1 Background

Recent advances in prevention and treatment of heart, stroke and vascular diseases, and a lower prevalence of some of their associated risk factors, have been associated with increased life expectancy and improved quality of life for many Australians. Despite these advances, cardiovascular disease remains the largest cause of premature death and death overall in Australia and most other developed nations. It is also responsible for a greater proportion of health and economic burden than any other disease in Australia. The major factors contributing to this burden are the managements related to cardiovascular conditions, e.g. hospitalisation, diagnostic and surgical procedures, cardiovascular co-morbidity increasing length of stay for other conditions, general practice consultations and prescriptions associated with cardiovascular conditions[1-4].

As cardiovascular disease is generally acknowledged as a disease of the elderly, the number of Australians with these diseases is likely to increase dramatically over the next 20 to 30 years as the average age of the population increases. The burden associated with the increase in heart failure, stroke and heart attack is therefore also likely to increase. Reducing the impact of this burden falls, to a great extent, on general practitioners. Being consulted by approximately 80% of Australians annually[5], and being the main access point to the health system for most Australians, there is an increasing expectation for GPs to be more active in lifestyle counselling and continually up-to-date regarding medications, tests and techniques available for prescription or referral to manage these conditions.

Although some of the risk factors associated with cardiovascular disease are physiological or familial, many premature deaths and a significant amount of morbidity could be reduced by decreasing risk behaviours such as tobacco smoking, physical inactivity and poor diet. These activities are also important in preventing and managing type 2 diabetes, lipid disorders and hypertension, all conditions which are associated with eventual development of other cardiovascular disease. A number of people exhibit a clustering of these risk behaviours, despite already being managed for one or more cardiovascular conditions by their general practitioner.

This report differs from previous BEACH reports which have to date been based largely on encounter data, i.e. on information about the overall problems managed and treatments provided in general practice. This report does include an examination of some encounter data, but only that pertaining to the topic of interest—cardiovascular problems. It does not provide detailed information on the management of specific cardiovascular problems as this topic was covered in an earlier report[6].

The current study includes information about encounters with patients for whom a cardiovascular disease was managed, the characteristics of patients with cardiovascular problems and the GPs who managed these conditions. It also investigates self-reported risk behaviours of patients at subsamples of encounters involving management of cardiovascular problems and the prevalence of cardiovascular-related problems in patients at general practice. Changes in the management of cardiovascular problems over the 10-year period 1990–91 to 1998–00 are also examined.

1.1 Aims

This report:

- describes the characteristics of GPs who managed patients with cardiovascular problems and defines the group of GPs most likely to see cardiovascular problems more frequently
- describes the encounters with these patients and the types of cardiovascular problems managed
- describes the characteristics of patients who have cardiovascular problems managed at general practice encounters
- describes changes in the management of cardiovascular problems over the 10-year period 1990–91 to 1998–00
- estimates the prevalence of cardiovascular-related problems in general practice
- examines selected self-reported risk behaviours of the general practice patients for whom cardiovascular problems were managed.

1.2 A review of the literature—risk factors, risk behaviours and cardiovascular problems

This study looks at general practice encounters at which a cardiovascular problem was managed. It further focuses on the risk behaviours of patients who have been identified as having a cardiovascular problem. For this reason, the main objective of this literature review is to describe the risk factors and risk behaviours which cause cardiovacular disease initially, and which exacerbate cardiovascular problems once established. The risk behaviours reported in the substudy populations are smoking, alcohol consumption and being overweight; risk factors such as hypertension, diabetes and lipid disorders are also examined. These topics are therefore examined more through the literature. Other risk factors and risk behaviours not reported on in this study, but nevertheless important in the development of cardiovascular disease, have been covered to a lesser degree. The topics are also presented from a health system perspective as much as from that of general practice. Although GPs may be well placed to effect change in risk behaviours through counselling, the problems associated with risk behaviours are systemic—the same risks affect the population at large as they do general practice patients in particular.

Cardiovascular disease (CVD) incorporates all diseases pertaining to the heart and blood vessels including coronary heart disease, stroke, heart failure and peripheral vascular disease[7]. It kills more Australians annually than any other disease[3]. Associated illness, disability and ensuing healthcare costs, which exceed those of any other disease, categorise cardiovascular disease as a major health and economic burden for Australia[2] and have resulted in its inclusion as one of Australia's National Health Priority Areas.

The underlying cause of many forms of cardiovascular disease is atherosclerosis, a common type of arteriosclerosis in which deposits of plaque containing cholesterol, lipoid material and lipophages are formed within large and medium-sized arteries[8]. Atherosclerotic plaques basically develop in three stages—dysfunction of the vascular endothelium, development of a fatty streak and fibrous cap formation[9]. These plaques

reduce the capacity of vessels to supply blood. The most serious effects occur when vessels supplying blood to the heart muscle become clogged, leading to angina or a heart attack, or to the brain which can result in a stroke. Peripheral vascular disease results from damaged or blocked vessels supplying blood to the legs or other peripheral structures.

Cardiovascular disease accounted for 39% of all deaths in Australia in 2000[10]. With around 80 deaths each day from coronary heart disease, it is the highest single cause of death in Australians aged less than 70 years. The second greatest killer is stroke, which claimed almost 12,500 Australians in 2000, averaging 9.7% of all deaths in Australia per year [10]. Stroke is also the principal cause of long-term disability in adults[3,7]. Peripheral vascular disease accounted for 1.6% of deaths from all causes in 1998–99 as well as over 700 amputations and 13,612 hospitalisations with an average length of stay of 8.0 days [10,11].

In 1993–94 cardiovascular disease accounted for 12% of the total health system costs representing the largest proportion of health system costs in Australia[1,4,10]. Public and private hospital costs due to cardiovascular diseases during that period totalled $1.5 billion, with a further $700 million spent on drugs for their treatment and prevention[3]. Cardiovascular diseases account for approximately 8% of all hospital separations each year[7,10,11]. They also comprised 11.1% of all problems managed by general practitioners annually between 1998–99 and 2000–01[12-14]. For each of these periods, almost 15% of all prescriptions provided by GPs were for cardiovascular medications, antihypertensives being the drugs most frequently prescribed. Nationally, one-fifth of all medications prescribed in the community in 1997 were for cardiovascular drugs[3]. By 1998 this figure had risen to almost one-quarter of all prescriptions[4].

Although advances in treatment and the introduction of prevention strategies have contributed to a steady decline in cardiovascular diseases over the past 30 years, these diseases remain the single largest cause of premature death and death overall in Australia. As cardiovascular diseases most commonly affect older persons, the progressive ageing of Australia's population is likely to result in an increasing demand on the healthcare system in the future, as levels of drug treatment and other interventions, and their associated costs continue to rise[7].

Risk factors

Cardiovascular diseases share a number of risk factors, both physiological and behavioural. Some of these risk factors such as age, gender and family history cannot be influenced, but many risk factors are reducible or preventable. Cigarette smoking, excessive alcohol consumption, poor nutrition, physical inactivity, excess weight, hypertension, hyperlipidaemia, stress and diabetes[3,15] are all risk factors for heart disease. Atrial fibrillation is a further risk factor for stroke. Some population groups have a higher risk of cardiovascular disease than other groups and risk factors themselves can be influenced by other characteristics such as education, living conditions, economic resources, working conditions, social influences and access to health care[3].

Age and sex

Increasing age and being male are risk factors for cardiovascular disease. The prevalence of cardiovascular disease increases with age, particularly between the ages of 40 and 70 years. For example, in 1995 over 60% of people aged 75 and over had a cardiovascular condition compared with less than 9% of those aged 35 or under[3]. The risk for both sexes increases greatly with age, but at any age the risk for males exceeds that for females. A

report based on data from the Framingham Heart Study[16] found that at all ages men had a higher lifetime risk of developing coronary heart disease than women. Before age 40 the risk is low (1.2% in men, 0.2% in women). However, at age 40 the risk for men is about one in two (48.6%) and one in three for women (31.7%)[3,16]. Even at age 70, the risk for men is one in three, and for women it is one in four.

While the overall mortality rate from heart disease has declined in recent years, the decline has been greater for men than for women. Since 1984, more women than men (in absolute numbers) have died each year from heart disease, a fact accounted for by the greater number of women than men in the population, particularly among the elderly[17]. In many cases, age helps to level out the sex differences in risk for cardiovascular disease.

Sex and age differences have been observed in survival outcome following hospitalisation for myocardial infarction (MI)[18]. In patients under 50 years old, women had an almost threefold higher risk for death than men, although this odds ratio decreased with advancing age such that it was close to 1.00 by 70 years of age. Women often have a worse prognosis compared with men once heart disease has been confirmed[17], but men are still at far greater risk of developing heart disease initially. A recent study of Finnish men and women found that men were three times more likely to develop heart disease and five times more likely to die of heart disease than women[19]. Differences in major cardiovascular risk factors (smoking, cholesterol levels, body mass index and diabetes), particularly in the youngest participants, accounted for half of the sex difference in incidence and mortality. In general, risk factors carry a similar level of risk for both men and women. One exception is diabetes—not only is diabetes more strongly related to heart disease in women than in men, but also it removes their health advantage with respect to heart disease[17].

Family history

Several studies have concluded that family history of cardiovascular disease is in itself an independent risk factor, as well as having additive effects when combined with other risk factors[20,21]. Family history of stroke has been found to be an independent risk factor for all stroke types combined and for cerebral infarction, especially among individuals with a parental history of stroke[22-24]. Other studies have concluded that most early cardiovascular events occur in families with a positive family history of cardiovascular disease[25,26], that family history is the most important risk factor in prevention of coronary heart disease from youth[27] and that family history may be a favourable indicator of familial burden for coronary heart disease in situations where genetic or clinical information is unavailable[28].

A positive family history of coronary heart disease indicates a high risk for premature coronary heart disease independent of traditional and non-traditional risk factors[29,30]. Szamosi et al. (1999) examined children of parents with premature coronary heart disease (observed prior to 45 years of age) for body fat, blood pressure, blood cholesterol and blood sugar levels, and concluded that risk factors of atherosclerosis are detectable in children and adolescents of high-risk families[31]. Although genetic factors are certainly involved, the family environment can add to the risks, as many family members often share the same behavioural patterns of diet, alcohol consumption, smoking and level of physical activity[20,22,23]. Family history should feature prominently in targeting high-risk subjects for the prevention of cardiovascular disease[25,30,32].

Smoking

Smoking of tobacco products increases the risk of cardiovascular diseases as well as a range of cancers and other morbidity[3]. It is the risk factor associated with the greatest disease burden in Australia[33] with approximately 13% of deaths from cardiovascular disease attributed to its practice[1,34]. Smoking is regarded as the most important modifiable risk factor because it approximately doubles the risk of death from a heart attack and is entirely preventable[7,35,36]. Furthermore, cessation of smoking reduces the risk of heart, stroke and peripheral vascular disease within 2 to 5 years, to levels similar to those for people who have never smoked[7].

Cigarette smoking contributes to the development of coronary atherosclerosis by damaging the vascular endothelium. Nicotine alters the expression of a number of endothelial genes whose products help to regulate vascular tone, leading to endothelial cell dysfunction and the gradual onset of coronary atherosclerosis[37]. It increases arterial wall stiffness reducing blood vessel distensibility and compliance, and is associated with increased fibrinogen levels, increased platelet aggregation, increased hematocrit and decreased high-density lipoprotein (HDL) cholesterol levels[36]. Smoking has been found to acutely affect cerebral blood flow[38] and, when combined with other risk factors, considerably increases the risk of cardiovascular diseases even in the young. Smoking, even of short duration and moderate consumption, has been found to have a detrimental impact on lipid profiles of boys as young as 15 to 18 years[39]. The risk of myocardial infarction from smoking in women aged 16 to 44 years is considerable, heavy smokers with other risk factors being particularly at risk[40]. Stroke risk is greatly increased for teenage girls and young women who smoke in combination with the use of oral contraceptives[36,41-43].

In Australia, the number of people who smoke daily is gradually declining. In 1995, 24% of adults were daily smokers[44]. In 1998, 21.8% smoked daily, and by 2001 this percentage had reached 19.5%[45]. Similarly, the percentage of adults who have ceased smoking or have never smoked has gradually increased[45].

Because smoking has been identified as the single most important cause of premature death in developed countries and is rapidly becoming a major health concern in most developing countries, the World Health Organization (WHO) is increasing efforts to control what it describes as a 'global tobacco epidemic'[46]. Its aim through studies such as the MONICA Project, a multinational study set up to monitor the trends and determinants in cardiovascular disease, is to determine whether changing prevalence is resulting from initiation or cessation of smoking and which groups should be targeted for public health intervention strategies. Many of the countries in the study showed a reduction in the prevalence of smoking. However, population groups from some countries, particularly men in China and eastern and central Europe, and women in many populations, have an increasing prevalence, a factor attributed to powerful socioeconomic forces resulting from a global economy[46].

Two Australian communities participated in the MONICA Project (Perth and Newcastle). Over the 10-year period of the study there was a significant reduction in the prevalence of smoking[47], a decreasing trend reported in the number of daily smokers, an increase in the number of people who have never smoked, and an increase in the number of former smokers for both population groups[46]. These trends supported the findings of the 2001 National Drug Strategy Household Survey. The study concluded that reducing the prevalence of smoking should remain a high public health priority in all countries.

Alcohol

Consumption of alcohol has long been considered a risk factor for cardiovascular disease. Level of consumption and patterns of drinking produce different health outcomes, some negative and some positive. The negative effects of excessive long-term alcohol consumption are cardiac arrhythmias[36], haemorrhagic stroke[41], aortic stiffness[48], systemic hypertension[36,41,49], congestive cardiomyopathy, cerebral vascular incidents and reduced left ventricular ejection fraction[49]. Consuming sporadic large amounts or 'binge' drinking can result in embolic stroke or acute myocardial infarction[50]. The rapid change in both systolic and diastolic blood pressure during and after intoxication may increase the likelihood of strokes even in young drinkers[51].

During recent years, many observational studies have reported a demonstrated cardioprotective effect of consuming small to moderate amounts of alcohol regularly[49,50,52-56]. Consuming 10–20g per day (1–2 standard drinks) for women or 20–30g per day (2–3 standard drinks) for men is associated with a 30–50% morbidity and mortality risk reduction for cardiovascular disease[49,52,54,55,57,58]. The protective effects of alcohol consumption are attributed to an ethanol-induced increase in HDL cholesterol[49,53,54], to the antioxidant effects[52,54] particularly of red wine[49,56] and dark beer[49], and to platelet anti-aggregation agents[53,54,56]. Several studies have reported a J-shaped dose-response curve where those who consume 1–2 standard drinks of alcohol per day have a lower relative risk of developing cardiovascular diseases than non-drinkers, although the risk increases again after a level of more than 5 drinks per day is consumed[36,41,49,59]. National Health and Medical Research Council (NHMRC) guidelines suggest that two or fewer standard drinks per day for women and four or fewer per day for men are low risk levels for alcohol consumption[10].

For this reason, promotion of alcohol as a cardioprotective tool should be viewed with caution, particularly among population groups who have higher rates of diabetes and hypertension, or whose literacy skills make the 'beneficial limit' difficult to comprehend[60]. Low socioeconomic status and education level are often associated with groups who have experienced alcohol abuse as a problem either collectively or for addictive individuals[60,61]. Apart from the direct negative physical effects of excessive alcohol consumption, the increased injury and mortality from road accidents, violence, accidental falls, fires, drownings, suffocations and inhalations, suicides and self-inflicted injuries take a huge toll on the population, and categorise alcohol abuse as harmful even for those not alcohol-dependent[33,62]. Alcohol's protective benefits are specifically associated with cardiovascular disease. It remains detrimental where other causes of morbidity and mortality are concerned[62].

Diet and nutrition

Dietary patterns also provide potential risk factors for cardiovascular disease. The risk is attributed to a range of dietary components which, when combined over time, can either cause or adversely affect other physiological conditions. Diabetes, hypertension, excess body weight, blood cholesterol and antioxidant levels are all affected by dietary intake and are all involved with the disease processes of cardiovascular morbidity[3]. In Australia, diet-related diseases and their risk factors result more from over consumption and sedentary life styles than from under-nutrition[2]. This is resulting in a gradual rise in the number of overweight and obese Australians[10]. A proportion of some population groups, such as the Indigenous, still suffer from under-nutrition, although generally the population's mean nutrient intake meets the NHMRC's Recommended Dietary Intake (RDI) levels for most vitamins and minerals in all age groups[2,63]. There is certainly room

for improvement in this area, however, as 'on any day, over half of males aged 12–44 years and approximately a third of children aged 4–11 years do not eat fruit or fruit products, and more than 20% of children under 12 years do not eat any vegetables or vegetable products'[2]. For many Indigenous Australians, poor diet is often the result of nutritious food being unavailable in some of the more remote areas[64] but for other Australians it seems simply the result of poor choices. GPs may have more success in counselling individuals that good dietary habits from a young age help prevent cardiovascular (and many other) problems in the long term.

High dietary intake of saturated fatty acids (from cheese, butter, margarine, meat, milk and pastries), trans fatty acids (from some margarines, meat and meat products) and cholesterol (from eggs, meat, milk and poultry) increase blood cholesterol and contribute to an increased risk of coronary heart disease. Dietary intake of alcohol and salt should also be moderated. An increase in blood pressure is associated with high consumption of dietary salt[4].

A study by Hu at al. (2000) concluded that two main dietary patterns significantly predict the incidence of coronary heart disease, independent of other lifestyle variables. The study found strong evidence that a diet high in vegetables, fruit, legumes, whole grains, fish and poultry, and low in red meat, processed meat, high-fat dairy products and refined grains may reduce the risk of coronary heart disease[65]. Other studies agree that a diet high in fruits and vegetables[36,41,66], whole grains[67], and fish containing omega-3 fatty acids[68] may decrease the risk of stroke. Diets high in sodium and low in potassium can lead to hypertension, and those high in saturated fats cause obesity and hypercholesterolaemia, which also adversely affect blood pressure levels[41]. Excess intake of carbohydrates with a high glycaemic index such as potatoes and white bread have been associated with an increased risk of type 2 diabetes and coronary heart disease[65]. A study by Liu at al. (2000) concluded that a higher intake of whole grain foods was associated with a lower risk of ischaemic stroke among women, independent of other known CVD risk factors[67]. Dewailly at al. (2001) attribute the consumption of marine products rich in n-3 (omega 3) fatty acids, the traditional diet of the Inuit, to the low mortality rate from ischaemic heart disease in the Inuit population[69].

Folic acid, particularly in conjunction with vitamin B12[70], has recently been recogised as an effective therapy for reducing plasma homocysteine, which, in elevated concentrations is associated with cardiovascular disease[71-73]. Dietary sources of folate include meat (especially liver), yeast extract, fruits and vegetables, cereals and other grain products[74,75]. Consuming fibre contained in fruits, vegetables and especially cereals, also has a beneficial affect on cardiovascular health[76]. Fernandez (2001) cites several studies documenting that dietary fibre lowers the risk of coronary heart disease[77]. High fibre intake has been associated with reductions in serum cholesterol[77,78], with oat fibre in particular tending to lower plasma total and LDL cholesterol[76,79]. Giacco at al. (2000) also report benefits of a high fibre diet for type 1 diabetic patients, in particular, an improvement in glycaemic control and reduction of hypoglycaemic events[80].

Between 1960 and 1990 evidence gathered by the WHO suggested that the Mediterranean population were being positively influenced by some factor affecting their health[81]. Of particular interest were the lower death rates and longer life expectancy occurring in Greece, specifically Crete[82,83]. The traditional diets (pre-1960) of people in these regions, particularly in rural areas, consist of a high intake of fruits, wild edible greens and other vegetables, nuts, beans, seeds, cereals, olive oil and olives, fish, cheese, moderate amounts of red wine, and low amounts of milk and red meat[82,84,85]. This style of diet is low in saturated fat, moderately high in unsaturated fat, and high in fibre, flavonoids and other

antioxidants[81,86] and it is considered likely that these properties may be responsible for the cardioprotective effect observed[82,85,87]. Wasling (1999) suggests this effect may also be due to what is missing from the diet—'the animal fats, margarine, cakes, sweets, biscuits and manufactured foods that are characteristic of the British diet'[88]. The Lyon Diet Heart Study found that a Mediterranean-style diet reduced subsequent cardiovascular events in patients following an initial myocardial infarction[85,87,89]. Fuentes at al. (2001) found that the Mediterranean style diet improved endothelial function in hypercholesterolemic men[90]. However, subsequent studies suggest that the Mediterranean model may be losing favour in its traditional regions, becoming more restricted to older people and to rural areas because younger, urbanised people are departing from it[84,91].

Physical activity

Poor nutrition coupled with physical inactivity can often lead to excess weight or obesity, also a risk factor for cardiovascular disease. However, physical inactivity is recognised as an independent risk factor for coronary artery disease. The American Heart Association expounds the beneficial effects of physical activity—exercise training increases both maximum cardiac output and the ability of muscles to extract and use oxygen from blood; improves haemodynamic, hormonal, metabolic, neurological and respiratory function; helps control blood lipid abnormalities, diabetes and obesity by favourably altering lipid and carbohydrate metabolism; affects the distribution of adipose tissue; and assists in the prevention of osteoporosis and certain neoplastic diseases, notably colon cancer[92].

Apart from the health benefits of physical activity, being active greatly reduces the financial burden of healthcare costs. Physical inactivity accounts for 6% of the total burden of disease and injury among Australian males and 8% among females, and ranks second only to tobacco use in terms of the burden of disease in Australia. The direct healthcare cost attributable to physical inactivity is approximately $377 million annually, comprising an estimated $161 million for coronary heart disease, $101 million for stroke and $28 million for type 2 diabetes[4].

Studies by Hu at al. (2000, 2001) concluded that physical activity, including moderate-intensity exercise such as walking, is associated with substantial risk reduction of total and ischemic stroke[93] and cardiovascular events among diabetic women[94]. Wannamethee at al. (1999) recommend light or moderate physical activities such as walking, gardening, light swimming or cycling undertaken regularly to reduce mortality and heart attacks in older men with and without diagnosed cardiovascular disease[95]. Similar conclusions were drawn by Hakim at al. (1999) in the Honolulu Heart Program, which targeted physically capable elderly men. Their findings suggested that important health benefits, particularly a reduced risk of coronary heart disease, could be derived by encouraging the elderly to walk[96].

Wei at al. (1999, 2000) concluded that low cardiorespiratory fitness and physical inactivity[97] were strong and independent predictors of cardiovascular disease, comparable in importance with diabetes as a risk factor[98] and that physical activity is associated with decreased risk of developing diabetes[99,100]. They found that, generally, fit and active individuals were at much lower risk of morbidity, mortality and loss of function than sedentary and unfit persons[101]. Other studies have shown a relationship between mild-to-moderate physical activity levels and favourable lipid profiles in men[102], and that cardiorespiratory fitness achieved through physical activity attenuates many of the health risks associated with overweight or obesity[103]. Lee at al. (1998, 1999) supported these findings, reporting that lean, unfit men had a higher level of all-cause and cardiovascular mortality than men who were fit and obese[104,105]. Lakka at al. (2001) found

good cardiorespiratory fitness to be associated with slower progression of early atherosclerosis in middle-aged men[106].

Yet, although all these benefits continue to be substantiated by ongoing studies, Australians are spending less time each week walking or participating in other moderate or vigorous physical activity even though the majority believe that doing so would be beneficial to their health. The results of the National Physical Activity Survey in 1999 (a follow-up to the 1997 Survey) show that 88% of Australians aged 18–75 years believe they could be healthier by being more active, and 92% believe their health could be improved by spending 30 minutes each day on moderate physical activity[107]. However, the survey also reported that the average amount of time spent each week on all forms of physical activity has fallen in recent years. In particular, the level of vigorous activity had fallen from an average of 91 minutes per person each week in 1997 to 65 minutes per person in 1999, and that the proportion of Australians doing enough physical activity each week to provide a health benefit had also fallen.

Overweight and obesity

The decline in activity levels coincides with a continuing high level of overweight and obesity across the community, which the National Physical Activity Survey reports as 44%[107]. The AIHW (2001) reports similar findings for 1999–00, noting 60% of Australians aged 25 years and over were overweight with 20% of these being classified as obese[4]. In 1995, Australian men on average weighed 3.6 kg more than their counterparts in 1980. Women weighed on average 4.8 kg more[2]. In terms of total disease burden, overweight and obesity are responsible for approximately 4.3% in both males and females in Australia[4].

The estimate of overweight and obese Australians reported in the National Physical Activity Survey[107] is supported by BEACH data over the first 3 years of its collection. BEACH collects the self-reported height and weight of patients to estimate their body mass index (BMI) which is summarised in the program's annual reports. In 1998–99, BEACH showed 51.2% of adults over 18 years to be overweight (32.8%) or obese (18.4%)[14]. In 1999–2000, BEACH reported 52.5% to be overweight (33.1%) or obese (19.4%)[12]and in the 2000–2001 BEACH year, 54.3% were found to be overweight (34.1%) or obese (20.2%)[13]. All of the above studies used BMI as the classification for being overweight or obese, as proposed by the WHO as a simple measure of obesity[108]. BMI is calculated by dividing a person's weight (in kilograms) by their height squared (in m^2). A person is generally considered to be overweight if their BMI is ≥ 25, and a BMI of ≥ 30 is considered to be obese[4]. Because the calculation of a raised BMI does not distinguish between weight mass from fat, muscle or heavy bone structure, the WHO also regard waist circumference as a useful measure of increased risk due to overweight and obesity[108]. A waist circumference of 94 cm for men and 80 cm for women indicates increased risk, and circumferences exceeding 102 cm for men and 88 cm for women indicate substantially increased risk[4]. Waist-to-hip ratio (WHR) is another commonly used anthropometric measure to indicate obesity, the American Heart Association recommending a WHR > 0.80 as an indicator of obesity for women and > 1.0 for men[109].

Excess body weight carries with it a higher risk of ill health because of the effects of increased body fat on conditions related to life expectancy, particularly cardiovascular conditions such as coronary heart disease, congestive heart failure, stroke and type 2 diabetes[4,33]. Hypertension and adverse lipid profiles associated with excess body weight also increase the risk of coronary heart disease[4,36,108]. In particular there is a strong association between intra-abdominal adiposity and the development of such diseases as

type 2 diabetes[108]. Although being overweight is an important risk factor, the central obesity fat patterning characterised by abdominal fat deposition seems to present a greater risk than the 'pear shape' patterning of hip and thigh fat deposition in the occurrence of stroke[36,41] and other atherosclerotic disease[110].

Results from both the Honolulu Heart Program and the Framingham study indicate that obesity is an independent risk factor for stroke, as reported at the American Heart Association prevention conference in 1997 [36,110], a claim supported by Goldstein at al. (2001)[36]. Fitzgerald and Jarrett (1992) reported from the Whitehall study data that BMI was a predictor for stroke in both smokers and non-smokers, and that a BMI above 24 in combination with smoking accounts for 60% of strokes in men up to 65 years of age[111]. Rexrode at al. (1997, 1998) concluded that waist–hip ratio and waist circumference are independently associated with risk of coronary heart disease[112], and that both obesity and weight gain in women are important risk factors for ischaemic and total stroke[113].

Yet despite increasing knowledge and education about obesity, nutrition, exercise, and the hazards associated with being overweight, the prevalence of obesity in many countries is at a level that is now considered pandemic[114,115]. The WHO has described this increasing prevalence as a major public health problem for developed countries and an increasing number of developing countries[116]. The health economic consequences to these countries has been estimated at 3–5% of their total health budget[117]. A study by Quesenberry at al. (1998) showed a direct association between BMI and annual rates of in-patient days, number and costs of outpatient visits, costs of outpatient pharmacy and radiology services, and total costs. Relative to a BMI of 20 to 24.9, mean annual costs were 25% greater for those with a BMI of 30 to 34.9 and 44% greater for those with a BMI ≥ 35. The authors attributed these elevated costs to the association between BMI and coronary heart disease, hypertension and diabetes[118].

Australia is at present second only to the United States as the most overweight nation in the world[119,120], with the United Kingdom a close third[120]. Such a large proportion of the US population is overweight that in 2000 the American Heart Association began stressing the importance for adults of trying to maintain their current weight rather than just urging the overweight to slim down[121]. In December 2001, the US surgeon-general warned that obesity could soon overtake smoking as the leading cause of preventable deaths in America with over 60% of Americans now considered overweight, and that the situation is so serious that it is countering progress made in fighting cancer and heart disease[122]. The International Obesity Task Force, extrapolating from existing data, has made a projection that by the year 2025 obesity levels could reach 45–50% in the United States, 30–40% in Australia, England and Mauritius, and over 20% in Brazil[120]. Studies previously referred to in this work suggest that in Australia, if the situation remains unchecked, this level is likely to be achieved, and probably sooner than the year indicated[4,12-14,107].

A trend of increased prevalence of obesity in children is also of concern. Childhood obesity increases the risk of adult obesity and of the associated cardiovascular disease risk factors of hypertension, diabetes and dyslipidaemia[123]. Again, the United States leads the rest of the world in the prevalence of overweight youth[119,120]. In the past 30 years the percentage of overweight children in the United States has doubled, from 15% to 32%[120]. But although American children have the 'gold medal for fatness'[119] Australian children are very close behind. Magarey at al. (2001) reported that the rate of overweight and obese children in Australia had doubled in the decade between 1985 and 1995 to a level where 19.5% of boys and 21.1% of girls between 7 and 15 years of age are overweight or obese[124]. What is more disturbing, Magarey at al. showed that the number of children

classified as obese in the overweight group has trebled in that decade[124]. Children of obese parents have more than double the risk of obesity in adulthood[4]. Although it may be conjecture to assume that overweight children remain overweight and eventually become overweight adults, it is likely that this may be the case for a substantial number when consideration is given to the effort required in changing the diet and lifestyle habits established in childhood.

There is, however, evidence that the risks to these children are not postponed until adulthood. Results from the Taipei Children Heart Study showed 70% of obese boys had one, and 25% had two or more, CVD risk factors other than obesity. Obese girls had a higher prevalence of CVD risk factor clustering and a significantly higher prevalence of high blood pressure than non-obese girls[123]. Similar findings were reported from the National Heart, Lung, and Blood Institute Growth and Health Study. Overweight was associated with increased risk factor levels and with increased clustering of risk factors in 9–10 year old girls, and greater central adiposity was associated with higher levels of risk factors and increased clustering[125]. Morrison at al. (1999) concluded from the Princeton School Study that the trend towards increased obesity in children could potentially reverse the recent decline in morbidity from cardiovascular disease[126].

Obesity is presenting serious consequences in terms of personal and economic costs, and the solution will require commitment and education. The desire among overweight individuals to reduce their weight is obvious from the amount they spend on diet programs[127] and weight loss gimmicks. Telephone surveys have reported that more than two-thirds of Americans are attempting to lose or maintain weight, but only 20% of those trying to reduce their weight were using the recommended combination of calorie/ kilojule reduction and 150 minutes of moderate physical activity per week[127,128].

Although reducing weight is beneficial to health generally, many studies have shown specific benefits. Clinically significant long-term reductions in blood pressure and reduced risk for hypertension can be achieved with even modest weight loss[129]. In Phase II of the Trials of Hypertension Prevention, Stevens at al. (2001) observed a linear association between weight reduction and reduction in blood pressure: for every kilogram of body weight lost, systolic and diastolic blood pressures were reduced by 1 mmHg and 1.4 mmHg, respectively. The more weight lost, the greater the reductions in blood pressure levels[127,129].

Given the difficulty in reducing weight once the problem has manifested, prevention strategies would seem to be most worthwhile, particularly when aimed at the young. Singapore's 'Fit and Trim' program, based on activities promoting healthy eating habits and increased physical activity, has started to reduce the prevalence of overweight and obesity in primary, secondary and junior college students since its introduction into schools. The International Obesity Task Force is hopeful that, despite cultural differences, such programs might be as successful if introduced in other nations' schools[120].

Hypertension

Hypertension refers to an increase in the forces exerted by blood onto the walls of the arteries, and for this reason, is often referred to as high blood pressure. The pressure of the blood on arterial walls depends on the energy of the heart action, the elasticity of the arterial walls and the volume and viscosity of the blood. The maximum pressure occurs near the end of the stroke output of the heart's left ventricle and is called maximum or systolic pressure. The minimum pressure occurs late in ventricular diastole and is called the minimum or diastolic pressure[8]. Blood pressure is usually expressed as systolic/ diastolic in mmHg, e.g. 120/80 mmHg, stated as '120 over 80'.

The WHO has recently released a classification for the clinical management of hypertension as systolic blood pressure (SBP) ≥ 140mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg and/or receiving medication for high blood pressure. Previously, high blood pressure in Australia was defined as SBP ≥ 160 mmHg and/or DBP ≥ 95 mmHg and /or receiving medication for high blood pressure[4]. Many Australian studies have therefore quoted estimates of prevalence at the latter level. As we now use the WHO classification, the validity of comparisons across studies is affected where previous and future studies are based on different classifications for defining hypertension.

Hypertension is the most common cardiovascular disorder and is considered both as a disease category and as one of the major risk factors for stroke, coronary heart disease and heart failure[4,33,130]. As the level of blood pressure increases so does the corresponding risk level[7,33]. Although systolic blood pressure is a stronger predictor of death due to coronary heart disease, both systolic and diastolic blood pressures are predictors of heart, stroke and vascular diseases at all ages[7,131].

Prevalence of hypertension increases with age. The major risk factors are obesity, poor nutrition, excessive intake of sodium and alcohol, and lack of physical activity[130]. In 1999–00, approximately 31% of men and 26% of women in Australia aged 25 years and over had hypertension. For those aged 65–74 years, 70% of men and 67% of women had high blood pressure and/or were on treatment for hypertension[4]. Approximately 25% of Americans have hypertension[129] which is slightly higher than the international trend—the WHO estimates that hypertension currently affects about 20% of the adult population worldwide[130].

Hypertension has a major role in the pathogenesis of atherosclerosis[132], and is estimated to account for about 5.5% of the total burden of disease and injury among Australians[4,33] with most of this burden attributed to ischaemic heart disease and stroke. The burden of risk for men starts in the 15–24 age group, for women in the 25–34 age group, and rises steadily with age for both. The burden for men is higher across all age groups except those 70 and over, where it is much higher for women[33]. Since 1998, hypertension has accounted for approximately 6% of all problems managed by GPs in Australia[12-14]. There is evidence that this burden could be eased by adopting healthy lifestyle changes[133-136]. Oncken at al. (2001) found that cessation of smoking reduces systolic blood pressure[134]. Dickey and Janick (2001) reported a reduction in both systolic and diastolic blood pressure from weight reduction and reduced dietary sodium intake[133]. Moreau at al. (2001) reported that just 30 minutes per day of moderate-intensity physical activity is effective in lowering systolic blood pressure[134]. Non-pharmacological methods in combination with antihypertensive medications increase the success of achieving target levels for blood pressure reduction[137].

Many studies have examined the efficacy of treatments and medications for patients with hypertension, in consideration of the social and economic resources required to manage this predominantly 'elderly' disease in an ageing population. There is consensus that current treatments are effective and safe for long-term use and, when taken correctly, do reduce the development of severe hypertension, stroke, congestive heart failure, and other coronary heart disease[26,131,138,139]. However, control of hypertension remains poor in many countries, especially when the patient is elderly, where multidrug regimens are involved or where patients experience side effects from antihypertensive medications[129,140]. Since 1980 there has been a significant decline in the prevalence of hypertension in Australia. The rate of men aged 25–64 years with high blood pressure has

fallen from 45% in 1980 to 22% in 1999–00. The rate for women in that age group has fallen from 29% in 1980 to 16% in 1995 and has remained steady[2,4,33].

Promoting lifestyles which prevent the initial development of hypertension should still be the objective of healthcare providers in both developed and developing countries[129,133].

Hyperlipidaemia

Hyperlipidaemia is a general term for elevated concentrations of any or all of the lipids in the plasma, including triglycerides and cholesterol. Lipids are fats and fatlike substances which are stored in the body and serve as a source of fuel. They are an important constituent of cell structure and include fatty acids, neutral fats, waxes and steroids. Compound lipids comprise the glycolipids, lipoproteins and phospholipids[8]. The role of blood lipids such as cholesterol in the development of atherosclerosis and subsequent cardiovascular disease were established in the Framingham Heart Study in 1960 and high blood cholesterol is now considered a major modifiable risk factor for developing coronary artery disease[141,142].

Cholesterol is a lipid which the body needs to repair cell membranes, insulate nerves, manufacture vitamin D on the skin's surface and produce certain hormones such as oestrogen and testosterone. About two-thirds of the body's cholesterol is manufactured in the liver, production being stimulated by saturated fat. It is also obtained through diets which include animal fats found in meat, poultry, fish and dairy products[143,144]. Cholesterol is transported through the blood in lipoproteins categorised by their size[143]. Low-density lipoproteins (LDL) and high-density lipoproteins (HDL) both have an important role in maintaining health.

Low-density lipoprotein (LDL) transports about 75% of the blood's cholesterol to the body's cells and is normally harmless. It becomes problematic, however, when it penetrates the artery walls where it can interact through oxidation with oxygen-free radicals, particles which the body releases naturally but which increase when the body is exposed to environmental toxins such as cigarette smoke. These molecules are essential in fighting bacteria but, in excess, can become destructive because they are missing an electron and therefore tend to bind with any other molecule. If LDL collects on arterial walls, free radicals can attack and oxidise with it, modifying its form. The new oxidised LDL triggers an immune system response where white blood cells gather at the site forming a fatty plaque and causing inflammation. This process of plaque deposits building up on arterial walls which, over time, restrict the blood flow is known as atherosclerosis[143,145]. The reduced blood flow starves the heart of oxygen which can eventually cause angina, myocardial infarction or death[144].

The function of high-density lipoproteins (HDL) is to remove cholesterol from the arterial walls and return it to the liver which, apart from producing cholesterol, also removes it from the blood. It is removed by special proteins called LDL receptors which are normally present on the liver surface[143,145]. Because of their differing functions, LDL cholesterol has become known as 'bad' and HDL as 'good' cholesterol[144,145]. Total cholesterol level is therefore not a good reflector of cardiovascular disease risk because it consists of both LDL and HDL cholesterol which have opposing effects on cardiovascular risk[146]. The best risk assessment is obtained through separate measurements of these two levels[146-148]. The Framingham study demonstrated the total/HDL cholesterol ratio to be the most efficient lipid profile for predicting coronary disease[142]. Rizos and Mikhailidis (2001) concluded from evidence presented in the Veterans Affairs High Density Lipoprotein Cholesterol Intervention Trial (VA-HIT) and the Bezafibrate Infarction Prevention Trial (BIP) that levels of total, LDL and HDL cholesterol, and triglycerides, are all predictors of risk of

cerebrovascular events[149]. Wannamethee at al. (2000) found an association between higher levels of HDL cholesterol and a significant decrease in risk of non-fatal stroke[150].

For optimum health, high levels of HDL are promoted while LDL should be kept to a minimum[148]. As the body makes enough cholesterol to perform the functions for which it is required, any added cholesterol from dietary sources has the potential to create more LDL than the body can remove through normal processes. Dietary fatty acids directly influence the susceptibility of lipoproteins to oxidation which subsequently affects other triggers in the inflammatory process and formation of plaques[9]. Some people also have a reduced number of LDL receptors on the liver cell surfaces which impede their ability to dispose of LDL cholesterol from the blood. This can be a genetic predisposition that can make 'high cholesterol' a familial problem, and affected family members tend to develop atherosclerosis in early adulthood[145]. In most cases, however, raised LDL cholesterol levels are usually caused by diet[146], particularly the consumption of foods high in saturated fats.

High dietary intake of saturated fats impedes the liver's LDL receptor activity which thus raises the LDL cholesterol levels in the blood. Unsaturated fats, either polyunsaturated or mono-unsaturated, do not raise LDL cholesterol and may even lower it in some cases. Oils high in mono-unsaturated fats such as olive or canola oil contain antioxidants which can scavenge free radicals and protect against peroxidation[151], offering a protective effect against coronary heart disease[144,145]. However, excess calories from any source are stored as fat deposits so the body becoming overweight, and particularly obese, will also raise cholesterol levels.

Diet modification is the preferred initial choice for prevention and treatment of hypercholesterolaemia[152,153], but the best method of dietary intervention continues to be debated. Diets focusing on reducing total fat and cholesterol often result in reducing both LDL and HDL cholesterol, whereas those which focus on calorie reduction but not on saturated fat reduction have little effect on lowering LDL cholesterol levels[152]. However, raising the HDL cholesterol level may be just as, if not more important. Boden (2000)[154] reported that raising HDL while maintaining levels of LDL cholesterol was responsible for benefits which included significant reductions in death from coronary artery disease, non-fatal myocardial infarction (MI), stroke, transient ischaemic attack and carotid endarterectomy. From the VA-HIT study he concluded, 'for every 1% increase in HDL-C, there was a 3% reduction in death or MI, a therapeutic benefit that eclipses the benefit associated with LDL-C reduction'. The best approach appears be a diet which lowers LDL cholesterol and raises HDL cholesterol levels.

Although dietary therapy remains the first line of treatment for high cholesterol, drug therapy is available for those patients considered at risk for coronary heart disease[153]. There is an abundance of literature from many clinical trials which report evidence that lipid-modifying treatments have a positive effect on cardiovascular disease risk from hyperlipidaemia. Many secondary prevention studies report the benefits of lipid-lowering medications, particularly for patients with pre-existing coronary heart disease and diabetes (the Scandinavian Simvastatin Survival Study(4S)[155-157], the Cholesterol and Recurrent Events (CARE) Trial[158], the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study[159], the Veterans Affairs High-Density Lipoprotein Cholesterol Intervention Trial (VA-HIT)[160], the Helsinki Heart Study[161], the West of Scotland Coronary Prevention Study (WOSCOPS)[162,163], and the Air Force/Texas Coronary Atherosclerosis Prevention Study[164]). Results from many of these studies show a significant reduction (around 30%) in relative risk of major coronary events such as fatal and non-fatal myocardial infarction for patients with elevated lipids treated with lipid

modifying medications[148]. Statins, particularly, as demonstrated in many of these trials, reduce the risk of stroke[165] and transient ischaemic attacks in patients with coronary disease, and their efficacy extends to subgroups such as diabetics, women and the elderly[165,166]. The anti-inflammatory and antioxidant properties of statins reduce the incidence of ischaemic stroke by stabilising atherosclerotic plaques[167,168] and through antithrombotic actions[167,168].

The availability of lipid-lowering medications has reduced the severity and frequency of cardiovascular events for a large proportion of the population. However, given the health and economic costs associated with more than 6 million adult Australians (aged 25 years and over) having a cholesterol level higher than 5.5 mmol/L[4], promoting strategies for awareness and prevention should continue as a priority for healthcare planners.

Diabetes

Diabetes is a general term representing a collection of related metabolic disorders characterised by excessive urine excretion (polyuria) and high blood glucose levels (hyperglycaemia). It is caused by defective pancreatic islets which inhibit the secretion of the hormone insulin, inhibit the insulin action, or both[4,8]. Diabetes is associated with long-term dysfunction of organs such as the heart, eyes, kidneys, nerves and blood vessels. Peripheral vascular disease associated with diabetes often results in lower limb ulceration, gangrene and amputation in more severe cases[169].

There are three main types of diabetes. Type 1 or insulin-dependent diabetes mellitus (IDDM) is the result of auto-immune destruction of the pancreatic islets that produce insulin. Individuals with type 1 diabetes require insulin injections daily for survival[4]. The peak age of onset is 12 years, but onset can occur at any age[8]. Type 2, or non-insulin-dependent diabetes mellitus (NIDDM) is characterised by insulin resistance and/or abnormal insulin secretion[4]. Basal insulin secretion levels are maintained or reduced, but insulin release in response to glucose load is delayed or reduced. Defective glucose receptors on the beta cells of pancreatic islets may be involved. Obesity and physical inactivity are strongly associated with its onset, as is a genetic predisposition. The peak age of onset is generally 50–60 years. Type 2 diabetes accounts for about 85–90% of all diabetes[4]. Gestational diabetes is caused by carbohydrate intolerance and is first recognised during pregnancy[8]. Approximately 4–6% of women not previously diagnosed are affected, and have a greater risk of developing type 2 diabetes in later life[4].

Diabetes is both a risk factor for, and shares other common risk factors with, cardiovascular disease. In particular, type 2 and gestational diabetes are promoted by behavioural risk factors such as poor nutrition, excess weight and physical inactivity. The effects of abnormal blood lipid levels, smoking, central obesity and hypertension are all amplified by the presence of diabetes. Cardiovascular risk factors relatively specific to people with diabetes include longer duration of diabetes during adulthood, raised blood glucose concentrations and microalbuminuria[170]. People with diabetes and microalbuminuria have a higher risk of coronary morbidity and mortality than people with normal levels of urinary albumin and a similar duration of diabetes. Clinical proteinuria increases the risk of major cardiac events in type 1 and type 2 diabetes compared with individuals with the same type of diabetes having normal albumin excretion[170]. Abnormal lipoprotein metabolism advances the development of coronary artery disease in type 1 diabetes[171].

A clustering of risk factors, known as the metabolic syndrome, is more likely among diabetes sufferers[4]. Conversely, people with the metabolic syndrome are at increased risk of developing diabetes and cardiovascular disease[172]. There is no international definition

for the metabolic syndrome but the WHO suggests a working definition as having glucose intolerance, impaired glucose tolerance or diabetes mellitus and/or insulin resistance together with two or more of the following: impaired glucose regulation; central obesity, hypertriglyceridemia and high blood pressure[172]. Other definitions add low high-density lipoprotein to this list[173,174]. This clustering has been variously labelled as the 'metabolic syndrome', the 'insulin resistant syndrome'[172] or 'syndrome X'[172,175,176]. Each individual component of the cluster group carries a risk of CVD but in combination they become much more powerful[172]. Using 2000 census data, the 3rd National Health and Nutrition Examination Survey (ATPIII) determined that about 47 million US residents have the metabolic syndrome, with prevalence increasing with age from around 6.7% of 20–29 year olds to around 43% of those aged 60 or older[173].

Apart from being the seventh leading cause of death in Australia[169], diabetes is the most common cause of blindness in persons under 60 years of age, the most common cause of non-traumatic amputations, and the second most common reason for the commencement of renal dialysis[6]. The risk of heart disease and stroke is two to four times higher for diabetics than for people without diabetes[177]. In Australia in 1998, diabetes was the second most common additional cause of death where ischaemic heart disease was the underlying cause[1].

Along with obesity, diabetes is reaching epidemic proportions in the United States, Australia, New Zealand and other developed nations[178]. For males, most nations have experienced increases in the death rates from diabetes since the mid–1950s, with the most dramatic of these occurring in Denmark (164%), Italy (83%) and Spain (80%)[179].

The 1995 National Health Survey[44] reported 430,700 Australian people (2.4% of the population) diagnosed as diabetics and estimated that the number had doubled since the 1980s. Based on this assessment, the 1998 National Health Priority Areas report on diabetes mellitus predicted that the number diagnosed with diabetes would rise to 770,000 by the year 2000, would reach 950,000 by 2010 and would exceed 1 million within 15 to 20 years of the 1995 survey[169]. However, just 2 years after the 1998 report, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) found about 940,000 Australians aged over 25 (8.0% of males and 7.0% of females) were diagnosed with diabetes[177]. It would seem that the 'one million' mark will be exceeded much sooner than estimated, and may indeed have already been exceeded—both studies[44,177] reported that for each diagnosed diabetic there was another person with diabetes as yet undiagnosed. As in many other countries, Australia's Indigenous population has a much higher rate of diabetes than other Australians[169]. In the 30–54 age group, more than 20% of Indigenous Australians are estimated to have diabetes[179]. The annual incidence of type 1 diabetes in New South Wales children aged 0–14 years has increased on average by 3.2% each year since 1990[180].

The burden of this disease is also increasing at a similar rate. In 1993–94, the estimated health system cost attributed to diabetes was 2.2% of total health expenditure for that year[33]. By 1996 this had increased to 5%, a figure considered to be an underestimation as it was based on self-reported information[4]. The main contributors to this cost increase are the large number of hospitalisations either directly related to diabetes or where diabetes is a secondary diagnosis. The average length of stay in hospital for diabetes in 1998–99 was almost twice as long as for non-diabetic conditions, accounting for almost 1% of all occupied hospital beds on any given day[4]. Diabetes is the eighth most common problem managed by Australian doctors each year in general practice[4,12-14,181] and is managed at nearly 2% of all encounters[4,12-14]. A study by Overland at al. (2000) reported that over a 5-year period, diabetics accounted for 3.0% of the population but had used 5.5% of

general practitioner services, and a three-fold to four-fold increase in the use of specialist and consultant physician services compared with their non-diabetic counterparts[182]. Although the financial cost of 7,887 diabetes-related lower-limb amputations between 1995 and 1998 can be readily determined, the personal cost cannot[183].

Pharmacological managements are advancing and studies such as the Catopril Prevention Project (CAPP)[184] and the Heart Outcomes Prevention Evaluation (HOPE) study[185,186] report benefits of treatment with angiotensin-converting enzyme (ACE) inhibitors, specifically catopril and ramipril respectively, which include reduced risk of complications related to diabetes, and reduced new diagnoses of diabetes itself among those at high risk. It is reassuring to have access to these medications, but given the escalation in costs associated with the increase in diabetes incidence, promoting lifestyle changes might be a better initial approach to management. Studies in Sweden, China and Finland[187,188] have concluded that changes in diet and exercise habits can prevent or delay the onset of type 2 diabetes for high-risk subjects; however, lifestyle changes have to be sustained and willingness to do this ultimately rests with the individual.

Stress

Stress as a contributing factor in development of cardiovascular disease is somewhat controversial as it is not always clear whether 'stress' refers to physical, mental, emotional or psychological stress. Regardless of the type of stress involved, stress is predominantly a secondary rather than primary risk factor for cardiovascular disease[189].

The majority of medical literature refers to physical stress in relation to cardiovascular disease—the increased demands for energy and oxygen placed on the heart during physical stress are measurable and reproducible. The effects of physical stress on the cardiovascular system depend on the general fitness of the individual. Physical exercise which places a greater demand on the heart muscle to pump oxygenated blood around the body is very beneficial to the cardiovascular health of the individual. Indeed, the lack of physical activity caused by sedentary lifestyle is considered to be a major risk to cardiovascular health. However, if there is underlying heart disease from other causes, stress from physical effort can be dangerous. During physical exertion, the heart beats more rapidly to meet the body's increased demands. If coronary arteries are partially blocked by atherosclerotic plaques, they cannot supply the heart's extra requirement for oxygenated blood. This 'starving' of oxygen is called ischaemia and can cause angina pain or, in severe cases, the heart muscle to infarct[190]. Although physical stress can induce a heart attack in an individual with existing coronary heart disease, it does not cause heart disease in a normal, healthy person.

Other types of stress—mental, emotional, psychological—can be caused by a variety of factors and can be detrimental to long-term cardiovascular health. Individuals may perceive situations differently, but whatever the triggers, their physiological responses to mental effort, anger, fright, grief, anxiety and so on are similar. The sympathetic nervous system mediates the discharge and release of adrenal medullary hormones in response to these stressors, which results in increased blood pressure and cardiac output, increased blood flow to skeletal muscles, decreased flow to the viscera, and increased rate of glycolysis and blood glucose concentration[8]. Homocysteine levels, heart rate and blood pressure have been shown to increase in women exposed to mental and emotional stress-inducing tests[191]. Homocysteine is an amino acid strongly associated with cardiovascular disease because of its potential to damage cells lining arterial walls, contributing to the development of arterial plaques. Stoney (1999) concluded that the rise in homocysteine concentration may be sympathetically mediated and may therefore be an important factor

in the relationship between psychological stress and cardiovascular disease risk. Homocysteine level has also been associated with stroke risk in other works[192]. Sarabi and Lind[193] reported that endothelium-dependent vasodilation in young healthy people is also affected by mental stress. Carroll at al. (2001)[194] found that heightened blood pressure reactions to mental stress contributed to the development of high blood pressure on 10-year follow-up of participants in the Whitehall Study. Cordero et al[195] suggest that ischaemia can be initiated through a neurocardiac pathway and that stress can trigger the link between the brain and the heart involved in pathogenic processes such as coronary vasospasm, ischaemia and arrhythmias even in hearts lacking structural disease.

Although these works support a possible primary link between the physiological responses to stress and the long-term development of heart disease, there is considerable evidence that stress is detrimental to health where cardiovascular disease is already present. Various studies have concluded that mental stress impairs systolic function by inducing transient myocardial ischaemia[196]; combined increases in cardiac demand and concomitant reduced myocardial blood supply may contribute to myocardial ischaemia with mental stress[197]; excessive sympathetic reactivity to stress may be aetiologically important in stroke, especially ischaemic stroke[198]; psychological distress is a predictor of fatal ischaemic stroke[199]; cardiovascular reactivity to psychological challenge plays a role in the etiology of hypertension[200]; mental stress could contribute to Coronary Artery Disease (CAD) progression and acute coronary syndromes in patients with cardiovascular disease[201]; acute psychological stress may elicit a hypercoagulable state in elderly subjects with cardiovascular disease which could promote progression of atherosclerosis and acute coronary thrombosis[202]; and both chronic and acute psychological stress can be detrimental to the patient with CAD[203].

Chronic mental, emotional or psychological stress can therefore play a prominent role in the development of cardiovascular disease, and methods of reducing stress are widely promoted. Some individuals are more likely to be affected by stress-induced cardiovascular problems, particularly those of 'type A' behaviour pattern (competitive achievement orientation, sense of urgency in general life, easily aroused to anger/hostility)[204,205], those with 'job strain'[206,207], those in stressful marital relationships[208,209], sufferers of depression[206] or pessimistic/superstitious outlook[210], or those who have experienced war or natural disasters[206,211]. Pickering (2001) regards the common link for all these factors as a perceived loss of control over one's environment[206]. This perception may also apply to persons following an initial cardiovascular event and may affect their recovery. Cossette at al. (2001) concluded that post-myocardial infarction interventions that reduce psychological distress have the potential to improve long-term prognosis and psychological status for both men and women[212]. It seems that, regardless of its role in the aetiology of cardiovascular disease, stress is a major factor in the progression of established illness.

Atrial fibrillation

Atrial fibrillation (AF) is an arrhythmia in which very small areas of the atrial myocardium are in various uncoordinated stages of depolarisation and repolarisation. Multiple re-entry circuits within the atrial myocardium cause the atria to quiver continuously in a chaotic pattern instead of intermittently contracting[8]. This rapid activity within the atria removes normal sinus node control of the heart rhythm causing rapid and irregular atrial activity[213]. The result is a totally irregular, often rapid, ventricular rate[8].

AF is the most common arrhythmia encountered in clinical practice. It accounts for approximately one-third of all hospitalisations for cardiac rhythm disturbances[214] and its prevalence increases with age. About 2% of the general population are affected, increasing to 5% in people over 65 years and 10% of those over 75 years. Episodes may occur singularly, as a series of recurrent episodes (paroxysmal AF), or continuously (permanent or chronic AF)[215]. When an underlying cause can't be identified, the condition is called 'lone' AF, and this type affects about 10% of chronic AF sufferers[216]. However, AF may be a sign of underlying heart disease and is associated with an increased risk of systemic thromboembolism and stroke[215]. This increased risk occurs because the loss of effective atrial contraction alters the normal flow of blood, often resulting in stasis and triggering of the coagulation cascade. As a result, fibrin thrombi form within the atria and atrial appendages which can dislodge and travel through the systemic circulation to the brain causing ischemic stroke[217].

The incidence of AF is greater in men than in women, but advancing age levels this difference. Women with AF are more likely to have underlying valvular disease, whereas men are more likely to have underlying coronary artery disease[218]. The risk of stroke is substantially increased for both men and women with AF, particularly in the first year after diagnosis[219,220]. Stroke in persons with AF is generally more severe and induces higher mortality[220]. In Framingham Heart Study cohorts, AF was associated with a 50% to 90% mortality risk after adjustment for age and pre-existing cardiovascular conditions such as hypertension, myocardial infarction, congestive heart failure and smoking[220,221]. For people with AF, risk factors for stroke include history of congestive heart disease (relative risk (RR) 1.4), increasing age (RR 1.4), history of hypertension (RR 1.6), history of diabetes (RR 1.7), and prior stroke or transient ischaemic attack (RR 2.5)[222]. Prior myocardial infarction, rheumatic heart disease and poor left ventricular function are also factors associated with an increased risk of stroke in AF[216].

Socioeconomic influences

In 1992, the National Health Strategy cited a number of studies which offered evidence that low socioeconomic status is strongly associated with high mortality rates, poorer health status and higher levels of risk behaviours which increase exposure to a number of health problems including cardiovascular disease and some cancers[15].

People in lower socioeconomic groups are more likely to die from CVD than people in higher socioeconomic groups. In 1997, people aged 25–64 years living in the most disadvantaged group died from CVD at around twice the rate of those living in the least disadvantaged group[4]. The 1995 National Health Survey reported that 82% of women in the lowest socioeconomic group had a CVD risk factor (i.e. tobacco smoking, high blood pressure, overweight or obesity, physical inactivity) compared with 69% in the highest socioeconomic group. Almost 13% of women in the lowest group had three or more risk factors compared with 7% of women in the highest group[44]. Men in the lowest socioeconomic group were twice as likely to have three or more risk factors than men in the highest socioeconomic group (18% and 9% respectively) although there was no significant difference between the two male groups for one CVD risk factor. Men and women in the lowest socioeconomic group (37% and 40% respectively) were more likely to partake in no physical activity in their leisure time than those in the highest socioeconomic group (27% and 29%)[44]. Women in lower socioeconomic groups are more likely to be overweight (53%) or obese (24%) than women in the highest group (44% and 14% respectively) although there were no significant differences between overweight and obese men in these two groups. For type 2 diabetes, prevalence in the lowest socioeconomic group was almost 2.5 times higher, and deaths where diabetes was the

underlying cause of death were 44% higher than those in the highest socioeconomic group[44].

The 1998 National Drug Strategy Household Survey reported that 27% of individuals from the lowest socioeconomic background smoked daily compared with 18% of those from the highest socioeconomic background[223]. Higher levels of smoking were associated with being unemployed and with having a lower level of education. Those with no qualifications (26%) were more than twice as likely to smoke daily compared with those with tertiary qualifications (12%). Persons with less than 12 years of education were almost twice as likely to be physically inactive as those with a Higher School Certificate or equivalent, or with tertiary qualifications[4,44]. Results from a Finnish study support the higher incidence of smoking among the poorer educated, observing that the educational discrepancy already begins to emerge at the upper stage comprehensive level (12–15 years of age)[224]. Other studies have concluded that men with heightened cardiovascular responsiveness to stress who were born into poor families, had little education or had low incomes had the greatest atherosclerotic progression[225], that poorer socioeconomic circumstance, particularly with early-life adversity, was associated with greater stroke risk[226], and that chronic stress, hostility, depression, level of social support and socioeconomic status play a direct role in organic coronary artery disease pathology[227].

Population groups

Some population groups have higher prevalence of cardiovascular disease and risk factors than others. People born in Australia are more likely to die from CVD than Australian residents who were born overseas, but certain migrant groups have a higher prevalence of conditions such as diabetes than their Australian-born counterparts. Diabetes is very common among peoples from many Pacific Islands, Asian Indians, Chinese and other Asian groups, and people from southern Europe[4].

Various ethnic groups may have a physiological predisposition for some of the risk factors associated with CVD. US-based studies have shown hypertension to be more frequent and blood pressure levels to be higher in black participants than in their white counterparts[228]. The incidence of stroke in the US and UK black populations is approximately twice that of the white population after adjusting for age, sex and socioeconomic status[228,229]. The Northern Manhattan Stroke Study (NOMASS) demonstrated race–ethnic differences in stroke incidence whereby blacks had a 2.4-fold increased annual stroke incidence and Caribbean Hispanics a 2-fold increased annual stroke incidence compared with whites living in the same community[229]. Sacco at al. cite literature which has consistently shown race–ethnic disparity in the prevalence of cardiovascular risk factors. Blacks have the highest prevalence of hypertension regardless of geographic location and diabetes is more common among black populations, whereas coronary artery disease and atrial fibrillation are more common in whites[229,230]. Physiological causes are not always the explanation for high prevalence of some conditions—Polynesians have a high prevalence of type 2 diabetes, attributed more to their high level of obesity because studies have concluded that they are not intrinsically insulin-resistant as a group[231].

For some population groups, higher prevalence of CVD and CVD risk factors may be a reflection of a combination of physiological predisposition and socioeconomic circumstances. In the 1996 Australian Census, Aboriginal and Torres Strait Islander peoples were shown to be disadvantaged across a range of socioeconomic factors. They experienced lower incomes, higher rates of unemployment, poorer educational outcomes and lower rates of home ownership than other Australians, all of which can affect general

health and wellbeing[232]. Australia's Indigenous peoples also experience higher death rates from all causes than other Australians. Not only are they twice as likely to die from CVD than other Australians, but also they have substantially higher levels of chronic heart disease (CHD) and stroke than Indigenous populations in New Zealand or the United States[4].

Compared with other Australians, Aboriginal and Torres Strait Islander peoples have higher rates of hospitalisation for CHD and stroke (2–4 times) and greater length of stay for CVD conditions. They die from CVD at twice the rate of other Australians, the greatest disparity occurring among those aged 25–64 years, where death rates are seven and ten times those of other Australian men and women respectively. Indigenous peoples have one of the highest rates of rheumatic heart disease in the world, with hospitalisation rates in 1998–99 for this disease being 20–25 times higher than those for other Australians. They have one of the highest rates of prevalence for type 2 diabetes in the world. Overall prevalence for Indigenous Australians is 2–4 times higher than for other Australians, particularly for the 25–55 age group. In 1995, the self-reported prevalence for this age group was 7–8 times higher than for other Australians. In 1997 and 1998, Indigenous deaths where diabetes was the underlying cause occurred at almost three times the rate of other Australians[4].

Indigenous Australians also have higher levels of risk behaviour for CVD. They are more likely to report no physical activity in their leisure time, and those aged 18 years or over are almost twice as likely to smoke as other Australians[4]. Around 56% of Indigenous men and 46% of Indigenous women in 1998 were defined as current smokers compared with 29% of other Australian men and 24% of other Australian women[223]. Although there is little difference between overweight Indigenous men (62%) and other Australian men (63%) the levels of obesity within these groups differ, with Indigenous men at 25% and other Australian men at 18%[4,223]. For women, more Indigenous women were overweight (60%) or obese (28%) than other Australian women (49% and 18% respectively). Although Indigenous Australians are more likely to abstain from alcohol than other Australians (51% compared with 45%) those who do drink are more likely to consume harmful quantities (8% compared with 3%)[4].

Most of the literature referred to above underscores the importance of prevention as a key factor in the management of cardiovascular disease internationally. Promotion at all levels—from government to practitioners to classrooms to homes—is vital if the gains made in cardiovascular health are to be maintained or improved in the future. In acknowledgement of the substantial opportunity for GPs to observe and influence the risk behaviours of their patients, the Joint Advisory Group on General Practice and Population Health has developed the 'SNAP' initiative for general practice which aims to 'reduce the health and socioeconomic impact of Smoking, poor Nutrition, harmful and hazardous Alcohol use and Physical inactivity on patients and the community through a systematice approach to behavioural interventions in primary care'[233]. Education about risk behaviours and individual responsibility should be promoted from the earliest age, and general practice is an excellent avenue for both introducing information and positively reinforcing it over the duration of the GP–patient relationship.