

Ischaemic Heart Disease/Chest Pain

Part 3: Chronic IHD

Editor: Christine Connors

Authors: This chapter is made up of extracts (with permission from the authors and publisher) from W Walsh, I Ring, A Brown, A Boyden and S Couzos, 'Ischaemic Heart Disease', in *Aboriginal Primary Health Care: An evidence-based approach*, second edition, Oxford University Press, Melbourne. The authors acknowledge Prof Lindon Wing, Prof Andrew Tonkin, Ass/Prof Stephen Colaguirri, and Ass/Prof David Sullivan.

[Editor: Readers can refer to the full chapter of the above mentioned work for detailed information on the prevention and management of ischaemic heart disease, treatment goals and targets, case management, program implementation, data collection and performance indicators.]

Implementation summary

Cardiovascular conditions, particularly ischaemic heart disease, are the major cause of death for Aboriginal and Torres Strait Islander people.

Comprehensive primary health care, including broad population health education and ongoing preventive care, and access to appropriate specialist and hospital services is fundamental, but not sufficient, to bring about an improvement in cardiovascular health for Aboriginal and Torres Strait Islander people. Approaches should also include the involvement of Aboriginal people and Torres Strait Islanders in determining their own communities' health priorities and how they will be addressed, and in designing and delivering a range of services.

Burden of suffering

Diseases of the cardiovascular system are the biggest single cause of deaths and of excess deaths for the Aboriginal and Torres Strait Islander population.^{1,2,3,4}

Cardiovascular conditions account for 30% of both deaths and of excess deaths of Aboriginal and Torres Strait Islander peoples. Over half (57%) of cardiovascular disease (CVD) deaths in the Aboriginal and Torres Strait Islander population are due to ischaemic heart disease (IHD). Age-specific CVD mortality rates for Aboriginal and Torres Strait Islander people are higher than for the non-Indigenous populations throughout adult life and are seven to twelve times higher than the overall Australian population in the 25–54 year age group.

The average age of death from CVD in Aboriginal and Torres Strait Islander people is 59 compared with 79 for the non-Indigenous population in 1998.⁵

Hospital admission rates of Aboriginal and Torres Strait Islander people for CVD are two to three times higher than for the rest of the Australian population, with Indigenous males having higher admission rates than females. The average age at admission was 47 years, almost 20 years younger than for the total population in 1998–99.^{2,4}

In the last thirty-five years the age-standardised mortality from IHD has fallen by 70% in the total Australian population. Although there may have been some reduction in mortality from heart disease in Indigenous females, there does not appear to have been a significant reduction in heart disease mortality in Indigenous males between 1991–97.

Cardiovascular health comparisons with the Maori population in New Zealand and Native Americans in the United States of America have demonstrated that IHD mortality is 1.5 times higher in the Australian Indigenous population than in Maoris and 2.6 times higher than in Native Americans. Mortality rates from IHD in Maoris fell from levels in 1974 (which were above the current Australian Indigenous rates) to a third below those rates by 1990–94, suggesting that rapid and sizeable reductions in IHD mortality in Indigenous populations are possible.⁶

There are some explanations for the greater and growing relative disadvantage faced by Indigenous Australians in cardiovascular morbidity and mortality. There have been very large increases in the prevalence of type 2 diabetes. This is a potent risk factor which more than doubles the likelihood of developing IHD. Smoking rates are more than twice those of the non-Indigenous population (51% compared to 23%).⁷

Obesity is also more common, with Indigenous Australians far more likely to have BMI >30 than non-Indigenous Australians. In 1995, Indigenous Australian adults were more likely to be physically inactive in their leisure time (42% females were inactive compared with 38% of males).

Conventional risk factors may only explain approximately 60–70% of the IHD burden. A number of studies have demonstrated that psychosocial stress — including social isolation, poverty, hopelessness and lack of empowerment — are associated with significant increases in the prevalence of IHD.^{8,9,10,11} Lower socioeconomic status is also associated with increased levels of high-risk behaviour and therefore increased prevalence of cardiovascular risk factors. The high prevalence of IHD in low socioeconomic populations has also been related to suboptimal intra-uterine development and low birth weight (LBW).¹¹

Chronic psychosocial stress has also been related to prognosis in patients with IHD. The mortality rate in those who have suffered acute myocardial infarction (AMI) is higher if depression is present, as well as a lack of quality social support.^{12,13,14} Depression and psychosocial factors (psychological traits such as hostility, depression and anxiety, work characteristics, and social supports) also predict the development of CVD (myocardial infarction or coronary related death) in initially healthy people, as shown in systematic reviews of cohort studies.^{15,16}

Diagnosis of stable angina

It is important to have a high index of suspicion concerning symptoms suggesting angina, as patients may confuse them with indigestion, unfitnes and the like. Many patients may delay seeking help from health providers until symptoms have become quite limiting. Initial investigation is a twelve-lead electrocardiogram (ECG). This is often normal in those with stable IHD. However, it may show non-specific ST-T wave changes or evidence of an old myocardial infarction with the presence of Q-waves. The presence of an abnormal ECG significantly increases the likelihood of the patient having IHD, especially in males. Chest X-ray is frequently not helpful in the diagnosis of IHD as heart size is usually normal. The diagnosis of IHD in those with suspected angina is usually confirmed with non-invasive exercise ECG, otherwise known as a 'stress test'.

Some patients may present with atypical chest pain which has some features of angina but the diagnosis is not clear. Stress testing is useful in these patients, as patients with non-cardiac cause of chest pain would be expected to have normal exercise tests. Patients who develop early symptoms of angina during exercise with marked ST-segment depression on the ECG are likely to have multi-vessel disease and need more aggressive management. Patients who perform well with minimal ECG changes at a good level of exercise have a good prognosis and can usually be managed conservatively with medications only. In some cases the test is not diagnostic, or patients may be unable to exercise adequately because of co-morbidity. In these cases nuclear stress

myocardial perfusion imaging (NSMPI) is very helpful as pharmacological provocation can be used to induce myocardial ischaemia. An alternative is stress echocardiography.

Patients with stable symptoms should be referred for further assessment, including coronary angiography, if: (a) their anginal symptoms are interfering with their normal lifestyle, or (b) they have a moderately or strongly positive exercise ECG or evidence on NSMPI of significant myocardial ischaemia.

A note on acute coronary syndromes

[Editor: Acute ischaemic heart disease is covered in an earlier chapter. Warren Walsh adds: 'In short, the current correct term is acute coronary syndromes, which are divided into ST segment elevation and non-ST segment elevation acute coronary syndromes (ACS). ST segment elevation ACS is a medical emergency, which requires the prompt administration of thrombolytic drug, out of hospital if necessary as described in the chest pain protocol. Non-ST Segment elevation ACS includes unstable angina (UA) and non-ST segment elevation myocardial infarction (NSTSEMI). The treatment of UA and NSTSEMI is the same. These patients need admission to hospital but there is time to allow transfer.']

Effectiveness of prevention

The prevention of IHD has been divided into the following two approaches. Primary prevention strategies relate to interventions that may prevent the onset of IHD, especially in people who have increased risk factors for CVD. Secondary prevention strategies relate to optimal strategies to prevent further deterioration in those who have already been diagnosed with CVD, and who are therefore at future risk of another cardiovascular event.

Primary prevention

[Editor: There is strong evidence supporting the impact of primary prevention. For information about smoking cessation, weight reduction, diet changes and physical activity refer to the relevant sections in this reference book or to Aboriginal Primary Health Care: An evidence-based approach.]

Secondary prevention of coronary heart disease

A summary of the relative risk reduction in CV endpoints attributed to secondary prevention interventions is shown in table 1 on the following page.

Crucial need for early detection of ischaemic heart disease

There is evidence that the prevalence of pre-existing and undiagnosed CHD, even in young Aboriginal people (<37 years), is high.¹⁷ The rate of autopsy-examined sudden death attributable to IHD was 5.5 times higher for Aboriginal people than for non-Aboriginal people in the NT.¹⁸ A high prevalence of undiagnosed IHD was detected by ECG screening in one Kimberley population survey.¹⁹ However, screening the Aboriginal and Torres Strait Islander population with ECG has not been recommended and is unlikely to be useful. The use of an initial ECG in the assessment of newly diagnosed clients with diabetes is recommended. Early detection will facilitate further diagnostic investigations. An exercise ECG in clients with stable symptoms to investigate the severity of ischaemia may improve outcomes if interventions occur at a time when the risk of complication and progression is lower.

Modifying diet

There is evidence that advising people with CHD to eat more fish (fish oil or fish oil capsules), fruit and vegetables, bread, pasta, potatoes, and olive oil (i.e. a more 'Mediterranean' diet) leads to survival benefits.²⁰ The evidence supporting a low fat or high-fibre diet in reducing mortality from CHD is less compelling²⁰, but there is strong evidence these interventions (including weight reduction) can reduce CV risk factors such as hypertension, hyperlipidaemia and possibly prevent diabetes.

There may be a need to advise moderation of alcohol intake because of its impact on diet and weight. Studies have consistently shown that blood pressure increases in direct proportion to alcohol intake, and reducing heavy alcohol consumption will reduce blood pressure.

Increasing physical activity

There is evidence from a number of systematic reviews that physical activity is effective in the primary prevention of CHD.²¹ All people should aim to participate in moderate intensity physical activity for 30 minutes or more on most or all days of the week for health benefit. The evidence for the effectiveness of exercise alone to reduce CV outcomes in those with existing CHD is not compelling. However, when combined in cardiac rehabilitation programs with other forms of secondary prevention, physical activity significantly reduced CV mortality by 20–25%.²⁰ Physical activity can increase the risk for sudden death, but this risk in the individual is outweighed by the benefits. Healthy Aboriginal sportsmen aged 15–37 years in the NT had a 40 times higher risk of sudden death than their non-Aboriginal counterparts in Victoria in a cases series from 1982–96.¹⁷ All deaths occurred in the hottest time of the year, and were attributable to underlying IHD.

[Editor: Community strategies to reduce this risk include playing sport during cooler times of the day (late afternoon, evening), installing lights at football fields, ensuring sufficient fluid intake both prior to and during the games, avoiding alcohol the night prior to games, and local health staff providing health education, including recognition of CVS symptoms, during preseason training.]

Psychological or stress management

There is some evidence to support the effectiveness of stress management using relaxation therapy and other counselling strategies to reduce rates of AMI and death in people with CHD.²⁰ In two meta-analyses of randomised controlled trials, psycho-educational (health education and stress management) programs for coronary heart disease patients yielded: reductions in cardiac mortality; recurrence of myocardial infarction; and significant positive effects on blood pressure, cholesterol, body weight, smoking behavior, physical exercise, and eating habits.^{22,23} The applicability of these findings to Aboriginal and Torres Strait Islander populations is unclear, given that social and emotional support can take a number of forms, and most determinants have to do with socioeconomic disparities which are beyond the control of individuals.

Smoking cessation

The primary and secondary prevention of CV events has been demonstrated in many observational studies. Patients with CHD who stop smoking reduce their risk of recurrent coronary events or premature death by 50% compared with continuing smokers.²⁰

Aspirin

Some of the strongest evidence for the prevention of further CV events in those with established CHD pertains to the use of aspirin.²⁴ Aspirin, even in relatively low doses reduces the risk of serious vascular events in those with previous CV events (including AMI, angina) at doses as low as 75 mg/day, which is as effective as higher doses.²⁵ Unless contra-indicated, aspirin should be given to all patients indefinitely with acute coronary events or for secondary prevention in patients with chronic ischaemic disease. In those who are intolerant or allergic to aspirin alternative treatment is available with the antiplatelet agent clopidogrel (300 mg loading dose, 75 mg daily). Systematic review of trials have shown that clopidogrel is clinically equivalent to aspirin for reducing cardiovascular risk without the gastrointestinal side effects of aspirin.^{26,27}

β-blockers

Data from pooled meta-analyses suggest that early intervention and long-term treatment of patients following an acute coronary syndrome with beta-blockers significantly reduce the risk of patients suffering death, cardiac arrest or another myocardial infarction.²⁵ The American Heart

Association and the American College of Cardiology recommend the use of beta-blockers in all patients without contra-indications, post AMI or acute coronary syndrome for an indefinite period.²⁸

ACE inhibitors

ACE inhibitors should be considered for all patients suffering an AMI within 24 hours of onset, and continued for at least 5–6 weeks unless contra-indicated based on efficacy established in large clinical trials.²⁹ ACEi therapy as secondary prevention can reduce the rate of death, hospitalisation for heart failure and recurrent AMI for those with evidence of left ventricular dysfunction (e.g. heart failure).³⁰

Recent randomised controlled trials show that even those without heart failure can benefit from ACE inhibitor therapy.³¹ Treatment with ramipril was associated with significant declines in the rates of death, AMI, stroke, coronary revascularisation, cardiac arrest and heart failure as well as the risk of complications related to diabetes.

Controlling blood pressure

The benefits of treating hypertension in terms of reduced CVD mortality and morbidity are well established from randomised controlled trial evidence.³²

International and Australian blood pressure management guidelines emphasise that the management of hypertension should not only be influenced by an individual's blood pressure level, but also take into account the factors that worsen an individual's prognosis and increase the absolute risk of adverse CV outcomes.^{32,33} The numerous intervention trials that have demonstrated the clear benefits of blood pressure reduction in patients with hypertension in terms of reduced CV morbidity and mortality have not necessarily been designed and/or powered to determine blood pressure targets for intervention. The HOT study³⁴, did investigate targets but this showed no significant differences in CV outcomes between three blood pressure target groups (diastolic blood pressure <90, 85, or 80 mmHg respectively). Subgroup analysis, however, did demonstrate improved CVD outcomes in diabetics in the low target group.

Cholesterol-lowering agents

[Editor: There is very strong evidence for the benefit of lipid-lowering agents, particularly the 'statins.' The benefits and cost effectiveness are greatest for (but not limited to) secondary prevention in those with existing ischaemic heart disease. For more detail on this see the chapter on lipids in this book, or Aboriginal Primary Health Care: An evidence based approach.]

Integrating secondary prevention through cardiac rehabilitation

An important part of the post discharge management of CHD is enrolment in a cardiac rehabilitation (CR) program where emphasis is on secondary prevention and health education. There is now strong evidence, collated in a number of systematic reviews, that comprehensive risk factor management extends overall survival, improves quality of life, decreases the need for interventional procedures (such as angioplasty and bypass grafting) and reduces the incidence of subsequent myocardial infarction. The most significant benefits of CR programs are a reduction in longer-term mortality. Overseas studies have shown significant reductions in non-fatal reinfarction rates of 61% and between 25–30% in total cardiac events for those who undertake CR (table 2).³⁵

These ratings are dependent upon demonstration of benefits from randomised controlled trials (1,2) and/or supported by observational studies (3). While all are supported by authoritative opinion, none is solely dependent on such opinion (4).

The National Heart Foundation of Australia recommends that, unless contraindicated, CR and secondary prevention programs should be offered to all patients with CVD.³⁶ However, evidence

suggests that Aboriginal people access CR to a very limited degree compared to the non-Aboriginal population³⁷

Case management

Stable angina

Risk factor modification (increased physical exercise, dietary modification including reduction in fat intake, and smoking cessation) has been found to be effective in those with stable angina.³⁸ Most patients require initiation of a statin drug for lipid control, since benefits have been shown with patients who have a total cholesterol of 4 mmol/L or greater in patients with known CAD.³⁹

Smoking is a powerful risk factor for coronary artery disease and cessation is essential. Aspirin has been shown to be quite beneficial in stable angina²⁴

Beta-adrenergic blocking agents have also been demonstrated to improve ischaemic symptoms and prognosis in acute coronary syndromes. Despite a lack of mortality trials using beta-blockers in chronic stable angina there is no reason to believe why efficacy would not apply to these patients⁴⁰

Calcium channel blockers may also be used in patients with chronic stable angina⁴¹ These have been shown to improve symptoms but there is controversy about their effect on prognosis. Long-acting drugs such as verapamil or diltiazem, slow A-V conduction and hence heart rate, and have been demonstrated to be very effective in providing symptomatic control. Long-acting nitrates are also of benefit symptomatically. Topical nitrates are especially useful for nocturnal symptoms. There is no evidence that nitrates improve prognosis but they do enhance symptomatic control.

Recent studies have suggested that ACE inhibitors may also be beneficial in patients with stable IHD. A large study of the ACE inhibitor ramipril in patients who are at high vascular risk demonstrated significant improvement in clinical outcome independent of blood pressure reduction. ACE inhibitors are particularly beneficial in patients who have left ventricular dysfunction post AMI or who have clinical heart failure. In the HOPE study of 9297 patients without clinical heart failure, ramipril significantly reduced the risk of composite endpoint of cardiovascular death, AMI and stroke from 17.7% to 14.1%, a relative risk reduction of 22% (P<0.01). This is the first of the large ACE inhibitor trials in patients with chronic IHD and other trials are currently in progress using other ACE inhibitors⁴²

Unstable angina

[Editor: These patients will present with increasing frequency or severity of angina. ECG shows no ST segment elevation. Due to the high risk factor profile of many Aboriginal and Torres Strait Islander people, patients presenting with unstable angina should be managed in consultation with the appropriate specialist, and should be admitted to hospital for further investigation. Also see note on acute coronary syndromes above.]

Patients need to have their future CV risk stratified for management purposes. Numerous studies have now identified that elevated cardiac troponin I or T or CKMB, new or reversible ST segment depression on ECG, and recurrent anginal symptoms are indicative of high risk. The presence of clinical heart failure and depressed LV function (e.g. echocardiography) also signifies high risk. These patients require referral to a tertiary centre for coronary angiography and consideration of revascularisation.⁴³ Three large randomised trials have shown significant reductions in the combined endpoint of recurrent AMI, in-hospital mortality and hospital readmission when an invasive approach is carried out in this high-risk population^{44,45}

Intermediate-risk patients are those with a history of previous myocardial infarction, previous bypass surgery or coronary angioplasty and/or have diabetes. Aboriginal and Torres Strait Islander

patients who are at intermediate risk should also be referred for coronary angiography. Patients who lack the above features, have normal cardiac markers or enzymes and a normal ECG are deemed to be at low risk and can be managed locally without the need for referral to a tertiary institution. These patients do not benefit from an invasive strategy.

Implementation of programs

There are clearly many barriers to the appropriate care of Aboriginal and Torres Strait Islander clients with established cardiovascular disease. Whilst data is limited, there is evidence that Aboriginal patients frequently delay presentation to hospitals with ST-segment AMI.⁴⁶ Similar differential delays were noted for the performance of diagnostic ECGs and the delivery of nitrate therapy, heparin and lipid-lowering drugs.

The reasons for the delay in presentation are complex and relate to cultural, education and distance factors. Typical AMI symptoms appear less likely to be recorded for Aboriginal and Torres Strait Islander patients, often because of communication difficulties with mainstream health providers.^{46,47}

There is evidence that Aboriginal people are significantly less likely to undergo procedures such as coronary artery bypass surgery or coronary angioplasty compared to the non-Aboriginal population.⁴⁵

The structure and benefits of cardiac rehabilitation (CR) programs both in Australia and overseas have been reviewed⁴⁸, yet there is little appraisal of the provision of cardiac services to Indigenous population groups. During the development of CR services in the Top End of the NT, it was demonstrated that only 8% of eligible clients admitted to the Royal Darwin Hospital were recruited to rehabilitation services.⁴⁹

A number of Cardiac Rehabilitation resources have been developed for use in rural, remote, Aboriginal and Torres Strait Islander communities. The 'Heart Story'⁵⁰ is a manual developed for health workers to use with Aboriginal people who are at risk or who have suffered a cardiac event. 'Promoting heart health'⁵¹ is an educational resource manual for rural and remote health workers. The Heart Health Manual training resource for Aboriginal health workers has recently been released.⁵²

There is insufficient evidence of the impact and potential benefits of targeted cardiovascular prevention programs within remote Aboriginal communities or for the Aboriginal and Torres Strait Islander population within larger metropolitan centres.

Between 1995 and 1998, a systematic treatment program was developed in a remote NT Aboriginal community to modify renal and CV disease.⁵³ The main focus of the trial included health education about diet, exercise, health behaviours and medical therapy. The study found that the introduction of a systematic treatment program was associated with improvements in blood pressure, stabilisation of renal function, decreases in the rates of renal failure and all-cause mortality (including cardiovascular deaths). It was concluded that a systematic approach to the treatment of cardiovascular and renal disease risk factors, with screening and treatment algorithms and clear goals was of tremendous value.

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