

Department of Social Protection

# Ischaemic Heart Disease

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# 1. Overview and Definition of Ischaemic Heart Disease

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## 1.1 Overview

Ischaemic Heart Disease is one of the groups of conditions which are known as cardiovascular disease - the others being cerebrovascular disease, hypertension, heart failure and rheumatic disease (World Health Organisation, 2009). Ischaemic Heart Disease is defined by a joint International Society and Federation of Cardiology and World Health Organisation task force as 'myocardial impairment due to an imbalance between coronary blood flow and myocardial requirements caused by changes in the coronary circulation' (Warrell et al, 2004).

In Ireland, as throughout the UK, Europe, the US and most of the developed world, cardiovascular disease is the leading cause of death, with around 30% of annual worldwide deaths resulting from these conditions. Of these, approximately 44% are directly due to ischaemic heart disease. Whilst cardiovascular diseases have traditionally been considered as diseases of the 'developed' world, the prevalence of these conditions is decreasing in developed countries due to improvements in prevention, diagnosis and treatment, and changes in lifestyle such as the reduction in smoking rates. In contrast, due to increasing rates of urbanisation and adoption of 'western' culture, the prevalence of cardiovascular disease in developing countries is rapidly increasing (World Health Organisation, 2009). It is expected that over 80% of the future increase in world wide mortality rates for heart disease will be in third world and developing countries, with prevalence rates in developed countries falling or remaining static.

## 1.2 Definition of Ischaemic Heart Disease

Ischaemic heart disease (I.H.D.) is a condition which results from reduced blood supply to the heart muscle. This usually involves impairment of blood flow through the coronary arteries, most commonly caused by atherosclerotic narrowing, but occasionally due to arterial spasm (Warrell et al, 2004).

The reduction in blood supply to the heart muscle can result in a number of clinical presentations:

- Chronic stable angina (angina pectoris)
- Acute coronary syndromes such as myocardial infarction and unstable angina
- Chronic ischaemic heart disease such as silent myocardial ischaemia.
- Sudden cardiac death

## 1.3 Chronic Stable Angina

The condition usually described as angina is more recently fully named as chronic stable angina, but is also known as angina pectoris; effort angina; angina of effort.

### 1.3.1 Definition of Chronic Stable Angina

Chronic stable angina was first identified by Heberden in 1768 who described the condition as resulting in a pain that had a “sense of strangling and anxiety”, further stating that individuals suffering from angina “are seized while they are walking (more especially if it be uphill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it were to extinguish life, if it were to increase or continue; but the moment they stand still, this uneasiness vanishes....The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni, and often more inclined to the left than to the right side. It likewise very frequently extends from the breast to the middle of the left arm” (Heberden, 1768 as cited in Warrell et al, 2004).

Many individuals describe angina pain as a ‘tight, crushing ache’ rather than a pain specifically. The pain may be central or sometimes left sided, and may radiate to the left arm, the front of the neck, lower jaw or teeth. The pain is most commonly caused by coronary artery insufficiency due to atherosclerotic disease, but can also be more rarely caused by conditions unrelated to atherosclerosis including valvular heart disease and uncontrolled hypertension.

Stable angina can be distinguished from other ischaemic heart diseases by factors which provoke or relieve the symptoms. Symptoms often occur on exercise, especially in cold or windy weather, or after a meal, or when an affected individual experiences emotional distress. It should be noted that atypical symptoms can occur, particularly in women, the elderly and diabetics.

Chronic stable angina is relieved by rest. If angina symptoms do not subside on rest, an alternative diagnosis should be sought.

## 1.4 Definition of Acute Coronary Syndromes

At any point during the development of atherosclerotic conditions, plaque may detach or tear from the vessel wall prompting platelet aggregation and thrombus formation. If the artery is occluded, the blood flow to the myocardium is compromised and necrosis occurs. This results in changes which can be detected on electrocardiogram and is known as an **acute-ST elevation myocardial infarction (STEMI)**. If the artery is only partially occluded, or only for a short period of time then the blood flow is either not severely or intermittently compromised. This results in some damage to the myocardium, but no significant changes on electrocardiogram and is known as a **non-ST elevation myocardial infarction (NSTEMI)**. The presence of necrosis in the myocardial tissue can be detected by a rise in a cardiac specific serum biomarker such as troponin. When myocardial ischaemia is present, but necrosis is absent (evidenced by a normal troponin level), this is known as **unstable angina** (National Clinical Guideline Centre for Acute and Chronic Conditions, 2009).

It should be noted that there are new diagnostic definitions for myocardial infarctions which were published in 2007, and have been accepted as universal (National Clinical Guideline Centre for Acute and Chronic Conditions, 2009). These are described at an overview level below, but further detail can be found in the following two documents:

Thygesen K, Alpert JS, White HD behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction (2007) 'Universal definition of myocardial infarction' *European Heart Journal* 2007 28(20):2525-2538; doi:10.1093/eurheartj/ehm355 Full text can be accessed at [http://eurheartj.oxfordjournals.org/cgi/content/full/28/20/2525?ikey=c930985104c248160053959392bc6a2c34b6b8d2&keytype2=tf\\_ipsecsha](http://eurheartj.oxfordjournals.org/cgi/content/full/28/20/2525?ikey=c930985104c248160053959392bc6a2c34b6b8d2&keytype2=tf_ipsecsha) (link accessed November 2009)

Atar, D. (2008) 'New definition of myocardial infarction' in *British Medical Journal* ;337:a3078 doi: 10.1136/bmj.a3078 Full text can be accessed at <http://www.abbott.no/Admin/Public/Download.aspx?file=Files%2fFiler%2fAbbottForum2009%2fKlin+kjemi+lmmu+kjemi%2fTrop I Atar BMJ.pdf> (link accessed November 2009)

#### 1.4.1 Definition of Myocardial Infarction

Despite the introduction of new universal definitions as to what constitutes a myocardial infarction, and the factors which differentiate unstable angina from a MI, there is still a degree of ambiguity as to the specific diagnostic criteria which are applied to each acute cardiac syndrome (SIGN, 2007).

The new European Society of Cardiology and American College of Cardiology definition of myocardial infarction (Thygesen et al, 2007) states that the term can be applied when there is evidence of myocardial necrosis consistent with myocardial ischaemia, listing a number of criteria which if met, means the formal diagnosis of myocardial infarction can be used. These criteria include:

- Detection of a rise and/or fall of cardiac biomarkers (usually troponin) to assess the degree of necrotic damage to the myocardium, together with evidence of one or more of the following symptoms:
  - Symptoms of ischaemia
  - ECG changes indicating new ischaemia (e.g. left bundle branch block)
  - Development of pathological Q waves on an ECG
  - Imaging results indicating loss of myocardial tissue or abnormal wall motion.
- Sudden cardiac death involving cardiac arrest often with preceding symptoms of myocardial ischaemia and possibly accompanied with fresh evidence of ST elevation and evidence of fresh thrombus at coronary angiography or at autopsy

- Specific criteria for diagnosis of myocardial infarction in percutaneous cardiac infusion or coronary artery bypass grafting patients would be indicated where patients with a normal baseline troponin levels elevation of cardiac biomarkers above the 99<sup>th</sup> percentile Upper Reference Limit are indicative of periprocedural myocardial necrosis
- The above criteria also apply to patients undergoing Coronary Artery Bypass Graft
- Pathological findings of acute myocardial infarction.

Other diagnostic classifications also use serum troponin levels to indicate a threshold for diagnosis of myocardial infarction, but the levels used for each syndrome may differ (SIGN, 2007).

#### 1.4.2 Clinical Classification of Myocardial Infarction

There is a recent universal classification of the different types of myocardial infarction (Thygesen et al, 2007) which can occur. These are:

- Type 1—Spontaneous myocardial infarction related to ischaemia caused by a primary coronary event, such as plaque fissuring or rupture
- Type 2—Myocardial infarction secondary to ischaemia resulting from an imbalance between oxygen demand and supply, such as coronary spasm
- Type 3—Sudden death from cardiac disease with symptoms of myocardial ischaemia, accompanied by new ST elevation or left bundle branch block, or verified coronary thrombus by angiography. In this type of myocardial infarction death occurs before blood samples can be obtained
- Type 4—Myocardial infarction associated with primary percutaneous coronary intervention
- Type 5—Myocardial infarction associated with coronary artery bypass graft

### 1.5 Diagnostic Classification of Ischaemic Heart Disease - International Classification of Diseases; 10<sup>th</sup> Edition (ICD-10) Classification

The International Classification of Diseases, 10<sup>th</sup> Edition (World Health Organisation 2007) contains the following classifications under the heading of Ischaemic Heart Disease:

- **120 - Angina pectoris** (including unstable angina)
- **121 - Acute myocardial infarction**
- **122: Subsequent myocardial infarction**



- **123: Certain current complications following acute myocardial infarction**
- **124: Other acute ischaemic heart diseases** including Coronary thrombosis not resulting in myocardial infarction, Dressler's syndrome, Other forms of acute ischaemic heart disease
- **125: Chronic ischaemic heart disease** including Atherosclerotic cardiovascular and heart disease, Aneurysm, Ischaemic Cardiomyopathy, Silent myocardial ischaemia and Other forms of chronic ischaemic heart disease

## 1.6 Prevention of Cardiovascular Disease

There has been an increasing recognition in recent guidelines that rather than concentrating on the prevention of ischaemic heart disease (IHD) specifically, prevention measures should be aimed at the spectrum of cardiovascular disease (CVD) to emphasise the importance of stroke prevention as well as coronary artery disease and ischaemic conditions.

Prevention measures are focused towards two groups of individuals:

- *Primary prevention* involves intervention in individuals without clinical evidence of CVD.
- *Secondary prevention* involves patients with known CVD who should all be considered for risk reduction measures – e.g. with aspirin or statin therapy).

Within each group risk factors can be divided in to two groups – modifiable (for which prevention measures apply), and non-modifiable factors (such as age or gender).

### 1.6.1 Primary Prevention: Assessment of Risk for Cardiovascular Disease

There are a number of groups of people who can be assumed to be automatically at high risk, as their 10 year risk of developing cardiovascular disease is at least 20% (NHS Institute for Innovation and Improvement, 2009). These are individuals with one or more of the following factors:

- People 75 years of age or older
- Established cardiovascular disease
- Familial hypercholesterolaemia
- Type 2 diabetes mellitus and older than 40 years of age with any additional risk factor (e.g. overweight, smoker, hypertensive, history of CVD etc).

For individuals who do not fall in to the above groups, there are a number of calculators and scoring systems available which assess relative risk. The use of which specific scoring or assessment system may depend on local policy, but also

on the background factors of the individual being assessed. Some scoring systems omit criteria which may be influential in affecting an individual's risk of developing CVD. For example, the Framingham risk equations which are included as electronic decision support in many practice systems omit social deprivation as a factor (SIGN, 2007), despite the fact that the mortality from coronary heart disease is high in this group.

For individuals without diabetes mellitus, the scoring system available from the Joint British Cardiac Societies may be appropriate. This can be located at [www.bhsoc.org](http://www.bhsoc.org).

For individuals who have Type 2 diabetes mellitus and are older than 40 years of age the United Kingdom Prospective Diabetes Study (UKPDS) risk engine may be appropriate. This can be located at [www.dtu.ox.ac.uk](http://www.dtu.ox.ac.uk). This calculator is not considered appropriate for individuals with diabetes under the age of 40 years, where clinical judgement should be used (NHS Institute for Innovation and Improvement, 2009)

[Links accessed November 2009]

## 1.6.2 Primary Prevention: Lifestyle Measures

Lifestyle measures for primary prevention of cardiovascular disease aim to optimally manage risk factors which can be modified.

These include:

### Diet

- Consumption of a 'Mediterranean' style diet
- Consumption of two to four portions of oily fish per week to equate to 7g of omega 3 fatty acids per week
- Supplements of antioxidants, beta-carotene or folic acid are not recommended
- Healthy eating advice tailored to the individual and their family should be provided

### Alcohol Consumption

- Consumption of alcohol should be kept within safe limits and binge drinking avoided

### Physical Activity

- Regular physical activity to increase exercise capacity should be undertaken.
- Individuals should aim to take 20-30 minutes exercise to the point of slight breathlessness daily

- A stepped programme should be devised with the individual in order to allow the individual to gradually reach this target

### Smoking Cessation

- All individuals should be advised to cease to smoke and be given appropriate help and advice to support the individual in this aim (e.g. support groups, pharmacotherapy)

### Weight Management

- All individuals should be given appropriate advice and support to help the individual achieve and maintain a healthy weight

## 1.6.3 Secondary Prevention: Recommendations Post Myocardial Infarction

All individuals who have had an acute myocardial infarction should have secondary prevention measures initiated in terms of the lifestyle measures detailed under section 1.6.2 above, as well as appropriate preventative pharmacotherapies. These may include:

- ACE (angiotensin-converting enzyme) inhibitor
- Aspirin
- Beta-blocker
- Statin therapies.

Please also see section 6 **Treatment**.

## 2. Epidemiology

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### 2.1 Overview

The mortality rate in Ireland due to all forms of cardiovascular disease (which includes coronary heart disease (CHD), stroke and other circulatory diseases) is approximately 10,000 annually. This figure accounts for 36% of all deaths, making this group of conditions the commonest cause of fatalities in Ireland. Coronary Heart Disease is also the leading cause of death in the UK, most of Europe, and the US.

The death rate for coronary heart disease has fallen in Ireland in the last two decades, in common with other industrialised societies. This is mainly due to improvements in both primary and secondary treatment (for example, cholesterol and blood pressure management and improved care following myocardial infarction), and changes in lifestyle (for example, a reduction in smoking). However, there is a risk that other lifestyle impacts (increasing obesity, decreased physical exercise and an increasing prevalence of diabetes) may cause these rates to rise again.

- 50% of these deaths are due to coronary heart disease.
- 22% of premature deaths (i.e. those aged 65 or less) are due to Cardiovascular Disease.

(Kabir et al, 2007; Irish Heart Foundation, 2009)

### 2.2 Epidemiology of Chronic Stable Angina

Stable Angina is the most prevalent form of coronary heart disease, increasing with age, and thought to be of slightly higher prevalence in women than in men (Daly et al, 2006). Figures from the UK (2006 Health Survey for England) indicate that the prevalence rates suggest that approximately 8% of men and 3% of women aged 55 to 64 report current or past episodes of angina, rising to approximately 14% of men and 8% of women in the 65 to 74 age group (British Heart Foundation, 2008).

Fox et al (2006) suggested that the prevalence rate of angina across Europe increases sharply with age in both sexes from 0.1–1% in women aged 45–54 to 10–15% in women aged 65–74 and from 2–5% in men aged 45–54 to 10–20% in men aged 65–74.

## 2.3 Epidemiology of Acute Coronary Syndromes

### 2.3.1 Unstable Angina

There is little epidemiological information available which specifically covers unstable angina, due to the close relation with non-ST elevation myocardial infarction.

### 2.3.2 Myocardial Infarction

Statistical information regarding the occurrence of myocardial infarction in Ireland is limited. Whilst, the annual incidence of myocardial infarction (MI) for men aged in the UK in the age group 30-69 is about 600 per 100 000 and for women about 200 per 100 000 (Cooper et al, 2007); it is estimated that the incidence in Ireland is higher (British Heart Foundation, 2004). Although these rates are falling (as they are in all developed countries), the death rate from coronary heart disease in the UK and Ireland is still high when compared internationally (Cooper et al, 2007).

Death rates are increased:

- In deprived areas compared to affluent areas (although this gap has narrowed in recent years)
- In men aged <75 compared with women (three times more likely)
- In certain ethnic groups – e.g. individuals of Indian, Pakistani and Bangladesi decent have a 50% higher death rate compared with the general population (Wild and McKeigue, 1997)
- In certain regions and areas – for example Scotland has a higher death rate from coronary heart disease compared to other countries in the UK.

## 3. Aetiology

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### 3.1 Overview

The risk of developing ischaemic heart disease is influenced by a number of factors. These include:

- Increasing Age
- Male gender
- Diet
- Physical inactivity
- Obesity
- Alcohol consumption
- Psychosocial Wellbeing
- Hypertension
- Elevated serum Cholesterol levels
- Diabetes
- Ethnic origin
- Smoking

### 3.2 Aetiology of Ischaemic Heart Disease

Chronic Stable angina results from the development of focal atherosclerotic plaques in the intimal layer of the epicardial coronary artery. The plaques impair the coronary lumen and may limit blood flow to the myocardium, especially during periods of increased myocardial oxygen demand. Although at rest a reduction of 75% in the capacity of the lumen is required to precipitate symptoms, during exertion symptoms may occur when the lumen is impaired by as little as 30%.

Plaque-fissure in a minor atherosclerotic lesion may breach the artery's internal elastic lamina, leading to platelet deposition, thrombus formation, reduction in blood flow and possibly coronary dissection and acute occlusion. This process may change the pattern of angina from stable to unstable, and/or lead to an acute myocardial infarction.

Acute myocardial Infarctions are caused by irreversible necrosis of cardiac muscle: -

- Nearly always (in 90%) due to occlusion of a coronary artery by

atherosclerosis, with or without superadded thrombus.

- Rarely due to coronary embolus (e.g. in atrial fibrillation or infective endocarditis).

The extent of necrosis which occurs may be limited by the degree of collateral blood supply. Coping mechanisms supplied by collateral vessels are better developed in an individual who has an existing history of chronic stable angina. In this case the infarct is often then smaller than would occur in an individual who has developed a sudden occlusion.

## 4. Diagnosis

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### 4.1 Chronic Stable Angina

The diagnosis of chronic stable angina involves clinical assessment, laboratory investigations and a number of specific cardiac investigations which may be invasive or non invasive. Initial diagnosis and assessment is usually undertaken in an outpatient setting. An important aspect of the diagnosis is confirming the extent of ischaemic heart disease, and therefore an indication of the prognosis for the individual in order to identify those who are progressing to more acute coronary symptoms.

In many cases a working diagnosis of angina can be formed on clinical history alone, with subsequent investigations confirming the extent of the condition. However, it should be noted that a significant proportion of individuals who present with chest pain may not have angina, and early identification of alternative diagnoses should be sought (SIGN, 2007).

#### 4.1.1 Clinical Features

There are four areas which indicate features characteristic of angina:

- **Location** is often retrosternal or left side of chest and can radiate to left arm, neck, jaw or back
- **Character:** Type of discomfort is often described as a pressure, tightness, or a dull or heavy pain. The pain may be strangling or constricting.
- **Duration:** Typically the symptoms last up to several minutes after exertion or emotional stress has stopped. Symptoms rarely last longer than 10 minutes. If an individual reports longer attacks, an alternative diagnosis should be sought.
- **Relation to exertion:** Angina is typically exacerbated by exertion or emotional stress and eased with rest. The symptoms may be precipitated by cold weather or following a meal.

Atypical symptoms (more common in older individuals, women and diabetics) include epigastric pain, breathlessness, or nausea (Fox et al, 2006; SIGN, 2007).

Angina is characteristically relieved by glyceryl trinitrate.

**Individuals who report chest pain at rest or on minimal exertion may have unstable angina and should be considered for hospital admission.**

Angina can be graded by severity using the Canadian Cardiovascular Society (CCS) class scale (Campeau, 1976). The classes of severity are shown in table 1 below:



Class	Description
I	Ordinary activity such as walking or climbing stairs does not always precipitate angina.
II	Angina precipitated by emotion, cold weather or meals and by walking upstairs.
III	Marked limitation of ordinary physical activity
IV	Inability to carry out any physical activity without discomfort. Anginal symptoms may be present at rest

**Table 1: Canadian Cardiac Society Angina Grading Scale**

#### 4.1.2 Other History

An individual presenting with symptoms indicative of angina should have a full cardiovascular risk assessment. This should additionally include evaluation of:

- Body Mass Index or waist circumference
- Heart Murmur
- Psychosocial factors such as depression or social isolation
- Physical activity levels.

#### 4.1.3 Complications

Complications of chronic stable angina include escalation to other unstable angina and/or myocardial infarction. Individuals who are affected by chronic stable angina often suffer anxiety and depression, a reduction in general health or functional ability, and a reduced quality of life (SIGN, 2007). Studies have indicated individuals with angina may have poor symptom control, do not undertake opportunities for lifestyle modifications, and require ongoing frequent medical contact (Smith et al, 2002).

#### 4.1.4 Physical Examination

Physical examination is often normal, unless the patient is seen during an episode of pain, when tachycardia or transient arrhythmia may be present.

Features which may indicate predisposing factors include:

- Signs of hyperlipidaemia
- Evidence of vascular disease
- Elevated BP.

- Pallor of anaemia
- Evidence of a previous MI
- Conditions other than coronary heart disease which can occur with angina including aortic stenosis, uncontrolled atrial fibrillation, cardiomyopathy

This may include signs of severe ischaemic myocardial dysfunction and/or cardiac failure such as a raised jugular venous pressure, ankle oedema, basal lung crackles, displaced apex beat, resting tachycardia or pulsus alternans.

#### 4.1.5 Investigations

##### Blood tests

Investigations which are recommended in order to assess the extent of angina and as an input to form a treatment plan:

- Haemoglobin level – to exclude anaemia
- Serum creatinine and estimated glomerular filtration rate — to identify renal impairment (NHS Institute for Innovation and Improvement, 2009)
- Fasting blood glucose
- Lipid profile
- Thyroid function (if indicated by clinical history or physical examination)

[SIGN, 2007]

##### Chest X-ray

Radiography is not routinely recommended, as the results are usually normal. A chest x-ray should only be requested in individuals with suspected heart failure, valvular disease, or pulmonary disease (Fox et al, 2006).

##### Resting 12 lead Electrocardiography

A resting 12 lead ECG should be performed to determine if any arrhythmias, signs of myocardial ischaemia, or evidence of prior myocardial infarction are present (SIGN, 2007; NHS Institute for Innovation and Improvement, 2009), however it should be noted that a normal ECG does not exclude cardiovascular disease as ECG results can be normal in 30% of individuals who present with a typical history of angina.

##### Exercise ECG

An exercise ECG is primarily used to assess prognosis, however this investigation may be unsuitable for some individuals such as those with impaired mobility. High risk findings are

- A low threshold for the development of ischaemic changes

- Widespread, marked or prolonged ischaemic changes
- A fall in BP during exercise
- Exercise induced arrhythmia

In contrast, while not excluding ischaemic heart disease, an exercise ECG that remains normal or only changes nearing the completion of the test is indicative of a good prognosis.

Stress ECG testing has a specificity of 70%. The sensitivity is 90% in men with chest discomfort suggesting angina, this is similar in women but specificity is below 70%.

### **Stress echocardiography and radionuclide scintigraphy**

These investigations are useful to assess regional myocardial ischaemia or viability in certain subgroups (e.g. those unable to exercise, or with abnormal conduction on resting ECG). The stress is either induced pharmacologically (with dobutamine or dipyridamole infusion or by exercise).

The most accurate are stress echocardiography and myocardial perfusion imaging with single-photon emission computed tomography (SPECT) or PET. However, these tests are more expensive than simple stress testing with ECG.

### **Coronary angiography**

This invasive procedure demonstrates coronary artery anatomy (a prerequisite to by-pass graft or angioplasty). Although low risk, the complication rate for this procedure is higher with unstable angina, aortic valve disease, acute MI or cardiogenic shock.

### **Other Investigations**

Newer investigation methods for angina may be available from specialist services which include magnetic resonance perfusion imaging (MRI) and multislice computed tomography (CT) scans.

### **Screening Tools**

Whilst a number of screening tools have been developed to assess the severity of angina, their use is not routinely recommended (SIGN, 2007).

## **4.2 Diagnosis of Acute Coronary Syndromes**

There are five main factors which should be considered in forming a diagnosis of an acute coronary syndrome (American Heart Association/American College of Cardiology (AHA/ACC)):

- Nature of Symptoms

- History
- Age
- Gender
- Number of traditional cardiovascular risk factors present

Key diagnostic investigations involve 12 lead echocardiography, often repeated at intervals during the diagnostic phase; and measurement of cardiac enzymes (usually troponin), again often repeated at intervals during the diagnostic phase.

## 4.3 Unstable Angina

### 4.3.1 Clinical Features

Unstable angina is differentiated from stable angina in that the pain is often of more recent onset with increasing frequency and severity, and unlike stable angina is often not relieved by rest, and can occur at rest and on minimal exertion. Attacks are often prolonged and only partially relieved by GTN.

There are several variations to unstable angina which may present with similar symptoms:

#### **Prinzmetal's variant angina**

This occurs at rest and in response to cold, often at a consistent time of day or night, and is related to augmented coronary artery tone and/or spasm. The condition is rapidly relieved by GTN, but may be provoked by acetylcholine. It is associated with S-T segment elevation indicating transmural ischaemia and can occur in structurally normal coronary arteries or with variable degrees of coronary artery stenosis.

#### **Angina with syncope**

This is more common in the elderly and suggests severe coronary artery disease (or aortic valve stenosis).

#### **Syndrome X**

Syndrome X is typical angina provoked by emotion/anxiety, or of diurnal character. This condition results in normal epicardial arteries when imaged on coronary angiography. Microvasculature structure may be abnormal and may be seen by myocardial scintigraphy imaging. It is associated with ventricular hypertrophy, systemic hypertension, glucose intolerance and insulin resistance.

### 4.3.2 Physical Examination

Physical examination during a period of unstable angina may show symptoms such as tachycardia, abnormal or disordered heart rhythm, hypertension or hypotension; but often there are no physical signs or symptoms on examination.

### 4.3.3 Investigations

A 12 lead electrocardiogram may show changes in acute angina, but can often be normal. Serum cardiac enzyme investigations do not usually show any elevation from normal levels as unstable angina is not usually associated with actual tissue damage to the myocardium

## 4.4 Myocardial Infarction

### 4.4.1 Clinical Features

The first sign that a person has suffered a myocardial infarction may be sudden death – over 50% of all myocardial infarctions are rapidly fatal (Capewell et al, 2001). In those who survive, the features are: -

- Prolonged chest pain, similar in nature to angina, lasting several hours, in over 80% of patients. Pain may be slight or absent in the elderly and diabetics.
- Tachypnoea, breathlessness (and hyperventilation).
- Anxiety and apprehension.
- Sweating, nausea, sometimes vomiting and occasionally hiccoughs.
- Fall in BP, bradycardia and often elevation of the JVP; but sinus tachycardia in  $\frac{1}{3}$  of patients.
- Audible and palpable atrial gallop (A fourth heart sound due to forceful atrial ejection).
- Paradoxical (reversed) splitting of the second sound, plus 3rd or 4th sound.
- End-inspiratory crackles or frank pulmonary oedema.
- After 1-2 days, a pericardial friction rub.
- Necrosis causes moderate pyrexia 12 - 24 hours post-infarct.

It is important to note that diabetics, the elderly and women may have an atypical presentation of symptoms.

### 4.4.2 Complications

- *Dysrhythmias* occur in 90%. All types of dysrhythmia may occur especially within 24 hours of the acute event.

*Other complications include*

- *Cardiogenic shock* – severe hypotension; cold clammy skin; oliguria;

clouding of consciousness

- *Heart failure* – congestive, often causing hypoxaemia.
- *Mild mitral regurgitation* – in around 50% - due to abnormal papillary muscle geometry, malaligned mitral cusps or annular dilatation.
- *Early pericarditis* – with the advent of thrombolysis. (now only in 6 - 7%.)
- *Rupture* – of infarct (fatal haemopericardium – 15% of post-MI deaths);
  - of papillary muscle (mitral regurgitation - <1%);
  - of interventricular septum (acute right ventricular failure – 1 - 3%).
- *Embolism* – either systemic – usually cerebral or pulmonary (DVT).
- *Ventricular aneurysm* – reduced systolic ejection fraction, with paradoxical movements of the bulging section.
- *Dressler's syndrome* – recurrent pericarditis and pleurisy, 2 - 6 weeks post-MI. Responds to NSAIDs (and steroids if necessary).
- *Shoulder-hand syndrome* – frozen shoulder and Raynaud's phenomenon, usually on the left.

#### 4.4.3 Physical Examination

On physical examination the individual may be tachycardic, have abnormal heart rhythm, appear pale, clammy and breathless. Hypertension or hypotension may be present.

#### 4.4.4 Investigations

##### Resting 12 lead Electrocardiography

A resting 12 lead ECG should be performed to determine if any arrhythmias, signs of myocardial ischaemia, or evidence of prior myocardial infarction are present (SIGN, 2007; NHS Institute for Innovation and Improvement, 2009). It is important to note that in 20% of myocardial infarction little or no changes may be detected on an ECG, and in the early hours following a myocardial infarction changes may not be apparent. Repeated ECG recordings should therefore be obtained and compared with previous recordings.

##### Serum Cardiac Enzymes

- Aspartate aminotransferase - raised for 1 - 4 days.
- Creatine phosphokinase - raised for 2 - 3 days.
- Lactate dehydrogenase - raised for 2 weeks.

- Isoenzyme studies to confirm myocardial (rather than skeletal muscle) damage.

### Troponins (T & I):

Regulatory myocyte proteins – can be measured with a bedside test kit. The cardiac markers troponin T and troponin I are extremely sensitive to myocardial injury. Minimal damage can be detected allowing the detection of “micro-infarcts” where there is a rise in the troponin level without an increase in other cardiac enzymes. This has categorised many patients with very small rises in troponin levels as having sustained a myocardial infarction despite the absence of major tissue damage.

Since the introduction of troponin measurement many studies have changed the definition of myocardial infarction. Table 2 below shows current definitions and prognosis of acute coronary syndrome according to troponin concentration.

	12hr serum troponin concentration (ug/l )		
	<0.01	>0.01 and <1.0	>1.0
<b>(BCS) British Cardiac Society definition</b>	ACS with unstable angina	ACS with myocyte necrosis	ACS with clinical myocardial infarction
<b>(ECC/ACC) European Society of Cardiology/American College of Cardiology definition</b>	Unstable angina	Myocardial infarction	Myocardial infarction
<b>(World Health Organisation) definition</b>	Unstable angina	Unstable angina	Myocardial infarction
<b>30 day mortality</b>	4.5%	10.4%	12.9%
<b>6 month mortality</b>	8.6%	18.7%	19.2%

**Table 2: Definition of Acute Coronary Syndrome according to troponin concentration**

#### 4.4.5 Other Investigations

Blood Tests: Polymorph leucocytosis with raised ESR and CRP.

Investigations – if doubt regarding the diagnosis remains:

- Echocardiography
- Radionuclide scintigraphy
- Coronary angiography

## 5. Differential Diagnosis and Comorbidity

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### 5.1 Differential Diagnosis

The differential diagnoses of ischaemic heart disease syndromes include other ischaemic heart conditions, and non cardiac conditions which affect the chest wall, oesophagus and lungs. Such conditions include:

- **Other Ischaemic Heart Conditions** e.g. a progression from stable angina to myocardial infarction
- **Prinzmetal's angina** — a form of angina possibly caused by vasospasm, which is not precipitated by exercise, and has a more severe and longer duration than chronic stable angina. This condition is often associated with unusual appearances on an ECG.
- **Da Costa's syndrome** – Also known as neurocirculatory asthenia; effort syndrome; soldier's heart; cardiac neurosis. This condition is a disordered action of the heart resulting in chest wall pain with the addition of anxiety, dizziness, lassitude, sighing and hyperventilation. This syndrome may affect a subject who also experiences angina.
- **Chest wall pain** - Usually well localised, sharp and fleeting, rarely midline.
- **Bornholm disease** (epidemic pleurodynia, Devil's grip) – This condition initially presents with severe pain, but fever quickly develops. Headache and malaise are common. Recovery is rapid although relapses may continue for several weeks
- **Tietze's syndrome** - pain and swelling of costal cartilages (rare). Pain and tenderness at the costochondral junctions are more common – this is often mistakenly referred to as Tietze's syndrome.
- **Spinal disorders** – cervical spine disorders may cause anterior chest/axilla/arm pain associated with limitation of movement, muscle weakness, reduced or absent arm reflexes. Thoracic spine disorders may result in referred pain.
- **Gastrointestinal or oesophageal disorders** – e.g. **Acid (oesophageal) reflux** and **dyspepsia** which will normally be relieved by antacids.
- **Pleural Pain** – from infection, pulmonary embolus
- **Musculoskeletal Disorders**
- **Psychological Disorders** – panic attacks, anxiety.



## 5.2 Comorbidity

### 5.2.1 Comorbid Conditions

There are a number of which will indicate a poorer outcome and prognosis in individuals with ischaemic heart disease. These include:

- Diabetes
- Hyperlipidemias
- Hypertensive disease
- Lung disease
- Obesity
- Peripheral vascular disease
- Renal disease
- Smoking
- Other cardiovascular disease e.g. stroke

### 5.2.2 Assessment of Risk Prior to Non Cardiac Surgery

An individual with ischaemic heart disease is at increased risk of an ischemic event, when undergoing surgery of any kind. This may mean that additional pre-admission screening is indicated in these individuals. Risk can be assessed using a number of simple screening tools such as the Revised Cardiac Risk Index (Lee, 1999). This suggests that moderate to high risk patients are those having any of the following risk factors:

- High risk surgery – intraperitoneal, intrathoracic or suprainguinal vascularsurgery
- History of ischaemic heart disease
- History of congestive heart failure
- History of cerebrovascular disease
- Presence of insulin dependent diabetes
- Serum creatine level of >180 micromol/l

The risk of adverse effects increases from 0.1% in an individual with none of the above factors, 1.3% in an individual with one of the above factors to 9% in an individual with all of the above factors.

## 6. Treatment

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The objectives of treatment regardless of the type of ischaemic heart condition are threefold – identification and control of risk, symptom relief and optimal management to improve prognosis

### 6.1 Treatment Options for Chronic Stable Angina

#### 6.1.1 Non-Pharmacological Management of Chronic Stable Angina

There are a number of measures which can be beneficial to individuals with angina. These fall into two main groups; patient education and general risk reduction measures:

##### Patient Education

- Understanding the importance of treatment compliance – poor compliance is shown by evidence to result in a poorer outcome
- Understanding of medications, their use, risks and side effects
- Understanding the importance of risk reduction measures and any lifestyle modifications which are required.

##### Risk Reduction Measures:

- Smoking Cessation: Transdermal nicotine is safe in patients with IHD. [14]. The individual should be aware that the cardiac risks of being an overweight ex-smoker are less than those of a thin smoker.
- Weight reduction (lowers BP and lipid levels), until BMI is as close to normal as achievable.
- Regular physical exercise to the development, but not beyond, the onset of symptoms
- A diet low in salt and high in fruit, vegetables, cereals and poly/mono-unsaturated oils in preference to animal fat.
- Blood Pressure Control

#### 6.1.2 Pharmacological Management of Chronic Stable Angina

A number of pharmacological interventions are used in the treatment for angina. These may be used singly, or in combination to achieve maximum benefit.

##### Low Dose Aspirin

Unless contraindicated, all individuals who have been diagnosed with angina should

receive low dose anti-platelet therapy in the form of aspirin 75-150mg daily. There is a question as to the value of enteric coated aspirin in preventing gastrointestinal complications, given their greater cost (SIGN, 2007).

### **Beta Blockers**

Beta blockers should be used as a first line therapy for the relief of symptoms in chronic stable angina (SIGN, 2007). They work by improving coronary flow, but are contraindicated in a number of conditions including sinus bradycardia, a history of asthma or bronchospasm, severe hypertension, severe peripheral arterial disease and uncontrolled heart failure (SIGN, 2007; NHS Institute for Innovation and Improvement, 2009).

Preparations commonly used include atenolol, bisoprolol, or metoprolol.

### **Calcium Channel Blockers**

Calcium-channel blockers reduce afterload and coronary artery spasm by inducing smooth muscle relaxation (SIGN, 2007) They have been shown to be as effective as beta blockers in minimising angina symptoms. The choice of specific drug will be influenced by contraindications, side effects and the presence of comorbidities.

### **Potassium Channel Activators**

Clinical trials indicate that Nicorandil significantly reduces unplanned admissions, morbidity and mortality from coronary heart disease in individuals with angina. The drug acts by relaxing smooth muscle, causing venous and arterial (including coronary) vasodilatation. SIGN (2007) suggest that it is cost effective to include this therapy for all angina patients, on the basis that the increased cost of prescribing is offset by decreased hospital admission costs.

### **Nitrates**

This group of drugs work by reducing preload/venous pooling and afterload/BP, which results in increased coronary perfusion.

- Sublingual – aerosol spray has a longer life, whereas tablet can be removed once attack relieved, reducing side-effects of headache, flushing and postural dizziness. Can be used prophylactically.
- Long-acting mononitrates – an oral form of nitrate with good bioavailability.

### **Lipid-lowering Therapy with Statins**

Statin therapy has been shown in a number of trails to be beneficial to individuals with angina, resulting in reduced mortality, subsequent myocardial infarction, coronary revascularisation and fatal or non fatal stroke.

Available evidence strongly supports the long term use of statins and aspirin in individuals with chronic stable angina.

## Hypertension

Angiotensin converting enzyme (ACE) inhibitors should be considered for blood pressure management if appropriate.

### 6.1.3 Surgical Interventions for Chronic Angina - Coronary Artery Bypass Graft and Percutaneous Intervention Procedures

Surgical intervention in the form of revascularisation can be considered for patients who have continuing angina symptoms despite attempted non-pharmacological and pharmacological therapies. A decision as to the best surgical procedure is typically made once a coronary angiography has been performed, and depends on factors such as anatomy, co-existing conditions (cardiac and non-cardiac), the individual's preferences and local policies and procedures.

There are two main intervention approaches. Traditionally, coronary artery by-pass grafting (CABG) has been used, but more recently percutaneous intervention (PI or PCI) involving either angioplasty (dilatation of the affected vessel using a fine balloon) or 'stenting' (the use of a fine lattice scaffold to expand the vessel wall) has gained in popularity.

Initial trials comparing CABG and PCI / stenting for multi-vessel angina over 3 – 5 years follow-up have shown no evidence of a major difference in the risk of death or MI, and little difference in assessments of physical activity, exercise tolerance, employment status or quality of life, meaning that PCI has become more widely used. However recent trails show that although initial postoperative outcome is similar with both procedures, the risk of major adverse cardiac or cerebrovascular events at 12 months is higher with PCI (Serruys et al, 2009).

## 6.2 Treatment Options for Myocardial Infarction

The aim of treatment for myocardial infarction is to restore impaired blood flow to the myocardium as quickly as possible. This can be achieved either through the use of percutaneous coronary intervention measures, which are ideally delivered within the first 90 minutes following presentation, or through the use of thrombolytic agents, within the firsts 12 hours of symptom onset.

Treatment may involve pharmacological therapies alone, but may involve interventional cardiac procedures such as percutaneous coronary intervention or coronary artery bypass grafting.

**It should be noted that determining the optimal treatment for in an individual with a myocardial infarction is an extremely complex process that takes in to account a number of variables in addition to the traditional cardiac risk factors.**

Examples of these factors are listed below.

- Type of myocardial infarction (STEMI or NSTEMI)

- The extent, distribution and severity of an individual's cardiovascular disease
- The outcome of risk assessment and stratification
- Left ventricular function
- The presence of comorbid conditions
- Relative risks of interventional measures (e.g. revascularisation)
- Availability of specialist beds and interventions (e.g. PCI)

The treatment strategies which are used also vary not only with respect to the individual's characteristics, but with the coronary unit procedures and policies, the extreme pace of evolution in evidence based practice in cardiovascular medicine, and the delivery of healthcare from a systems point of view.

The Canadian Cardiovascular Society, commenting on the 2007 focused update of the American College of Cardiology and American Heart Association 2004 guidelines for the management of ST elevation myocardial infarction, stated that clinical guidelines regarding the treatment of myocardial infarction needed to be focused on the quality and availability of expertise in the geographic area in which treatment is being provided, and be specific to that health care delivery system in order to achieve high quality treatment. They further commented that the rapidity of development in this area meant that care guidelines were often out of date soon after publication (Canadian Cardiovascular Society, 2009).

**This protocol is therefore not written with the intent of providing the level of detail necessary for determining treatment options for a specific individual but merely to provide an overview of the main treatment options available.**

### 6.2.1 Initial Management

There are a number of initial management strategies that can be undertaken which aid in minimising myocardial damage. These include maintaining adequate oxygen saturation with oxygen therapy as required ( $O_2$  saturation >90%), ensuring pain control is achieved using morphine and nitrate therapy to reduce sympathetic pain activity and therefore help reduce myocardial strain, and the early use of the following pharmacotherapies where indicated (noting possible complications e.g. bleeding, possibility of surgery etc.):

- Aspirin – this acts as a platelet aggregation/activation inhibitor and has been shown to reduce mortality and the incidence of non-fatal MI (Cairns et al, 1985), with a 30-50% reduction in coronary events (Antithrombotic Trialists' Collaboration, 2002).
- Anti-thrombotic agents such as clopidogrel and low molecular weight heparin. These are often initiated by pre-hospital care staff such as paramedics
- Beta Blockers

Myocardial Infarction must be considered a medical emergency, and the best outcomes are achieved through treatment in a specialist coronary care unit.

### **Percutaneous Coronary Intervention**

Percutaneous coronary intervention achieved using coronary angiography to place stents (either bare metal or drug eluting) is one method of revascularisation. Many units now have teams able to perform these procedures on a 24/7 basis. This is a less invasive procedure than surgical coronary artery bypass grafting and can be performed under a local anaesthetic.

### **Coronary Artery Bypass Graft (CABG)**

CABG is a surgical intervention performed under general anaesthetic and has been the preferred method of treatment for individuals with extensive ischaemic disease (e.g. multiple vessel, associated poor left ventricular function) or a significantly narrowed main stem artery (National Clinical Guideline Centre for Acute and Chronic Conditions, 2009). There are multiple trials which compare PCI with CABG however the evidence is not directly comparable due to the fact that the individuals selected for each procedure are not directly comparable themselves.

The recommendations in the *draft* NICE guideline for Acute Coronary Syndromes indicate that the choice of revascularisation procedure should continue to be made based on clinical judgement of the individual's risk and characteristics, influenced by location of care and the individual's choice (National Clinical Guideline Centre for Acute and Chronic Conditions, 2009).

### **Thrombolytic Therapy**

Where revascularisation procedures are contraindicated or not available within 90 minutes of presentation, thrombolytic therapies should be initiated. These should be commenced within 12 hours of the first onset of symptoms.

There are a number of contraindications which should be assessed in terms of risk. These include previous cerebral haemorrhage or ischaemic stroke, suspected aortic dissection, active bleeding, or significant trauma (especially closed head or facial) in the last 3 months. The contraindications are due to the increased risk of bleeding with thrombolytic therapies and anti-platelet agents.

The evidence regarding routine interventions following initial treatment with thrombolytic therapies is complex, but routine interventions are not generally recommended at this point.

### **Cardiogenic Shock**

Cardiogenic shock occurs in 5% of people surviving the first hour after an acute MI, with a mortality of 50% to 80% in the first 48 hours. The presence of cardiogenic shock indicates the need for urgent PCI or thrombolytic therapies. PCI (rescue PCI) should be initiated if thrombolytic therapies fail but cardiogenic shock continues. If this is not successful, urgent progression to coronary artery bypass grafting is indicated.

## 6.3 Ongoing care: Cardiac Rehabilitation and Risk Factor Modification

The World Health Organisation defined cardiac rehabilitation in 1993 as 'The sum of activities required to influence favourably the underlying cause of the disease, as well as to provide the best possible physical, mental and social conditions, so that they [the people] may, by their own efforts preserve or resume as normal a place as possible in the community'. The World Health Organisation definition further stated that cardiac rehabilitation is just one factor in secondary prevention of cardiovascular disease.

SIGN (2002) reiterated this point by stating that cardiac rehabilitation was a partnership between an individual and a multidisciplinary team in order to help the individual achieve optimal physical and psychosocial health.

Cardiac Rehabilitation encompasses a number of factors, which commence during an individual's inpatient admission following initial presentation, and continue post discharge into the community. It should be noted that cardiac rehabilitation services differ from region to region as they are affected by external service issues such as funding. Meta-analyses of randomised trials have shown that cardiac rehabilitation reduces mortality by 20 – 25% (Taylor et al, 2004; British Heart Foundation, 2005) and that they improve mobility and perception of health, as well as facilitating the return to work and continued employment of those capable (Engblom et al, 1997).

Rehabilitation is often considered in terms of phases:

- Phase 1: Pre-discharge counselling by a cardiac rehabilitation nurse, Specific advice to correct risk factors, reassurance and patient education
- Phase 2: Early post discharge period interventions aimed at reducing psychosocial factors which may poorly affect prognosis (e.g. psychological distress, depression, lack of social support)
- Phase 3: Community based individually tailored programmes including individual or group activities aimed at reducing risk factors. This may include smoking cessation activities, physical activity programmes, weight management programmes and occupational and vocational activities aimed at resuming work activities
- Phase 4: Long term management of cardiac risk factors

## 6.4 Occupational Factors

It is important to reiterate that ischaemic heart disease does not usually affect the individual's ability to undertake occupational activities in order to avoid anxiety and a loss of confidence for occupational activities. Failure to return to work following an episode of ischaemic heart disease, or a delay in returning, is associated with a poorer outcome and reduced quality of life (British Heart Foundation, 2005). Medical views disabilities and re-employment are derived mainly from medical variables (such as cardiac status and co-morbidity) which are not necessarily

accurate predictors of future occupational activity. For example, there is no evidence to state that the more severe the ischaemia, or damage post myocardial infarction, the more likely it is that an individual will not return to work (Soejima, 1999).

Evidence suggests that the greater impact on an individual's ability to return to work are reinforcement of positive psychological and social factors, accompanied by early discharge followed by prompt rehabilitation. An individual may have many perceptions about their ability to return to work – for example it is common to believe that occupations which have physical activity or jobs of certain natures will not be available to an individual with ischaemic heart disease. This is almost always not the case. Occupations which do not have physical activity have been shown to carry almost twice the risk of developing cardiovascular disease (Benowitz, 1992), and only a very small number of occupations absolutely preclude individuals with heart disease. Whilst it is not possible to continue an occupation as a deep sea diver for example, it is possible to continue to fly as a professional pilot subject to a medical examination.

In commenting on patients' misconceptions, Professor Lewin (in a chapter on psychological factors in cardiac rehabilitation) has stated that: "Lengthy periods of work avoidance make anxiety worse, and work is often an important source of self-validation and social support" (Lewin, 1995). It is also important to note that poverty is a strong predictor of early mortality in IHD and dependence on income replacement payments may result in a poorer prognosis.

Many of the psychosocial and occupational yellow, blue, orange and black flag indicators of poor prognosis which are used in areas such as chronic pain also be applied to assessing prognosis in cardiac rehabilitation. Please refer to the **chronic pain** protocol for further information on the use of the 'flag' indicators for prognosis.

A good overview of the effects of psychosocial factors in ischemic heart disease can be found in a presentation by Professor Lewin which can be accessed at <http://www.cardiacrehabilitation.org.uk/a2z/Donated%20Oral%20presentations/Psychological%20Factors%20-%20Lewin%20R/> [link accessed November 2009].



## 7. Prognosis (Main Prognostic Factors)

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### 7.1 Prognosis of Chronic Stable Angina

There are a number of factors which influence the prognosis for an individual with chronic stable angina. An individual with angina has an increased risk of progression to more severe ischaemic heart disease, however with optimal management, the symptoms of angina can be reduced and the prognosis improved.

The Framingham Heart Study (Fox et al, 2006) found that for individuals with chronic stable angina the risk of adverse outcome was:

- Non-fatal myocardial infarction - 14.3% in men, and 5.5% for women
- Coronary heart disease death over 2 years - 6.2% in men, and 3.8% in women

Factors which will result in a poorer prognosis for stable angina include:

- A greater extent and severity of coronary artery disease; previous myocardial infarction; impaired left ventricular function
- Low exercise or effort tolerance
- Presence of comorbidities
- Time since onset of symptoms – new onset angina has a poorer prognosis than long term stable angina
- Poor adherence to medication therapy
- Failure to undertake lifestyle modification recommendations

[Thadani, 2006; Trujillo and Dobesh, 2007; O'Toole, 2008; BMJ Best Practice 2009].

### 7.2 Prognosis of Acute Coronary Syndromes

Despite advances in treatment over the last two decades, the prognosis for individuals with acute coronary syndromes is still poor. The prognosis is particularly poor in individuals who have acute coronary syndromes with elevated troponin levels, but the mortality rate is still elevated in individuals with non-elevated troponin levels such as unstable angina (SIGN, 2007).

#### 7.2.1 Unstable Angina

It is thought that the presence of ST segment deviation on ECG is a more accurate predictor of poor prognosis than raised troponin levels (Granger et al, 2003; Eagle et al, 2004, SIGN, 2007). Mortality rates are estimated as 4.8% over a 6 month period

in individuals without a ST segment derivation, however these rates rise in the period of 6-12 months to exceed the mortality rates for individuals with ST segment derivations. In the period beyond 12 months it is thought that mortality and morbidity in individuals with NSTEMI coronary syndromes can be as high as 10%. This is possibly due to ongoing atherosclerotic disease processes (Eagle, 2003; Chang 2004)

## 7.2.2 Poor Prognostic Factors in Unstable Angina

Markers of poor prognosis in unstable angina include:

Adverse prognostic markers in patients with unstable angina include:

- Prolonged pain at rest >20 minutes
- Presence of comorbidities
- Short term poor prognostic markers (e.g. indicating an impending MI) include a exacerbation of ischaemic symptoms over a 48 hour period
- pulmonary oedema or new mitral regurgitation
- Age >75 years
- Altered ECG readings including presence of bundle branch block, sustained ventricular tachycardia or ST changes
- Elevated serum troponin levels

(SIGN, 2007; BMJ Best Practice 2009)

## 7.2.3 Myocardial Infarction

The mortality and morbidity rates in myocardial infarction vary considerably dependent on the individual's risk factors and lifestyle at presentation, and the effect of risk lowering strategies (such as lifestyle changes and pharmacotherapies) over a long term period following the initial myocardial infarction.

Prognosis, particularly for individuals who have ST-elevation type myocardial infarction also depends on the period of time from initial onset of symptoms to presentation, and from presentation to treatment.

The following mortality rates have been suggested following a myocardial infarction:

- Within one year of having a first MI: 25% of men and 38% of women will die
- Within 6 years of having a first MI:
  - 18% of men and 35% of women will have another MI,
  - 22% of men and 46% of women will have heart failure

- 7% of men and 6% of women will die suddenly

(AMA, 2004)

#### 7.2.4 Poor Prognostic Factors following Myocardial Infarction

Factors indicating a poorer prognosis following a myocardial infarction include:

- Increasing age
- Female gender
- Extensive infarction (determined by magnitude of changes in cardiac enzymes or widespread ECG changes)
- Mechanical complications (e.g. VSD, free wall rupture or acute mitral regurgitation)
- Anterior infarction (especially if associated with atrioventricular block)
- Cardiogenic shock
- Arrhythmias (ventricular, atrial fibrillation)
- Increased heart rate
- Peripheral vascular disease
- Inferior infarction with right ventricular involvement
- Diabetes mellitus
- Renal Impairment
- Previous angina or MI
- Impaired ventricular function (3<sup>rd</sup> sound or pulmonary oedema)
- Failure to re-perfuse with thrombolysis
- Recurrent infarction/ischaemia while still an in-patient
- Electrical instability (e.g. secondary ventricular tachycardia or fibrillation)
- Depressed heart rate variability
- Abnormal late potentials
- Cerebrovascular disease

[SIGN, 2007; BMJ Best Practice, 2009; National Clinical Guideline Centre for Acute and Chronic Conditions, 2009]

## 8. Information Gathering at the In Person Assessment

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### 8.1 Main Disabling Effects of Angina

Since physical signs are often absent or sparse, assessment of the degree of disability relies heavily on the history and, in particular, on an assessment of the limitations imposed on daily activities, especially on walking and climbing stairs. In more severely affected individuals there may be restriction of some aspects of personal care such as dressing, washing and bathing/showering.

The main disabling effect of angina is exercise / effort limitation due to chest pain, breathlessness and fatigue. This restricts walking distance on the flat, and up stairs or inclines, as well as producing intolerance of cold and windy weather. The exercise intolerance is likely to prevent heavy manual work.

In some individuals the persisting disability is due to illness behaviour. After diagnosis most patients divert more attention to how they feel, scanning their body for any minor symptoms or somatic sensations. For most, these preoccupations diminish over time but those patients in whom they do not are at high risk of developing illness behaviour.

Symptoms of anxiety and panic closely mimic symptoms of angina. Recurrent attacks of atypical chest pain, dizziness, constant tiredness, palpitations and episodic dyspnoea can occur without objective evidence of cardiac dysfunction.

Individuals who have no symptoms or signs of cardiac dysfunction and who can achieve a good workload on exercise testing, with no adverse features, have a very low risk of (further) cardiac events. This applies particularly to younger individuals and is reflected in the DVLA guidelines. In the absence of symptoms and signs of cardiovascular dysfunction, an ability to reach stage 4 of the Bruce protocol without anti-anginal medication for 48 hours previously, enables vocational driving to be resumed (DVLA, 2007).

[Draft Note: The vocational driving statement above is applicable as per the Driver and Vehicle Licensing Agency in the UK. DSP to confirm if this sentence is still correct with respect to Ireland].

When work is resumed, the levels and duration of activity should be increased progressively. It may be helpful to arrange temporary shorter hours, (curtailing both ends of the day to avoid rush hour travelling), and to define with management (and colleagues) a period over which the hours will be extended to the full working day.

Those with stable angina can safely work within their limitations of physical fitness, but should not be put in situations where their angina may be readily provoked and guidance about the psychological stresses associated with managerial duties should be individually tailored (Baxter et al, 2000). Modification of hectic work patterns marked by long hours, competitiveness, time urgency and aggression (Type A behaviour) as part of other stress reduction measures may be beneficial. However,

returning to their own work may be less of a problem than trying a new role or task. Similar considerations apply to shift work and permanent night working. Those with angina find it easier to rise from their bed during the warmer daytime, to have some family contact, and to travel to and from work in quieter times than their day-shift colleagues.

In terms of disability assessment, the functional areas of walking and walking up and down stairs will be significantly affected by more severe effort limitation. Exemption advice may well be warranted in some of these severely affected cases.

### **Post-CABG**

Although the results of coronary artery bypass surgery are good with regard to symptoms and life expectancy, the effect on return to work is less clear cut. UK trials showed a net work gain following CABG (Westaby et al, 1979). However, the majority of trials within the USA and also a trial in Spain showed that CABG is associated with work cessation. The consensus of opinion is that the numbers returning to work post-CABG is disappointing, with many patients not returning to work - even though on purely medical grounds they appear fit to do so.

Non-cardiac causes of invalidism are as important as cardiac causes. A patient's subjective evaluation of their health is a greater determinant of their return to work than their clinically assessed physical capacity. A positive attitude is important for return to work. Of patients who expected problems with work,  $\frac{2}{3}$  actually reported no work problems 12 months after their return to work (Crooq and Lavine, 1979).

Negative expectations of return to work were closely related to patients' reactions to the initial illness, particularly in relation to the patients' images of themselves as being 'damaged'. Those with prolonged symptoms prior to surgery, especially those with limited education and/or income, commonly show a "damaged self" concept (Grundle, 1980).

Psychosocial problems are most important, with the psychological effects of surgery, the reluctance of employers to re-employ those who have been off work for a considerable time before surgery and the opportunity that the operation may provide to take early retirement, all appearing to play a part (Cay, 1995; Broustet, 1995). Economic incentives also play a role in the decision making process. Patients with lower incomes have smaller differentials between their salaries and income replacement payments:

30% of patients referred to an intensive rehabilitation programme were judged to be emotionally distressed post-CABG. The distressed group did not differ in regard to disease status or physical capacity from non-distressed patients. However, they did experience more angina both in daily life and when exposed to a maximal exercise stress test. In the distressed group half the number of patients were employed and twice the number were in receipt of a disability pension compared to the initially non-distressed group.

### **Post-PCI**

PTCA is more successful in returning people to work than CABG (Sobrinho, 1995). However, patients' worries with regard to the risk of re-stenosis, true re-stenosis and

complications of anticoagulant therapy played a part in preventing post-PTCA return to work (Broustet, 1995).

### **Predictors of Employment Status after Cardiac Surgery**

One variable that is a predictor of return to work is pre-operative angina class described by interview. 79% of patients with angina classes 1 and 2 (i.e. those who could walk 200m or climb one flight of stairs at an ordinary pace without chest pain or discomfort) returned to work, compared to 56% of those with angina classes 3 or 4. Persons with less fatigue post-operatively are more likely to return to work (80%) compared to those with greater fatigue (58%) (Grundle et al, 1980).

General psychological and attitudinal predictors of return to work were greater pre-operative job satisfaction, higher well-being scores and lower helplessness scores. One of the strongest predictors of a return to work was a positive answer to the question "Do you feel that you will be able to return to work following your surgery?": of those answering "Yes" 80% returned to work, compared to 40% of those giving uncertain replies and 38% of those giving negative replies.

Higher education, higher family income and less use of religion as social support were also predictive of return to work post-CABG (Grundle et al, 1980).

Patients more likely to return to work are those: without post-operative angina.

- working before surgery.
- under 50 years of age.
- Literate
- with professional or executive employment before surgery

(Speziale, 1996)

### **Summary**

- Returning to work is an important part of regaining quality of life
- Failure to return to work often relates more to the patient's health beliefs than to the condition of their heart
- Health professionals should address the issue of returning to work early and positively by tackling mistaken health beliefs
- Patients should be given simple advice to facilitate a phased return to work
- Cardiac Rehabilitation programmes should include assessment and treatment strategies to facilitate early return to work.

## 8.2 Main Disabling Effects of Myocardial Infarction

CABG or PCI allow patients to return to work earlier and at a level of fitness higher than that present before operation.

Where indicated, cardiac pacing, valve replacement or cardiac transplantation may improve cardiovascular fitness. Despite rehabilitation programmes, some patients do not make a full recovery. Though chronic physical disability after MI is unusual, when it does occur it is due to angina, dyspnoea or fatigue from unrelieved myocardial ischaemia and pump failure.

The risk of sudden disability and death from ventricular fibrillation is the major factor affecting work capacity. The risk is proportional to the degree of myocardial damage. Continuing myocardial ischaemia also worsens the prognosis. Subjects with continuing severe disability, poor left ventricular function or a progressive cardiac disorder (e.g. dilated cardiomyopathy) should generally be advised to retire. In contrast, subjects with good ventricular function, a stable rhythm and minimal disability usually do well and should be encouraged to return or prepare for work after 4 - 6 weeks, though longer may be required in some cases (Baxter, 2000).

If CABG is performed, return to work (when possible) is usually 1 - 2 months post-operation. After less traumatic procedures (e.g. pacemaker implant or PCI), return to work is much quicker, sometimes after only 48 hours (British Heart Foundation, 2005).

Psychological difficulties may be experienced, reducing morale and delaying recovery. Anxieties of both the patient and spouse impair the ability of individuals surviving MI to return to work. Half have some anxiety or depression, and half of these have severe symptoms persisting (if untreated) a year later. In general, physical activity is good for the heart, so activities causing no symptoms can be undertaken safely and artificial restrictions are unnecessary. However, after MI not all will be able to return to their original job. In light engineering after one year, about half of those returning to work were fully fit, requiring no job change, whereas one tenth of all those returning had severe limitations requiring a change of role. Work responding to emergency calls should be avoided. Heavy physical work, repetitive use of stairs, piece-rate work, technical skill and the stress of responsibility may all need to be avoided. It has been shown that altering Type A behaviour reduces cardiac morbidity and mortality in post-MI patients. However, if employees were managing rapid and tightly paced repetitive work (e.g. on assembly lines) before MI, they may well manage afterwards if not impaired by angina or dyspnoea.

## 9. Analysis of Effect on Functional Ability

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Eligibility to the Department of Social and Family Affairs various Illness-related schemes and the Activation Programme, is determined primarily by the degree of Ability/Disability and its expected duration.

The degree of Ability/Disability assessed, using the Indicators in 9.1, can be depicted on the Ability/Disability Profile illustrated in 9.2.

In many cases of IHD clinical signs may be few or even absent. Assessment of functional ability relies heavily on the recognised steps of Disability Analysis i.e. clinical history, activities of an average day, observed behaviour and unlike in many other disabilities less on clinical findings. Particular care should be given to obtaining a clear account of activities such as walking, climbing stairs and in more severe cases should include any difficulties with personal care such as washing, dressing and bathing. Variability should also be considered.

### 9.1 Indicators of Ability/Disability

#### Normal

- Symptoms and treatment do not suggest a diagnosis of angina
- Discharged from cardiac clinic
- GTN medication may be out of date or left at home
- No restriction in day to day activities particularly with walking and stairs
- No dyspnoea at rest or on activity during assessment process
- Cardiovascular examination likely to be normal

#### Mild

- Discharged from cardiac clinic
- Stable on cardiac medication and symptoms well controlled
- Successfully completed cardiac rehabilitation course
- Successful revascularisation procedure at least six months ago
- Minimal restriction of average day activities particularly walking and climbing stairs
- No evidence of dyspnoea during the assessment process
- Examination of the cardiovascular system likely to be normal



## **Moderate**

- Attendance at hospital cardiology clinic
- An emergency admission to hospital in the last two years
- Attending or recently completed a cardiac rehabilitation course
- On regular treatment with appropriate cardiac drugs
- Experiences regular symptoms of angina requiring GTN spray for relief
- Avoids rushing and walks at own pace
- May use GTN prophylactically
- No evidence of dyspnoea at rest or in the course of the assessment process
- Cardiovascular examination likely to be normal

## **Severe**

- Multiple emergency cardiac admissions to hospital in the last two years
- Frequent symptoms of angina on mild/moderate exertion
- Treatment with high dosage combinations of cardiac drugs
- May be awaiting surgical intervention for angina
- May have some restriction in walking distance and on climbing stairs on a day to day basis
- There may be evidence of dyspnoea during the course of the examination process e.g. climbing on and off the examination couch, which can be equated to climbing stairs
- Clinical examination of the cardiovascular system is likely to be normal

## **Profound**

- May be under regular review at a cardiology clinic
- Previous unsuccessful surgical intervention for angina
- On maximum dosage of cardiac combination medication
- Severely restricted exercise tolerance ,may rarely leave home
- Cannot manage stairs at home
- May require help with self care e.g. bathing and dressing

- May look grey and unwell
- Likely to be breathless at rest and on speaking
- Where examination findings are needed to provide confirmatory evidence the techniques should be minimally intrusive

## 9.2 Ability/Disability Profile

Indicate the degree to which the Claimant's condition has affected their ability in ALL of the following areas.					
	Normal	Mild	Moderate	Severe	Profound
Mental health/Behaviour	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Learning/Intelligence	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Consciousness/Seizures	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Balance/Co-ordination	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Vision	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hearing	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Speech	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Continence	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Reaching	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Manual dexterity	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Lifting/Carrying	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Bending/Kneeling/Squatting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sitting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Standing	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Climbing stairs/Ladders	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Walking	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

## 10. Summary of Scheme Criteria

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Scheme eligibility criteria are maintained on the DSP website and are accessible from the following links:

- **Carer's Allowance**  
[http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers\\_all.aspx](http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers_all.aspx)
- **Carer's Benefit**  
[http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers\\_ben.aspx](http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers_ben.aspx)
- **Disability Allowance**  
<http://www.welfare.ie/EN/OperationalGuidelines/Pages/disall.aspx>
- **Disablement Benefit**  
[http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib\\_disableb.aspx](http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib_disableb.aspx)
- **Domiciliary Care Allowance**  
<http://www.welfare.ie/EN/Schemes/IllnessDisabilityAndCaring/Carers/DomiciliaryCareAllowance/Pages/DomiciliaryCareAllowance.aspx>
- **Illness Benefit**  
<http://www.welfare.ie/EN/OperationalGuidelines/Pages/illben.aspx>
- **Injury Benefit**  
[http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib\\_injuryb.aspx](http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib_injuryb.aspx)
- **Invalidity Pension**  
<http://www.welfare.ie/EN/OperationalGuidelines/Pages/invalidity.aspx>
- **Respite Care Grant**  
<http://www.welfare.ie/EN/OperationalGuidelines/Pages/respitegrant.aspx>

## Appendix A - Risk Factors for IHD

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### A.1 Modifiable Factors

#### Elevated cholesterol

- 1/3 of UK population have a high blood cholesterol (> 6.5mmol/L).
- Inverse relationship between high-density lipoprotein (HDL) levels and IHD.
- Raised blood triglyceride levels are a weak risk factor.

#### Smoking

- Increased risk of IHD from smoking is dose-related.
- Those smoking 20 or more cigarettes per day have a 2–3 times greater risk of developing a major coronary event than the general population.
- Increases thrombogenesis, vasoconstriction, BP, heart rate and myocardial oxygen demand.
- Promotes atherosclerosis and provokes cardiac arrhythmias.
- Reduces oxygen-carrying capacity.

#### Obesity

- Correlation between weight, raised BP & blood cholesterol, non-insulin diabetes mellitus (NIDDM) and low levels of physical activity.

#### Diabetes Mellitus

- IHD develops at a younger age in diabetics and is more severe and diffuse in age-matched controls.
- Premature IHD in insulin-dependent diabetics.
- In NIDDM, risk of developing IHD is 2 – 4 times higher than in the general population.

#### Systemic hypertension

- Each 5mmHg reduction in diastolic BP reduces the risk of IHD by approximately 16%.

#### Sex hormones

- Combined oral contraceptives approximately triple the risk of IHD.

### Psychological factors

- Stress and Type A personality (aggression, competitiveness and hostility) are risk factors for IHD.
- Anxiety and depression are important predictors for IHD.

### Physical activity

- Regular aerobic exercise of moderate intensity may reduce the incidence of IHD by 20 – 40%. Physical inactivity roughly doubles the risk of IHD (and is a major risk factor for stroke) (British Heart Foundation, 2005).

### Clotting and alcohol

- Some patients with IHD have increased levels of inhibitors of plasminogen activators.
- Alcohol in low dose increases endogenous thrombolysis, reduces platelet adhesion and increases circulating levels of HDL.

### Infection

- Infection with Chlamydia pneumoniae (a common respiratory pathogen) seems linked to the presence of atherosclerotic IHD.

## 10.2 Non-Modifiable Factors

### Gender

- IHD morbidity in males twice that in females.
- IHD occurs approximately 10 years earlier in men than women.
- Endogenous oestrogen is protective, but after the menopause the incidence of IHD in women (unless on HRT) rises steeply and parallels that seen in men.

### Family history

- IHD in a first-degree relative aged less than 70 years gives an odds ratio 2 – 4 times that of a control population.
- Positive family history (premature IHD death or onset of symptoms/diagnosis before 55 years in men or 60 years in women) reduces the age of onset of IHD.

### Race

- Incidence of premature death from IHD is higher in Asians (and lower in Afro-Caribbeans) living in this country than in the indigenous population.

### **Geography**

- Death rates from IHD are higher in Northern Ireland, Scotland and the north of England than in the rest of the UK.

### **Social class**

- Socio-economic gradients in IHD are widening.
- Premature death from IHD is three times more likely for male unskilled workers than for members of the professions, and twice as likely for the wives of manual workers than the wives of non-manual workers.

## Appendix A - Bruce Protocols

### B.1.1 Full (Standard) Bruce Protocol

Stage	Speed (mph)	Gradient (%)	Duration (min)	Cumulative time (min)
I	1.7	10	3	3
II	2.5	12	3	6
III	3.4	14	3	9
IV	4.2	16	3	12
V	5.0	18	3	15
VI	5.5	20	3	18
VII	6.0	22	3	21

### B.1.2 Modified Bruce Protocol

Stage	Speed (mph)	Gradient (%)	Duration (min)	Cumulative time (min)
I	1.7	0	3	3
II	1.7	5	3	6
III	1.7	10	3	9
IV	2.5	12	3	12
V	3.4	14	3	15
VI	4.2	16	3	18
VII	5.0	18	3	21



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