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WHAT ALL YOU SHOULD KNOW ABOUT HEART AND HOW TO KEEP IT FIT

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Abstract: *Due to the increasing number of men and women who are unaware about the working of heart and hear t disease an d surgery and how can the heart valve and coronary artery bypass surgery is done,. The person who had come for health checkup were told about heart, how it works, its valve, the effect of valve on heart , how the heart valves are check and how can they be cured by different surgery. They were told about cigarette smoking, alcohol drinks, fat consumption, nature of physical exercise and other important things and what the burden of smoking for our economy is. And how can we improve the heart and keep it healthy by this teaching we could save lot.*

Keywords: *Heart, Valve, Surgery, Cigarette, Alcohol, Fat, Exercise*

INTRODUCTION

The heart is divided into a right and left portion. The right portion receives blood (which is impure) from the body and pumps the same to the lungs for purification (oxygenation). The impure blood enters the heart from two large veins called the superior and inferior vena cava. The blood from these veins enters an upper chamber known as the Right Atrium. This chamber also receives impure blood from the heart veins through the coronary sinus. The right atrium pumps this blood into the Right Ventricle or the lower chamber through a Tricuspid Valve. The tricuspid valve prevents blood from flowing from the right ventricle to the right atrium. The right ventricle pumps blood into the Pulmonary Artery. The Pulmonary Valve prevents blood from leaking back into the right ventricle. The pulmonary artery carries impure blood to the right and left lungs. The left half of the heart collects and pumps pure (oxygenated) blood from the lungs to all parts of the body. The blood from the lungs enters the heart from four veins called the Pulmonary Veins. These veins bring the blood into the upper chamber called the Left Atrium. The left atrium pumps blood through the Mitral Valve into the left ventricle. The mitral valve prevents blood from leaking back from the left ventricle to the left atrium. The left ventricle pumps the blood into the Aorta which circulates to all parts of the body. The Aortic Valve prevents blood from leaking back into the left ventricle. The four valves in the heart are the Mitral and Aortic valves on the left

side and Tricuspid and Pulmonary valve on the right side. Of these, the mitral valve is most commonly affected by diseases. The Aortic valve is second most commonly affected heart valve. The tricuspid valve is never diseased alone. Disease of the tricuspid valve always accompanies diseases of the mitral valve.

CAUSES OF THE VALVE DISEASES

The most common cause of valve diseases in children and adults is known as Rheumatic Heart Disease. This problem occurs because of the poor hygienic conditions, malnutrition and infection by microorganism (Streptococcos). Rheumatic Fever produces sore throat, joint pains, swelling of the joints, fever and others symptoms. Repeated attacks of such symptoms may cause heart valve disease in the long run. This rheumatic heart disease affects the mitral valve most commonly, the Aortic valve and the Tricuspid valve in the order. The Pulmonary valve is almost never affected by long standing rheumatic heart disease (RHD). This may cause narrowing or leakage of one or more valves.

The other most common cause of valves disease is birth defects. This abnormality may affect the Aortic valve or the mitral valve. They usually cause narrowing of the aortic valve or leakage of the mitral valve.

EFFECTS OF VALVE DISEASE

Mitral Stenosis (MS)

This means narrowing of the Mitral valve. As a result blood cannot pass into the left ventricle and becomes dammed in the left atrium and pulmonary veins. The pressure in the left atrium also increases and in long standing mitral stenosis the pressure in the pulmonary artery also increases. The valve may also become hardened (calcified). Clots may also be found in the left atrium because of the stagnation of blood. The patient suffers from reduced blood flow to the body and increased pressure in the lungs which causes shortness of breath, rapid beating of the heart (palpitation) and heart failure. It may also lead to paralytic stroke if blood clots or calcium particles go away in the blood circulation. The patient may also develop spitting of blood (haemoptysis).

Mitral Regurgitation (MR)

Mitral regurgitation means leaking of blood from left ventricle to left atrium. The effects of such a leak are similar to the affects of mitral stenosis (MS).

However, mild Mitral Regurgitation (MR) can be tolerated without any serious consequences for a long time. If the leak is severe the patient develops enlargement of the heart, shortness of breath, palpitations and heart failure. Both stenosis and mitral regurgitation may be present together.

Aortic Stenosis (AS)

Narrowing of the Aortic valve particularly in children is usually due to birth defects. However, rheumatic heart disease can also cause narrowing of the aortic valve. As a result, pressure in the left ventricle increases. The left ventricle becomes very thick. The flow of blood to the body decreases. The valve may also become calcified. As a consequence of these, the patient may suffer fainting attacks, shortness of breath and palpitation. Sudden death has also been known to occur in such patients.

Aortic Regurgitation (AR)

Leaking of the aortic valve causes return of blood from Aorta to the left ventricle. The heart becomes enlarged and the ventricle becomes thick. Blood flow to the body decreases and the patient suffers from low blood flow. This causes breathlessness, palpitation and heart failure. Both aortic stenosis and aortic regurgitation may be present together and is usually due to rheumatic heart disease (RHD). Hardening of the aortic valve (calcification) occurs more often in older patients. This may cause paralytic strokes or heart attacks.

Tricuspid Stenosis (TS)

Tricuspid stenosis is usually caused by rheumatic heart disease (RHD). Because of this, there is obstruction to blood passing from the right atricum to the right ventricle. This results in enlargement of the liver, swelling on the feet, fluid accumulation in the abdomen and heart failure.

Tricuspid Regurgitation (TS)

This may be due to enlargement of the right ventricle or due to effects of rheumatic heart disease, because the leak blood returns from the right ventricle to the right atrium. This is always seen together with mitral valve disease. The patient may suffer the same consequences as in tricuspid stenosis.

SYMPTOMS

The patient experiences some difficulties when one of the heart valves is damaged. These difficulties are known as symptoms. One of the most common symptoms is breathlessness. In the early stages breathlessness is experienced on exercise like running, cycling, climbing

stairs etc. However, as the disease progresses and becomes more pronounced, the patient experiences breathlessness by just walking to the bathroom and sometimes even at rest. In extreme cases patient may be unable to lie down and may experience breathlessness just sitting up.

The other most common symptoms is a thumping in the chest known as palpitation. This is due to rapid and forceful beating of the heart. Heart beat becomes irregular and fast in some patients with valve disease. Again palpitation may come on with exercise or may even occur at rest. The next common symptoms is tiredness (Fatiguability). This may be experienced in the early stages and becomes more pronounced as the disease progresses. Patients with mitral valve problems may also have blood spitting. This is because of the severe narrowing of the valve in mitral stenosis (MS). Patients with aortic valve disease may notice giddiness, black out or fainting attacks. Patients with tricuspid valve disease may notice swelling on the feet, abdominal distension and bluish discolouration. In patients with calcified valves or in patients with clots inside the heart, paralytic strokes may occur resulting in paralysis of one limb or one side of the body or loss of speech. Permanent damage is sometimes quite likely.

TESTS FOR VALVE DISEASES

Most heart valve disease patients can be diagnosed with a x-ray of the chest, ECG and Echocardiography. Echocardiography may have to be done by passing a tube into the stomach and examining from inside. This is known as Transesophageal Echocardiography (TEE). This may be necessary in patients who have suspicion of involvement of more than one valve; for example the mitral valve and tricuspid valve or mitral valve and aortic valve. These tests do not cause much discomfort. Occasionally cardiac catheterization or angiography may be required in older patients. In this a tube is passed from the groin into the heart where pressure is recorded and x-ray are taken to see the damage caused to the valves. The patient will need to stay in the hospital for a day. CT Scan and MRI Scan may not be required for diagnosis. With echocardiography the severity or seriousness of the problem can also be estimated, in order to recommend proper treatment, When the disease is mild no operation may be required.

TREATMENT

Generally patients with heart valve problems develop heart failure. Because of this they retain fluids in the body which causes breathlessness and swelling on the feet. Treatment

with medication is meant to improve heart function and to remove fluid from the body. Drugs like Digoxin and others which increases the flow of urine (like Lasix and Ditide) may be prescribed. Patients are also advised to restrict salt intake and fluid intake to 800 ml to 1000 ml (1 litre) per day. This includes fluids such as tea, coffee, fruit juice, milk and water that is taken in a 24 hour period. Seriously ill patients may require to restrict their activity in order to remain symptom free. In patients who have irregular heart beat (Atrial fibrillation). Drugs may be given to reduce the possibility of blood clotting. This will prevent the occurrence of strokes or paralysis. These drugs have to be taken carefully and require constant monitoring. During treatment with these drugs the patient is likely to bleed excessively during menstrual period or if an injury occurs.

Operation is recommended for patients who have serious problems. Before operation patients should make sure that they do not have any infection in the teeth or elsewhere such as the ear in children and urinary tract in adults. The patient should be admitted to the hospital a day or two before operation. If your doctor recommends operation, it is better to get it done as early as possible before developing a complication which will require emergency operation with more risk to life.

Narrowing of valves such as mitral stenosis (MS) or aortic stenosisi (AS) can be opened up without a major operation by using a balloon catheter. This is known as Balloon Valvuloplasty. This is done by the Cardiologist in the Cardiac Catheterisation Laboratory. In this procedure a tube is passed from the groin into the heart and maneuvered into the mitral valve. A balloon is passed through this tube into the mitral valve. When the balloon is inflated it forces open the narrowed valve. This operation avoids a scar, however it is not possible to use this in all patients. This procedure costs between Rs. 230,000 to Rs. 40,000 in various hospitals. This procedure may also fail to open the valve or may cause a tear in the valve resulting in leakage. Such patients may require emergency operation to save their lives.

OPERATIONS FOR HEART VALVE DISEASES

Narrowing of the Mitral Valve or Mitral Stenosis

The operation for mitral stenosis (MS) is known as Closed Mitral Valvotomy (CMV). It is usually recommended for patients with only narrowing of the valve without calcification and in young patients who have a normal regular heart beat. The operation can be performed in any major hospital by a qualified surgeon. Here a finger is passed under controlled

conditions and an instrument called the dilator is introduced into the valve and the valve is forcibly opened. This operation is more successful than balloon valvuloplasty. This operation costs very less in a hospital like AIIMS and can be done free of charge for poor and needy patients. Patients who have irregular heart beat but do not have a clot in the heart can also undergo this operation provided a Transesophageal Echocardiography (TEE) shows that there is no clot. However, they have to be treated with blood thinning drugs (anticoagulant) for one and a half month before such an operation.

Open Heart Surgery

It the mitral valve is leaking or if the aortic valve is diseased or the tricuspid valve is diseased the patient will need open heart surgery. Here the heart has to be stopped and circulation of blood and oxygenation of blood will be taken over temporarily by a machine (Heart Lung Machine) so that surgeon can look inside the heart and perform a procedure to correct the problem.

Mitral Valve

Open heart surgery on the Mitral valve may be Open Mitral Commissurotomy (OMC), Mitral Valve repair or replacement. Open Mitral Commissurotomy (OMC) is possible if the valve is narrowed and is not calcified.

Mitral Valve Repair

In this operation correction of mitral stenosis (MS) and mitral rgurgitation (leaking valves) is done under vision. This operation is suitable for young patients, women and those who do not have calcification. The surgeon will reconstruct the valve so that its function is returned to near normal. The advantage is that the patient retains his own natural valve. He will not require anticoagulant medicine for all his life. He will also not require expensive hospital tests. The operation also costs about ½ to 1/3 of the cost of valve replacement with an artificial valve. This operation is also best suited for young patients, women and elderly people.

Mitral Valve Replacement

Replacement of the mitral valve becomes necessary when the valve is severely damaged or calcified. It may also be required in patients who are undergoing a secong operation. In this, surgeon will remove the mitral valve and replace it with an artificial valve (Prosthetic valve), a valve made from animal tissue (Bioprosthesis or with a human valve taken from a dead

person (homograft). The cost of such operation is much more than valve repair, In addition the patient will require to take anticoagulant medicines for the rest of his life. The patient will also require blood and other tests to monitor the function of the valve at regular intervals.

Aortic Valve

In young patients with narrowing of the Aortic valve a balloon dilatation can be done. However, this operation is not very successful. Most patients with aortic valve problem will require open heart surgery. Diseased aortic valve can be repaired or replaced.

Aortic Valve Repair

This operation can be performed only in some centres and by experienced surgeons. It is most difficult. However, it helps the patients to retain his own natural valve. It is also less expensive. The operation may also fail to correct the abnormality permanently. This means that the patient may require a second operation after several years if the valves is damaged again.

Aortic Valve Replacement

The major operation for Aortic Valve disease is aortic valve replacement. In this, surgeon removes the aortic valve and replaces with an artificial valve prosthasis, bioprosthetic valve, homograft or autograft. In young patients and in women and in elderly people it is better to have the valve replaced by human valve (homo-graft) or by the patients won pulmonary valve (Autograft). This operation with homograft or autograft replacement can not be done in all centres. It can be done only in some centres where the surgeon is experienced and homograft valves are available. The advantages are that this is a natural valve and is expected to function normally. The autograft or the patients own valves is also likely to grow in children and be free of complications and is therefore the best choice for Aortic valve replacement.

In most centres the aortic valve is replaced by an artificial valve (prosthetic valve). Againthe patient needs to be on anticoagulant medication throughout his life after this operation. It is also necessary to take precautions to avoid infection. Prosthetic valve replacement is twice as expensive as homograft or autograft valve replacement.

Tricuspid Valve Repair

The Tricuspid valve is almost always repaired. It does not require replacement. However, minor degree of leakage may persist even after repair.

COMPLICATIONS

In good centres generally there are no complications. However, because of the nature of the operation and because the heart has to be stopped for a period of time there is a risk to life. This risk may be anywhere between 3 to 15% depending on the type of operation performed. For closed mitral valvotomy the risk to life is less than one per cent. For Mitral valve replacement risk to life is 6-8. For Mitral valve repair it is 4-5%. For Aortic valve replacement it is 3 to 5% and for double valve replacement (Aortic and Mitral Valve) it is 10-12%. In patients who require replacement of Aortic and Mirtal valve and repair of Tricuspid valve risk to life is 15%.

Patients may also develop bleeding soon after surgery and may require to be returned to the operating room for stopping bleeding. Damage to brain may cause prolonged unconsciousness and paralysis. Damage to the lungs, liver and kidneys is also possible in the operation. These complications are less in patients who come before heart failure has set in. It is therefore; better to get the operation done at the earliest opportunity after the diagnosis is made.

A 60% decline in stroke and 50% decline in coronary artery disease (CAD) mortality over the past 25 years have been documented in the United States, with similar decreases in Finland and many other western countries. The dramatic decline in cardiovascular diseases (CAD) in the West is attributed to aggressive modification of lifestyle by the entire population rather than to high-tech hospital care of individual cardiac patients. Several interventions have been proven or shown likely to alter the risk of CVD. These include those dir5ected at cigarette smoking, diet, and low density lipoprotein (LDL), hypertension, platelet adhesion, diabetes, physical inactivity, high-density lipoprotein, (HDL), triglycerides, obesity, and estrogen replacement therapy. An epidemic of CAD is underway in India. Life style modification for the entire population as part of a population-based strategy offers the best hope of arresting and reversing the epidemic of CAD among Indians. This strategy aims to reduce the smoking rate and lower the serum cholesterol and blood pressure levels of the entire population by emphasizing the perils of tobacco abuse, the importance of consumption of healthy foods, and the need for regular exercise. This strategy is more likely to be practical and successful in India than the extensive use of expensive medical technology, which is beyond the reach of the overwhelming majority of Indians.

The rise and fall in the CAD mortality in the Western world in the latter half of the twentieth century correlates directly with changes in lifestyle in the society, rather than changes in the genetic pool. In India, similar lifestyle transformation with enormous significance is taking place; the middle class is undergoing tremendous changes in lifestyle and socioeconomic factors such as acquiring cars and consuming increasing amounts of alcohol and tobacco. Eating patterns are also changing rapidly with greater use of fast foods, meats, and fats. These changes are leading to sedentary habits and increased consumption of unhealthy foods. An epidemic of CAD is already underway in India because of these factors. Experiences in the West clearly show amazing success in reversing the CAD epidemic through aggressive modification of lifestyle. For example, the age-standardized mortality rate (SMR) for CAD in the US has declined by more than 50% in the past 25 years. Of this decline, about 30% is attributed to a modest reduction of serum cholesterol level in the entire American population, mainly through reduced intake of saturated fat. About 24% of the decline is attributed to reduction in smoking and 8% to the treatment of hypertension. Other advances in treatments such as coronary care units, cardiopulmonary resuscitation, coronary angioplasty and bypass surgery have had only a small impact. This reviews discusses the various aspects of lifestyle modification, which can be implemented immediately with minimal cost in India.

Cardiovascular Risk Factors –Priority for Intervention

The 27th Bethesda Conference on Matching the Intensity of Risk Factor Management with the Hazards for Coronary Disease Events is a landmark document that offers clear guidelines for management of risk factors. The priority of intervention for the various risk factors according to the degree of benefits is given in Table 1.

Crucial Role of Cigarette smoking in Cardiovascular Diseases

Cessation of smoking is given the top priority. The US Surgeon General's report in 1964 first established the epidemiological relationship between smoking and coronary artery disease. The 1989 Surgeon General's report presented definitive data from observational, case-control, and cohort studies, that smoking increases cardiovascular disease (CVD) mortality by 50%. More importantly, a linear relationship exists between cardiovascular risk and cigarettes consumed. An average smoker dies 3 years earlier than a non-smoker, and a person at "high risk" for CAD (due to other risk factors) dies 10 to 15years earlier if he or she

smokes. A patient who continues to smoke after a myocardial infarction (MI) has an increased risk of death and reinfarction ranging from 22 to 47%. Smoking has clearly been implicated in bypass graft atherosclerosis and thrombosis. Continued smoking after a bypass graft is associated with a 2-fold increase in the relative risk of death and MI.

Smoking damages the cardiovascular system in at least two ways. One is a short term effect produced by agents that have an immediate effect on the circulatory system, probably related to the thrombotic effects of smoking and vasoconstriction caused by nicotine. Smoking exerts its thrombogenic effects by inducing an elevation in blood fibrinogen concentration, enhancing platelet reactivity, and increasing whole blood viscosity by inducing secondary polycythemia. Smoking also accelerates the atherogenic process both in duration and a dose-dependent fashion. The second is a long-term effect, mostly determined by cumulative consumption, probably involving an increased rate of atherosclerosis that may be irreversible. Smoking promotes oxidation of LDL and lowers HDL. Recently, a 10% decrease in HDL levels in children of smoking parents has been reported. Monoclonal proliferation of vascular smooth muscle cells induced by different components of cigarette smoke or their metabolites is yet another mechanism.

Economic burden of Active and Passive Cigarette Smoking

In the US smoking is the leading preventable cause of death killing, 420,000 smokers annually. The total cost of medical services for smokers amounts to 50 billion annually, with another 50 billion in lost wages due to morbidity and mortality associated with smoking. Environmental exposure to second-hand smoke accounts for another 40,000 CAD deaths annually. Parental smoking is an important preventable cause of morbidity and mortality of American children; it result5s in an annual direct medical expenditure of 4.6 billion and loss-of-life cost of 8.2 billion. These startling data provide additional reason for elimination of smoking by parents of young children. No wonder the US tobacco companies were eager to reach an agreement to pay 368.5 billion in return for immunity from litigation in the US courts and to cover the cost associated with the treatment of tobacco-related health problems!

Economic and Health Benefits of Smoking Reduction

Clinical data accumulated over the past 20 years strongly suggest that smoking cessation reduces the of CVD events. The impacts of smoking cessation accrue rapidly when heart

disease and stroke are considered. The excess risk of MI or stroke falls by 50% within the first 2 years after stopping smoking. The decline in risk of overall CVD mortality is greatest in the several months after smoking cessation and continues to decline more gradually over the ensuing several years. Early benefit may derive from improving the prothrombotic state or result of retarding or reversing the progression of atheromata.

In the US, it has been estimated that with a 1% reduction in smoking prevalence there would be 924 fewer hospitalizations for MI and 538 for stroke, resulting in an immediate savings of 44 million, in the first year alone. A seven year program that reduces smoking prevalence by 1% reduction in smoking prevalence there would be 924 fever hospitalizations for MI and 538 for stroke, resulting in an immediate saving s of 44 million, in the first year alone. A seven year program that reduces smoking prevalence by 1% per year would result in a total of 63,840 fewer hospitalization for Mi and 34,366 fewer for stroke, resulting in a total savings of 3.20 bi8llion in costs and would prevent 13,100 out-of-hospital cardiac deaths. Creating a new nonsmoker reduces anticipated medical costs associated with MI and stroke by 47 in the first year and by 853 during the next seven years. This degree of smoking reduction was successfully achieved in California, where the Proposition 99 Anti-tobacco Education Program accelerated the historical decline in smoking prevalence by 1% per year for the past seven years. A some what similar reduction in smoking was achieved by raising taxes on tobacco in Canada, where most adolescent children cannot afford to buy a pack of cigarettes that cost more than $4.

Differing Trends in smoking rates in East and West

Cigarette smoking has been declining in North America and Western Europe, but rapidly increasing in Eastern Europe and Asia. For example, over the past 30 years, the smoking rate in the US has come down from 55% to 29% in men and from 33% to 23%in women. America cigarette exports have grown 260% in the past decade, more than 40% of US tobacco exports are sole in Asia, where smoking rates remain high. Three-fourths of the Vietnamese men and two-thirds of the Chinese and Japanese men are cigarette smokers. Cigarette smoking is an important health problem in India where tobacco is used in many different ways such as bidi, Zarda, Gutka, huka, and betel leaves, in addition to cigarettes.

The decline in cigarette smoking in the US has recently leveled off and some preliminary data suggest more American girls than boys are smoking cigarette. President Clinton has initiated a national campaign to reduce the smoking rates in children by 60% in the next 10

years. His proposal includes an increase in price and or tax of $1.50 per pack to make cigarettes unaffordable to children. There is an urgent need for such an initiative in India since tobacco use typically begins in childhood and nonsmoking children rarely become adult smokers.

Table 1: Cardiovascular Risk Factors. Priority for Intervention

Class 1: Factors for which interventions have been proved to lower coronary artery disease risk.
Class 2: Factors for which interventions are likely to lower coronary artery disease risk.
Class 3: Factors that, if modified, might lower coronary artery disease risk.
Class 4: Factors that cannot be modified or for which modification would be unlikely to lower coronary artery disease risk.

Class 1 Cigarette Smoking High LDL Cholesterol High fat/cholesterol diet Hypertension Left ventricular hypertrophy (LVH) Thrombogenic factors	Class 3 Psychosocial factors Lipoprotein (a) Homocysteine Oxidative stress No alcohol consumption.
Class 2 Diabetes mellitus Physical inactivity Low HDL cholesterol High triglycerides; small, dense LDL Obesity Postmenopausal status (women)	Class 4 Age Male gender Low socioeconomic status Family history of early-onset CVD

Intervention Strategies for Smoking Cessation

Although more than 80% of smokers are aware that smoking has adverse health affects. Many underestimate the hazards. The different intervention strategies include self-help, physician advice or nurse counseling to quit, and pharmacological therapy such as transdermal nicotine patch, nicotine gum, clonidine, etc. These interventions can be combined for better results. The smokers may be motivated to quit if the self-help materials such as those prepared by the American Heart Association, American Lung Association and American Cancer Society are widely disseminated. It is estimated that 90% of Americans who successfully quit smoking do so using individual methods of smoking cessation rather than organized programs. This does not imply that other types of smoking cessation interventions are not necessary. Organized smoking cessation programs may be more

helpful for heavy smokers. Most smokers who quit using self-help strategies may relapse, but this should not be viewed as a failure. It may be reassuring to know that, on an average, smokers attempt to quit three or four times before they maintain abstinence. Predictors of the outcome of smoking cessation efforts are Motivation to quit; intention to quit; confidence in quitting; and degree of nicotine addiction. The doctor's role in encouraging the patient to quit cigarette and beedi is given in Table 2.

Table 2 : Physician Counseling guidelines for smoking Cessation

- Ask about smoking at every clinical visit
- Advise all smokers to stop
- Assist the patient by advising a quit date and providing self-help materials
- Arrange follow-up visits to assess smoking status and encourage continued abstinence

Dietary Modifications

The results of 50 years of intensive research worldwide support the conclusion that diet is the major environmental cause of atherosclerosis and cardiovascular diseases. A high caloric density of diet, often due to high fat content combined with limited physical activity, contributes to obesity, insulin resistance, and dyslipidemia. All these abnormalities increase the risk of CAD. Salt intake in susceptible persons is associated with elevated blood pressure, the foremost risk factor for stroke.

Dominant Role of Serum Cholesterol in CAD

Elevated serum cholesterol level is the strongest risk factor for CAD. The mean level of cholesterol in umbilical blood of newborns worldwide is 75 mg/dl, which rises to 150 mg/dl in two weeks and remains at that level until approximately 20 years of age, when it starts to gradually rise again in most populations. However, in most native African, Latin American, and many Asian populations, the serum levels of cholesterol do not rise. These groups have a virtual absence of CAD. For example, despite a very high prevalence of hypertension and cigarette smoking, the Cad rate in China is one eighth, and Japan one tenth, the CAD rate of the UK. Yet, a 1% difference in the level of serum cholesterol results in a 3% difference in the risk of CAD even within the desirable range of cholesterol level. For an extreme example, an increase in blood cholesterol from 147 mg/dl to 182 mg/dl among Chinese in Shanghai is associated with a 4-fold increase in CAD. The difference in the levels of serum cholesterol ranging from 116 mg/dl in rural China to 235 mg/dl in urban UK offer the best

plausible explanation for the 13-fold difference in CAD rates among different population. Therefore, the optimum level of cholesterol appears to be 150 to 160 mg/dl, especially for Asians, much lower than the 200 mg/dl considered desirable in the Western society.

Fats and Fatty Acids

Contrary to common belief, the contribution of dietary cholesterol to serum cholesterol is small (<10 mg/dl). The average adult American and European consume daily about 500 mg of cholesterol (about the size of five toothpicks), which is hardly a calorie. On the contrary, dietary fat contributes to as much as 100 mg/dl of serum cholesterol. Fats are substances composed of a combination of fatty acids, which are classified as saturated (SAFA), monounsaturated (MUFA) or polyunsaturated (PUFA), depending upon the location and number of double bonds. ^Dietary excess of SAFAs is the largest contributor to serum cholesterol worldwide. SAFAs suppress LDL receptor activity, resulting in marked elevation of LDL. Substitution of 1% carbohydrate calories with SAFAs raises cholesterol by approximately 1.5 mg/dl, whereas PUFAs and MUFAs lower it by 0.5 mg/dl. SAFAs also increase HDL, and PUGAs decrease it; MUFAs are neutral on HDL.

Table 3 : Atherogenicity, Thrombogenicity and Percentage of Fatty Acids In Common Cooking Oils and Dietary Fats

Oil/ fat	PUFA	MUFA	SAFA	LDLResponse in mg/dl	Index of Atherogenicity	Index of Thrombogenicity
Coconut Oil	2	6	92	+36	13.63	6.18
Palm Oil	10	40	50	+12	0.88	1.74
Olive Oil	11	72	17	-15	0.14	0.32
Soybean Oil	61	24	15	-24	NA	NA
Peanut Oil	32	50	14	NA	NA	NA
Corn Oil	59	28	13	-24	NA	NA
Sunflower Oil	96	19	12	-30	0.07	0.28
Safflower Oil	78	13	9	-30	NA	NA
Canola Oil	32	62	6	-25	NA	NA
Beef fat	2	39	51	+5	0.72	1.06
Pork fat (lard)	10	45	51	0	0.60	1.37
Butter fat	3	28	69	+5	2.03	2.07
British Diet	NA	NA	NA	NA	0.93	1.21

The current US caloric intake averages 34% total fat, 14% MUFA, 12% SAFA, and 6% PUFA. The recommended fat intake is <30% of the calories, with up to 15% from MUFA, up to 10% from PUFA and the remainder from SAFA (< 10% in Step I and <7% in Step II diet). Since the

average consumption of fat in India is much lower than in the West (about 20-25% of the calories), this dietary guideline may be too liberal. About 7% to 8% of calories from each of the three fatty acid categories appear to be more appropriate for Indians.

Atherogenic Effects of SAFAs

Whereas serum cholesterol is the strongest risk factor for CAD, SAFAs are its largest contributor. Therefore SAFAs are the essential predisposing factor fro CAD, acting through both atherogenic and thrombogenic mechanisms. However, only three SAFAs with chain lengths 12-16 have cholesterol-raising properties. These are lauric acid (C12:0), myristic acid (C14:0) and palmitic acid (C16:0). These three fatty acids account for only 25-30% of the total fat but 60-70% of SAFAs, in Western diets. Palmiticx acid is the principal SAFA in palm oil and animal fats. Myristic acid is the most powerfdul cholesterol-raising SAFA, being capable of raising LDL from 70 mg/dl to 200 mg/dl. The cholesterol-raising ability of myristic acid is 50% more, and lauric acid is 33% less, than that of palmitic acid, the most common fatty acid in human diet. Replacement of 1% of energy from carbohydrate by 1% energy from myristic acid raises blood cholesterol by 2.3 mg/dl, compared to 1.5 mg/dl with palmitic and 1.0 mg/dl with lauric acid. Most of the risk in cholesterol is due to an increase in LDL, the respective contribution from HDL being 0.8 mg/dl, 0.4 mg/dl and 0.6 mg/dl. 13 Lauric acid is the principal SAFA in coconut and palm kernel oils, both containing 49%. The major sources of myristic acid are butter, coconut oil, and palm kernel oil, each containing about 18%. These three fats are more atherogenic and thrombogenic than lard and beef tallow, the latter two containing only 2 to 3% of myristic acid. Atherogenicity and thrombogenicity and the percentages of various fatty acids in common cooking oils are provided in Table 3.

Antiatherogenic effects of MUFAs

Diets high in MUFAs (loeic acid C18”1) restore LDL receptor activity and lower cholesterol. In Mediterranean countries, the high intake of MUFAs in the from of olive oil is inversely related to CAD as well as total mortality. A diet high in oleic acid generates LDL that is resistant to oxidation, and decreases thrombogenicity by lowering Plasminogen Activator Inhibitor (PAI-I) levels. Olive oil and canola oil are rich sources of MUGA, but meat and dairy products, which are also rich in SAFAs, provide most of the MUFAs in the Western diet. Mustard oil is high in MUFAs but also high in erucic acid, which is known to have toxic effects on the hea.

Antiatherogenic Effects of Omega-…… PUFAs

Linoleic acid (C18:2), the major fatty acid in omega-6 PUFAs, inhibits the hepatia synthesis of apo B-xontaining lipoproteins and lowers serum cholesterol by 2.0 mg/dl for every 1% of the SAFA calories substituted. Between 1963 and 1990 the CAD mortality rate in the US declined by 54%. About a third of this decline is attributed to an increase in the consumption of PUFA from 3% to 6% of the calories, resulting in a 6% to 8% decrease in population level of serum cholesterol. Vegetable oils such` as soybean, corn and cottonseed are primary sources of -6 PUFA and its average consumption in the Western diet is 6% to 8% of the calories. The two undesirable effects of PUFA are lowering of HDL and increased susceptibility for peroxidation.

The Anti-thrombogenic Effects of Omega-3 PUFAs

The principal effect of increased intake of omega-3 PUFAs is antithrombogenic, whereas that of omega-6 PUFA is antiatherogenic. Dietary consumption of omega-3 PUFAs reduces platelet and monocyte reactivity, lowers blood pressure, and lowers blood levels of triglycerides and homocysteine. Rich sources of omega-3 PUFAs include walnuts, canola oil, soybean oil, and fatty fish (sardine, salmon, mackerel, etc.). Recently an intake of one fatty fishmeal per week such as 80 g of salmon was shown to reduce the risk of primary cardiac arrest by 50%. In a secondary prevention trial of 605 French men recovering from MI, there was a 70% reduction in total and cardiac death during a follow-up of 27 months in those who received an experimental “Mediterranean diet” using canola oil-based margarine, enriched with omega-3 PUFA. Since a very high ratio of omega-6 to omega-3 PUFA increases thrombogenicity, a ration of 4 to 55 appears to be optimum in reducing the risk of CAD.

Table 4 : Eating Pattern: General Guidelines (DHHS/AHA/NCEP)

- Avoid too much fat, saturated fatty acids, and cholesterol
- Eat foods with adequate complex carbohydrates and starch
- Avoid too much simple sugar
- Avoid too much sodium
- If you drink alcoholic beverages, do so in moderation
  - 1 drink for women
  - 2 drink for men
- Eat a variety of foods
  - Dietary energy (calorie) levels needed to reach or maintain a desirable body weight and waist.
  - An average of 30% of total calories or less from all fat (20-25% for Indians)
  - Less than 10% of total calories from saturated fatty acids (7-8% for Indians)
  - Less than 200 mg of cholesterol per day

Atherogenic Effects of Trans Saturated Fatty Acids (TRAFAs)

Hydrogenation of vegetable oils converts some PUFAs to TRAFAs (elaidic acid), which have a significant adverse effect on lipoproteins. These include a decrease in HDL and increases in LDL, triglycerides, and lipoprotein (a). TRAFAs in the West are doughnuts, Danish pastry, fried chicken, vegetable shortenings, and hard margarine. Butter contains 60% SAFAs, whereas stick margarine contains only 16% TRAFAs and the tub form even less. Therefore, the fat spread of choice is soft margarine. The impact of TRAFAs on the American diet is small as it contributes only 2% of the calories, but may be higher in those developing countries where unhealthy margarine containing 50% TRAFAs is sold. Therefore, the TRAFA content of vegetable ghee and other hydrogenated fats in India deserves scientific scrutiny.

Antioxidants

Extensive laboratory data indicate that the oxidative modification of LDL accelerates the atherogenic process by recruiting monocyte macrophage, stimulating autoantibodies, stimulating LDL uptake by macrophages and increasing vascular tone and coagulability. Iron, copper, zinc and SAFAs all are likely to increase oxidative potential, whereas antioxidants, both in vivo and in vitro, can increase LDL resistance to oxidation. In epicemiological studies, diets high in vitamin C, vitamin E and beta-carotene are protective against coronary heart disease with the clearest effect for vitamin E Many other dietary and non-dietary factors may reduce LDL oxidation, including selenium, estrogen, flavenoids, magnesium and monosaturated fat.

The value of vitamin supplementation continues to be unresolved. Although the Finnish Alpha-Tocopherol Beta-Carotene Cancer Prevention Study failed to lower the CAD risk among middle-aged male smokers, the Cambridge Heart Antioxidant (CHAOS) Study has clearly demonstrated a 47% reduction in recurrent clinical events in CAD patients who received Vitamin E at a daily dose of 400 to 800 IU. Despite these uncertainties, about 44% of the cardiologists in the US routinely take antioxidants and 37% of them recommend it to their patients. In addition, 28% of these cardiologists take both antioxidants and aspirin, virtually all of them prophylactically. Only 2% of them had CAD and the remainder had a very low risk factor profile.

Food for the Heart

The greatest reductions in the risk of CAD can be achieved by lowering the serum cholesterol level by reducing the intake of SAFAs. This goal is best accomplished by avoiding

butter and ghee, replacing full fat milk with skim milk, and consuming less of dairy fat, and more of fatty fish and fiber-rich foods. No more than tow servings a day of lean meat and shellfish may be used. One serving is 2 oz – the size of a woman's palm or a deck of cards. In-creased consumption of MUFAs such as canola (low erucic acid), olive, high-oleic varieties of safflower or sunflower oils is advisable. Unless and until a beneficial effect is clearly demonstrated, the liberal use of palm oil and coconut products should be discouraged, especially in those whose diet contains other sources of SAFAs. However, in those with negligible intake of fish, meat, milk and dairy fat, modest use (>5% of the calories) of such oils may be preferable to no fat at all. Anti-thrombogenic effects of omega-3 PUFAs may be critically important in the middle-aged to prevent heart attacks. This is hest accomplished by the weekly or bi-weekly use of fatty fish (like salmon, mackerel, farm-raised catfish, etc). Consumption of fish is preferable to taking large number of fish oil capsules. Similarly, daily consumption of five or more servings of fruits and vegetables would provide most of the necessary antioxidants and are preferable to vitamin supplements.

Several studies have shown that a diet rich in fruit and vegetables and low in SAFAs and high-fat dairy products can substantially lower blood pressure. Since fruits and vegetables are rich in potassium, a liberal intake of these foods can b e recommended for the prevention and treatment of hypertension, especially in those who are unable to reduce their intake of sodium. Good sources of potassium include bananas, orange, backed potato, beans, fish, and dairy products, While you can get an overdose of potassium from pills, you can't get an overdose of potassium from food. General dietary guidelines are given in Table 4.

Physical Activity and Exercise

Physical inactivity has recently become a major target of preventive medicine. Approximately 12% of the premature mortality in the US is attributable to physical inactivity, which is associated with at least a 2-fold increase in the risk for CAD. It is difficult to measure physical activity, and consequently, it is difficulty to quantify the relationship between the amount of exercise and CAD risk. Nevertheless, over 50 studies have established that physical activity, either on the job or during leisure time, reduces the risk of CAD events in both men and women. Reduction in the risk appears to be greatest between non-active and moderately active individuals. Less benefit occurs with increases from

moderate to extreme amounts of total energy expenditure. Although any physical activity appears to be of benefit, those activities of higher intensity (>7 Kcal/min) such as brisk walking. Jogging, or heavy gardening appear to be more protective.

Studies have suggested that the “blood thinning” effects of exercise provide another reason for patients to stay active. Exercise probably exerts its beneficial effect through a variety of direct and indirect mechanisms. Physical training improves the myocardial supply/demand relationship, lowers triglycerides, raises HDL cholesterol, lowers blood pressure, decreases platelet aggregation and improves other clotting factors. Studies of exercise in both animals and humans with established coronary atherosclerosis demonstrate slowing of atherosclerotic progression, and in some circumstances, actual reversal of the process. Several meta-analyes of randomized trials support a 20 to 30% reduction in coronary disease deaths with regular aerobic exercise. In addition to preventing heart disease, exercise can also lower blood pressure and prevent stroke, diabetes, osteoporosis, osteoarthritis and possibly colon cancer and breast cancer. The following guidelines will help you stay physically active without much exertion.

- Take a walk after dinner instead of watching television.
- Take an activity break at work-get up and stretch, walk around and give your muscles and mind a chance to relax.
- Take the stairs in lieu of the elevator.
- Walk instead of driving short distances
- Use fewer labor saving devices, doing housework or yard work
- Become more physically active throughout the day.

The Role of the Physician in Increasing Physical Activity

Sedentary living is a serious and pervasive health problem. Getting patients moving is always a challenge. Many sedentary patients would like to become more active but do not know how to begin. Perceived lack of time is the most commonly cited barrier to participation. Many persons can readily insert shorter bouts of lifestyle activity into busy schedules. Remember that doing something is better than nothing.

The American College of Sports Medicine, Center for Disease Control, and the US Surgeon General have jointly recommended that “Physicians should routinely counsel sedentary patients to accumulate 30 minutes of moderate-intensity activity, equivalent to walking at 3

to 4 mph, for most healthy adults – on most, preferably all, days of the week. "Many physicians do not feel adequately prepared to prescribe exercise to their patients. The counseling style for exercise has to be empathetic, supportive and encouraging. Once the stage is set, it is best to establish small, attainable initial goals with the patient. The prescription should be very specific, achievable, and realistic (Table 5). Recording prescribed physical activity in patient charts can help with follow-up office visits, since despite the best intentions, many persons who begin an exercise program will not stay with it over time.

Obesity

Obesity is one of the most inconsistent risk factors of CAD. Obesity is a predictor of CAD in univariate analysis, but not when serum lipids, diabetes and hypertension are included in multivariate analyses. Analysis of the relationship is difficult because obesity itself predisposes to diabetes, hypertension, decreased HDL and in creased triglyceride concentrations. Obesity also predisposes one to avoid exercise. The current availability of highly palatable, calorically dense foods combined with a sedentary lifestyle promote weight gain. If the daily caloric intake exceeded expenditure by 5%, the result would be an increase of approximately 20 lbs. in a year. Obesity in childhood appears to increase the risk of subsequent morbidity, whether or not obesity persists into adulthood. Obesity is a cholesterol-dependent risk factor, not an independent risk factor. The total number of calories is an important factor from the standpoint of body weight, but fat calories or fat grams is the important item from the standpoint of atherosclerotic disease. Snacks just before bedtime are rarely fat or calorie-friendly except that fruit snacks are permitted for those who take an early dinner (typically Americans eat dinner between 5:30 and 6:30 P.M.). Smoking and alcohol consumption are inversely associated with adiposity. No study has specifically examined the effect of weight loss or the type of weight loss on coronary artery disease events. However, deficit of 500 to 600 Kcal per day is usually well tolerated. A combination of diet and exercise is more effective than either alone.

Although a body mass index (BMI) of >27 is considered normal, recent data suggest that a BMI of > 22 is the optimum. An increase of BMI from > 21 to > 27 is associated with a 3-fole increase in risk of CAD in women and a 6-fold increase in risk of diabetes in men. There is no better monitor for fat consumption than body weight. Distribution of body fat rather than the absolute weight appears to be a more important predictor of heart disease, with truncal

distribution of body fat being worse than peripheral distribution. Visceral (central, abdominal, or apple-type) obesity, which can be quantified by the waist to hip ratio (WHR), is a precursor to insulin resistance and hypertension. This pattern has been shown to markedly increase CAD risk. The desirable WHR is < 0.9 for men and <0.8 for women. It is uncertain if hip circumference yields any useful information, waist circumference as the sole measurement has been recommended. The metabolic complications of obesity increase when the waist circumference is >80 cms in women men and > 94 cms in men. The risk is substantially increased if this circumference is > 88 cms in women and > 102 cms in men.

Postmenopausal Status

Although CAD develops about a decade later in women than in men, more women than men die from CAD in the US. Endogenous estrogen not only protects against the development of coronary atherosclerosis, but also prevents plaque rapture and MI. Estrogen might also have a direct effect on the vessel wall. Case-control and cohort studies suggest that postmenopausal estrogen replacement result in a 50% reduction in the risk of developing CAD. The reduction risk is even greater fro subsequent coronary events among women with established coronary disease. Estrogen replacement therapy (ERT) raises HDL and lowers LDL, although it modestly elevates serum triglycerides. ERT can lower elevated lipoprotein by 50%. Since Indian women have high rates of CAD and high levels of lipoprotein , ERT should be given strong consideration in postmenopausal women with or at high risk of CAD.

Alcohol

Moderate alcohol consumption – one or two drinks per day appear to reduce the risk of Cad by 40 to 50%. All alcoholic beverages (wine, beer and spirits) probably protect against CAD and additional protection by specific beverages are minor. Beer and wine appear to have the same protective effect, and red wine is not more protective than white wine. The apparent differences in benefits in different studies are related to user traits and drinking patterns. For example, men drink more than women. American men are more likely to drink liquor or beer, whereas some women are more likely to drink wine. Wine drinkers have the largest proportion of whites and college graduates and the smallest proportions of smokers; beer drinkers are generally heavier drinkers, and persons with high drinking variability.

Mechanism of benefits of Alcohol

Among populations with high cholesterol and fat intake, such as the French, wine consumption is more strongly related to reduced risk of CAD than is total alcohol consumption. Both red wine and grape wine inhibit platelet activity in vivo, lending credence to the idea that some derivative of grape juice is beneficial.

Alcohol appears predominantly to reduce the incidence of Mi and sudden cardiac death and has less effect on the incidence of angina pectoris. This may be due to alcohol's effect on vascular reactivity or on homeostatic factors, thereby reducing acute events rather than reducing coronary atherosclerosis progression. About 50% of the benefits of alcohol are due to raising HDL (both HDL2 and HDL3 subtypes). However, the benefits appear to be limited to those with low HDL (<40 mg/dl) or high LDL (>200 mg/dl). An additional 18% of the cardiac protection is attributable to a decrease in LDL, but this is counterbalanced by a 17% increase in risk due to increased systolic blood pressure. The regular use of three or more drinks of alcohol per day is an important risk factor for hypertension. As much as 10% of hypertension is attributable to alcohol, more than all the secondary causes of hypertension considered together. Thus, although alcohol is a vasodilator at low doses, it is a pressor at high does.

Dangers of Alcohol Excess

There is no doubt that heavy alcohol use is harmful because of adverse effects on the lover, heart (alcoholic cardiomyopathy, hypertension and arrhytghmia), and other organs. The adverse effect of alcohol appears to be more concentrated in people of low socio-economic status. In addition, alcohol consumption tends to occur along with other activities like smoking cigarettes, which has implications for the heart and other organs. Heavy drinkers are more likely than teetotalers to smoke cigarettes and engage in unhealthy behaviors, which consequently increases the risk to death from all causes. Alcohol intake is hard to measure and information about its consumption obtained at any one time may be misleading as a measure of long-term alcohol exposure.

The recent documentation of a decrease in longevity in Russia is attributed to alcohol abuse. Russians have one of the highest annual per person alcohol consumption rates (14 liters), and they often engage in binge drinking, a behavior that increases the risk of stroke, cardiac arrhythmia, and fatal alcohol poisoning. A recent Finish study has shown that men who

drink 6 or more bottle of beer at a time hve a substantially higher risk of death (3-fold higher all-cause death: 7-fold higher death from violence, suicides, injuries, poisoning; and 7-fold higher fatal MI) after adjusting for age and total alcohol consumption. While the choice of whether or not to drink alcohol remains a personal one, the following guidelines may be helpful:

- People who do not drink at all should not start.
- Light to moderate drinkers should feel sate in continuing one or two drinks per day, but never more than three drinks at a time.
- Heavy drinkers should reduce their intake of alcohol, and if they cannot control their drinking, they should stop altogether.

In France, renowned for liberal consumption of wine and no guidelines for drinking, the following motto has been popular; “One glass is OK. Two glasses is too much, Three glasses spell trouble. “Drinking should not be anyone’s main strategy for preventing CAD.

Dramatic Reduction in CVD Rates from Lifestyle Modification : Lessons from Finland

In the north Karelia and Kuopio provinces of Finland, the CAD mortality declined between 1972 and 1992 by 55% in men and 68% in women. The changes in three major risk factors during the 20 years explained 43% decline in men and 49% in women and underscore the tremendous benefits of smoking cessation, controlling blood pressure, and lowering cholesterol.

In men, the relative role of the three risk factors is as follows: 26% decline in CAD mortality for a 13% decrease in serum cholesterol level; 15% decline for a 9% decrease in diastolic blood pressure; 10% decline for a 16% decrease in smoking. The greater than expected decline in CAD mortality may have been due to modification of other risk factors such as physical inactivity, obesity, dietary pattern, and flavonoid intake, the impact of which were not analysed.

Other Interventions to Reduce CVD Burden

A variety of other intervention have been documented to drastically reduce the risk of CVD. An estimate of benefits from various interventions in reducing the risk of a first Mi is given in Table6. Maintaining ideal body weight, avoiding tobacco and exercising daily can halve the risk of a CVD event. Further halving of the CVD risk can be achieved by a 10% reduction in serum cholesterol and 10% increase in HDL, along with low dose aspirin and estrogen

replacement therapy (in postmenopausal women). The benefits of these interventions appear to be far greater than that of coronary revascularization procedures. Although such procedures are highly effective in relieving severe angina, their impact on preventing MI or death is small. Safe and effective medications are also now available to lower LDL by as much 60%, in those who are unsuccessful was maximum modification of lifestyle.

CONCLUSION

Diabetes mellitus, atherosclerosis hyperlipidemia, hypertension, physical inactivity, obesity, and insulin resistance are highly interrelated. Each is an independent risk factor fro atherosclerotic events, although the pathogenic mechanisms involved are unresolved. The magnitude of the risk reduction with exercise,diet, lipid modification, and smoking cessation is similar to other medical therapies fro CAD such as aspirin, beta-blockers, and coronary bypass surgery. Smoking is perhaps the most important and least genetic of all risk factors. Smoking augments the severity of other risk factors such as dyslipidemia and hypertension and markedly increases the incidence of and mortality from CAD. A prescription for your health is " Live an active life feel better, look better and work better".

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