by Dr Matti Tolonen and Dr Pentti Raaste

GUIDELINES FOR GOOD SELF-MANAGEMENT OF CARDIOVASCULAR DISEASES

INTRODUCTION
"THE EMPEROR'S NEW CLOTHES" DISCLOSED

"The Emperor's New Clothes" (Danish: Kejserens nye Klæder) is a short tale by Hans Christian Andersen about two weavers who promise an Emperor a new suit of clothes invisible to those unfit for their positions or incompetent. When the Emperor parades before his subjects in his new clothes, a child cries out, "But he isn't wearing anything at all!"

The diet-cholesterol-heart theory is "The Emperor's New Clothes". As a matter of fact, there is no scientific evidence for a conclusion that dietary saturated fat is associated with an increased risk of coronary heart disease or other cardiovascular diseases. Neither is there conclusive evidence that cardiovascular diseases are caused by cholesterol. Thus, cholesterol-lowering drugs target neither the origin nor the mechanisms of the disease. We believe that the origin may be infective and related to deficiency of omega-3 fatty acids, vitamins and other protective micronutrients, and that the mechanisms of the disease are chronic low-grade inflammation and oxidative stress, which should both be suppressed. To this end we recommend regular exercise, at least 2,5 hours per week, and a healthy diet which provides plentiful antioxidants. Modest reductions in dietary salt could substantially reduce cardiovascular events and medical costs and should be a public health target. We also recommend dietary supplements, such as fish oil, berberine, carnosine, vitamins D, folic acid along with other vitamins and minerals.

INDEX OF TERMS

AA: arachidonic acid, an omega-6 fatty acid, involved in cellular signalling as a lipid second messenger. AA in the human body usually comes from dietary animal sources, meat, eggs, dairy, or is synthesized from linoleic acid, another omega-6 fatty acid.

AGEs: advanced glycation endproducts, the result of a chain of chemical reactions after an initial glycation reaction. AGEs are known to play a role as pro-inflammatory mediators, e.g. in diabetes.

ALA: alpha linolenic acid, an omega-3 fatty acid found in many common vegetable oils. It is biologically inferior to EPA and DHA, found in fish oil.

Angina pectoris: commonly known as angina, is severe chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle generally due to obstruction or spasm of the coronary arteries (the heart's blood vessels).

Antioxidant: a molecule capable of slowing or preventing the oxidation of other molecules. Oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent. Oxidation reactions can produce free radicals, which start chain reactions (cascades) that damage cells. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions by being oxidized themselves.

Atherosclerosis, arteriosclerosis: commonly referred to as a hardening or furring of the arteries. It is caused by the formation of multiple plaques within the arteries in response to a chronic inflammatory process in the walls of arteries. The origin of the syndrome remains unknown.

CHD: coronary heart disease, caused by atheromatous plaques within the walls of the coronary arteries that supply the myocardium (the muscle of the heart) with oxygen and nutrients.

Coronary arteries: blood vessels supplying blood to the heart muscle.

Claudication: literally "limping" (Latin) indicates impairment in walking.

Sclerosis: a hardening of tissue and other anatomical features.

DHA: docosahexaenoic acid, an omega-3 fatty acid in fish oil.

E-EPA: the ethyl ester of eicosapentaenoic acid (EPA), an omega-3 fatty acid in fish oil.

EPA: eicosapentaenoic acid, an omega-3 fatty acid in fish oil. EPA and E-EPA are anti-inflammatory and antidepressive fatty acids.

Free radicals: atoms, molecules, or ions with unpaired electrons on an open shell configuration. The unpaired electrons cause them to be highly chemically reactive. See oxidative stress.

Homocysteine: a toxic amino acid, which the body makes from proteins in meats. Deficiencies of the vitamins folic acid, B6, or B12 can lead to high homocysteine levels in blood and tissues.

Inotrope: an agent that alters the force or energy of muscular contractions. Positively inotropic agents, like digitalis and berberine, increase the strength of muscular contraction.

Interleukins (IL-1–20): a group of cytokines (secreted proteins/signaling molecules) that are used extensively in cellular communication. IL-1, IL-6, IL-8 and TNF-a. are pro-inflammatory and IL-4 and IL-10 are anti-inflammatory cytokines.

Mitochondria: cellular "cellular power plants" which generate most of the cell's supply of adenosine triphosphate (ATP).

MPO: myeloperoxidase, a protein secreted by white blood cells. A risk indicator of CHD.

Myocardial infarction, heart attack.

NO: nitric oxide, a gas is an important signaling molecule in the body. Keeps the arteries flexible.

Oxidative stress: caused by an imbalance between the production of reactive oxygen and a biological system's ability to readily detoxify the reactive intermediates or easily repair the resulting damage.

Pathogenesis: step by step development of a disease and the chain of events leading to that disease, due to a series of changes in the structure and /or function of a cell/tissue/organ being caused by a microbial, chemical or physical agent.

PLA2s, Phospholipases A2: enzymes that release fatty acids from the second carbon group of glycerol. Genetically overactive PLA2s promote and maintain inflammation in the endothelium and lead to endothelial dysfunction. E-EPA counter- fights PLA2s.

RLP-C: remnant-like particles of cholesterol, an indicator of CHD.

Statins: a class of popular drugs that lower cholesterol levels in people.

Telomeres: "age clocks" located at the end of chromosomes. Their fast shortening speeds up cell death and cuts peoples life expectancy. Increased dietary intake of marine omega-3 fatty acids is associated with prolonged survival in patients with coronary heart disease.

HEART AND ARTERY (CARDIOVASCULAR) DISEASES

Cardiovascular diseases are common in Western populations: every third man, and every tenth woman has or will be diagnosed with a clinical cardiovascular disease. Moreover, as we age, almost everyone's arteries will become more or less stiff and narrower (sclerotic), thus impairing the blood circulation. The most common causes of death in industrialised nations are heart attacks and strokes. Cardiovascular diseases kill 80% of diabetics and 70% of all those aged over 75.

The origins of arterial diseases are obscure. It is known, however, that inflammation and calcification (not cholesterol, as commonly thought) are the main mechanisms in the narrowing and hardening of arteries. In the heart, impaired circulation may lead to heart burn (angina pectoris), and a total occlusion of a coronary artery will cause a myocardial infarction (heart attack). A reduced blood flow in the brain can cause a stroke.

Deaths from cardiovascular diseases are largely due to coronary heart disease (CHD) and stroke, but in addition, the patient's other arteries usually also harden and narrow, resulting in circulatory problems, especially in the lower extremities. In this case a symptom may be intermittent claudication. Capillary sclerosis of the retina of the eye also indicates CHD.

Our aim with this guide is to give easy-to-follow instructions, in order to reduce the risk of cardiovascular diseases, or potentially to slow down an alteration, which is already in progress. Simply lowering cholesterol readings is not enough. We hope to highlight the importance of prevention and non-medicated self-treatment. We also believe that the vast majority of cholesterol-lowering drugs would become unnecessary if people would eat a low-carb diet, exercise, take enough antioxidants and achieve an ideal ratio between omega-6 and omega-3 fatty acids in their blood. We are convinced that if you follow our guidelines you may need less or no medication and avoid its unwanted side-effects.

The mystifying cause of arterial diseases

The conventional wisdom is still firmly entrenched in the diet-cholesterol-heart theory, created in the 1950s. We question this hypothesis, as it has many flaws and weaknesses, and the true causes of arteriosclerosis still remain too obscure to form a robust conclusion. We humbly admit that we understand very little about the pathogenesis of arteriosclerosis. While no definite cause has been identified so far, the doctors have listed a long list of so called risk factors, or indicators, in all about 250, which more or less associate with the disease without actually causing it. Therefore modification of these indicators (e.g., lowering of cholesterol) cannot be expected to give much benefit.

Most popular (classic) risk indicators include: smoking; eating saturated fats; hypertension; lack of exercise, large waist circumference; "apple" body shape; non-genetic form of transmission; persistent, long-term, intense psychological stress; dysfunction of the fat and glucose metabolism; calcification of arterial walls; inflammatory diseases; accumulation of homocysteine in the blood and tissues. Recently new risk factors have emerged: micro-organism infections and the lack of sunshine and consequent vitamin D insufficiency. We will present some other "new" risk indicators later in this guide.

On the highest level of the "risk pyramid" of a heart attack are diabetics and those suffering from metabolic syndrome, who in the past have already suffered a heart attack. On the next level down are diabetics and those with a metabolic syndrome who have not yet experienced a heart attack. Their risk is similar to those non-diabetics who have survived a myocardial infarction. The common denominators in the risk pyramid are insulin resistance and dysfunction of fat and glucose metabolism. Further down the pyramid are those people who have one or several of the aforementioned risk factors.

Arteriosclerosis usually starts early in life, sometimes during childhood or teenage, and stays latent for decades without necessarily manifesting any symptoms or sensations. When symptoms eventually appear, the disease has progressed quite far, and the changes are largely irreversible.

Coronary arteries, which supply oxygen for the heart itself are, when young, flexible and elastic like lycra; they are highly resistant to pressure and they enlarge with the pulse waves. However, with age the elasticity decreases, blood pressure rises, and the blood does not circulate in the arteries as freely as before. A result of this can be either a stroke or a heart attack. The hardening of arteries (sclerosis) is largely due to impaired dysfunction of the endothelium in the inner cell layer in the arterial wall. Therefore the emphasis of the primary and secondary prevention should be targeted to endothelial functions.

A stealthy, chronic inflammation

As far back as 1856, the father of immunology, the German Rudolf Virchow, wrote that arteriosclerosis is an inflammatory process, and inflammation usually has an infective cause. This view was forgotten and was sidelined for decades by the cholesterol boom, but it has now resurfaced again. Therefore, the prevention and treatment of CHD must specifically target something other than cholesterol, namely inflammation, which can be diagnosed from blood tests, e.g., the value of C-reactive protein (CRP), preferably high sensitivity CRP (hsCRP). Despite the reference value for CRP being under 6, even a value of 3 indicates a stealthy inflammation. Other useful indicators of inflammation include the interleukins 1 and 6 (IL-1, IL-6) and the tumour necrosis factor alpha (TNF-a).

A key event in atherosclerosis is a maladaptive inflammatory response to subendothelial lipoproteins. A crucial aspect of this response is a failure to resolve inflammation. Defects in these processes promote the progression of atherosclerotic lesions into dangerous plaques, which can trigger atherothrombotic vascular disease (Figure 1). This revolution in our thinking about the pathophysiology of atherosclerosis has now begun to provide clinical insight and practical tools that may aid patient management.

We would like to elaborate further about the infective origin and the role of inflammation in process of arteriosclerosis: If there were no resistance, attacks by germs would continue to anabated until complete destruction of the artery occurred. Luckily, the body has some defense mechanisms to counteract foreign invaders. Different cellular, inflammatory and immunological mechanisms come into play in reaction to invasion, and damage the germs. A key element is the accumulation of immune cells called macrophages that are filled with cholesterol, which gives the cells a foamy appearance. Macrophages engulf and destroy germs. Foam cells are not dangerous as such, but can become a problem when they accumulate at particular foci thus creating a

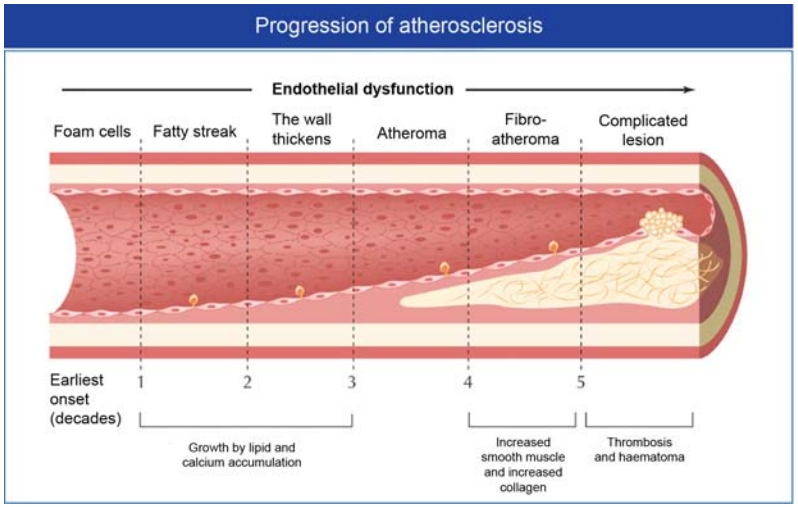

Figure 1.

necrotic centre of the atherosclerosis. If the fibrous cap that prevents the necrotic centre from spilling into the lumen of a vessel ruptures, a thrombus can form which can lead to emboli occluding smaller vessels. The occlusion of small vessels results in ischemia, and contributes to stroke and myocardial infarction, two of the leading causes of cardiovascular-related death. We will later present therapeutic strategies designed to boost inflammation resolution.

Oxidative stress

In the 1950s Professor Denham Harman (University of Nebraska), presented a theory about the oxidative stress caused by free radicals resulting in cell damage. Today his theory has been generally accepted as correct in medicine and biology.

Oxidative stress is caused by external and internal free radicals, which make the fats turn rancid, including LDL-cholesterol. Cholesterol as such is entirely inert (harmless), but when oxidised, it starts to stick to the inside of arteries, impairing the blood circulation. Luckily, oxidative stress can be prevented, suppressed and quenched by strengthening the body´s antioxidative capacity by means of a healthy diet providing plentiful antioxidants, and also by taking antioxidants as supplements, like alpha lipoic acid, berberine, carnosine, selenium, ubiquinone (CoQ10) and vitamins C and E, as we will discuss later in more detail.

Cholesterol and Triglycerides

Laymen and most healthcare professionals alike consider cholesterol as the main culprits of cardiovascular diseases. Million of people take cholesterol-lowering drugs and statins, the vast majority probably needlessly. The role of cholesterol, in our view, is enormously exaggerated. Cholesterol is a necessary building block for cells, which, when not oxidised or glycated, is entirely harmless. There are many different fractions of cholesterol, out of which HDL is considered 'good', while LDL, sdLDL (small dense LDL) and very

low density LDL (VLDL) are considered 'bad'. Oxidised LDL (oxLDL), can constrict and stiffen arteries. It is toxic, and therefore the body produces antibodies against it. The more there is LDL in the blood, the more easily it will oxidise. However, the oxidation of LDL can be prevented with antioxidants, of which there are plenty in vegetable-based foods (e.g. in salads, fruits and berries), and dietary supplements.

Increased blood glucose glycates LDL to form AGE-LDL, which, like oxLDL, sticks to the endothelium of arteries. The good news is that vitamin B6 (pyroxin), berberine and carnosine are able to prevent and suppress glycation.

In addition to cholesterol, there are other "bad" fats, which need to be targeted in the prevention and treatment of CHD. Another fat called triglyceride (TG) is a significant risk indicator in CHD. Thirty per cent of men under 65 years and 14 per cent of women of the same age have their blood plasma significantly increased TGs (2,0 mmol/l or more). They originate from the fats made up by the liver from alcohol and dietary carbohydrates. The liver converts, that is, any extra carbohydrates and alcohol, that are not immediately used for energy, into fats. For this reason we advise avoiding too much carbohydrates and alcohol; moreover, they may cause a fatty liver.

Although the commonly used statins effectively lower cholesterol, they also have unwanted side-effects, like worsening the balance between omega-6 and omega-3 fatty acids in the blood. Statins also prevent the synthesis of ubiquinone (coenzyme Q10) in the liver thus decreasing its content in the blood. These facts add further weight to the use of omega-3 fatty acids and CoQ10 as supplements to statins.

Statins and other cholesterol lowering drugs, in the usual doses, do not reduce TGs significantly, while the omega-3 fatty acids in fish oil – EPA and DHA - do. Therefore fish oil was recently added to the doctors´ guidelines for treatment of dyslipidemias (disturbed fat profile in the blood) and artery diseases.

Contrary to popular belief, the omega-3 fatty acid ALA found in vegetable oils (rapeseed, flaxseed etc.) is not as effective as the omega-3s in fish oil, EPA and DHA. In fact, they prevent effectively the synthesis of TGs in the liver and at the same time they prevent fatty liver. The ratio of cholesterol and TGs – non-HDL/triglyseride + LDL can give a better picture of the risk of illness than simply measuring one or the other (cholesterol or triglyserides).

Atherogenic metabolic triad

Hyperinsulinemia, elevated apolipoprotein B (Apo B) and sdLDL, in the same individual, are strong indicators for CHD. The triad is usual both in diabetics and in those with metabolic syndrome. Ethylesterised fish oil lowers simultaneously all the different factors in this imminent triad.

Increased homocysteine

Homocysteine is a toxic amino acid, which the body makes from proteins in meats. Homocysteine is associated to arteriosclerosis, loss of memory and Alzheimer´s disease and other types of dementia. Substances that counteract homocysteine include folic acid, and vitamins B6 and B12. Folic acid appears mainly in vegetable foods, but it is unfortunately not part of most ageing men's diets. For them, we recommend vitamins that will lower homocysteine. The readings can easily be obtained from a blood sample. Whenever the reading exceeds 7 micromoles/litre (µmo/L), we recommend vitamin supplements, although the laboratory reference values are 5–15 µmo/l.

The Calcification of Arteries

Calcium deposits accumulate into the arterial walls and lead to the hardening of the arteries. This has been ascertained by computer scanning which also allows for giving the so-called Agastson's score as a measure of the calcification. If calcium is not discovered, the score as well as the risk of a heart attack equals zero, while a bigger score indicates a risk of heart attack and death. Vitamin D deficiency associates with calcification of arteries, and conversely, a sufficient intake of this vitamin (or sunshine) may prevent this. To this end, the daily requirement for vitamin D for an adult is around 50 µg, i.e. about 7 times the current official recommendation (7.5 µg/day). Also intake of vitamin C (500 to 1000mg daily) may decrease the risk of arterial calcification.

Psychological Stress

Long-term stress may increase the risk of a heart attack. E-EPA (1000mg daily) has been found to balance stress hormones on the hypothalamic-pituitary-adrenal axis (HPA-axis) and hence reduces stress symptoms and the risk of CHD.

Other Risk Factors

An apparently healthy person – who has not been diagnosed with any of the aforementioned risk factors – can suffer a sudden heart attack. It can come with no warning, like a lightning bolt. There are many new explanations for this:

Angiotensin, a protein, causes blood vessels to constrict, and drives blood pressure up. It is part of the renin-angiotensin system, which is a major target for drugs that lower blood pressure. Angiotensin also stimulates the release of aldosterone from the adrenal cortex. Aldosterone promotes sodium retention in the distal nephron, in the kidney, which also drives blood pressure up. Berberine counterfights angiotensin, which explains many of berberine´s benefits in cardiovascular diseases and diabetes.

Fibrinogen, a protein in blood plasma, causes blood to clot by breaking down into fibrine. That in turn is a fibrous protein which promotes blood clotting.

Endothelin, is a peptide, which is produced in arterial walls, kidneys, retina and in the heart muscle. Endothelin thickens the heart muscle and stiffens arterial walls. The use of insulin when treating diabetes promotes formation of endothelin; excessive insulin can therefore be harmful. The detrimental effects of endothelin can be minimised by losing weight, exercising and taking fish oil and carnosine supplements.

Lipoprotein (a) [Lp(a)] is formed by an LDL-particle, with an apoprotein (a) attached to its apolipoprotein B. Lp(a) is an independent risk indicator in atherosclerosis. The amount of it in blood is independent of ageing, cholesterol, blood pressure, diet and exercise. The concentration is determined by hereditary factors, and it is quite stable in each individual (at<250mg/L). A larger concentration indicates an increased risk of cardiovascular disease. Lp(a) decreases with niacin and omega-3 fatty acids, but not with statins. This is yet another reason to take fish oil in cardiovascular diseases.

Myeloperoxidase (MPO), is a protein secreted by white blood cells. The function of MPO is to destroy harmful bacteria, which have made their way into the bloodstream. At the same time, however, it may inflame the inner walls of the arteries, constricting them and impairing the circulation up to ten years before the appearance of clinical cardiovascular disease. This favours the hypothesis of the

infectious origin of arterial diseases.

MPO adds to the formation of the toxic substances called Advanced Glycation End products (AGEs) in the body. MPO oxidises LDL cholesterol and simultaneously MPO itself causes inflammation in the arteries. In addition, MPO alters "good" HDL cholesterol in such a way that its protective effect is lost, especially in diabetics. MPO also reduces the amount of nitric oxide (NO) in the blood, which results in the lessening of arterial flexibility. Carnosine, on the other hand, increases the formation of NO in blood, which prevents hypertension, amongst other things. Both carnosine and E-EPA counteract MPO, which supports the use of these dietary supplements in cardioprotection. MPO and other "new" risk factors can be measured from the blood, e.g. in physical checkups.

Omega-6/omega-3 ratio, calculated from analysis of fatty acids in the blood, is another way of estimating the risk of heart disease. Heart patients often have less omega-3 in their blood than healthy people, partly due to low dietary intake and partly due to a genetically overactive enzyme called Phospholipase A2 (PLA2). The enzyme breaks down fatty acids in cell membranes, which in addition liberate fatty acids of the omega-6 family, especially arachidonic acid (AA). It promotes and maintains inflammation in the endothelium and leads to endothelial dysfunction. The ratio between AA and EPA (AA/EPA) is a useful indicator of the risk of future heart disease and of the progression of an existing condition. The larger is the ratio, the worse is the prognosis. The AA/EPA ratio should not exceed 3:1 in a healthy individual and not 2:1 in a heart patient. Whenever the ratio is larger, we advise intake of E-EPA fish oil so that the ratio comes down to ideal levels. If the AA/EPA ratio goes under 1:1, the E-EPA dosage needs to be reduced. E-EPA suppresses the overactive PLA2 as well as inflammation, thus protecting the heart and arteries. The overactive PLA2 is also known to increase the risk of depression.

Endothelial dysfunction as mechanism of the artery disease

The risk indicators mentioned above are "partners in crime", interacting with each other and strengthening and maintaining each other. This leads to a vicious circle: an arterial disease in its early stages feeds on itself. We consider it very important that people learn how to break this vicious circle through lifestyle changes, at which point the prognosis for the disease will improve markedly. Not all of those suffering from arteriosclerosis possess the common risk factors, but they all do have calcium in their artery walls, as well as quiet chronic inflammation and endothelial dysfunction in the arterial walls. The understanding of these factors is vital so that we are able to prevent the illness and manage it adequately.

Arteriosclerosis always begins with an endothelial inflammation and the activation of immune cells, which may lead to the changes shown in diagram 1. If a person has some of the risk factors mentioned above, this will speed up the damage. In this case the endothelial cells' telomeres shorten more quickly than usual and the mitochondria are damaged. When the damage occurs in the coronary arteries, the heart muscle cells suffer from acidosis (low pH). Changes in the mitochondrial membranes are crucial in the onset and worsening of cardiovascular diseases. Dietary fats have a major influence on the condition and functions of ageing mitochondria. Especially the omega-3 fatty acids and ubiquinone protect mitochondria. Therefore we suggest complementing the current treatment with cardioprotective dietary supplements to protect the telomeres and mitochondria.

Meals which contain large amounts of trans and omega-6 fats will stiffen arteries a few hours after eating, and cola drinks will do this within half an hour of drinking. If a person eats this type of pro-inflammatory junk food often, the arteries will harden and stiffen, especially if his or her glucose metabolism is impaired. However, those people who consume large amounts of fish and fish oil have arteries that stay more elastic than average, even if they eat greasy meals. Omega-3 fatty acids suppress inflammation and thus prevent the hardening of arteries. This is why these supplements are beneficial, especially for diabetics.

HOW IS ATHEROSCLEROSIS DIAGNOSED?

Blood tests can give an indication about the risk factors of the disease, but there is no convenient reliable diagnostic examination for asymptomatic persons. Therefore the true prevalence of CHD in the population remains unknown. The statistics are based on diagnosis and mortality of symptomatic subjects. In scientific studies they measure the thickness of the carotid artery wall by ultrasound or determine the speed of the pulse wave from an upper arm artery to the foot artery. An ophthalmologist can see arterial sclerosis by looking at the retina, where the arteries are visible in their natural state. CHD may affect the results of an electrocardiography (ECG) test, especially during exercise. More precisely, the disease can be diagnosed by angiography or by computer scanning and giving an Agastson´s score.

SIMULTANEOUS OTHER DISEASES – CO-MORBIDITY

Many heart patients have simultaneously high blood pressure, insulin resistance, metabolic syndrome and diabetes. About 80% of diabetics become ill with and die of myocardial infarction or stroke, even when they are being treated according to the recommended treatment guidelines. The most common causes of death are CHD (45%) other heart diseases (15%) and stroke (10%). One in three heart patient suffers from depression, at some point of time, and the heart and blood pressure medications seem to increase the risk of and worsen depression. One of the common denominators for these diseases is overactivity of the PLA2-enzyme family, which can be dampened with E-EPA, as we mentioned earlier. Heart patients also run a risk of contracting Alzheimer´s disease or vascular dementia. A heart attack will increase the risk of stroke 44-fold over the course of a month, and over the course of three years additionally 2 to 3-fold. This risk may be lowered by taking dietary supplements.

PRINCIPLES OF PREVENTION

Practical tips for prevention of heart attacks:

- Measure your waist circumference (the upper limit: men, 90cm; women, 80cm). Exceeding the limits indicates that you are overweight.
- If you are overweight (obese) begin a diet and maintain a sensible weight.
- Follow a low-carb diet. Familiarise yourself with the glycaemic index and load of carbohydrates.
- Do not smoke, consume alcohol only in moderation and do not eat sweet treats.
- Exercise regularly at least for an hour a day (an average 7 hours a week).
- If you belong to a risk group or you already have CHD, or you have had an infarction or stroke, take daily and continuously the supplements recommended in this guide. They may save your life.
- If there is diabetes in your family and you are overweight, get your blood sugar measured both after fasting and after a glucose tolerance test; also request an HbA1c and an insulin secretion test. You should also check your blood glucose levels 30 to 45 minutes after meal. This postprandial or HbA1c test may uncover prediabetes of which you may not be aware.
- If you have any of the following: impaired glucose tolerance, insulin resistance, diabetes, hypertension or metabolic syndrome, ensure you manage them with care (view our guide Guidelines for Good Management of Diabetes).

DIETARY SUPPLEMENTS

Omega-3 fatty acids EPA and DHA in fish oil

The American Heart Association (AHA) and the world's leading cardiologists recommend fish oil as a dietary supplement to prevent and treat heart and artery diseases. UK's critical watchdog, the National Institute for Health and Clinical Excellence (NICE), recommends that doctors prescribe ethylesterised fish oil to patients who have had a heart attack during the last three months, especially if they do not eat fatty fish 2 to 4 times a week. Many top cardiologists recommend a combination of E-EPA and E-DHA for prevention of cardiovascular disease (at least 500mg/day) and treatment (approx 1g/day). For patients with high triglycerides (TG) the recommendation is 3 to 4g/day; this dose usually lowers the readings by 20 to 50%. Two fatty fish meals a week may contain up to 500 mg omega-3 (EPA and DHA combined), but it is not enough for heart patients with high TGs. In these patients, the ethylesterised fish oils almost halve the risk of sudden cardiac death.

Foto: Osmo Lehtinen

A low concentration of EPA + DHA in the blood is an independent risk factor for a sudden heart attack, which is adjustable, according to professor William S. Harris from Missouri University, one of the writers of the AHA guidelines. He recommends for heart patients 850 mg daily as a combination of E-EPA and E-DHA. It goes well as an extra treatment to statins.

"Combined treatment of statins and fish oil is a safe and effective way of improving the fat profile in blood and therefore also the patient's prognosis as opposed to statins only", write the leading US cardiologists in the journal "Mayo Clinic Proceedings".

So far, the largest fish oil study in the world, JELIS, suggests that E-EPA, 1800 mg a day taken together with statins, prevents heart attacks and angina pectoris pain considerably more effectively than statins alone (see diagram 2). According to JELIS, E-EPA also reduces significantly the risk of recurrent stroke. So, at present there are two excellent options for heart patients, either E-EPA alone or a combination of E-EPA + E-DHA. Both options provide powerful protection against heart attack and stroke. In contrast, traditional omega-3 products, such as cod liver oil, are not as effective as the new generation fish oils.

The effect of the new fish oils goes "beyond cholesterol", i.e. prevention and suppression of the principal mechanism of the disease, i.e. chronic low-grade inflammation. The omega-3 fatty acids are converted in the body into powerful anti-inflammatory compounds (resolvins docosatirenes and maresins). Resolvins are produced even more when aspirin and EPA are taken together. For this reason, we recommend E-EPA or E-EPA + E-DHA for people using aspirin for cardioprotection.

Omega-3-fatty acids

1. Reduce intestinal (visceral) fat
2. Increase the Omega-3 index
3. Reduce AA/EPA ratio
4. Reduce elevated triglycerides in the blood
5. Reduce Apo B; sdLDL; remnant cholesterol particles; endothelin, and myeloperoksidase
6. Prevent and suppress inflammation
7. Stabilise arterial plaques and prevents their ruptures
8. Prevent arrythmias
9. Prevent blood clots
10. Protect mitochondria from the side effects of statins
11. Protect telomeres and thus prolong survival of patients with coronary heart disease.

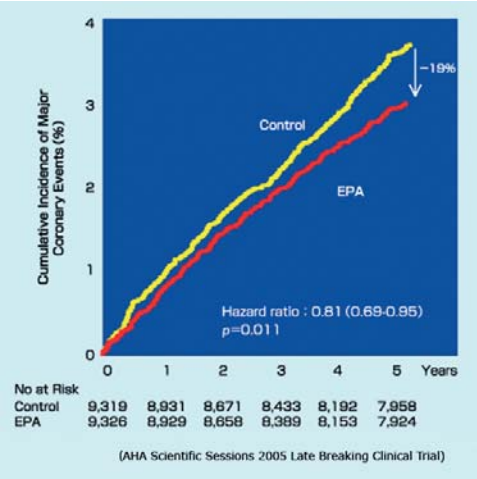

Diagram 2. JELIS-study showed, that E-EPA (1800mg per day), taken with statins prevents every fifth heart attack over five years.

Berberine

Berberine is a plant-derived folk medicine, which has been used for over 2,000 years in the Orient. Recently, scientists in the West have shown that berberine is a powerful positive inotropic substance (like digitalis), which increases the strength of contraction of heart muscle (myocardial contractility). As such, we recommend berberine to support cardiac function in conditions such as CHD, heart failure, myocardial infarction and cardiomyopathy. Berberine also lowers elevated blood glucose readings and HbA1c. Moreover, berberine lowers elevated cholesterol like statins although by a distinct mechanism, without risk of side effects. The effective and safe daily dose is 1 to 1,5 grams.

Carnosine

Carnosine is a cardioprotective protein (peptide) that occurs naturally in humans, e.g. in the brain and heart muscle. When taken as a supplement carnosine prevents the acidification of cardiac muscle cells, which is always present in arterial disease (when the heart is lacking in oxygen). Carnosine also produces nitric oxide in the body, which maintains arterial wall elasticity. In this way carnosine prevents and treats hypertension. Carnosine activates vitagenes, which prevent premature ageing. We recommend 400 to 800mg/per day for prevention and 800 to1200mg/per day for treatment. Carnosin does not have side effects, and it can be taken with all medications and other dietary supplements.

Magnesium

Magnesium is an essential mineral for the heart and arteries. Heart patients often suffer from a sub-clinical magnesium deficiency which, when corrected, improves significantly the patient's prognosis. A latent magnesium deficiency may cause arrhythmia and conversely, magnesium supplements may prevent it. Magnesium deficiency may be detected in a blood test up to five years before a clinical myocardial infarction manifests itself.

Magnesium also works like statins, in other words, it prevents the liver from producing too much cholesterol. Magnesium also protects the heart from the side effects of the drug digitalis (Digoxin).

A long-standing magnesium deficiency can cause the hardening of the aorta and the carotid artery. Magnesium strengthens the inside walls of the arteries and prevents them collapsing inwards (like a rusted exhaust pipe), which is often an immediate cause of heart attack and stroke. A heart patient needs magnesium 350 to 700mg per day as supplement. Magnesium works most efficiently when taken together with folic acid, and vitamins B6 and B12.

Selenium

Selenium is an excellent and versatile antioxidant, which protects both the heart and the arteries from oxidative stress. Organic selenium is more effective than inorganic. A suitable daily dose of organic selenium in self-treatment is 100 to 200 microgrammes (µg).

Chromium

Organic chromium, e.g. as picolinate or nicotinate, may prevent the increase in LDL-cholesterol and normalise glucose metabolism. Chromium is especially useful as a weight control agent in obesity, diabetes and metabolic syndrome. A heart patient's daily need is around 100 to 300µg.

Vitamin D

Vitamin D is crucial for prevention of arterial calcification, but for this, around 50µg per day is needed. The safe upper limit in continuous use is 250µg/day (adults). Heart patients should have their serum vitamin D concentration (25-OHD) analysed in order to ensure sufficient intake. The optimal level is between 35 and 50ng/mL (about 90 and 130nmol/L). The upper safe limit is 100ng/ml or 250nmol/L.

Folic acid (folate) and other B-vitamins

Low vitamin B status has been linked to inflammation, oxidative stress, and an increased risk of cardiovascular diseases. 300 microgrammes (µg) folic acid is the recommendation for healthy people, but a heart patients may need as much as 800µg/day. It is impossible to get that much from food, so a supplement is necessary. Folic acid and other B-vitamins are anti-inflammatory, anti-oxidative, and they lower the homocysteine, protecting thus the heart, the blood vessels and the brain.

Vitamin B3, or niacin

This is the most effective substance known to increase "good" HDL-cholesterol. Niacin lowers high triglycerides and lipoprotein A. Combining niacin with statins protects against heart-related deaths better than statins on their own. In fact, the FDA has recently approved this combination as a medicine. The European Consensus Panel also recommends the combination of niacin and statins for prevention and treatment of CHD and diabetes.

Ubiquinone (Coenzyme Q10, CoQ10)

This vitamin-like substance helps the cells' power plants, mitochondria, to produce energy. CoQ also prevents damage to mitochondria produced by statins. Because of this we recommend CoQ10 (100–300mg/day) for all heart patient who are on statins.

Vitamins C and E

Vitamin C is a water-soluble, and vitamin E a fat-soluble antioxidant. They both prevent oxidative stress and chronic inflammation. One can safely take 500 to 1000mg of vitamin C and 100 to 200mg of vitamin E daily on a daily basis.

Alpha lipoic acid

Alpha lipoic acid is another versatile and effective universal antioxidant, which is recommended in doses of 300–600mg per day to combat and prevent oxidative stress and inflammation.

All the aforementioned dietary supplements can be taken continuously on a daily basis, and if need be, together with any medication. These supplements do not cause any side effects when used according to the guidelines given here. On the contrary, they protect the liver, brain and heart from the side effects of drugs.

In this guide we provide practical advice and guidance for self-treatment of cardiovascular diseases, so that everyone could take care of their health and wellbeing. We intend to impart the latest information on cardiovascular risk factors and to advise on how they can be mitigated naturally. We emphasise the importance of self-care and the responsibility everyone has for their own health. We emphasise a healthy lifestyle, regular exercise, avoiding smoking, salt and excessive alcohol and a healthy and varied diet, which aids permanent weight-control. We especially highlight the dangers of fat deposits around the waist area and visceral fat, and recommend that you reduce it through exercise, low-carb diet and supplements. In this guide, we do not cover drug treatments, except where supplements can be used to strengthen the positive effects of some drugs and where they can prevent side-effects, e.g. of cholesterol-lowering statins.

Matti Tolonen (right), MD, is docent at the University of Helsinki. He is reknowned as one of the pioneers of complementary or integrative medicine. He resides on Costa del Sol, Spain. **Pentti Raaste**, MD, is a GP with a private practice in Fuengirola, on the Costa del Sol in Spain. His contact number is +34 952 47 52 90.

Foto: Osmo Lehtinen