

GUIDELINES

Investigation and management of stable angina: revised guidelines 1998

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In October 1991 the joint audit committee of the British Cardiac Society and the Royal College of Physicians of London set up a working group to review present practices in the investigation and management of angina and to identify potential audit issues in the care of patients presenting with this complaint. The findings of the working group were published as a summary in 1993 and in the form of a monograph in 1994.^{1,2} The present document represents the first revision of these guidelines. The need for revision was driven by three factors: first, progress in our understanding of the epidemiology, pathology, and treatment of angina; second, continuing developments in the methodology and format of guidelines; and third, a reappraisal of the target of the guidelines in the light of developments in medical practice.

Methodology

The original guidelines were drawn up by a working party selected to contain representatives of a wide variety of different cardiological practices, general practice, academic cardiology and epidemiology, and with advice from patient groups and purchaser representatives. Members were asked to prepare monographs on particular topics based both on their own experience and on a review of the literature. The monographs were then discussed in committee and a consensus synthesis of guidelines undertaken. For the processes of revision a working group representative of a similarly wide spectrum of experience was identified and asked: (1) to review the existing guidelines; (2) suggest corrections and amendments; (3) identify new data in the form of original papers, new guidelines or systematic reviews; and (4) attend a meeting to discuss the relative priorities and nature of amendments. The working group also took specific account of: feedback collected since the publication of the original guidelines; systematic reviews and guidelines published since that date, in particular the guidelines on primary care management of stable angina produced by the North of England Evidence Based Guideline Development Project³ and the report of the task force of the European Society of Cardiology⁴; and specific literature searches designed to answer particular questions which emerged during the

debate. The current guidelines now explicitly state the level of evidence on which (in the opinion of the working group) each of the recommendations made is based. Category A recommendations are based on large randomised controlled trials or meta-analyses. Category B recommendations are based on smaller trials, or large observational studies. Category C recommendations are based on other evidence including small studies and consensus.

Who are the guidelines intended for?

The guidelines are ultimately intended for the patient. They are intended to present an evidence based and judiciously interpreted account of the standards of care which the authors would wish to receive themselves or for their own relatives. This is likely to be delivered in the context of a continuum of care that encompasses primary, secondary, and tertiary care delivery systems, and by medical, nursing, and other professionals. It is recognised, and indeed expected, that they will be used as a basis for more specific and detailed guidelines appropriate to individual circumstances and user groups. Where valid cost effectiveness data are available for comparing alternative treatment strategies, they will be presented. It is not, however, within the scope of these guidelines to attempt cost effectiveness comparisons with treatments for other diseases. It is accepted that where resources are limited it may be necessary to deny treatment even of proven efficacy to particular patients. However, when this is necessary it should be done openly and explicitly, and patients should be given the opportunity to make alternative arrangements.

What is the legal status of the guidelines?

The guidelines represent a consensus of expert opinion based on specified evidence available at the time they were compiled. Of necessity much of this evidence will have been obtained from large randomised trials, and its applicability to individual patients will be a matter of professional judgment. It is emphasised that these are intended as guidelines rather than as mandatory management protocols. It is also emphasised that the guidelines do not cover

A list of the participants in the revision workshop and those who contributed written comments is given at the end of the paper

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treatment regarded as experimental, or drugs for which a product licence was not in force at the time they were compiled.

Pathology and epidemiology of angina

Angina is a symptom, consisting of a characteristic chest pain precipitated by exertion, that is usually the result of partial obstruction of a coronary artery by atheroma. Many people with coronary atheroma do not have angina, either because the atheroma is not causing obstruction, or because gradual obstruction has been compensated for by a collateral circulation. Thus although the majority of people with angina have coronary atheroma, many of the complications of coronary atheroma, including myocardial infarction and sudden death, occur in people without any previous known angina history.⁵ Coronary atheroma is associated with several factors including smoking, a raised plasma cholesterol concentration, high blood pressure, and diabetes.^{6,7} It is more common in men than in premenopausal women, and increases in prevalence and extent with age. The prevalence of coronary artery disease varies between different ethnic groups, being higher in Indo-Asians and lower in east Asians and African Caribbeans compared to whites.⁸ In a few patients, angina is not caused by coronary artery disease but by aortic stenosis or hypertrophic cardiomyopathy. Angina can be made worse by anaemia or hyperthyroidism.

It is common for the severity of anginal symptoms to vary with time, or even from day to day. At the same time, underlying coronary atheroma may progress, either by an increase in the severity of pre-existing lesions or by the formation of new stenoses (narrowings). Under certain circumstances coronary atheroma can be induced to regress. "Stable" angina is a relative term used in contrast to "unstable" angina where symptoms are progressively increasing in severity over a short time period, and there is an increased risk of complications such as death or myocardial infarction. Symptom severity is an imperfect guide to the severity or progression of coronary atheroma.⁹

Population studies which have used general practice records, nitrate prescription or both as a marker for angina indicate a population prevalence in the United Kingdom of approximately 2%.^{10,11} Surveys based on questionnaires have shown a prevalence of between 5–10% in men aged 40 to 60 years.^{12,13} Angina prevalence increases with age, and there is evidence for a demographic shift whereby age specific prevalence is falling in younger patients but increasing in those aged 65 or over.¹⁴

The most reliable incidence estimates (new cases per population per year, from a study which routinely used exercise testing and a cardiologist interview) are from 0.44/1000/year (age 31–40 years) to 2.32/1000/year (age 61–70 years) in men, and from 0.08/1000/year (age 31–40 years) to 1.01/1000/year in women.¹⁵ Applying these results to the UK population gives an estimate of approximately 22 000 new angina cases per year. For the reasons described above these figures system-

atically underestimate the true prevalence and incidence of coronary atheroma. In men, angina is a less common initial manifestation of ischaemic heart disease than myocardial infarction, but the converse is true in women. In men with angina the average attributable increase in mortality is around 2% per year, which is similar to that seen after myocardial infarction. In women the prognosis with angina is better but the prognosis after myocardial infarction is worse. Although patients with angina are more likely to have myocardial infarction, only about one in five myocardial infarcts is preceded by angina.¹⁶

Clinical diagnosis of angina

The diagnosis of angina is usually made on the basis of the clinical history, informed by an assessment of risk factors for coronary atheroma.^{17,18} The most characteristic clinical feature of angina is retrosternal chest pain precipitated by physical or emotional exertion. It is relieved by rest. The pain is usually described as burning, squeezing, or pressing. Sometimes the sensation is of breathlessness rather than pain. The discomfort may be experienced alternatively or additionally in the arms, epigastrium, jaw, or back. The relation to exertion is more characteristic than the precise site. Angina is often worse on effort in cold weather or after food. Pain that is independent of physical activity, or persists for long periods at rest, is rarely angina. Angina is usually relieved by glyceryl trinitrate but this is not a specific response. Angina is termed unstable if it is brought on by progressively less physical exertion over a short period of time, often culminating in episodes of angina precipitated by minimal exertion or even at rest.¹⁹ A classification of the severity of angina symptoms is helpful. The most commonly used is that devised by the Canadian Cardiovascular Society.²⁰

The association between typical angina symptoms and coronary artery obstruction is stronger in men than in women.²¹ The presence of risk markers such as hypercholesterolaemia, hypertension, a history of smoking, or a family history of ischaemic heart disease makes it more likely that a chest pain is anginal in origin. There are no physical signs of angina or coronary atheroma, but patients should be examined for other possible causes of angina such as aortic stenosis and for features of hyperlipidaemia. An accurate clinical diagnosis is an essential step in the investigation and management of angina.

Assessment of risk factors

A minimum assessment of risk factors should include: recording of a family history of angina, hypertension or diabetes; recording of body mass index; measurement of blood pressure; urine testing for glucose; and measurement of plasma lipids, preferably including high density lipoprotein (HDL) cholesterol and triglycerides [B].

Confirmation of diagnosis

A resting 12 lead electrocardiogram (ECG) should be recorded [B]. Although this is of low sensitivity in diagnosing coronary artery disease, an abnormal 12 lead ECG identified a patient subgroup with a substantially higher risk of death or myocardial infarction.²²

EXERCISE TESTING

Exercise testing, if practicable, is important both to provide objective confirmation of exercise induced ischaemia and as an independent indicator of prognosis [A].²³⁻²⁴ It will usually take the form of treadmill exercise according to a standard protocol with ECG monitoring. The Bruce protocol is the one most commonly used in the UK.²⁵ An exercise ECG is conventionally said to be positive if it shows 1 mm or more of reversible plane ST segment depression. Also important are whether or not the exercise induces typical symptoms, the amount of exercise the patient can achieve, the presence of a normal or abnormal blood pressure response to exercise, and the appearance of the patient while exercising.²⁶⁻²⁷ Exercise electrocardiography cannot be regarded in isolation as an effective screening test for ischaemic heart disease. Patients with non-obstructive coronary artery disease or a well developed collateral circulation may have negative exercise tests; on the other hand exercise testing of a population with a low prevalence of ischaemic heart disease will produce a high proportion of false positive tests.²⁸ False positive exercise recordings are also more common in women. The working group emphasised the importance of exercise testing in the evaluation of patients with suspected angina, but were convinced that it should be preceded by careful clinical evaluation, and the result interpreted by trained clinicians. In patients with a clinical diagnosis of angina it is not necessary to wait for exercise testing to be carried out before instituting antianginal treatment [A].²⁹ Similarly, antianginal treatment should not be withdrawn when carrying out exercise testing unless this is specified for particular purposes—for example, the issue of a public service vehicle licence [B].

MYOCARDIAL PERFUSION SCANNING

Myocardial perfusion scanning involves the injection, at peak exercise or during stress induced using adenosine, dipyridamole, or dobutamine, of a radionuclide which is taken up by the myocardium. The creation of a myocardial perfusion image depends upon the ability of a radiopharmaceutical to be distributed throughout the myocardium in proportion to regional blood flow. Typically, two sets of images are acquired using a gamma camera, one reflecting perfusion at peak stress and the other at resting perfusion. Radiopharmaceuticals distribute uniformly in normally perfused myocardium. In areas supplied by coronary arteries with functionally significant stenoses there is a stress defect that improves on rest imaging (reversible defect). Stress defects that fail to improve on rest imaging (fixed defects) generally represent infarcted areas.

Myocardial perfusion imaging has a higher diagnostic sensitivity and specificity for coronary artery disease than exercise electrocardiography (80% and 92% *v* 68% and 84%, respectively).³⁰ Comparisons using receiver operating characteristic curves have also confirmed the superior diagnostic capabilities of perfusion imaging.³¹ The requirements for expensive equipment and appropriately trained staff, together with the radiation burden to the patient, mean that perfusion imaging is usually employed for diagnosis when exercise electrocardiography is unhelpful or leaves doubt. This may occur when the resting ECG is abnormal, equivocal ST segment changes occur with exercise, exercise electrocardiography is normal despite a high pretest probability of disease, abnormal ST segments are seen despite a low pretest probability of disease, or only submaximal exercise has been achieved [B].

There is evidence that myocardial perfusion imaging provides prognostic information which is more reliable than either exercise electrocardiography or coronary angiography.³²⁻³³ A normal stress perfusion study predicts a favourable prognosis for cardiac event rate (less than 1% per year), even in patients with known coronary disease.³⁴⁻³⁶ Conversely severe and extensive reversible ischaemia predicts an adverse prognosis.³⁷

STRESS ECHOCARDIOGRAPHY

Stress echocardiography involves the imaging of areas of altered myocardial contractility under conditions of exercise or pharmacological stress. As with myocardial perfusion scanning, the object is to improve the sensitivity and specificity of conventional exercise electrocardiography. Stress echocardiography and myocardial perfusion imaging probably provide comparable results in the diagnosis of coronary disease but perfusion imaging may provide more reliable information about prognosis.³⁸⁻³⁹

CORONARY ANGIOGRAPHY

Coronary angiography gives a uniquely detailed anatomical record of the coronary arteries and their stenoses. Strictly speaking, it does not diagnose either coronary atheroma (since vessel wall disease may be present when the lumen is normal) or myocardial ischaemia (since it does not give full information about coronary flow). It provides information valuable in risk stratification and it is an essential prelude to interventions such as angioplasty or coronary artery bypass grafting.

Coronary angiography is an invasive investigation involving arterial puncture and the introduction of catheters which are passed through the aorta to the mouths of the coronary arteries. It is usually performed on a day case basis. The overall mortality associated with the procedure is of the order of 1 in 2000 and the serious complication rate is about 1%.⁴⁰

In many countries coronary angiography is regarded as a routine investigation in patients with clinical features of angina.⁴ In the UK, access to coronary angiography is limited for

many patients by lack of resources or by purchasing restrictions. Several attempts have been made to evaluate the "appropriateness" of referral for coronary angiography, based on criteria such as symptom severity, exercise test performance, and adequacy of medical treatment.⁴¹⁻⁴⁴ These criteria can be used to form the basis of a scale of priority for investigation; however, for prospective planning as opposed to retrospective audit, a numerical scale, where the decision point can be adjusted in response to available resources, is more likely to be helpful than a simple division into "appropriate" and "not appropriate".

Numerous attempts have also been made to define "appropriate" numbers of investigations for a given size of population.⁴⁵⁻⁴⁶ These have inevitably been based on consensus rather than on objective evidence, and are further complicated by the marked variations in rates of investigation between different districts in the same geographical area.⁴⁷ Where rationing of resources is inevitable, it should be based on clear, explicit, and objective clinical criteria.

Risk stratification

The investigation and treatment of the patient with angina has the dual objectives of relieving symptoms and of improving prognosis. Prognosis will be improved both by correcting risk factors for coronary atheroma (secondary prevention) and, where appropriate, by myocardial revascularisation. Risk stratification is important both for choosing therapeutic options and for allocating resources.

Age is important; the older the patient with ischaemic heart disease the greater the risk of an ischaemic event and the more likely a fatal outcome.⁵⁻⁷ On the other hand, older patients have lower demands of physical exertion and a more stoical approach to symptoms.

Family history of heart attack and diabetes are independent predictors of death from coronary heart disease.⁴⁸⁻⁴⁹

The presence of *angina symptoms* in a patient with known ischaemic heart disease worsens the prognosis.⁷⁻⁵⁰ The link between symptom severity and prognosis is more complex, with some studies showing a poor correlation,⁵⁰ while others have shown a worse outcome in patients with the most severe symptoms.⁵¹ This might be explained in part by confounding between symptoms of angina and those of heart failure, and in part by the tendency of patients with more severe symptoms to receive prompt and more intensive care.

However, subjective assessment of symptoms is variable and *objectively assessed exercise tolerance* is more reliable as a predictor. A good performance on exercise testing is generally associated with a good prognosis.²⁶ As described above, *myocardial perfusion imaging* is an even more reliable predictor of outcome.

Evidence of myocardial damage in the form of ECG changes or a reduced left ventricular ejection fraction indicates a worse prognosis.⁵¹

The extent and distribution of coronary artery stenoses, as revealed by *coronary angiography*, predict outcome. Patients with left main coronary stenosis or three vessel coronary

disease have a poorer prognosis, while patients with angiographically normal vessels or a single stenosis have a good outlook.⁴⁹⁻⁵¹

Investigation summary

- Clinical assessment is paramount and should include a full assessment of risk factors for coronary atheroma [A]
- A baseline 12 lead ECG is useful [B]
- With the exception of patients who are very frail or who have serious intercurrent illness, non-invasive assessment of myocardial ischaemia by one of the methods described is strongly recommended [A]
- Angiography is justifiable in all cases where it would alter patient management [B]. Where resources are limited priority should be given to patients who have severe symptoms despite adequate medical management, who have objective evidence of extensive ischaemia or of ischaemia precipitated at a low work load, and in patients whose independence or livelihood is in jeopardy [A]

An investigation algorithm is shown in fig 1. In an earlier version of these guidelines it was suggested that the application of this algorithm would involve approximately 3000 exercise tests and 1000 diagnostic coronary angiograms per year per million of the population. These estimates were based on hospital referral patterns at the time; subsequent experience has suggested that they were considerable underestimates.⁵²⁻⁵³

Treatment

Treatment of the patient with angina can be considered on four levels:

- Management of underlying risk factors for coronary atheroma
- Control of symptoms with medical treatment
- Coronary revascularisation
- Rehabilitation.

CONTROL OF RISK FACTORS

There is now excellent evidence that control of risk factors in patients with angina leads to an improved outcome both in terms of a reduced incidence of ischaemic events and of improved survival. Recommendations include:

- Advice on stopping smoking [A]
- Adequate control of blood pressure following British Hypertension Society guidelines⁵⁴ [A]
- Lowering of plasma cholesterol by either dietary or pharmacological means⁵⁵⁻⁵⁶ [A]
- Regular exercise, control of diabetes, and weight reduction towards an ideal body mass index [C]
- Prescription of low dose aspirin unless contraindicated or not tolerated⁵⁷⁻⁵⁸ [A]

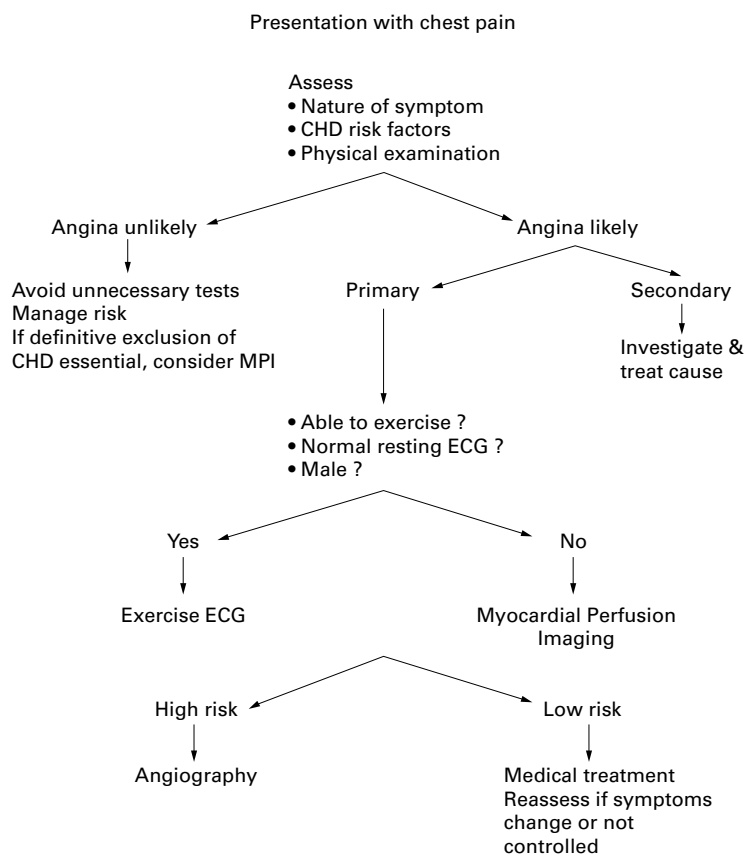


Figure 1 Algorithm of investigation in stable angina. CHD, coronary heart disease; MPI, myocardial perfusion imaging.

Detailed dietary advice is beyond the scope of this paper. There is some clinical trial evidence to support the use of antioxidant vitamins and this is currently being evaluated in a large randomised trial.⁵⁹

It is recognised that the evidence for a beneficial effect of aspirin is less strong when consideration of trials is restricted solely to those performed in patients with stable angina, but the working group saw no good reason for considering these in isolation from other

groups of patients with atheromatous vascular disease. Further justification and references to support these recommendations are given elsewhere.^{3 4}

MEDICAL CONTROL OF SYMPTOMS

This can be divided into immediate symptom control and background antianginal medication. Sublingual glyceryl trinitrate remains the standard treatment for immediate symptom control [B]. Classes of agents available for background antianginal medication are listed in table 1. For each of the agents listed there is clinical trial evidence for efficacy in symptom control versus placebo.⁶⁰⁻⁶⁹ There is no direct evidence that any of the agents listed has a significant effect on the incidence of sudden death or myocardial infarction when prescribed for the treatment of angina. This reflects the absence of clinical trials with sufficient power to evaluate these outcomes. β Blockers have been shown to reduce mortality when prescribed to patients before or following myocardial infarction.^{70 71} Verapamil has been shown to reduce the number of new cardiovascular events following myocardial infarction in a subgroup of patients without heart failure.⁷² Although there have been numerous comparisons between different classes of antianginal agents in terms of symptom reduction, these have almost always involved fixed dose comparisons; there is little evidence for the intrinsic superiority of one class over another in terms of symptom relief. It is clear, however, that individual patients differ markedly in their experience of side effects, and in practice the choice of a particular agent will often be determined by patient acceptability. Similarly, differences between agents within a given class tend to reflect differences in dose schedule and side effects rather than efficacy—a detailed description is beyond the scope of the present paper.

It is a commonly held belief that a combination of two antianginal agents makes for enhanced antianginal efficacy while minimising side effects. There is some support for this

Table 1 Classes of agents available for background antianginal medication

Pharmacological class	Examples	Main mode of action	Contraindications	Comment
β Adrenoceptor antagonist	Propranolol, timolol, atenolol, metoprolol, nadolol, oxprenolol, pindolol, bisoprolol	Attenuates heart rate increase on exercise	Asthma; caution in heart failure, peripheral vascular disease, diabetes	No convincing evidence differences between agents affect efficacy, but they may affect acceptability
Organic nitrate	Isosorbide mononitrate	NO donor, vasodilator, reduces pre- and afterload		Give once or twice daily to avoid tachyphylaxis. Increase dose slowly to avoid headache
Calcium antagonist class II: dihydropyridine type	Nifedipine, amlodipine, felodipine, nicardipine, nisoldipine	Vasodilator, reduces pre- and afterload	Caution in heart failure; avoid as monotherapy in unstable angina	
Calcium antagonist class I and III: rate slowing type	Verapamil, diltiazem	Attenuate heart rate increase on exercise; vasodilator, reduce (mainly) afterload	Risk of bradycardia if combined with β blocker; caution in heart failure	
Potassium channel activator	Nicorandil	NO donor, vasodilator, reduces pre- and afterload		Induces myocardial preconditioning; clinical significance uncertain

NO, nitric oxide.

from clinical trials,^{69 70} but combination treatment has seldom been compared with an optimised dose of either agent alone, and it is sensible to evaluate the effect of an adequate dose of a single agent before moving on to another single agent or to combination therapy [C]. As a general rule background antianginal agents should be tailed off rather than discontinued abruptly, unless they are causing serious side effects. There is very little evidence that the use of three or more antianginal agents provides any additional advantage.

- Use sublingual nitrate for immediate symptom control [A]
- Background antianginal medication will improve symptoms [A]
- β Blockers may reduce incidence of cardiac events and improve survival [B]
- Evaluate the effect of an adequate dose of a single agent before moving on to another single agent or to combination treatment [C]

REVASCULARISATION

Coronary revascularisation is nearly always done to relieve symptoms, and in some cases to improve survival. There is clinical trial evidence that surgical revascularisation by coronary bypass grafting gives a better long term survival than medical management in patients with left main coronary stenosis, three vessel coronary disease, or two vessel disease where one of the vessels was in the proximal left anterior descending artery. Benefit was greater in patients with impaired left ventricular function despite the greater perioperative mortality.^{51 73 74} There is currently no evidence that coronary angioplasty in the context of stable angina improves survival, but in randomised trials it has been shown to give better symptomatic relief than medical treatment.^{75 76} The vast majority of coronary angioplasty procedures are done in patients with single or two vessel disease who would be expected to have an excellent short to medium term survival. In these patients angioplasty and bypass grafting give similar relief of symptoms for the same low mortality rates, although angioplasty patients are more likely to remain on some antianginal medication and more likely to require a second procedure.^{77 78} In patients with multivessel disease the outcome of angioplasty and bypass grafting is similar in terms of early symptom and operative mortality, but more angioplasty patients require a second procedure and they are more likely to have recurrent angina within five years.⁷⁹ Coronary stenting is an integral part of modern angioplasty practice. There is a consensus that it has reduced the immediate complication rate and reduced the rate of restenosis, though direct clinical trial evidence is lacking.^{75 76} Randomised trials of elective stenting have tended to show lower angina recurrence and second procedure rates at the price of more vascular complications.^{80 81} The latter are particularly associated with intensive

anticoagulant regimens that have now been superseded.⁸²

The overall average mortality for elective coronary bypass grafting or coronary angioplasty in stable angina is around 1%. The 1997 UK mortality rate for all patients having coronary bypass grafting was 3%, but this includes unstable angina and emergencies. In cost benefit analyses the need for repeat procedures reduces the cost advantage of coronary angioplasty over bypass grafting until this becomes insignificant at about five years.⁸³ It is important to note that the patients entered into these trials were those in whom both angioplasty and bypass grafting were technically feasible, and that neither stenting nor statins were available when the trial began. Angioplasty is not usually suitable for patients with unprotected left main stenosis, diffuse three vessel disease, or for some patients with chronically occluded coronary arteries.

Coronary revascularisation rates, whether by angioplasty or bypass grafting, have increased much more slowly in the UK than in several other countries. As for coronary angiography, this reflects both limited resources and limited purchasing. On the one hand, there is little doubt that patient and doctor preference is increasingly towards an interventionist approach; on the other, rationing of limited resources in the context of a free National Health Service is legitimate, particularly where the procedure is being performed to improve quality of life rather than to prolong survival.⁸⁴ As for coronary angiography there are major differences between health districts in revascularisation rates which are hard to explain rationally.⁵² In many centres an imbalance between rate of referral and refunded throughput has led to the accumulation of long waiting lists for coronary bypass surgery. The situation has been further complicated by the Patients' Charter requirements for all patients to receive treatment within one year. Several attempts have been made to devise scoring systems to determine whether patients should be offered surgical revascularisation; if this approach is adopted it is important that the criteria should be explicit, objective, and shared with patients and purchasers alike.⁸⁵ As an absolute minimum patients with severe symptoms in whom bypass grafting can objectively be shown to improve survival should be able to have surgery without delay.

REHABILITATION

Full restoration of patients to satisfactory social function and work may require more than relief of symptoms and correction of risk factors. Modern concepts of rehabilitation emphasise the importance of early explanation, full involvement of the patient and his or her family in the rehabilitation process, attention to psychological aspects of the diagnosis, and long term reinforcement of life style changes. These are discussed in more detail in the British Cardiac Society guidelines on cardiac rehabilitation.⁸⁶

Treatment summary

- Both angioplasty and bypass surgery give effective symptom relief [A]
- Bypass surgery improves one and five year survival in selected patient groups [A]
- Patients suitable for either intervention have similar operative mortality and success rates, but angioplasty patients are more likely to need repeat intervention [A]
- Intensive risk factor reduction is appropriate before and after intervention [A]

Early attention to correction of risk factors is important and longer term risk factor control must be maintained even when symptoms have been satisfactorily relieved. Medical treatment will usually be indicated as a first step, and while investigations are being completed. Choice of antianginal agents will be influenced by individual patient tolerance and the pattern of side effects. Combinations of more than two background antianginal agents can seldom be justified. Data are currently lacking concerning the ability of individual antianginal agents to prevent mortality or cardiovascular complications. Revascularisation may be indicated for prognostic reasons in patients with specific patterns of coronary disease, because medical treatment has been ineffective in controlling symptoms, or because of patient preference. There is currently a large gap in UK provision for revascularisation between what could be justified for the relief of symptoms and what can be funded under the National Health Service. Some form of rationing seems inevitable, and if introduced this should be objective and explicit.

Current management standards

PRIMARY CARE

General practitioners are the points of presentation for most patients with chest pain. General practice assessment should include:

- Clinical assessment of symptoms, in the light of knowledge of the patient, his family and environment
- Clinical examination to identify other possible causes of chest pain and other causes of angina (for example, anaemia, valve disease)
- Assessment of coronary risk factors (family history, smoking, diabetes, hypertension).

Further investigation at the general practitioner level should include a baseline 12 lead ECG and cholesterol/HDL cholesterol measurement.

Management at the general practice level focuses on advice/explanation, risk factor reduction (especially smoking), and medical treatment which would normally include low dose aspirin, sublingual glyceryl trinitrate, one or more background antianginal agents as discussed above, and, where appropriate, a lipid lowering agent.

The working party recommends that all newly diagnosed cases of angina should be referred for an objective assessment of myocar-

dial ischaemia by one of the methods described above, unless contraindicated by intercurrent illness, other disability, or patient preference. Such referral would usually be to a physician with special interest and training in cardiology. Open access referral for stress testing may be appropriate, provided there is an adequate mechanism for expert review and consultation if necessary. It is not intended that the physician to whom such referral is made should take over continuing care of the patient unless this is specifically requested. Referral is also indicated where the diagnosis is in doubt or where a positive diagnosis would have major implications for the patient's livelihood.

Referral for treatment is indicated for patients of any age with severe, unstable or rapidly progressive symptoms, for patients with secondary angina from a remediable cause, or for patients with unacceptable symptoms despite adequate medical treatment.

SECONDARY CARE

Secondary care may be provided by a physician with special interest and training in cardiology, or by a specialist cardiology unit acting in a secondary care role.

Facilities available at a secondary care referral centre should include:

- Advice available from a consultant or other specialist
- Exercise electrocardiography to confirm the diagnosis and for risk stratification
- Other non-invasive techniques, including myocardial perfusion imaging, as discussed above
- Access to a wider range of facilities for risk assessment and modification such as a lipid clinic
- Cardiac care unit with dedicated beds and monitoring facilities.

Management at secondary care level is essentially an extension of that at primary care level and should be a collaborative venture with the primary care team.

Referral from secondary to specialist cardiac care is indicated when intervention by angioplasty or bypass surgery is felt to be necessary on the basis of symptom severity or the severity of ischaemia as assessed by non-invasive testing. Referral may also be indicated when the diagnosis is in doubt, particularly in patients with recurrent hospital admissions for atypical symptoms. Where secondary care is provided by a number of physicians, only one of whom has specialist training in cardiology, it is desirable that referrals to a specialist cardiac centre should be channelled through the specialist physician. A close working relationship between secondary and specialist cardiac care is essential.

SPECIALIST CARDIAC CARE

In addition to providing expert advice, one of the major functions of specialist cardiac referral centres is to perform invasive investigations with a view to possible cardiac intervention. The principal resources required for this are a catheter laboratory suite, cardiac surgery operating facilities, an intensive care unit, and

associated inpatient beds. The extent to which investigative and interventional facilities can be separated has been debated; the risk of diagnostic angiography is small but it is accepted that angioplasty needs effective surgical back up. In practice specialist cardiac centres need to duplicate many of the non-invasive facilities of secondary care centres, and many units function as combined secondary and specialist care centres.

Specialist cardiac centres need to be organised so as to respond rapidly to emergencies; at the same time they must be efficient in dealing with routine cases. They need to set high educational and audit standards.

Audit points

These have been divided into three groups: "process audit" deals with the organisation of medical care; "personal care audit" deals with the services provided to the patient; and "outcome audit" attempts to measure whether the condition itself was altered as a result.

GENERAL PRACTICE

Ways of identifying angina patients for audit

Age/sex/disease register.

Nitrate prescriptions as marker for diagnosis of ischaemic heart disease.

Process audit

Practice policy/agreed referral policy with local centre?

Recording of standard dataset?

Referral rate monitored?

Prescription policy monitored?

Personal care audit

Where appropriate investigations requested?

Was appropriate advice given to patient?

Was family screened for risk factors?

Was referral made? Was it appropriate?

Is patient being followed up?

Outcome audit

The following clinical outcome measures are proposed as a basis for audit. They are primarily intended for patients under the age of 70 years without severe intercurrent illness and on appropriate medication.

Class 1

Symptoms—New York Heart Association (NYHA) or Canadian Cardiovascular Society (CCS) class 0 or 1.

Exercise test—completes Bruce stage 3 or equivalent.

Cholesterol—< 5.2 mmol/l.

Blood pressure—< 140/90 mm Hg.

Able to work or look after dependents.

Class 2

Symptoms—NYHA or CCS class 2 or less.

Exercise test—completes Bruce stage 2 or equivalent.

Cholesterol—< 5.2 mmol/l.

Blood pressure—< 140/90 mm Hg.

Able to work or look after dependents.

Class 3

Any other outcome.

SECONDARY CARE CENTRES

Ways of identifying angina patients

Outpatient/inpatient diagnostic register.

Process audit

Agreed referral protocol with local practitioners?

Priority for urgent referrals?

Outpatient waiting times?

Recording of standard dataset?

Exercise electrocardiography utilisation and reporting.

Seniority of doctor seeing patient.

Prescribing policy?

Referrals monitored?

Return visits monitored?

Discharge summaries timely and complete?

Participation in external audit scheme?

Personal care audit

Were appropriate investigations requested?

Were results recorded?

Was investigation treatment plan made and communicated to general practitioner and patient?

Was follow up appropriate?

Was specialist referral made?

Was it appropriate?

Outcome audit

As for primary care plus:

Emergency admission of patients referred with stable angina.

Re-referral rate.

SPECIALIST CARDIAC CENTRES

Ways of identifying angina patients

Outpatient/inpatient diagnostic register.

Process audit points

Agreed referral protocol with secondary centres?

Incoming referrals monitored?

Waiting times monitored, priority for urgent referrals?

Indications for angiography/angioplasty/surgery recorded and monitored?

Results/complications of angiography/angioplasty/surgery monitored?

Participation in external audit schemes?

Personal care audit

As for secondary care plus:

Indications for invasive investigation recorded? Results of invasive investigations recorded and communicated?

Indications for angioplasty/surgery recorded?

Outcome of angioplasty/surgery recorded and communicated?

Follow up plans recorded?

Outcome audit

As for secondary care.

Participants in the revision workshop and those providing written comments

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