Medical Services

ISCHAEMIC HEART DISEASE

Version 2 Final

Medical Services

Document control

Version history

Version	Date	Comments
2 Final	9 October 2007	Signed off by Medical Services Contract Management Team
2e (draft)	23 August 2007	Comments
2d (draft)	22 August 2007	Formatting
2c (draft)	11 June 2007	External review
2b (draft)	3 May 2007	Internal QA
2a (draft)	5 April 2007	Initial Draft

Changes since last version

1Introduction

Definition

Ischaemic heart disease (I.H.D.) involves impairment of blood flow through the coronary arteries, most commonly by atherosclerotic narrowing. Clinical presentations include silent ischaemia, angina pectoris (stable angina), acute coronary syndromes (unstable angina, myocardial infarction), and sudden cardiac death. [1]

Coronary heart disease (CHD) kills more than 117,000 people in the U.K. every year (around 1 in 5 deaths in males and 1 in 6 in females). More than 1.4 million people suffer from angina and 275,000 people have a heart attack annually.

Cardiovascular disease (CVD) is the main cause of death in the U.K. totaling 238,000 (39%) deaths, half of which are due to CHD and a quarter from stroke. The Government is committed to reducing the death rate from coronary heart disease and stroke and related diseases in people under 75 by at least 40% (to 83.8 deaths per 100,000 population) by 2010.

Every second – a patient is prescribed a course of lipid lowering drug (statin)
Every minute – 380 patients are prescribed a heart drug
Every hour – 50 hospital inpatients receive treatment for CHD
Every day – 250 patients undergo a coronary artery bypass or angioplasty. [2]

Many individuals with CHD are asymptomatic until the onset of a major event, such as a heart attack. This explains the emphasis on the identification and appropriate management of risk factors to reduce the prevalence of coronary heart disease.

Risk is influenced by;-

Gender and Age
Diet
Physical activity
Obesity
Alcohol
Psychosocial Wellbeing
Blood Pressure
Blood Cholesterol
Diabetes
Ethnic origin

The focus of prevention in recent guidelines has switched from ischaemic heart disease (IHD) to cardiovascular disease (CVD) to emphasise the importance of stroke prevention as well.
Primary prevention involves intervention in individuals without clinical evidence of CVD.
S*econdary prevention* involves patients with known CVD who should all be considered for risk reduction therapy – e.g. with aspirin or statins).

The recently revised Joint British Societies' guidelines [3] on prevention of cardiovascular disease in clinical practice recommend that cardiovascular disease prevention should focus not only, but also, on people with established cardiovascular disease, people with diabetes mellitus and those with CVD risk of 20% or greater over 10 years. The guidelines recommend that more intensive lifestyle intervention and the appropriate use of antihypertensive, lipid lowering, glucose lowering and other cardiovascular protective therapies should also be used to reduce overall cardiovascular risk in those with:

- Elevated blood pressure: equal to or above 160 mm Hg systolic or 100 mm Hg diastolic, or lesser degrees of blood pressure elevation (140 / 90) if there is end (target) organ damage
- Elevated total cholesterol (TC) to greater than 5 mmol/L and low density lipoprotein (LDL) cholesterol equal to or above 3.5mmol/L.
- Familial dyslipidaemia, e.g. familial hypercholesterolaemia or familial combined hyperlipidaemia.

Risk assessment charts estimating absolute coronary or cardiovascular risk should be used to identify high risk people for primary prevention and to recommend appropriate therapy. They are an aid to making clinical decisions about how intensively to intervene on lifestyle and whether to use antihypertensive and lipid lowering medication. They should not replace clinical judgment.

The charts have their limitations.

- All charts are based on groups of people with untreated levels of blood pressure. Some use the total cholesterol and LDL cholesterol concentrations while others use the LDL to HDL ratio or TC to HDL ratio. Charts are not appropriate for patients who have existing diseases which already put them at high risk, e.g. pre-existing cardiovascular disease, familial lipid disorders, renal dysfunction or established hypertension or diabetes with associated target organ damage.
- The charts should not be used to decide whether to introduce antihypertensive medication when blood pressure is persistently at or above 160/100 or when end organ damage due to hypertension is present. In both cases antihypertensive medication is recommended regardless of CHD risk.
- They are likely to underestimate risk in people with a family history of premature CHD or in people from certain high-risk ethnic groups such as South Asians.
- Inaccuracies in estimating risk may occur in people already taking antihypertensive treatment. [4]

Current guidelines for management of Primary and Secondary prevention of cardiovascular disease are detailed in the table below. [3]

Cardiovascular risk	Lifestyle	Drug Therapy	Treatment Goals	Follow up
CVD risk clinically determined >20% Secondary prevention	Intensive lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation interventions. Lifestye advice should be given simultaneously with drug treatment	**In all patients** Aspirin or other antiplatelet drug. Intensive statin therapy **In all patients with CHD** An ACE inhibitor **Following an MI** A beta blocker **With hypertension** Antihypertensive drug therapy	TC < 5mmol/L LDL < 3mmol/L BP Reduced to <140 mmHg systolic and /or <90 mmHg diastolic	Risk factor monitoring every 3 to 6 months
CVD risk calculated at>20% Primary intervention	Intensive lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation interventions. Lifestye advice should be given simultaneously with drug treatment	Aspirin 40mg simvastatin or equivalent dose of pravastatin if simvastatin contraindicated Anti-hypertensive drug therapy in hypertensive individuals	TC < 5mmol/L LDL < 3mmol/L BP Reduced to <140 mmHg systolic and /or <90 mmHg diastolic	Risk factor monitoring every 6 to 12 months
CVD risk calculated 10-20% Primary prevention	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation interventions. Lifestyle advice should be given3-6 months prior to initiating drug treatment.	Drug treatment indicated for people with extreme risk factors. People with isolated high risk factor either TC>8mm/l or BP>160/100mmHg should have these risk factors treated and considered for drug therapy to reduce levels or other modifiable factors		Risk assessments every 1-5 years depending on clinical circumstances.

2 Angina

Synonyms / other terms: angina pectoris; effort angina; angina of effort.

Description

Angina pectoris, often simply known as angina, is a clinical syndrome characterised by discomfort in the chest, shoulder, back, arm, or jaw. [5] Angina is usually caused by coronary artery atherosclerotic disease. Rarer causes include valvular heart disease, hypertrophic cardiomyopathy, uncontrolled hypertension, or vasospasm or endothelial dysfunction not related to atherosclerosis.

Stable angina is defined as regular or predictable angina symptoms that have been occurring for over 2 months. Symptoms are transient and are typically provoked by exertion, and alleviated by rest or glyceryl trinitrate (GTN). Other precipitants include cold weather, eating, or emotional distress.

Unstable angina is diagnosed if there is a rapid decline in exercise capacity or if there are episodes of pain at rest. This is usually associated with atherosclerotic plaque instability and, as myocardial infarction and death may ensue, should be assessed and treated urgently, usually requiring hospital admission.

Aetiology

Myocardial oxygen consumption is determined by systolic wall tension, contractile state, heart rate and the presence of ventricular hypertrophy. At rest, a coronary artery atherosclerotic stenosis must reduce luminal diameter by over 75% to affect flow, but maximal flow (during exercise) may be reduced when the lumen is impaired by as little as 30%. In practice, a lesion reducing diameter by 50% or more on coronary arteriogram is judged significant. Coronary artery spasm may temporarily reduce the 'angina threshold'. When a coronary artery is either stenosed or occluded, collateral vessels may dilate rapidly, offering an alternative route of myocardial perfusion. 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, or lead to an acute myocardial infarction.

Prevalence

The prevalence of stable angina remains unclear. [6] [7] Epidemiological studies in the UK estimate that 6–16% of men and 3–10% of women aged 65–74 years have experienced angina. [8] [9]

Annually, about 1% of the population visit their general practitioner with symptoms of angina [10] and 23 000 people with new anginal symptoms present to their general practitioner each year in the UK. [11] These studies did not distinguish between stable and unstable angina. [8] [9] [11]

Diagnosis

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.

Clinical History (Stable Angina)

Patients with stable angina should have the diagnosis made, where possible, following a carefully performed clinical assessment. Clinical history is the key component in the evaluation of the patient with angina. Often the diagnosis can be made on the basis of clinical history alone. There are several typical characteristics of stable angina which should raise awareness of the likelihood of underlying CHD. These include: [6]

- Type of discomfort is often described as tight, dull or heavy
- Location is often retrosternal or left side of chest and can radiate to left arm, neck jaw or back
- Relation to exertion. Angina is characteristically brought on with exertion or emotional stress and eased with rest
- Duration Typically the symptoms last up to several minutes after exertion or emotional stress has stopped
- Other factors Angina may be precipitated by cold weather or following a meal

Angina can be graded by severity using the Canadian Cardiovascular Society (CCS) class scale. [12]

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

CCS class was linearly associated with angiographic findings, revascularization rates, mortality and nonfatal myocardial infarction. These findings support the importance of a four-level grading of symptom severity among angina patients.

Breathlessness

The predominant features described by some patients are discomfort and heaviness rather than pain. Chest discomfort, irrespective of site, is more likely to be angina when precipitated by exertion and relieved by rest. It is also characteristically relieved by glyceryl trinitrate. Not all patients will present with pain or discomfort and the clinician should be aware of other symptoms such as breathlessness, fatigue and belching which may be the initial presenting symptom.

Physical Signs

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

Features of predisposing factors may be present and include: -

- Stigmata of hyperlipidaemia: -
    - Corneal arcus (juvenilis).
    - Xanthelasmata and xanthomata.
- Elevated BP.
- Pallor of anaemia

Evidence of a previous MI

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.

Investigations

Resting ECG

- Very limited diagnostic value
- Can be normal in 30% of those with a typical history of angina..
- Minor ST-T segment changes are common in the general population.
- Widespread changes indicate severe, diffuse coronary disease and a poor prognosis.
- Resting ECG changes do not of themselves establish or refute the diagnosis.

Chest X-ray

- Usually normal.
- Enlarged heart may indicate left ventricular dysfunction or aneurysmal dilatation.

Blood tests

- To plan treatment and assess prognosis
- FBC,
- Urea and electrolytes,
- glucose,
- lipids,
- Thyroid Function Tests
- Liver Function Tests.

Exercise ECG

An exercise ECG is primarily used to assess prognosis. 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 CAD, 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% and sensitivity is 90% in men with chest discomfort suggesting angina. The sensitivity is similar in women but specificity is below 70%.

- Needs specialist supervision and full resuscitation facilities.
- Treadmill is preferable to bicycle ergometer as it is supervisor controlled.
- Symptoms, arrhythmias and BP are monitored as well and these (and work rate achieved) influence prognosis.
- It is often performed during the recovery phase of a myocardial infarction or in subjects with a history of unstable angina.
- Should comply with a recognised protocol (normally, modified or full Bruce Protocol [Appendix B].
- "Double product" (peak heart rate x peak systolic BP) correlates well with maximum myocardial oxygen consumption (MVO_2).

Stress echocardiography and radionuclide scintigraphy

These tests 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 induced pharmacologically (with dobutamine or dipyridamole infusion). [6]
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 demonstrates coronary artery anatomy (a prerequisite to by-pass graft or angioplasty).

It is invasive, but low risk (morbidity of 0.8%, mortality of 0.12% in elective patients).

The complication rate is higher with unstable angina, aortic valve disease, acute MI or cardiogenic shock. [13]

Coronary angiography is best performed where there are facilities to proceed immediately to PCI (e.g. angiography with or without stenting) of suitable lesions.

Differential Diagnosis

A full description of the differential diagnosis is given in the protocol on Chest Pain, but a summary is included here:

Da Costa's syndrome – (synonyms: - neurocirculatory asthenia; effort syndrome; soldier's heart; cardiac neurosis; disordered action of the heart) is 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. It is rarely in the midline.

Bornholm disease (synonyms: epidemic pleurodynia, Devil's grip) – presents with severe pain, but fever quickly develops. Headache and malaise are common but there is usually rapid recovery, though relapses frequently continue for several weeks.

Tietze's syndrome with pain and swelling of costal cartilages is rare.
However, pain and tenderness at the costochondral junctions is more common and often mistakenly referred to as Tietze's syndrome.

Disease of the *cervical spine* causing anterior chest/axilla/arm pain is usually associated with limitation of movement, muscle weakness and reduced or absent arm reflexes. It often has dermatome-pattern distribution.

*Gastrointestinal pain is o*ften related to eating certain foods or time since eating. Acid (oesophageal) reflux and dyspepsia will normally be relieved by antacids. Endoscopy, ultrasound and oesophageal scanning can confirm the underlying gastrointestinal pathology.

Other Types of Angina

Unstable angina

Modern synonym for this and acute myocardial infarction is acute coronary syndrome (ACS).

History will be of recent onset (within 2-4 weeks) with increasing frequency and severity.

Attacks are often prolonged or only partially relieved by GTN.

May be due to an unstable atheromatous plaque and this may lead to intermediate high-risk acute coronary syndrome (with myocardial damage). There is significant risk of progression to acute MI or sudden death and urgent specialist referral is advised.

Prinzmetal's variant angina

This occurs at rest and in response to cold, often at a consistent time of day or night.

It is related to augmented coronary artery tone and/or spasm.

It is rapidly relieved by GTN. It 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.

The syncope suggests severe coronary artery disease (or aortic valve stenosis).

Syndrome X

Typical angina provoked by emotion/anxiety, or of diurnal character.

Normal epicardial arteries 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.

Treatment

The objectives of treatment are threefold.

- To identify and control the risk factors

- To relieve the symptoms

- To improve the prognosis

Medical Management

General measures for risk factors

- Stop smoking. Transdermal nicotine is safe in patients with IHD. [14]

- 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.

- Cardiac risks of being an overweight ex-smoker are less than those of a

thin smoker.

Symptom Control

Nitrates – reduce preload/venous pooling and afterload/BP, thus increasing 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* – oral form with good bioavailability.

Cardioselective β-blockade – lowers MVO_2 by reduction of myocardial contractility, pulse rate and BP. Reduces frequency and severity of attacks and incidence of major cardiac events, so improves prognosis.
Beta blockers should be used as first line therapy for the relief of symptoms of stable angina

Calcium-channel blockers – reduce afterload and coronary artery spasm. Avoid nifedipine without β-blockade. Diltiazem is considered the best of the first-generation drugs. Verapamil is of use if β-blocker contra-indicated or not tolerated.
Patients with Prinzmetal angina should be treated with a dihydropyridine derivative calcium channel blocker.
When adequate control of anginal symptoms is not achieved with beta-blockade a calcium channel blocker should be added.

Nicorandil – combines properties of a nitrate and a potassium channel activator – relaxes smooth muscle, thus causing venous and arterial (including coronary) vasodilatation.

Improve Prognosis

Low-dose aspirin (if no specific contraindication) – 75mg daily usually avoids gastrointestinal side-effects, and lowers the risk of a subsequent vascular event.

Lipid-lowering therapy with Statins
A meta-analysis of data from 14 randomised trials of statins involving 90,056 patients including patients with stable angina has shown the overall benefit of statin therapy. There was a significant reduction in all cause and coronary mortality, myocardial infarction, the need for coronary revascularisation and fatal or non fatal stroke.

HRT - post-menopausal women already on HRT should continue it, since long-term use protects against IHD events. However, HRT should not be started because there is no overall cardiovascular benefit (but an increase in the risk of IHD events and gallbladder disease) in the early years of therapy. [15]

'Stepped' therapy

All– aspirin and GTN as required, plus lipid-lowering therapy if indicated.

Most – as 'all' plus β-blocker (or calcium-channel blocker), including treatment of hypertension

Some– (10 - 20%) – as 'all' plus β-blocker <u>and</u> calcium-channel blocker or oral nitrate.

Resistant patients– as 'all' plus β-blocker and calcium-channel blocker <u>and</u> oral nitrate, and possibly also Nicorandil. [28]

Surgical Management

There are two indications for surgical management. These are, either to relieve symptoms that are significantly affecting quality of life or to improve life expectancy (e.g. in patients with left main stem disease.

Coronary Revascularisation

Coronary artery by-pass grafting (CABG)

CABG uses sections of autologous veins (eg, saphenous) or, preferably, arteries to bypass diseased segments. At 1 yr, about 85% of venous bypass grafts are patent, but after 10 yr, as many as 97% of internal mammary artery grafts are patent. Arteries also hypertrophy to accommodate increased flow. CABG is preferred for patients with left main artery disease, 3-vessel disease, or diabetes mellitus.

CABG is typically performed during cardiopulmonary bypass with the heart stopped. A bypass machine pumps and oxygenates blood. Risks of the procedure include stroke and MI. For patients with a normal-sized heart, no history of MI, good ventricular function, and no additional risk factors, risk is < 5% for perioperative MI, 2 to 3% for stroke, and ≤ 1% for mortality. Risk increases with age and presence of underlying disease. Operative mortality rate is 3 to 5 times higher for a second bypass than for the first; thus, timing of the first bypass should be optimal.

After cardiopulmonary bypass, about 25 to 30% of patients develop cognitive dysfunction, possibly caused by microemboli originating in the bypass machine. Dysfunction ranges from mild to severe and may persist for weeks to years. CABG is very effective in selected patients with angina. The ideal candidate has severe angina pectoris and localized disease, with an otherwise healthy heart. 75-90% are free of angina after successful surgery, but 4% per annum experience recurrent angina. Exercise stress testing shows positive correlation between graft patency and improved exercise tolerance, but exercise tolerance sometimes remains improved despite graft closure. [5]
CABG reduces the need for drug treatment and increases work capability. [16]

Percutaneous Interventions (PCI)

Now more PCI procedures are carried out worldwide than CABG. PCI (eg, angioplasty, stenting) should be considered if angina persists despite drug therapy and worsens quality of life or if anatomic lesions (noted during angiography) put a patient at high risk of mortality. The choice between PCI and CABG surgery depends on extent and location of anatomic lesions, the experience of the surgeon and hospital, and, to some extent, patient preference. PCI is usually preferred for 1- or 2-vessel disease with suitable anatomic lesions. Lesions that are long or near bifurcation points are often not amenable to PCI. Most PCI is done with stenting rather than balloon angioplasty alone, and as stent technology improves, PCI is being used for more complicated cases. Risk is comparable with that for CABG. Mortality rate is 1 to 3%; MI rate is 3 to 5%. In < 3%, intimal dissection causes obstruction requiring emergency CABG. After stenting, aspirin is supplemented with clopidogrel for at least 1 month, but preferably 6 to 17 months, and a statin is added if not already being used. About 5 to 15% of stents reocclude in a few days or weeks, requiring placement of a new stent inside the original or CABG. Occasionally, occluded stents are asymptomatic and angiography 1 year later shows an apparently normal lumen in about 30% of these affected vessels. Patients may quickly return to work and usual activities, but strenuous activities should be avoided for 6 weeks. It carries early mortality rate of 0.5 - 1% and complication rate of 3 - 5%.

Trials comparing CABG and PCI / stenting for multi-vessel disease 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. [17]

Percutaneous myocardial revascularisation (PMR)

Transcutaneous myocardial revascularisation (TMR)

Both procedures use laser technology, and create small 1-2mm channels into the myocardium.

- either - from the left ventricular cavity using a percutaneous retrograde arterial approach (PMR);

- or - from the epicardium using an open-chest left thoracotomy approach (TMR).

Currently, studies are limited to patients in whom conventional revascularisation (CABG or PCI) is not technically feasible.

Symptomatic improvement is seen in most patients, confirmed objectively on exercise testing.

However, favourable prognostic data are lacking; the results of the DIRECT trial suggest that laser revascularisation does not significantly improve life expectancy.

Prognosis

Stable Angina

Aggressive risk factor modification, effective drug therapy and successful revascularisation have reduced risk and improved prognosis:

Extent of disease	Annual mortality
Single-vessel	1 - 2%
3-vessel	3.5 - 7.5%
Left main-stem	10 - 13%

By 4 years after revascularisation, 60% of patients with chronic stable angina were free of angina and their health related quality of life (HRQOL) was the same as or better than that of the general population. However, the HRQOL of patients with new or persistent angina, was worse than that in control group of the community. [18]

Unstable Angina

For patients with unstable angina (or non-Q wave infarction), there is a 10 – 15% risk of death or (further) infarction in the first 30 days after the onset. A further 35 – 50% will experience recurrent ischaemia. If this persists despite intensive medical therapy, coronary angiography followed by PCI or CABG is undertaken. All high-risk patients should undergo angiography, as the prognosis for those with multi-vessel disease and impaired left ventricular function is improved by surgery.

After stabilisation, whether medical and/or surgical, secondary risk-reduction measures should be implemented with help to stop smoking; dietary advice; increased physical activity; aspirin therapy; treatment of hypertension and, in most cases, cholesterol lowering with a statin. Patients stabilised with medical treatment alone remain at risk from IHD, so most should undergo treadmill exercise ECG assessment.

Main Disabling Effects

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. [19]

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 [20] 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 the IB-PCA the functional areas 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. [16] 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 underline expected problems with work, $^2/_3$ actually reported no work problems 12 months after their return to work. [22]

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. [23]

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 [24] [25] 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. [26] 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. [25]

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%). [23]

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. [23]

Patients more likely to return to work are those: [27]

- without post-operative angina.
- working before surgery.
- under 50 years of age.
- Literate.
- with professional or executive employment before surgery.

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.

3 Myocardial Infarction (Acute Coronary Syndromes)

Description

The acute coronary syndromes encompass a spectrum of unstable coronary artery disease from unstable angina to transmural myocardial infarction. All have a common aetiology in the formation of thrombus on an inflamed, torn or ruptured atheromatous plaque. The principles behind the presentation, investigation and management of these syndromes are similar with important distinctions depending on the category of acute coronary syndrome.

Aetiology

MI is 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).
- Can follow plaque fissure (see section on Angina) or coronary artery spasm.
- Extent of necrosis is limited by the degree of collateral blood supply - better developed after a history of chronic stable angina, so the infarct is usually then smaller than in a patient with a sudden occlusion.

Incidence

The true incidence of MI is unknown; the distinction between a definite MI and an episode of unstable angina is often only possible retrospectively. There were approximately 150,000 deaths from IHD in the UK in 1995; this represents a quarter of all deaths. More than 1 in 15 men die of IHD before age 65 years – accounting for over a quarter of all premature deaths. Women follow the same trend, but lag men by about 10 years. [28] The Whitehall study showed that the incidence was higher in social classes 4 & 5 than in classes 1, 2 & 3. [29]

1	Professional / executive management
2	Middle management
3	Junior management / skilled manual workers
4	Semi-skilled / unskilled / apprentices
5	Unemployed / long term sick

Diagnosis

Clinical Features

The first sign that a person has suffered a myocardial infarction may be sudden death. 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 ⅓ 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.

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.

Investigations

ECG:

– S-T elevation, convex upwards; T-wave inversion; then pathological Q-wave.
– Reciprocal changes in leads facing the opposite side of the heart from the infarct.
– Positive predictive accuracy of 80%, so a normal ECG does not exclude MI.

Serum enzymes:

– Aspartate aminotransferase - raised for 1 - 4 days.
– Creatine phosphokinase - raised for 2 - 3 days.
– Lactate dehydogenase - 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.
Current definitions and prognosis of acute coronary syndrome according to troponinT concentration.

12hr serum troponin concentration (ug/l)

	<0.01	>0.01 and <1.0	>1.0
BCS definition	ACS with unstable angina	ACS with myocyte necrosis	ACS with clinical myocardial infarction
ESC/ACC definition	Unstable angina	Myocardial infarction	Myocardial infarction
WHO definition	Unstable angina	Unstable angina	Myocardial infarction
30 day mortality	4.5%	10.4%	12.9%
6 month mortality	8.6%	18.7%	19.2%

BCS-British Cardiac society
ESC-European Society of Cardiology
ACC-American College of Cardiology

Other blood tests: -

– Polymorph leucocytosis with raised ESR and CRP.
Echocardiography, radionuclide scintigraphy and coronary angiography: -If doubt remains.

Treatment

The immediate priorities in management are to

- Achieve pain relief

- Prevent early death from a dysrhythmia

- Minimise myocardial damage

- Prevent or treat complications

General Measures

- Myocardial Infarction must be considered a medical emergency

- Hospital admission and transfer to a coronary care unit.

- ECG monitor and intravenous (IV) line.

- Oxygen – 2 - 4 L/min via nasal cannulae (not necessary in uncomplicated MI).

- Bed rest only necessary as long as pain, evidence of arrhythmia or haemodynamic disturbance continue.

IV Opiate Analgesia with Prophylactic Anti-emetic

- Diamorphine 2.5–5mg or morphine sulphate 5–10mg.

- Prochlorperazine 10mg, metoclopramide 10mg or cyclizine 50mg.

- Additional benefit of vasodilatation.

- Reduces likelihood of arrythmias (relaxation) and subendocardial ischaemia (lowered catecholamine levels).

- Short-acting benzodiazepine (e.g. lorazepam 0.5 - 1.0mg) added for anxious patients.

Antiplatelet Therapy

- Early aspirin 300mg, then 75mg daily.
- Reduces all-cause mortality including reinfarction and non-fatal stroke. [28]
- Clopidrogel 75mg added in non-Q wave MI.

Thrombolysis

- Rapid IV administration of a fibrinolytic agent reduces early mortality and improves long-term prognosis, by achieving early reperfusion. [30]
- Few absolute (and only a limited number of relative) contraindications.
- Choice of agent is influenced by cost – accelerated t-PA is superior to streptokinase but prohibitively expensive for routine use.

3.1.1 Anticoagulation

- Subcutaneous or IV low molecular weight heparin, followed by warfarin.
- In patients treated with thrombolysis, formal anticoagulation is now restricted to those with:
- a high risk of, or existing, ventricular mural thrombus (e.g. extensive MI or sustained atrial arrythmias);
- poor left ventricular function (aneurysm or ejection fraction < 40%); cardiogenic shock.

Cardioselective β-blockade

- Lowers MVO_2 by reduced myocardial contractility, pulse rate, BP, cardiac output and work.
- Reduces all - cause mortality, including sudden death and non-fatal reinfarction.
- Atenolol 25 - 50mg daily often used.

IV Nitrates

- In doses sufficient to reduce systolic BP to 100mmHg.
- Benefits as for angina.

[Formatted: Bullets and Numbering]

ACE Inhibitors

- Start within 24 hours.

- Reduce all-cause mortality, including late sudden death and reinfarction.

- Benefit occurs in addition to that from β-blockers used concurrently.

Calcium Channel Blockade

- Diltiazem for those with non-Q wave MI.

- Verapamil for those in whom β-blockers are contraindicated.

HRT

- As with angina, postmenopausal women already on HRT should continue it.

- HRT should not be initiated, since postmenopausal women who started HRT after a recent MI had an increased risk of early cardiac events (death, MI or unstable angina), largely due to an excess of unstable angina.

Other Measures

Anti-arrhythmic agents, diuretics, other vasodilators, magnesium, inotropic or mechanical support, valve replacement DC cardioversion or endocardial pacing may be indicated. Automated internal cardiac defibrillators (ICDs) are being increasingly used. They function as a pacemaker but can also treat ventricular tachyarrythmias and reduce the risk of sudden death in at risk patients.

Primary Angioplasty (with or without Stenting)

- Improves vessel patency and myocardial salvage.

- Reduces reinfarction rate and incidence of cerebral haemorrhage.

- Requires a huge 24-hour resource of personnel and facilities for widespread application. In centres with 24 hour access to a catheter laboratory, early angioplasty is the treatment of choice

Prognosis

ECG Factors

The prognosis is worse for non-Q wave than for Q-wave infarction.
Early, 30-day and late post-MI mortality is related to the territory and extent of the ECG changes:

	30-day mortality	12-month mortality
Limited inferior infarcts	4.5%	6.7%
Widespread anterior infarcts (with bundle branch block)	19.6%	25.6%

Killip Classification

(Killip studied the complications of those surviving the initial acute myocardial infarction event and graded the frequency of complications (%) related to the mortality rate at 30 days post MI [%])

I	No evidence of heart failure	85% [6%]
II	3rd sound and bibasal crackles	13% [17%]
III	Frank pulmonary oedema	1% [38%]
IV	Cardiogenic shock	1% [81%]

Poor Prognostic Factors

- Increasing age

- Female gender

- Extensive infarction (determined by 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)

- Inferior infarction with right ventricular involvement

- Diabetes mellitus

- Previous angina or MI

- Impaired ventricular function (3rd 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.

Rehabilitation and Risk Factor Modification

The World Health Organisation defined cardiac rehabilitation (CR) 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 patients] may, by their own efforts preserve or resume as normal a place as possible in the community'.

- Predischarge counselling by a cardiac rehabilitation nurse.

- Specific advice to correct risk factors.

- Formal rehabilitation programme and group activity to achieve progressive increase in dynamic (isotonic) exercise (e.g. walking, swimming or cycling). Meta-analyses of randomised trials have shown that cardiac rehabilitation reduces mortality by 20 – 25%. [31]

- "Mediterranean diet" with oily fish twice a week [32], and statins if fasting cholesterol over 5 mmol/L.

- Fibrates to reduce triglycerides and cholesterol in combined hyperlipidaemia.

- Post-MI exercise testing to stratify risk, help plan the exercise programme, and determine the need for invasive investigation.

- Primary indication for coronary angiography and revascularisation is symptomatic recurrent ischaemia, intractable arrhythmia or poor treadmill performance (which can be assessed by a simple scoring system).

- Depression is a risk factor for future coronary events .Depressive symptoms following acute coronary events are associated with increased cardiovascular morbidity and impairment in quality of life .Depressed mood should always be assessed and treatment with selective serotonin uptake inhibitors considered in patients with severe depression.

Rehabilitation Programmes

The most recent Cochrane review (2001) of 48 randomised controlled trials found that Cardiac Rehabilitation resulted in a 20% reduction in all cause mortality and a 26% reduction in cardiac mortality at 2-5 years.

Rehabilitation programmes have been shown to improve mobility and perception of health, as well as facilitating the return to work and continued employment of those capable. [33]

At their conception, rehabilitation programmes were no more than physical training programmes. Subsequently the need for psychological rehabilitation has been recognised. It has been suggested that psychological counselling before surgery and during post-op rehabilitation, should be carried out to ease the obsession with early retirement and prevent possible ensuing psychopathological disturbances. [34]

It is important that the patient should not be given conflicting advice with regard to work as uncertainty increases anxiety and results in loss of confidence for physical work. [35] Doctors' views on disabilities and re-employment are derived mainly from medical variables (cardiac status and co-morbidity), whereas the patients' views are based on their overall health status, former job status, job satisfaction, and negative incentives for return to work. [36] There is a need for better doctor education, to prevent placing unnecessary physical restrictions on patients whose hospital course and early evaluations suggest that they are at low risk for recurrence of a cardiac event.

Early discharge followed by prompt rehabilitation has been shown to get a large number of patients back to work more quickly. Haskell et al suggest return to work one week after angioplasty and 7 weeks after CABG. Return to work evaluation can be performed within 5 weeks and exercise testing gives the most useful information. The British Heart Foundation's recommendation for return to work after cardiac illness is that patients should return to work when they feel physically and psychologically able to do so. [37]

Optimum times for asymptomatic, uncomplicated patients are:

- Angioplasty - 1 week for sedentary occupations, one month for strenuous occupations.
- MI - 4-6 weeks.
- CABG - 4-8 weeks.

It is important to establish whether unwarranted fears or illness behaviour motivate a patient who is seeking to leave employment.

In commenting on patients' misconceptions, Bob 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." [35]   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.

Main Disabling Effects

See corresponding section under "Unstable Angina".

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. [20]

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. [37]

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.

Appendix A - Risk Factors for IHD

Reversible 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 arrythmias.

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). [38]

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.

Permanent 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. [39]

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 B - Bruce Protocols

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

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

4 References

1. Merck Manual Professional, Ischaemic Heart Disease

2. British Heart Foundation Statistics. May 2006

3. Joint British Societies' Guidelines on Prevention of Cardiovascular Disease in Clinical Practice. British Cardiac Society, British Hypertension Society, Diabetes UK, Heart UK, Primary Care Cardiovascular Society, The Stroke Association. Heart: 2004, Vol 91, (Suppl V), v1 - v52

4. How to use the coronary risk prediction charts for primary prevention. http//www.patient.co.uk/showdoc/40002451/

5. Merck Manual Professional, Coronary Artery Disease : Angina Pectoris 2005 : chapter 73

6. Gibbons RJ, Abrams J, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for the management of Patients with Chronic Stable Angina). 2002

7. British Heart Foundation prevalence rates and morbidity [cited 13 August 2005].

8. Joint Health Surveys Unit. Health survey for England 1998. The Stationery Office: London, 1999.

9. Gill D, Mayou R, Dawes M, et al. Presentation, management and course of angina and suspected angina in primary care. *J Psychosom Res* 1999;46:349–358.

10. Royal College of General Practitioners, the Office of Population Censuses and Surveys and the Department of Health. *Morbidity statistics from general practice: fourth national study 1991–1992*. HMSO: London, 1995.

11. Gandhi MM, Lampe FC, Wood DA. Incidence, clinical characteristics, and short- term prognosis of angina pectoris. *Br Heart J* 1995;73:193–198.

12. Campeau L. Grading of angina pectoris. Circulation 1976;54(3): 522 – 23

13. SIGN (Scottish Intercollegiate Guidelines Network) 96 Management of stable angina.

14. O'Toole, L. (2005) *Angina (stable).* Clinical Evidence. Volume 14

Formatted: Bullets and Numbering

15. Hulley S, Grady D, Bush T, Furberg C, Herrington D, Riggs B, Vittinghoff E. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) Research Group. *JAMA* 1998; 280: 605-13.

16 Westaby S, Sapsford RN, Bentall HH. Return to work and quality of life after surgery for coronary artery disease. *Br Med J* 1979; 2: 1028-31.

17 British Heart Foundation. *Factfile 10/1997.* By-pass surgery or angioplasty for angina? London, 1997.

18. Brorsson B, Bernstein SJ, Brook RH, Werkö L. Quality of life of patients with chronic stable angina before and four years after coronary revascularisation compared with a normal population. *Heart* 2002; 87: 140-145.

19. Drivers Medical Unit. At a Glance Guide to the Current Medical Standards of Fitness to Drive. DVLA, Swansea, April 2007.

20. Baxter PJ, Petch MC. Chapter 18. Cardiovascular Disorders. *Fitness for Work* (3rd edition, ed Cox RAF, Edwards FC, Palmer K). Oxford University Press, 2000.

21. Julian DG. Chapter 1 Medical back ground to cardiac rehabilitation. *Cardiac Rehabilitation* (ed Jones D, West R). London: BMJ Publishing Group, 1995.

22. Crooq SH, Levine S. *The heart patient recovers.* New York: Human Sciences Press, 1977: 102-6.

23. Grundle MG, Reeves BR, Tate S, Raft D, McLaurin MD. Psychosocial outcomes after coronary artery surgery. *Am J Psychiatry* 1980; 137: 12 1591-1594.

24. Cay E. Chapter 2. Goals of rehabilitation. *Cardiac Rehabilitation* (ed Jones D, West R). London: BMJ Publishing Group, 1995.

25. Broustet JP, Douard H. Chapter 6. Rehabilitation after cardiac surgery. *Cardiac Rehabilitation* (ed Jones D, West R). London: BMJ Publishing Group, 1995.

26. Sobrino J A, Merino JL, et al. "[The return to work after coronary revascularization.]" *Revista Espanola de Cardiologia* 1995; 48: 183-6.

27. Speziale G, Bilotta F, et al. Return to work and quality of life measurement in coronary artery bypass grafting. *Eur J Cardio-Thoracic Surg* 1996; 10: 852-8

28. Gray HH, Dawkins KD, Morgan JM, Simpson IA. Lecture Notes on Cardiology, (4th edition). Chapter 8 – Coronary Heart Disease. Oxford: Blackwell Science, 2002.

29. Rose G, Marmot MG. Social class and coronary heart disease.
    Brit Heart J 1981; 45: 13-19.

30. British Heart Foundation. Factfile 12/1999. Emergency! Bust the Clot!
    Reducing pain to needle time in Acute Myocardial Infarction. London, 1999.

31. British Heart Foundation. Factfile 09/2000. Cardiac Rehabilitation.
    London, 2000.

32. Eccles M, Rousseau N, Adams P, Thomas L. Evidence-based guideline for
    the primary care management of stable angina. *Family Practice* 2001; 18:
    217-222.

33. Engblom E, Korpilahti V et al. Quality of life and return to work 5 years after
    coronary artery bypass surgery. Long-term results of cardiac rehabilitation.
    J. Cardiopulm Rehab 1997; 17: 29-36.

34. Kramer C, Gammerler-Schulte H. Psychosocial predictors for initiating a
    pension procedure after bypass operation. Rehabilitation 1998; 37: 21-7.

35. Lewin B. Chapter 4. Psychological factors in cardiac rehabilitation. Cardiac
    Rehabilitation (ed Jones D, West R). London: BMJ Publishing Group, 1995.

36. Mittag O, Kolenda KD. Return to work after myocardial infarction/coronary
    artery bypass grafting: patients' and physicians' initial viewpoints and
    outcome 12 months later. *Social Science & Medicine*   2001; 52: 1441-50.

37. British Heart Foundation. *Factfile 07/2005.* Return to work after cardiac
    illness. London, 2005.

38. British Heart Foundation. *Factfile 04/2001.* Physical Activity and the Heart:
    An Update. London, 2001.

39. British Heart Foundation. *Factfile 01/1995.* Family history and coronary
    heart disease – and what constitutes a positive history. London, 1995.