

14. STRATEGY OF PREVENTION OF CARDIOVASCULAR DISEASE

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We all have a dream: all of us want to live a long, healthy, and successful life in full possession of our physical and mental faculties, surrounded by family and friends. We all want to be respected, to feel that we are needed, and to be useful and happy at work and in family life. Lives cut off before their time, people we care for often, and increasingly leaving us for eternity while young or middle-aged, sometimes at the peak of their capacities, leaving painful and continuously open wounds in families, society, and the economy. All of us bear a responsibility for changing this situation.

The most often publicized North-Karelia Program was set into motion in Finland in 1972 in response to a local petition. The program achieved its success rate through cooperation on all levels of government, and offered urgent and effective assistance in reducing an extremely high level of mortality from coronary heart disease. While this program ran, it reduced the mortality rate from coronary heart disease among the male population by about 50%. The program focused on organizing ongoing care for people with hypertension, and on a public information campaign that increased the ratio of the people overall for whom physical fitness became an element in their lifestyle, while reducing the number of cigarette smokers at the same time. A national dietary and nutritional policy also was put into operation. It included a full-scale public education program combined with promoting the manufacture of an increased variety of healthy foods and introducing healthy diets to public meal services. In other words, there is an existing model that can be adapted to most Central-European conditions and, if it is implemented consistently, success is guaranteed.

However, good programs alone are not enough. Success also requires the kind of cooperation by all of society that we have been able to muster several times before in our history, and which has seen us through some hard times. We are currently in the midst of one of these historical watersheds. We only will be able to close down our

transition to a new political system and become a successful and integral part of Europe if we can prevent the loss of our supreme resource, the creative power of our citizens, and if our population stops declining. This requires determination and innovative program designs by government and proactive participation by all of society in the belief that it can be done. To see results in ten to twenty years, we need to go into motion immediately, in our own private lives as well as on a society-wide scale. The only real guarantee of a successful future for Hungary is our people, people who are aware of what needs to be done and are ready to act for their own sake and for the sake of their children!

14.1 The Hungarian situation

The overall state of public health in Hungary has been in continuous decline for more than thirty years. In 1999, average life expectancy at birth for the male population was 66.37 years, and for the females it was 75.24 years, well below the European Union average (where in 1997 the rate for males was 74.84 years and for females it was 81.24 years), and also lower than corresponding data from neighboring Central and Eastern European nations. The mortality rate for middle-aged males is particularly high, even in worldwide comparisons. Although there has been a slight improvement here in recent years, the consensus of opinion is that a substantive breakthrough in this very slowly improving trend will require society-wide and coordinated intervention.

The leading causes of mortality in people under the age of 65 (which qualifies as premature) are disorders of the circulatory system (the same as for overall mortality). Within these disorders, high blood pressure plays a decisive role in the onset of coronary heart disease (primarily myocardial infarction) and cerebrovascular diseases (stroke and other cerebrovascular accidents). Among Hungarians, premature mortality due to coronary heart disease is three times the average rate for the European Union, and it is four times that average for cerebrovascular diseases. Mortality due to malignant neoplasms is also far in excess of the EU average (1.8-times higher). Among men, lung cancer is the type of tumour causing the highest death rate, while among females the prime killers are breast and cervical cancer. Hungary's premature male mortality rate from lung cancer and premature female mortality rate from cervical cancer are particularly high when compared to the EU average. The male premature lung cancer mortality rate is 2.5 times over the EU average, and the female premature cervical cancer mortality rate is 3.5 times higher. At the same time, premature mortality due to malignant colon and colorectal tumours has been rising in both men and women. Another acute problem is the 7-8-fold increase in premature mortality due to chronic liver diseases and cirrhosis of the liver - mostly alcohol-induced - over the past 30 years. By the mid-1990s this mortality rate was

sevenfold (7-fold!) the EU average. Death due to violence needs to be cut in half to meet the EU average. Within the causes of violent death, premature mortality due to suicide continues to be more than double the average level among EU-residents, even though it has declined continuously over the past decade.

Underlying causes include unhealthy lifestyles, shortcomings in health habits, environmental pollution, the country's economic development level, difficulties with performance, social inequality, and a substandard level of healthcare. Many people smoke cigarettes and are heavy smokers to boot, consume unhealthy foods, are completely sedentary, consume excessive amounts of alcohol, pursue self-destructive lifestyles, pay no attention to their health, avoid screening programmes, and do not utilize other forms of healthcare either. The factor that *ab ovo* causes differences in lifestyle is social inequality - the scale of differences between the poorest and richest strata - principally the result of differences in the education level, and the ensuing employment and income conditions. Health problems appear cumulatively among disadvantaged social groups, the groups that were the losers when the political system changed. Traditional relationships have lost their adhesive power, and century-long generational bonds have collapsed. Rural residents are even less healthy than urban ones.

14.2 The New Hungarian Program: Goals, Tasks, and Projects

Contemporary health promotion/public health programs are based on the following principles:

- Individual health is determined predominantly by environment and lifestyle.
- Determinants of health relate to one another in a complex manner and the predominant factors are generally not pathology-specific - in other words, they can trigger any of a number of diseases.
- An individual's immediate environment (family, workplace, leisure time, etc.) is able to influence personal lifestyles (which have a decisive influence on health), because of the individual's natural need to be part of a community and to meet community expectations.
- Improving public health requires action by all of society, including inter-sectoral cooperation and acceptance of responsibility, and a partnership between all players that make up society (central government, local government, and grass roots communities).

14.2.1 The Five National Targets to reach by 2010

Health has to become a supreme human value for the vast majority of the public, which in turn has to be made ready to act to maintain its health. Decision-makers, both in legislation and in setting the budget, have to give a top priority to improving the nation's health.

- 1 Conditions for healthy development have to be guaranteed for upcoming generations, from conception to adulthood.
- 2 Years of life spent in good health have to be extended for both males and females.
- 3 Average life expectancy at birth has to be extended to at least 70 years for men and 78 years for women.
- 4 Social inequalities and differences in life expectancy at birth have to be reduced.

14.2.2 Ten Priority National Tasks

- 1 Education for health, heightening education and health consciousness
- 2 Early detection of diseases of major public health importance by introducing targeted population screening
- 3 Widespread dissemination of healthy nutrition
- 4 Promoting a physically active lifestyle essential to healthy life
- 5 Combating addictions (excessive alcohol consumption, tobacco smoking, drugs)
- 6 Establishing and maintaining equal opportunity to a healthy life
- 7 Improving epidemiological safety
- 8 Improving food safety conditions - preparing for new challenges
- 9 Evolving an environment conducive to health, thereby reducing death due to external causes
- 10 Improving the healthcare system with a focus on public health considerations

14.2.3 Seventeen Programs to reach the targets and implement the tasks

The 17 implementation programs include ones that are targeted specifically at combating disorders that play the greatest role in quality-of-life deterioration and mortality (cardiovascular diseases, tumours, mental disorders, and mobility impairments). There are also non-specific action plans and key measures that target health determinants (education, nutrition, exercise, tobacco smoking, drug use and alcohol consumption). By implementing them, health can be improved and the risks of multiple diseases and disorders can be reduced.

- 1 Reducing mortality due to myocardial infarction
- 2 Reducing mortality due to cerebrovascular diseases
- 3 Reducing tumour morbidity
- 4 Preventing mental disorders
- 5 Reducing the burden of mobility disorders borne by individuals and society
- 6 Guaranteeing a healthy start in life and childhood
- 7 Promoting equity in health for multiply disadvantaged population groups
- 8 Evolving a healthy environment

- 9 Improving epidemiological safety
- 10 Improving food safety
- 11 Reducing mortality due to external causes
- 12 Expanding population screening programs
- 13 Promoting healthy nutrition
- 14 Reducing smoking
- 15 Health promotion in education
- 16 Encouraging a physically active lifestyle
- 17 Reducing alcohol consumption and drug use

14.2.3.1 Reducing Mortality Due to Myocardial Infarction:

The Target:

To reduce mortality due to coronary heart disease among the under-65 population by 20% by 2010.

The Situation:

Diseases of the circulatory system are the leading cause of death in Hungary. Coronary heart diseases, in particular myocardial infarction are the list leaders. Circulatory system disorders are responsible for one out of every three deaths among the under-65 population. Self-destructive lifestyles - typically cigarette smoking; unhealthy nutrition and a lack of exercise; neglected, insufficiently, or poorly treated hypertension; and often the shortcomings of acute care, whether occurring separately or in combination, put all of us at increased risk or further reduce our chances for a fuller, healthy and creative life.

While, in Western Europe, changes in nutritional habits, an increased interest in exercise, and up-to-date drug therapies have reduced mortality due to circulatory system disorders (coronary heart disease, cerebrovascular accidents, peripheral vascular disorders, and aorta anomalies) by 40-50% since 1970, mortality rates in Hungary have increased. As a result of these two opposing trends, in 1997 premature deaths (occurring at ages 0-64) from circulatory disorders including coronary heart disease were three times over the average for the European Union, and even exceeded corresponding average mortality rates for the populaces of the Central and East European nations.

All three of the key factors - lifestyle, environment and healthcare - probably contributed to the trends evolving over past decades. As far as the healthcare service is concerned, shortcomings in preventive, curative, and rehabilitation services for cardiovascular patients were probably major contributors to the deterioration in cardiovascular health, though there also have been encouraging results in recent years. From 1997 there has been a small but steady decline in coronary heart disease among men aged 15 to 64, including a drop in mortality from acute myocardial infarction. Today, the rate of properly treated hypertensive patients ranges from 17% to

28%, depending on geographic region and the particular population being surveyed. This rate can be pushed up to 90% with the proper continuing education of physicians and with patient education.

Other nations report a great deal of success in preventing coronary heart disease. Finland instituted a program called The North-Karelia Program in 1972, in response to a local plea. The program, which offered urgent and effective help in reducing an extremely high mortality rate from coronary heart disease, achieved its success rate because of cooperation by all levels of government. Results show that in 1972 some 52% of the male population smoked cigarettes, a figure that declined to 32% by 1992. Dietary changes succeeded in reducing the total cholesterol level by 15% among the population of North-Karelia. The program also involved continuous care for hypertensive patients, and an information campaign that increased the portion of the public that exercised regularly. During the time the program ran, the coronary heart disease mortality rate among the male population of North-Karelia dropped by about 50%. Between 1970 and 1996, mortality from coronary heart disease dropped by about 65% among under-65 males and females. (In 1970 the death rate of Finnish males from coronary heart disease was 2.4 times higher than it was for Hungarian males, while today it is just slightly more than half that rate, i.e., 58%)

A working group of leading professionals came together over two years ago, and were given the task of designing the best national program it could, tangibly to reduce Hungary's mortality rate from myocardial infarction within a 10 year time frame.

Goals up to 2010:

- Reducing mortality due to coronary heart disease among the under-65 population by 20%.
- Increasing the effectiveness of regular screening for hypertension.
- Increasing the rate of hypertensive patients under regular treatment to at least 85%.
- Introducing effective treatment of hypertension to increase the rate of patients whose blood pressure is lower than the threshold value of 140/90 Hgmm to at least 50%.
- Advancing methods for the primary and secondary prevention of cardiovascular diseases: promoting healthy nutrition, promoting physical exercise, reducing cigarette smoking, increasing the focus on health improvement within the educational system.

Indicators:

- **Myocardial infarction mortality data.** Risk factors: data on cigarette smoking, diet, nutrition, exercise (see corresponding sub-programs).
- **Coronary heart disease morbidity data (incidence, prevalence).** Rate of hypertensive patients receiving regular treatment, rate of patients with satisfactory blood pressure readings.

Actions:

- 1 Surveying the prevalence of high blood pressure and of risk factors, conducting population surveys and collecting morbidity data.
- 2 Screening the population for high blood pressure (see separate section).
- 3 Improving the quality in primary health care for hypertension.
- 4 Running a satisfactory pharmaceutical care program for hypertensive patients on an ongoing basis.
- 5 Designing, establishing, and operating a national registry of hypertensive patients and the setting up the information system needed for it.
- 6 Elaborating the organizational and operational conditions for hypertension treatment facilities, and accrediting their operation.
- 7 Introducing extension continuing education and training programs both for patients, for the public at large, as well as for physicians and allied health personnel.
- 8 Improving the conditions for emergency care.
- 9 Healthy nutrition: reducing cholesterol levels and obesity.
- 10 Promoting physical exercise.
- 11 Reducing smoking.
- 12 Health promotion in the educational system.

14.2.3.2 Reducing Mortality due to cerebrovascular diseases

The Target:

To reduce permanent damage and mortality from cerebrovascular diseases, and to decrease CVD incidence.

The Situation:

Stroke is the third leading cause of death in Hungary following coronary artery occlusions and tumours, just as in the developed nations of the world. International trends and a World Health Organization projection strongly suggest that as populations of industrial societies grow older, there will be a shift in the leading causes of mortality, and stroke will become even more significant as a leading cause of death.

In the 1950s, death due to stroke was more or less equal to the average for Europe. But from then on, until 1980, the stroke mortality rate in Hungary increased enormously, significantly exceeding the European average, and was the sixth highest in Europe. Despite a steady decline since the early 1980s, the stroke mortality rate is still more than double the European Union average, for both males and females. In 1999, over 19,000 Hungarians died of strokes.

The stroke mortality rate is particularly high among the under-65s, nearly 4.5 times over the EU average for males and 3.5 times higher for females.

Hemorrhages or blood clots resulting in permanent damage (non-transient ischemic attacks) lead to death within 30 days in 12-20% of cases and to death within one year in 25-30%. The overall stroke survival rate for all types of stroke is no more than 2-3 years following the onset of the accident. About 60-70% of people suffering stroke are not taken to specialized hospital wards. The result is a roughly 4% higher mortality rate during the acute phase of the attack than occurs in facilities specialized in strokes, which translates into over 500 avoidable deaths each year.

Another consequence of cerebrovascular diseases is permanent damage to health in 60% of cases overall, manifest not only in physical symptoms (paralysis, difficulties in understanding or vocalizing speech, loss of the ability to swallow), but also in injury to thought processes in 20% of cases, and in depression in 40%. Quality of life deteriorates significantly.

Brain damage is one of the most significant factors behind the deterioration in quality of life among the elderly. A stroke qualifies as a disaster, not only for the patient but also for the family and for society in general. The condition requires long-term care, which makes it one of the most expensive, and physically and emotionally exhausting of all conditions.

The problem of care for stroke patients first became a centre of focus in 1988, and in 1992 it was widely publicized with the announcement of a National Stroke Program. However, figures on incidence in 2001 show the continued urgent need to elaborate and introduce an updated strategy enabling the nation to significantly reduce the overall incidence of stroke, the mortality due to stroke, and the other burdens incurred.

Goals up to 2010:

- Reducing acute stroke mortality by 30%.
- Reducing morbidity by 30%.
- Treating a larger ratio of stroke patients in specialized wards or facilities.
- Broadly promoting the following in working towards the primary and secondary prevention of vascular disorders: promoting healthy nutrition, promoting physical exercise, reducing smoking and alcohol consumption, increasing the focus on health promotion within the educational system.
- Improving the effectiveness of screening for hypertension, and treating hypertensive patients.
- Creating a framework for effective rehabilitation.
- Designing the criteria for centrally accrediting stroke wards by 2002, and enforcing them subsequently. Re-establishing a national database suitable for epidemiological studies

Indicators:

- Stroke mortality data.
- Stroke morbidity data (incidence, prevalence).
- Risk factors: data on smoking, diet, nutrition, physical exercise (see corresponding sub- programs).
- Frequency of referral to specialized facility.
- Frequency of permanent damage to health.
- Frequency of post-stroke depression, suicide.

Actions:

- 1 Finishing the development of a domestic stroke hospital network corresponding to European norms to treat CVAs.
- 2 Providing the diagnostic conditions needed for the operation of stroke wards.
- 3 Elaborating the methodology for circulatory (vascular) screening programs and providing the funding for their operation; starting up stroke genetic programs.
- 4 Designing a national stroke registry.
- 5 Updating undergraduate, postgraduate and continuing medical education in keeping with the National Stroke Program; introducing new distance-learning programs in this area.
- 6 The “Tele-Stroke” Program.
- 7 Designing various rehabilitation programs to include funding models.
- 8 Developing home healthcare with an alarm system.
- 9 Designing and introducing programs of communication with the public. Providing information to teachers.
- 10 Heightening the fight against cigarette smoking, to include a priority no-smoking program for people under the age of 35.
- 11 Reducing excessive alcohol consumption.
- 12 Promoting healthy nutrition.
- 13 Promoting physical exercise.
- 14 Developing a healthy lifestyle.
- 15 Screening the public for hypertension, focusing particularly on the under-55s, and screening the over-65s for atrial fibrillation.
- 16 Treating hypertension properly.
- 17 Introducing a program of regular pharmaceutical care for hypertensive patients (pages 1-15 copied from “Healthy Nation - Public Health Program”, Ministry of Health, Social Affairs and Family Protection, Hungary).

14.3 Primary prevention

Primary prevention generally means the effort to modify risk factors or prevent their development with the aim of delaying or preventing new-onset CHD.

14.3.1 Concept of Risk Factors

The concept of risk factors constitutes a major advance for developing strategies for preventing CHD. The **major risk factors** are **cigarette smoking, hypertension, high serum cholesterol and various cholesterol fractions, low levels of high-density lipoprotein (HDL) cholesterol, and diabetes mellitus.** **Advancing age** is also included as a risk factor because of increased absolute risk with aging.

Factors other than those listed as major risk factors increase the likelihood for developing CHD. Among these, which have been studied at Framingham or elsewhere, are obesity, physical inactivity, family history of premature CHD, hypertriglyceridemia, small low-density lipoprotein (LDL) particles, increased lipoprotein (a) (Lp[a]), increased serum homocysteine, and abnormalities in several coagulation factors.

Despite the potential importance of these other factors, they are not included in the risk charts for both theoretical and practical reasons. Nonetheless, they deserve some comment and consideration of reasons for omission.

Framingham research reveals that both **obesity and physical inactivity** are positively associated with risk for CHD. Even so, Framingham data suggest that obesity and physical inactivity exert much of their adverse influence on development of CHD through the major risk factors. Certainly it is possible that some of the increased risk imparted by obesity and physical inactivity results from mechanisms unrelated to the major risk factors. However, these mechanisms are not well understood, and it is difficult to define the risk imparted by these 2 factors independent of their influence on the major risk factors. Certainly both public health and clinical efforts to promote desirable body weight and regular exercise deserve a high priority in prevention.

Family history undoubtedly gives useful information about an individual's risk status. However, the independent effect of a positive family history is difficult to determine. Almost certainly familial influences on risk status are mediated in part through blood pressure and serum lipoprotein levels. Even so, a positive family history of premature CHD cannot be ignored in clinical evaluation. Not only should such a history increase awareness that an individual is at greater risk, but it calls for evaluation of other family members who may carry heritable risk factors.

Framingham risk scores do not include **serum triglyceride levels.** Much research confirms that elevated serum triglycerides are significantly correlated with risk. However, a controversy has raged for many years over whether elevated triglycerides are an independent risk factor. For example, triglyceride levels are inversely correlated with serum HDL-cholesterol levels. This approach does not necessarily mean that triglyceride-rich lipoproteins are not atherogenic. There is growing evidence that certain species of these lipoproteins are in fact atherogenic and probably should be targets of therapy. Even so, for risk assessment, HDL-cholesterol levels reflect a significant portion of the risk imparted by higher serum-triglyceride concentrations.

Another lipoprotein abnormality, small dense LDL particles, is likewise strongly associated with low serum HDL-cholesterol levels. Small dense LDL particles may promote atherosclerosis.

Future research will be required to define the independent contributions of the 3 components of the atherogenic lipoprotein phenotype - elevated triglyceride-rich lipoproteins, small LDL particles, and reduced HDL cholesterol - to overall CHD risk. Use of serum HDL-cholesterol levels to define the risk accompanying this complex phenotype is undoubtedly an oversimplification, but this drawback is partially offset by clinical usefulness.

Lp(a) may be still another lipid risk factor. Several reports indicate that elevated serum Lp(a) concentrations are associated with high risk for CHD. Although other reports fail to document a significant link between Lp(a) levels and CHD rates, the preponderance of the evidence seems to support a significant relationship. However, before measurements of Lp(a) levels can be routinely used in risk prediction, a stronger link between Lp(a) and atherogenesis must be established, and accurate and inexpensive measurements must be widely available.

Another category of candidate risk factors includes **abnormalities in several coagulation factors**. Among these factors are platelet hyperreactivity, high levels of hemostatic proteins (**fibrinogen** and factor IV), defective **fibrinolysis**, and **hyperviscosity** of the blood. The most extensive epidemiological data link plasma fibrinogen concentrations to CHD risk.

In addition, evidence suggests that plasma markers for endothelial cell injury and inflammation may be predictors of acute coronary events. Research on these various factors promises to provide new insights into the pathogenesis of CHD, but their quantitative roles have not been determined sufficiently to include them in risk prediction equations. Moreover, accurate measurements of these coagulation factors are not yet widely available to practicing physicians.

In recent years there has been a growing interest in the possibility that a condition called **insulin resistance** underlies several metabolic risk factors, predisposing the individual to premature CHD. Insulin resistance refers to a generalized metabolic disorder in which various tissues are resistant to normal levels of plasma insulin. Metabolic abnormalities include defective glucose uptake by skeletal muscle, increased release of free fatty acids by adipose tissue, overproduction of glucose by the liver, and hypersecretion of insulin by pancreatic β -cells. The presence of insulin resistance can usually be detected clinically by truncal (or abdominal) obesity and hyperinsulinemia. CHD risk factors often present in patients with insulin resistance include the atherogenic lipoprotein phenotype, hypertension, impaired glucose tolerance, and a prothrombotic state. This clustering of several metabolic risk factors in a single patient has been termed the metabolic syndrome.

The final risk predictor is serum homocysteine level. Persons with the rare congenital disorder homocysteinuria develop severe arterial disease; this discovery gave rise to the theory that high homocysteine levels may be a cause of CHD. Furthermore, according to several studies, moderately elevated serum levels of homocysteine in the general population are positively associated with CHD occurrence. In addition, patients with a genetic defect in an enzyme producing high homocysteine levels also appear to be at increased risk for CHD. Whether measurement of plasma homocysteine concentrations is clinically useful in risk stratification is uncertain but worthy of further investigation.

According to the most recent US panel (JAMA, 2001), the two major modalities of LDL-lowering therapy are therapeutic lifestyle changes (TLC) and drug therapy (Table 2). The TLC Diet stresses reductions in saturated fat and cholesterol intakes. When the metabolic syndrome or its associated lipid risk factors (elevated triglyceride or low HDL cholesterol) are present, TLC also stresses weight reduction and increased physical activity. The table below defines LDL cholesterol goals and cutpoints for initiation of TLC and for drug consideration for persons with three categories of risk: CHD and CHD risk equivalents; multiple (2+) risk factors (10-year risk 10-20% and <10%); and 0-1 risk factor.

TLC⁺:

- Reduced intakes of saturated fats (<7% of total calories) and cholesterol (<200 mg per day)
- Therapeutic options for enhancing LDL lowering such as plant stanols/sterols (2 g/day) and increased viscous (soluble) fiber (10-25 g/day)
- Weight reduction
- Increased physical activity

14.4 Secondary prevention

The term secondary prevention denotes therapy to reduce recurrent CHD events and decrease coronary mortality in patients with established CHD. Secondary prevention strategy is aimed at both control of risk factors and direct therapeutic protection of coronary arteries from plaque eruption. This dual approach has led some investigators to view secondary prevention efforts as treatment of coronary artery disease. Although there may be a slim distinction between secondary prevention and high-risk primary prevention, once a patient has exhibited clinical atherosclerotic disease, he or she is unequivocally at very high risk for developing new acute coronary events. For purposes of secondary prevention, manifest atherosclerotic disease includes angina pectoris or history of documented myocardial infarction, history of coronary artery procedures (bypass graft, angioplasty), peripheral arterial disease, aortic aneurysm, symptomatic coronary artery disease.

Recent clinical trials demonstrate that LDL-lowering therapy reduces total mortality, coronary mortality, major coronary events, coronary artery procedures, and stroke in persons with established CHD. An LDL cholesterol level of < 2.6 mmol/l is optimal; therefore, ATP III specifies an

Table 1. Risk factors and recommendations for preventing

Risk intervention	Recommendations	
Smoking: Goal complete cessation	Ask about smoking status as part of routine evaluation. Reinforce nonsmoking status. Strongly encourage patient and family to stop smoking. Provide counseling, nicotine replacement, and formal cessation programs as appropriate.	
Blood pressure control: Goal <140/90 mm Hg or <130/85 mm Hg if heart failure, renal insufficiency or diabetes	Measure blood pressure in all adults at least every 2 years. Promote lifestyle modification: weight control, physical activity, moderation in alcohol intake, and moderate sodium restriction. If blood pressure > 140/90 mm Hg after 3 months of lifestyle modification or if initial blood pressure >160/100 mm Hg or >130/85 mm Hg with heart failure, renal insufficiency or diabetes, add blood pressure medication. Individualize therapy to patient's age, race, need for drugs with specific benefits, etc.	
Cholesterol management: Primary goal LDL <4.1 mmol/l if 0-1 risk factors or LDL <3.3 mmol/l if 2 risk factors.	Ask about dietary habits as part of routine evaluation. Measure total, cholesterol, HDL cholesterol and triglycerides in all adults 20 y and assess positive and negative risk factors at least every 5 years. For all persons: promote therapeutic lifestyle changes (TLC), AHA Step I diet (<30% fat, <10% saturated fat, <7.75 mmol/l cholesterol), weight control, and physical activity.	
	If LDL: 4.1 mmol/l with 0-1 risk factors; or 3.3 mmol/l on 2 occasions with 2 risk factors; then Start Step II diet (<30% fat, <7% saturated fat, <5.2 mmol/l cholesterol) and weight control. Rule out secondary causes of high LDL (LFTs, TFTs, UA). If LDL: 4.1 mmol/l plus 2 risk factors; or 4.9 mmol/l; or 5.7 mmol/l in men <35 y; or in premenopausal women; then consider adding drug therapy to diet therapy for LDL levels > those listed above that persist despite Step II diet.	Risk factors that modify LDL goals: age (men 45 y, women 55 y or postmenopausal), hypertension, smoking, HDL < 1.04 mmol/l, family history of CHD in first-degree relatives (in male relatives <55 y, female relatives <65 y)
Physical activity: Goal Exercise regularly 3 - 4 times / week for 30-60 minutes	Ask about physical activity status and exercise habits as part of routine evaluation. Encourage 30 minutes of vigorous-intensity dynamic exercise 3 to 4 times per week as well as increased physical activity in daily life style activities (e.g., walking breaks at work, gardening, household work). Advise medically supervised programs for those with low functional capacity and/or comorbidities.	
Weight management: Goal BMI 21-25 kg/m²	Measure patient's weight and height, BMI, and waist-to-hip ratio at each visit as part of routine evaluation. Start weight management and physical activity as appropriate. Desirable BMI range: 21-25 kg/m ² . Desirable waist circumference <101 cm in men and <91 cm in women.	
Diabetes management: Near normal fasting	Appropriate hypoglycemic therapy to achieve near normal fasting plasma glucose as indicated by HbA1c. Treatment of other risks (e.g., physical activity, weight management, blood pressure and for cholesterol management see recommendations for patients with coronary disease).	

TG indicates triglycerides; LTs, liver function tests; TFTs, thyroid function tests; UA, uric acid; CHD, coronary heart disease; and BMI, body mass index.

Table 2. LDL-lowering therapy according to recent US panel (JAMA, 2001)

Risk category*	LDL goal	LDL level at which to initiate therapeutic lifestyle changes (TLC+)	LDL level at which to consider drug therapy
CHD or CHD risk equivalents (10-year risk < 20%)	< 2.6 mmol/l	> 2.6 mmol/l	> 3.3 mmol/l
2+ risk factors (10-year risk < 20 %)	< 3.3 mmol/l	> 3.3 mmol/l	10-year risk 10-20 %: > 3.3 mmol/l; 10-year risk < 10 %: > 4.1 mmol/l
0-1 risk factor**	< 4.1 mmol/l	> 4.1 mmol/l	>4.9 mmol/l

*Some authorities recommend use of LDL-lowering drugs in this category if an LDL cholesterol < 2.6 mmol/l cannot be achieved by therapeutic lifestyle changes. Others prefer use of drugs that primarily modify triglycerides and HDL, e.g., nicotinic acid or fibrate. Clinical judgment also may call for deferring drug therapy in this subcategory.

**Almost all people with 0-1 risk factor have a 10-year risk <10%, thus 10-year risk assessment in people with 0-1 risk factor is not necessary.

LDL cholesterol < 2.6 mmol/l as the goal of therapy in secondary prevention. This goal is supported by clinical trials with both clinical and angiographic endpoints and by prospective epidemiological studies. The same goal should apply for persons with CHD risk equivalents. When persons are hospitalized for acute coronary syndromes or coronary procedures, lipid measures should be taken on admission or within 24 hours. These values can guide the physician on initiation of LDL-lowering therapy before or at discharge. Adjustment of therapy may be needed after 12 weeks.

For persons with CHD and CHD risk equivalents, the goal is to attain an LDL cholesterol level < 2.6 mmol/l. Most CHD patients will need LDL-lowering drug therapy. Other lipid risk factors may also warrant consideration of drug treatment. Whether or not lipid-modifying drugs are used, non-lipid risk factors require attention and favorable modification. In persons admitted to the hospital for a major coronary event, LDL cholesterol should be measured on admission or within 24 hours. This value can be used for treatment decisions.

In general, persons hospitalized for a coronary event or procedure should be discharged on drug therapy if the LDL cholesterol is > 3.3 mmol/l. If the LDL is 2.6- 3.3 mmol/l, clinical judgment should be used in deciding whether to initiate drug treatment at discharge, recognizing that LDL cholesterol levels begin to decline in the first few hours after an event and are significantly decreased by 24-48 hours and may remain low for many weeks. Thus, the initial LDL cholesterol level obtained in the hospital may be substantially lower than is usual for the patient.

Some authorities hold drug therapy should be initiated whenever a patient hospitalized for a CHD-related illness is found to have an LDL cholesterol > 2.6 mmol/l. Initiation of drug therapy at the time of hospital discharge has two advantages. First, at that time patients are particularly motivated to undertake and adhere to risk-lowering interventions; and second, failure to initiate indicated therapy early is one of the causes of a large "treatment gap," because outpatient follow-up is often less consistent and more fragmented.