UNIT 11 NUTRITIONAL MANAGEMENT OF CORONARY HEART DISEASES

Structure

11.1 INTRODUCTION

In the previous unit, you must have realized that nutrition is a basic prerequisite for maintaining a desirable body weight and that diet has a major role in the prevention and treatment of several degenerative diseases. We have learnt that ovetweight can be one of the risk factors Tor cardiovascular disease (CVD). Risk factors as we know are conditions and habits that have been dcmonstralcd to be associated with the increased probability of a disease. We will study about cardiovascular diseases in this unit and learn about the genetic and environmental factors, which lead to elevated serum lipids, elevated blood pressure and development of other clinical manifestations associated with coronary artery disease (CAD). This unit will also elaborate upon the dietary management of various forms of heart disease.

Objectives

After studying this unit, you will be able tn:

- describe the various forms of coronary heart diseases,
- enumerate the risk factors (genetic and environmental) in causation of CHD,
- discuss the etiology, symptoms, as well as, complications of various forms of heart disease(s),
- elaborate on the objectives of dietary management and the nutrition care process, and
- explain the treatment, management and prevention of disease with emphasis on behaviour modification.

11.2 CORONARY HEART DISEASES (CHD)

Coronary heart disease is a broad term comprising of a spectrum of diseases associated with disorders of the circulation, heart muscles or the vessels of the heart in particular. We all come across certain common terms associated with heart disease which are very often used inter-changeably. You must have heard of terms such as coronary heart disease, coronary artery disease, ischemic heart disease being used synonymously. But have you ever realized that they do not mean exactly the same. Well, the term coronary heart disease as discussed above encompasses all the diseases of the heart i.e. those associated with the blood, circulation, as well as, the structure. *Coronary artery disease (CAD) refers to diseases of the arteries, generally resulting from blockage of the arteries.* Ischemic heart diseases (IHD) are usually a consequence of coronary artery diseases such as myocardial infarction is a consequence of progressive atherosclerosis. We shall be using these terms in this unit. The diseases of the heart mentioned above can be congenital or acquired. In this unit, we shall discuss about the acquired forms of CHD i.e. those, which develop as a result of dietary errors or sedentary life-style practices. The major forms include dyslipidemias, atherosclerosis, hypertension, angina pectoris, myocardial infarction, congestive cardiac failure and rheumatic heart disease. The cardiovascular diseases are of prime importance as we see that the incidence of these diseases is rising at an enormous rate are going to account for an appreciable proportion of mortality and morbidity in the adults and now appearing in the young. Table 11.1 highlights briefly the various types of CHD.

Table 11.1: Common disorders and complications of Coronoary Heart Diseases (CHD)

Disorders/Complications of CHD	Definition
Dyslipidemia	Abnormal lipid levels in the blood.
Atherosclerosis	A thickening and narrowing of the walls of the large and medium sized blood vessels caused due elevated to lipids and cholesterol.
Hypertension	Higher than normal blood pressure.
Angina Pectoris	A characteristic pain or discomfort in the chest.
Myocardial Infarction	An area of necrosis (dying/dead cells of the myocardium) in a tissue.
Congestive Cardiac Failure	A clinical syndrome caused by heart disease characterized by breathlessness, chest pain and abnormal sodium and water retention.
Rheumatoid Heart Disease (RHD)	A complication of rheumatic fever and occurs after attacks of this fever.

We begin our discussion on CHD by studying about the prevalence, etiology and pathophysiology of the disease condition.

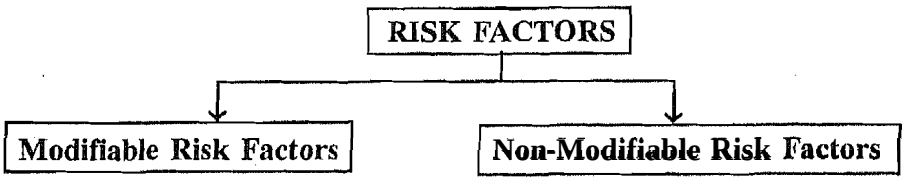
11.2.1 Prevalence

Incidence of obesity, diabetes and CHD is increasing in India in almost all age groups. At present, obesity, which is a major risk factor for hyperlipidemia and atherosclerosis, is present in 14% of the rural and 27% of urban population. This is so because urban population is more prosper'ous, lead a sedentary lifestyle and generally prefer consuming refined foodstuffs. Hypertension (high blood pressure) – a risk factor for IHD is prevalent in 5% rural and 5-15% urban population. Similar is the situation with diabetes mellitus, which is closely associated with cardio-myopathies (diseases of cardiac muscles) such as congestive heart failure.

But why is the incidence of coronary heart diseases increasing at a rapid rate every year? Are there any factors, which may predispose Asians particulary Indians to an early onset of heart diseases? Let us read further to learn about the causative factors associated with the development of heart diseases in various age groups.

11.2.2 Etiology: *Cardiovascular Risk Factors*

It must be coming in your mind several times that why do some people suffer from heart disease while others do not? Well the most obvious reason is that they are more susceptible due to the presence of certain risk factors. Now what is a risk factor and what are the risk factors for CHD? The factors that play arole in causing or increasing the risk of getting the cardiovascular disease are called *risk factors.* Figure 11.1 illustrates these risk factors. As you may have noticed in the figure, these factors are classified as *modifiable* and *non-modifiable* risk factors. *Modifiable risk factors* are those, which we have control over. For example, obesity, smoking, high blood pressure, high cholesterol, physical inactivity etc. By themselves, they are major risk factors, which increase our risk of developing CHD. Positive healthy living, smoke-free air, good nutrition, regular physical activity, and supportive living and working environments can go a long way in preventing CHD. *Non-modifiable risk factors* are those that we have no control over such as hereditary, age, gender etc. We will talk about some of these common risk factors in the following text .



1. *Behavioural*
   - Smoking
   - Sedentary Lifestyle Habits
   - Dietary Errors

2. *Physiological*
   - Hyperlipidemia
   - Hypertension
   - Obesity
   - Diabetes Mellitus
   - Hyperuricaemia and Gout
   - Fibrinogen
   - Platelet Aggregation
   - Lipoprotein (a)
   - Homecystiene Levels
   - Low Birth Weight

3. *Psychological*
   - Stress
   - Low Socio-Economic Strata
   - Type A Personality

4. *Geographic*
   - Soft Drinking Water
   - Cold Weather

1. Age
2. Sex
3. Heredity
4. Endomorphic Body Build

Figure 11.1: Risk *factors for* Coronary Heart Diseases

1. *Family history:* People who already have the disease in their family are more prone to getting heart disease. Genetic factors greatly influence the risk of developing premature cardiovascular diseases.

2. *Obesity:* As you know obesity or excessive weight is the primary cause of cardiovascular disease. It is an independent risk factor for heart disease. Obesity in generally associated with elevated triglyceride, elevated low density lipids, increased blood pressure and impaired glucose tolerance. Weight reduction improves these abnormalities. Keeping the body mass index within the normal range (18.5-24.9) can be helpful in retarding the onset of CAD. Remember we read about BMI in unit 9. Further, android form of obesity makes us more prone to heart diseases as compared to the gynoid form of obesity. Thus, abdominal fat is considered more harmful than fat on the hips as you may recall studying in Unit 9. This can be measured by waist/hip ratio (WHR). Normal WHR is 0.85 for females and 1.0 for males.

3. *Hypertension or high blood pressure:* It is also one of the risk factors of cardiovascular disease and is frequently accompanied by hyperlipidemia (excess lipids in the blood). Increased coronary artery wall tension is believed to accelerate the atherosclerotic process by stimulating arterial smooth muscle cell hyperplasia and hypertrophy with resultant fibromuscular thickening.

4. *Diabetes:* Sustained hyperglycemia is associated with tissue damage and cardiomyopathies. You shall study about diabetes and its associalion with cardiovascular diseases in the next unit. Control of blood glucose levels is important to prevent heart disease.

5. *Age:* Earlier men less than 55 years were more prone but now heart disease has caught up with a younger age-group of 30 years also. In fact, autopsy studies have indicated that the process of atherosclerosis can begin as early as at two years of age and that the sites of blockage may get predetermined in the womb of hyper-cholesterolernic women.

6. *Smoking and tobacco:* Cigarette smoking and tobacco is a major independent risk factor for myocardial infarction and cardiac failure. Coronary artery disease has been seen in 80% of the smokers. Inhaling nicotine, carbon monoxide and various other pollutants narrow the coronary arteries thus reducing the blood flow to the heart muscle. It deserves special attention in the prevention of cardiovascular disease.

7. *Alcohol:* Excessive amount of alcohol is also a risk factor. Alcohol shows a positive relationship between the amount of alcohol consumed and blood pressure levels, hence it is best to avoid it or take it in moderation.

8. *Lack of physical activity*: Sedentary and un-exercised people are more prone to CVD.

9. Syndrome X is a cluster of conditions such as central abdominal obesity, diabetes, dyslipidemia or hypertension with elevated triglycerides, decreased HDL and blood sugar abnormalitiesñall harmful for cardiovascular disease.

10. *Plasma fibrinogen and Lipoprotein(a)*: Plasma fibrinogen is closely associated with blockage in the arteries due to blood clot formation. Serum lipoprotein (a) which is a genetically inherited mutant of plasminogen, is a discriminant marker of early, asymptomatic atherosclerotic plaques in the carotid arteries and aorta of hypercholesterolernic individuals.

11. *Psychological, social, cultural and religious factors* indirectly influence the risk of cardiovascular diseases by their effects on kind of food and quantity of food consumed, cigarette and alcohol consumed. I-Iighly competitive job stress and physical exercise, people who are impatient, workaholic (Type A personalities) can cause greater harmful effects on the heart and its vessels.

In the discussion above we have highlighted the most common risk factors which are associated with heart diseases. It would be good to note here that newer risk factors

are being identified everyday. Risk factors such as viral infections, low birth weight and blood homocystiene levels are currently being investigated to understand the high prevalence rate in the urban poor of the developing nations and the early onset of CHD. Let us now study about the basic disease process in brief.

11.2.3 Pathophysiology of CWD

We all know that heart attack i.e. myocardial infarction is not the beginning but a last stage representing acute clinical manifestation of CHD. Several clinical trials and autopsy studies have indicated that the process of developing atherosclerotic lesions can begin as early as during infancy and that it may take several decades for the lesions to develop into fatty streaks and fibrous plaques that ultimately cause stenosis . (complete blockage) of the arteries. Diffuse intimal thickening during infancy which is considered to be a normal physiological and not a pathological process can result in the initialization of early clinical manifestations which may appear in the smooth muscle cell layer between the endothelium and the internal elastic lamina. These lesions may progress and develop into fatty streaks to reach their maximum extent in the aortas over a period of two decades among individuals having elevated cholesterol and/or triglyceride levels. There is also focal proliferation of smooth muscle cells which are termed as gelatinous lesions because they have a low lipid but high water content. Some of these lesions may become large and develop a grayish opaque center while remaining soft and translucent around the edges. These are referred to as the transitional lesions. These lesions at times develop a fibrous cap with atheromatous lipids in the center and are known as fibrous plaques. Such fibrous plaques may coalesce together resulting in blockage of the arteries and hence reduced flow of blood to the tissues. The irritating presence of plaques may cause injury to the intima of the arteries which may result in thrombosis. Myocardial infarction/cerebral stroke is the ultimate result of stenosis in the arteries.

In the next section, we shall discuss about the role of different types of heart diseases, their symptoms, treatment and dietary management. But before we go on to this topic let us recapitulate what we have learnt so far.

Checlc Your Progress Exercise 1

1. What do you understand by the term CHD? Enlist some major forms of acquired heart diseases.

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2. Enumerate the modifiable risk factors that increase the risk for developing heart diseases.

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3. Enlist the major stages of CHD development.

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4. What do you understand by the term Syndrome X?

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11.3 COMMON DISORDERS OF CORONARY HEART DISEASES AND THEIR MANAGEMENT

In this section, we shall deal with different types of heart diseases, their etiology, symptoms, complications, as well as, nutrition and dietary management goals. We shall begin our discussions with dyslipidemia/hyperlipidemia which are directly and most closely associated with the development of atherosclerosis. Subsequently we shall learn about hypertension, angina pectoris, myocardial infarction, congestive cardiac failure and rheumatic heart diseases. However, the planning of diets for these disease conditions shall be taken up in the Practical Manual (MFNL-005). So here let us begin our discussion with dyslipidemia.

11.3.1 Dyslipidemia or Hyperlipidemia

It has been known for over five decades now that dyslipidemia is associated with increased severity and prevalence of atherosclerosis. Dyslipidemia is frequently seen in the form of increased concentration of either cholesterol or the triglycerides or frequently the both. There are several types of blood lipid disorders which hold different risks and require somewhat different treatment methods. The proportions and total amounts of specific lipoproteins present in the blood are used in diagnosing hyperlipidemia.

The characteristics, as well as, classification of the lipoprotein disorders have been given by *Fredrickson etal (1967).* Also you would recall reading about this in Nutritional Biochemistry Course (MFN-002), in Unit 7. We suggest you to go back to Unit 7 and refresh yourself on the classification of major lipid disorders and their characteristics. However, for a quick recapitulation we have elucidated the characteristics of some important lipoproteins here.

Blood lipids (cholesterol, triglycerides and phospholipids) being insoluble in blood need a ship to travel in our body; it therefore usually gets bound to proteins and form complex particles called *lipoproteins* which vary in size, composition and density. There are five classes of lipoproteins in the blood. These include:

1. *Chylomicrons*: These are formed in the intestines when a fatty meal is taken. These pass into the blood through the lymphatics. It contains nearly 90% of triglycerides and 5% cholesterol.
2. *Very Low Density Lipoproteins (VLDL)*: These are produced by the liver and are the main transporters of triglycerides. VLDL generates most of the LDL in the plasma. It contains about 60% of triglycerides and 10% cholesterol.
3. *Intermediary Density Lipoprotein (IDL):* This is rich in cholesterol and triglyceride. It contains about 40% of triglycerides and 10% cholesterol.
4. *Low Density Lipoproteins (LDL):* This type of lipoprotein is most artherogenic of all the lipoproteins and is responsible for the atherosclerosis in the vessels. It has 10% of triglycerides and 45% of cholesterol.
5. *High Density Lipoprotein (HDL)*: The high HDL content is associated with decrease in the risk of atherosclerosis. It contains about 3% triglycerides and 20% cholesterol.

Besides these lipoproteins, the other parameters, which are of interest in CHD, include:

- *Total Cholesterol:* Serum totnl cholesterol equals the sum of HDL-cholesterol (HDL-c), VLDL-cholesterol (VLDL-c) and LDL-cholesterol (LDL-c). High level of cholesterol is associated with a high risk of atherosclerosis.
- *Triglycerides*: The triglyceride-rich lipoproteins include chylomicrons, VLDL and intermediate density lipoproteins (IDL). Their levels increase by many dietary factors such as excess calories, fat, carbohydrates and alcohol. Indians are

genetically more susceptible to developing hypertriglyceridemia as compared to Europeans primarily due to our cereal based diets.

- *Apoproteins*: Apoproteins are closely related to lipids as they maintain the solution of lipoprotein lipids in plasma. Measurement of apoprotein levels aids in diagnosing disorders of lipids and preventing the risk of cardiovascular disease. The apoproteins include A_I, A_{II}, B_{100}, B_{48}, C_I, C_{II}, C_{III}, D and E. The apoproteins A_I and A_{II} are associated with HDL (the good lipoprotein) and the rest are associated with not so good lipoproteins.

It would be interesting to note here that the clinical reports of most cardiac patients would indicate the levels of the abovc-mentioned parameters. As a dietician, you should be able to interpret and utilize this information for planning the dietary regime of the patient. For your reference, the normal and elevated levels (indicative of disease condition) for some of these parameters are given in Box 11.1.

Box 11.1 Classification of Lipid/Lipoprotein Levels

Classification of Total Cholesterol

Totnl Cholesterol (mg/dl)	Interpretation
< 200	Desirable
200 - 239	Boderline
≥ 240	High

Classification of Triglyceride levels

Triglyceride (mgl dl)	Interpretation
< 150	Normal
150 - 199	Borderline
200 - 499	High
≥ 500	Very High

Classification of HDL-c *

HDL-c (mgl dl)	Interpretation
< 40	Low
40 - 60	Normal
≥ 60	High

Classification of LDL-c**

LDL-c (mgl dl)	Interpretation
< 100	Optimal
100 - 129	Near Optimal
13b - 159	Borderline
160 - 189	High
≥ 190	Very High

Classification of cholesterol: HDL-c*

Cholesterol:HDL-c	Interpretation
3.3 - 4.4	Low Risk
4.5 - 7.1	Average Risk
7.2 - 11.0	Moderate Risk
> 11.0	High Risk

* High Density Lipoprotein cholesterol
** Low Density Lipoprotein cholesterol

Source: National Cholesterol Education Program ATP III report 2005.

Next, we move on to the etiology or the factors leading specifically to hyperlipidemia or specific types of lipoproteins. You would find the factors elaborated below are the same discussed earlier under sub-section 11.2.2.

Etiology

The causative factors of dyslipidemia/hyperlipidemia may be environmental (dietary/ lifestyle), genetic or secondary to certain disease conditions or drugs. We have already studied these but let us briefly touch upon them.

1. *Environmental factors:* These include diets liigh in saturated fats, excessive calorie intake, alcohol intake andsedentary lifestyle. A change in diet and lifestyle can help to normalize elevated blood lipid levels.
2. *Genetic defects:* These lead to familial hyperlipidemia. In such cases, the lipid levels may be alarmingly high and risk of CHD very high. Myocardial infarction can occur within the first two decades of life. Dietary modifications along with drug therapy are beneficial.
3. *Hyperlipidemia secondary to other conditions*: The disorders that upset lipid metabolism include poorly controlled diabetes mellitus, kidney disease (nephrosis and end-stage renal disease), liver disease, hypothyroidism and use of drugs like oral contraceptives, thiazide diuretics, corticosteroids could be a cause of the lipid imbalances.

We will now proceed over to the common symptoms elicited with elevated serum cholesterol and triglycerides levels in the blood.

Symptoms

The main symptom is presence of *xanthoma*. This is a yellowish swelling, nodule or plaque in the skin resulting from deposits of fat. There are various types of xanthorna for example, in type 2b, the xanthoma may be on the hand, buttocks, knees, upper eyelids. In type 3, the planar xanthomas may be seen in the creases of the palms of hands and fingers. In type 5, the xanthoma may be present on the back of the neck or buttocks.

Complications

The complications include:

- Pancreatitis
- Progressive atherosclerosis or asymptomatic coronary disease (we shall be studying about this in the next section).

Excess of triglycerides (hypertriglyceridemia) and cholesterol (hypercholesterolemia) are the usual problems for increase in very low-density lipoprotein cholestrol (VLDLc), low-density lipoprotein cholesterol (LDLc) levels. The following are possible causes of elevated triglycerides and cholesterol and reduced HDLc levels-

Possible causes of elevated triglycerides:

- Obesity
- Uncontrolled diabetes
- Genetic
- Drugs
- Alcohol
- Hypothyroidism
- Liver disease

Possible causes of elevated cholesterol:

- Excess fat in the diet (saturated and cholesterol)
- Overweight/Obesity

Possible causes of reduced Serum HDL levels:

- Cigarette smoking
- Obesity
- Lack of exercise
- Uncontrolled diabetes
- Hypothyroidism
- Hypertriglyceridemia
- Genetic factors
- Drugs (Progestational agents, steroids etc.)
- Liver disease

Keeping the etiology and causative factors in mind let us proceed over to the dietary management of hypercholesterolemia.

Treatment and Management of Dyslipidemia/Hyperlipidemia:

Maintenance of blood lipids within the normal range is the most essential objective to prevent the onset/progression of atherosclerosis. According to the National Cholesterol Education Programme (NCEP), USA it is pertinent to lay stress on dietary and lifestyle modifications to control the manifestations associated with elevated lipids whether or not the patient is on drug therapy. Let us now proceed over to the dietary management of hyperlipidemia and understand the role of nutrients in increasing blood lipids (except HDLc) and triglyceride levels. We will begin with identification of objectives for the nutritional care process.

Goals of Dietary Treatment:

The goals of dietary management (alone or conjunction with exercise or with lipid lowering drugs) are to reduce the total fat, saturated fat and cholesterol intake. This is an attempt to reduce total cholesterol, LDL and triglyceride levels, thus also reducing the risk of atherosclerosis and modifying its progression in subjects with the disease.

We read in the section above that there are several environmental factors apart from genetic attributes that increase the probability of developing dyslipidemia. For example, obesity is a high risk factor and one of the important causes of this is the diet. We know that when we take more food than what we require it is accumulated as fat in our body. This raises the lipid levels in the blood, which deposits in the arteries and the excess calories are converted to fat in the body, which results in excess weight a very important cause of obesity. This when compounded by wrong lifestyles (physically inactive, smoking, intake of alcohol, stress etc.) worsens the already harmful effects of imbalanced food. The fats in the food, as we already know, give twice as much calories (9 Kcal/g of fat) as proteins and carbohydrates (4 Kcal/g of proteins and carbohydrates). So very rich foods containing excess fat are the first culprits and then other nutrients as well could be involved. We will learn about these also. So let us start with dietary fats.

Dietary Fats: There is a consistent support for the hypothesis that both the quantity and quality of fat are directly associated with the elevation of most blood lipids particularly LDLc. It has largely been observed that high intake of fat particularly saturated fat results in elevation of serum total cholesterol particularly LDLc. The foods that we consume contain cholesterol, saturated, monosaturated and polyunsaturated fatty acids. They may be invisible like fats present in various foods or visible like fats used in cooking etc. Let us learn about each of these in detail, We will begin with dietary cholesterol.

a) *Cholesterol:* It is a natural component of foods such as mutton, pork, ham, sausages, lamb, chicken, eggs (yellow), whole milk, cheese, ice-cream, butter and desi ghee. Cholesterol is present only in animal kingdom and does not exist in vegetable kingdom. Increased cholesterol in blood is called *hypercholesterolaemia,* which leads to atherosclerosis.

Foods rich in cholesterol
Mutton, pork, ham, sausages, lamb, chicken, egg (yellow), glandular meat (brain, liver and kidneys), whole milk, cheese, ice-cream, butter, desi ghee.

b) *Saturated Fatty Acids (SFA):* These are found mostly in animal fats as white marble-like solid at room temperature. Red meats are rich in it. Others sources of saturated fats are milk fat, butler, ghee, coconut oil, palm oil, margarine and hydrogenated fats (vanaspati). These saturated fats in the diet also give rise to high LDL, thus leading to atherosclerosis. The three saturated fatty acids lauric acid, myristic acid and palmitic acids increase cholesterol levels. The energy provided from saturated fats should always be < 10 % of the total calories.

Foods rich in saturated fatty acids (SFA)
Milk fat, butter, pure ghee, coconut oil, palm oil, margarine, vanaspati, red meats (mutton)

(c) *Monounsaturated fats (MUFA)*: These are liquid at room temperature, the highest food source being olive oil, canola oil, rapeseed oil, to some extent mustard oil. MUFA is an excellent fat as it reduces the LDL levels and increases the good HDL levels and cholesterol, thus preventing atherosclerosis. Oleic acid is a monounsaturated fatty acid of great clinical relevance.

Oils high in monounsaturated fatty Acid (MUFA)
Canola oil, olive oil and rapeseed oil.

(d) Polyunsaturated *Fatty Acids* (PUFA): These are also liquid at room temperature. There are two main types of dietary PUFA's of significance:

a) linoleic acids (LA/n-6) present in good amounts in safflower, sunflower, corn and sesame oil.

b) Alpha linolenic (ALNA/n-3) fish oils, to some extent olive oil, mustard and rapeseed oil.

The ratio of n-6:n-3 between 5-10 is considered healthy. This can be obtained by using a mixture of two oils. Combination of safflower, corn, sunflower or sesame oil (rich in n-6) with equal portions of mustard oil or rapeseed oil (rich source of n-3) can give a ratio between 5-10. This is not artherogenic and hence healthy for the heart. Fish oils and fish also contain n-3 and are beneficial for the heart as they decrease plasma triglycerides. Table 11.2 enlists the rich sources of PUFA.

Table 11.2: Rich sources of polyunsaturated fatty acids (PUFA)

Rich in n-6 **(Linoleic acid)**	Rich **in n-3** (alpha linolenic acid)
Safflower, sunflower, sesame, corn oil	Canola, olive oil, rapeseed oil, mustard oil, soyabean oil, fish oil, (mackerel, sardines, trout and tuna), wheat, bajra, green leafy vegetables, methi, mustard (rai), almonds, Black gram, cow pea (lobia), rajmah and soya.

Monounsaturated fatty acids have a plasma cholesterol lowering effect. Polyunsaturated fatty acids also have a cholesterol decreasing effect. Omega 3 fatty acids are excellent for the heart as they:

1. reduce platelet aggregation and monocyte adherence
2. modify plasma lipids, and
3. lower blood pressure

To sum up, we now know that saturated fats and dietary cholesterol increase the level of LDL and cholesterol in the blood. If there is a decrease of 1% calories from SFA, the blood cholesterol level decreases by 3 mg/dl. The monounsaturated fatty acids (MUFA) are good as they decrease the LDL levels and cholesterol in the blood. The polyunsaturated fats (PUFA) decrease LDL levels. Thus, the energy provided from fats should remain less than **30%** of the total calories; of which saturated and polyunsaturated fatty acids each should provide less than 10% of the total calories; while mono-unsaturated fatty acids may provide the remaining i.e. > 10% of the total calories.

Table 11.3 gives the composition of various fatty acid in foods to help you gain more knowledge regarding fatty acids. This would also help you to choose the type of foods low in saturated fats and rich in n-6 and n-3 polyunsaturated fats,

Table 11.3: Composition of common fats and oils (g/100 g)

	Satu-rated	Mono-unsaturated	Polyunsaturated n-6	n-3	Predominant Fatty Acids
Coconut	90	7	2	< 0.5	Saturated
Ghee	65	32	2	< 1.0	Saturated
Vanaspati (hydrogenated fat)	24	19	3	< 0.5	Saturated
Red palm oil	50	40	9	< 0.5	Saturated+ Monounsaturated (raw)
Palm oil	45	44	10	< 0.5	Saturated+ Monounsaturated
Oilve oil	13	76	10	< 0.5	Monounsaturated
Groundnut	24	50	25	< 05	Monounsaturated
Rape/Mustard	8	16	12	10.0	Monounsaturated
Sesame	15	42	42	10	Mono and Polyunsaturated
Rice bran	22	41	35	1.5	Mono and Polyunsnturated
Cotton seed	22	25	52	1.0	Polyunsaturated
Corn	12	32	55	1.0	Polyunsaturated
Sunflower	13	27	60	< 0.5	Polyunsaturated
Safflower	13	17	60	< 0.5	Polyunsaturated
Soyabean	15	27	53	5.0	Polyunsaturated

Let us learn about an important type of fat, which has an important place in the Indian diets in view of its characteristic flavour, and excellent shortening properties, which improves the texture, and acceptability of several foods. You must have guessed it right. It is available in the Indian market in the form of vanaspati, which is a type of hydrogenated fat.

Hydrogenated Fat

All vanaspati preparations are the result of hydrogenation of oils, where unsaturated fat is converted to saturated fat for its flavour and long shelf life. This is often preferred by housewives, as it is an imitation of pure ghee. However, it is saturated in nature and contains trans fatty acids. Trans fatty acids, are known to raise LDL in blood thus enhancing atherosclerosis. This is the reason why hydrogenated fats are harmful to the heart.

Next, we will proceed to the type and quality of carbohydrates, which can help in controlling hyperlipidemia. Simple carbohydrates as we know when consumed above the requirements can result in elevated levels of VLDLc and triglycerides.

Carbohydrates: As you have already read that carbohydrates provide 4 Kcal/g of energy in our diets. Since we take large amounts of carbohydrates, these provide 60-70%of our total calorie needs of the body. If taken in excess, it is converted to fat in the body. You would recall reading about different types of carbohydrates, that form an essential part of our diet. Let us brush up our knowledge about these in this sub-section and find out the role of carbohydratesin heart diseases. The dietary sources of carbohydrates are as follows:

1) *Monosaccharides:* You are aware that monosaccharides exist mainly as glucose and fructose in our diets. Fruits, vegetable, honey, jaggery are good sources of monosaccharides.

2) *Disaccharides:* Sucrose (common known as sugar) is the commonest of them all and present in table sugar. Lactose is found naturally in milk and milk products, Maltose is the product of hydrolysis of starch and is found in sprouted wheat and barley.

3) *Oligosaccharides*: These are found in plant seeds mainly legumes, beans and peas.

4) *Polysaccharides*: Starch is one of the main carbohydrates found in our diets and comes from cereals, potato, bananas etc.

Next, let us get to know about their role.

Role of Carbohydrates

It is important to know about these carbohydrates, as they all differ in their digestive properties. The rate of absorption is variable. Monosaccharides gel absorbed the fastest and polysaccharides get absorbed the slowest. This is because polysaccharides contain more fibre. The latter are good for many disorders like intestinal diseases, diabetes and even cardiac problems. Fibre is beneficial for cardiovascular disease and is found as water-insoluble and water-soluble type. The benefits and sources of the same are given in Table 11.4.

Table 11.4: Benefits **of fibre in diet**

Type of Fibre	Water Insoluble (Cellulose)	Water Soluble **(Gums,** pectins, **mucilages)**
Benefits **reduces**	Forms bulk of stool, helps in regulation of bowel movement gives satiety value.	Helps in increasing viscosity of foods, absorption of nutrients, reduces post-prandial plasma glucose increase tissue insulin sensitivity and insulin receptors, reduces serum cholesterol and serum triglycerides. Gives a satiety value.
Source	Whole wheat products, Bajra, Ragi, Maize, Green leafy vegetables, fniits with skin and seeds	Oats, whole pulses, whale grains, apples, pears, citrus fruits and vegetables like potato, bhindi, Sound as pectins, gums or mucilages and isabgol.

Soluble fibers like pectins, gums and mucilages have shown reduction in cholesterol levels. Intake of about 20-40 g of soluble fibre has proven to be beneficial. As you can see, legumes, oats, whole grains, fruits (apples, pears, and citrus fruits), and vegetables along with psyllium (isabgol) are a rich source of soluble fibre. Soyabeans are a good source of fibre and soya proteins have estrogenic effect, which causes of lipid lowering. A recent analysis of 38 completed trials showed the beneficial affects of soya protein to be in the amount of 47g/day.

Proteins: While the quantity of protein does not impose any significant impact on the serum lipoproteins, it is the quality of protein, which may be of significance. Patients should be advised to consume plant origin proteins over those of animal origin. This is in view of the facl that plant origin foods, which are good sources of protein, are generally rich sources of dietary fibre, have low amounts of saturated Eat and are devoid of cholesterol, Egg white and lean meats (meat without fat) should be the preferred options in case of animal foods.

Of late tremendous amount of research is being carried out to find out the potential of micronutrients and trace elements in the treatment, management and prevention of dyslipidemia. Let us brief ourselves on these aspects too.

Vitamins: Antioxidants and flavonoids, natural vitamin E, vitamins C and A are nutrients (vitamins) that scavenge cell-damaging free radicals and act as antioxidants as you may recall studying earlier in Unit 7. It is important to know this in view of the fact that damage through free radicals is quite pronounced among patients with Syndrome X – a risk factor for cardiovascular disease. Vitamin **A** is present in good amounts in green and yellow fruits and vegetables and lycopene in tomatoes and anthocyanin in grapes and berries. Vitamin E rich foods include buck wheat (7.9 μg), corn (5.8 μg), almonds (24.4 μg), sunflower seeds (52.1 μg), spinach (3.0 μg) and soyabcan (93.7 μg). Hence, vitamins (E, C and A) containing foods, bright yellow fruits and vegetables like papaya, orange, mango, strawberry, tomato, carrots and green leafy vegetables like methi and spinach, cabbage, red wines, tea and soyabcan are excellent foods because of their antioxidant properties.

Minerals: The three most important minerals are chromium, zinc and magnesium. These minerals play a critical role in maintaining proper insulin function. Deficiency of these minerals increase the risk of Syndrome X – a risk factor for cardiovascular disease. Excess of sodium intake and lack of potassium have been seen to play an important role in hypertension. Low intakes of calcium can also be a risk for cardiac disorder. Sodium added to the food or sodium-rich foods need to be restricted in cardiovascular diseases.

Antioxidants and Flavanoids: You must have already read about different antioxidants present in our foods. The body makes use of a grcut variety of antioxidants and free radical scavengers for different purposes and to protecl tissues with different needs. Vitamins A, C and E have important antioxidant functions as you have already studied above. The B vitamins, although not technically antioxidants often acts as a co-factor with antioxidants. Flavonoids are naturally occurring in fruits, vegetables, tea and wine.

Let us next summarize about some important guidelines that can help in planning diets for hyperlipidemic patients.

Dietary *guidelines* fur *hyperlipidemic patients–A summary.*

- Calories: to maintain ideal body weight.
- Carbohydrates should constitute 55-65% of calories with emphasis on polysacchnrides (complex carbohydrates)
- Sugar less than 10% of total calories
- Dietary fibre : > 40 g/day
- Proteins: 15-20% of modified energy
- Fat: < 15-20% of total energy
- Dietary cholesterol : < 200-300 mg/day

It is important to note here that the dietary modifications need to be individualized in each patient and these should be tailored lo the abnormalities of the specific component. Initially lipid disorders are treated by diet modification and physical exercise. After 3-6 months, if there is no improvement then drugs are given in addition to the diet and exercise. A diet history prior to dietary changes must be assessed. The approximate intake of cholesterol, total fat, unsaturated fat, and alcohol, simple and complex carbohydrates should be assessed and accordingly the subject tackled. Some dietary tips that may be of help for hyperlipideinic patients include:

- Cholesterol and fat intake could be decreased by:
- Avoiding whole milk, cheese, curds made from whole milk. Skimmed milk or toned milk may be used.
- Organ meats (brain, liver, kidneys), egg yolks, cold meats, canned and sausages, ham, frankfurters, peanut butter should be avoided instead fish and poultry (baked and steamed) can be taken.

- Baked foods made with refined flour (maida) like cookies, patties, pastries, cakes, samosas etc. must be avoided. Whole wheat flour snacks may be encouraged.
- All fats especially butter, margarine, cream, coconut oil, hydrogenated fats must be avoided. Instead, oils rich in polyunsaturated fatty acids (safflower, soyabean, sunflower) and monounsaturated fatty acids (olive oil, peanut oil, rapeseed oil) may be used.
- Fresh fruits, canned or dried fruits (limited amounts) could be consumed and fruits with cream, butter, ice creams or dips avoided. Vegetables could be consumed except root vegetables in large quantities.

Triglycerides could be decreased by:

- limiting foods high in fats
- decreasing sugar and sugar containing foods (carbonated beverages, fruit drinks, sweet snacks and desserts, honey, jam, jelly, chocolates and candy)
- decreasing alcohol intake
- reducing portion size
- striving for reducing weight, and
- increased physical exercise

Having understood the etiology, symptoms and dietary management of dyslipideinia we will now proceed to learn about an important clinical manifestation of elevated lipids that remains asymptomatic for a long time - atherosclerosis. Remember we acquainted ourselves about this disease in sub-section 11.2.3.

11.3.2 Atherosclerosis: A Coronary Artery Disease

Atherosclerosis *is an arterial lesion characterized by patchy thickening of the intima (innermost coal of artery) comprising of fat and layers of collagen like fibres.* This is a slow or progressive disease, degenerative in nature affecting small and large arteries and weaken them leading to proliferation. This leads to problems in smooth flow of blood. These deposits are referred to as *plaque* as illustrated in Figure 11.2. The plaque reduces the size of the lumen of the artery and consequently, the amount of blood flow. The reduced blood flow causes an inadequate nutrient and oxygen supply and water removal from the tissues, leading to a condition referred to as *ischemia.* These condition causes pain in the chest which is referred to as angina pectoris and it radiates down the left arm. When the lumen narrows so much so that a blood clot occurs in a coronary artery and blood flow is cut off, a heart attack can result. The dead tissue that results is called an *infarct,* The heart muscle that receives the blood is the myocardium. Thus, such an attack is referred to as an acute *myocardial infarction* (MI).

Atherosclerosis is thus categorized as a continumm of as fatty streaks, intermediate lesions, fibrous plaques and complicated lesions. There are 5 phases to atherosclerosis:

Phase I: Asymptomaticphase, consists of fatly streaks which are non-obstructive, lipidfilled cells.

Phase 2: Consists of plaque with high lipid content and prone to rupture usually the type of lipid is LDLc.

Phase 3: Acute complicated phase with rupture and non-occlusive thrombus.

Phase 4: Acute complicated lesions with occlusive thrombus, which are associated with angina/mycordial infarction and even sudden death.

Phase 5: Fibrotic or occlusive lesion. Large thrombi can cause serious acute defects.

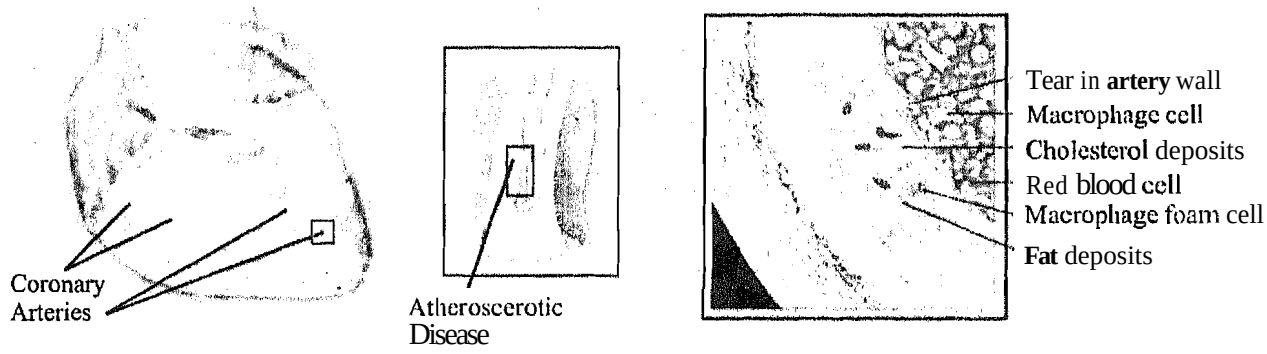

Figure 1.2: Progression of plaque in coronary artery

Having understood the development and progression of atherosclerosis, next let us get to know what are the causes for this degenerative disease.

Etiology

Various factors are responsible for atherosclerosis. These include:

1. *Hyperlipidemia*: Excess circulating fats in blood especially the low density lipoprotein (LDL) and low levels of high density lipoprotein (HDL) can predispose to atherosclerosis.
2. ***Hypertension:*** HT can accelerate atherosclerosis and cause complications.
3. ***Diabetes mellitus***: An important risk factor commonly associated with hypertension, due to abnormalities of coagulation, platelet adhesion and aggregation, increased oxidative stress, and abnormalities in vessel vasomotion can be a high risk for atherosclerosis.
4. ***Obesity:*** Excessive triglycerides (hyperglyceridemia) and LDLc levels ore commonly present in obese and lower HDL levels are a great independent risk factors 'for atherosclerosis.
5. ***Lifestyle:*** Low physical activity, cigarette smoking could affect tlie rate of atherosclerosis, increased CAD risk, On the other hand, regular exercise is seen lo be protective.
6. ***Factors causing endothelial damage:*** Elevated blood homocysteine a (genetically determined) and viral infections of lungs could damage the endothelial and cause injury and hence lead to atherosclerosis.

What then are the symptoms and complications linked with atherosclerosis? Let us find out.

Symptoms

Excessive weight, hypertension, high levels of cholesterol and triglycerides.

Complications

Myocardial infarction, systolic and diastolic dysfunction, inflammatory problems (pericarditis), stroke, gangrene (death and decay of body tissue) and aneurism (blood filled dilation of a blood vessel).

Now that we know tlie pathophysiology of atherosclerosis, let us learn about the nutritional management goals of this disorder.

Nutritional Management Goals

The nutritional management goals include:

- Reduction of weight if overweight or obese
- Reduction in the intake of total fat, saturated fat and cholesterol

- Medication if required for treating lipid disorders and controlling BP
- Lifestyle changes–increase in physical exercise, moderation in alcohol intake. No smoking, restricting coffee
- Consuming a balanced adequate diet, rich in calcium, chromium, iron and zinc
- Medical management is through various lipid lowering drugs

Dietary management and the nutrient requirements during atherosclerosis remain the same as for the management of dyslipidemia. Hence, we shall not go into the details here.

In our subsequent discussions, we will learn about a major clinical manifestation of progressive atherosclerosis i.e myocardial infarction. We will also update ourselves regarding some disorders commonly observed among patients suffering from myocardial infarction. These include hypertension and angina pectoris.

Before we proceed further, it would be good to recapitulate your understanding on the aspects covered so far through the check your progress exercise 2.

Check Your Progress Exercise **2**

1. List the various types of lipids in blood and also explain the role of apoproteins in CHD.

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2. Explain the following:

   a) Trans fatty acids .....

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   b) Ratio of n-3 and n-6 fatty acids in diet. Give two rich sources of each.

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3. Why is it essential to have water-soluble fibre in the diet?

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4. Discuss the dietary guidelines for carbohydrates during atherosclerosis.

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As mentioned earlier, in this section we shall discuss about an important acute, often life threatening clinical manifestation of progressive atherosclerosis viz., myocardial infarction. We shall also learn about a chronic degenerative heart disease which is frequently observed among poorly managed survivors of myocardial infarction i.e congestive cardiac failure. However, the management of these conditions is often challenging due to the presence of high blood pressure and chest discomfort (angina pectoris). Hence, let us first proceed with hypertension.

11.3.3 Hypertension (HT)

As you have seen in the above sections, hypertension is one of the major risk factors for cardiovascular disease. It is the most common public health problem and often referred to as a silent killer. If untreated, it can lead to a major health set back and cause many complications. In this section, we will know more about this disease condition.

We all have experienced our heart beat on several occasions. We can hear it more sharply with a stethoscope. The sound you hear is 'lup dup'. The first sound *lup*, you may recall reading in the Applied Physiology Course (MFN-001), in Unit 4, is the *systolic* sound, which occurs when the heart conlracts and pushes out the blood into the various parts of the body. This denotes the higher range of pressure called systolic blood pressure (SBP) which is measured as millimeters of mercury (mm Hg). The sound *dup* is the relaxation period of the heart when the blood enters the heart chambers. This is the *diastolic blood pressure* (DBP) and denotes the lower blood pressure.

To understand hypertension we must have an idea about normal and high blood pressure range. Table 11.5 presents the classification given by Joint National Committee (JNC), USA for the detection, evaluation and treatment of high blood pressure.

Table **11.5:** Classification **of** blood pressures and stages **of hypertension** in adults

Blood pressure **range SBP/DBP**	Classification
120/80	Normal (optimal)
120-129180-84	Prehypertension (normal)
130-139/85-89	Prehypertension (Borderline HT)
> 140/90	Hypertension
Stage I 140-159/90-99	Hypertension (Stage I)
Stage II 160-1791100-109	Hypertension (Stage II)
Stage III > 180/110	Hypertension (Stage III)

Source: Joint **National** Committee **on** Hypertension (2003)

High blood pressure is one of the leading causes of kidney failure, also commonly called *end-stage renal disease* (ESRD) about which you will learn later **in** this course in Unit 16. Major complications of hypertension includes:

- Kidney disease
- End-stage renal disease
- Hardened arteries
- Angina
- Left ventricular hypertrophy
- Left-side heart failure
- Cerebrovascular disease
- Cerebral haemorrhage
- Impaired vision
- Kidney failure
- Heart disease
- Cardiovascular disease
- Heart attack
- Heart failure
- Stroke
- Eye complications
- Retinal damage
- Death

Unlike many other diseases, hypertension develops insidiously (without **symptoms).** In 90% of hypertension cases, the cause is unknown. This condition is called essential *orprimary hypertension.* Rest of *the* cases with hypertension do have **an identifiable**

cause and are said to have *secondary* hypertension. This means that hypertension is caused by some other problem such as diabetes, thyroid and adrenal gland problems and kidney disease. Figure 11.3 presents the classification and manifestations of hypertension

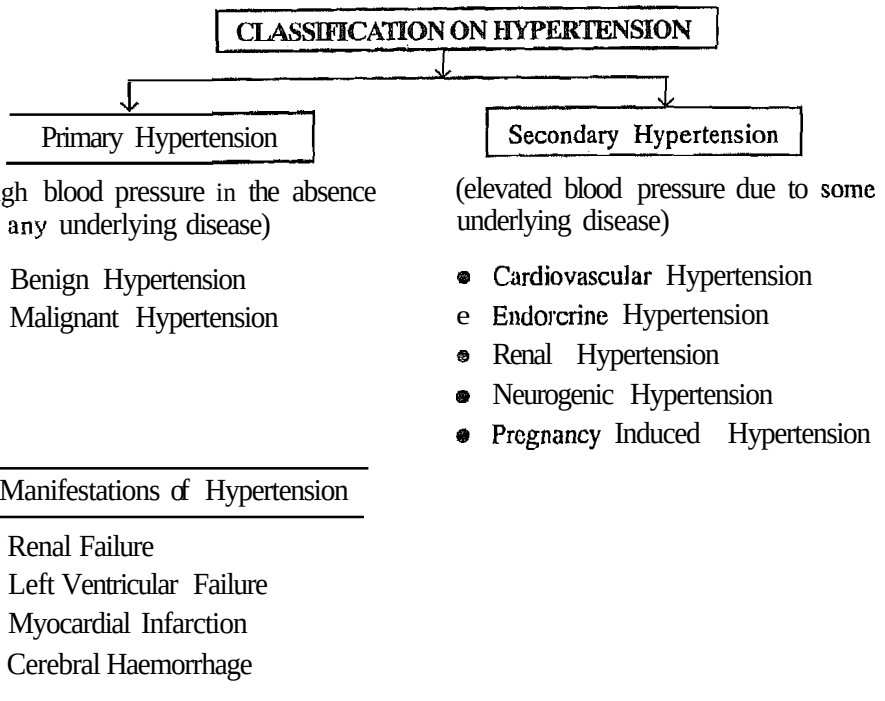

Figure 11.3: Classification and manifestation of hypertension

Hypertension can be damaging due to two effects:

1. Increased work load on the heart, and
2. Effect of hypertension on arteries

The increased work load on the heart causes hypertrophy (increase in size of tissue) of the cardiac muscle. The left ventricle continues to beat against the high blood pressure resulting in the enlargement of heart (enlargement is due to increase in muscle formation). As hypertension becomes severe, the heart becomes weaker,

High blood pressure causes coronary sclerosis (hardening In early stages, fatty lesions in the inner surface of the artery occur and this damages the inner surface of the artery (intima). Once the damage occurs, the endothelial cells swell and the smooth muscle below gets affected causing atherosclerosis (deposits of cholesterol, calcium on fibrous matter) leading to hard arteries, which loose their distensibility and could rupture. The deposits (plaque) may protrude in the lumen causing problems with blood flow and roughness of the surface. This could cause clots (thrombus) and damage to the organs.

Let us then learn about the causative factors of hypertension.

Etiology

Earlier in the unit, we learnt about the various risk factors of cardiovascular disease. We will now briefly mention the causes related to hypertension.

1. *Genetic factors*: Currently it is believed that there is polygenic inheritance and when environmental factors are not healthy, hypertension is precipitated.
2. Body weight and height: Hypertension increases with increase in the weight and height. Hence those who are obese have higher blood pressure values. Increase in BMI increases hypertension.
3. Age: Increases steeply with age. Now scientists have found shifts in BP. It is found in adolescents and the young as well.
4. *Gender*: Rise is greater in men than women but after menopause, the difference decreases.

5. Factors that may increase reabsorption by sodium can cause hypertension.

6. ***Changes in rennin-angiotensin:*** Aldosterone system and excretion of adrenocorticoids and prolactin may affect blood pressure.

7. *Hyperinsulinemia* of obese may influence blood pressure susceptibility through renal sodium reabsorption and transport.

8. ***Dietary factors:*** Excess calories, fats especially saturated fat and cholesterol in large quantities can increase blood pressure. Refined carbohydrates (sugars) could have an effect but studies in humans are inconclusive. High fibre intakes are beneficial (soluble fibre). Possible role of chloride, low potassium (K) and high sodium diets is a suspect. Less calcium and magnesium in diet could cause hypertension.

9. ***Modern lifestyle:*** Sedentary life devoid of exercise, stress, smoking, tobacco intake, alcohol are pointing towards increases in blood pressure.

We will now proceed over to the management of hypertension.

Treatment and Management of Hypertension

The first choice of treatment and management of primary hypertension is through behaviour modifications pertaining to food choices and lifestyle pattern. Patients who cannot maintain near normal levels despite dietary and life-style disorders are prescribed medications. Dietary management is important even for such patients in order to avoid drug dependency, side effects and dosage. We shall now learn in detail about the dietary management of hypertension. However, let us first identify the objectives of nutritional management.

Objectives of Nutritional Management

The objective of riutritional management of hypertension includes:

- To achieve gradual weight loss in overweight and obese individuals and maintain weight slightly below the normal levels,
- To reduce sodium intake and maintain fluid and electrolyte balance,
- To maintain adequate nutrition,
- To lead a healthy lifestyle (no smoking, alcohol consumption but a high physical activity), and
- To slow down the onset of complications.

In order to meet the above objectives, we need to understand the nutrient requirements during hypertension. Let us start with the calorie requirements.

Energy: Calorie requirement should be based on the concept of maintaining an ideal body weight. Excess calories through fats and carbohydrates have to be reduced so that the weight is maintained.

Proteins: Anormal protein intake is recommended. Protein should contribute 15-20% of the total energy needs. Excess non-vegetarian foods especially red meat and egg yolks could be avoided as it has greater proportion of saturated fatty acids.

Fats: The fats incorporated in the diet should be rich in unsaturated fatty acids and should not provide more than 20% of the total energy (refer dietary management of dyslipidemiafor details).

Carbohydrates: About ***60-65%*** energy should be provided from carbohydrates which are polysaccharides (complex carbohydrates) rather than simple sugars (monosaccharides and disaccharides).

Let us now learn about the most important aspect of dietary management i.e. the intake of minerals and electrolytes, which are closely associated with the maintenance of blood volume. Hypervolemia as we know would cause greater pressure on the arteries.

Minerals and Electrolytes: Minerals and electrolytes of clinical significance include calcium, sodium and potassium. Let us read about them one by one.

- *Calcium* (Ca): Adequate calcium intake is an essential part of the treatment and this could be ensured through intakes of milk and milk products and green vegetable as well as adequate cereals and pulse intakes.

- *Sodium:* Studies have shown that sodium restriction along with weight reduction is effective in controlling mild to moderate hypertension (1-2 g/day) along with diuretics recommended. Depending on the severity of hypertension, different levels of sodium intake can be recommended. These include:

  - *Mild Sodium restriction:* 2-3 g sodium (2000-3000 mg). Salt may be used lightly in cooking, but no salt at the table is allowed. There is no restriction on naturally occurring fresh foods but processed foods should be avoided.

  - *Moderate Sodium restriction:* 1 g sodium (1000 mg). In addition to the above restrictions, some control in naturally occurring fresh foods and no salt in cooking is added. Vegetables with high sodium content are limited in use, canned vegetables and baked products are avoided. Meat and milk products are used in moderate amounts.

  - *Strict Sodium restriction:* 0.5 g sodium (500 mg). Apart from the restrictions stated above, meat, milk and eggs are allowed in small portions and vegetables with higher sodium content are avoided.

  - *Severe Sodium restriction:* 0.25 g sodium (250 mg). This level is too restrictive and nutritionally inadequate and realistic to be used practically. In this, restricted quantities of meat and eggs are used only occasionally. Table 11.6 presents details on low sodium foods.

Table 11.6: Low sodium diet (Less then 0.5 g of sodium)

Foods	**Remarks**
Bread or chapattis or wheat, rice, maize, jowar, bajra or ragi, breakfast cereals, pulses, fish, chicken, milk (toned), vegetables-potato, sweet potato, tomato, gourds, cauliflower, cabbage, carrots.	Permitted unsalted, Avoid table and cooking salt.

  - Potassium: Increasing the potassium content in the diet lowers the blood pressure and improves hypertension. This could be done by increasing fruits and vegetables in the diet, which are rich in both potassium and fibre content.

Fluids: Fluid restriction is necessary only if oedema is present. Dehydration may be observed in some patients on diuretics. Thus, normal amount of fluids especially in the form of plain drinking water can be taken.

Thus, remember the following points while chalking out a patient care plan for hypertensives.

- *Lifestyle changes:* Avoiding smoking, use of tobacco, and excess alcohol intake. Physical activity like walking, 4 times a week for 40 minutes, is beneficial.

- *Medications:* Diuretics, calcium channel blockers and others should be consumed regularly.

- *Nutritious balanced diet:* The diet of a hypertensive should be nutritious. It should be low in calories (if required) and fat with a normal protein content. It should be low in sodium but rich in potassium, calcium, magnesium and fibre. Currently the DASH diets are recommended. These are rich in fruits and vegetables, non-fat dairy products and low in total as well as saturated fats,

The lifestyle modifications to manage hypertension are highlighted in Table 11.7.

Table 11.7: Lifestyle modifications to manage hypertension

Modification	Recommendation
Weight Reduction	Maintain normal Body weight (BMI : 18.5 to 24.9)
Adopt Dietary Approach to Stop Hypertension (DASH)	Consume diet rich in fruits, vegetables and low fat dairy products with a reduced content of saturated and total fat
Dietary Sodium Reduction	Reduce sodium intake to no more than 6 g sodium chloride
Physical Activity	Engage in regular aerobic physical activity such as brisk walk (at least **30** minutes per day, most days)
Alcohol Consumption	Limit to no more than two drinks per day

Source: IAMA (2003)*

With this, we end our discussion on hypertension. Next, we will proceed towards understanding the details of managing angina pectoris.

11.3.4 Angina Pectoris

Chest discomfort is often reported by most patients especially those which are chronic cases of dyslipidemia and/or hypertension. Like diarrhoea and fever, angina pectoris is a symptom and can appear in any cardiovascular disease condition. It is a tight choking feeling in the chest brought about by effort or less often by excitement. It is worse in cold weather or after heavy meals and is due to lack of blood to heart muscles. The angina could be stable or unstable. The stable angina shows no changes in the patterns of frequency or severity. The unstable angina becomes increasingly severe and the pain develops with less and less effort. It is sometimes called the 'brittle angina'.

Most common causes associated with angina pectoris are enumerated herewith:

- The usual cause of angina is the narrowing of the major coronary artery due to atherosclerosis.
- Systemic hypertension increases myocardial demand and if the supply of blood to the heart muscles is less, it can cause angina.
- Heart disease: In late stages of aortic stenosis (narrowing of aorta) it can precipitate an anginal attack.
- Anaemia: The heart gets less oxygen due to'lack of haemoglobin in anaemia.
- Thyroid disease: Thyroid disease is associated with angina.

Angina pectoris presents itself in the form of specific symptoms which tend to re-occur after a particular level of activity or exertion. The most typical symptom(s) and complications are mentioned next.

Symptoms

The pain of angina is usually over the center of the chest (below the sternum) but can be felt from epigastrum to the jaw and arms. It is brought about by exertion sometimes by stress and is relieved by rest. The duration of angina is short and can be relieved in three minutes on rest.

Complications

It is a symptom giving a warning of impending myocardial infarction, sudden cardiac death or even ischemic necrosis of the brain leading to cerebral stroke.

Now that we have learnt about the causes, symptoms of angina, let us get to know how to manage this condition.

Treatment and** dietary **management

Proper and careful treatment of the underlying cause (usually dyslipidemia, advanced atherosclerosis or sever chronic hypertension) is imperative to prevent the occurrence of any acute ischemic event namely myocardial infarction/stroke. Dietary management is the key component in preventing the progression of underlying disease condition. The most vital objectives of dietary and life style management include:

- To maintain ideal weight for age
- To lower blood pressure through drugs and diet control
- To avoid exertion and unnecessary stress
- To follow a prudent diet /**DASH** diet

The nutrient requirement here are the same as discussed earlier, however, to sum up it can be said that we need to restrict calories from total fats (particularly saturated fats) and from simple carbohydrates; avoid glandularmeats @rain, liver, kidneys etc.); use less salt in cooking and avoid salt sprinklers. Use a 'prudent' / **DASH** diet as explained under the hypertension section.

Let us move on to the next disorder, which is often referred to as the last stage in development of CAD i.e. myocardial infarction. It is an irreversible form of CAD and often results in decompensated changes in the structure and function of the heart. We should read and understand this critical disease carefully as it is often life threatening and most of the patients are hospitalized in the intensive cardiac care unit.

11.3.5 Myocardial Infarction (MI)

It is an initial acute phase of cardiovascular disease caused by the blockage of a coronary artery supplying blood to the heart. Figure 11.3, shows the progression of this disease. Myocardial infarction generally occurs when fibrous plaques coalesce together with blood clots to result in complete blockage or stenosis in artery supplying oxygen and nutrients through blood to the heart. The heart tissue denied blood because of this blockage, ultimately dies as indicated in blue colour in Figure 11.4. Now what causes it? Let us see.

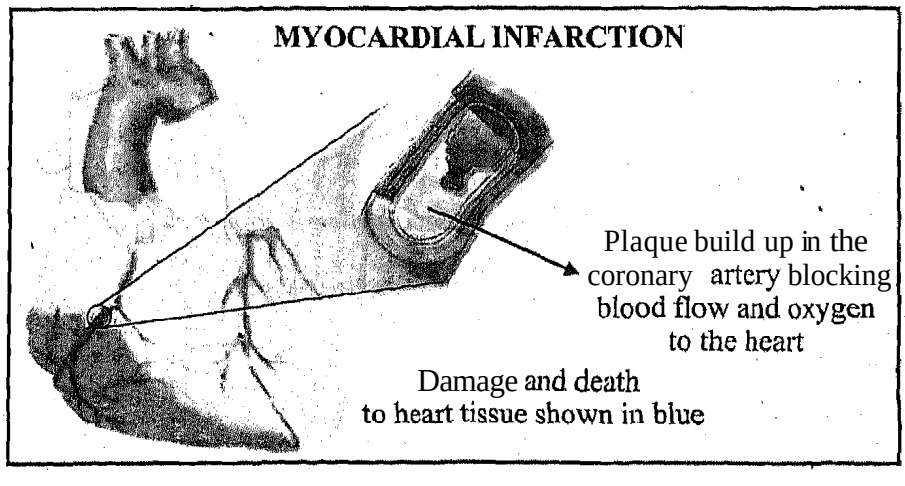

Figure 11.4: Infarction in the heart tissue

Persistently elevated blood lipids particularly LDLc, serum triglycerides, chronic hypertension and alterations in the balance between prostacyclins (a prostaglandin)

and thromboxanes are most directly associated in the development of occlusion of an artery.

Myocardial infarction is a critical disease with high frequencies of morbidity and mortality. Both medical and dietary management play a crucial role in managing the condition, as well as, in preventing re-occurrence/death. Proper nutritional care is also imperative if the patient needs to be operated to improve his quality of life. Let us understand some important aspects of dietary care in detail.

Dietary Management

Patients who suffer from an attack of inyocardial infarction are hospitalized and are usually kept under strict medical supervision. During the initial 24 to 48 hours most patients are on intravenous support and if needed on an artificial ventilator to meet their oxygen requirements. Initially oral food intake is restricted and not recommended as the ailing heart cannot support the oxygen and absorption of food. Oral food intake is resumed based on several cardiac function tests which help in assessing the residual functional capacity of the heart after injury. A low fat soft diet is generally recommended and during the initial stages foods may be introduced in very small quantities every hour or after every two hours. Observation of the patient during consumption of food and at least till ½ to 1 hour after eating is essential to rule out the elicitation of angina pectoris or another attack of myocardial infarction.

Let us now understand the nutrient requirements and overall dietary management of MI patients. We will first identify the objectives of the nutrition care process. You may want to read the pathophysiology discussed in section 11.3.

Objectives

The objectives of dietary management of myocardial infarction patients are as follows:

- to provide rest to the injured heart
- to maintain an optimum nutritional status
- to achieve and maintain a desirable body weight
- to prevent the development of another attack of MI

Let us then learn as to how can we fulfill tlie above mentioned objectives through suitable modifications in the nutrient intake. We will start with the calorie requirements of the patient.

Energy: As mentioned above, patient who have recently suffered from an attack of myocardial infarction are hospitalized in the intensive cardiac care unit wherein their movement is strictly restricted and they generally are advised not to socialize a lot. Thus, the energy expenditure on physical activity is very low or negligible. The diet should therefore provide enough calories to meet the basal requirements, hence a low-calorie diet is recommended. Other benefits of providing a low calorie diet include: reduction in the adipose tissue mass particularly among obese patients and hence reduced oxygen requirements of the body (tissues); reduction in the requirement of oxygen associated with ingestion, digestion and assimilation of food. The energy intake may initially begin with 800 Kcal wliich can be slowly progressed to a 1200 Kcal diet till the patient is discharged. Thereafter, the patient's energy intake should be governed on the maintenance of body weight wliich is preferably 1 to 2 kg below IBW.

We will now brief ourselves on the contribution of macronutrients towards the total energy intake.

Protein, Carbohydrates and ***Fat:*** The protein intake generally remains the same as per the RDI i.e. 1.0 gm protein per kg body weight per day. Adequate amount of proteins are necessary to promote regeneration of the n'ecrotic tissues in the myocardium, As we had mentioned earlier emphasis should be laid on plant proteins and low fat animal products (skimmed milk, low-fat paneer, chicken, fish and other marine foods).

Majority of MI patients are also hyperlipidemic and have elevated serum triglyceride levels. In such cases, the calorie contribution from fat should not be above 20% and the dietary cholesterol intake should remain below 200 mg per day. Details on the type of fat and food sources have been mentioned in section 11.3 earlier in the dietary management of hyperlipidemia. You may recapitulate from details mentioned in Table 11.7 on how to reduce fat and cholesterol intake.

Table 11.8 : How to **reduced** fat and **cholesterol** intake

	Avoid	Restrict	Use
Milk	Whole milk, cheese, ice cream	3% fat milk, processed curds	Skimmed milk
Meat	Organ meats, sausages, cold cuts	Fish canned in oil, shrimps, mutton with fat	Fish and marine food

Carbohydrates should provide 60% of the total energy. However, emphasis should be laid on the inclusion of easy-to-digest simple carbohydrates, which are low in fibre. Low fibre cereals, roots and tubers should be served in a soft well cooked/ blended form (purees etc.).

Vitamins *and Minerals*: ***The*** requirement of vitamins and minerals is largely governed by the existing nutritional status and the clinical parameters of the patient. Mild to moderate sodium restriction is generally recommended if the patient is hypertensive or is at risk of developing oedema due to congestive cardiac failure. Inclusion of low fibre, low sodium fruits and vegetables can help in providing good amounts of iron and B-group vitamins particularly folic acid and vitamin B_{12}.

So far, we have discussed the nutrient requirements during myocardial infarction. Mentioned below are a few useful tips that would be helpful in chalking out the nutrition care process of the patient.

Nutrition Support and other Considerations

You must have understood by now that the nutrient requirements of a MI patient vary from time of getting hospitalized in an emergency to the time of getting discharged. Patients when admitted to the intensive cardiac unit are critically ill and could be on life-support system.

They are initially kept on intravenous fluids to maintain a desirable blood volume and also to supply some amount of calories. As the condition improves; the patients may either be put on enteral tube feeding (intubated with ventilator lo supply oxygen) or introduced small sips of full-fluids after every 1 to 2 hours. ***The diet is gradually progressed to a semi-soft and then a soft one.*** The patients are closely observed when on a soft to normal diet, a few days before discharge. Thus, great care must be taken regarding the consistency and quantity of food being served to the patient,

The patient should be advised to cat slowly and adhere to a small frequent meal pattern even after discharge. Rest after meals should be advocated and the patient should avoid all forms of activity after meals. If the palient is overweight/ obese and needs to be operated, a low calorie diet to aid in weight reduction is a must. As a dietician you must be vigilant if the patient is also a diabetic as his insulin requirements may fluctuate drastically during the post MI period. Proper dietary counseling must be provided, particularly if congestive cardiac failure is present. In our next section, we shall learn about the causes, symptoms, treatment and management of congestive

cardiac failure–a decompensated heart disease that frequently develops among patients of MI after several years of rehabilitation.

In this section we read about the management of hypertension, angina pectoris and myocardial infarction. It must be clear to you that some aspects of dietary management may differ in the disease condition and the diet must thus be individualized. Let us now attempt the check your progress exercise 3 before we proceed further.

Check Your Progress Exercise 3

1. What are the two types of hypertension? Enlist any two complications.

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2. Enumerate the factors which contribute towarcls dyslipidemia?

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3. What do you understand by the term 'Atherosclerosis'? Enumerate its risk factors.

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4. What specific dietary measures would you recommend to a patient suffering from atherosclerotic plaque?

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5. What is Myocardial Infarction? Discuss about the carbohydrate intake during MI.

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So far we have learnt about hyperlipidemia, atherosclerosis, angina pectoris, hypertension and myocardial infarction. Most of these diseases except myocardial infarction are chronic in nature and usually reversible. Myocardial infarction is of course an acute form of CHD and causes irreversible damage. In this section we will proceed to learn about yet another decompensated heart disease viz., congestive cardiac failure.

11.3.6 Congestive Cardiac Failure (CCF)

It is an end stage heart disease and a significant contributor to morbidity and mortality particularly in the elderly population. Congestive cardiac failure is a form of chronic decompensated disease as the condition may develop over a period of time. It results from an injury to the heart muscle due to atherosclerosis, hypertension or rheumatic fever resulting in progressive weakening of the heart muscle. This leads to inadequate

blood circulation and hence insufficient supply of nutrients and oxygen to the tissues. This leads to malnutrition and underweight. If oedema is present, it masks the state of malnutrition. Mostly haemodynamic derangements are present. Congestive cardiac failure causes left ventricular systolic dysfunction over a period of time. The heart muscle – myocardium gets weakened progressively and is not able to maintain normal cardiac blood output or blood circulation. Firstly, it can cause pulmonary oedema causing breathing problems of pumping out blood fast enough resulting in blood accumulation on the right side of the heart, affecting normal circulation. This affects the normal flow of fluid between tissue space and blood vessel which thus starts accumulating in the tissue causing oedema. The two hormones in the body that control water balance also fail. (Antidiuretic hormone and the aldosterone). The aldosterone conserves sodium and water and worsens the oedema. Reduced blood circulation effects the metabolism of tissues resulting in increased cells breakdown and potassium is released. Increased potassium in the cell increases the osmotic pressure and sodium tries to replace potassium causing more water retention.

Congestive heart failure develops over a period of time when the necrotic tissues are not replaced by functional connective cells thereby affecting the contraction and relaxation capacity of the heart. Weakness of the heart muscles can be due to varied reasons some of which are discussed below.

Etiology

The causes of this disorder can be numerous. The risk factors which are known are:

- Chronic hypertension
- Left ventricular hypertrophy
- Coronary heart disease (recurrent episodes of IHD particularly myocardial infarction)
- Diabetes
- Advancing age
- Viral damage
- Alcohol abuse

  Injury

The characteristic signs and symptoms which develop due to the inadequate pumping capacity of the failing heart are being discussed next.

Symptoms

Congestive cardiac failure is a progressive form of cardiomyopathy. The most classical symptom is fluid imbalance due to inadequate cardiac output which results in cardiac/ pulmonary oedema which may latter involve other organs in the abdomen.

Since the haemodynamic changes may or may not develop at the same time, the clinical symptoms can vary a great deal from patient to patient. The most common symptoms seen are:

- Fatigue, faintness and weakness
- Swelling of feet and ankles
- Shortness of breath even after lying down, loss of appetite, indigestion, nausea and vomiting
- Congestion
- Inadequate cardiac output
- Altered fluid balance (oedema)

- Cardiac cachexia (severe malnutrition)
- Decreased urine production

One manifestation of congestive cardiac failure is decreased exercise tolerance, depending on the level of compensation. This leads to symptoms of shortness of breath, upon exertion orthopnea (inability to breathe easily), and oedema. However, these symptoms occur primarily in the late stages of cardiomyopathy. Notably, atrial fibrillation and ventricular dysrhythmias may be early signs of myocardial disease. Apart from these, other noticeable symptoms are:

• Weight gain (due to oedema)	• Swelling of feet and ankles
• Swelling of the abdomen	• Pronounced neck veins
• Difficulty in sleeping	• Sensation of feeling the heart beat (palpitations)
• Irregular or rapid pulse rate	• Decreased alertness or concentration
• Cough	• Decreased urine production

The above mentioned complications ultimately result in total failure of the heart (circulatory collapse), and circulatory problems causing organ damage (heart, lungs, kidney).

Impaired cardiac function could also lead to cardiomegaly (enlargement of heart), tachycardia (rapid pulse rate), heart rhythm problems, growth retardation in children. Poor peripheral perfusion results in cold extremities, weak pulse, low blood pressure. If pulmonary oedema is present, it can cause wheezing and problems in breathing.

Next, let us learn about the treatment of this condition.

Treatment

A judicious and careful co-ordination between oxygen support, drug therapy and nutrient intake can help in alleviating majority of the symptoms associated with congcslive cardiacfailure thereby reducing the frequency of morbidities and mortalities associated with this disease. ACE inhibitors, diuretics, beta blockers and digitalis drugs are generally used in conjunction with dietary management and oxygen support (severe category hospitalized patients). Surgical management involves heart transplantation, cardiomyoplasty, mechanical support and dialysis. Most of these are conducted as life-saving strategies. Whatever may be the choice of treatment, it generally revolves around the following objectives.

- To provide relief from the symptoms
- To improve the quality of life
- Prolong life by reducing, stopping or reversing left ventricular dysfunction
- To maintain adequate nutritional status.

In our subsequent discussion, we shall elaborate upon the nutrient requirements of the patients. We will also highlight some important aspects of dietary management and nutrition support that can help in the treatment1delaying the progression of congestive cardiac failure.

Dietary Managemerrt

Nutritional care is a little difficult in congestive cardiac failure. This is because oedema complicates the nutritional assessment of the subject. Because of cardiac cachexia the subject can loose about 10-15% of the usual weight and there is depletion of lean body mass. Poor reserves of essential vitamins, calcium and iron have also been observed. Thus, in view of the symptoms and treatment objectives, let us first identify the goals of dietary management.

Dietary Management

The objectives of dietary management include:

- to minimize stress workload on the heart
- to correct and maintain fluid and electrolyte imbalance
- to maintain a desirable body weight, and
- to maintain an optimum nutritional status.

Keeping the above objectives in mind let us proceed over to the nutrient requirements of the patient. We shall begin with the energy needs and then proceed lo the macro/ micronutrient intake which would be most feasible for the patient. Let us move on to the calorie requirement.

Energy: Composition of the calorie requirements on the basis of body weight is usually not feasible due to the presence of oedema. Thus energy requirements are generally based on the residual cardiac function and the usual body weight of the patient. The need for oxygen support is also an important factor for determining the energy intake needs to be sufficient to meet nutritional needs and prevent deterioration in nutritional status and at the same time care should be taken to avoid a positive energy balance unless so desired. This is so because excessive body mass increases the cardiac workload and weight gain from increased fat stores should be avoided. Gain in adiposity should be distinguished from short term fluctuations in body weight caused by changes in fluid balance. Patients on artificial oxygen support systems and/or those who are obese are recommended 1200 Kcal diet. Ambulatory and /or normal weight patients are usually able to tolerate around 10-25 Kcal/kg IBW or usual body weight per day. Not much efforts are made to increase weight among severely undernourished patients as increased food intake would impose enhanced burden on the heart.

Let us now proceed to the contribution of various other nutrients to meet the above discussed objectives. We will begin with protein.

Protein: The protein requirements remain the same as healthy adult men and women, About 0.8 - 1g of protein per kg usual or ideal body weight should be incorporated in the diet orally or by special methods of feeding. Since congestive cardiac failure is a form of cardiomyopathy and there is weakness of cardiac muscles, it is essential to supply good amounts of dietary proteins particularly high biological value proteins to facilitate tissue synthesis. Emphasis needs to be laid an plant proteins, which are at the same time low in sodium, rather than animal proteins.

Carbohydrate: While the quantity of carbohydrate remains almost the same as per the RDI i.e. 60% of the total energy, the quality of carbohydrate needs to be modified. Since the requirement of oxygen for the digestion and metabolism of complex carbohydrates is much higher as compared to that of simple carbohydrates, it is recommended that the diet should be very low in fiber and should provide good amount of simple carbohydrates (semolina, refined four, rice, dehusked pulses, papaya, mango, brinjal, pumpkin, gourd etc.). Whole cereals and pulses, legumes, lotus-stem, cabbage, soyaflour should be avoided.

Fat: The quantity and quality of fat would be governed by the severity of hyperlipidemia and adiposity. Emphasis, as always should be laid on oils rich in MUFA's and PUFA's . Fats rich in SFA's should be avoided. You may refer to sub-section 11.3.1 for details. In any case fats should not provide more than 20% of the total energy and the diet should be low in cholesterol (< 200 mg/day) depending upon the lipid profile of the patient.

Minerals: Since sodium and potassium are the major electrolytes associated with oedema, it is important that sodium intake should be 135-145 meq/L and potassium intake should be 3.5-5.0 meq/L. Mild to moderate sodium restriction (2.0 - 3.0 g Na per **day)** is often beneficial for most patients. Restriction of table salt and cooking salt is recommended for all patients. High sodium fruits and vegetables such as fenugreek leaves, lettuce, spinach, beetroot, tomato, grapes, lichi, musk melon, as well as, processed foods and preserves should be avoided. The extent of sodium restriction should be recommended by the dietician depending upon the severity of retention of sodium and water.

Vitamins: The requirements of all vitamins remains the same as per the RDI. If the patient is also suffering from hyperlipidemia/ atherosclerosis, liberal intake of vitamin A, vitamin C and folic acid may be helpful.

Fluids: Fluid intake should be monitored in accordance with urine output and severity of oedema. Fluid restriction is more important if the patient is not receiving diuretics. Patients on diuretics may consume normal amounts of fluids i.e. around 1.5 litres per day. The fluid allowance for patients not being prescribed diuretics can be estimated by any of the two methods given herewith:

I. Using the formula:

Fluid allowance	=	urine output (Previous 24 hrs.)	+	500 ml (basal losses)	+ losses of fluids due to diarrhoea vomiting (if any)

II. Weighing the patient everyday to find out gain in weight due to fluid retention and hence restricting the requisite amount (if required).

Some other considerations are highlighted next.

Other considerations

- Subjects with congestive cardiac failure often tolerate small frequent meals better than larger infrequent meals as these are tiring to consume, can contribute to abdominal distention and markedly increase oxygen consumption.
- Alternative seasonings and flavouring agents such as mild herbs and condiments may be used spairingly if sodium restriction is moderate to severe in order to ensure adequate food intake.
- The menu should be planned by keeping in mind the fluid allowance for the day.
- The patient should be advised to chew the food slowly. Sweating and chest discomfort are indicators of oxygen deficiency. Food ingestion should be stopped in such situations.
- Meals should be soft and well cooked. Raw food should be completely avoided. A liquid diet may be gradually progressed to a semi-soft, soft and a normal diet.
- If the patient is on ventilator, oral intake may not be feasible. In such situations; enteral parenteral tube feeding should be started.

The dietary management details discussed in the text above are usually recommended for congestive heart failure patients not suffering from liver or renal failure. Dialysis

is the treatment of choice in case of renal failure. In such situations restrictions on sodium and fluid may be less rigid.

Next, we shall proceed to yet another form of corollary heart disease i.e. rheumatic heart disease which generally affects children.

11.3.7 Rheumatic Heart Disease (RHD)

Rheumatic Heart Disease (RHD) is a very common cause of cardiovascular disorder in children and adolescents in India. This disease involves damage to the entire heart and its membranes. It is a complication of rheumatic fever (resulting from an untreated Streptococcus throat injection)'and usually occurs after attacks of rheumatic fever. Rheumatic fever can damage the heart valves. If the heart valves are damaged, they will fail to open and close properly, When this damage is permanent, the condition is called *Rheumatic* ***Heart Disease.***

What are the symptoms and complications linked with rheumatic heart disease ? Let us find out.

Symptoms

Symptoms generally appear after 1 to 6 weeks of the fever and sometimes the infection may have been too mild to have been recognized. The symptoins are fever, fatigue, shortness of breath, fainting, palpitation and chest pain. Swollen, tender, red, painful nodules or small protuberances may appear. There could be red, raised, lattice-like sash and uncontrolled movements of arms, legs and facial muscles.

Complications

Inflammation of lining of heart (pericarditis), anaemia, heart ,enlargement, valve deformities (mitral and tricuspid valves), embolism, arrythmia, abdominal pain, fever, arthritis etc.

Having learnt about the symptoms, complications..Let us also review the dietary considerations.

The diet should be nutritious and without restrictions except in the patient with congestive heart failure, whose fluid and ,sodium intake should be restricted. Potassium supplementation may be necessary because of the mineralocorticoid effect of corticosteroid and the diuretics (if used).

Finally let us dwell a bit further on the preventive strategies for coronary heart diseases.

11.4 PREVENTION OF CORONARY HEART DISEASES

In view of the steep rise in the incidence of chronic degenerative coronary heart diseases, several programmes have been formulated to reduce their incidence. The most important organizations/programmes which have contributed significantly include World Health Organization and National Cholesterol Education Programme. Some key aspects which have been propounded in all scientific auras pertaining to prevention of CHD and must comprise the counseling sessions are being elucidated in our subsequent discussions.

Nutrition guidelines for prevention of heart disease as suggested by WHO are highlighted in Table 11.9.

Details regarding nutrient intake and food choice checklists that can be used as effective tools for modifying the dietary intake of the masses at large are given in Table 11.10.

Table 11.9 : Dietary recommendations for the prevention of coronary heart disease (WHO) year

Calories	: Sufficient to maintain ideal body weight for height
Total fat	: 15-30% of calories
Cholesterol	: < 300 mg/day
SFA	: < 10% of total calories
PUFA	: < 8% of total calories
P/S ratio	: **0.8-1.0**
Linoleic Acid (LA/n6)	: 3-7% of total calories
Alpha linolenic acid (ALNA/n3)	: < 1 % of total calories
LA/ALNA ratio	: 5-10
Proteins	: 10-15% of total calories
Carbohydrates	: 55-65%, an with etnphasis on complex carbohydrates
Sugars	: ≤ 10% of total calories
Salt	: 5-7 g/day
Dietary fibre	: 40 g/day

Table 11.10 : Dietary recommendations (W.H.O.) - upper and lower limit for various foods and nutrients

	Upper Limit	Lower Limit
Total fat % energy (Kcal)	30%	15%
SFA (Saturated Fatty Acid)	8-10%	0%
PUFA (Polyunsaturated Fatty Acid)	5-8%	LA-%
MUFA (Mono Unsaturated Fatty Acid)		ALNA – 0.5% The difference
Cholesterol (mg/day)	< 300 mg	0
Total proteins % energy	15%	10%
Total CHO (Carbohydrates) % energy	75%	55%
Complex CHO % energy	70%	50%
Total dietary fibre (g / day)	40	27
NSP (Non Starch Polysaccharides) g / day	24	16
Free Sugar (g / day)	10	0
Salt (g / day)	6	—
Fruits and vegetables	400 g/day	—
Pulses, nuts and seeds	30 g/day	—

Since, excessive consumption of alcohol is a risk factor for cardiac heart diseases, consumption of alcohol should be strictly monitored. The caloric value of some soft and hard drinks is given in Table 11.11, which can serve as a handy guide for cardiac patients.

Table 11.11: Caloric value of hard and soft drinks

		Quantity (ml)	Carbohydrate(g)	Alcohol(g)	Calories
1.	Beer	250 (1 glass)	13	10	122
2.	Brandy	30 (1 peg)		14	98
3.	Gin	30 (1 peg)	—	12	84
4.	Rum	30 (1 peg)	—	14	98
5.	Whisky	30 (1 peg)	—	13	91
6.	Champagne (dry)	30 (peg)	2	10	78
7.	Red wine	100	3	10	82
8.	White wine	100	3	9	75
9.	Sherry	100	2	5	43
10.	Port	30	4	5	51
11.	Cola	30	21	—	84
12.	Aerated drinks (Orange/Yellow)	300 (1 bottle)	2	—	84
13.	Plain soda	300 (1 bottle)	0	0	—

The information/guidelines presented in the section above, we hope would go a long way in helping you plan, counsel patients suffering from coronary heart diseases.

In this section we learnt about the etiology, symptoms and management of a decompensated heart disease which is frequently found among the elderly. We also briefed ourselves on rheumatic heart disease which generally affects our pediatric population. Attempting the check your progress exercise will help you in recapitulating and giving a better understanding of these disease conditions.

Check Your Progress Exercise 4

1. Describe CCF and enumerate any four causative factors,

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2. Elaborate upon the electrolyte requirements during CCE

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3. What is rheumatic heart disease? Enlist any two objectives of dietary management during RHD.

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11.5 LET US SUM UP

In this unit, we learnt about their eliological factors, metabolic alterations, clinical manifestations and dietary management of the disease of heart. Cardiovascular diseases, hypertension, dyslipidemia, atherosclerosis, myocardial infarction, congestive cardiac failure and RHD are a group of cardiac diseases about which we had discussed briefly in this unit.

Finally, we focussed on the various dietary recommendations proposed by WHO for the prevention of heart diseases, as well as, dietary guidelines of the American Heart Association (AHP).

11.6 GLOSSARY

Aneurism : a localized, pathological, blood-filled dilation of a blood vessel caused by a disease or weakening of the vessel's wall

Angina pectoris : a recurring pain or discomfort in the chest that happens when some part of the heart does not receive enough blood.

Antioxidant : a group of vitamins including vitamin C, E, selenium and carotenoids.

Arteriosclerosis : sclerosis or thickening of the walls of the smaller arteries

Atherosclerosis : a hardening of the walls of the arteries caused by fatty deposits that build on the inner walls of the arteries which interfere with blood flow.

Carditis : inflammation of the heart tissues.

Coronary Heart Disease : disease involving the network of blood vessles that surround the heart and serves the myocardium.

Diuretics : a substance or drug that rends to increase the urine discharge.

Embolt/Embolus : abnormal particle (such as a blood clot or air bubble) circulating in the bloodstream.

End Stage Renal Disease : severe kidney dysfunction reached when kidney function is reduced to 10% or less of normal function.

Epitopes : the part of a foreign organism (or its proteins) that is being recognized by the immune system and targeted by antibodies, cytotoxic T cells or both.

Essential Hypertension : hypertension of unknown etiology.

Glycemic Index : a ranking of carbohydrate-containing foods, based on the food's effect on blood glucose compared with a standard reference food. It measures how much blood sugar increases over a period of 2 or 3 hours after a meal.

Homocysteine : an amino acid that occurs naturally in the body with high levels as risk factor for coronary artery disease.

Infarction/Infarct : an area of coagulation necrosis in a tissue due to local ischemia resulting from obstruction of circulation to the area.

Ischemia : deficiency of blood in a tissue, due to functional obstruction or actual obstruction of a blood vessel.

Linoleic Acid : an omega-6 fatty acid that serves as the parent compound in the synthesis of other ω-6 fatty acids such as arachidonic acid.

Linolenic Acid : a liquid polyunsaturated fatty acid that occurs in some plant oils.

Nephrosis : a syndrome characterized by oedema and large amounts of protein in the urine and usually increased blood cliolesterol.

Non-starch Polysaccharides : the main components of dietary fibre and includes celluclose, hemicellulose, pectins and gums.

Pancarditis : an inflammation of the entire heart (the epicardium, the myocardium and the endocardium).

Pericarditis : an inflammation of the membrane that surrounds the heart.

Pharyngitis : inflammation of the pharynx – the portion of the throat that lies just beyond the back of the roof of the mouth.

Prostacyclins : A prostaglandin produced in the walls of blood vessels that acts as a vasodilator and inhibits platelet aggregation.

Secondary Hypertension : hypertension secondary to some other disease state.

Xanthacalasma : xanthoma of the eyelids.

Xanthoma : yellowish coloured lesions on the skin containing cholesterol and fats, often associated with inherited disorders of lipid metabolism.

11.7 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1. CHD refers to disorders of the circulation, heart muscles or the blood vessels (valves/arteries, veins, capillaries). The tnost commonly occurring forms of CHD include dyslipidemias, atherosclerosis, hypertension, angina pectoris, MI, congestive heart failure.

2. Several risk factors have been identified with CHD; the major modifiable risk factors being: dietary habits, obesity, hypertension, diabetes, smoking, tobacco and alcohol abuse, as well as, lack of physical exercise.

3. Major stages in the development of CAD include: Proliferation of the smooth muscle cells, Fatty streaks, Gelalinous lesions, Fibrous plaques, Thrombosis, and Occlusion of an artery.

4. Syndrome X is a defined as a collection of clinical manifestations such as abdominal obesity, diabetes, dyslipidemia, hypertension and elevated triglycerides, decreased HDL all of which are risk factors for CVD.

Check Your Progress Exercise 2

1. The various types of lipids in the blood are: chylomicrons, VLDL, LDL, IDL, HDL Cholesterol and Triglycerides. Apoproteins maintain the solution of lipoprotein lipids in plasma. Measurement of their level aids in diagnosing lipid disorders and preventing the risk of CVD.

2. a) Transfatty acids arise during hydrogenation of vegetable oils. They are known to raise LDL levels in the blood.

   b) n_3 fatty acids (alpha-linolenic acid) and n_6 fatty acids (linoleic acid) are polyunsatuated fatty acids whose ideal ratio should be 5-10.

   n_3 rich **food** sources – Canola oil, Fish oil

   n_6 rich food sources – Safflower, Sun flower oil

3. Water soluble fibres such as gums, pectins etc. are beneficial in the treatment management and prevention of CHD as they enhance satiety, help in weight reduction, reduce LDLc, VLDLc, triglycerides and post prandial plasma glucose levels. It also increases tissue insulin sensitivity.

4. Dietary guidelines regarding carbohydrate intake during atherosclerosisinclude: 55-65% of calories from CHO with emphasis on polysaccharides, Sugar less than 10%of total calories, Dietary fibre should be >40 g/day.

Check Your Progress Exercise 3

1. Hypertension has been classified into 2 types: I-Iypertension arising due to unknown causes in called primary hypertension. Hypertension caused by other problems like diabetes, changes in the functioning of thyroid is called secondary hypertension. Common complications involve the heart and renal system.

2. The causes for dyslipidemia are: genetic, environmental or secondary to disease condilions or drugs.

3. Atherosclerosis is a slow, progressive and degenerative disease of the arteries, characterized b y patchy thickening of the intima of arleries with fat and layers of collagen like fibres. Risk factors include hyperlipidemia, obesity, hypertension, dietary errors and sedentary lifestyle.

4. Specific dietary measures to be taken during atherosclerosis include: Moderate calorie restriction to achieve and maintain an ideal body weight, reduction in total fat, proteins 15%-20% of the total calories (high biological value and preferably from plants) and increased intake of fruits and vegetables (preferably with their edible peels) along with reduction in salt intake.

5. Myocardial infarction (MI) or heart attack is an acute form of CVD caused due to blockage of a coronary artery supplying blood to the heart, resulting in cardiac tissue death. MI patients should preferably be given simple carbohydrates and the intake of indigestible carbohydrates i.e dietary fibre should be minimized. Thus, refined cereals such as maida, rice, sago, arrowroot should be preferred over whole cereals.

Check Your Progress Exercise 4

1. The main feature of CCF is left ventricular systolic dysfunction over a slow period of time. The causes being hypertension, left ventricular hypertrophy, diabetes, CHD.

2. In view of oedema due to impaired circulation it may be imperative to restrict the sodium intake. Mild to moderate sodium restriction (2-3 g/day) is usually recommended.

3. Rheumatic Heart disease stems from an untreated streptococcus throat infection in childhood, resulting in Rheumatic fever and gradually progressing to cardiac value damage, which is permanent.