

The investigation and management of stable angina

REPORT OF A WORKING PARTY OF THE JOINT AUDIT COMMITTEE OF THE BRITISH CARDIAC SOCIETY AND THE ROYAL COLLEGE OF PHYSICIANS OF LONDON

ABSTRACT—This paper is a summary of present practices in the investigation and management of angina. The mechanism, incidence, and prevalence of angina are reviewed, the roles of invasive and non-invasive investigations assessed, and the indications for coronary angioplasty and coronary bypass grafting discussed. Basic audit data sets for primary, secondary, and tertiary care are proposed and potential audit initiatives suggested.

Angina is a common symptom in both general and hospital practice. 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 symptom. This paper summarises the discussions and conclusions of the working group. Its full working papers are being edited for separate publication.

The cause of angina

Angina is usually the result of partial obstruction of a coronary artery by atheroma. Coronary atheroma is associated with several factors that include smoking, a raised plasma cholesterol concentration, high blood pressure, and diabetes (Table 1). It is more common in men, and increases in prevalence and extent with age. Coronary obstruction may develop gradually, or may occur rapidly as a result of thrombosis at the site of an atheromatous plaque in the vessel wall. It is possible for coronary atheroma to exist without causing obstruction and therefore be present without symptoms of angina. In a few patients, angina is due not to coronary artery disease but to aortic stenosis or hypertrophic cardiomyopathy. Angina can be made worse by anaemia or hyperthyroidism.

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Table 1. Principal factors associated with coronary atheroma

Smoking
Raised plasma total cholesterol and/or low density lipoprotein concentrations
Low plasma high density lipoprotein concentrations
Hypertension
Diabetes/glucose intolerance

The clinical diagnosis of angina

The most characteristic clinical feature of angina is retrosternal chest pain precipitated by physical or emotional exertion. It is relieved by rest [1,2]. 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 relationship to exertion is more characteristic than the precise site. Angina is often worse on effort in cold weather or after food. Pain which 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.

The association between 'typical' anginal symptoms and coronary artery obstruction is stronger in men than in women. 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. The discovery of localised chest tenderness often makes possible the positive diagnosis of musculoskeletal chest pain. An accurate *clinical* diagnosis is an essential step in the investigation and management of angina.

The incidence and prevalence of angina

The population *prevalence* (total cases per 100 population) of angina has been estimated at 1.1% of all patients in general practice aged between 30 and 59 [3], and 2.6% of all patients over 30 [4]. Estimates of

prevalence in middle-aged men based on answers to questionnaires range from 3.6% [5], 4.3% [6], to 7.9% [7]. The differences in these estimates can be explained, at least in part, by differences in the age of the study population: angina is more prevalent with increasing age. At all ages, angina is more prevalent in men than women.

The most reliable incidence estimates (new cases per 1,000 population per year), from a study which routinely used exercise testing and a cardiologist interview [8], are from 0.44/1,000/year (age 31–40) to 2.32/1,000/year (age 61–70) in men, and from 0.08/1,000/year (age 31–40) to 1.01/1,000/year (age 61–70) in women. Applying these results to the UK population gives an estimate of approximately 22,000 new angina cases per year.

In one study, 14% of *new* cases of angina developed complications (myocardial infarction or death) within six months from the time of presentation [9]. In two studies reported in the 1970s, the annual incidence of death or myocardial infarction in patients with *stable* angina ranged from 3% to 4.6% [10,11]; more recent data on unselected populations are not available.

The relation between angina, myocardial infarction and sudden death

Myocardial infarction results from the sudden complete obstruction of a coronary artery, usually by thrombus. The case fatality rate is about 30%, higher in the elderly, and 50% of deaths occur before hospital admission can be effected. Surviving patients often have permanent impairment of left ventricular function. Data from clinical trials in myocardial infarction indicate that about 25% of patients under the age of 70 presenting with myocardial infarction have previously recognised angina [12]; the proportion rises to 50% in older patients. The implication is that in the majority of young infarct patients thrombosis occurs in association with coronary atheroma which has not previously caused sufficient coronary obstruction to lead to angina. On the other hand, patients with symptomatic angina are at a greater risk of infarction than people without symptoms.

Apart from myocardial infarction, sudden death, often apparently associated with exertion, is more common in patients with angina. It is presumed to be due to a lethal arrhythmia resulting from sudden myocardial ischaemia.

Confirmation of diagnosis and risk stratification

The *resting 12 lead electrocardiogram* is important in diagnosing myocardial infarction, but insensitive in identifying other patients with coronary artery disease. An abnormal 12 lead ECG identifies a patient subgroup with a substantially higher risk of death or myocardial infarction, but a normal resting 12 lead ECG is not uncommon in patients with severe angina.

Exercise testing with electrocardiographic monitoring cannot be regarded *in isolation* as an effective screening test for ischaemic heart disease. The working group agreed that exercise electrocardiography should only be carried out after careful clinical evaluation, and the results interpreted by trained clinicians. This applies especially to populations with a low prevalence of ischaemic heart disease in which the proportion of false positive tests will be high [13]. False positive exercise recordings are also more common in women [14].

The discriminating ability of exercise electrocardiography is enhanced by qualified supervision during the recording. Time to the onset of electrocardiographic changes and/or symptoms, the overall exercise time, the blood pressure response, and the persistence into recovery of the electrocardiographic changes are all important [15,16].

In addition to its role in the diagnosis of ischaemic heart disease, exercise evaluation has an important role in risk stratification of patients in whom the diagnosis has already been made. This is further discussed below.

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 bypass grafting.

Radionuclide studies, in the form of perfusion scanning with thallium or other radionuclides, are sometimes a useful adjunct to exercise electrocardiography, particularly in patients whose resting electrocardiogram is abnormal.

Risk stratification is important both for choosing therapeutic options and for allocating resources.

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

Symptoms: severe symptoms, especially if accompanied by significant lifestyle limitation, indicate poorer prognosis. However, subjective assessment of symptoms is variable, and objectively assessed exercise tolerance is more reliable as a predictor. A good performance on exercise testing generally associated with a good prognosis [17,18].

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

Coronary arteriography: the extent and distribution of coronary arteriographic lesions predict outcome. Patients with left main coronary stenosis or three-vessel coronary disease have a poorer prognosis; patients with angiographically normal vessels or single stenoses have a good prognosis.

An algorithm for the assessment of patients with a clinical diagnosis of angina is shown in Figure 1. Applying this algorithm on a nationwide basis would require approximately 3,000 exercise tests per million of population per year, and 700–1,000 coronary angiographic studies per million of population per year [8,19]. These figures do not allow for other possible indications for these procedures.

Treatment

The objectives for treatment are:

- improving patient survival;
- enhancing quality of life.

Mortality rates for ischaemic heart disease in the USA, Scandinavia, and now the UK have been falling over the past few years. There are many possible reasons besides the specific effects of treatment, and it has been suggested that lifestyle changes leading to reductions in cigarette smoking and in serum cholesterol may account for at least half of the mortality reduction [20]. Aspirin, thrombolytic therapy, and beta-blockade improve survival. Treatment of hypertension, and of patients with heart failure using angiotensin converting enzyme inhibitors, also improves outcome [21–24]. Reduction in mortality has not, however, been accompanied by a similar reduction in morbidity: either because patients who would formerly have died now survive with angina, or because subjective appreciation of acceptable and unacceptable symptoms has changed.

Coronary bypass grafting improves survival in patients with left main coronary artery stenosis, with three-vessel coronary disease, particularly when this is associated with impaired left ventricular function, and in two-vessel coronary disease if one of the vessels involved is the left anterior descending coronary artery [25]. The effects of coronary bypass grafting on survival are greater in severely symptomatic than in mildly symptomatic patients. Coronary angioplasty has not been proven to prolong life.

Medical therapy for angina improves exercise tolerance and quality of life. There are sometimes theoretical and practical reasons for combining two different families of antianginal drugs, for example beta-blockers and calcium antagonists; evidence for an additional effect of adding a third class of drugs is scant.

Many patients with mild to moderate symptoms of angina are currently treated by general practitioners or general physicians. Patients presenting to cardiologists are likely to have more severe symptoms. Both coronary angioplasty and coronary bypass grafting are effective in relieving symptoms of angina, and both have low operative mortality rates (< 1% fatal outcome for elective surgery or angioplasty in good risk groups). About one-quarter of patients receiving angioplasty will develop restenosis over the next six months, and about one-sixth will have further angio-

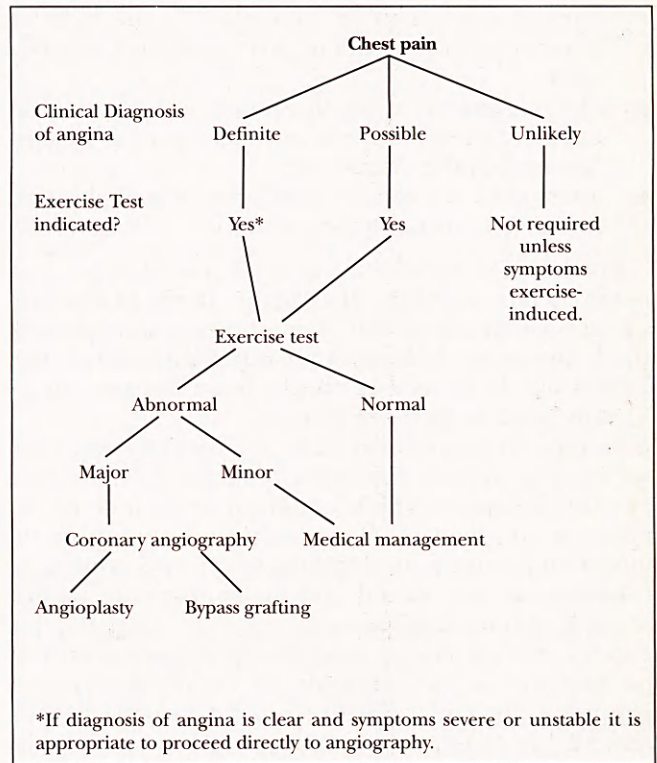


Fig 1. Flow chart for investigation of patient with suspected angina

plasty or coronary bypass grafting. Long waiting times for coronary bypass grafting are a relevant factor in choosing angioplasty for some patients.

There are marked variations in rates of coronary angiography [26], coronary angioplasty, and coronary bypass grafting in different regions and within parts of the same region. They exist despite apparent similarities in selection criteria. There is little doubt that, where facilities are available, patient and doctor preference is for an increasingly interventionist approach. UK operation rates for coronary angioplasty are 148 per million per year and for coronary bypass grafting 220 per million per year. They contrast with USA annual rates of approximately 1,000 per million for each procedure. A report of a joint cardiology committee of the Royal College of Physicians of London and the Royal College of Surgeons of England has recommended provision of facilities for 500 bypass operations and 300 angioplasty procedures per million of the population per year [27].

Current management standards

Primary care

General practitioners are the point 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/or other causes of angina (anaemia, valve disease, etc);
- assessment of coronary risk factors (family history, smoking, diabetes, hypertension).

Further investigation at general practitioner level might include a 12 lead ECG (either in the practice or via an open access service) and cholesterol/high density lipoprotein cholesterol measurement. (Note: the ECG is not to exclude ischaemic heart disease, but to identify possible high-risk patients.)

Management at general practice level focuses on advice/explanation, risk factor reduction [28,29], and medical treatment which would normally include, as appropriate, beta blockers, calcium antagonists or long-acting nitrate, in addition to glyceryl trinitrate. Low-dose aspirin would also be appropriate in the absence of contraindications.

Referral for specialist assessment is indicated for patients 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 therapy (Table 2). Referral is also indicated where the diagnosis is in doubt, or where a positive diagnosis would have major implications for the patient's livelihood (for example, heavy goods vehicle (HGV) drivers).

The working party recommends that all newly diagnosed cases of angina in patients under the age of 70 should have access to cardiological referral for further evaluation and, if appropriate, exercise electrocardiography. Treatment with antianginal drugs, if indicated, should *not* be withheld pending such referral. (In this context 'cardiological' referral means referral to a physician with special interest and training in cardiology.) 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.

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 echocardiography and radionuclide ventriculography and perfusion scanning;
- access to a wider range of facilities for risk assessment and modification, such as a lipid clinic;

Table 2. Target times for secondary referral

Patients with unstable or crescendo angina	Immediate or within 7 days (depending on clinical picture)
Patients with known angina whose symptom profile is worsening despite medication	7 days to 1 month
Patients with stable angina well controlled on medication	< 3 months
Patients with chest pain of uncertain cause, possibly angina	< 3 months
Patients with established angina whose lack of confidence is inhibiting a normal lifestyle	< 3 months
Patients with probable non-cardiac pain for clarification of diagnosis	< 3 months

- 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 will often be a collaborative venture with the primary care team.

Most cases of *acute myocardial infarction* are managed in secondary referral centres, ie district general hospitals. Referral from secondary to tertiary care is indicated when intervention by angioplasty or bypass surgery is felt to be necessary because 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, referrals to a tertiary centre should be channelled through the specialist physician.

A close working relationship between secondary and tertiary care is essential. There is a danger that the interposition of a secondary care step may introduce delay when speed is of the essence; conversely it is important that tertiary care centres should not become congested with cases that could equally well be managed elsewhere.

Tertiary care

In addition to providing expert advice, one of the major functions of tertiary care referral centres is to perform invasive investigations with a view to possible cardiac intervention. The principal facilities 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 surgi-

cal back-up. In practice, tertiary care centres need to duplicate many of the non-invasive facilities of secondary care centres, and many units function as combined secondary and tertiary care centres.

Tertiary care 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.

Basic data sets

Agreement on the amount and nature of essential information which should be recorded about an individual patient and his or her illness constitutes a basic or minimum data set. This information is important as a means of communicating between doctors, for example in the context of a referral letter, and can also be invaluable for audit and research. The suggested data sets listed in Tables 3–5 for patients presenting with angina should be regarded as minimal, and may need to be expanded in the light of experience.

Audit points

The following list of audit points is intended as a series of suggestions for audit rather than as a comprehensive prescription. Experience indicates that audit is often more successful if a limited number of points are tackled intensively than if a large number of issues are investigated half-heartedly.

Primary care

- Does the practice maintain an age/sex/disease register, and is angina one of the registered disorders?
- Is this register programmed to record nitrate prescriptions?
- What is the proportion of patients with a diagnosis of suspected angina in whose notes the standard data set (*p* 00) is recorded?
- Does the practice have a written policy for prescribing for angina, and is this policy monitored?
- Has the practice any agreed written policy on referral for further investigation of angina? Is referral monitored?

For individual patients in primary care

- Is there a written or computer record that:
 - risk stratification was undertaken for this patient;
 - investigations appropriate to the level of risk were requested;
 - appropriate advice was given about modifiable risk factors such as smoking or obesity;
 - appropriate referral was made according to the agreed referral policy;
 - a management plan was made for the patient, including a firm appointment for review?

Table 3. Basic data set: primary care

Patient identification: age, gender, ethnic group
Nature and history of present complaint
Relevant past or current medical history, eg asthma, diabetes
Medication
Family history
Smoking history
Occupation
Physical examination
Blood pressure
Any other significant social or medical factors
Working diagnosis
Investigations (optional):
urinalysis
plasma total + HDL cholesterol
haemoglobin
12 lead ECG

Table 4. Basic data set: secondary care

As for primary care data set plus urinalysis, plasma total + HDL cholesterol, haemoglobin, 12 lead ECG
Exercise electrocardiogram (if appropriate) documented in terms of protocol used, medication at time of test (if any), duration of exercise, ECG changes (if any), heart rate and blood pressure response and symptoms, reason for stopping test. Results of perfusion scan if available
Assessment of left ventricular function; clinical, echocardiographic or radionuclide ventriculogram
Written plan for future investigation and management
Reason for referral to tertiary centre

Table 5. Basic data set: tertiary care

As for secondary care level, plus:–
Documentation of indications for and results of coronary angiography, if performed
Documentation of indication for and results of PCTA/CABG, if performed
Written plan for future investigation and management, including referral back to secondary and primary care

Audit at secondary care centres

- Does the centre maintain an inpatient/outpatient diagnostic register of patients with coronary artery disease?
- Has the centre agreed a referral protocol with local general practitioners?
- Has the centre a system for giving priority to urgent referrals?

What is the proportion of patients categorised as urgent who have to wait for more than a week before being seen?

What is the proportion of patients not categorised as urgent who have to wait for more than eight weeks?

What is the proportion of standard data recorded in the medical records of a representative sample of patients?

What is the proportion of patients with an agreed level of risk stratification who have had an exercise electrocardiogram?

What is the proportion of patients who have not been seen by a consultant by the end of their second visit?

Is there a written prescribing policy for the outpatient management of angina? Is it monitored?

Are return visits monitored to ensure care is appropriately shared between the centre and general practice?

Does the secondary care centre participate in any external audit scheme?

Is there a written policy about referral to a tertiary care cardiac centre?

For audit of individual patients at the secondary care centre

Is there written or computer evidence that:
 risk stratification was performed for this patient;
 appropriate investigations were requested and the results recorded;
 the results of investigations and the treatment plan were communicated to the general practitioner, and that the patient has knowledge of this plan;
 the plan includes a firm statement about who is primarily responsible for the continuing care of the patient;
 if the patient was considered appropriate for referral to a tertiary care centre, that this referral was made?

Audit at tertiary care centres

All the audit measures for secondary care centres apply, but in addition:

is there a written policy for angiography, angioplasty and coronary bypass surgery;
 are there systems for monitoring rates and outcomes of these procedures?

For audit of individual patients at the tertiary centre

Is there written or computer evidence that:
 the indications for invasive investigation have been recorded;
 the results of such investigations have been recorded and communicated to the patient;
 the indications for angioplasty or bypass surgery have been recorded;
 the outcome of angioplasty or bypass surgery has been recorded, and plans for future management

communicated to the patient, general practitioner, and referring consultant?

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References

- 1 Heberden W. On Angina Pectoris. *Med Tr Roy Coll Phys* 1768, tr W Heberden Jr, 1818.
- 2 Matthews MB. Clinical Diagnosis. In *Angina Pectoris* (2nd edn). ed. Julian DG. Edinburgh, Churchill Livingstone, 1985.
- 3 Research committee, Northern Region Faculty, Royal College of General Practitioners. Study of angina in patients aged 30-59 in general practice. *Br Med J* 1982;285:1319-22.
- 4 Cannon PJ, Cannell PA, Stockley IH, *et al*. Prevalence of angina as assessed by a survey of prescriptions for nitrates. *Lancet* 1988;1:979-81.
- 5 Reid DD, Brett GZ, Hamilton PJS, *et al*. Cardiorespiratory disease and diabetes among middle-aged male civil servants. A study of screening and intervention. *Lancet* 1974;ii:469-73.
- 6 WHO European Collaborative Group. Multifactorial trial in the prevention of coronary heart disease. 1. Recruitment and critical findings. *Eur Heart J* 1980;1:73-80.
- 7 Shaper AG, Cook DG, Walker M, MacFarlane PW. Prevalence of ischaemic heart disease in middle-aged British men. *Br Heart J* 1984;51:595-605.
- 8 Gandhi MM, Lampe F, Wood DA. Incidence of stable angina pectoris. *Eur Heart J* 1992;13:181-9.
- 9 Duncan B, Fulton M, Morrison SL, *et al*. Prognosis of new and worsening angina pectoris. *Br Med J* 1976;1:981-5.
- 10 Fry J. The natural history of angina in a general practice. *J Roy Coll Gen Prac* 1976;26:643-8.
- 11 Kannel WB, Feinleib M. Natural history of angina in the Framingham study. Prognosis and survival. *Am J Cardiol* 1972;29:154-62.
- 12 Wilcox RG, Von der Liffe G, Olsson CG, *et al*. Trial of tissue plasminogen activator for mortality reduction in acute myocardial infarction. Anglo Scandinavian Study of Early Thrombolysis (ASSET). *Lancet* 1988;ii:525-30.
- 13 Froelicher VF, Thompson AG, Wolthius A, *et al*. Angiographic abnormalities in asymptomatic aircrewmen with electrocardiographic abnormalities. *Amer J Cardiol* 1974;39:32-40.
- 14 Melin JA, Wijns W, VanButsele RJ, *et al*. Alternative diagnostic strategies for coronary artery disease in women. *Circulation* 1985;71:535-42.

- 15 Detry JMR, Robert A, Luwaert TR, *et al.* Diagnostic value of computerised exercise testing in men without previous myocardial infarction. *Eur Heart J* 1985;**6**:227-38.
- 16 Mark DB, Shaw L, Harrell FE, *et al.* Prognostic value of a treadmill exercise score in outpatients with suspected coronary disease. *N Engl J Med* 1991;**325**:849-53.
- 17 Gordon DJ, Ekelund LG, Karon JM, *et al.* Predictive value of the exercise test for mortality in North American men. *Circulation* 1986;**74**:252-60.
- 18 Weiner DA, Ryan TJ, McCabe CH, *et al.* Prognostic importance of a clinical profile and exercise test in medically treated patients with coronary artery disease. *J Am Coll Cardiol* 1984;**3**:772-9.
- 19 MacRae CA, Marber MS, Keywood C, Joy M. The need for invasive cardiologist assessment and intervention; a ten-year review. *Br Heart J* 1992: in press.
- 20 Goldman L, Cook EF. Effects of medical intervention and lifestyle changes on mortality rates from ischaemic heart disease. *Ann Intern Med* 1984;**101**:825-36.
- 21 Nyman I, Larsson H, Wallentin L, and research group on instability in coronary disease in south east Sweden. Prevention of serious cardiac events by low dose aspirin in patients with silent myocardial ischaemia. *Lancet* 1992;**340**:497-501.
- 22 Norwegian multicentre study group. Timolol-induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. *N Engl J Med* 1981;**304**:801-7.
- 23 ISIS-2 Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988;**ii**:349-60.
- 24 Report of the British Hypertension Working Party: Treating mild hypertension. *Br Med J* 1989;**298**:694-8.
- 25 Joint AHA/ACC Task Force: Guidelines and indications for coronary artery bypass graft surgery. *J Am Coll Cardiol* 1991;**17**:543-89.
- 26 Gray D, Hampton JR, Bernstein SJ, *et al.* Audit of coronary angiography and bypass surgery. *Lancet* 1990;**335**:1317-20.
- 27 Fourth Report of a Joint Cardiology Committee of the Royal College of Physicians of London and the Royal College of Surgeons of England. Provision of services for the diagnosis and treatment of heart disease. *Br Heart J* 1992;**67**:106-16.
- 28 Royal College of General Practitioners. Guidelines for the management of hyperlipidaemia in general practice. *Occasional Paper No. 55*, London, Royal College of General Practitioners, 1992.
- 29 Rossouw JE, Lewis B, Rifkind BM. The value of lowering cholesterol after myocardial infarction. *N Engl J Med* 1990;**323**:1112-8.

This report, including background papers submitted to the Joint Audit Committee, is to be published as a book at a later date.