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Strategies for the prevention of coronary heart disease: A policy statement of the European Atherosclerosis Society

STUDY GROUP, EUROPEAN ATHEROSCLEROSIS SOCIETY*

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Of the highest priority for preventive medicine in Europe is to achieve a major reduction in coronary heart disease (CHD) and other manifestations of atherosclerosis. To this end a policy based upon reduction of risk factors has been formulated by experts from 19 countries: it employs complementary strategies directed to CHD-prone populations as a whole and to individuals at particular risk. The population strategy includes improved nutrition, avoidance of smoking, blood pressure reduction and promotion of suitable exercise. These should be based upon health education for all age groups and actions by governmental and supranational agencies (including implementation of food labelling, smoking control measures and wide provision of exercise facilities). Only measures directed to the population can reach the large proportion of people at mild to moderate risk of CHD.

To provide care for individuals at particular risk of CHD, case finding requires that risk factor assessment, including measurement of plasma cholesterol and blood pressure, be included in full medical examinations. The relative merits of other modes of case finding, including selective screening and general screening, are discussed. Decisions concerning management of elevated lipid levels should be influenced by overall cardiovascular risk, by a family history of CHD and by age. Lipid lowering dietary and drug therapy should take these variables into account in addition to the extent and type of the hyperlipidaemia. Cholesterol levels exceeding about 5·2 mmol l^{-1} (200 mg dl^{-1}) deserve consideration, and the goal of therapy should be to reduce levels towards this value. For most persons with levels of 5·2–6·5 mmol l^{-1} (200–250 mg dl^{-1}) dietary advice and correction of other risk factors are appropriate, i.e. management comprises reinforcement of population strategy. Dietary recommendations for the control of hyperlipidaemia are reviewed, and indications for drug therapy are presented. Non-pharmacological methods for reducing mildly elevated blood pressure are discussed, and reduction of CHD risk in diabetes is reviewed.

Implementation of these recommendations will be furthered by their endorsement by cardiologists and other physicians.

Introduction

The Study Group recognizes that prevention of atherosclerosis and its clinical manifestations such as coronary heart disease (CHD) are fundamental goals in improving the health of most European countries. CHD in most patients develops on the basis of advanced coronary atherosclerosis. The latter may exist silently for long periods without clinical manifestations. The Study Group recognizes the existence of several factors that determine the risk of CHD, including hyperlipidaemia, smoking, high blood pressure, obesity, diabetes mellitus, physical activity, adverse psychosocial influences and thrombogenic factors. The following report is based on the concordant views of experts from nineteen European countries. It is concerned with strategies for the prevention of CHD by the control of risk factors in populations and in individuals.

Interaction between population strategy and individual strategy for reduction of CHD risk

Two strategies for risk factor reduction have been proposed. The *population* strategy is based on the recognition that in the majority of European countries, most CHD results from exposure of a large proportion of the population to moderately elevated levels of risk factors. Hence this strategy seeks to improve the health-orientated behaviour, i.e. nutrition, smoking and exercise of the whole population. It is directed to reduction of the risk of CHD

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in the entire population. The population strategy has been the subject of reports from the WHO* and national bodies and the Study Group fully endorses these policies.

The *individual or high-risk* strategy seeks to identify by widespread use of clinical and laboratory tests the minority of persons within the population who are at particular risk of CHD; it is directed to high *relative* risk of CHD. The Study Group also fully endorses this policy.

The population and individual strategies are complementary, and there is reason to anticipate that implementation of each may enhance the effectiveness of the other. The two strategies represent different ways of bringing an essentially similar package of preventive measures to the population. Each has its strengths and limitations. Cigarette smoking, overweight and unbalanced diet are so common that ongoing mass education is essential in reaching the large numbers of persons with these characteristics; this educational approach can and should be strongly reinforced in individual clinical practice. Similarly, undesirable or overtly high levels of serum cholesterol are exceedingly prevalent; in many countries more than 50% of young and middle aged persons have cholesterol levels > 200 mg dl^{-1} (5·2 mmol l^{-1}). On the other hand, the minority of the population with pronounced risk factors, e.g. moderate or severe hypertension, major genetic hyperlipidaemias or marked obesity require individual therapy in a clinical setting; for such persons the moderate changes in health habits comprising the population strategy are often inadequate.

The individual ('high-risk') strategy

The availability of effective dietary and pharmacological measures for control of metabolic risk factors, and the substantial evidence of consequent reduction in CHD, imply a commitment to identify persons at high risk with a view to effective individual therapy. This aspect of preventive medicine is an established part of good clinical practice.

Alternative modes of identifying persons at high risk may be distinguished:

(1) *Selective screening* is the targetting of risk factor assessment on persons likely to have positive findings and on those in whom hyperlipidaemia is likely to be particularly hazardous. Selection criteria for such screening include the presence of the following characteristics:

positive family history of cardiovascular disease (CVD) particularly occurring at relatively early age (< 50 years),
family history of hyperlipidaemia,
presence of definite or suspected xanthomas,
presence of xanthelasmas or corneal arcus in persons aged < 40 years,
obesity,
diabetes mellitus,
hypertension,
smoking,
gout.

(1) From the viewpoint of the community, selective screening is not sufficient as a means of reducing the incidence of CHD; a large proportion of persons at high risk would not be examined if assessment, were confined to those having the characteristics listed. However, from the viewpoint of the individual with one or more of these characteristics, risk factor assessment is recommended and positive findings may be anticipated in a substantial proportion.

(2) *Case-finding, opportunistic screening* or the *health check* refers to the sound practice of including risk factor assessment in a full clinical examination e.g. at first attendance, or during employment medical examination (including entry into the armed services) or, when appropiate, during consultation for intercurrent medical problems. Over the course of several years the goal is to recognize most people with levels of risk factors requiring treatment. By doing so in the context of the doctor–patient relationship, compliance with advice and treatment are furthered. Case finding is best conducted at primary health care level and in hospitals, since this affords an opportunity to reach as large a proportion of the population as possible. By extending the process over several years the impact on clinical work-load and laboratory costs is minimized; the introduction of the high-risk strategy will, as is desirable, occur in phase with development of the population strategy.

(3) *General screening* refers to the direct invitation to the public to undergo blood pressure measurement, laboratory tests or other investigations. In some countries such activities are already in operation for selected age-groups, utilizing existing screening facilities and trained primary health care personnel who follow up high-risk persons. In other

*Reference to the following WHO publications is made: *Prevention of coronary heart disease*, Report of a WHO Expert Committee, Technical Report Series 678, 1982; *Primary prevention of coronary heart disease*, Report on a WHO meeting, EURO Report and Studies 98, 1984; *Community prevention and control of cardiovascular diseases*, Report of a WHO Expert Committee, Technical Report Series 732, 1986.

countries its abrupt institution would create the problem of generating a very large case-load, possibly before the medical profession has had time to assimilate the implications of risk factor reduction at the clinical level. Screening should only be carried out if provisions have been made for treatment and follow-up by medical practitioners.

In summary, the case-finding approach, coupled with selective 'screening', provide the optimal means of detecting those at particular risk in most countries at the present time. It should be emphasized that detection of increased risk implies a commitment to provide continuing preventive care. Further, since many risk-related variables tend to increase with age in high incidence populations, it is appropriate to repeat assessment of risk factors especially when borderline values have been noted on initial measurement.

RISK VARIABLES AS CONTINUOUS VARIABLES, AND THE SELECTION OF ACTION LIMITS

The majority of large-scale epidemiological studies show that the relation between most risk factor levels and CHD incidence is continuous and linear or curvilinear, without evidence of a threshold demarcating a boundary between low-risk and high-risk ranges. None of the growing body of knowledge concerning the mechanisms of atherogenesis encourages the view that threshold levels of risk factors are biologically plausible. This absence of 'natural' cut-points, for example for plasma cholesterol level, blood pressure or relative body weight does not interfere with implementation of the population strategy, where the aim is to shift the population distribution. For the high-risk strategy, however, clinical practice is facilitated by provision of practical cut-off points or action-limits. It is all-important to use such action limits flexibly. The use of action limits when the risk factor is a continuous variable is based on somewhat arbitrary choice. In Table 1 therefore, the levels cited as action limits for lipid-lowering therapy, should be regarded only as guidelines; it will be seen that several known clinical, biometric and laboratory findings interact in determining individual risk. Good clinical judgement requires that all of these be taken into account in deciding on a treatment programme for the patient.

The Study Group has considered the options of basing action limits on a selected quantile of the risk factor distribution or of defining absolute levels relevant to all European countries. The interaction between lipoproteins and the arterial wall is presumably a function of their plasma concentration, favouring the selection of absolute levels. The use of quantiles of the distribution, on the other hand, would lead to differences in cut-off points between countries and would serve only to define the proportion of each population deemed to have elevated lipid levels. In this statement, therefore, cut-off points are based upon absolute levels.

Several of the variables appearing in Table 1 require brief comment:

(1) *Age*. As the atherosclerotic process commences early in life its prevention requires detection and management of risk factors at as early an age as possible. Conversely, some forms of hyperlipidaemia, e.g. familial combined hyperlipidaemia, type III hyperlipidaemia and familial hypertriglyceridaemia, are not expressed until early adult life, hence assessment in adolescence or in the third decade may be falsely reassuring.

The intensity of therapeutic measures should be greatest in younger persons. However, life-style counselling may have stronger motivating power when an individual comes closer to the age at which CHD prevalence becomes high and is perceived as a health risk. In any case it would be arbitrary to define an upper age beyond which treatement should be withheld. An intrinsically important role of the population strategy, for younger persons, is to prevent the rise of risk-related variables with age (e.g. blood pressure, serum cholesterol) and to prevent the adoption of deleterious habits such as smoking. When risk factors are detected for the first time in older persons, less-intensive treatment may be appropriate. In older patients, the target of treatment, be it body weight, blood pressure or plasma lipid level, may be less exacting.

(2) *Sex*. At all ages, but especially in young persons, the risk of CHD is higher in males than in females. This too may influence the choice and target of therapy. However, the potential effect of certain oral contraceptive preparations on CVD risk in young women, especially in cigarette smokers, must be borne in mind.

(3) CHD tends to aggregate in families. A *positive family history* of CHD justifies more vigorous attention to risk factors, particularly if family members were affected at an early age and/or if multiple relatives were affected.

(4) The multifactorial nature of CHD indicates that *all modifiable risk factors should receive attention*. In using the action-limits for lipid levels given in Table 1, the entry 'smoking' implies that the hyperlipidaemia requires particularly careful treatment; it is also

Table 1 Guidelines for management of hyperlipidaemia

A	Cholesterol 200–250 mg dl^{-1} Triglyceride < 200 mg dl^{-1}	Assess overall risk of CHD, taking into account family history of CHD, hypertension, diabetes, male sex, younger age, smoking, low HDL cholesterol e.g. < 35 mg dl^{-1}	Restrict food energy if overweight; give nutritional advice and correct other risk factors if present (see Section on 'Components of the population strategy')
B	Cholesterol 250–300 mg dl^{-1} Triglyceride < 200 mg dl^{-1}	Assess overall risk of CHD as under A	Restrict food energy if overweight; prescribe lipid lowering diet and monitor response and compliance. If cholesterol remains high, consider use of lipid-lowering drug.
C	Cholesterol < 200 mg dl^{-1} Triglyceride 200–500 mg dl^{-1}	Seek underlying causes of hypertriglyceridaemia, e.g. obesity, excessive alcohol intake, diuretics, beta blockers, exogenous oestrogens, diabetes	Restrict dietary energy if overweight; deal with underlying causes if present. Prescribe and monitor lipid-lowering diet. Monitor cholesterol and triglyceride levels.
D	Cholesterol 200–300 mg dl^{-1} Triglyceride 200–500 mg dl^{-1}	Assess overall risk of CHD as in A. Seek underlying causes of hypertriglyceridaemia as in C.	Restrict dietary energy if overweight; deal with underlying causes of hypertriglyceridaemia if present and proceed according to A or B. Prescribe and monitor lipid-lowering diet. If serum lipid response is inadequate and overall CHD risk is high, consider use of lipid-lowering drug.
E	Cholesterol > 300 mg/dl and/or Triglyceride > 500 mg/dl		Consider referral to lipid clinic or to specialized physician for investigation and initiation of treatment by diet and if necessary, drugs.

Cholesterol: 200 mg dl^{-1} = 5·2 mmol l^{-1}, 250 mg dl^{-1} = 6·5 mmol l^{-1}, 300 mg dl^{-1} = 7·8 mmol l^{-1}.
HDL cholesterol: 35 mg dl^{-1} = 0·9 mmol l^{-1}.
Triglyceride: 200 mg dl^{-1} = 2·3 mmol l^{-1}, 500 mg dl^{-1} = 5·6 mmol l^{-1}.

necessary that the smoking habit should receive equal attention to the hyperlipidaemia.

(5) Similarly, the patient with *hypertension*, and the persisting cigarette smoker, require not only treatment and counselling for these conditions but careful attention to their plasma lipids, in view of their additive influence on CHD risk. During drug therapy for hypertension lipid levels should be monitored regularly as chlorthalidone, thiazides and some beta-blockers may tend to elevate plasma lipids and to depress HDL cholesterol. If such drugs are employed, these changes may be minimized by diet, and serial measurement of lipid levels is advised.

(6) Independently of other risk factors, *diabetes mellitus* confers a two-fold increase in the risk of CHD; hence hyperlipidaemia, smoking, and hypertension should be sought and assiduously treated in diabetics (see below).

(7) *Overweight* (body mass index* > 25) confers an increased risk of CHD, and more pronounced obesity is a risk factor independent of other risk-related variables. The control of overweight of all degrees is justified in its own right; in addition, weight reduction is important in ameliorating abnormal lipid and lipoprotein levels, hypertension, hyperuricaemia and non-insulin dependent diabetes.

LIPIDS AND LIPOPROTEINS IN RELATION TO CHD RISK

Cholesterol

The relation between plasma cholesterol level and CHD risk is strong and consistent in a large number of studies and is independent of other risk factors. Genetic, experimental, epidemiologic, and clinical trial evidence is concordant in indicating that elevated cholesterol levels due to high levels of LDL-cholesterol play a causal role in atherosclerotic heart disease. It has been further established that lowering elevated plasma cholesterol levels will reduce the risk of heart attack in men with levels in the upper part of the distribution seen in most European countries. From this observation, together with much evidence from other sources on the causal link between abnormal lipid levels and atherosclerosis, it is justifiable to extend the recommendation to reduce plasma cholesterol, or to maintain low levels throughout life. The accurate measurement of cholesterol level is a mandatory part of CHD risk assessment, and cholesterol levels exceeding about 200 mg dl^{-1} (5·2 mmol l^{-1}) deserve consideration, particularly in young adults. As discussed in the previous section, risk assessment and preventive measures are determined by a person's overall risk and are influenced by age and other variables. It is important to stress that for the majority of adults with cholesterol levels in the range 200–250 mg dl^{-1} (5·2–6·5 mmol l^{-1}) the appropriate management will include nutritional counselling, advice against smoking, guidance concerning weight control and physical exercise, i.e. it comprises reinforcement of population strategy.

A major reason for selecting the value of 200 mg dl^{-1} is that it is concordant with the longitudinal study on 325 000 white men in the Multiple Risk Factor Intervention Trial Screening Programme. In this study there was an increase in CHD mortality from the lowest quintile to the highest. Thus mortality was least in men in the lowest quintile, who had serum cholesterol levels < 182 mg dl^{-1} (4·7 mmol l^{-1}). The value of 200 mg dl^{-1} is also compatible with the data from the Seven Countries longitudinal study by Keys in which, similarly, CHD increased from the lowest part of the distribution of serum cholesterol. While it is true that the relation between cholesterol level and CHD risk varied in some other studies, these two investigations are uniquely powerful by virtue of very large sample size. They provide no evidence of a threshold level of serum cholestrol within the range seen in high incidence populations.

The Study Group concurs with the statement made by the NIH Consensus Development Conference 'Lowering Blood Cholesterol to Prevent Heart Disease' that 'it is a goal to encourage reduction of the blood cholesterol to approximately 180 mg dl^{-1} for adults under the age of 30 years and to approximately 200 mg dl^{-1} for individuals aged 30 or older. This is recognized as a realistic 'target' level that should be possible to achieve . . .'*.

It is the Study Group's view that the varying distributions of lipoproteins in different countries do not influence their biological significance. It was felt that it would not be meaningful to regard a cholesterol level of for example 225 mg dl^{-1} as normal in one country and pathological in another.

The action limit mentioned would, because of the distribution of cholesterol levels in some countries, define a majority of the population as having undesirable levels. There is nothing intrinsically erroneous about such a consequence. As is stated in

*Body mass index = weight/height^2, where weight is in kg and height in m.

*NIH Consensus Development Panel. Lowering blood cholesterol to prevent heart disease. *JAMA* 1985; 253: 2080–6.

the section below on 'Management of hyperlipidaemia', the role of the physician in dealing with persons with cholesterol levels in the range 200–250 mg dl^{-1} is most often confined to reaffirming the population strategy.

Triglyceride

Many patients with CHD have high triglyceride levels. However, unlike high cholesterol levels, elevated triglyceride has yet to be shown to be a cause of atherosclerosis. In some patients hypertriglyceridaemia indicated the presence of lipoprotein abnormalities that may be related to atherosclerosis. There is reason to suspect that among the triglyceride-rich lipoproteins certain subclasses (remnant particles) play a causal role in atherogenesis. In the absence of controlled trial evidence the levels shown in Table 1 are meant as provisional guidelines. When hypertriglyceridaemia can be shown to be a manifestation of type III (remnant) hyperlipoproteinaemia or of familial combined hyperlipidaemia, both of which are strongly associated with CHD, effective therapy is clearly indicated. When triglyceride exceeds 1000 mg dl^{-1} (11 mmol l^{-1}) there is a risk of pancreatitis; vigorous therapy is needed to lessen this risk.

Overall, the available data did not permit the Study Group to make firm recommendations concerning triglyceride measurements in risk assessment, nor concerning therapy of elevated triglyceride levels. However, the extensive Stockholm Prospective Survey and some of the smaller longitudinal studies have suggested that triglyceride level is related to CHD risk, independent of serum cholesterol. It must be emphasized that many of the causes of hypertriglyceridaemia themselves merit therapy irrespective of associated lipid abnormalities, for example obesity, alcohol abuse and uncontrolled diabetes. Current practice is that triglyceride levels must be measured in the fully-fasting state, which may necessitate a further attendance at the surgery or clinic. Triglyceride levels show greater intraperson variability than cholesterol or HDL-cholesterol levels.

HDL-cholesterol

In many, but not all epidemiological studies, levels of HDL-cholesterol showed a strong inverse relation with CHD risk. For reasons not fully understood, low HDL-cholesterol concentrations in many individuals may indicate an increased risk for heart attack. It has been suggested that HDL or a small subclass within HDL plays an important role in initiating the reverse transport of cholesterol, i.e. in mobilizing cholesterol from tissues.

Measurement of HDL cholesterol will identify persons in whom mildly-elevated blood cholesterol is due to high levels of HDL cholesterol. Low HDL cholesterol levels are often associated with lack of exercise, obesity, cigarette smoking and hypertriglyceridaemia. These conditions require correction in their own right, by appropriate counselling.

In the view of some authorities the level of HDL cholesterol may be taken into account in deciding upon the intensity with which hyperlipidaemia should be treated. Interpretation of HDL-cholesterol levels is limited by constraints similar to those referred to above in the section on triglyceride levels. Furthermore evidence is lacking as yet that raising HDL-cholesterol levels will reduce the risk of CHD in man. The size of the tissue pools of cholesterol is not independently related to HDL cholesterol levels. The cut-off point of 35 mg dl^{-1} (0·9 mmol l^{-1}) (Table 1) should be regarded as a provisional guideline. Men have lower HDL-cholesterol than women.

In summary, cholesterol measurement in a random blood sample is mandatory to assessment of CHD risk. However, depending upon possible geographical variation in the relative importance of certain risk factors, local variations in practice are likely. Financial considerations will also influence the extent of investigations. In some centres it is considered preferable to measure cholesterol, triglyceride and HDL-cholesterol. If triglyceride is included the individual must attend in the fasted state.

As Table 1 indicates, lipoprotein and other risk factors must be considered together when deciding upon management of the patient. Subjects in group A have moderately increased lipoprotein-associated risk. By contrast, persons in group E are at high risk for CHD and it is often advisable that they be referred to a specialized physician for investigation and initiation of treatment. Persons belonging to groups B to E should be followed up, and regular lipid measurements made to assess the effectiveness of treatment.

MANAGEMENT OF HYPERLIPIDAEMIA

The initial clinical approach includes the detection and treatment of underlying causes. Hyperlipidaemia secondary to hypothyroidism or excessive alcohol use is remarkably frequent, and several other causes are well known, including commonly

used drugs. Most often clinical features identify such disorders, but selective use of laboratory tests may be necessary. Dietary management is necessary for all primary hyperlipidaemias and suffices as the sole therapy for the majority of persons with elevated levels. For those with serum cholesterol levels in the range 200–250 mg dl^{-1} the role of the clinician is, usually, to reaffirm and endorse the measures comprising the population strategy (see the section on 'Components of the population strategy' below. In a minority judged to be at particular risk of CHD or of progression of existing CHD, however, more active intervention may be appropriate. The dietary measures are qualitatively the same as those referred to in the section on 'Nutrition' below, but may be modified according to the serum lipid response. For patients with severe hypertriglyceridaemia (chylomicronaemia syndrome or familial hypertriglyceridaemia) emphasis is placed on reduction in total fat intake. Written dietary instructions are a necessary but not a sufficient part of nutritional treatment. A dietitian, supported by the patient's physician, is best able to instruct the patient, adapt the diet to energy needs and food preferences, and (if response is inadequate) to monitor ongoing compliance.

In two situations in particular, drug therapy should be considered in addition to diet for patients deemed to be at high risk of CHD on overall assessment. One is the presence of a major genetic hyperlipidaemic state, usually familial hypercholesterolaemia, or type III (remnant) hyperlipoproteinaemia. The other situation in which drug therapy should be considered is exemplified by the patient with moderate hyperlipidaemia, judged to be at high risk of CHD, in whom assiduous attempts at dietary control fail to achieve acceptable levels of serum lipids. With experience it is usually possible to decide upon the need for drug therapy initially. Where there is uncertainty it is strongly recommended that introduction of drug therapy be deferred pending the outcome of effective dietary counselling. Monotherapy is preferred, but resistant familial hypercholesterolaemia not infrequently requires treatment with two drugs possessing different modes of action. The goal of therapy should be to minimize lipoprotein-mediated risk. The constraints are twofold: the effectiveness of treatment and the physician's clinical judgement of the balance between benefit and risk. As treatment of hyperlipidaemia is a long-term commitment, careful diagnosis and informed judgement as to the risk–benefit ratio of therapy are essential preconditions.

DIAGNOSTIC LABORATORY PROCEDURES

The criteria for laboratory assessment of risk factors include relevance, accuracy and precision, and also cost and speed of analysis. Considerable progress has been made toward these goals in the past decade. Meticulous quality control is necessary, not least because patients may remain under observation for decades. An advance of great potential has been the development of dry chemistry techniques permitting satisfactory measurement of cholesterol, triglyceride and glucose on capillary blood samples. These procedures permit very rapid analysis at low capital and recurrent cost, and are suitable for the clinic or general practice. They promise to facilitate both case finding and the monitoring of therapy of hyperlipidaemia, goals already achieved in diabetic care.

Serum lipids and lipoproteins are usually measured in the fasted state. In the follow-up of pure hypercholesterolaemia fasting is unnecessary. Pronounced hyperlipidaemia or dyslipoproteinaemia must be characterized by at least two measurements to facilitate proper diagnosis and to establish a clear baseline before choosing and initiating treatment.

DIABETES MELLITUS

Although diabetes mellitus is recognized as an independent CHD risk factor, the mechanisms underlying the association between hyperglycaemia and atherosclerotic heart disease are incompletely understood. In particular it is not clear, as yet, whether a causal link exists between abnormal blood glucose concentration and CHD. However, it is well documented that, by improving blood glucose control, plasma cholesterol and triglyceride decrease and the level of HDL increases. In addition it is important to reduce CHD risk in diabetic patients by controlling other risk factors often associated with diabetes such as hyperlipidaemia, hypertension, smoking and obesity.

Recently the diabetic diet has been reconsidered and more emphasis has been given to reduction of saturated fat intake, which is replaced by complex carbohydrate. Fruit, vegetables and particularly legumes should be recommended since these are also fibre-rich. A high complex carbohydrate, high fibre diet is effective in reducing plasma cholesterol levels as well as blood glucose concentration.

MANAGEMENT OF SMOKING IN HIGH RISK PERSONS

Particular efforts should be made to help persons at high risk to quit smoking. The basic principles

are those outlined in the section on 'smoking' below; in addition, intense individual counselling should be offered. Referral to a smoking cessation clinic may prove useful, if available.

Components of the population strategy

In this section the scope of measures for control of cigarette smoking, and for promotion of healthy eating habits and exercise habits are outlined. Necessary though these measures are, it must be recognized that nutritional change in particular will require years for its full implementation. This time course will facilitate adoption of the criteria for optimal nutrition by the food producing and distributive industries.

NUTRITION

The Study Group adopted the following recommendations for the general population and for high risk individuals:

(a) control of overweight by decreasing energy intake and by exercise suitable for the age and cardiorespiratory fitness of the individual;

(b) reduction of total fat intake to 30% or less of total dietary energy;

(c) reduction of the intake of saturated fatty acids (contained in foods of animal origin, hydrogenated oils and certain vegetable products) to less than 10% of total dietary energy;

(d) reduction of dietary cholesterol to less than 300 mg day^{-1};

(e) increased consumption of complex carbohydrates;

(f) encouragement of the use of oleic acid (a cis-monounsaturated fatty acid) and of linoleic acid (an all-cis-polyunsaturated fatty acid);

(g) increased intake of fruit, vegetable and cereal-fibre, with some emphasis on legumes;

(h) moderation in salt intake.

Implementation of these nutritional recommendations is a central component of the strategy for reduction of CHD and is a priority within preventive medicine at the present time. The gradual, progressive achievement of this nutritional policy will permit congruent changes in patterns of food consumption and of production and marketing.

The Study Group recommends a strategy involving the following components, requiring determined application through involvement of the medical and allied professions, government, health and education agencies, the food production and distribution industries and local self-help groups:

(1) Nutritional education at all levels of schooling, to provide adequate understanding of healthy eating habits and of the content of major nutrients in foods.

(2) Adult education through publications by the European Atherosclerosis Society, government agencies, health education councils, and the media. Establishment of lay self-help groups (with medical and nutritional backing) through local authorities, hospitals, adult education institutions and churches. Provision of understanding of healthy eating, of food composition, of informed purchasing of foods and of appropriate recipes, menus and cooking techniques.

(3) Statutory requirements for simple, informative food labelling.

(4) Availability of low saturated fat, high complex carbohydrate, high fibre options in institutional catering and restaurants.

(5) Constructive interaction with the food industry to promote mutual understanding of goals and constraints.

(6) Interactions with government and supranational bodies to encourage health-oriented use of food policies, including subsidies and tariffs.

NON-PHARMACOLOGICAL TREATMENT OF HYPERTENSION

While drug treatment of hypertension is beyond the scope of these recommendations, it is relevant to summarize the Study Group's position on non-pharmacological approaches. These are relevant both to population strategy and to individual care. Even a small reduction in mean blood pressure in the population may have a substantial effect on the risk of CVD. Many principles are common to the management of hyperlipidaemia and hypertension. The major demonstrable benefits in trials of drug treatment of hypertension have been reduction of the frequency of stroke, cardiac failure and renal disease.

(1) Reduction of overweight and obesity in the mildly hypertensive patient commonly lessens systolic and diastolic pressure and is an essential component of management.

(2) Excessive use of alcohol is strikingly correlated with elevated blood pressure and with stroke mortality. In some patients abstention from alcohol or restraint in its use results in substantial reduction of blood pressure.

(3) The results of trials of moderate restriction of dietary sodium on blood pressure have not been

entirely consistent, though it is likely that a significant reduction in blood pressure is more regularly achieved in persons with elevated pressures than in normotensives. There is no evidence of adverse effects of moderate salt restriction on the majority of people. There is limited evidence that blood pressure reduction is more effectively achieved by sodium restriction accompanied by a high potassium intake. Potassium supplementation is particularly relevant when patients are also receiving potassium losing diuretics, and when compliance with sodium restriction is poor. The high content of fruit and vegetables implicit in the dietary recommendations listed in the section on 'Nutrition' above result in a substantial potassium intake.

(4) There is limited evidence that regular physical exercise has a small antihypertensive effect. The several potential health benefits of appropriate regular exercise justify its inclusion in comprehensive recommendations.

SMOKING

Prevention and cessation

Cigarette smokers are twice as likely as non-smokers to die before the age of 65 years. They are twice as often in hospital or off work because of illness. Cigarette smoking is the most important known cause of respiratory disease and cancer, and is a major risk factor for coronary heart disease, stroke, peripheral vascular disease and aneurysym, particularly in populations with high mean cholesterol levels.

Current trends in smoking

There is wide variation in smoking habits and in smoking trends in European countries. The number of male smokers is falling, largely due to increased cessation of smoking. The level of smoking among women is stabilizing and may soon follow the male pattern. However, there is in most countries an increase in smoking among young males and females, a trend which can be attributed to the powerful advertising influence of the tobacco companies.

Better educated people are stopping in larger numbers and fewer are beginning to smoke. It is hoped that their example will eventually extend to other educational classes.

There is increasing social and political pressure against smoking in public places, and an increasing awareness of the unpleasant and adverse health effects of passive smoking.

The community control of smoking

Governments should adopt the following measures:

1. Progressive increases in tobacco taxation.
2. Suppression of advertising, sponsorship and indirect advertising by the tobacco companies.
3. Support of public education programmes.
4. Control of smoking in hospitals, schools, public offices, places of entertainment, restaurants and other public places; where necessary, demarcation of separate smoking areas.
5. Encouragement of tobacco companies to diversify.
6. In tobacco producing countries, withdrawal of subsidies for tobacco production; encouragement of alternative crops by financial and other means.
7. Influencing of national and especially supranational bodies to adopt a more active policy against smoking, an approach which now requires greater urgency because of recent accession to the EEC of the tobacco growing countries Greece, Spain and Portugal.

Public education

The public must be informed about the adverse health consequences of smoking. This requires increased investment of funds in public education programmes. Television, newspapers, magazines, books and posters have a place. Government-sponsored and voluntary health agencies can both contribute. Following the abolition of tobacco advertising, newspapers and magazines will be more able to stress the health consequences of smoking.

Education of doctors and other health professionals

Firm advice from a doctor or other well informed health professional is more effective in inducing people to stop smoking than any other technique available. Smoking as a health hazard should receive more prominence in undergraduate and postgraduate medical education. All doctors should routinely proffer well informed advice on the subject.

Doctors, like teachers and other members of the caring professions, should be encouraged not to smoke, at least in public. They should be fully informed about effective techniques to induce their patients to stop smoking. Professional medical organizations should lobby government about the necessity of smoking control.

Social pressures against smoking

The increasing unpopularity of smoking in public

places stems from the growing concern expressed by the public and by a large number of civic and voluntary organizations and pressure groups. An increase of such social pressures should be encouraged by a variety of appropriate public actions, including lobbying of public representatives, and out-spoken criticism of promotional activities. Parents, caring professions and organizations should be more aggressive about the influence of tobacco advertising and sponsorship on young people. Opinion leaders in clubs and sports organizations should be seen to oppose tobacco sponsorship.

Control of smoking in industry and in the workplace

The advantages of a non-smoking policy on the factory floor and in the workplace should be stressed in industry and commerce. Improved productivity, less absenteeism and reduced cleaning and replacement costs can be added to the benefits of better health among the workers. A non-smoking policy in the workplace has been adopted with success by a number of firms, particularly in the United States.

Schools

Our biggest challenge is to prevent the smoking habit. This will have the greatest impact on community control of smoking. It can best be achieved in schools and in other institutions where young people congregate. Children can be very effective in influencing parental smoking if the children are well informed about the hazards of smoking.

PHYSICAL ACTIVITY

Most epidemiological studies, though not all, have shown an inverse relation between habitual aerobic exercise and CHD mortality. Data from controlled trials is sparse, and is rendered inconclusive by relatively weak statistical power and high drop-out rates. In addition to the probable protective effect agains CHD, regular exercise may have other objective health advantages, including reduction of obesity, lessened risk of osteoporosis with advancing age and enhanced cardio-respiratory and musculoskeletal fitness. Subjectively, there is improved well-being and physically active persons commonly report lessening of tension.

Proof of a causal role of exercise in reducing the risk of CHD is incomplete, and it has not been entirely excluded that self-selection may account in part for the inverse relation described. Nevertheless, the Study Group regards the evidence of a favourable effect as being sufficient to justify the recommendation of regular, frequent, appropriate aerobic exercise as part of healthy life style.

Exercise is not without hazard. CHD events, including sudden death, are more common during and immediately after exercise than at rest. Overall, however, sudden cardiac death is substantially less common in active people than in sedentary people.

The amount and type of exercise must, therefore, take into account each individual's pre-existing fitness, age, cardio-respiratory health and the presence of musculoskeletal problems. Young fit persons should be encouraged to maintain fitness with advancing age. Unfit persons should carry out self-assessment. For this purpose simple questionnaires are available that take into account cardiovascular symptoms, family history, presence of evident risk factors and current fitness; depending on the result they may proceed with a slowly progressive programme of exercise, allowing at least six months to achieve fitness. Evidence from the questionnaire of increased cardiovascular risk should prompt the individual to seek medical assessment before undertaking more than mild exercise.

In assessment of middle-aged persons before undertaking substantial exercise, the presence of CHD risk factors is one helpful guideline. In addition most physicians use exercise stress electrocardiography in such assessment. However, it must be emphasized that the sensitivity and specificity of the exercise test in asymptomatic persons are unsatisfactory.

Adequate regular activity should be the norm from early life. It should be avoided during even mild intercurrent illness. The extent is best determined by informed subjective criteria. Some experts advise monitoring pulse rate during exercise, aiming at 60% of maximum heart rate during acquisition of fitness and at 70–75% of maximum heart rate for maintenance.

The distinction between aerobic and non-aerobic exercise should more widely recognized. The former, on which cardiovascular fitness largely depends, consists of a large number of repetitions of movements against relatively low resistance. Suitable examples are brisk walking, jogging, running, swimming, cycling, cross-country skiing, calisthenics and vigorous dancing. Ball games are also suitable, with the provision that the intensity and duration of exercise should be limited by the subjective criteria mentioned and not by competitiveness.

To encourage the widespread adoption of regular exercise, it is recommended that local authorities and central government provide suitable facilities

widely. Examples are the provision of cycle paths in urban areas, reservation of thoroughfares as pedestrian precincts, the availability of school sports facilities to adults when not in use by schoolchildren and development of forest pathways and other rural exercise facilities.

ALCOHOL AND CHD RISK

The evidence relating alcohol intake to CHD is complex. This is mainly due to concomitant abuses which accompany alcohol intake and which often vary between different cultural and ethnic groups.

The Study Group recognize that, in addition to damage to the liver, excessive alcohol intake may directly damage the myocardium causing conduction defects and sudden death. Furthermore, several studies have shown that chronic alcohol use increases the risk of hypertension, stroke and certain cancers. In some obese persons alcohol is a major source of dietary energy. Thiamine deficiency heart disease is often associated with alcohol abuse.

Heavy intake of alcohol, for the reasons shown and because of its deleterious effects on personal and social behaviour, must be strongly discouraged. There is no evidence that moderate alcohol intake (not more than 25–30 g per day) increases the risk of coronary atherosclerosis and CHD. On the other hand people who tend to develop hypertriglyceridaemia on chronic exposure to alcohol must be warned about the possible risks.

The role of cardiologists and other physicians

Cardiologists and other physicians have a leadership role in promoting policies to prevent and reduce CHD. A more positive attitude of cardiologists to the prevention of CHD will greatly further this goal.

Physicians, and cardiologists in particular, should influence those in training. It is remarkable how little attention is currently given by consultants to detailed aspects of coronary prevention when instructing registrars, senior house officers and house physicians. Examinations are a poor way of ensuring training in prevention. This training should start at undergraduate level, but many undergraduate courses provide little time for such instruction. Thus physicians who are responsible for teaching at under- or post-graduate level should specifically identify to those in training their responsibility for promulgating CHD prevention.

The implementation of these recommendations will be enhanced by their endorsement by physicians and especially by cardiologists.

Research needs

There are several important emergent fields for research into the understanding of the pathogenesis of CHD. These include:

(1) Identification of the relative risk of CHD associated with abnormalities of the coagulation–thrombosis system. Particular attention is needed to confirm and quantify the predictive values of raised fibrinogen, factor VII and factor VIII, and newer measures of platelet adhesiveness and blood viscosity. More information is needed concerning the inter-relationship between lipid abnormalities and these thrombogenic factors. The pathophysiology of fibrinogen and fibrinolysis also requires additional study in man.

(2) The relation between specific dietary, plasma and tissue fatty acids and CHD needs to be examined in greater depth. Low dietary and tissue concentrations of the essential fatty acid, linoleic acid, is associated with a high incidence of CHD; while this relationship is partly confounded by cigarette smoking, it is an independent risk factor for CHD. The possibility that high consumption of olive oil may have a favourable effect on lipoprotein concentrations and on CHD requires further exploration. A high consumption of marine oils, particularly eicosopentaenoic acid, may reduce the risk of thrombogenesis and its effects on the incidence of CHD requires formal study.

(3) The development of 'genetic probes' needs more support. Use of monoclonal antibodies to identify susceptible individuals on the basis of receptor abnormalities is one need. Another is to identify genetic influences that protect against the development of atherosclerosis and CHD and that appear to operate favourably in long-lived families.

(4) Improvement of techniques for imaging atheromatous plaques is an urgent need. The development of magnetic resonance to permit precise measurement of coronary atheroma in relation to regression studies is one area for development. This will facilitate study of the rate of progression and regression of coronary lesions and the influence of risk factor reduction of these processes.

(5) The mechanisms through which cigarette smoking lead to CHD have still to be elucidated. More accurate identification of constituents of smoke that influence endothelial function, thrombogenesis,

sympathetic activity and myocardial metabolism are required.

(6) Further information is required as to the optimal dietary distribution of saturated fatty acids, cis-monounsaturated fatty acids and essential polyunsaturated fatty acids of the ω-6 and ω-3 series required to minimize CHD risk. Such research should take into account their effects on plasma lipoproteins, and on platelet function and blood pressure.

(7) The significance of hypertriglyceridaemia for the processes of atherogenesis requires further evaluation at the levels of epidemiology, arterial wall metabolism of triglyceride-rich lipoproteins and their metabolic products, and interaction with thrombogenic mechanisms.

(8) The apparent association between low levels of HDL cholesterol and CHD requires fuller evaluation. It should be established whether serum levels of HDL and/or of its subclasses or components are rate limiting to reverse cholesterol transport. Further studies are required of its suggested relationship to tissue cholesterol pools. These may help establish whether epidemiological associations between HDL and CHD represent a causal relation.

(9) More research is needed in the area of psychological and sociopsychological traits and behavioural characteristics as possible risk factors for CHD.

Footnote: This article was written under the responsibility of the signatories and does not necessarily reflect the official views of the European Society of Cardiology.

Appendix

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