



Scottish Intercollegiate Guidelines Network

51

Management of Stable Angina

A national clinical guideline

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April 2001

KEY TO EVIDENCE STATEMENTS AND GRADES OF RECOMMENDATIONS

The definitions of the types of evidence and the grading of recommendations used in this guideline originate from the US Agency for Health Care Policy and Research¹ and are set out in the following tables.

STATEMENTS OF EVIDENCE

Ia	Evidence obtained from meta-analysis of randomised controlled trials.
Ib	Evidence obtained from at least one randomised controlled trial.
IIa	Evidence obtained from at least one well-designed controlled study without randomisation.
IIb	Evidence obtained from at least one other type of well-designed quasi-experimental study.
III	Evidence obtained from well-designed non-experimental descriptive studies, such as comparative studies, correlation studies and case studies.
IV	Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities.

GRADES OF RECOMMENDATIONS

A	Requires at least one randomised controlled trial as part of a body of literature of overall good quality and consistency addressing the specific recommendation. <i>(Evidence levels Ia, Ib)</i>
B	Requires the availability of well conducted clinical studies but no randomised clinical trials on the topic of recommendation. <i>(Evidence levels IIa, IIb, III)</i>
C	Requires evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities. Indicates an absence of directly applicable clinical studies of good quality. <i>(Evidence level IV)</i>

GOOD PRACTICE POINTS

<input checked="" type="checkbox"/>	Recommended best practice based on the clinical experience of the guideline development group.
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1 Introduction

1.1 WHY IS ANGINA IMPORTANT?

Coronary heart disease (CHD) represents the most common single cause of adult death in the United Kingdom after all causes of cancer taken together. In Scotland, mortality rates from CHD compare unfavourably with other countries. Our 13,400 annual CHD deaths account for 25% and 20% respectively of all male and female fatalities, almost one in six being below the age of 65.²

The prevalence of men and women with angina or myocardial infarction (MI) is shown in Table 1. It is positively associated with socio-economic deprivation and rises with age. The incidence is hard to measure but is probably around 3.5% per year in adult men and around 3% per year in adult women.³

Table 1

PREVALENCE OF ANGINA AND MYOCARDIAL INFARCTION (MI) IN SCOTLAND, 1998

		Age Group					
		15-24	25-34	35-44	45-54	55-64	65-74
MEN	Angina (%)	0	0	0.8	4.4	13.7	20.6
	MI (%)	0	0.1	0.2	4.0	9.4	16.8
	Either (%)	0	0.1	0.8	6.6	16.1	27.3
WOMEN	Angina	0	0.2	0.4	3.6	10.3	15.4
	MI	0	0	0.2	1.0	4.0	7.4
	Either	0	0.2	0.6	3.8	10.7	17.3

The majority of those with CHD are managed in the community. Continuous Morbidity Recording data shows that the average GP has around 45 consultations per thousand practice population per year with patients where the first reason is angina.⁴ However, this does not account for consultations for the management of risk factors nor for nurse contacts. The burden on primary care should be considered in the context of the prevalence of the disease and the high prevalence of risk factors in the CHD population.

In order to identify individuals at particular risk, and those requiring further assessment in the form of coronary angiography with a view to a revascularisation procedure, angina demands early recognition, diagnostic investigation and appropriate treatment.

1.2 THE NEED FOR A GUIDELINE

Several baseline surveys and audits of performance have reported a considerable gap between normal practice and optimum secondary prevention with regard to aspirin, lipids, blood pressure, smoking, diet, weight and exercise for all patients with coronary heart disease.⁵⁻¹⁰ This appears to be particularly true for patients with angina alone (e.g. 53% of angina only patients on aspirin compared to 85% with recent myocardial infarction).⁹ Overall, cholesterol concentrations have been reported as outwith optimal limits for 71-84% of patients and blood pressure levels for 17-37%.^{5,7,9} With regard to lifestyle, two thirds of patients are overweight, most are physically inactive and consume poor diets and nearly 20% continue to smoke.¹¹⁻¹³

1.3 ANGINA IS A SYMPTOM, NOT A DIAGNOSIS

Angina most commonly takes the form of chest discomfort provoked by effort or emotion and relieved by rest. The index symptom may however affect a site of radiation, occurring for example as isolated throat tightness or arm heaviness. Exertional breathlessness may likewise represent an anginal equivalent. When severe, angina may be accompanied by autonomic features such as fear, sweating and nausea. These symptoms are clearly not specific, and it may be difficult to distinguish patients with gastro-oesophageal reflux disease, musculoskeletal discomfort or pulmonary disease. The coronary risk factor profile may be helpful in this regard, as chest discomfort is more likely to represent coronary artery disease in an individual with two or more existing risk factors, e.g. cigarette smoking, hypertension, diabetes mellitus, hypercholesterolaemia, a family history of premature coronary artery disease, or the presence of other acquired vascular disease.¹⁴

The application of the term angina, implying the presence of coronary artery disease, is solely an interpretation of the history given by the patient. Accordingly, if the clinical impression is of angina, due consideration should be given to further investigation in order to establish the likelihood and extent of underlying coronary disease. Potential associated cardiac and cardiovascular conditions such as valvular heart disease and hypertension should be identified, as these present important implications for both the investigation and management of angina.

Angina usually reflects atherosclerotic coronary artery disease. However, individuals with aortic stenosis, hypertensive heart disease and hypertrophic cardiomyopathy may volunteer typical symptoms in the absence of coronary disease. Also, there are patients who experience recurrent angina despite being demonstrated to have a structurally normal heart with angiographically normal coronary arteries.

1.4 THE SCOPE OF THE GUIDELINE: STABLE vs UNSTABLE ANGINA

This guideline addresses the management of stable angina pectoris. The information and advice given does not apply to the patient with unstable angina describing prolonged episodes of severe angina, increasingly frequent angina, or angina at rest. Also, angina recurring early following initially successful coronary artery bypass grafting (CABG), or percutaneous transluminal coronary angioplasty (PTCA), falls outwith the context of stable angina, and should prompt early specialist referral.

The focus of this guideline is therefore on angina presenting as a stable symptom for the first time, and the management of angina in primary care. It is assumed that the underlying diagnosis is coronary artery disease. Areas covered include investigation, risk factor identification and management, drug treatment, and referral to secondary care. The recommendations are for primary and secondary health care professionals and apply to adult patients attending general practice or hospital clinics. There is no upper age limit to patients to whom this guideline may apply.

The guideline development group assumes that health care professionals will use general medical knowledge and clinical judgement in applying the principles and specific recommendations of this document to the management of individual patients. Recommendations may not be appropriate for use in all circumstances; all patients should continue to be considered as individuals and many may fall outside the scope of this guideline. This may be particularly true in those of advanced age or with multiple medical problems. Decisions to adopt any particular recommendation from the national guideline locally can only be made in the light of available resources.

2 Investigation and referral

2.1 INITIAL ASSESSMENT

The history and examination and investigation of any patient with angina will necessarily be comprehensive including the patient's understanding of symptoms and expectations of treatment. It may include the following points of detail in patients with suspected or known angina:

History:

- precipitants of anginal attacks
- stability of symptoms
- smoking history
- occupations
- assessment of the intensity, length and regularity of exercise
- basic dietary assessment
- alcohol intake
- drug history
- family history.

Examination:

- weight and height (to allow calculation of BMI) or waist / hip ratio
- blood pressure
- presence of murmurs, especially that of aortic stenosis
- evidence of hyperlipidaemia
- evidence of peripheral vascular disease and carotid bruits (especially in diabetes).

It is important that precipitating factors are identified and discussed with the patient so that anginal pain may be prevented by the appropriate use of short-acting nitrates.

- Factors that precipitate angina should be enquired about and discussed with the patient.

2.2 INITIAL TESTS

The initial tests that should be considered in a patient with angina represent a consensus of good practice.

- Patients with suspected angina should have:
 - haemoglobin measured to identify those with an underlying anaemia
 - fasting blood glucose measured to identify those with previously undiagnosed diabetes mellitus
 - a full lipid profile.

Thyroid function tests are frequently recommended in text books but the guideline development group, after taking expert advice, decided that there was no justification for recommending *routine* assessment of thyroid function. This should be performed in cases where there is clinical suspicion of thyroid disease.

2.3 RESTING 12- LEAD ELECTROCARDIOGRAM (ECG)

A resting 12-lead ECG is used to provide information on rhythm, presence of heart block, previous myocardial infarction and myocardial hypertrophy and ischaemia.

The presence of an abnormal ECG supports a clinical diagnosis of coronary artery disease.¹⁵ ST/T abnormalities have been correlated with abnormalities of left ventricular function and left anterior descending artery stenosis. QRS abnormalities have been associated with abnormal findings on angiography.

III

An abnormal ECG also identifies a patient at higher risk of suffering new cardiac events in the subsequent year. However, a normal ECG *does not exclude* coronary artery disease. In a review of 109 patients who had normal ECGs, 39% still had cardiac pain and 90% of those subjected to angiography showed significant coronary artery disease.¹⁵

III

B Patients with angina should have a resting 12-lead ECG.

B Patients with angina and an abnormal resting 12-lead ECG should be considered for urgent referral and investigation.

It is important to consider where an ECG is obtained, the equipment and its operation and who is to interpret the ECG. Many general practitioners will have their own 12-lead ECG machine and it is the responsibility of the individual doctor to decide whether the ECG is of sufficient quality to enable satisfactory interpretation and whether they have sufficient skills to undertake this. Studies in general practice have demonstrated that most general practitioners are capable of deciding whether an ECG is abnormal or not.¹⁶ However, some general practitioners may need the reassurance of a hospital-based service, for example, faxing an ECG to a cardiologist, referral to an open access ECG clinic, or referral to a rapid access chest pain clinic.

2.4 FURTHER INVESTIGATION

The aim of further investigation is to provide diagnostic and prognostic information and to identify patients who may benefit from further intervention.

Exercise tolerance testing (ETT) has been shown to be of value in assessing prognosis of patients with coronary artery disease.¹⁷⁻²⁰ An ETT is also helpful in patients at high risk of CHD, where a positive test can provide useful prognostic information.¹⁷

III

Exercise and stress test indications of adverse prognosis:

- Poor maximal exercise capacity^{17, 18, 21}
- Limited systolic blood pressure response i.e. fall or no rise from baseline^{22, 23}
- ≥ 1 mm ST depression during stage 2 or less;¹⁸
or ≥ 2 mm ST depression at any time.

An ETT is a low risk investigation even in patients with known ischaemic heart disease, but serious complications occur in 2-4 per 1,000 tests. Death may occur at a rate of 1-5 per 10,000 tests.²⁵

III

An ETT is a poor diagnostic test in low-risk populations. The CASS study concluded that the value of the test is limited in a heterogeneous population of patients with angina and that exercise testing should not be regarded as a screening test.^{21, 26} An exercise ECG was best performed with patients on treatment to improve the specificity of the test and to avoid angiography in those who are well controlled on medical treatment.²⁷

IIa & III

B Patients with angina should be referred for exercise tolerance testing for risk stratification.

B Patients having an exercise tolerance test to assess prognosis should have the test while taking their normal medication.

- Patients should *not* be referred for an ETT if:
 - they are on maximal medical treatment and still have angina symptoms
 - the diagnosis of CHD is unlikely (these patients should be referred to a cardiologist: see section 2.5)

- they are physically incapable of performing the test
- they may have aortic stenosis or cardiomyopathy
- results of stress testing would not affect management.
- All patients should have the ETT test and its implications explained to them so they may have the opportunity of declining the test.
- Minimising delay in carrying out exercise tolerance testing will keep patient anxiety to a minimum.

Referral for exercise tolerance testing will be influenced by local arrangements. Open access facilities are used in some areas and have been shown to be used appropriately by general practitioners especially where a local guideline is available. Other areas may have open access chest pain clinics where ETT is available.²⁸ Otherwise ETT is available generally through referral to a cardiologist or general physician. There is no evidence to suggest which of these approaches is in the best interests of patients or makes best use of resources.

If conventional exercise testing is precluded by immobility, pharmacological stress myocardial perfusion imaging is a valuable alternative, but only available through specialist referral in certain centres.²⁹

2.5 REFERRAL TO A CARDIOLOGIST

The decision to refer a patient with suspected or confirmed angina, secondary to coronary artery disease, is an important aspect of management. A number of clinical factors should lead the general practitioner to refer a patient with angina because these patients might benefit from early investigation or revascularisation. These are:

- patients who appear to have had a previous MI on their initial ECG or other abnormality that the general practitioner considers significant
- patients who fail to respond to medical treatment having already had an ETT
- patients who have an ejection systolic murmur suggesting aortic stenosis.

In addition, patients with the following symptoms may have unstable angina and should be considered for urgent referral:

- pain on minimal exertion
- pain at rest which may occur at night
- angina which appears to be progressing rapidly despite increasing medical treatment.

Further clinical reasons to refer a patient to a cardiologist include:

- to confirm or refute a diagnosis in patients with uncertain or atypical symptoms
- to identify whether they would fall into a group that might benefit from further investigation and treatment
- to advise on the management of an individual patient and particularly where the patient has not responded to the modification of risk factors and treatment outline recommended in this guideline
- the presence of a number of adverse risk factors or a strong family history
- patient preference for early referral
- problems with employment, life insurance or unacceptable interference with lifestyle
- significant co-morbidity, for example, diabetes mellitus.

Note: *not all patients need to be referred. Some patients may not wish to be referred.*

- It is most important not to delay treatment as outlined in this guideline while awaiting referral.
- Hospital outpatient clinics should discharge patients who are at low risk or who are otherwise not suitable for further investigations back to general practitioners. Clinics should provide a clear management plan to enable general practitioners to manage their patients' symptoms and advise about the point at which further referral is appropriate.

3 Management of risk factors

The guideline covers the management of seven modifiable risk factors for patients with angina:

- smoking
- hypertension
- dietary factors
- overweight and obesity
- cholesterol
- physical activity
- excess alcohol consumption.

Although not a modifiable risk factor, *per se*, those angina sufferers who are also diabetic are at increased risk of developing CHD and are therefore discussed in this section.

It is important to encourage the family, and not only the individual, to modify their lifestyle, as this is more likely to be effective.³⁰

This section specifically excludes consideration of the management of anxiety, panic attacks, depression and their associated behaviours, vocational and other psychosocial issues. These would normally fall under the umbrella of rehabilitation but they cannot be divorced from other aspects of risk factor modification and secondary prevention. They will be addressed in greater detail in the forthcoming Scottish Needs Assessment Programme (SNAP) report and SIGN guideline on cardiac rehabilitation.

3.1 SMOKING

Most of the papers which considered smoking as a risk factor, and the benefits of smoking cessation, consider coronary heart disease or coronary heart disease risk factors rather than stable angina specifically. In observational studies, cigarette smoking has been shown to be strongly associated with the likelihood of developing a heart attack, although the relationship with angina is not as strong.³¹⁻³³ The risk is dose related in both men and women.³⁴⁻³⁶ Smoking amplifies the effect of other risk factors, thereby promoting acute cardiovascular events.³⁷ Events related to thrombus formation, plaque instability and arrhythmia are all influenced by cigarette smoking.

One observational study in those with angina indicates the benefits of smoking cessation.³⁸ Those who continued had around five times the risk of a coronary event over ten years than those who quit. The benefit falls with increasing age. Observational studies also show that patients post MI who continue to smoke are at an increased risk of death of around 50% over five years compared to those who stop.^{32, 39} Patients who have had CABG and who smoke have also been shown to have a reduced survival⁴⁰ and an increase in non-fatal MI and angina relative to non-smokers. Thus, there is observational evidence in various groups of CHD patients that smoking cessation is beneficial.

There is debate about how quickly risk decreases following smoking cessation. The British Doctor's Study carried out in a general population showed a halving of risk at 2-3 years and a non-smoker's risk at ten years.⁴¹ The British Regional Heart Study, also carried out in a general population, showed a slower attenuation of risk.⁴² Case control studies in men following MI have shown that there is a substantial (around 50%) reduction in risk of death over three years among those who stop smoking which seems to increase more slowly over time.⁴³ In women following MI, the risk in those who stop smoking becomes similar to those who had never smoked after three years and the decrease in risk is most marked early in the cessation period.⁴⁴

B All patients with angina who smoke should be advised to stop.

III

Tobacco smoking is a complex behaviour, with pharmacological, social and psychological components. Nicotine addiction has been compared with addiction to heroin or cocaine.^{45, 46} The Government White Paper “*Smoking Kills*” has been underpinned by the allocation of resources to each Health Board to provide a GP referral service and a smoker’s self-referral service for specialist counselling, advice and support. *Advice is available from the Health Education Board for Scotland (HEBS) Smokeline: Tel. 0800 848 484.*

Several systematic reviews of RCTs and observational studies have shown good evidence of effectiveness for: brief advice from a doctor, structured interventions from nurses, individual group counselling and individualised self-help materials. There is insufficient evidence of effectiveness for: aversive conditioning, acupuncture, hypnotherapy, mecamylamine or exercise.⁴⁷⁻⁴⁹

Ia

The antidepressants bupropion and mirtazapine increased quit rates in a small number of trials. The two bupropion trials, however, involved only heavy smokers with behavioural support.⁵⁰

A Brief advice from a health professional, tailored self-help materials, individual and group counselling and antidepressants (with behavioural support) can increase rates of smoking cessation.

Nicotine replacement therapy (NRT) in addition to brief advice from a health professional can double quit rates in those motivated to quit⁵¹⁻⁵³ but there is no increased quit rate associated with use for longer than eight weeks. NRT has been shown to be safe in those with CHD.^{54, 55}

Ib

B Nicotine replacement therapy is recommended as part of a smoking cessation program in patients with angina.

Extrapolated from evidence in the general population

Many manufacturers of NRT emphasise caution in the prescription of these products to patients with known cardiovascular disease. Nicotine may contribute to cardiovascular disease, presumably by hemodynamic consequences of sympathetic neural stimulation and systemic catecholamine release. However, analyses have now documented the lack of association between NRT and acute cardiovascular events and the risks of NRT for smokers, even for those with underlying cardiovascular disease, are small and are substantially outweighed by the potential benefits of smoking cessation.^{56, 57}

Ib

3.2 HYPERTENSION

Epidemiological data indicate that continued hypertension following the onset of CHD increases risk of a cardiac event and that reduction of blood pressure reduces risk.^{58, 59}

III

The British Hypertension Society (BHS) guidelines consider that patients with stable angina all have high CHD risk, and the threshold for starting antihypertensive treatment is systolic blood pressure averaging ≥ 140 or diastolic blood pressure averaging ≥ 90 .⁶⁰ The BHS sets the target blood pressure once antihypertensive treatment is started at the same level for angina patients as for others who do not have diabetes, namely optimal blood pressure $< 140/85$, and audit standard blood pressure $< 150/90$.

IV

The literature search failed to identify any RCTs of blood pressure reduction specifically in those with CHD. However a meta-analysis of 15 RCTs in subjects over 60 years old, some of which had a proportion of patients with CHD, showed the benefits were substantial. No differential treatment effects could be established for groups with different risk factors, pre-existing cardiovascular disease or competing co-morbidities.⁶¹

Ia

Lifestyle modification should be an integral part of management of hypertension. There is evidence that increasing exercise,⁶² reducing weight,⁶³ alcohol consumption⁶³ and sodium consumption⁶³ are all effective at reducing blood pressure.

Ia & Ib

B All those with angina should have blood pressure assessed and managed.

The management of hypertension is considered in the revised British Hypertension Society guidelines⁶⁰ and the SIGN guideline on Hypertension in Older People.⁶⁵

3.3 DIETARY FACTORS

Diet is an important contributor to many risk factors including high blood pressure, obesity and impaired glucose tolerance. There is evidence in the post-MI patient that dietary change may have a benefit beyond changes in lipid levels. Increasing the consumption of oil-rich fish, eating a “Mediterranean” diet (rich in fruits, vegetables, cereals and fish and low in saturated fat) and increasing consumption of fruit, vegetables, nuts and grains can be successful in reducing the risk of future cardiac events and in managing obesity.⁶⁶⁻⁶⁸

Ib

B Patients with angina should modify their diet in line with healthy eating advice:

- increase fruit and vegetable consumption to five portions per day
- increase consumption of oil-rich fish to three portions per week
- decrease total fat consumption, increasing the proportion of monounsaturated fat
- increase starchy food intake and reduce sugary food intake.

Extrapolated from evidence in post MI patients

Lifestyle measures should continue beyond three months, irrespective of the need for pharmacological treatment.

Dietary change in CHD patients is discussed more fully in the SIGN guideline on Secondary Prevention of Coronary Heart Disease following Myocardial Infarction⁸⁰ and in the Scottish Diet Report.⁶⁹ Further help and patient literature can be obtained from “Towards Healthy Eating” – a HEBS resource pack for primary care professionals. An example diet sheet produced by dietitians in Scotland is included in the SIGN guideline on lipids and the primary prevention of CHD and is also available on the SIGN website.⁷⁰ A detailed discussion of strategies for implementing healthier eating may be found in Eating for Health – A Diet Action Plan for Scotland.⁷¹

3.4 OBESITY AND OVERWEIGHT

Obesity and overweight have an adverse influence on many coronary risk factors including blood pressure, cholesterol, triglycerides and glucose tolerance. In addition obesity increases myocardial oxygen demand. The Scottish Heart Health Study follow-up over eight years showed a two-fold increase in risk of developing CHD between the highest quintile of BMI and the lowest in men.⁷² There was a U-shaped curve for women, with the lowest risk for those with BMI 22-26 and a 40% increase in risk for those with BMI > 29. However, observational studies show an inverse relationship between re-infarction and BMI in those following MI.

IIb

C All patients with CHD should be actively encouraged to lose weight towards BMI < 25.

Extrapolated from evidence in the general population

3.5 CHOLESTEROL

The Scandinavian Simvastatin Survival Study (4S) demonstrated that cholesterol lowering with simvastatin significantly reduced cardiovascular and all cause mortality in patients with angina, not only post-myocardial infarction, and with a total cholesterol > 5.4 mmol/l.⁷³ Other RCTs of statin therapy in the secondary prevention of CHD have not specifically included patients with angina but all showed a reduction in relative risk of cardiac events irrespective of the starting level.^{74, 75}

Ib

Angiographic studies have also shown the benefit of lipid lowering in terms of plaque stabilisation, regression and less progression. These trends were also linked to a reduction in coronary events⁷⁶⁻⁷⁸

The guideline development group recommends that patients with angina should have their cholesterol measured and there is sufficient evidence to warrant cholesterol lowering in patients with symptomatic angina, as in patients after a myocardial infarction.

The Joint British recommendations on Prevention of Coronary Heart Disease in Clinical Practice,⁷⁹ the SIGN guideline on secondary prevention of CHD following MI⁸⁰ and the North of England guideline⁸¹ all recommend that patients who have total cholesterol of ≥ 5.0 mmol/l or low density lipoprotein (LDL) ≥ 3.0 mmol/l should be offered lipid lowering therapy.

IV

C All patients with angina should have their cholesterol level measured.
If total cholesterol (TC) is ≥ 5.0 mmol/l:

C appropriate dietary measures should be recommended and a random non-fasting cholesterol measurement repeated after 6-12 weeks.

A If required, drug therapy should then be initiated to reduce TC to < 5.0 mmol/l.

The subsequent management of the patient with hypercholesterolaemia is beyond the scope of this guideline. (See the *British Hyperlipidaemia Society guideline on management of hyperlipidaemia*.⁸²)

3.6 PHYSICAL ACTIVITY

Light to moderate physical activity in healthy adults has been shown to reduce the risk of all cause mortality and morbidity.^{11, 78, 83} Observational studies show that those with higher physical activity levels are less likely to develop CHD than those who are more sedentary, and that those who increase their physical activity decrease their chance of developing CHD irrespective of their starting level.

III

Multiple RCTs in patients with chronic stable angina have consistently shown a significant improvement in exercise tolerance in those in the exercise group.^{12, 84-86} The number of RCTs carried out to demonstrate an improvement in symptomatology is small and the numbers involved are also small. Two of three studies^{84, 87} showed a reduction in symptoms and one did not,⁸⁸ although each showed a reduction in the objective measurements of ischaemia.

Ib

Increasing exercise levels can also be part of a weight control programme and increased physical activity reduces blood pressure (see section 3.2).

The safety of exercise in this group of patients is well reviewed in an American guideline which recommends risk stratification to inform decisions about the type and intensity of exercise to be recommended and the level of supervision required.⁸⁹

IV

The best way to provide an exercise programme to maximise perseverance with exercise over time is less well understood but the best effects in terms of reduced mortality are seen when exercise is offered as part of a multifactor lifestyle intervention programme.⁸⁹ Continued compliance is improved when there is attention to interpersonal relationships between patient and provider⁹⁰ and increasing patient involvement in decision making.⁹¹⁻⁹³

III

B All those with CHD should be encouraged to increase their aerobic exercise levels within the limits set by their disease state.

B Patients should be involved in decisions about exercise in order to improve perseverance.

3.7 EXCESS ALCOHOL CONSUMPTION

The risk of CHD is up to one third less in those who drink alcohol than in abstainers.⁹⁴ There is no increase in effect with alcohol consumption over three units per day. However all cause mortality increases with consumption greater than three units per day.⁹⁴ The subject is well reviewed in a report of a working group of the Royal Colleges of Physicians, Psychiatrists and General Practitioners.⁹⁵ Alcohol depresses myocardial function.⁹⁶ Chronic alcohol consumption

III

is associated with raised blood pressure and reducing consumption lowers blood pressure (see *section 3.2*). There is no indication to encourage those who do not consume alcohol to do so.

Brief interventions having as their core, assessment of intake, information about the harms and hazards of excessive consumption, and clear advice for the patient have been shown to reduce alcohol intake by over 20% in the group of people with raised alcohol consumption.⁹⁷

III

B Patients with CHD who consume alcohol should be encouraged to limit their consumption to three units per day for men and two units per day for women.

3.8 DIABETES

Diabetes increases the risk for CHD 2-4 fold.⁹⁸⁻¹⁰⁰ In type 2 diabetes (which represents over 90% of patients with diabetes) the increased risk is apparent at the time of diagnosis and is independent of the duration of the diagnosed diabetes.¹⁰¹⁻¹⁰³ The increase in risk is greater for women than men.^{98, 104} Risk rises with increasing glucose levels and this extends well below conventional levels for the diagnosis of diabetes.¹⁰⁵⁻¹⁰⁷

III

C Diabetic patients with angina should make efforts to optimise glycaemic control.

Further information can be found in the SIGN guideline on the management of diabetic cardiovascular disease,¹⁰⁸ which is currently under review.

4 Drug treatment

Drug therapy in patients with stable angina falls into three main categories:

- secondary prophylactic treatment
- short term control of angina symptoms
- long term prevention of angina symptoms.

The range of drugs, dosages, contraindications and side effects are described in the British National Formulary (BNF). All recommendations for treatment in this guideline apply only in the absence of recognised contraindications, side effects and interactions as documented in the BNF.

It is important that health professionals endeavour to ensure that patients comply with treatment and that any side effects they may experience are enquired about and documented. The guideline development group believes that within any drug class, patients should, in principle, be treated with the cheapest preparation that controls their symptoms, that they can comply with, and that they can tolerate.

4.1 SECONDARY PROPHYLACTIC TREATMENT

4.1.1 ASPIRIN THERAPY

Aspirin is used as an antiplatelet drug for the secondary prevention of vascular events in patients with angina. The aim of secondary prophylactic treatment is to minimise the patient's risk of subsequent vascular events. The use of aspirin in high-risk groups lowers the risk of subsequent vascular events.

The Swedish Angina Pectoris Aspirin Trial (SAPAT) randomised 2,035 patients with a history of at least one month of exertional angina to receive 75 mg aspirin daily. The results demonstrated a relative risk reduction in primary endpoints (non-fatal MI, fatal MI or sudden death) in the treatment group of 34% after 72 months. Absolute risk reduction was 4% (95% confidence intervals 1-7%) All patients were treated with β -blockers. A subgroup analysis of the US Physicians' Health Study showed that aspirin therapy greatly reduced the risk of first MI in patients with stable angina ($p < 0.001$).¹¹⁰

Ib

The Antiplatelet Trialists Collaboration's meta-analysis demonstrated benefits for patients with coronary heart disease being treated with aspirin¹¹¹ and included patients with stable angina. The rates of myocardial infarction, stroke and vascular death in the antiplatelet group were 10.6% compared with 14.4% in the control group, an absolute reduction of 3.8% in the risk of a subsequent vascular event. There was a similar picture from the other three high-risk groups: prior myocardial infarction; prior stroke or transient ischaemic attack; other CHD events (unstable angina, stable angina, post-coronary artery bypass graft).

Ia

The aspirin dose used in these trials ranged from 75-300 mg daily, however a more recent meta-analysis showed no difference in risk reduction across doses of aspirin up to 500 mg daily.¹¹² There is no evidence that medium doses (< 500 mg daily) in excess of 75 mg are likely to provide greater benefits in patients with stable angina.

A Patients with stable angina should be treated with aspirin 75 mg daily.

The most common side effect related to aspirin is dyspepsia. Non-statistically significant increases in the incidence of gastro-intestinal bleeding have been reported with increasing doses of aspirin in some studies.¹¹³ Enteric-coated preparations of aspirin at doses greater than 75 mg have been shown, on endoscopy or stool analysis, to produce fewer gastro-intestinal erosions in healthy volunteers.¹¹⁴⁻¹¹⁷ One larger clinical study, however, appeared to show that at average daily doses of 325 mg or less, the risks of gastric or duodenal bleeding with standard and enteric-coated aspirin were the same.¹¹⁸ Enteric-coated preparations are also more expensive than standard preparations of aspirin.

Persistent dyspepsia may be treated with antacids, H₂ antagonists or proton pump inhibitors, although only misoprostil has been demonstrated to be effective in the primary prevention of nonsteroidal anti-inflammatory drug-induced gastric ulceration.¹¹⁹

Ia

True aspirin allergy manifests itself as angio-oedema or bronchospasm, but is rare. An alternative antiplatelet agent in these circumstances is clopidogrel, which prevents platelet aggregation by blocking the adenosine diphosphate (ADP) receptor. Clopidogrel is an effective¹²⁰, but more costly alternative to aspirin and should therefore be reserved for cases of allergy or intolerance to low-dose aspirin.

Ib

A In the event of true aspirin intolerance or allergy, clopidogrel 75 mg daily should be considered.

4.1.2 STATINS

See section 3.4

4.1.3 ACE INHIBITORS

Data from the Heart Outcomes Prevention Evaluation (HOPE)¹²¹ study which used ramipril 10 mg daily suggests that there may be additional benefits of ACE inhibition of patients who are considered at high risk of cardiovascular events, over the age of 55 years **and** with a history of CHD. Further trial evidence is awaited to clarify the role of this class of drugs in this patient group.

Ib

A sub-study of low dose (2.5 mg daily) versus high dose (10 mg daily) has yet to be reported.

4.2 SHORT TERM CONTROL OF ANGINA SYMPTOMS

Sublingual glyceryl trinitrate (GTN) is an acceptable treatment of short term symptom control where patients have been educated on its appropriate use. Key points for discussion with patients include:

- why it has been prescribed
- how to use the product (spray or tablets)
- when to use GTN to treat chest pain and to prevent pain if it can be anticipated
- likely side effects and their management
- what to do if pain persists after three doses over 15 minutes
- the need for backup supplies (GTN may be purchased from pharmacies).

All patients with symptomatic coronary heart disease should be prescribed sublingual GTN and be educated in its use.

4.3 LONG TERM PREVENTION OF ANGINA SYMPTOMS

Four groups of drugs are used in the long term prevention of chronic stable angina symptoms:

- β-blockers
- calcium channel blockers
- long-acting nitrates
- potassium channel opening drugs.

4.3.1 β-BLOCKERS

β-blockers have been shown to be as effective in the prevention of long-term angina symptoms as the other available classes of drugs. Patients receiving β-blockers (either singly or in combination therapy) benefited equally¹²²⁻¹²⁴ or significantly more¹²⁵⁻¹²⁷ in terms of anginal relief than patients on alternative monotherapies.

Ia & Ib

In addition, β-blockade in high risk patients reduces cardiovascular mortality and morbidity. Supporting evidence is drawn from post-myocardial infarction trials and trials of patients taking

Ia & Ib

β -blockers for any reason.^{128, 129} Long term β -blockade remains an effective and well-tolerated treatment that reduces mortality and morbidity in patients after myocardial infarction. Patients who have had a myocardial infarction or currently have angina and are given β -blockers have a lower rate of mortality and morbidity.¹³⁰

Ia & Ib

There is conflicting evidence on whether β -blockers are better tolerated than calcium channel blockers. One meta-analysis reported that calcium channel blockers were associated with a greater number of adverse events compared with β -blockers in trials of patients with stable angina.¹²² Other papers have indicated frequent adverse effects with β -blockers and attributed lower adverse-effect profiles to calcium channel blockers.^{131, 132}

B Patients who require regular symptomatic treatment should be treated initially with a β -blocker (unless specifically contraindicated).

Acute withdrawal of β -blockers has been associated with an increase in coronary events in the months after stopping treatment in hypertensive patients.¹³³

IIb

B Patients should be warned not to stop β -blockers suddenly or allow them to run out.

4.3.2 MONOTHERAPY IN PATIENTS INTOLERANT OF β -BLOCKERS

Several studies in patients with angina have shown calcium channel blockers, long-acting nitrates and potassium channel opening drugs to be effective first line agents compared with placebo.¹³⁴⁻¹³⁸ However, differences in patient selection, study design and drug dosages all prevent critical comparisons being made and none of these studies is large enough to warrant a definitive conclusion that any single class of drug is more effective.¹²⁴

Rate-limiting calcium channel blockers (e.g. diltiazem or verapamil) are considered to be safer than short-acting dihydropyridines, which may lead to tachycardia in some patients. There is limited evidence of the effectiveness of verapamil in reducing mortality and morbidity following MI: subgroup analysis in two trials indicated a marginal benefit in patients with normal left ventricular function.^{139, 140}

Ib

C Patients intolerant of β -blockers and who show no left ventricular systolic dysfunction should be treated with a rate-limiting calcium channel blocker, a long-acting dihydropyridine, a nitrate or a potassium channel opening agent.

Extrapolated from evidence in post MI patients

4.3.3 APPROPRIATE USE OF NITRATES

Oral nitrates are effective as long term symptomatic therapy when used as a sustained release preparation, dosage three times daily or as an eccentric twice daily preparation (e.g. 8am and 4pm or 2pm and 10pm).^{137, 141, 142} Both isosorbide dinitrate and mononitrate have been shown in controlled trials to be superior to placebo in controlling symptomatic angina.^{137, 143} Limited comparison with a sustained release preparation of verapamil found no difference in efficacy, provided the nitrate is used in an eccentric dosage, allowing a nitrate-free period of 6-8 hours to obviate nitrate tolerance.¹⁴⁴

Ib

The dinitrate is metabolised to mononitrate, and as a wider variety of mononitrate preparations are available, the guideline development group considers that isosorbide mononitrate in an eccentric twice daily dosage is the preferred regimen.

There is conflicting evidence as to the effectiveness of nitrate patches in the treatment of angina¹⁴⁴⁻¹⁴⁷ and they are considerably more expensive than other therapies. Comparison of patches with placebo has not clearly demonstrated effectiveness and continuous patches have been shown to be ineffective.¹⁴⁸ High dose patches are more effective than lower dose patches when used with a patch free interval.¹⁴⁹

A Oral nitrates can be used as a satisfactory monotherapy, provided they are used in a way which avoids nitrate tolerance (e.g. in an eccentric dosage).

4.3.4 CHOOSING A SECOND DRUG

There is evidence from randomised controlled trials to support the use of isosorbide mononitrate or a calcium channel blocker as second line agent to a β -blocker.¹⁵⁰⁻¹⁵⁵ Although one study demonstrated the effectiveness of adding diltiazem to a β -blocker, the cautions cited in the BNF should be observed.¹⁵⁶

Ib

A In patients taking β -blockers, add isosorbide mononitrate, a long-acting dihydropyridine or diltiazem.

There is no clear evidence to support recommendations of other drug combinations, however the use of more than one drug from any one therapeutic class should be avoided.

4.3.5 CHOOSING A THIRD DRUG

Studies on the effects of triple dose combinations do not demonstrate any significant advantage over dual therapy.¹⁵⁷

Consideration should be given to referral to a cardiologist on initiation of treatment, if the patient is perceived to be at increased risk, (see section 2.5) if at any stage medical treatment fails to control symptoms or if these symptoms limit the patients' desired activities.

4.4 ANGINA IN PATIENTS WITH CHRONIC HEART FAILURE OR ATRIAL FIBRILLATION

Some patients with angina will have chronic heart failure and, among these, atrial fibrillation is common. See the SIGN guideline on *Diagnosis and Treatment of Heart Failure due to Left Ventricular Systolic Dysfunction*.

5 Implementation and audit

5.1 STATEMENT OF INTENT

This guideline is not intended to be construed or to serve as a standard of medical care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. These parameters of practice should be considered guidelines only. Adherence to them will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement regarding a particular clinical procedure or treatment plan must be made by the doctor in light of the clinical data presented by the patient and the diagnostic and treatment options available. However, it is advised that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

Implementation of national clinical guidelines is the responsibility of each NHS Trust and is an essential part of clinical governance. It is acknowledged that every Trust cannot implement every guideline immediately on publication, but mechanisms should be in place to ensure that the care provided is reviewed against the guideline recommendations and the reasons for any differences assessed and, where appropriate, addressed. These discussions should involve both clinical staff and management. Local arrangements may then be made to implement the national guideline in individual hospitals, units and practices, and to monitor compliance. This may be done by a variety of means including patient-specific reminders, continuing education and training, and clinical audit.

Given the substantial body of evidence on how patients with angina should be treated, attention needs to be focused on implementing this evidence. The size of this task is considerable. The Scottish Health Survey found the overall prevalence of ischaemic heart disease in Scotland among male and female patients aged from 16 to 74 years to be 6.4% and 4.6% respectively.³

5.2 SYSTEMS TO IMPROVE CARE FOR PATIENTS WITH ANGINA

Given its prevalence, most patients with stable angina are cared for most of the time in general practice.^{159, 160} Several systems of care have been suggested as means to improve on routine care. These include nurse-run clinics, cardiac liaison nurses, postal prompts to general practitioners and patients, audits and other data collection exercises, computer prompts for use during consultations, expanded roles for community pharmacists and multidisciplinary approaches with input from dietitians and physiotherapists. To date, only some of these approaches have been subject to robust evaluations. There have been five randomised trials either wholly in primary care or at the primary/secondary care interface.

5.3 STRUCTURED CARE AND FOLLOW-UP WITH NURSE-LED CLINICS

Two studies suggest benefits from a structured approach to care. Both evaluated the effects of follow-up by trained nurses in primary care. In the first study in Belfast health visitors were trained to give personal health education on diet, exercise and smoking and refer uncontrolled hypertensive patients to their GP.¹⁶¹⁻¹⁶³ After two years they reported significantly more physical activity (44% vs. 24% active daily) and better diet in the intervention group, but no changes in smoking, blood pressure or lipids. Patients reported less angina and scored better for physical mobility on the Nottingham Health Profile. Total mortality was also reduced in the intervention group (odds ratio 0.43, 95% confidence intervals 0.22 to 0.85).

Ib & III

In the second study, in Grampian, nurse-led secondary prevention clinics were used to promote medical and lifestyle components of secondary prevention.^{164, 165} At one year, significantly more patients took aspirin (81% vs. 66%), had better blood pressure treatment (91% vs. 85%)

Ib

and lipid treatment (29% vs. 16%), were moderately physically active (42% vs. 31%) and had low fat diets (56% vs. 49%), but there were no differences in smoking. The clinics improved patients' health, especially the physical and functioning scales of the SF-36 questionnaire and fewer patients required hospital admission (20% vs. 28%).

Ib

B Structured care and follow-up should be provided for patients with coronary heart disease in primary care.

5.4 AUDIT AND FEEDBACK

A Cochrane review has concluded that audit and feedback may be effective in improving the practice of healthcare professionals, especially prescribing. With respect to management of angina, one randomised trial has been conducted, which evaluated feedback to general practitioners of their aspirin prescribing to patients with coronary heart disease.¹⁶⁷ Feedback and education were given during a practice meeting and further self audit was facilitated. After four months, aspirin prescribing was significantly higher in the intervention group (58% vs. 50%).

General practices should audit their prescribing of secondary preventive drugs to patients with coronary heart disease. Audit should be facilitated and results fed back with appropriate education.

5.5 IMPROVING COMMUNICATION AT THE PRIMARY/SECONDARY CARE INTERFACE

A number of problems have been identified at the interface of primary and secondary care that can lead to problems for patients with coronary heart disease, including poor communication, misinformation, and poorly defined remits for relevant staff.¹⁶⁸ Recommendations for good practice on communication and information have been given in the SIGN *Immediate Discharge Document (under review in 2001)*.¹⁶⁹

Two studies have evaluated systems to improve care at the interface of primary and secondary care after hospital admissions with myocardial infarction or angina or referral to chest pain clinics. In the Southampton integrated care project (SHIP) study, cardiac liaison nurses promoted more follow-up in general practice but this did not translate into improvements in treatment or risk factors at one year.¹⁷⁰ In another study, postal prompts to patients and general practitioners after hospital discharge appeared to improve prescribing of some secondary preventive drugs, but the differences were not statistically significant. Further research is needed.

5.6 OTHER APPROACHES

There is no direct evidence to indicate whether the other approaches suggested in section 5.2 are effective at improving care for patients with angina or coronary heart disease. A Cochrane Review on the expanded role of pharmacists in general found studies that supported their role in patient counselling and physician education but had concerns about the limited number and quality of studies so came to no definite conclusions.¹⁷² Although promising, further research should be undertaken. The roles of data collection exercises, computer prompts and multidisciplinary approaches in primary care all require evaluation.

5.7 RECOMMENDATIONS FOR AUDIT

5.7.1 OUTCOME INDICATORS

- Symptom control assessed by patients.
- Emergency admission for CHD.
- Rates of sudden death, myocardial infarction, angioplasty, CABG.
- Rates of smoking cessation.
- Achievement of blood pressure, total cholesterol, weight and physical activity targets.

5.7.2 KEY POINTS FOR AUDIT

- Identification and register of all CHD patients (including history of MI, angioplasty, CABG).
- System in place for annual review of identified CHD patients.
- Structured questionnaires used to measure
 - alcohol intake
 - exercise level
 - diet.
- Proportion of those with CHD - not MI, angioplasty or CABG - who have had a risk assessment.
- Proportion of those not on aspirin without a documented reason.
- Proportion of those not on an ACE Inhibitor without a documented reason.
- Proportion of those who have had blood pressure recorded since diagnosis with angina.
- Proportion of those with hypertension (defined as locally agreed) who are being treated.
- Proportion of those treated for hypertension for at least six months who have reached an agreed target level.
- Proportion of those with angina with a recorded cholesterol concentration.
- Proportion of those treated for hypercholesterolaemia for at least six months who have reached the target.
- Proportion of those with a cholesterol level above a locally agreed target.
- Proportion of those with a cholesterol level above a locally agreed target who are being treated.
- Proportion of smokers, who have stage of change identified and help offered if ready to change.
- Proportion of those with above optimal alcohol intake who have been offered a structured intervention.
- Proportion of those with a less than adequate exercise level who have been offered exercise referral / advice.
- Proportion of those requiring dietary change who have been offered structured advice.

5.8 RECOMMENDATIONS FOR FURTHER RESEARCH

- A large scale RCT to examine efficacy and safety of the different classes of anti-anginal drugs as monotherapy and in combination.
- An RCT to examine the benefits of weight reduction and exercise or fitness programmes in angina.
- A rigorous evaluation of models of care, e.g. open access ECG and exercise testing vs. referral to a clinic, to include outcome and economic evaluations.

6 Information for patients

EXAMPLE ADVICE LEAFLET FOR PATIENTS WITH STABLE ANGINA

What is angina?

Angina Pectoris is a discomfort or pain usually felt in the chest, that comes from the heart muscle. You normally feel it as a tightness, heaviness, weight, pressure or some similar feeling. It may also spread to the throat, jaw, shoulders or back. Sometimes you might also notice aching or tingling in your arms or hands, or breathlessness when you have angina.

You will usually get your angina by doing something energetic or by getting angry or excited or upset. You might find it is worse in cold weather.

Angina is caused by the heart muscle not getting enough blood. This happens because of a narrowing in the blood vessels (the coronary arteries) which bring blood to the heart muscle. Normally, enough blood flow occurs to satisfy the heart muscle at rest or during light activity. With more energetic activity (or when you get angry or excited) the heart has to pump harder and faster and the muscle needs more blood. If the coronary arteries are narrowed the blood flow through them cannot increase and the heart complains about the blood supply not matching what it needs. You feel this as angina.

In many ways angina is like a muscle cramp in the arm or leg which also occurs when the working muscle does not get enough blood to match what it needs. That is why angina warns you to stop and rest for a few minutes or calm down a bit.

Patients with angina are looked after mostly by their GP.

What your GP will do:

- Your doctor will do some blood tests to make sure you are not anaemic, that you do not have diabetes, and to measure your blood cholesterol.
- Your doctor will arrange an **ECG** (NB Some people with a normal ECG have got coronary heart disease). Your doctor should discuss referring you to hospital for an exercise test to help to decide how severe the condition is. The severity of your symptoms does not indicate the severity of the disease in your coronary arteries. Ask your doctor about this test if you haven't had one. The test will help your doctor decide what treatment is best for you.
- Your doctor may refer you to a specialist for further tests if the exercise test is positive or if the diagnosis is uncertain.

The biggest risk for patients with angina is having a heart attack. If you follow the advice below your chances of getting a heart attack will be as low as possible - and your chance of doing all the things you want to without getting angina will be better.

What YOU can do:

- Any chest pain should be discussed with your GP.
- You should see your doctor **quickly** if your symptoms get any worse, especially if the pain is worse than usual, or comes on at night, or at rest.

Changing your lifestyle will improve your chance of not getting further problems:

- You should quit **smoking**. Nicotine patches will help people to quit if they are well motivated. They are of help when used over a limited time.
- If you are **overweight**, weight loss is sensible.
- **Regular exercise** is sensible. Aim for 30 minutes of moderate physical activity on most days. This can be done all at once or in smaller blocks of around 10 minutes. Build up gradually.
- Eat a **sensible diet**. Try to eat five portions of fresh fruit and vegetables a day and two to three portions of oil rich fish a week. Eat less fat and make as much of it as possible polyunsaturated or monounsaturated. Cut down on the sugary foods and eat more starchy foods (bread, rice, pasta etc).
- Drinking a small amount of alcohol each day is perfectly safe for people with angina or after a heart attack. The limits should be: *Men up to three units in one day; Women up to two units in one day.*
- Some drugs may improve the length of your life - if you are not taking them ask your doctor about them. These drugs are aspirin and ACE Inhibitors.
- Some drugs allow you to carry out the activities you want to without having chest pain. These include β -blockers, calcium antagonists and nitrates. **If you get chest pain that stops you doing things you want to, ask your doctor for help.**
- The GTN tablets or spray can prevent the pain of angina, so use them *before* any activities that you know will bring on an attack.
- If your **blood pressure** is raised, treatment will reduce your risk of a heart attack and stroke. If you don't know your blood pressure, or what it ought to be, ask your doctor or practice nurse.
- All people with angina should have their **cholesterol** checked. You may be able to keep your cholesterol level down by a low fat diet and a healthy lifestyle. However if your cholesterol level stays above 5mmol/l additional treatment may be required, usually with a drug called a statin. If you do not know your own cholesterol level ask your doctor or practice nurse.
- If you are diabetic you should try to keep your blood sugar levels under good control (as close to 4 mmol/l - 8 mmol/l as possible).
- If you have not had **pneumococcal immunisation** (to prevent chest infections) ask your doctor or practice nurse. Make sure you get your **flu jab** each autumn.
- You should discuss **driving** with your doctor and you must notify DVLC if you hold a PVC or LGV licence.

7 Development of the guideline

7.1 INTRODUCTION

SIGN is a collaborative network of clinicians and other health care professionals, funded by the Clinical Resource and Audit Group (CRAG) of the Scottish Executive Health Department. SIGN guidelines are developed by multidisciplinary groups of practising clinicians using a standard methodology, based on a systematic review of the evidence. Further details about SIGN and the guideline development methodology are contained in “SIGN 50; A Guideline developer’s handbook” available at www.sign.ac.uk.

7.2 THE GUIDELINE DEVELOPMENT GROUP

Dr Charles Swainson (Chairman)	<i>Consultant Renal Physician and Medical Director, Lothian University Hospitals NHS Trust</i>
Dr Neil Campbell (Methodologist)	<i>Cancer Research Campaign Primary Care Oncology Fellow, University of Aberdeen</i>
Dr Neil Dewhurst	<i>Consultant Cardiologist, Perth Royal Infirmary</i>
Dr Robert Finnie	<i>General Practitioner, Livingston</i>
Sister Janice Fraser	<i>Coronary Care Unit, Western Infirmary, Glasgow</i>
Mr Robin Harbour	<i>Information Manager, SIGN</i>
Professor Lee Kennedy	<i>Consultant Physician, Sunderland Royal Hospital</i>
Mr Stephen McGlynn	<i>Area Pharmacy Specialist, R&D, Western Infirmary, Glasgow</i>
Dr Donald McLeod	<i>Consultant Cardiologist, Queen Margaret Hospital, Dunfermline</i>
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Dr Ashley Mowat	<i>Consultant Gastroenterologist, Aberdeen Royal Infirmary</i>
Dr Moray Nairn	<i>Programme Manager, SIGN</i>
Dr Sue Vincent	<i>General Practitioner, Dundee</i>
Dr Stephen Walton	<i>Consultant Cardiologist, Aberdeen Royal Infirmary</i>

The membership of the guideline development group was confirmed following consultation with the member organisations of SIGN. Declarations of interests were made by all members of the guideline development group. Further details are available from the SIGN Executive.

7.3 SYSTEMATIC LITERATURE REVIEW

The guideline development group reviewed the medical literature and existing guidelines published by a wide range of other bodies, including the North of England evidence-based guideline for the Primary Care Management of Stable Angina.⁸¹ The group decided that the quality and depth of the systematic review undertaken for the North of England guideline was such as to make duplication of this work unnecessary. It was decided to base the SIGN guideline on the North of England guideline, adapting it to produce a guideline suitable for use within Scotland. SIGN acknowledges the debt to the North of England project group and extensive reference is made throughout the guideline to their original document. The SIGN guideline quotes additional references reviewed by the SIGN group.

Searches for other guidelines or systematic reviews were carried out covering key Internet sites, Embase, Healthstar, Medline, and Pascal. An additional search, covering the same databases, was carried out looking specifically at the management of stable angina and secondary prevention of coronary heart disease in primary care. The main searches were supplemented by material identified by individual members of the development group. All selected papers were evaluated using standard methodological checklists before conclusions were considered as evidence.

7.4 CONSULTATION AND PEER REVIEW

7.4.1 NATIONAL OPEN MEETING

A national open meeting is the main consultative phase of SIGN guideline development, at which the guideline development group present their draft recommendations for the first time. The national open meeting for this guideline was held on 27th September 1999 and was attended by 198 representatives of all the key specialties relevant to the guideline. The draft guideline was also available on the SIGN web site for a limited period at this stage to allow those unable to attend the meeting to contribute to the development of the guideline.

7.4.2 SPECIALIST REVIEW

The guideline was also reviewed in draft form by a panel of independent expert referees, who were asked to comment primarily on the comprehensiveness and accuracy of interpretation of the evidence base supporting the recommendations in the guideline. The Scottish Executive Coronary Heart Disease Task Group also provided comments on the guideline. SIGN is very grateful to all of these experts for their contribution to this guideline.

Dr Phil Adams	<i>Consultant Cardiologist, Royal Victoria Infirmary, Newcastle</i>
Dr Miles Fisher	<i>Consultant Physician, Royal Alexandra Hospital, Paisley</i>
Professor Charles Forbes	<i>Consultant Physician, Ninewells Hospital, Dundee</i>
Dr John Forfar	<i>Consultant Cardiologist, John Radcliffe Hospital, Oxford</i>
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Professor Lewis Ritchie	<i>Professor of General Practice, University of Aberdeen</i>
Dr Alex Watson	<i>General Practitioner, Westgate Health Centre, Dundee</i>

7.4.3 SIGN EDITORIAL GROUP

As a final quality control check, the guideline is reviewed by an Editorial Group comprising the relevant specialty representatives on SIGN Council to ensure that the peer reviewers' comments have been addressed adequately and that any risk of bias in the guideline development process as a whole has been minimised. The Editorial Group for this guideline was as follows:

Dr Jim Beattie	<i>Royal College of General Practitioners</i>
Dr Marion Bennie	<i>Royal Pharmaceutical Society of Great Britain (Scottish Department)</i>
Dr Doreen Campbell	<i>Senior Medical Officer, Scottish Executive Department of Health</i>
Dr Patricia Donald	<i>Primary Care Adviser to SIGN</i>
Professor Gordon Lowe	<i>Royal College of Physicians of Edinburgh</i>
Ms Juliet Miller	<i>Director of SIGN, Co-editor</i>
Professor James Petrie	<i>Chairman of SIGN, Co-editor</i>
Dr Joanna Wardlaw	<i>Royal College of Radiologists</i>
Mr Pete Wimpenny	<i>Centre for Nursing Practice Development, Robert Gordon University</i>

Each member of the guideline development group then approved the final guideline for publication.

7.5 ACKNOWLEDGEMENTS

The contribution of the following former members of the guideline development group is gratefully acknowledged:

Dr Ian MacLean	<i>Director of Public Health, Dumfries & Galloway Health Board</i>
Dr Nick Boon	<i>Consultant Cardiologist, Royal Infirmary of Edinburgh</i>
Dr Gavin Brown	<i>General Medicine, Fort William</i>
Dr Patricia Donald	<i>General Practitioner, Edinburgh</i>
Dr Ursula Guly	<i>Consultant in A&E Medicine, Ninewells Hospital, Dundee</i>
Dr Helen Oxenham	<i>Cardiologist, Royal Infirmary of Edinburgh</i>
Dr Howard Robson	<i>Consultant Physician, Cumberland Infirmary, Carlisle</i>
Dr Ian Starkey	<i>Consultant Cardiologist, Western General Hospital, Edinburgh</i>

Guideline development and literature review expertise, support, and facilitation were provided by the SIGN Executive:

Ms Francesca Chappell	<i>Information Officer</i>
Ms Gail Crosbie	<i>Communications Officer</i>
Dr Ali El-Ghorr	<i>Programme Manager</i>
Mrs Lesley Forsyth	<i>Conferences Coordinator</i>
Mr Robin Harbour	<i>Information Manager</i>
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Update to printed guideline

23 Jun 2003

Section 4.1.1 paragraph 2 wording changed from -

The Swedish Angina Pectoris Aspirin Trial (SAPAT) randomised 2,035 patients with a history of at least one month of exertional angina to receive 75 mg aspirin daily. The results demonstrated an absolute reduction in primary endpoints (non-fatal MI, fatal MI or sudden death) in the treatment group of 34% after 72 months. All patients were treated with beta-blockers. A subgroup analysis of the US Physicians' Health Study showed that aspirin therapy greatly reduced the risk of first MI in patients with stable angina ($p < 0.001$).¹¹⁰

to

The Swedish Angina Pectoris Aspirin Trial (SAPAT) randomised 2,035 patients with a history of at least one month of exertional angina to receive 75 mg aspirin daily. The results demonstrated a relative risk reduction in primary endpoints (non-fatal MI, fatal MI or sudden death) in the treatment group of 34% after 72 months. Absolute risk reduction was 4% (95% confidence intervals 1-7%). All patients were treated with beta-blockers. A subgroup analysis of the US Physicians' Health Study showed that aspirin therapy greatly reduced the risk of first MI in patients with stable angina ($p < 0.001$).¹¹⁰

SUSPECTED ANGINA

HISTORY

CLINICAL EXAMINATION

- Haemoglobin** to identify underlying anaemia
- Fasting **blood glucose** to identify previously undiagnosed diabetes mellitus
- Full **lipid profile**

- B Resting 12-lead ECG

A normal ECG does not exclude the possibility of CHD

Patients may be referred to a cardiologist at any point if symptoms or test results indicate.

The following should be specifically referred:

- patients who appear to have had a previous MI on their initial ECG or other abnormality that the general practitioner considers significant
- patients who fail to respond to medical treatment having already had an ETT
- patients who have an ejection systolic murmur suggesting aortic stenosis

Patients with pain on minimal exertion, pain at rest (which may occur at night) or angina which appears to be progressing rapidly despite increasing medical treatment may have **unstable angina** and should be considered for **immediate** referral.

- B **Exercise tolerance test (ETT)** for risk stratification

Myocardial perfusion imaging where ETT is not possible

- B ETT should be performed whilst patients are taking their normal medication

- Patients should not be referred for ETT if:
 - they are physically incapable of performing the test
 - they may have aortic stenosis or cardiomyopathy
 - the results of stress testing would not affect management

RISK FACTOR MANAGEMENT

▶ **SMOKING**

- B **All patients with angina who smoke should be advised to stop**

A Brief advice from a health professional, tailored self-help materials, individual and group counselling and antidepressants (with behavioural support) can increase smoking cessation

▶ **HYPERTENSION**

- B All patients with angina should have **blood pressure assessed and managed**

▶ **DIET**

- B Patients with angina should **modify their diet** in line with healthy eating advice

▶ **EXERCISE**

- B All those with CHD should be encouraged to **increase their aerobic exercise levels** within the limits set by their disease state

▶ **OVERWEIGHT**

- C All patients with CHD should be actively encouraged to **lose weight towards BMI < 25**

▶ **ALCOHOL**

- B Patients with CHD who consume alcohol should be encouraged to limit their consumption to **three units per day for men and two units per day for women**

▶ **LIPIDS**

- C All patients with angina should have a **cholesterol measurement**

If total cholesterol (TC) is ≥ 5.0 mmol/l

- C **Appropriate dietary measures** should be recommended and a random **non-fasting cholesterol measurement** repeated after 6-12 weeks

- C If required, **drug therapy** should then be initiated to reduce TC to < 5.0 mmol/l including a fall in TC of at least 1 mmol/l

DRUG THERAPY

▶ **SECONDARY PROPHYLACTIC TREATMENT**

- A Patients with stable angina should be treated with **aspirin 75 mg daily** (unless contraindicated)

▶ **SHORT TERM CONTROL OF ANGINA SYMPTOMS**

- All patients with symptomatic coronary heart disease should be prescribed **sublingual GTN** and should be educated in its use

▶ **LONG TERM PREVENTION OF ANGINA SYMPTOMS**

- B **Patients who require regular symptomatic treatment should be treated initially with a β -blocker** (unless specifically contraindicated)

- B Patients should be warned not to stop β -blockers suddenly or allow them to run out

- C **Patients intolerant of β -blockers** and who show no LVSD should be treated with:
 - a **rate limiting calcium channel blocker**
 - a **long-acting dihydropyridine**
 - a **nitrate**
 - or a **potassium channel opening agent**

- A If symptoms are not controlled in patients taking β -blockers, add:
 - **isosorbide mononitrate**
 - a **long-acting dihydropyridine**
 - or **diltiazem** (but observe the cautions in the BNF)

▶ **REFERRAL**

- Consideration should be given to referral to a cardiologist
 - on initiation of treatment
 - if the patient is perceived to be at increased risk
 - if at any stage treatment fails to control symptoms
 - or if these symptoms limit the patients' desired activities

KEY

A B C grade of recommendation

good practice point