# Stable angina pectoris
## evaluation and treatment

**The investigation and management of a patient presenting with chest pain are guided by the probability of the patient having significant coronary artery disease.**

**CON ARONEY**
MD, FRACP

Dr Aroney is Director of Cardiology, Holy Spirit Northside Hospital, Brisbane, Qld; Associate Professor of Medicine, University of Queensland; and a member of the Clinical Issues Committee, Heart Foundation of Australia.

Stable angina pectoris is a common manifestation of atherosclerotic coronary artery disease but its recognition may be difficult and the diagnosis greatly increases the risks of sudden death and myocardial infarction. Coronary artery disease (CAD) is a diffuse and progressive disease. Intracoronary ultrasound assessment of the coronary arteries of patients with at least one severe coronary lesion demonstrated atherosclerotic plaque in 93% of the entire coronary artery tree.[1] Its management, therefore, must include long term control of all coronary risk factors, aggressive medical management and surveillance for development of ischaemic and thrombotic complications in all vascular beds.[2,3]

The presence of stable angina increases the risk of myocardial infarction to about one in four in men (slightly less in women) in the next five years; the risk of death over eight years is 30% and about half of these deaths will be sudden. The severity of the anginal symptoms may not be an indication of the burden of coronary disease, and patients with mild symptoms sometimes have severe life threatening proximal left anterior descending artery or multivessel disease. The presence of coronary disease is associated with atherothrombotic disease and complications in the cerebrovascular and peripheral arteries, and these blood vessels should always be clinically assessed.

Unstable angina is caused by thrombosis or spasm of atherosclerotic coronary lesions. Clinically, a change in the pattern, severity or frequency of the discomfort is defined as unstable angina. It carries a high risk of major cardiac events, and is an indication for urgent assessment and even ambulance transfer in the event of prolonged discomfort. Patients manifesting anginal symptoms for the first time in the previous month are considered

**IN SUMMARY**

- **The probability of significant coronary artery disease (CAD) in a patient presenting with chest pain guides further investigation and management, and can be assessed with a simple diagnostic algorithm.**
- **The differential diagnoses of stable angina pectoris include musculoskeletal chest pain, gastro-oesophageal pain, peptic ulceration, biliary disorders and anxiety/hyperventilation.**
- **For patients with an intermediate pretest probability of CAD and mild or equivocal symptoms, assessment with a provocative stress study is useful for both diagnosis and risk stratification.**
- **Beta blockers are first line therapy for symptom control as they most effectively reduce exercise and stress induced tachycardia. Sublingual glyceryl trinitrate relieves anginal symptoms. Antiplatelet agents, ACE inhibitors and statins improve cardiovascular outcomes.**
- **Percutaneous coronary intervention or coronary artery bypass grafting is indicated particularly in patients with troublesome or refractory symptoms or where significant ischaemia is identified.**
- **Patients should be counselled regarding long term control of risk factors.**

unstable because some will have atherothrombotic disease with a definite risk of progression to myocardial infarction. These patients should be treated with beta blockers and aspirin and cardiac assessment should be obtained urgently.

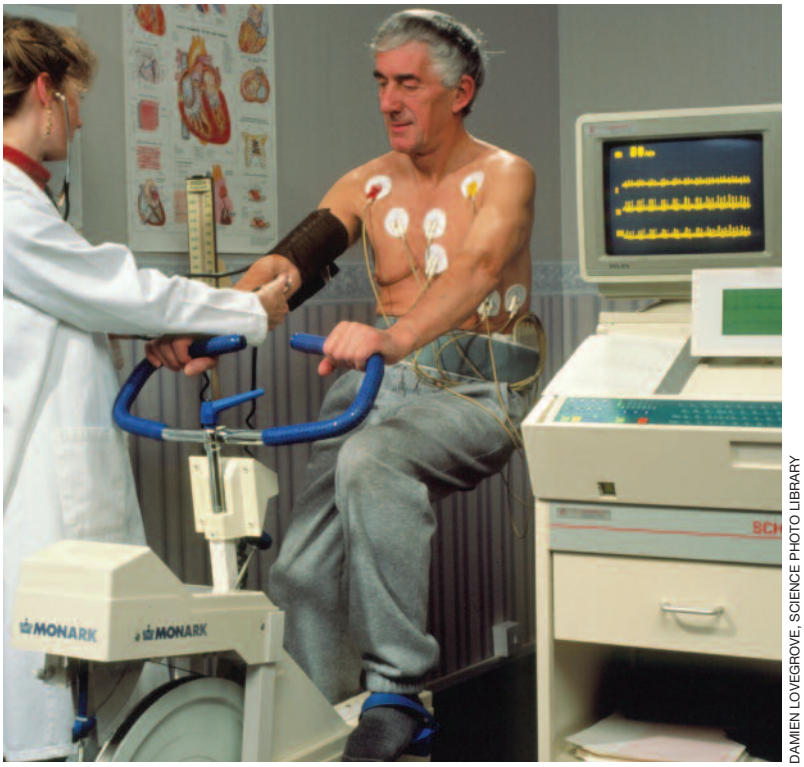
## Features and classification of stable angina

### Features

A patient with exertional chest discomfort in whom the pattern of pain has not changed in the past two months is considered to have stable angina pectoris. The four cardinal features of stable angina are:

- location – usually central or upper chest with or without radiation to the arms, neck or jaw; sometimes the discomfort is centred in the back, shoulders or epigastrium
- provocation/relief – the discomfort is provoked by exercise, emotional stress, cold weather or meals, and relieved with rest or glyceryl trinitrate
- character – the discomfort is often described as heavy or compressing, and may be mild or severe (severe symptoms usually reflect severe CAD; mild symptoms may reflect mild, moderate or severe CAD)
- duration – the discomfort usually lasts for one to 10 minutes after discontinuation of exertion.

Some patients, particularly women, the elderly and those with diabetes, may have atypical presentations such as discomfort in the shoulders, back or epigastrium. Stable angina may also be manifested by or associated with exertional dyspnoea, nausea or faintness.




DAMIEN LOVEGROVE, SCIENCE PHOTO LIBRARY

Figure. Exercise stress ECG testing (using either a bicycle or a treadmill) is useful for both diagnosis and risk stratification in patients with angina.

### Classification

The features described above are used to classify chest pain as typical, nontypical or noncardiac.[4]

- Typical angina (definite angina) – meets the three criteria of (1) substernal chest discomfort with a characteristic quality and duration that is (2) provoked by exertion or emotional stress and (3) relieved by rest or glyceryl trinitrate.
- Atypical angina (probable angina) – meets

**Table 1. Pretest likelihood of coronary artery disease in symptomatic patients, according to age and sex[4]***

| Age (years) | Nonanginal chest pain | | Atypical angina | | Typical angina | |
|---|---|---|---|---|---|---|
| | Men | Women | Men | Women | Men | Women |
| 30 to 39 | 4% | 2% | 34% | 12% | 76% | 26% |
| 40 to 49 | 13% | 3% | 51% | 22% | 87% | 55% |
| 50 to 59 | 20% | 7% | 65% | 31% | 93% | 73% |
| 60 to 69 | 27% | 14% | 72% | 51% | 94% | 86% |

* Each value represents the percentage of patients with significant coronary artery disease on catheterisation.
Table reproduced from Snow V, et al. Evaluation of primary care patients with chronic stable angina: guidelines from the American College of Physicians. *Ann Intern Med* 2004; 141: 57-64, with the permission of the American College of Physicians.

**Table 2. Differential diagnoses of stable angina**

| |
|---|
| Musculoskeletal chest pain |
| Gastro-oesophageal pain |
| Peptic ulceration |
| Biliary disorders |
| Anxiety and/or hyperventilation |

two of the three typical angina criteria.
- Noncardiac chest pain – meets one or none of the typical angina criteria.

## Probability of CAD

The probability that chest discomfort is due to obstructive CAD may be more than 90% in patients with typical anginal symptoms, particularly in middle aged and older patients.[4] Based on age, gender and symptoms, Table 1 provides the pretest likelihood (probability) of CAD in symptomatic patients.[4]

Cut off points applied to the pretest probability figures further classify the probability of significant CAD into low, intermediate and high:[4]
- low probability of CAD – values below 10%
- intermediate probability – values between 10 and 80%
- high probability – values above 80%.

**Table 3. Risk factors for stable angina**

| |
|---|
| **Modifiable** |
| Smoking |
| Physical inactivity |
| Abdominal obesity |
| Hypertension |
| Diabetes |
| Dyslipidaemia – elevated LDL-cholesterol, low HDL-cholesterol, elevated triglycerides |
| Renal disease, including microalbuminuria/proteinuria |
| **Nonmodifiable** |
| Older age |
| Male gender |
| Family history of premature CAD |

## Prognosis

The prognosis in a patient with angina is dependent on age, left ventricular (LV) function, the severity and location of coronary lesions (particularly left main, proximal left anterior descending artery and three-vessel disease) and the severity of LV ischaemia (remembering that mild angina can be a manifestation of severe ischaemia).

## Pathophysiology

The pathophysiology of angina pectoris is dependent on the balance of myocardial oxygen demand and supply. In addition to obstructive lesions reducing the oxygen supply, many comorbid conditions influence demand and supply of oxygen, and these should always be considered at initial diagnosis and during follow up.

Myocardial oxygen demand is increased in conditions such as tachycardia, hypertension, hyperthyroidism, fever, anxiety, aortic stenosis and hypertrophic cardiomyopathy. Myocardial oxygen supply is decreased in conditions such as anaemia, hyperviscosity (e.g. polycythaemia), obstructive sleep apnoea and pulmonary disease.

## Differential diagnoses

The differential diagnoses of stable angina are listed in Table 2. Recurrent localised or positional discomfort suggests a musculoskeletal cause. In patients with retrosternal discomfort, an upper gastrointestinal endoscopy is often useful when coronary investigations are negative.

## Assessment and investigation

### Risk factors

Modifiable and nonmodifiable risk factors that should be assessed in a patient with angina are listed in Table 3.

The metabolic syndrome is a constellation of risk factors that imparts a major risk for developing cardiovascular disease and diabetes.[5] The new International Diabetes Federation consensus definition of metabolic syndrome is based on the presence of abdominal obesity together with two of low HDL-cholesterol, elevated triglycerides, hypertension or previously treated hypertension, and elevated fasting blood glucose or previously diagnosed diabetes (see the box on metabolic syndrome on this page).[6] All of these components are risk factors for CAD, but the

**The International Diabetes Federation consensus definition of metabolic syndrome[6]**

The International Diabetes Federation defines metabolic syndrome as the combination of:
- abdominal obesity, defined as waistline circumference:
  - in Europids, ≥94 cm in men and ≥80 cm in women
  - in Asians, ≥90 cm in men and ≥80 cm in women

plus any two of:
- low HDL-cholesterol (<1.03 mmol/L in men and <1.29 mmol/L in women)
- high triglycerides (≥1.7 mmol/L)
- hypertension (systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg) or previously treated hypertension
- elevated fasting blood glucose (≥5.6 mmol/L) or previously diagnosed diabetes.

combination contributes a greatly increased risk. Lifestyle changes are capable of reducing the progression to type 2 diabetes and the development of adverse cardiovascular events.

### Physical examination

The physical examination of a patient with stable angina should pay particular attention to:

- obesity – particularly waist circumference, using the values in the box on the metabolic syndrome on page 46 for waistline circumference indicating abdominal obesity
- tendon xanthomas
- xanthelasmas
- corneal arcus in patients under 45 years
- reduced peripheral pulses, vascular bruits (especially carotid and femoral)
- hypertension
- signs of aortic stenosis or hypertrophic cardiomyopathy (both of these conditions may present with anginal symptoms).

### ECG

All patients with angina should have an ECG performed to evaluate for arrhythmias, previous myocardial infarction (Q waves), LV hypertrophy or ischaemic ST/T changes (Table 4). Also left bundle branch block or resting ST depression will mean that stress ECG testing will not be helpful, and the patient should be considered for a different provocative study.

### Pathology testing

A fasting blood sample should be taken for a full lipid profile (including LDL- and HDL-cholesterols) and blood glucose level, as well as full blood count, electrolyte levels, renal function, liver function tests (LFTs) and creatine kinase level (Table 4). These tests will screen for diabetes, anaemia and atherogenic dyslipidaemia as well as provide a baseline reference range for potential side effects of future medical treatment with antiplatelet and lipid lowering agents and ACE inhibitors. Patients will require long term follow up of LFTs and creatine kinase with lipid lowering therapies and of renal function and electrolytes with ACE inhibitor treatment.

### Echocardiography

Echocardiography should be considered if there is evidence of valvular heart disease, heart failure, previous myocardial infarction, complex ventricular arrhythmias, hypertrophic cardiomyopathy or pericardial disease.

### Stress testing

In very elderly or frail patients with multiple comorbidities, further investigations may not be helpful and medical management is usually preferred, unless symptoms are severe and refractory to treatment.

For patients with an intermediate pretest probability of CAD and mild or equivocal symptoms, assessment with an exercise stress ECG test or other provocative stress study is useful for both diagnosis and risk stratification (Figure). This further risk stratification is essential because mild anginal symptoms may be present in patients with severe underlying coronary disease.

Interpretation of an exercise ECG requires the patient to be able to achieve at least 80% of their predicted maximum heart rate and involves assessment of changes in the ST segment with exercise. Hence a stress test other than exercise should be considered in patients unable to achieve this heart rate level or who have features that affect the ST segment response to exercise (such as pre-existing ST abnormalities, left bundle branch block, LV hypertrophy or the taking of digoxin or β-blockers). Where progression of disease is being evalusated and diagnosis is less important, β-blockers may be continued.

Patients with severe symptoms or who have a high pretest probability of CAD should be referred for coronary angiography as this is the most cost effective option in these patients.[7]

Features on exercise stress ECG study that indicate a high prognostic risk are:

- inability to complete stage II of the Bruce protocol (a standardised multistage exercise test)
- 1 mm of ST depression before completing stage II of the Bruce protocol
- 2 mm ST depression or more at any time
- persistent ST depression for more than five minutes into recovery
- ventricular tachycardia or heart failure
- fall in blood pressure of 10 mmHg or more.

The presence of any of the above features is considered a strong indication for coronary angiography.

Patients should not be referred for exercise stress ECG testing if they have the following features:

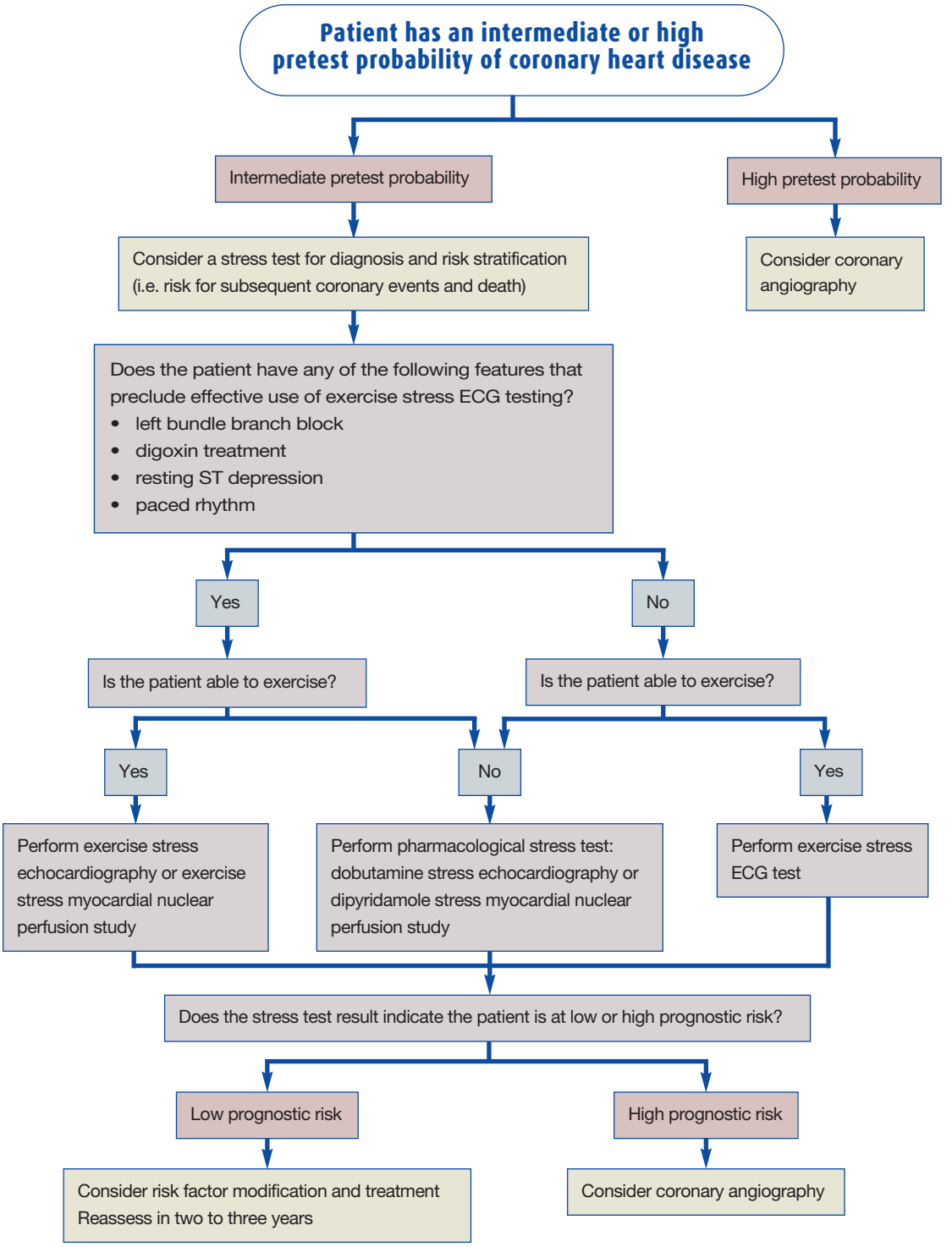
- inability to exercise
- left bundle branch block
- more than 1 mm ST depression at rest
- digoxin treatment
- paced rhythm
- Wolff–Parkinson–White syndrome

**Table 4. ECG and pathology testing in stable angina**

| ECG |
|---|
| Rate and rhythm |
| Q waves |
| Left ventricular hypertrophy |
| Left bundle branch block |
| Ischaemic changes |
| **Pathology testing** |
| Full blood count |
| Fasting serum lipid profile, including LDL-cholesterol and HDL-cholesterol |
| Fasting blood glucose |
| Electrolytes |
| Renal function tests |
| Thyroid function tests |
| Liver function tests |
| Creatine kinase |

**continued**

## Evaluation of possible stable angina pectoris



- high risk unstable angina (e.g. troponin elevation or ST deviation)
- heart failure
- uncontrolled hypertension.

The sensitivity of exercise stress ECG testing is only 65 to 70%, compared with 80 to 85% with a myocardial perfusion study or exercise stress echocardiography.[8] False positive stress ECG results are most common in women with atypical symptoms; in these patients, alternative testing should be considered.

Patients unable to exercise may be referred for dobutamine stress echocardiography, a dipyridamole stress myocardial nuclear perfusion study or, in selected cases, coronary angiography. Stress echocardiography and stress perfusion imaging should be considered in cases where the stress ECG is equivocal, and are also recommended to localise ischaemia in patients with known coronary anatomy and previous revascularisation. Stress echocardiography has a superior specificity to myocardial nuclear perfusion studies and provides accurate information on cardiac anatomy and function, whereas a myocardial nuclear perfusion study has superior sensitivity and a higher rate of technical success. The number, size and location of wall motion or perfusion abnormalities or LV dysfunction indicate the level of risk and the requirement for coronary angiography. A stress study should be considered every two to three years in patients with clinically stable angina to assess for progressive disease.

The evaluation of possible stable angina for patients with intermediate and high pretest probabilities of CAD is summarised in the flowchart on page 48.

### Angiography

The indications for coronary angiography in stable angina are listed in Table 5. International guidelines recommend that patients with a high pretest probability of CAD may be most cost effectively managed by direct referral for coronary angiography.[7] Other patients with difficult or recurrent symptoms may also prefer coronary angiography, which remains the gold standard for the diagnosis of coronary disease. Angiography may be combined with a 'pressure wire' assessment of fractional flow reserve in selected cases. This provides a functional assessment of intermediate lesions that closely correlates with myocardial perfusion study results.

**Table 5. Indications for coronary angiography in stable angina**

| |
|---|
| Refractory angina |
| Severe ischaemia on provocative study |
| High pretest probability of CAD |
| LV dilatation or dysfunction |
| High risk occupations (such as aircraft pilots) |
| Aortic stenosis or hypertrophic cardiomyopathy |
| Troublesome symptoms of uncertain aetiology after inconclusive noninvasive testing, where ischaemic heart disease needs to be excluded |
| Patient preference in selected cases |

## Management

The aims of treatment in a patient with stable angina are to control symptoms, reduce premature death and reduce myocardial infarction.

Low dose aspirin, ACE inhibitors and lipid lowering therapy reduce death and myocardial infarction in patients with proven CAD. Beta blockers are considered first line therapy for symptom control as they most effectively reduce exercise and stress induced tachycardia. An elevated baseline heart rate is also associated with coronary plaque rupture,[9] which may explain some of the benefit of β-blocker therapy. Second line therapies include long acting nitrates, nicorandil (Ikorel) or calcium channel blockers for symptom control. Patients with refractory pain despite multiple treatment may respond to perhexiline (Pexsig) therapy. New research in this area includes pure heart rate lowering agents (ivabradine) and newer metabolic agents (ranozaline).

Table 6 lists the currently available drugs used in the first, second and third line therapies of stable angina.

**Table 6. Medical treatment of stable angina**

**First line therapy**

Low dose aspirin (100 mg daily), or clopidogrel (Iscover, Plavix) where aspirin not tolerated

Beta blockers, or long acting calcium channel blockers where β-blockers are contraindicated – for symptom control

Sublingual glyceryl trinitrate (Anginine, Lycinate, Nitrolingual Pumpspray) – for anginal symptoms

**After diagnosis of CAD is confirmed:**

ACE inhibitors

Lipid lowering therapy (particularly statins), in high doses if tolerated

**Second line therapy**

Long acting nitrates (oral or transdermal)

Nicorandil (Ikorel)

Long acting calcium channel blockers

**Third line therapy**

Perhexiline (Pexsig)*

*After commencement of perhexiline, serum levels should be measured at 10 days, 30 days and then three-monthly to exclude toxicity.

### Lipid lowering therapy

Recent trials (such as the Heart Protection Study [HPS], Treating to New Targets [TNT] study, Pravastatin or Atorvastatin Evaluation and Infection Therapy [PROVE-IT] study and Reversal of Atherosclerosis With Aggressive Lipid Lowering [REVERSAL trial) have shown that high dose statin therapy provides the greatest clinical benefits and that these doses may greatly enhance plaque stability. LDL-cholesterol lowering therapy with a

## Table 7. Secondary prevention of stable angina

### Risk factor control

**Diet**

Low in saturated fat and trans-fatty acids

High in omega 3 fish oils

**Physical activity**

At least 30 minutes of moderate intensity physical activity on five or more days each week

**Smoking**

Cessation of smoking and avoidance of passive smoking

**Weight**

Desirable waist measurement of:

- ≤94 cm in men (≤90 cm for Asian men)
- 80 cm or less in women

**Lipid levels**

Use of lipid lowering therapy

Target LDL-cholesterol of <2.0 mmol/L

**Blood pressure**

Use of antihypertensive therapy

**Diabetes**

Identification of undiagnosed diabetes mellitus

Maintenance of optimal blood glucose level in patients with diabetes

### Management

**Medication use**

Aspirin or clopidogrel, ACE inhibitors, β-blockers, statins and glyceryl trinitrate

**Rehabilitation**

Access to cardiac rehabilitation services

**Unstable angina and prolonged chest pain**

A written plan

statin, in high doses if tolerated, should aim for a target LDL-cholesterol of less than 2.0 mmol/L in patients with documented coronary disease. Updated US guidelines (the *National Cholesterol Education Program Adult Treatment Panel III guidelines, 2004*) have recommended a lower target of less than 1.8 mmol/L in high risk patients with proven cardiovascular disease plus either multiple risk factors, poorly control - led risk factors, diabetes or other features of the metabolic syndrome.[10]

Patients with CAD and high serum triglycerides (greater than 2 mmol/L) should be managed with weight reduction, reduced alcohol intake, increased physical activity and smoking cessation, plus glycaemic control in diabetics. Where necessary, fibrate therapy should be considered in addition to statin therapy. Current research in this area includes phase 3 clinical trials of new cholesterol ester transfer protein inhibitors that greatly increase HDL-cholesterol while lowering serum triglycerides and further reducing LDL-cholesterol.

## The roles of fish oils, folate and vitamin E

Omega 3 fish oil supplements reduce the risk of cardiac events, particularly sudden cardiac death, and should be considered in all patients who are not already eating a diet rich in salmon and tuna.

Although elevated serum homocysteine is associated with greater cardiac risk, it is not proven to be causal and may be a bystander, being closely correlated with renal function and plasma fibrinogen, which are both known risk factors. Serum homocysteine is reduced with folate supplementation, but there is no current evidence that folate therapy improves patient outcomes in these patients. In fact, the largest and most recent study showed that combinations of supplementary folic acid and vitamins $B_6$ and $B_{12}$ actually increased the risk of myocardial infarction and stroke (the NORVIT study).[11] In this study, the risk of adverse events was highest in the subgroup with the highest baseline homocysteine level. Earlier studies of folate therapy also demonstrated no clinical benefit of folate therapy in patients with ischaemic heart disease or after ischaemic stroke.[12-14] Similarly, several large randomised studies have shown no benefit of vitamin E supplements in reducing cardiovascular events (the Heart Outcomes Prevention Evaluation [HOPE] study and the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico [GISSI] trial).[15,16]

It should be noted that management aimed at surrogate markers, such as serum homocysteine, has a poor track record when examined in cardiovascular trials. Many cardiologists advocated antiarrhythmic drugs to reduce ventricular ectopy in an effort to improve outcome, but when examined in a randomised clinical study the treated patients had an increased mortality (The Cardiac Arrhythmia Suppression Trial [CAST]).[17] The same appears to be true of folate therapy, and yet many cardiologists have advocated measuring serum homocysteine and then treatment with folate therapy in the absence of any proven benefit. The only arbiter of benefit is improved clinical outcome, not an improvement in a surrogate marker, and widespread advocacy of new treatments should not be promoted until benefit is proven. The widespread policy of measuring and treating elevated serum homocysteine with folate and B group vitamins should be discouraged at present until clinical benefit is proven.

## Treatment in patients with associated conditions

Patients with stable angina and associated hypertension are optimally treated with combination therapy that includes β-blockers and ACE inhibitors, although long acting calcium channel blockers and diuretics may also be required. Patients with the combination of angina, diabetes and hypertension have a very high cardiovascular event rate, and require optimal hypertensive and glycaemic control. Patients with associated heart failure, as well as being assessed for possible revascularisation, will particularly benefit from selected β-blockers (carvedilol [Dilatrend, Kredex], bisoprolol [Bicor] or extended release metoprolol [Toprol-XL]), ACE inhibitors and long acting nitrates, with diuretics where required.

### Revascularisation

Percutaneous coronary intervention or coronary artery bypass grafting is indicated particularly in patients with troublesome or refractory symptoms, and are more effective in controlling myocardial ischaemia than medical therapy.[18,19] Health status, severity of angina and major adverse cardiac events have been shown to be significantly improved by invasive management in elderly patients with chronic stable angina.[20]

Revascularisation with percutaneous intervention or bypass surgery is of most benefit when there is a large amount of myocardium at risk, and improves survival in patients with high risk ischaemic or anatomical features (such as left main disease, multivessel disease with impaired LV function and proximal left anterior descending artery disease). Revascularisation, particularly with percutaneous intervention, also reduces death and myocardial infarction in patients who develop unstable angina.

### Patient counselling

Patients should be counselled regarding risk factor control and the use of medications, including glyceryl trinitrate (Table 7). They should also be educated about how to manage unstable angina and prolonged chest pain, which carry a significant risk of myocardial infarction or sudden death. (see the patient handout 'What are the symptoms of heart attack?' on this page).

## Follow up visits

A patient's clinical status, risk factors and tolerance of and compliance with medical therapy should be assessed at each follow up visit. Questions that may be considered are listed below.

- Have the patient's anginal symptoms worsened? Do they occur more frequently, are they more severe, are they now occurring on less exertion?
- Has the patient developed dyspnoea?
- Has the patient's level of physical activity decreased?
- Is the patient tolerating therapy?
- Are risk factors being adequately addressed?
- Has the patient developed comorbid illnesses? Are any comorbid illnesses contributing to worsening symptoms?

## Conclusion

The pretest likelihood of CAD in patients with suspected stable angina pectoris is based on a simple diagnostic algorithm. The diagnosis is confirmed with either noninvasive or invasive testing, and risk stratification is essential even in patients with mild symptoms. Aggressive control of risk factors and medical therapy with antiplatelet agents, ACE inhibitors and statins improve cardiovascular outcomes. Revascularisation should be considered in patients with severe symptoms or with high risk features on noninvasive testing. MT

**DECLARATION OF INTEREST:** Dr Aroney has received support from Pfizer, Merck Sharp and Dohme, Sanofi-Synthelabo and Eli Lilly.

MedicineToday **PATIENT HANDOUT**

### What are the symptoms of heart attack? A guide for all adults

Heart attack is the single largest cause of death in Australia. The most common symptom is chest pain but this does not always occur – other symptoms include discomfort or tightness in the chest, arms, neck or jaw that lasts for 10 minutes or more, and breathlessness.

#### Do's

- If the discomfort lasts for 10 minutes or more then assume that you may be having a heart attack. Call an ambulance.
- Take one aspirin tablet (chewed or swallowed) while you are waiting for the ambulance. Everyone should have aspirin in the house.
- If you have a history of angina or previous heart attack and have glyceryl trinitrate tablets (Anginine or Lycinate) or spray (Nitrolingual Pumpspray), use this under the tongue when the discomfort begins. Continue to use either the tablets or spray every 5 minutes if the discomfort persists while you are waiting for the ambulance.
- If you live in a remote or rural area, it may be appropriate to have a relative or friend drive you to meet the ambulance. This should be discussed with the rural ambulance or Royal Flying Doctor Service.

#### Don'ts

- Don't assume that the discomfort is caused by indigestion. You may be right, or you may be making a fatal mistake instead. It's not worth the risk.
- Don't waste time by calling your local doctor. He or she will only advise you to call an ambulance.
- Never wait until the next morning or 'the next available appointment' before seeking help. Your heart can be permanently damaged in the meantime and may even stop beating. Don't take the chance – call an ambulance immediately.
- Don't be embarrassed if the pain turns out to be indigestion or due to another cause. Hospital doctors know that it is vitally important to rule out a heart attack and they will not be concerned if the problem turns out to be something else.
- Don't drive yourself or get someone to drive you to hospital. It is much safer to call an ambulance.

This patient handout was prepared by Dr Con Aroney.