Treatment of Dyslipidaemia in the Elderly

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ABSTRACT

Until recently, the benefits derived from treating dyslipidaemia in the elderly had not been established, as most trials demonstrating a reduction in events related to coronary heart disease have been biased towards younger patients. However, there is now evidence to suggest that age alone should no longer be considered a barrier to the instigation of lipid modifying therapies. Large clinical trials involving drugs of the statin class (HMG-CoA reductase inhibitors) have shown that there is no diminution of the effects of statin therapy with advancing age, even for patients aged up to 80 years. Although it has not been proven in controlled trials, elderly patients may even derive a greater benefit compared to younger patients, as the absolute risk of coronary heart disease increases with age. Furthermore, the statins reduce the risk of stroke and possibly reduce the chances of developing dementia. This review summarises therapies available for treating dyslipidaemia and possible modifications required in managing elderly patients. Methods of assessing an individual's overall risk for coronary heart disease and the use of this risk assessment as a guide to the selection of patients for treatment are also discussed.

J Pharm Pract Res 2002; 32: 188-93.

INTRODUCTION

It has been ten years since the treatment of dyslipidaemia was reviewed in this section of the *Journal*.[1] At that time, it was concluded that the assessment and treatment of dyslipidaemia in the elderly was largely uncharted territory. Fortunately, the decision to treat dyslipidaemia in the elderly can now be guided by the results of several large clinical trials. These trials suggest that the relative risk reduction in vascular events seen with HMG-CoA reductase inhibitors (statins) for middle-aged patients also applies to the elderly and extends to patients aged up to 80 years. Indeed, given that the absolute risk of vascular events increases with age, intervention in older patients should produce a greater absolute benefit. Cholesterol lowering therapy has been estimated to prevent approximately twice as many coronary heart disease (CHD) events and deaths in older patients compared with patients aged less than 65 years.[2]

As well as reducing CHD in the elderly, statins reduce the incidence of stroke and possibly reduce the risk of developing dementia. Statin therapy in the elderly is well tolerated and has also been projected to be cost-effective. Lifestyle modifications such as attention to diet and exercise should also be considered as important therapies for dyslipidaemia in the elderly. However, these interventions may require modification in the very elderly, especially those with poor nutrition and significant co-morbidities such as arthritis and airways disease.

The decision to treat dyslipidaemia should be based on the assessment of a patient's overall risk for a vascular event. Thus, abnormal lipid levels, alone, may not warrant aggressive drug treatment. The recently published Australian Lipid Management Guidelines have attempted to identify individuals assessed to be at high absolute risk for CHD[3] (Table 1).

Table 1. Australian Lipid Management Guideline criteria to identify patients at high absolute risk of CHD

1. Known coronary heart disease
2. Known manifestations of atherosclerosis: peripheral vascular disease, ischaemic cerebrovascular disease or abdominal aortic aneurysm
3. Diabetes mellitus
4. Chronic renal failure or renal transplantation
5. Aboriginal or Torres Strait Island persons
6. Familial hypercholesterolaemia or combined hyperlipidaemia
7. Absolute risk of 10-15% or greater in the next five years according to the New Zealand cardiovascular risk calculator
8. Increased absolute risk judged by LDL-C >4.0 mmol/L or TC >6.0 mmol/L plus two (or more) other risk factors: • HDL-C <1.0 mmol/L • significant family history • hypertension • overweight

CHD = coronary heart disease, LDL-C = low density lipoprotein cholesterol, TC = total cholesterol, HDL-C = high density lipoprotein cholesterol

Recommendations for target lipid levels for individuals at high absolute risk for CHD have also been made (Table 2). In contrast, the criteria for a financial subsidy for lipid-lowering treatment in Australia by the Pharmaceutical Benefits Scheme[4] (PBS) are shown in Table 3. These criteria have been criticised as it has been suggested that the contribution of age, smoking, the additive effects of multiple risk factors and the continuous relationship between risk factor intensity and CHD events are not adequately considered.[5] It has been estimated that up to 61% of Australians (aged 25–69 years) who have a greater than 10% 15-year CHD mortality risk are not eligible for PBS-subsidised lipid-lowering therapy, while 11% of people with less than 2.5% risk are still eligible.[6]

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Table 2. Australian Lipid Management Guideline target lipid levels for patients at high absolute risk of CHD

Lipid profile	Target level
Low density lipoprotein cholesterol (LDL-C)	<2.5 mmol/L
Total cholesterol (TC)	<4.0 mmol/L
High density lipoprotein cholesterol (HDL-C)	>1.0 mmol/L
Triglycerides (TG)	<2.0 mmol/L

CHD = coronary heart disease

In this article we present the findings of recent clinical studies which suggest that age alone should no longer be considered a barrier to treating dyslipidaemia. The pharmacological and non-pharmacological therapies available for treating dyslipidaemia in the elderly and the expected reduction in vascular events are discussed.

CHOLESTEROL LOWERING AND CHD EVENTS

It is now well established that statin therapy reduces morbidity and mortality when used as a primary[7,8] or secondary[9-11] intervention in middle-aged people. However, the major statin trials have tended to exclude people over 75 years. This is surprising, since CHD is strongly age-related. Whether statins can improve clinical outcomes in the elderly to a similar extent as in younger people, remains an important question.

Two recent cohort studies, one observational and one interventional, have provided conflicting results. The Honolulu Heart Program compared total cholesterol (TC) levels and all-cause mortality over 20 years in 3572 Japanese/American men aged 71–93 years.[12] Three separate Cox proportional hazard models were analysed with total mortality as the end point and the first quartile as the reference. The results were in agreement with findings in other observational studies, which showed that long-term persistence of low serum cholesterol concentrations actually increases the risk of death. That is, the earlier these elderly patients started to have lower TC concentrations, the greater the risk of death. The explanation for these results is not clear. The study raises the question of whether there is a difference in biological effect from a permanent, untreated, intrinsically low level of TC when compared with the effect in those who have a dietary or pharmacologically induced reduction of cholesterol. This issue was not addressed in the Honolulu Heart Program, however the authors questioned whether it is scientifically justified to lower TC levels below 4.65 mmol/L in elderly people. However the findings of this study have not been supported by the results of numerous interventional studies.

The second study from Salt Lake City, USA examined the effect of cholesterol lowering with statin therapy on mortality in a cohort of 7220 people with mean age 65 ± 12 years and with severe CHD (70% stenosis defined on angiography).[13] Statin therapy was prescribed to 24% of patients on an individual basis at the time of discharge and patients were followed for a mean of 3.3 years. Overall, group mortality was 16%. Statin therapy was associated with a reduced mortality in all age groups. Among individuals aged 80 years or more (n = 637), statin use was associated with a significantly lower mortality compared with non-statin use (9% vs 30%, p = 0.04). Similar results were observed in people aged 50–59, 60–69 and 70–79 years. Mortality rates in 783 individuals aged less than 50 years showed a similar trend (2% vs 7%, p = 0.35), but this did not reach statistical significance. These data suggest that a greater absolute benefit can be gained in the elderly from statin therapy. However, this was not a prospective, randomised study, which means that the decision to use or not use statin therapy may have been influenced by selection bias.

Table 3. PBS criteria for prescribing lipid-lowering drugs

Patient category	Lipid level for subsidy
Patients with existing coronary heart disease	TC >4 mmol/L
Other patients at high risk with one or more of the following:	
• Diabetes mellitus • Familial hypercholesterolaemia • Family history of CHD (first degree relative <60 years) • Hypertension • Peripheral vascular disease	TC >6.5 mmol/L or TC >5.5 mmol/L and HDL-C <1 mmol/L
Patients with HDL-C <1 mmol/L	TC >6.5 mmol/L
Patients not eligible under the above: • Men 35 to 75 years • Post-menopausal women <75 years	TC >7.5 mmol/L or TG >4 mmol/L
Other patients not included in the above	TC >9 mmol/L or TG >8 mmol/L

PBS = Pharmaceutical Benefits Scheme, TC = total cholesterol, CHD = coronary heart disease, HDL-C = high density lipoprotein cholesterol, TG = triglycerides

STATINS FOR SECONDARY PREVENTION OF CHD

At present it would appear that elderly patients with established CHD should be treated in a similar fashion to younger patients for the initiation of statin therapy. Subgroup analysis of the major interventional lipid-lowering trials for secondary prevention of CHD (4S, CARE, LIPID) have not demonstrated any diminution in the reduction of CHD events in patients aged at least up to 75 years (Table 4). The recent results of the Heart Protection Study also suggest that these effects extend to patients up to 80 years of age.[15,16] Two ongoing studies, PROSPER and FAME, will provide more information on which to base the decision for therapeutic intervention in the elderly.[17]

STATINS FOR PRIMARY PREVENTION OF CHD

There is less evidence from interventional trials to suggest that statin therapy for the primary prevention of CHD in elderly patients is as beneficial as in younger patients. As discussed, this is due to the absence of data from clinical trials in the elderly (Table 5). The results of the Heart Protection Study support the concept that statin therapy in the elderly reduces CHD events in patients who are at high risk of developing clinical cardiovascular disease. The case for the treatment of elderly patients for primary prevention of CHD is also strengthened by the results of the Cardiovascular Health Study.[18] This observational study found a 50% lower risk of CHD events and a 44% lower all-cause mortality for statin vs non-statin users in patients who were 65 years of age or older, free of cardiovascular disease at baseline, and subsequently followed for 7.3 years.

Table 4. Relative risk reduction for major CHD events in lipid-lowering trials in patients with a previous history of ischaemic heart disease

Study*	Drug	Age group (yrs)	RRR (%)	Interaction with age
4S	Simvastatin	35-64 65-70	34 34	NS
PPP (CARE + LIPID)	Pravastatin	31-55 55-64 65-75	27 17 27	NS
VA-HIT	Gemfibrozil	45-66 66-74	22 26	NS
HPS	Simvastatin	40-64 65-69 70-74 75-80	25 30 18 35	NS

CHD = coronary heart disease, RRR = relative risk reduction, NS = not significant; * Scandinavian Simvastatin Survival Study[9] (4S), Cholesterol and Recurrent Events (CARE) and Long Term Intervention with Pravastatin in Ischaemic Heart Disease (LIPID)-results from the Prospective Pravastatin Pooling Project[14] (PPP), Veterans Affairs High-Density Lipoprotein Intervention Trial[21] (VA-HIT), Medical Research Council/British Heart Foundation Heart Protection Study[16] (HPS)-included here as a secondary prevention study although not all those in the study had a previous clinical CHD event

Table 5. Relative risk reduction for major CHD events in lipid-lowering trials in patients with no previous history of ischaemic heart disease

Study*	Drug	Age group (yrs)	RRR (%)	Interaction with age
HHS	Gemfibrozil	40-55	34	-
WOSCOPS	Pravastatin	45-54 55-64	40 27	NS
AFCAPS/ TexCAPS	Lovastatin	43-60 61-73	50 33	NS

CHD = coronary heart disease, RRR = relative risk reduction, NS = not significant; * Helsinki Heart Study[20] (HHS), West of Scotland Coronary Prevention Study[7] (WOSCOPS), Air Force/Texas Coronary Atherosclerosis Prevention Study[8] (AFCAPS/TexCAPS)

EFFECT OF LOWERING TG & RAISING HDL-C

Recent results from the Dubbo Study have suggested that elevated triglyceride (TG) levels are not an independent risk factor for CHD and that the relationship between increased CHD events and elevated TG is explained by the commonly associated finding of a low level of high density lipoprotein cholesterol[19] (HDL-C). A low level of HDL-C is a recognised risk factor for CHD in the elderly. In this study the odds ratio for a myocardial infarction was significantly increased in those with a high TG level and low HDL-C level (1.62, 95% CI 1.06–2.29) whereas it was not significantly elevated in those with a high TG level and a high HDL-C level.

In comparison to studies where statins were used to lower low density lipoprotein cholesterol (LDL-C) there is relatively little information on the effects of lowering TG and increasing HDL-C in the elderly. In one primary prevention study of middle-aged men aged 40–55 years with low HDL-C levels, the Helsinki Heart Study, treatment with gemfibrozil reduced CHD events by 34% after five years[20] (Table 5). To date there have been no similar primary prevention trials in elderly patients. In a secondary prevention trial, the VA-HIT trial, gemfibrozil significantly reduced TG levels and increased HDL-C levels resulting in reduction in death from CHD in patients aged 66–74 years[21] (Table 4).

PRIMARY PREVENTION & ASSESSING CHD RISK

While there is now good evidence supporting the treatment of elderly patients with established CHD with statin therapy, the situation for primary prevention is not as clear. Although elderly patients are at greater risk of CHD events than younger patients, the risk still varies greatly among the elderly without CHD, so some patients would benefit more from preventative treatment than others.

There is a growing consensus of opinion that the use of lipid-lowering medications in patients without CHD should be reserved for those estimated to be at high risk for the development of CHD. Guidelines from the USA use the concept of 'CHD risk equivalents' to identify these high risk patients.[22] These 'CHD risk equivalents' include non-coronary artery forms of atherosclerosis, diabetes, and a 10-year risk for CHD >20% as defined by Framingham risk scoring. Using Framingham risk scoring it is possible to demonstrate the effects that aging has on an individual's CHD risk. For example, if two males aged 50 years and 70 years have identical systolic blood pressures (160 mmHg), TC levels (6.5 mmol/L) and HDL-C levels (1.0 mmol/L), the 10-year risk for CHD is calculated as 16% and 31%, respectively.[22] To more accurately quantify the atherosclerotic plaque burden it is possible to use non-invasive imaging techniques such as the measurement of carotid artery intimal thickness with doppler ultrasound, or coronary artery calcification by CT.[23]

Australian and New Zealand guidelines recommend that lipid-lowering therapy should be commenced not only for secondary prevention of CHD but also for primary prevention in individuals estimated to be at high risk of future CHD events[3] (Table 1). Patients free of CHD but with an absolute risk of a CHD event in the next five years of >10–15% according to the New Zealand cardiovascular risk calculator should be treated. This cardiovascular risk calculator derives a five-year risk level from the patient's gender, presence of diabetes, smoking status, blood pressure level and the ratio of TC/HDL-C using a colour coded card.[24]

COST-EFFECTIVENESS

The cost-effectiveness and cost saving benefits for secondary prevention of CHD with statin therapy in elderly patients projected up to an age of 84 years have been demonstrated in two studies.[25,26] Another study has suggested that gemfibrozil therapy for patients with established CHD and low HDL-C levels is also cost-effective.[27]

A recent analysis of data from the LIPID Study found that for secondary prevention with 40 mg/day of pravastatin in older people (aged 65–75 years), the number needed to treat to prevent one death from CHD was 35 (95% CI 24–67). In comparison, the number needed to treat for a similar effect in younger patients was 71 (95% CI 46–164).[2]

An Australian study has examined the cost-effectiveness of primary prevention with 40 mg/day of pravastatin, when prescribed according to PBS criteria in terms of dollars per year of life saved with a 20-year projection

from 1999.[6] The cost-effectiveness of pravastatin was found to improve dramatically with advancing age. This trend appeared to continue when extrapolated to patients aged up to 80 years. However, when a model which estimates CHD mortality according to multiple and continuous risk factors was used instead of PBS criteria, the cost-effectiveness, as well as the number of potential deaths saved from CHD, was markedly improved. For males with a ≥5% 15-year risk of CHD, the number of lives saved was estimated to be 12 000, with a cost per year of life saved of $23 000. In comparison, using PBS criteria, only 5900 lives were saved at a cost per year of life saved at $110 000. This discrepancy was partly explained by the fact that the PBS criteria do not take into account the strong contribution of age to CHD risk.[6]

DRUG THERAPIES

The various lipid-lowering drugs available in Australia are summarised in Table 6. Although there have been no large trials comparing the effects of the different classes of lipid-lowering drugs in the elderly, the statins are considered first-line therapy for lowering LDL-C. They reduce CHD events, save lives and are well tolerated. This class of drug may also have vascular protective actions independent of cholesterol reduction. Fibrates are useful for patients in whom TG-lowering and HDL-C raising is required and have also been demonstrated to reduce clinical CHD events. Combination therapy, usually in the form of a statin with another lipid-lowering drug, should be considered in high-risk patients with unacceptable lipid profiles on monotherapy.

Statins

The statins block the conversion of HMG-CoA to mevalonate, which is a precursor for cholesterol and non-sterol intermediates. Depending on the dose, most trials have demonstrated a 20–60% decrease in LDL-C, a modest TG lowering effect and a rise in HDL-C of 5–10%. The mechanisms by which statins bring about the latter effects are not known. Recent trials involving simvastatin or pravastatin have demonstrated a 17–25% decrease in CHD events in patients aged up to 80 years.[2,16] A meta-analysis of statin trials has shown a 24–34% reduction in the risk of stroke in secondary CHD trials but a non-significant reduction in primary prevention trials.[28] In the Heart Protection Study, simvastatin produced a highly significant 25% reduction in the incidence of stroke in patients at high risk of vascular disease.[16]

The optimum dose of the various statins and whether there are benefits of one statin over another in terms of reducing vascular events remains uncertain. Only three of the statins—simvastatin, pravastatin and lovastatin—have been shown to reduce CHD events in large clinical studies. There is some evidence to suggest that a higher proportion of patients treated with atorvastatin will achieve recommended lipid targets compared with simvastatin.[29]

The main potential side effects of treatment with statin drugs are myositis and elevations in liver enzymes. Except for pravastatin, all of the available statins are metabolised by the cytochrome P_{450} (CYP) system. Serum concentrations of these statins can potentially be increased in patients who are prescribed drugs which inhibit the CYP system, increasing the chances of myositis. Interaction of statins with foods, particularly grapefruit juice, which contain a CYP isoenzyme (CYP 3A4) inhibitor, may potentially have a similar effect. Despite this, the incidence of serious adverse events associated with the use of this class of drug is low and discontinuation rates due to adverse events are less than 10%. In the Heart Protection Study, an increase in creatine kinase greater than ten times the upper limit of normal and alanine aminotransferase (ALT) greater than three times the upper limit of normal was no different in 10 000 patients treated with simvastatin or 10 000 patients treated with placebo.[16] However, the possibility of myositis is increased when statins are used in combination with a fibrate or cyclosporin. Although liver dysfunction is rare, it is recommended that liver function tests are measured once, about six weeks after commencing therapy, and patients with pre-existing liver disease are monitored three-monthly.

Although reduced LDL-C is probably responsible for most of the vascular protection provided by statins, their full benefit may be partly the result of other mechanisms, referred to as pleiotropic effects. These may include anti-thrombotic, anti-atherogenic and anti-inflammatory effects, improvements in endothelial function and cytokine modulation. Recently, the MIRACL Study has further strengthened the case for a direct effect of statins on the vasculature, as atorvastatin reduced the rate of recurrent CHD events following acute coronary syndrome as early as 16 weeks after its initiation.[30] Other improved outcomes with statins may include a reduction in deep vein thrombosis,[31] and there is some preliminary evidence to suggest that the risk of developing dementia can be reduced.[32,33] Although an inconsistent

Table 6. Lipid-lowering medications available in Australia

Drug class	Generic name	Strength	Dosing	Brand names
Statins	Atorvastatin	10, 20, 40, 80 mg	Daily	Lipitor
	Fluvastatin	20, 40 mg		Vastin, Lescol
	Pravastatin	10, 20, 40 mg		Pravachol
	Simvastatin	5, 10, 20, 40, 80 mg		Lipex, Zocor
Fibrates	Gemfibrozil	600 mg	Twice daily	Lopid, Jezil, Lipazil, Gemhexal, Ausgem
Resins	Cholestyramine	4 g, 8 g sachets	Twice daily	Questran Lite
	Colestipol	5 g sachets		Colestid
Miscellaneous	Nicotinic acid*	250 mg	Three times daily	Nicotinic acid
	Probucol*	250 mg	Twice daily	Lurselle

* The usual therapeutic doses of Nicotinic acid and Probucol are approximately 1 g three times daily and 500 mg twice daily, respectively

finding, a recent observational study has suggested that statins may possibly reduce the risk of fracture.[34]

Fibrates

The main benefit of the fibrate class of drugs is their ability to lower TG levels and increase HDL-C levels. They work by activating the nuclear peroxisome proliferator activated alpha receptor, which results in an increased production of lipoprotein lipase. It is now established that low serum HDL-C is a risk factor for CHD in the elderly.

Fibrate therapy is associated with a decrease in TG of 25–35%, an increase in HDL-C of 11–14% and a decrease in LDL-C of approximately 11%. To date, no large trials have examined the effects of fibrate therapy on improvement in lipids or CHD events in patients aged over 75 years. In younger patients, the risk of myocardial infarction was reduced by 9–34% in four large clinical studies: WHO Clofibrate Study,[35] HHS,[20] BIP[36] and VA-HIT.[21]

Fibrates are generally well tolerated. In patients with renal impairment the dose should be reduced because of a greater risk of myositis.

Nicotinic Acid

Nicotinic acid is another drug that mainly works by lowering TG and increasing HDL-C. When used at doses of 3–4 g per day, HDL-C levels increase by 20–50% and TG levels decrease by 5–30%.[3] Nicotinic acid has beneficial effects on lipids by inhibiting the release of fatty acids from adipose tissue. The drug has a relatively high discontinuation rate due to its poor tolerability, the main side effects being hot flushes, itching and gastrointestinal side effects. However, with proper dose adjustments and patient education, the discontinuation rate can be reduced. Also, the simultaneous use of aspirin is effective, as side effects are prostaglandin mediated. The possibility of worsening glucose intolerance has been a barrier to the use of nicotinic acid in the past, but a recent study has demonstrated that lipid-modifying doses of nicotinic acid in patients with diabetes do not lead to a deterioration in glycaemic control over a 60-week period.[37]

Bile Acid Sequestrants

Bile acid sequestrants (resins) act by binding bile acids in the intestine. This results in an increased conversion of cholesterol to bile acids in the liver, a transient decrease in hepatic cholesterol content, an increased expression of hepatic LDL-C receptors and hence an increased clearance of LDL-C from the circulation. Total cholesterol levels can be decreased by 11–20%. However, high doses are required, which results in an increased incidence of side effects such as bloating and constipation. They can also reduce the absorption of other medications and should be taken three hours before or one hour after drugs which may interact. These problems preclude the use of bile acid sequestrants for many elderly patients.

Combination Therapy

The combination of a statin with either a fibrate, resin, or nicotinic acid, should be considered in patients who have not reached recommended lipid profile targets. Recently there has been increased concern about the risk of myositis with statin therapy, especially in combination with a fibrate. This potential side effect led to the withdrawal of cerivastatin in 2001. However, according to the Australian Lipid Management Guidelines the risk of myositis is almost certainly outweighed by the benefits of combination statin and fibrate therapy in people at high risk of a CHD event, especially those with an elevated LDL-C level and low HDL-C level.[3] Of note, this combination needs to be used with great caution in patients taking cyclosporin, erythromycin or protease inhibitors, as the risk of myositis is further increased.

The combination of a statin and nicotinic acid is generally well tolerated and can produce both clinical and angiographically measurable benefits in patients with CHD who have low HDL-C levels.[38]

A study of patients aged approximately 55 years at high risk of CHD events demonstrated that combination therapy involving a statin with either a fibrate or nicotinic acid decreases TC by 35%, LDL-C by 37%, TG by 62% and increases HDL-C by 23%. This combination therapy was not associated with any cases of severe myopathy or rhabdomyolysis, or drug-induced hepatitis over a 7.5-year period in 136 patients.[39] A discontinuation rate of around 10% was noted but this may be significantly higher in the elderly.

LIFESTYLE INTERVENTIONS

To date there have been no dedicated studies comparing the relative effectiveness of lifestyle interventions in the treatment of dyslipidaemia and/or in reducing cardiovascular events in older compared with younger people. However lifestyle interventions (summarised in Table 7) can contribute to a reduction in cardiovascular events independent of lipid-lowering, and these should be considered as an important component of therapy for most patients with dyslipidaemia. Lifestyle modification should be first-line management in patients with dyslipidaemia except in those with existing coronary artery disease and a TC >4.0 mmol/L where drug treatment should be commenced without awaiting the assessment of dietary intervention. Even after drug therapy has commenced, lifestyle interventions should continue as an important adjuvant to pharmacological measures. However, it may be difficult to instigate lifestyle modification in the elderly for several reasons. Firstly, nutrition in the elderly is often poor, and eating habits may be difficult to alter, especially if the benefits are not immediately obvious to the patient. Furthermore, an active exercise program may not be possible. Therefore the benefits of lifestyle modification in terms of improvement in lipids and reducing vascular events need to be balanced against practical limitations of initiating and maintaining these changes in the elderly.

CONCLUSION

The results of recent clinical trials suggest that treating dyslipidaemia in the elderly results in similar outcomes to those observed for younger patients. There is also evidence to suggest that the expected reduction in vascular events seen with statins is not just due to their lipid-lowering effects, but also to possible direct effects on the vasculature. Statins should be considered for secondary prevention of CHD events in all patients, irrespective of age.

In terms of primary prevention, lifestyle modification, if possible, remains first-line therapy for correcting dyslipidaemia. If the abnormality persists, a statin should be prescribed if LDL-C lowering is required. If the main

Table 7. Summary of lifestyle interventions for patients with dyslipidaemia*

1. Encourage healthy eating:

- Use spreads, dressings, and cooking oils containing polyunsaturated and monounsaturated fats
- Choose low fat milk and dairy products
- Adopt a 'Mediterranean' style diet, high in fresh fruit and vegetables, fish, poultry, cereal grains and unsalted nuts
- Have fish at least twice a week.
- Limit cholesterol-rich foods such as egg yolks
- Only eat lean meats

2. Stop smoking

3. Undertake 30 minutes of moderate-intensity physical activity daily

4. Aim for a body mass index (BMI) <25 and a waist circumference <90 cm in men and <80 cm in women

5. Limit alcohol intake

6. Reduce salt intake

*Modified from the Australian Lipid Management Guidelines [3]

lipid abnormality is elevated TG and low HDL-C then a fibrate such as gemfibrozil should be considered. Both these drugs are well tolerated in the elderly. Combination therapy, usually in the form of a statin with another lipid-lowering drug, is an option in high-risk patients with unacceptable lipid profiles on monotherapy. Although the absolute risk of clinical vascular events increases with age, the decision to start a statin should only be made after assessment of a patient's overall cardiovascular risk.

Competing interests: None declared

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Submitted: July 2002
Accepted after external peer review: July 2002