-responsive hypotension and signs of high CVP (as jugular venous distension) without pulmonary congestion. In these patients nitrates should be avoided.

The ST segment depression and T wave inversion that may occur in NSTEMI are less clearly related to the site of myocardial damage than the changes in STEMI. Remember also that use of modified limb leads for ECG recording may alter the morphology of the 12-lead ECG and in particular the modified inferior leads may not show true electrical activity from the inferior wall of the left ventricle.

### Laboratory tests

The other important components of diagnosis and risk assessment are laboratory tests.

### Cardiac troponins (troponin T and troponin I)

Cardiac-specific troponins are components of the contractile structure of myocardial cells. Because concentrations of troponin in the blood of healthy individuals are undetectably low, and cardiac-specific troponins measured by current assays do not arise from extra-cardiac sources, the troponins are very sensitive and specific markers of cardiac injury. In the context of a typical clinical presentation of ACS, troponin release provides evidence of myocardial damage and therefore indicates myocardial infarction rather than unstable angina. In addition troponin measurement provides useful assessment of risk. The greater the troponin concentration, the greater is the risk of a further event. A combination of ST segment depression on the ECG and raised troponin identifies a particularly high-risk group for subsequent MI and sudden cardiac death.



The release of troponin does not in itself indicate a diagnosis of ACS. Troponin release aids diagnosis and is a marker of risk when the history indicates a high probability of AMI. Troponin may be released in other life-threatening conditions presenting with chest pain, such as pulmonary embolism and aortic dissection, and also in myocarditis, acute or chronic heart failure, sustained tachyarrhythmia, renal failure and acute sepsis. As with all clinical evidence, it is essential that troponin results are interpreted in the context of the clinical history.

### **Creatine kinase (CK), aspartate transaminase (AST) and lactate dehydrogenase (LDH)**

These enzymes are released from cardiac muscle when it is damaged; however, they are released also from skeletal muscle when it is damaged or during prolonged, vigorous exercise. To help clarify whether elevated CK concentrations in the blood originate from cardiac or skeletal muscle, the specific iso-enzyme of CK in cardiac muscle (CK-MB) can be measured. In many hospitals, measurement of CK-MB is not available routinely.

The amount of CK release from myocardium (e.g. when measured on sequential blood samples over three days) can serve as an approximate measure of the amount of myocardial damage.

### **Echocardiography**

This can be useful in assessing the severity of LV impairment resulting from any AMI. It is particularly important and urgent in confirming RV dilatation and impairment when extensive RV infarction is suspected, and in identifying some complications of AMI, including acquired ventricular septal defect and severe mitral regurgitation, both of which may require urgent surgical correction.

## **Risk assessment**

The choice of treatment is determined largely by the extent of myocardial damage and by the risk of early further coronary events. Accurate risk assessment in ACS enables early treatment to reduce risk and thereby prevent some instances of cardiac arrest and sudden death.

## **Immediate treatment**

### **General measures in all acute coronary syndromes**

Start with a rapid clinical assessment and record an ECG. Give immediate treatment to relieve symptoms, limit myocardial damage and reduce the risk of cardiac arrest. Immediate general treatment for ACS comprises:

- aspirin 300 mg, orally, crushed or chewed, as soon as possible;
- nitroglycerine, as sublingual glyceryl trinitrate (tablet or spray) unless patient is hypotensive or extensive

RV infarction is suspected;

- oxygen, to achieve an arterial blood oxygen saturation of 94 - 98% (or 88 - 92% in the presence of COPD);
- relief of pain is of paramount importance and intravenous morphine (or diamorphine) should be given, titrated to control symptoms but avoiding sedation and respiratory depression.

Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Give an anti-emetic with opiate analgesia or if the patient has nausea.

### **Treatment of STEMI (or AMI with new LBBB)**

For patients presenting with STEMI within 12 h of symptom onset, mechanical or pharmacological reperfusion must be achieved without delay. The aim is to restore the blood supply to myocardium that has not yet been damaged irreversibly. Clinical trials have confirmed the effectiveness of reperfusion therapy in reducing infarct size, complications, and mortality from MI. Reperfusion therapy is most effective when undertaken early after the onset of myocardial infarction and the benefit diminishes progressively with delay.

The risk/benefit ratio for reperfusion therapy favours reperfusion therapy for those patients who are at highest risk of immediate major myocardial damage and death.

Beyond 12 h from the onset of chest pain, the risks of fibrinolytic therapy probably outweigh any small residual benefit, but emergency percutaneous coronary intervention (PCI) should be considered in this situation if there is ongoing clinical or ECG evidence of ischaemia.

### **Coronary reperfusion therapy**

In STEMI, coronary reperfusion may be achieved in one of two ways:

- Percutaneous coronary intervention (PCI) may be used to re-open the occluded artery. This is referred to as primary PCI.
- Fibrinolytic therapy may be given in an attempt to dissolve the occluding thrombus that precipitated the MI.

### **Primary PCI**

Primary PCI (PPCI) is the preferred treatment for STEMI if it can be performed by an experienced team in a timely manner. Coronary angiography is used to identify the occluded coronary artery, following which a guidewire is passed through the occluding thrombus, enabling a deflated balloon to be positioned at the site of occlusion and inflated to re-open the artery. Aspiration devices may

be used to remove thrombus from the vessel and glycoprotein IIb/IIIa inhibitors may be injected intravenously or directly into the coronary artery. It is usual practice to insert a stent into the segment of previously occluded artery, to reduce the risk of re-occlusion at this point.

Primary PCI is the most reliable method of re-opening of the culprit artery in the majority of patients. Coronary artery patency can be confirmed, secured and maintained. There is a lower risk of major, particularly intracerebral, bleeding than with fibrinolytic therapy.

For PPCI to provide reliable, timely reperfusion a fully-equipped catheter laboratory staffed by an experienced team must be available 24-h a day. A fail-safe pathway of communication and care must be implemented in each region in order that patients in whom STEMI is diagnosed can access the service, ideally by direct transfer to this facility. Primary PCI can then be offered to patients for whom a 'call-to-balloon' time of 120 min can be achieved (National Infarct Angioplasty Project [NIAP]). In patients who present within 2 hours of onset of chest pain the time from first medical contact to reperfusion should be less than 90 min. Longer delays are associated with higher mortality.

Where PPCI is not available immediately, the need to achieve reperfusion as early as possible remains a high priority and for those patients initial treatment by fibrinolytic therapy may offer the best chance of achieving early reperfusion.

**Platelet inhibition and anticoagulant therapy in PPCI**

In addition to aspirin, give all patients being transferred for PPCI:

- clopidogrel as a 600 mg loading dose; or
- prasugrel as a 60 mg loading dose (not if >75 years, < 60 kg, history of bleeding or stroke).

Anticoagulation with unfractionated or low molecular weight heparin is given in the catheter laboratory often with a glycoprotein IIb/IIIa inhibitor. Bivalirudin, a direct thrombin inhibitor may be chosen as an alternative.

**Typical indications for immediate reperfusion therapy for AMI**

**Presentation within 12 hours of onset of chest pain suggestive of AMI and:**

- ST segment elevation > 0.2 mV in 2 adjacent chest leads, or > 0.1 mV in 2 or more 'adjacent' limb leads; or
- Dominant R waves and ST depression in V1 - V3 (posterior infarction); or
- New-onset (or presumed new-onset) LBBB.

**Table 4.1 Typical indications for immediate reperfusion therapy for AMI**

**Typical contraindications to fibrinolytic therapy**

**Absolute**

- Previous haemorrhagic stroke
- Ischaemic stroke during the previous 6 months
- Central nervous system damage or neoplasm
- Recent (within 3 weeks) major surgery, head injury or other major trauma
- Active internal bleeding (menses excluded) or gastro-intestinal bleeding within the past month
- Known or suspected aortic dissection
- Known bleeding disorder

**Relative**

- Refractory hypertension (systolic blood pressure > 180 mmHg)
- Transient ischaemic attack in preceding 6 months
- Oral anticoagulant treatment
- Pregnancy or less than 1 week post-partum
- Non-compressible vascular puncture
- Active peptic ulcer disease
- Advanced liver disease
- Infective endocarditis
- Previous allergic reaction to the fibrinolytic drug to be used

**Table 4.2 Typical contraindications to fibrinolytic therapy**

**Fibrinolytic therapy**

Fibrinolytic therapy has been shown in large-scale clinical trials to provide substantial reduction in mortality from AMI when given during the first few hours after the onset of chest pain. One of the major advantages of fibrinolytic therapy is that it does not require a cardiac catheter laboratory or an operator skilled in angioplasty. Early reperfusion may be achieved by pre-hospital fibrinolytic therapy with resulting clinical benefit, particularly when transport times to hospital are very long. Early treatment may also be achieved by minimising door-to-needle time (time from arrival at hospital to administration of fibrinolytic therapy).



Fibrinolytic therapy carries a risk of bleeding, including cerebral haemorrhage, and not all patients can be given this treatment safely. Table 4.1 lists typical indications for reperfusion therapy and the typical contraindications to fibrinolytics are shown in Table 4.2. Most of these contraindications are relative; the experienced clinician will decide whether the benefit from fibrinolytic therapy outweighs the risk to the individual patient or whether emergency angiography with a view to primary PCI would be more appropriate.

Figure 4.7 describes the options for reperfusion therapy for STEMI in the form of an algorithm.

### Platelet inhibition and anticoagulant therapy with fibrinolytic therapy

Give all patients receiving a fibrinolytic agent for STEMI:

- aspirin 300 mg, and
- clopidogrel as a 600 mg loading dose, and
- antithrombin therapy: low molecular weight heparin (IV bolus then SC) or unfractionated heparin (full dose) or fondaparinux.

### Rescue angioplasty

In 20 - 30% of patients receiving a fibrinolytic for STEMI, reperfusion is not achieved. Observe patients closely with cardiac monitoring during and after administration of a fibrinolytic. Record a 12-lead ECG at 60 - 90 min after giving fibrinolytic therapy. Failure of ST segment elevation to resolve by more than 50% compared with the pre-treatment ECG suggests that fibrinolytic therapy has failed to re-open the culprit artery. Symptoms are a less accurate guide to reperfusion because most patients will have received opiate analgesia. Even after initially successful thrombolysis there is a significant risk of re-occlusion and patients should be admitted to a coronary care unit with continuous ECG monitoring.

In cases of failed reperfusion or re-occlusion/re-infarction transfer the patient immediately to a cardiac catheter laboratory for mechanical reperfusion (PCI). In failed thrombolysis this is referred to as rescue PCI and has been shown to improve event-free survival and reduce heart failure when compared to conservative therapy or repeat fibrinolytic therapy. Again rescue PCI must be performed without any time-delay in order to be effective.

The role of 'facilitated PCI' in which initial fibrinolytic therapy is followed by immediate coronary angiography and PCI remains a subject of ongoing debate. So far there is insufficient evidence in support of this strategy but trials are ongoing.

## Treatment of unstable angina and NSTEMI

The immediate treatment objectives in these syndromes are:

- To prevent new thrombus formation, which may occlude an artery and lead to, or extend, myocardial damage.
- To reduce myocardial oxygen demand, providing myocardial cells with a better chance of survival in the presence of a limited supply of oxygen and glucose.

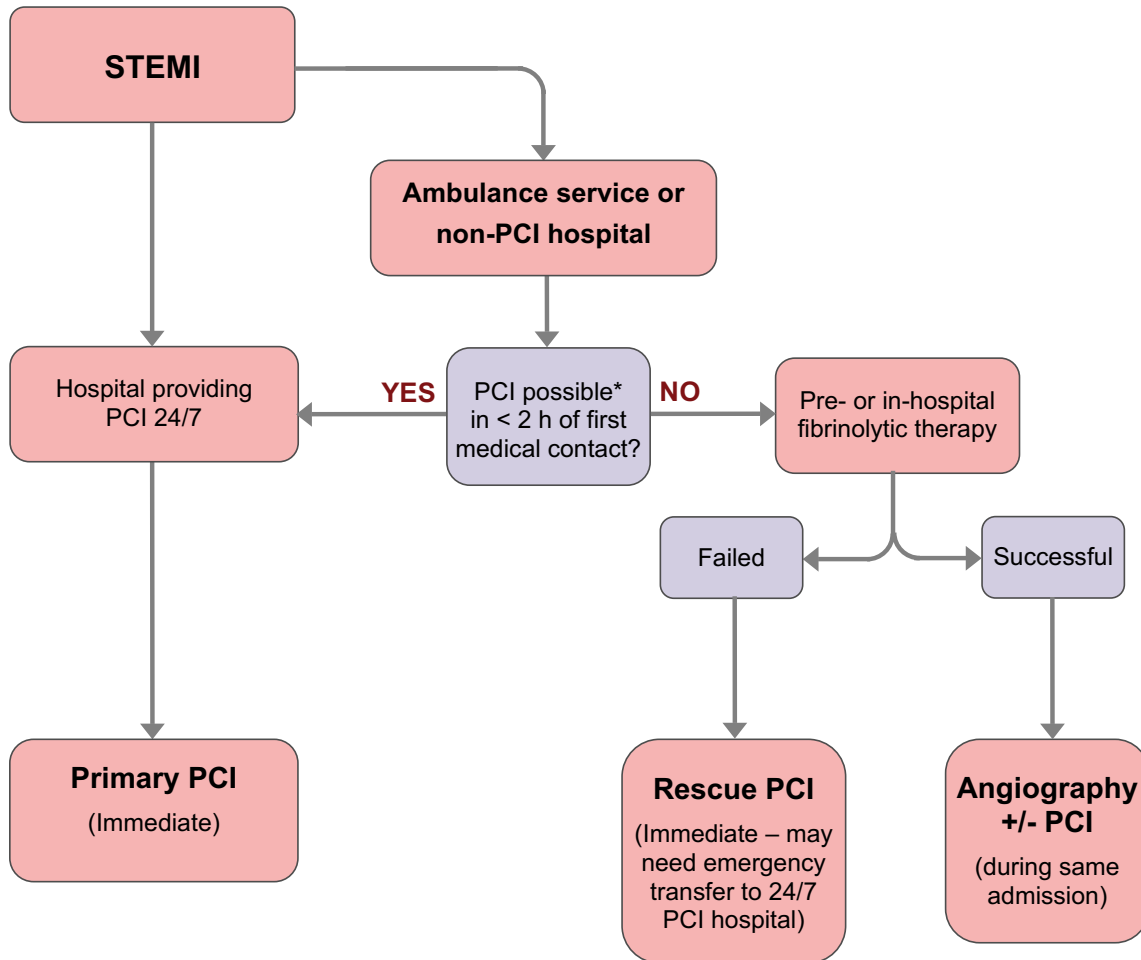
### Prevention of further thrombus formation

- Give subcutaneous low molecular weight heparin in therapeutic doses (weight-related, 12-hourly, dose reduction in renal impairment); or fondaparinux (once daily, contraindicated in severe renal impairment).
- Give aspirin 75 mg per day after the initial 300 mg loading dose.
- Start clopidogrel with a loading dose of at least 300 mg (consider 600 mg for rapid loading before urgent angiography), followed by 75 mg daily.
- In diabetic patients undergoing urgent PCI consider prasugrel (60 mg loading dose and then 10 mg daily) as a more effective alternative to clopidogrel.
- In high-risk patients, if early PCI is planned, consider starting a glycoprotein IIb/IIIa inhibitor (tirofiban or eptifibatide). The balance of ischaemic risk versus bleeding risk must be considered carefully.

### Reduction in oxygen demand

- Start beta-adrenoceptor blockade (unless contraindicated).
- Consider diltiazem if beta blockade contra-indicated.
- Avoid dihydropyridine calcium channel blockers (e.g. nifedipine).
- Consider intravenous or buccal nitrate if angina persists or recurs after sublingual nitrate.
- Consider early introduction of an ACEI, especially if there is left ventricular systolic impairment or heart failure.
- Treat complications such as heart failure or tachyarrhythmia promptly and effectively.

Access to reperfusion therapy for STEMI



\* In patients presenting < 2 h after onset of pain, time from first medical contact to PCI should be less than 90 min. If not achievable consider immediate fibrinolytic therapy.

Figure 4.7 Access to reperfusion therapy for STEMI

## Subsequent management of patients with acute coronary syndromes

### Suspected unstable angina – low risk patients

Patients with suspected unstable angina without a definite history of preceding angina of effort or myocardial infarction and without high-risk features at presentation (ECG and troponin levels normal after 6 - 8 h) should undergo early further risk assessment either by exercise testing or non-invasive imaging.

### Suspected angina – high risk unstable angina and NSTEMI

Patients with unstable angina and high-risk features (e.g. resting ST segment depression, high-risk features on exercise test or non-invasive imaging) should be considered for early investigation by invasive coronary angiography

Patients with NSTEMI should be regarded as a high-risk group, requiring early assessment by coronary angiography during the same hospital admission in the majority of cases, ideally within 72 h.

Many patients in both these groups will benefit from revascularisation by PCI. A few may require coronary artery bypass grafts (CABG).

Formal risk-scoring systems such as GRACE (Global Registry of Acute Coronary Events) should be used to guide clinical management. Those patients at the highest risk derive the greatest benefit from early intervention in terms of reducing further major cardiac events.

### STEMI

If fibrinolytic therapy has been used, many patients will be left with a severe stenosis or unstable plaque in the culprit coronary artery. PCI can stabilise this situation and reduce the risk of re-occlusion of the artery and resulting further myocardial infarction, cardiac arrest or sudden death. Coronary angiography and, if indicated, PCI should be undertaken early during the same hospital admission.

In patients with completed STEMI who have not been treated with reperfusion therapy (e.g. because of late presentation) it is usually recommended that coronary angiography is undertaken during the same hospital admission. Although the benefits of re-opening an occluded culprit artery late after STEMI are uncertain, there is often disease in other coronary vessels that can give rise to further major coronary events over subsequent months. Defining the severity and anatomy of such disease can help to identify those at highest risk, in whom early intervention may reduce that risk.

### Ventricular arrhythmia complicating acute coronary syndromes

When ventricular arrhythmia complicates an acute

coronary syndrome, interpret its significance in the context of the precise clinical setting and the time of onset of the arrhythmia. When VF/VT cardiac arrest occurs within the first 24 - 48 h after STEMI, and subsequent recovery is uncomplicated, the risk of another ventricular arrhythmia is relatively low and is determined by other factors, especially the severity of left ventricular impairment.

If VF/VT cardiac arrest occurs in the context of non-ST segment elevation ACS, there may be a continuing risk of further ventricular arrhythmia. If the arrhythmia has been caused by severe myocardial ischaemia, very urgent revascularisation is needed to prevent recurrence of the ischaemia and reduce the risk of resulting arrhythmia. If this is not achievable or if the arrhythmia has occurred without evidence of severe ischaemia, the patient will be at risk of recurrent ventricular arrhythmia and should be referred to a heart rhythm specialist with a view to insertion of an implantable cardioverter-defibrillator (ICD) **before discharge from hospital.**

Patients who have a VF/VT cardiac arrest as a late complication after myocardial infarction, or outside the context of an ACS, will be at risk of recurrent cardiac arrest and should be seen urgently by a heart rhythm specialist with a view to ICD implantation **before discharge from hospital.**

## Other complications of ACS

### Heart failure

Patients with heart failure complicating AMI or other ACS are at increased risk of deterioration, cardiac arrest and death: prompt, effective treatment of the heart failure is required to reduce risk. Give a loop diuretic (e.g. furosemide) and/or glyceryl trinitrate (sublingual and/or intravenous) for immediate symptomatic treatment. Give regular loop diuretic to maintain symptom control but review the need for this and the dose at least daily for the first few days. Ensure that angiotensin converting enzyme inhibitor (ACEI) treatment has been started and increase the dose as tolerated, until the target dose is achieved. In patients intolerant of ACEI, consider an angiotensin receptor blocker. Maintain beta blockade unless contraindicated or not tolerated. If more than mild LV systolic impairment is confirmed (ejection fraction 40% or less) consider addition of an aldosterone antagonist (e.g. eplerenone or spironolactone).

### Cardiogenic shock

This consists of severe hypotension with poor peripheral perfusion, often accompanied by pulmonary oedema, drowsiness or mental confusion due to poor cerebral under-perfusion and oliguria caused by poor renal perfusion. The mortality is very high, but can be reduced by early revascularisation by PCI.

Some patients may improve with inotropic therapy (e.g. dobutamine) but this requires initiation and supervision by those experienced in its use. Other treatments such as intra-aortic balloon pumping may be of benefit in selected patients, but require expert supervision.



When cardiogenic shock develops in a patient after STEMI, seek early expert assessment with a view to possible emergency PCI as this may be life-saving for some patients in this setting.

### Other cardiac arrhythmia

The treatment of other cardiac arrhythmia will be covered in more detail in Chapter 11.

When atrial fibrillation occurs in the context of an ACS it usually indicates some degree of left ventricular failure: treatment should address that as well as focusing on control of heart rate or rhythm.

When AV block occurs in the context of acute inferior wall myocardial infarction there is often excess vagal activity. QRS complexes are often narrow and heart rates may not be excessively slow. Treat symptomatic bradycardia in this setting with atropine and if necessary theophylline, and consider temporary cardiac pacing only if bradycardia and hypotension persist after atropine therapy. Complete AV block in this setting is usually transient and permanent cardiac pacing is often not necessary.

When AV block occurs in the context of acute anterior myocardial infarction this usually implies extensive myocardial injury and a poor prognosis. The QRS complexes are usually broad and the heart rate is usually slow and resistant to atropine. Temporary cardiac pacing is usually needed and should not be delayed. Many of those who survive this situation will require a permanent pacemaker.

### Cardiac rehabilitation

In all patients after an ACS, an effective programme of cardiac rehabilitation can speed the return to normal activity and encourage measures that will reduce future risk (see below). There is evidence that effective cardiac rehabilitation reduces the need for readmission to hospital. Cardiac rehabilitation is a continuous process, beginning in the cardiac care unit and progressing through to a community-based approach to lifestyle modification and secondary prevention.

### Secondary prevention

In patients with established coronary disease, general measures to reduce cardiovascular risk ('secondary prevention') can reduce the likelihood of future coronary events (including sudden cardiac death) and stroke.

### Anti-thrombotic therapy

Continued platelet inhibition is appropriate in all patients. They should receive low-dose aspirin (75 mg daily) for life, unless they have or develop a contra-indication. Give clopidogrel 75 mg daily (or prasugrel 10 mg daily) to patients with high-risk ACS and all those undergoing PCI;

current guidelines recommend treatment for a minimum of one year. Clopidogrel alone may be used in patients who are intolerant of aspirin. In patients who develop atrial fibrillation as a complication of ischaemic heart disease, there is an additional risk of thromboembolism from the left atrium. Warfarin is more effective than aspirin or clopidogrel in preventing intra-cardiac thrombus formation, and should be considered in addition to, or instead of platelet inhibition.

### Preservation of left ventricular function

Prognosis after AMI is determined partly by the severity of left ventricular impairment that results. Treatment after AMI with an ACEI can reduce the re-modelling that contributes to left ventricular dilatation and impairment, and where there is left ventricular systolic impairment, the use of ACEI therapy in adequate dose can reduce the risk and severity of subsequent heart failure, and the risk of future AMI and death. Echocardiographic examination of left ventricular function is appropriate during the first few days after an ACS to assess risk and identify those patients likely to benefit most from this treatment. The majority of patients should be considered for ACEI treatment during the first few days after AMI.

### Beta-adrenoceptor blockade

Treatment with a beta blocker, started early after AMI and continued, was shown many years ago to reduce mortality, so beta blockade that has been started in the acute phase of treatment is usually continued, often indefinitely. There is evidence that prior treatment with beta blockade may reduce the size of subsequent myocardial infarction, so in patients with coronary disease this treatment may have a 'cardioprotective' effect, and it may help to protect against other complications such as arrhythmia. In patients with heart failure and left ventricular systolic impairment there is evidence of symptomatic and prognostic benefit from some beta blocking drugs (e.g. bisoprolol, carvedilol).

### Reduction of cholesterol

Further reduction in risk can be achieved by effective suppression of cholesterol concentration in the blood; specifically, suppression of LDL cholesterol. Statins reduce the risk of most future coronary events by at least 30%. A low-fat, high-fibre diet and regular exercise will complement cholesterol suppression by drugs.

### Avoidance of smoking

At least as important in reducing risk, is the removal of other avoidable risk factors such as smoking. Information, encouragement and support for patients to help them to stop smoking should begin at an early stage after presentation with an ACS.

## Anti-hypertensive treatment

Effective control of raised blood pressure, using drugs as well as non-pharmacological methods, reduces the risk of stroke and of heart failure and contributes to some reduction in the risk of future coronary events.

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### Key learning points

- The acute coronary syndromes comprise unstable angina, non-ST-segment-elevation myocardial infarction, and ST-segment-elevation myocardial infarction.
- Give aspirin, nitroglycerine and morphine to patients presenting with acute coronary syndromes. Give oxygen to achieve SPO<sub>2</sub> of 94-98% (or 88-92% in the presence of COPD)
- Rapid initial assessment using the history, examination and 12-lead ECG will help to determine the diagnosis and immediate risk.
- Consider immediate reperfusion therapy in those patients with acute myocardial infarction accompanied by ST segment elevation or new LBBB.
- Effective assessment and immediate treatment of patients with acute coronary syndromes will reduce the risk of cardiac arrest and death.

## Further reading

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