

CLINICAL AND THERAPEUTIC NUTRITION

M.Sc. - 202



Directorate of Distance Education

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Published by: **Publishers Grid**

4857/24, Ansari Road, Darya ganj, New Delhi-110002.

Tel: 9899459633, 7982859204

E-mail: publishersgrid@gmail.com, work.publishersgrid@gmail.com

Printed by: A3 Digital Press

Edition : 2021

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1**INTRODUCTION TO MEDICAL
NUTRITION THERAPY****STRUCTURE**

- 1.1 Learning Objective
- 1.2 Introduction
- 1.3 Definitions and Role of Dietitian in Health Care
- 1.4 The Nutrition Care Process (NCP)
- 1.5 Importance of Coordinated Nutritional and Rehabilitation Services
- 1.6 Patient Care and Counseling
- 1.7 Let Us Sum Up
- 1.8 Glossary
- 1.9 Check Your Progress

1.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- describe the processes involved in nutritional care,
- learn how to evaluate the nutritional status of an individual,
- plan, implement and evaluate nutritional care based on the assessment,
- highlight the importance of patient care and counseling, and
- understand the importance of team approach in therapeutic nutrition.

1.2 INTRODUCTION

Nutrition is one of the basic components of life. It is an essential part of health care. You already know that good nutrition is essential for the maintenance of optimum health, prevention of disease and recovery from illness. Provision of proper attention to nutrition can remarkably alter the course of illness when it occurs.

In this unit, we shall deal with medical nutrition therapy. Nutrition is an integral part of the medical therapy as adequate nutrition support is essential to prevent an extended and complicated hospital stay. Working closely with the physician, the nutritionist/dietitian determines an individual's nutritional

therapy needs and plan of care. Who is a dietitian? What are his/her roles and responsibilities? How can patient care and counseling help to improve a patient's status? These are the issues which we shall study in this unit.

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This unit also focuses on nutritional care process, its components and its effectiveness. As you read on, you would realize that the important function of nutritional care is to ensure that all patients are adequately and appropriately nourished. We shall study about nutrition care process under the following sub-sections: nutritional assessment, nutritional care plan, implementation of the plan and evaluating the efficacy of the nutrition care plan.

1.3 DEFINITIONS AND ROLE OF DIETITIAN IN HEALTH CARE

We all are familiar with the word 'dietitian'. Who is a dietitian? What are the roles and responsibilities of a dietitian in a hospital setting?

Let us read and find out this and much more in this section. But before that, let get to know what we mean by the term dietetics?

1.3.1 Dietetics the Science and Art of Human Nutrition Care

Dietetics has been defined as the science and art of feeding individuals based on the principles of nutrition. It can also be said to be the "science and art of human nutritional care.

" Dietetics is a study of using the principles Of nutrition in planning suitable diets in health and disease, In other words, diet therapy and its application in patient related settings is a major focus of dietetics.

Thus, the field of dietetics can be related to:

- Nutrition care and intervention focused on the individual, and
- Nutrition care and intervention focused on the group.

Traditionally nutritionists have focused largely (or almost fully) on biological aspects of nutrition. However, we have realized over the years, that physiological biochemistry does not provide answers fully to the problems in human nutrition.

Thus, nutritionists are moving towards a comprehensive approach to human nutrition and societies, as well as, professionals from a variety of related fields have begun to increasingly recognize the central role in every aspect of life.

The rapid growth of scientific information and understanding of the inextricable nature of biological, sociological and psychological factors in human life are now making it obvious that we need a holistic or encompassing approach to human nutrition and dietetics.

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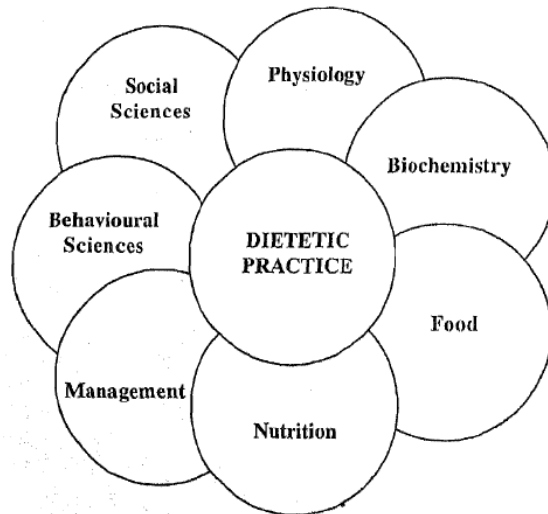


Figure 1.1: Dietetics multidisciplinary approach

The body can utilize nutrients only after food is eaten. Therefore, nutritionists and dietitians need to consider all those factors which influence or rather determine what, how, when, why and how much a person eats. The various biological, socio-cultural and environmental factors which affect food choices. If you examine which services/fields deals with these issues, you will move into the realm of sociology, ecology, anthropology and psychology.

Table 1.1: Factors affecting food choices

Biological Factors	Socio-cultural Factors	Environmental Factors
Nutritional needs	Education	Geography climate
Heredity	Understanding of nutrition	Season
Special physiological condition like pregnancy	health concept	Economics
Special diseases or abnormal conditions	Social class, status	Transportation
Taste preferences	Income'	Technology
Individuals cravings, likes and dislikes	Traditions, beliefs, values	Fuel availability
	Ideology, relation	
	Communication	
	Influence of business	
	Government (policies)	
	Professionals politics	

Dietetics optimizes the nutrition of populations and individuals. Dietetics, therefore require interdisciplinary approaches since the nutrition and diet counseling is not only science it is an art.

Let us next understand a few terms related with dietetics — Clinical Dietetics and Medical Nutrition Therapy (MNT). What are these and what is their role in nutrition care? Let us read and find out.

Clinical Dietetics is the application of dietetics in a hospital or health care institutional setting. Clinical dietetics focuses on an individual, nutrition support and symptom management.

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Medical Nutrition Therapy (MNT) is defined as the assessment of the nutritional status of a client followed by nutrition therapy ranging from diet modification to specialized nutrition support such as the administration of enteral and parenteral nutrition and monitoring to evaluate the patient.

MNT may also be defined as nutritional diagnostic, therapy and counseling services for the purpose of disease management.

MNT starts with the assessment of nutritional status of patient with a condition, illness or injury that puts them at risk. This includes the review and analysis of medical and diet history, laboratory values and anthropometric measurements. Based on the assessment, a nutrition care plan, most appropriate to manage the condition or treat the illness or injury is formulated. The MNT also includes intervention and evaluation of achievement of desired clinical outcomes. Appropriate medical nutrition therapy provided by the dietetics professional has been shown to result in health benefits and reduced health care costs.

Diet also plays a very crucial role in the health and well being of people. A good and balanced diet improves the quality of life to a great extent, Poor eating habits and inadequate food intake are the major causes of a lot of diseases. Nutrition and dietetics are fields related to this food and nutrition aspect of life.

The study of nutrition means an understanding of the various components of food and the role and requirement of each of these components for the body. One becomes aware of the right type of food which provides a balanced mix of the essential vitamins, minerals etc. It also involves a study of the processes by which the food is digested and absorbed in the body. This field finds application in medicine, veterinary, agriculture and public health.

Dietetics is the interpretation and communication of the science of nutrition to enable people to make informed and practical choices about food and lifestyle, in both health and disease. A dietitian will have training in both hospital and community settings as a part of their course. Most dietitians are employed in the hospitals, but may also work in the food industry, education, research and on a freelance basis. It is necessary for them to have a recognized degree; M.Sc. or post graduate diploma in nutrition and dietetics to work as a dietitian.

1.3.2 Role of Dietitian in Health Care

The role of the dietitian has come a long way since the early 1900s. Their role is still unknown to a lot of people. Some think that dietitians, as their name implies, only give out diets to make individuals lose weight, whereas this is only a small part of their role. The dietitian has a defined role concerning the ethical issues

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and dilemmas of nutrition care for patients. The dietitian is the link between the patient and the medical team or physician in assisting difficult decision making about nutrition care. A description of the dietitian's role in terms of managing the nutrition support of a terminally ill patient may be as follows: 'The dietitian needs to continue to play an essential role in evaluation and decision-making in the nutritional support of the terminal patient. No individual is better trained to interpret and coordinate nutrition issues between the patient and the other members of the healthcare team in this unique situation. The development of new feeding technologies, supplements, and interventions will continue to force difficult decisions to be made concerning the benefit of these modalities and the desires of the patient'.

Several medical and nutritional organizations have remarked on the role of the dietitian in nutrition care issues and dilemmas. These have been presented for your knowledge in Box I herewith.

Box 1 Role of Dietitian in Nutrition Care

Asian Society of Parenteral and Enteral Nutrition (ASPEN) states that the dietitian's role in nutrition care has been to recommend an adequate source and amount of balanced nutrients according to pre-established standards of care. A dilemma occurs when the disease state of the patient confounds the adequacy of nutritional support, which has resulted in the patient's malnourishment.

The American Dietetic Association (ADA) remarked on the role of the dietitian in feeding dilemmas as: the dietitian, like other healthcare professional, has an inherent ethical responsibility to respect the sanctity of life and the dignity and rights of all persons and to provide relief from suffering. It is the dietitian's responsibility to provide a combination of emotional support and technical nutrition advice on how best to achieve each patient's goals within legal parameters.

This statement affirms that dietitians have an active role in the care and support of any and all patients. It is not acceptable to sign off on a patient's medical record when the tough legal and medical decisions are to be made. Instead, the dietitian should be:

- informed on the rights and desires of the patient and/or family,
 - informed on the severity of illness and complications of treatments, inclusive of the benefits and burdens of feeding in all conceivable routes, active in the patient's care as the dietitian reporting on the nutritional status of the patient, as well as, the advisor to the physician and medical team, and
 - informed of legal decisions that may help determine the route of care for the patient, such as more aggressive or palliative care.
- Some of the situations that concern ethical decisions in nutrition care are as follows:

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difficulty of adequate nutritional support of malnourished patients,
problem of providing nourishment to competent patients who refuse feeding,
benefit vs burden questions, especially in terminally ill patients, and
incompetent patients who may or may not have families to help determine their wishes for feeding.

A common scenario that occurs in daily practice is providing adequate nutritional support to malnourished patients. The patients who are usually seriously ill may have complicating medical conditions that impede delivery of adequate nourishment. The dietitian struggles to provide adequate nutritional support in relation to the medical condition and the desires of the patient.

Dietitians are engaged in a variety of positions and in a number of work settings. Of course the largest proportion is involved/ engaged in food service and in patient care within hospitals or outpatients. However, some dietitians do work in food service for students, in the hotel industry, in employee cafeterias (industrial canteens), food and pharmaceutical companies, as well as, in community and public health services/ departments. Some are also in private practice and may be self-employed. Nowadays dietitians are also involved in marketing, sales and journalism. If you examine these activities you will find that the dietitians services focus on :

- Clinical services
- Public health/community nutrition
- Nutrition information/communication
- Food services
- Wellness/disease prevention
- Nutrition research.

Many dietitians are beginning to be involved in newer speciality areas such as sports nutrition, cardiovascular fitness, nutrition education of the public, prenatal nutrition, as well as, physical medicine and rehabilitation.

Thus dietitian's practice/roles are rapidly changing as the health needs of society change and as the health care system evolves and develops to meet societal needs. This necessitates dietitian's possessing a wide variety of skills. Beyond the technical knowledge and practical skills dietitians need to have communication and education skill (both oral and written), since they may be expected to plan, organize, implement and evaluate nutrition education for individuals, clients and groups.

In all of this the dietitian's involvement is not only in therapeutic nutrition i.e. rehabilitation but also health promotion and health maintenance. High-ranking competencies are needed to apply skills in communicating scientific information at a level appropriate to different audience. A good professional dietitian should

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also have the ability to select and/or develop nutrition education materials and approaches appropriate for a variety of target groups.

In food service systems, the dietitian will have many managerial roles to play e.g. orienting, training and developing staff, counseling subordinates, providing on-the-job and in-service training and continuing education that meet the needs of employees.

Another upcoming area is home health care where patient counseling, caregiver education, documentation, diet histories and developing a nutrition care plan are important activities.

Thus dietitians are in a 'helping' profession because the services they provide are beneficial to individuals and society and dedicated to improving the nutritional status of the people. Helping professions can be described as professionals that do something with knowledge e.g. communicating, interpreting and applying nutritional science to benefit the health of people.

Helping professional need a variety of skills:

- Techniques of interviewing
- Techniques of counseling
- Ability to relate to individuals, groups and individuals
- Effectiveness in bringing about change
- Capacity for self-understanding
- Establishment of professional, interdisciplinary relationship
- Knowledge of personality, group and societal dynamics

If a dietitian has these skills, she can assist others or herself, bc able to assess many (or all) dimensions of a problem, explore alternative solutions and stimulate action towards positive change and problem resolution.

Along with the role and responsibilities enumerated above, the dietitian is an important link in the chain of patient care decisions. Let us see how.

Consulting with Physicians

Usually, the dietitian's role in feeding dilemmas is seen as secondary and the physician's role is the focus. In reality, the dietitian is an important link in the chain of care decisions, often serving as a consultant or a fact-gatherer for the physician and/or medical team. Through the presentation of relevant information, the dietitian becomes a part of the decision-making body that assists patients in their care. A scenario of a dietitian consulting the medical team is described in this case example of the dietitian's role in an ethical dilemma regarding the allocation of two feeding pumps among seven critically ill patients who needed the pumps. The nutrition care dilemma in this case was an insufficient number of pumps available for the number of patients needing the pumps: A dilemma

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is the choice between two alternatives, neither being totally ideal. The medical team had to search for both medical and moral reasons for selecting one patient over the other for the use of the feeding pump. The choice of the team was to give the feeding pumps to those patients who were deteriorating the most quickly and where nutrition support was critical. As a patient improved, feeding was changed to oral methods to allow the feeding pump to help another patient. In other words, through careful planning and organization, the feeding pumps were rotated to the seven patients as medically necessary. A four-step process of moral judgment and action can be utilized to analyze the feeding pump allocation problem. The four-step process includes gathering relevant information, identifying the ethical dilemma, deciding what to do, and completing the action. The following list serves as an outline to this process:

- implement a pragmatic moral judgment: and action process.
- confer with other health professionals.

The dietician and the medical team ranked the patients in accordance to pump need, based on the following factors:

- **Present nutritional status:** The dietitian presents a nutritional assessment of the patient that may include information specific to ideal body weight, history of weight loss, pertinent laboratory values, and anthropometrical measurements.
- **History of diet and or tube feeding tolerance:** The dietitian presents the patient's history, which may include presence or history of emesis (vomiting), diarrhoea, fat malabsorption and food allergy and/or lactose intolerance.

Nature of Work — Other Activities

Dietitians, we read above, form an important part of the health care team within a hospital and are responsible for planning and organizing all activities for food service within the hospital. Apart from this, dietitians have direct responsibility for food service operations, where one food safety mistake can affect hundreds, and even thousands of people. The fact that many institutions are serving food to individuals, who may already be in a "high-risk" category for food-borne illness, makes the dietitian's role even more critical.

A few of the responsibilities include planning menus, purchasing and ordering food / equipment within budget, recruitment, education and evaluation of staff, observing and practicing all safety and sanitation rules strictly.

Dietitians have a direct contact with the public and other health professionals. Nutritionists and dietitians deal with people to inform and guide them about the diet they should take to improve the general health, to avoid certain diseases or to keep the existing ailments in control. People suffering from certain diseases need to take extra care of their eating habits and the kinds food they eat. Ignorance of this can aggravate the disease, whereas, adherence to the right diet can help in speedy recovery or stability of the condition. Major role of dietitians is to assist people in planning their meals depending upon their age, sickness or work routine.

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Dietitians counsel individuals and groups, organize the food service systems in hospitals, schools, hotels etc. Dietitians and nutritionists plan food and nutrition programmes and supervise the preparation and serving of meals. They help to prevent and treat illnesses by promoting healthy eating habits and recommending dietary modifications.

Dietitians can specialize in several areas such as administration, clinical dietetics, research and community dietetics. Let us understand these specializations.

- Administrative dietitians play a major role in large-scale meal planning and monitoring the food preparation process by applying the principles of nutrition and sound management in hospitals, schools, canteens etc. They take up the entire responsibility of their department and actively participate in planning, purchasing, preparation, distribution and service of meals. These dietitians select, train and direct food service supervisors and workers; prepare budget for food, equipment and supplies; enforce sanitary and safety regulations; and prepare records and reports. Increasingly, dietitians utilize computer programmes to plan meals that satisfy nutritional requirements and are economical at the same time.

Dietitians who are the directors of dietetic departments also decide on departmental policy, coordinate dietetic services with the activities of other departments, and are responsible for the dietetic department budget.

Clinical dietitians, sometimes called therapeutic dietitians, are associated with health care institutes, hospitals and nursing homes. Depending on the nutritional needs of the patient's on the basis of individual nutritional assessment they prepare the diet charts and monitor the results of diet therapy. They assess patient's nutritional needs, develop and implement nutrition care plans, evaluate and report the results. Clinical dietitians confer with doctors and other members of the health care team about patient's nutritional care, instruct patients and their families on the requirements and importance of their diets, and suggest ways to maintain these diets at home.

Technological advances in nutritional support for the critically ill have enhanced the clinical dietitian's role. In the hospital, dietitians oversee the preparation of custom-mixed high-nutrition formulas for patients who are critically or terminally ill and require special feeding through oral, enteral or parenteral route. In the home health field, they help develop and oversee sophisticated nutritional therapies for homebound patients who, because of surgery or illness, are unable to eat regular foods. In addition, clinical dietitians in nursing care facilities, small hospitals or correctional facilities may manage the food service department.

- Research dietitians work in the field of normal or therapeutic nutrition. Research dietitians seek ways to improve the nutrition of both healthy and sick people. They may study nutrition science and education, food management, food service systems and equipment, or how the body uses food. Other research projects may investigate the nutritional needs of the aging persons who have

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chronic diseases, or space travelers. Research dietitians need advanced training in this field and usually are employed in medical centers or educational facilities, or they may work in community health programmes.

- Community dietitians or nutritionists may counsel individuals and groups on sound nutrition practices to prevent disease, maintain health and rehabilitate persons recovering from illness. They may engage in teaching and research with a community health focus. This work covers areas such as special diets, meal planning and preparation, food budgeting and purchasing. Dietitians or nutritionists in this field usually are associated with community health programmes; they may be responsible for planning, developing, coordinating and administering a nutrition programme followed by proper evaluation.

Working in places such as public health clinics, home health agencies, health maintenance organizations, community dietitians evaluate individual needs, develop nutritional care plans and instruct individuals and their families. Dietitians working in home health agencies provide instruction on grocery shopping and food preparation to the elderly individuals with special needs, and children.

Increased public interest in nutrition has led to job opportunities in food manufacturing, advertising and marketing. In these areas, dietitians analyze foods, prepare literature for distribution, or report on issues such as the nutritional content of recipes, dietary fiber, or vitamin supplements.

- Consultant dietitians work under contract with healthcare facilities or in their own private practice. They perform nutrition screenings for their clients and offer advice on diet-related concerns such as weight loss or cholesterol reduction. Some work for wellness programmes, sports teams, supermarkets, and other nutrition-related businesses. They may consult with food service managers, providing expertise on sanitation, safety procedures, menu development, budgeting, and planning.
- Teaching (academic dietitians)

Dietitians possess knowledge on all aspects of nutrition and dietetics. They constantly keep themselves updated in the necessary information and knowledge which they transfer to the young interns/trainees under the internship programmes. They help translate theoretical concepts into applied aspects of dietetics (preventive and curative aspects of normal/ therapeutic nutrition).

Therefore, it must be evident, that the nature of work or activities undertaken by a dietitian may be multifarious. What about the work environment? As a dietitian, what would be your job and does your personality fit the job description. Read and find out for yourself.

A). Work Environment

Dietitians and nutritionists, who are associated with hospitals and clinics generally have regular work hours. At times, they may be required to work in shifts or on the weekend too. In this environment, they come in direct contact with patients and

advise them appropriate diet based on the illness.

In commercial food service, the working hours are usually irregular. The work in research is carried on in the laboratories while in most other assignments their office is located near food preparation areas. Certain time needs to be spent in kitchens which are usually hot.

There are good career opportunities in the food industry too. Lot of food companies employ nutritionists and dietitians to check the nutritional quality of the food products, for new product development and for marketing related advice. There is teamwork involved in experimenting on flavours and preparations.

B). The Job

The dietitians undertake the practical application of nutrition with both individuals and population groups to promote the well being of individuals and communities to prevent nutrition related problems. They are also involved in the diagnosis and dietary treatment of disease.

Dietitians work with people who have special dietary needs, inform the general public about nutrition, give unbiased advice, evaluate and improve treatments and educate clients, doctors, nurses, health professionals and community groups.

Dietitians can work in a variety of areas, as already mentioned earlier, many of these are in the hospitals or in the community as 'Clinical Dietitians', 'Nutrition/Health Educators' or as 'Managers'. Both hospital and community dietitians educate people who need special diets as part of their medical treatment, for example patients with kidney disease, food allergies, eating disorders, diabetes, HIV/AIDS, oncology and gastroenterology. There are also opportunities for dietitians to work outside the hospital setting in a variety of different areas such as food industry, education, research, business, charities, media, freelance work.

C). Personality

Dietitians have special skills in translating scientific and medical decisions related to food and health to inform the general public. They also play an important role in health promotion. A variety of skills need to be possessed by them which include: techniques of interviewing, counseling, ability to relate to individuals and groups, effectiveness in bringing about change, capacity for self-understanding, establishment of professional inter-disciplinary relationship and knowledge of personality, group and societal dynamics.

In all, a dietitian would need to have an interest in science, people and food, an ability to explain complex things simply, a positive and motivating attitude, non-discriminatory approach, as well as, patience and a sense of humour. If a dietitian has these skills, she can assist others or herself to be able to assess many (or all) dimensions of a problem, explore alternative solutions and stimulate action towards positive change and problem resolution. So then, as a student of dietetics,

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we hope you have the personality and interest to work and excel in this area.

With this, we end our study of dietetics and the role of a dietitian. Next, we will explore the nutritional care process. But, first let us recapitulate what we have learnt so far.

1.4 THE NUTRITIONAL CARE PROCESS (NCP)

The nutritional care process is a systematic and logical approach to ensure effective and successful nutrition intervention. The American Dietetic Association (ADA) defines the nutrition care process as ' a systematic problem-solving method that dietetic professionals use to critically think and make decisions to address nutrition related problems and provide safe and effective quality nutrition care'.

The purpose of the NCP is to give the dietetic professionals a consistent and systematic structure and method by which to think critically and make decisions. It also assists dietetics professionals to scientifically and holistically manage nutrition care, thus helping patient's better meet their health and nutritional goals.

Here it is important to emphasize that the nutrition care process is a standardized process for dietetic professionals and not a means to provide standardized care. Standardized process here refers to a consistent structure and framework used to provide nutrition care, whereas standard care infers that all patients/clients receive the same care.

Thus, the nutrition care process supports and promotes individualized care not standardized care, The nutrition care process acknowledges the common dimensions of practice by the following:

- defining a common language that allows nutrition practice to be more measurable,
- creating a format that enables the process to generate qualitative and quantitative data that can then be analyzed and interpreted, and
- servicing as a structure to validate nutrition care and showing how the nutrition care that was provided does what it intends to do.

Working closely with the physician, you as a dietetic professional should determine an individual's nutritional therapy needs and plan of care. The relationship between the patient/client/group and dietetic professional is at the core of the nutrition care process.

Therefore, nutrition care provided by dietitians or other qualified dietetic professionals should always reflect both the state of the science and the state of the art of dietetic practice to meet the individualized needs of each patient/client/group.

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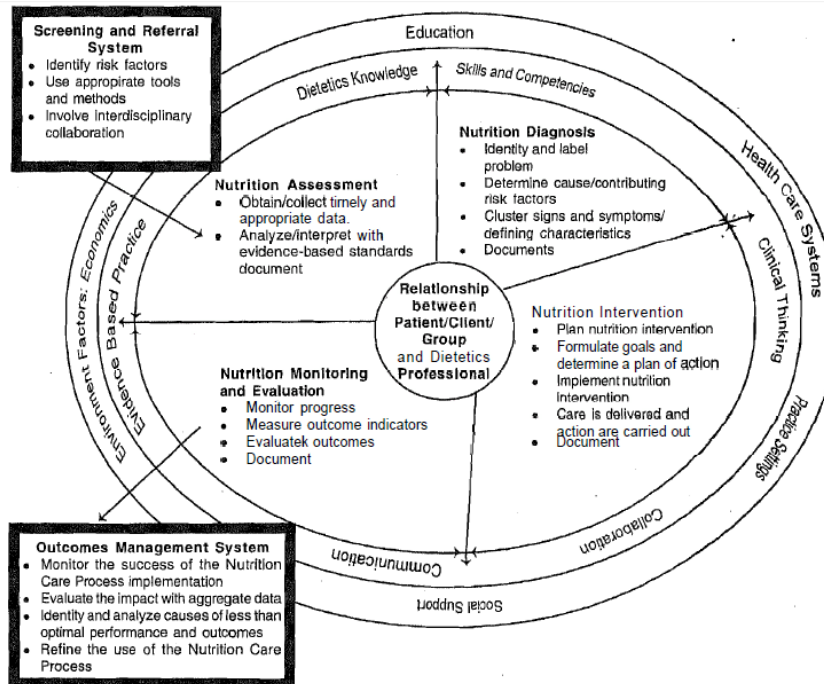


Figure 1.2: The nutrition care process and mode

The other factors that influence and impact on the quality of nutrition care are also highlighted in Figure 1.2. The strengths and abilities that dietetics professional bring to the process namely dietetic knowledge, skills and competencies, critical thinking, collaboration and communication, evidence-based practice are highlighted in the middle ring of the process.. Environmental factors particularly practice setting, health care system, social system and economics influence the process. The nutrition care process consisting of four distinct, but interrelated and connected steps include:

- a) Nutrition assessment
- b) Nutrition diagnosis
- c) Nutrition intervention, and
- d) Nutrition monitoring and evaluation

Documentation is equally important in the nutrition care process. Let us now discuss each of these steps in detail.

1.4.1 Nutrition Assessment

The nutritional care process, you would realize, begins with nutritional assessment. Nutrition assessment is the evaluation of an individual's nutritional status and nutrient requirements. It is a systematic process of obtaining, verifying, and interpreting data in order to make decisions about the nature and cause of nutrition-related problems. It is an ongoing, dynamic

process that involves not only initial data collection, but also continual reassessment and analysis of patients/clients/ groups needs. The purpose of nutrition assessment is to:

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obtain adequate information in order to identify nutrition-related problems, define accurately an individual's nutritional status, determine the level of nutritional support that individuals need, and monitor changes in the nutritional status and the effect of nutritional intervention.

How is this done?

This is based on the interpretation of information obtained from the diet history, medical history, review of symptoms and physical, clinical, examination, including anthropometric measurements and laboratory data. Often, this process is referred to as the 'ABCD' analysis, where:

A stands for Anthropometric measures: It measures growth in children and shows changes in weight in all populations that can reflect diseases and help to monitor progress in fat loss or gain. Box 2 included in this unit highlights some of these measures, particularly those which are important from the clinical and therapeutic nutrition point of view.

B for Biochemical investigations: These help to reveal nutrients and metabolites in blood and /or urine, and/or faeces that indicate an infection or a disease.

C for Clinical analysis: This analysis includes a complete physical examination and a medical history. The physical examination begins with the patient's general appearance.

Nutrition-oriented aspects of the physical examination focus on the skin, head, hair, eyes, mouth, nails, extremities, abdomen, skeletal muscle and fat stores.

D for Diet history and nutrient intake: This is used to evaluate diet for nutrient or food intake. Common methods used include the 24-hour diet recall, diet history, food frequency questionnaire, weighing method etc.

Nutritional assessment is important in the nutrition care process because acute and chronic malnutrition (both under and overnutrition) are common clinical findings. Malnutrition, we know, interferes with an individual's growth, development, general health and recovery from illness.

Box 2 Anthropometric Measures

Anthropometry involves obtaining physical measurements of an individual and relating them to standards that reflect the growth and development of an individual. Anthropometric measurements, therefore, involve taking physical

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measurements of the body, such as, height, weight, head circumference, girth measurement, or skinfold measurement. Anthropometric data are most valuable when they reflect accurate measurements and are recorded over a period of time. It is important to maintain proper equipments and careful techniques.

Three types of measurements are common in clinical practice — height, weight and body composition.

Let us study how are these useful for us and a few proper techniques for measuring these.

Both height and weight are the useful measures/indicators in determining nutritional status in adults. Height and weight measurements in children are evaluated against various norms. These are recorded as percentiles which reflect the percentage of the total population of children of the same sex who are at or below the same height or weight at that age. This allows the child's growth at every age to be monitored. Now let us see how we can measure length/height of children as well as adults.

Length -and Height — Measurements of height can be obtained using a direct or indirect approach. In the direct method, a fixed measuring stick is used against the wall or platform clinic scales are used. This is done when the person is able to stand while indirect methods are used for persons who cannot stand such as individuals with cerebral palsy or muscular dystrophy or those who are elderly. These indirect methods are arm span, recumbent length (that is, when one is lying down) and knee height measurements. Recumbent bed height measurements using a tape measure may be appropriate for institutionalized individuals who are comatose, critically ill or unable to be moved. Recumbent length measurements are also used for infants and children younger than 2 or 3 years of age. Careful measurement of length at each check-up hence, gives a clear indication of a child's growth rate.

Weight — As we have seen earlier, weight is a critical measure in nutrition assessment. It is used to assess children's growth, predict energy expenditure and protein requirements. Also, it helps to determine the body composition. Individuals should be weighed without shoes and in light clothing preferably on a beam balance scale. Body weight may be assessed by several methods including:

a) Ideal Weight for Height

Ideal weight for height can be determined from reference standards such as life insurance tables. Ideal weight for height can also be determined using the Hamwi method. According to this, the ideal weight for height for both the sexes is as follows:

- Females —100 lbs for the first 5 ft of height and 5 lbs for every inch over 5 ft.
- Males — 106 lbs for the first 5 ft of height and 6 lbs for every inch over 5 ft.

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Weight is then adjusted according to whether the person has a large or small frame as follows:

Large frame - Add 10%

Small frame - Subtract 10%

Now that you have understood how to find out ideal weight for height, we suggest you to calculate it for yourself. What do you derive out of this? Does the knowledge of Ideal Body Weight is of any use to you? Well, if you know your actual body weight, you can easily assess your nutritional status. Let us see how.

Significance of measured weights: Percent deviation from standard (percent ideal body weight) assesses the degree of malnutrition (underlover).

$$\% \text{ Ideal Body Weight} = \frac{\text{Actual Weight}}{\text{Ideal Body Weight}} \times 100$$

The table below presents you with criteria to determine nutritional status based on % IBW. Have a look at it and check it out for yourself.

Table 1.2: Criteria for assessing degree of malnutrition

IBW (%)	Interpretation of Nutritional Status
≥ 130 %	Obese
110% - 120%	Overweight
80% - 90%	Mild malnutrition
70% - 79%	Moderate malnutrition
< 69%	Severe malnutrition

b) Usual Body Weight

This may be a more useful parameter than ideal body weight for those who are ill. Comparing present weight to usual body weight allows changes in weight status to be assessed. A rapid weight loss or gain is significant.

c) Body Mass Index (BMI)

The Body Mass Index defines the level of adiposity according to the relationship in weight to height. It eliminates dependence on frame size. The formula for deriving BMI is:

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$$\text{BMI} = \frac{\text{Weight (in Kg)}}{\text{Height (in metres)}^2}$$

The BMI is a more accurate measure of body fat than weight alone. It is the quickest and most accepted measure of obesity.

The normal range of BMI is between 18.5 and 25. A BMI value less than 18.5 denotes under nutrition BMI values of 25-30 is considered overweight. Those with a BMI greater than 30 are obese. As a dietitian, you should routinely assess height and weight and determine BMI for patients under your care.

Before we move on to the study of body composition and other measurements, let us get to know another type of measurement, used for the anthropometric assessment of infants and children. It is referred to as head circumference. This measurement is taken using a flexible tape measure put snugly around the head. This measure is amongst other useful indicators of normal growth and development, especially from birth till age 3.

- **Body Composition I**

Various aspects of body size and composition can be measured which provide a good indication of body leanness and fatness in leanness measurements. The validity of these measurements depends on the accuracy of the measuring technique.

- **Skinfold Measurements**

These measurements serve a variety of purposes, the most important being indicator of body fat. As you are already aware that a significant amount of the body's fat stores are right beneath the skin (referred to as subcutaneous fat). Hence determination of the sizes of the skinfolds at various sites around the body can give a good indication of body fatness.

These measurements are useful in cases of illness. Can you think how? Well, this is because the maintenance of fat stores in a patient's body may be a valuable indicator of dietary adequacy.

Now how to determine these measurements?

These measurements are done with special calipers. Let us now have a look at the different types of skinfold measurements.

(a) Triceps Skinfold Measurements (TSF)

The TSF is measured with a caliper that measures the thickness of the skinfold over the triceps muscle of the arm not in predominant use. The thickness of the TSF gives an indication of subcutaneous fat and is considered an index of stored energy.

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Table 1.3: Triceps skinfold

Sex	Triceps Skinfold (mm)					
	Standard	90%	80%	70%	60%	50%
Female	16.5	14.9	13.2	11.6	9.9	8.3
Male	12.5	11.3	10	8.8	7.5	6.3

Percentages less than the standard, as indicated in Table 1.4, may be interpreted as mild, moderate or severe deficit.

b) Mid-Upper Arm Circumference (MUAC)

The MUAC measurement of the arm is taken with a metric tape measure at the midpoint between the acromion bone (a portion of the shoulder blade or the scapula that overhangs the rotator cuff and humerus— the upper arm bone) and the olecranon bones (the proximal part of the ulna bone which forms the elbow joint) on the arm not in predominant use. This measurement represents both muscle and fat stores. It is used to calculate mid arm muscle circumference (MAMC).

c) Mid-Arm Muscle Circumference (MAMC)

The mid-arm muscle circumference is used to estimate skeletal muscle mass and is calculated from the MUAC and the TSE

$$\text{MAMC (cm)} = \text{MUAC (cm)} - [3.14 \times \text{TSE (in cm)}]$$

Table 1.4: Mid-arm muscle circumference (PVLAMC)

Sex	Mid-Arm Muscle Circumference (cm)					
	Standard	90%	80%	70%	60%	50%
Female	23.2	20.9	18.6	16.2	13.9	11.6
Male	25.3	22.8	20.2	17.7	15.2	12.6

Percentages less than the standard, as indicated in Table 1.3, may be interpreted as mild, moderate or severe depletion.

d) Waist to Circumference Ratio (WHR)

This ratio differentiates between android and gynoid obesity. What do you understand by android obesity and gynoid obesity? Well, android obesity, also known as apple shaped fat distribution, refers to the centric fat distribution patterns with increased disposition towards the abdominal and waist area. While gynoid obesity refers to the fat distribution at the hips and thighs. It is also referred to as pear shaped fat distribution.

The waist circumference is the smallest circumference between the nipples and the top of the thighs. The hip circumference is the largest circumference between the waist and the knees. A WHR of 1.0 or greater in men and 0.8 or

greater in women is indicative of android obesity. This is an increased risk for obesity-related diseases.

e) Bioelectrical Impedance Analysis (BIA)

This is used for body fat analysis. BIA involves attaching electrodes to the extremities of a patient. A small electrical current is passed through the electrodes. Electrical and resistance measurements are obtained. It is a body composition analysis technique based on the principle that compared to fatty tissue, lean tissue has a higher electrical conductivity and lower impedance. Impedance is the opposition to the electric current and is the inverse of conductance. It is a safe, non-invasive and rapid means of assessing body composition. Though truncal fat cannot be assessed very accurately,

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	Normal Appearance	Signs Associated with Malnutrition	Possible Disorder or Nutrient Deficiency	Possible Non-Nutritional Problem
Hair	Shiny; firm; not easily plucked	Lack of natural shine; dull and dry Thin and sparse Dyspigmented Flag sign Easily plucked (no pain)	Kwashiorkor and less commonly, marasmus	Excessive bleaching of hair Alopecia
Face	Uniform skin colour; smooth, healthy appearance; no facial swelling	Nasolabial seborrhoea (scaling of skin around the nostrils) Swollen face (moon face) Paleness	Riboflavin Kwashiorkor	Acne vulgaris
Eyes	Bright, clear, shiny; no sores at corners of eye-lids; healthy, pink, and moist membranes; no prominent blood vessels or mound of tissue or sclera	Pale conjunctiva Bitot's spots Conjunctival xerosis (dryness) Corneal xerosis (dullness) Keratomalacia (corneal softening) Redness and fissuring of eyelid corners Corneal arcus (white ring around eye) Xanthelasma (small, yellowish lumps around eyes)	Anaemia (e.g., iron) Vitamin A Riboflavin, Pyridoxine Hyperlipidemia	Bloodshot eyes from exposure to weather, lack of sleep, smoke exposure, or alcohol
Lips	Smooth, not chapped or swollen	Angular cheilosis (white or pink lesions at corners of mouth)	Riboflavin	Excessive salivation from ill-fitting dentures
Tongue	Deep red in appearance, not swollen or smooth	Magenta tongue (purplish) Filiform papillae Atrophy or hypertrophy Red tongue	Riboflavin Folic acid Niacin	Leukoplakia

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Teeth	No cavities; no pain; bright	Mottled enamel Caries (cavities) Missing teeth	Fluorosis Excessive sugar in-take	Malocclusion Periodontal disease Health habits
Gums	Healthy, red, do not bleed; Not swollen	Spongy, bleeding Receding gums	Vitamin C	Periodontal disease
Glands	Face not swollen	Thyroid enlargement (front of neck swollen) Parotid enlargement (swollen cheeks)	Iodine Starvation Bulimia	Allergic or inflammatory enlargement of thyroid
Nervous system	Psychological stability, Normal reflexes	Psychomotor changes Mental confusion Sensory loss Motor weakness Loss of positional sense Loss of vibration Loss of ankle and knee jerks Burning and tingling of hands and feet (paresthesia) Dementia	Kwashiorkor Thiamine Niacin	Vitamin B ₁₂

Table 1.5: Physical signs of malnutrition

In the first step in the nutrition care process, we have learnt that assessing nutritional status is crucial. Assessment provides the foundation for the nutrition diagnosis which is the next step in the nutrition care process.

1.4.2 Nutrition Diagnosis

Nutrition diagnosis is the identification and labeling that describes an actual occurrence, risk of, or potential for developing a nutrition problem that dietetics professionals are responsible for treating independently. At the end of the assessment step, data are clustered, analyzed and synthesized. This will reveal a nutrition diagnostic category from which to formulate a specific nutrition diagnostic statement. Analyzing the assessment data and naming the nutrition diagnosis provide a link to setting realistic and measurable expected outcome, selecting appropriate interventions, and tracking progress in attaining those expected outcomes.

It is important to remember that nutrition diagnosis changes as the patients/ clients / groups response changes.

Once the nutritional care plan is formulated, it is easy to implement.

1.4.3 Nutrition Intervention

Nutrition intervention, the third step of the nutrition care process, is a specific set of activities and associated materials used to address the problem identified in the step above. Nutrition interventions are purposefully planned

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actions, designed with the intent of changing a nutrition-related behaviour, risk factor, environmental condition, or aspects of health status for an individual, target group or the community at large. This step involves

- a) selecting
- b) planning, and
- c) implementing appropriate actions to meet patient's/clients/groups nutrition needs.

The selection of nutrition intervention is influenced by nutrition diagnosis and provides the basis upon which outcomes are measured and evaluated.

Planning the nutrition intervention involves formulating and determining the plan of action. For this, we need to first prioritize the nutrition diagnosis based on the severity of the problem, safety, need of the patient etc. Next, identify science-based ideal goals and objectives. These objectives should be in behavioural form, realistic and should be appropriate to the educational levels, as well as, the economic and social resources of the patients and their family. Further, determine patient-focused expected outcomes for each nutrition diagnosis. The expected outcomes are the desired change(s) to be achieved over time because of nutrition intervention. For example, Increasing or decreasing weight, blood pressure, laboratory values etc. These expected outcomes should be clear and concise and should be written in observable and measurable terms. Finally, defining and selecting specific intervention strategies that focus on the etiology of the problem is included in the planning phase.

Once we have formulated a plan of action, the next component is to implement the action plan i.e. care is delivered and actions are carried out. Implementation translates assessment data into strategies, activities or interventions that will enable the patient to meet objectives established. This might include prescribing a diet, nutrition counseling and educating the patient, providing food or nutritional supplements or changing the mode of feeding, and advice on financial or food resources. The care process is a continuous one. The initial plan may have to be altered as the condition of the patient changes or as and when new needs are identified. Interventions should be specific to established problems or objectives.

Implementation, therefore, is the action phase of the nutrition care process. In the implementation phase the dietetic professional communicates the plan of nutrition care, carry out the plan of nutrition care, and continue data collection and modify the plan of care as required. We as dietetics professionals may actually do the intervention, or may include delegating or coordinating the nutrition care with other providers. We need to work collaboratively with the patients/client/groups, family or caregiver to create and implement

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a realistic plan that has a good probability of positively influencing the diagnosis/problem. This client-driven process is a key element in the success of this step.

Having implemented the nutritional care plan, we also need to monitor and evaluate to assess where we are and whether we are on track or not. This is the last step in the nutrition care process.

1.4.4 Nutrition Monitoring and Evaluation

Monitoring and evaluation is an essential step in the nutritional care process and is very important too. Monitoring specifically refers to the review and measurement of the patient/client/group status at a predetermined follow-up point with regards to the nutrition diagnosis, intervention plan, goals and outcomes. Evaluation, on the other hand, is the systematic comparison of current findings with previous status, intervention goals or a reference standard.

The purpose of monitoring and evaluation is to determine the degree to which progress is being made and goals or desired outcomes of nutrition care are being met. This step makes the nutritional care plan effective and responsive to the patient's needs.

The purpose of monitoring and evaluation is to determine the degree to which progress is being made and goals. This step makes the nutritional care plan effective and responsive to the patient's needs.

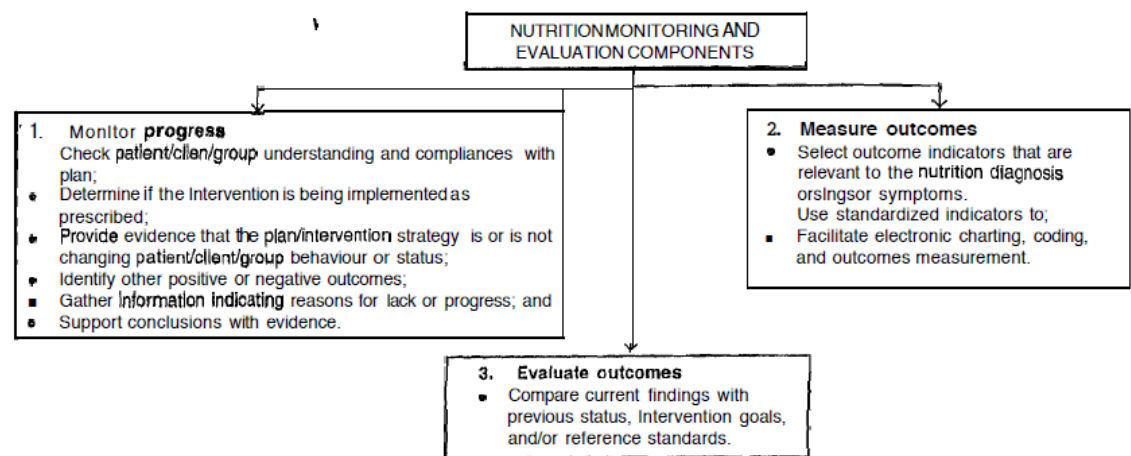


Figure 1.3: Nutrition monitoring and evaluation components

An evaluation of the extent to which the patient's nutritional requirements

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are being met can be done for example by means of the nutritional index (NI). This index calculates the extent to which an actual intake of a specific nutrient meets the recommended/desirable intake for a particular patient. NI can be calculated as:

$$NI = \frac{\text{Actual Intake of the Nutrient} - \text{Desirable Intake}}{\text{Desirable Intake}}$$

Now, how to interpret the value obtained from NI. If the actual daily intake exceeds the desirable intake, the NI is stated as a positive percentage while if less than the desirable intake, then it is stated as a negative percentage. Several negative NI days is an indicator of objectives not being met and that the care needs to be evaluated and changed.

Besides nutrition monitoring and evaluation, documentation too is an important part of the nutrition care process.

1.4.5 Documentation

Documentation is an essential aspect of the nutrition care process. It helps the patient to understand the nutritional care plan and their role in this process. It helps to ensure that nutritional care will be relevant, complete and effective. It also serves as a communication with the other members of the health care team. The documentation should be complete, clear, concise, legible and accurate. A format frequently used for medical record documentation is the problem-oriented medical record (POMR). This provides a vehicle for recognition of all the patient's problems and for coordination of the activities of all members of the health care team. It consists of four major parts — the database, a problem list, the initial care plan and progress notes.

Entries into the medical record can be done in many styles. One of the most common forms is the 'SOAP' note (Subjective, Objective, Assessment and Plan). Various health professionals, dietitians, physicians, nurses and social workers routinely collect much of this information. This entire team of health care professionals ensures that all aspects of nutritional care are noted in place as a part of the total health record. Let us see what SOAP is:

Subjective — the data includes information obtained from the patient or the patient's family regarding the problem.

Objective data — the data is gathered from tests, analysis, diagnostic procedures and observations by health care team.

Assessment — interpretation of patient's status is based on subjective and objective data.

Plan — specific plans are stated for dealing with each problem such as specific treatment plans, nutritional care plans, modified diet, nutrition counseling goals etc.

The clinical dietician should document the actual nutritional care provided. This should include the type of diet, adjustments for intolerance of the diet and diet instructions given.

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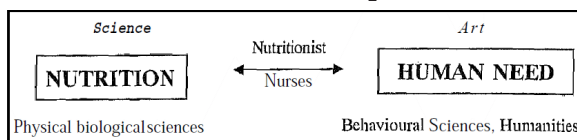
1.5 IMPORTANCE OF COORDINATED NUTRITIONAL AND REHABILITATION SERVICES

In our previous section, we learnt about the four step nutritional care process and the advantages of planning and documentation involved in such a process. Here, in this section, we shall deal with the rehabilitative services and the nutritional care for the patient. What do we mean by the terms rehabilitation and nutritional care? What is their significance and what is the role of health care professionals, especially dietitians in providing these services to the patients. Also, how can patients effectively utilize these services?

These are a few of the issues with which we are going to deal in this section.

So let us first understand the meaning and significance of nutritional care and rehabilitation

Nutritional care can be recognized as both a science and art. Can you tell why? It is considered as a science because the rapid advances in scientific knowledge provides all health care providers with a strong foundation on which the professional practice is based. Advances in nutritional sciences provide for such a base for nutritional care that is comprehensive, collaborative and continuing. It can be referred to as an art (an exceptional ability to conduct human activity) since it involves knowing and caring about people and their needs. Hence, it becomes all the more essential and imperative that all health care practitioners must base their work on sound scientific knowledge as well as, their patient's needs. Therefore, you would realize, as well as, appreciate the collaborative efforts in many aspects of both nutritionist and nurses working together and functioning as catalysts. This significant role that brings scientific knowledge and skill together to bear on patient's nutritional needs can be represented as:



All individuals want to have a long and healthy life. There is no doubt that adoption of healthy nutrition lifestyle and the practice of good nutrition habits would help eliminate many health problems caused by malnutrition. Here, the role of a health professional or a dietitian comes into picture. A health professional needs to have a sound knowledge about nutrition and must be

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able to apply the principles of sound nutrition practice to cater to the needs of patients. These include all diet-related questions and complaints to which clear and simple explanations must be provided by the health professional.

In many cases, a patient has to undergo diet therapy which becomes a part of their medical treatment. In such instances, the eating habits need to be changed, and the patient will require advice or instructions for the dietary modifications. This information is provided to them by a dietitian and from other health care professional.

The dietitian and the nurse hold unique position on the health care team in relation to the patient's nutritional needs. Their roles are enhancing as their team responsibilities expand. The dietitian determines nutritional care needs in relation to medical diagnosis and care, as well as, individual patient needs. The nurse assists the dietitian with this nutritional care applying it in the general nursing care. By this, we can realize that in many respects, these two health professionals are closest to the patient and the family and have the opportunity to determine many of the patient's needs. They are the ones who coordinate services and help the patient understand and participate in personal care. Hence, individualized care must be the focus of therapy. In fact, the doctor, nurse and dietitian working together as a team provide the best possible nutritional care.

Now let us move on to the concept of rehabilitation.

You would realize that some patients have problems that seriously limit their ability to function normally in everyday activities. In such cases, special planning can help them achieve and maintain their optimal level of functioning. The care that aims to prevent further disabilities and to restore function is called as rehabilitation. The planning that emphasizes rehabilitation is often beneficial to the clients with cardiovascular, respiratory and neurologic disorders. Nutritional rehabilitation focuses on maintaining adequate nutritional status and adjusting daily activities related to eating.

The patient is the focus of the team endeavour and must be included as an active and participating member. The patient himself is the one who probably has the greatest interest in his/her care plan. She/he can work better with health team members if he is informed about his current nutritional status, the relationship between his food habits and nutritional status to his health. He is also needed to be informed about the care services, which the various health team members will provide for him and the resources available for us. Sharing the nutritional care plan and goals with family members helps in clarifying their role in assisting the patient. This plan also facilitates the communication between health-team members such as nurses and dietitians who work as colleagues and frequently meet to discuss the patient's nutritional needs. Often, the communication process involves consultations and referrals among health team members. The consultation is provided to

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a patient from a dietitian or nutritionist who develops a care plan to assist the Client to make more appropriate food choices. While referrals are the written verbal information about the patient's problem and/or nutritional needs. The purposes behind making the referral are many. Let us see what these are. Referrals are made for:

- A specific kind of therapy
- Rehabilitation or training
- Education, and
- Special community services

Now, what is the information that must form a part of the referral? Well, one should first identify the patient's problem or need. This must be followed by his food habits, appetite, nutritional needs and diet instruction, and special instructions for feeding. Having looked at the importance of nutritional care and rehabilitative services, next let us focus on the other important aspect of patient care i.e. nutrition/diet counseling.

1.6 PATIENT CARE AND COUNSELING

Since past three decades, there has been an increased emphasis on setting standards of practice to ensue the delivery of quality patient care. An increased focus has been there on cost control in health care settings, for effectively evaluating patient care programmes based, on two factors — cost effectiveness and provision of nutritional services. Within dietetics, models of quality patient care have standards for identifying patients requiring increased nutritional support or education, determining patient care priorities and spelling out the degree of care required with increasing concerns about health care costs.

Further, counseling is one of the most useful methods for assisting an individual to arrive at a solution of his/her problems. In this section, we shall get to know about the patient care process and the science and art behind dietetic counseling. So let us get started to learn about patient care.

1.6.1 Patient Care

The primary basic principle in nutritional practice to be valid must be person/patient- centered. It must be based on initial and continuing identified needs and updated constantly with the patient, in order to provide essential physical care and support personal needs for maintaining self-esteem. The health care team in this process, as you would already know, involves a physician, dietitian, nurse and Other health care professionals. There are 5 distinct yet constantly interacting phases in the care process. These include:

1. Assessment: A broad base of relative information about the patient's

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nutritional status, food habits, and life situation provides the necessary knowledge for making valid initial assessments. Useful information may come from a variety of sources, such as the patient himself, patient's chart, family, relatives, friends, hospital staff and related research.

2. **Analysis:** The data collected must be analyzed to determine specific patient needs, on the basis of which a list of problems may be formed.
3. **Planning Care:** The plan for care must always be based on personal needs and goals of the patient, as well as, on the identified medical care requirements.
4. **Implementing Care:** The patient care plan is put into action according to realistic and appropriate activities. In this case, nutritional care and education will involve decisions and actions.
5. **Evaluating and Recording Care:** The results are checked carefully (with each activity being carried out) to see if identified needs have been met. Hence any appropriate revision of the plan can be made as needed for continuing care, These results are recorded in the patient's medical record. A clear documentation of all the activities is essential.

With a brief knowledge about the patient care process, we now move on to a detailed overview on counseling its scope, process and approaches.

1.6.2 Counseling

The term 'counseling' or 'nutrition/ diet counseling' is a broader term than teaching. It is one of the most useful methods for assisting an individual to arrive at a solution of his/her problems. It is a personal meeting of two individuals—the counselor, who assists in analyzing and understanding the problem and the counselee, who has a problem and needs assistance in arriving at a solution for this problem. It has been described as: (1) an internal process for the counselee, (2) a sequence of events, and (3) the elements of interpersonal relationship between counselor and counselee.

What is the role of counseling in patient care? Does it help to improve the existing state of the patient? What is the role of dietitian in it? Let us read and find out.

Nutrition or diet counseling is a primary educational activity of the dietitian. It incorporates the idea of working with a patient, encouraging him to make changes in his pattern of living that he sees as desirable and attainable and supporting him through- out the process. It is a process that assists people in learning about themselves, their environment and methods of handling their roles and relationships. It involves problem solving, identifying goals and change, counselor assist individuals with the decision-making process, resolving interpersonal concerns and helping them to learn

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newways of dealing with and adjusting to life situation. Counseling aims to help clients make and sustain desired changes over time. It is based on two premises:

- each person controls his own life and behaviour, and
- each individual has a background of personal interactions, socialization and education that he/she uses to make choices about their behaviour.

Counseling is explored as a four-stage process. The first stage concentrates on the development of a trusting, helping relationship between the counselor and the counselee. The remaining three stages focus on problem-solving, Dietetic counseling includes in its scope behaviour modification, counseling and cognitions, nutrition counseling and multicultural communications.

The health professional, including the dietitian uses the knowledge and skills to assist patients to identify problems, discover and list possible solutions, consider the consequences of each alternative, choose a solution and incorporate it into their daily activities.

In most instances, it is important to outline a plan to provide patient education or counseling. Some of these areas include:

- i) reinforcement of sound eating habits,
- ii) positive suggestions to improve poor habits,
- iii) discussion of reasons for diet modifications,
- iv) guidance and practice in planning meals meeting specific diet modifications,
- v) training in various feeding techniques, and
- vi) explanation of various assessment and treatment techniques.

There are almost 40 different therapy models or approaches but a few are most commonly used. Let us then understand various prevalent theories and approaches that are relevant to counseling.

Theories and Approaches Relevant to Counseling

Few theories and approaches relevant to counseling are reviewed herewith.

Reality Theory

Developed in the 1960s by William Glasser, a psychiatrist, reality therapists view human nature in terms of behaviour. They believe that human behaviour is motivated by two common basic needs: (a) the need to love and be loved, and (b) the need to feel worthwhile to ourselves.. and others. People are responsible for their behaviour and behaving in a responsible manner helps people fulfill their needs. Clients are helped and encouraged to make value judgments about their own behaviour. Once the chosen behaviour is viewed as responsible, clients' feelings about their behaviour tend to become positive.

This approach can help the dietetic practitioner to use a structure approach for assisting a client to change inappropriate eating behaviours.

Behavioural Counseling

This evolved from the early theories of behaviourism. The focus is on examining current behaviours and learning new ones. It is believed that feelings and thoughts may come before the behaviour, nor after. For example, a person feels upset, so he/ she eats.

Cognitive — Behavioural Approaches

They include psychoeducation and rational-emotive therapy. The goal is to identify problem behaviour and irrational beliefs and then to design strategies for immediate action plans. Psychoeducational therapy specifically involves a process of learning about oneself, gaining self-understanding and self-knowledge.

Once the client has progressed in the understanding, he/she will be in a position to regulate his/her behaviour in accordance with some standard. This therapy is intended to teach the individual to 'manage physical and mental impulses.

The rational-emotive therapy is based on the premise that negative self-talk and irrational ideas are a major cause of emotion-related difficulties. The therapy aims to provide the client with an insight to stimulate logic and emotion simultaneously in the direction of the planned change. For example, when working with a patient with high serum cholesterol, the dietitian would help the client to: (a) think that the cholesterol levels are very high, (b) feel emotions like concern or fear in order to get the patient to make an effort toward change and sustain it, and (c) to encourage patient that foods lower in fat and cholesterol are to be preferred.

The Family Nutrition Approach

This involves relatives/family who live in the client's household; in assisting the client to make necessary dietary changes to prevent or to control diet-responsive diseases and to maintain client adherence to nutrition advice over the long-term. Family counseling is used very commonly when working with children and adolescents. Family counseling is appropriate where the client's problems are related to his relationship or function in the family. Working with the family helps to achieve improvement faster and prevent lapses than if you treated/worked with the person alone.

Directive and Non-directive Counseling

Directive Counseling tends to be appropriate when the counselor is aware of the problem and/or is concerned about the behaviour of the patient but

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the latter is unaware about the problem and is avoiding acknowledging it. In contrast, non-directive counseling is more appropriate when the patient or the counsellee has insight and says that the counselor's help is needed to solve the problem.

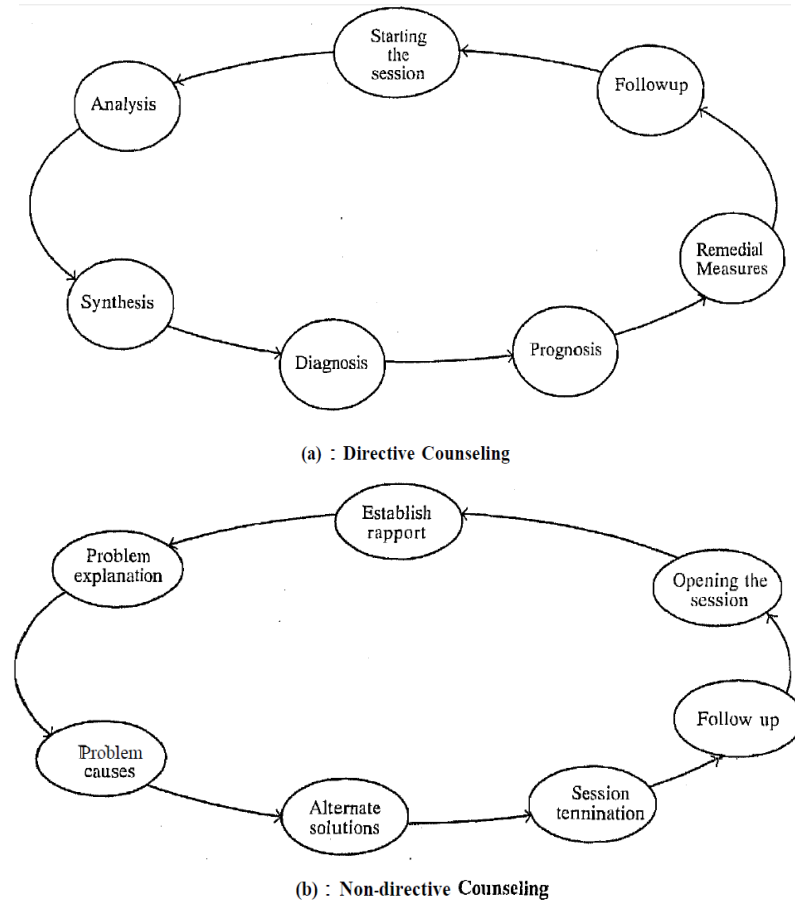


Figure 1.4: Directive and non-directive counseling process

The non-directive approach is often called 'client-centered'. A basic assumption is that humans are basically rational, socialized and realistic. If a person's needs for a positive regard from others and for positive self-regard are satisfied, the individual can realize the inherent tendency he/she possesses towards realizing their potential for growth and self-actualization. Counseling releases the potentials and capacities of the individual.

One of the assumptions is the relationship between the counselor and the client. The client cannot be helped only by listening to the knowledge the counselor possesses or to the counselor's explanation of the client's behaviour or the personality. Prescribing "cures" or corrective behaviours are not considered to be of a lasting value. The relationship that is most helpful is the one that enables the patient (client) to discover within himself/herself the capacity to change and grow. Using this relationship four specific characteristics are desirable: acceptance, congruence, understanding and

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the ability to communicate these to the clients.

The counselor should accept the clients as individuals, as they are. When a counselor accepts the person unconditionally and non-judgmentally, then the patient begins to trust the counselor. Note: Trust is focused on predictability, genuine concern and faithfulness.

Good counselors are integrated, consistent with no contradictions between what they say and what they are. The counselor's verbal and non-verbal behaviours should be consistent. Empathy is essential to non-directive therapy. Thus, the counselor needs to be a good listener, have intuition, provide feedback on the data, feelings, as well as, provide motivation and inspiration.

In directive counseling, the counselor initiates the discussion. Clients tend to be more likely to become resistant or defensive. Thus the counselor should be very sensitive to all verbal and non-verbal behaviour, as well as, supportant. Such a relationship (directive counseling) is more appropriate between managers and subordinate rather than between dietitian and client. In general, directive counseling techniques are used to expose poor employee performance about which employees are unaware or unwilling to expose it themselves.

Let us next learn about the different counseling strategies.

The counseling strategies which may serve to be useful are described herewith.

Counseling Strategies

The counseling strategies which may serve to be useful are described herewith.

- Individual Counseling: Individual counseling is personal counseling. The first step in this is to establish a sense of trust and a therapeutic alliance with the patient so as to ensure a productive counseling session. A counselor can use several techniques to enhance the process of learning. These are:

- Clarify goals at the beginning of the session

- Start instruction in a positive manner

- Approach the patient in a competent, quietly enthusiastic manner

- Keep the session patient-centered

- Focus on the topic to be covered

- Adjust counseling approach as the need arises

- Find out if the client understands what he is being told

- Give honest, sincere praise for successes

- Use teaching techniques that impart on more than one of the client's senses and actively involve him.

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- **Group Cocounseling:** Group counseling is a technique where a group of person is counseled by employing group interaction method for arriving at a solution to the problem common to the group. All the group members are given an opportunity to discuss their problems together, in a free atmosphere. .

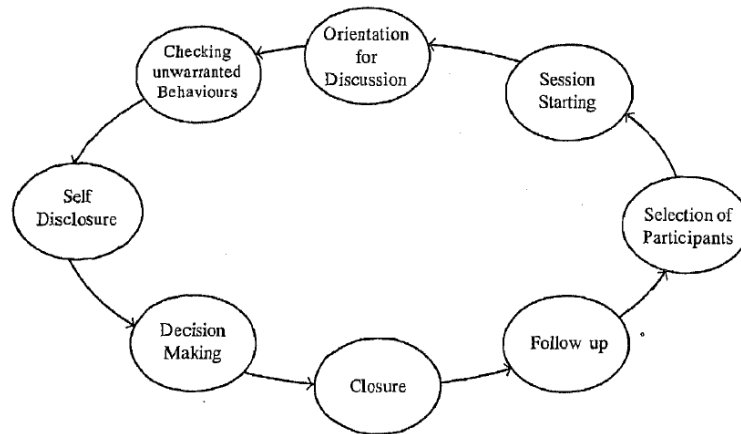


Figure 1.5: The group counseling process

Group counseling can be provided via formal classroom sessions or small group sessions. An active participation of group members facilitates the process of learning. Specific techniques are used for the purpose of instruction and these could be a lecture either with or without additional teaching aids or a role-play, demonstration and practical sessions.

An important strategy could be to conduct small group meetings for behavioural change encouraging full participation. The group atmosphere provides support and motivation to members to help them achieve their individual goals. Recommended actions often seem more acceptable when group members tell how they were helped by those actions. Also, learning in groups is sometimes more interesting and 'fun' than in a one-to-one setting. However, a person benefits from a group only if he can identify with it. In cases, such as these, individualized nutrition counseling sessions are preferred. So you would have realized how much important it is to motivate the patients and to maintain their interest during counseling sessions and ensure behaviour modifications. Let us next have a look at a few factors which play a major role in contributing towards motivation.

Psychological factors: Depression, anxiety or phobia induced by illness, life-style changes or medication effects, may hinder the ability to comply with the desired health behaviour changes.

Psychosocial factors: These may prevent patients from expressing concern for their health. They may lack confidence in the health professional or simply be unable to cope with dietary changes at that particular point of time because of the degree of illness or personal problems.

Physical factors: The drugs or illness may induce pain, fatigue or depression

which might block desire or ability to follow health care instructions.

Personal factors: There may be a language barrier or a lack of transportation or money for clinic visits.

Counselor-related factors: A personality conflict may exist between the patient and counselor.

Next, let us review a few tips for counseling children and elderly.

Children:

Assess the child's stage of development.

Adjust counseling for a child's dependency needs, lack of experience and the development tasks faced by him.

Have a cheerful and enthusiastic approach for better adaptation of nutrition and dietary practices.

Provide them with opportunities to learn by playing games, painting, reading stories, using puppets, handling and tasting food.

Since they have not developed many ingrained habits, they learn more quickly and so teaching must be made more interesting.

Elderly:

Emphasize and build on established dietary practices and attitudes.

Focus on the positive influence of a good diet so as to motivate them on making changes in the dietary habits. The focus, in most cases must be on good health.

Encourage their full participation in counseling programme.

Establish rapport with them so as to ensure their interaction and discussion on various issues.

Utilize the benefits of group discussions to bring more reluctant persons into the group and reduce anxiety related to educational programme.

Concerning an appropriate mode of feeding and the training and education needs for the patient, staff and family to carry it out.

In this section we learnt about the significance and methods of patient counseling. We end our discussions on this chapter here about the nutrition care process.

1.7 LET US SUM UP

In this unit, you have learnt how to render effective nutritional care in therapeutic nutrition. The primary goal of diet therapy is to achieve or maintain optimal nutrition status. The nutrition care process is a systematic and logical approach to ensure effective and successful nutrition intervention. The basic steps in the process include assessing nutrition status, interpreting

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assessment data to determine nutrient requirements, developing a plan of action for nutritional needs, implementing and evaluating the plan along with documentation of the entire process.

Further, the unit focused on the scope, process, approaches and strategies common to dietetic counseling.

1.8 GLOSSARY

Android obesity	: the centric fat distribution pattern with an increased disposition towards the abdominal and waist area.
Anticonvulsants	: drugs that prevent, reduce or stop convulsions or seizures.
Cerebral Palsy	: a group of chronic conditions affecting body movements and muscle coordination.
Clinical dietetics	: the application of dietetics in a hospital or health care institutional setting.
Diet history	: a review of an individual's usual pattern of food intake and the food selection variables that dictate the food intake.
Diet therapy	: the role of food and nutrition in the treatment of various diseases and disorders also known as therapeutic nutrition.
Dietetics	: a science and art of feeding individuals based on the principles of nutrition.
Gynoid obesity	: the fat distribution as the hips and thighs.
Leukoplakia	: a precancerous lesion that develops on the tongue to the inside of the cheek as a response to chronic irritation.
Medical Nutrition Therapy	: the assessment of the nutritional status of a client followed by nutrition therapy ranging from diet modification to the administration of enteral and parenteral nutrition.
Muscular therapy	: a group of diseases involving muscle deterioration.
Nutritional index	: the extent to which an actual intake of a specific nutrient meets the recommended desirable intake for a particular patient.
Oncology	: the branch of medicine concerned with the study and treatment of tumors.

Over-the-counter drugs : drugs those are available without a prescription.

1.9 CHECK YOUR PROGRESS

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- 1). Define the following terms:
 - a) Dietetics
 - b) Medical Nutrition Therapy
 - c) Therapeutic Nutrition
- 2). Diet and nutrition plays a crucial role in the health and well being of people. Explain.
- 3). What is a nutritional care process? List the steps involved.
- 4). What do you understand by ABCD analysis?
- 5). What is meant by SOAP note?

ADAPTATION OF THERAPEUTIC DIETS

STRUCTURE

- 2.1 Learning Objective
- 2.2 Introduction
- 2.3 Therapeutic Diets
- 2.4 Types of Dietary Adaptations for Therapeutic Needs
- 2.5 Normal Nutrition : Base of Therapeutic Diet
- 2.6 Diet Prescription
- 2.7 Constructing Therapeutic Diets
- 2.8 Routine Hospital Diets
- 2.9 Mode of Feeding
- 2.10 Let Us Sum Up
- 2.11 Glossary
- 2.12 Check Your Progress

2.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- discuss the purpose(s) of therapeutic diet adaptations,
- explain the different ways by which the normal diet can be modified to suit therapeutic needs,
- plan a diet prescription,
- describe the principles of general hospital diets — normal, liquid and soft diets, and
- elaborate on the different modes of feeding.

2.2 INTRODUCTION

We already discussed the role of a clinical dietitian and the various steps or processes necessary for rendering effective nutritional care.

This unit deals with therapeutic diets and the ways in which the normal

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diet of an individual can be modified to suit therapeutic needs. The clinical dietitian is educated and trained to interpret the science of nutrition to enhance the quality of life of individuals and groups in health and disease. Each diet prescribed for an individual has its own rationale and purpose. You as a dietitian should have the knowledge, skills and attitudes to ensure quality of work.

In this unit, we shall learn about different therapeutic diets that are available in hospital for specific disease conditions. Also, we shall deal with the different modes of feeding, through oral enteral and parenteral route.

2.3 THERAPEUTIC DIETS

Therapeutic diets are adaptations of the normal or regular diet. In other words, it is a diet for a patient suffering from a specific disease such as heart failure, hypertension, renal failure, diabetes etc.

Why do you think that the diet has to be changed in these conditions? What changes must be frequently made? These are a few issues which we shall deal in this subsection.

Well, you already know that there are certain diseases which can be cured by food or nutrient concentrates, such as deficiency diseases. In diseases such as diabetes, making alterations in the diet can help to control the extent of the disease and prevent the onset of complications.

Similarly, in genetic diseases simple dietary modifications can keep a check on the progression of the disease and symptoms which otherwise could be fatal. The disease process also influences both the quality and quantity of the diet. The other aspects that may require changes include meal frequency. These changes result as a consequence of the following reasons:

- loss of appetite and therefore low intake,
- feel more hungry and therefore an increase in the intake, and
- problems with mastication, swallowing, digestion or absorption of food or specific nutrients (due to structural and/or functional changes) leading to changes in types of food that can be tolerated, as well as, feeding frequency.

Purpose of Therapeutic Dietary Adaptations

A therapeutic diet is a quantitative/ qualitative modified version of a basic nutritious diet which has been tailored to suit the changing nutritional needs of a patient/ disease condition. The regular or normal diet may be modified for one or more of the following reasons:

- to maintain or restore optimum nutritional status,
- to provide rest or relieve an affected organ (e.g. soft or liquid diet in

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gastritis),

to adjust to the body's ability to digest, absorb, metabolize or excrete (e.g. a low fat diet for fat malabsorption),

to adjust to tolerance of food intake by mouth (e.g. tube feeding for patients with cancer of oesophagus),

to adjust to mechanical difficulties (e.g. soft diet for patients with denture problems), and

to increase or decrease body weight/body composition (e.g. high calorie, low calorie etc.).

While going through the above points you must have come across the terms like soft diet, liquid diet, tube feedings etc.

What are these and what do we mean by these? Don't panic, we shall get to know about these terms later in this unit.

The modified diet may reduce symptoms, make the patient more comfortable or improve the quality of life.

2.4 TYPES OF DIETARY ADAPTATIONS FOR THERAPEUTIC NEEDS

Normal nutrition is the foundation upon which therapeutic modifications are based. We have already discussed in previous sections about the purpose of dietary adaptations. The adaptations of the normal diet to suit therapeutic needs may take the following forms:

Change in consistency of foods, such as liquid diet, soft diet, low fibre diet, high fibre diet.

Increase or decrease in energy value of the diet such as low calorie diet for weight reduction, high calorie diet for burns.

Increase or decrease in specific nutrients or type of food consumed, such as sodium restricted diet, lactose restricted diet, high fibre diet, high potassium diet.

Elimination of spices and condiments, such as bland diets.

Omission of specific foods such as allergy diets, gluten free diet.

Adjustment in the ratio and balance of proteins, fats and carbohydrate such as diabetic diet, ketogenic diet, renal diet and cholesterol-lowering diets.

Rearrangement of the number and frequency of the meals such as diabetic diet, ostectomy diet, diet for peptic ulcer disease.

Test diets: These are single meals or diets lasting one or few days that are given to patients in connection with certain tests e.g. the fat absorption test used to determine if steatorrhoea is present.

Change in feeding intervals i.e., meal frequency.

Having reviewed the types of dietary adaptations next let us get to know the basis of planning therapeutic diets.

2.5 NORMAL NUTRITION : BASE OF THERAPEUTIC DIET

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Normal nutrition is the foundation upon which the therapeutic modifications are based. The primary principle of diet/nutrition therapy is that it is based on the patient's normal nutritional requirements. Any therapeutic diet is only a modification of the normal nutritional needs of an individual to suit what his/her specific condition requires. A person's 'diet' is defined as that person's intake of food and drink. All detailed dietary modifications should be presented with choices, clear guidelines, menu guidance and supporting information as to alternatives possible.

Patients should be encouraged to understand the key relationship between a food and a diet. The value of a food depends on the amount of nutrient in the food and the frequency with which the food is consumed. Dietary changes necessary are more likely to be followed if clear explanations and simple instructions are provided as to why the diet has to be changed. The Recommended Dietary Allowances (RDA) are often used as a basis for evaluating the adequacy of therapeutic diets. Nutrient requirements specific to a particular disease state or a disorder must be kept in mind when planning the diet.

As a dietitian, you also need to remember that an individual's diet is affected by various factors such as lifestyle, income, knowledge, taste preferences, religious beliefs and various other socio cultural factors. Failure to account for these could result in an impractical therapeutic diet planning.

2.6 DIET PRESCRIPTION

The diet prescription designates the type, amount and frequency of feeding based on an individual's disease process and disease management goals. The disease may require a calorie level or other restriction to be implemented. It may also limit or increase various components of the diet such as carbohydrate, protein, fat, vitamins, minerals, fibre, phytonutrients or water. Another aspect which the dietetic prescription takes into account includes the economic status, food habits (such as vegetarian, ovo- vegetarian, non-vegetarian), food intolerances (such as lactose intolerance, gluten- sensitive enteropathy), allergy (such as milk, eggs), occupation of the patient and meal timings. Next, let us have a look at each of these.

- **Economic Status** : It is one of the important practical considerations to be

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kept in mind while formulating a diet prescription. During an acute illness, a few expensive items may be permissible but for more prolonged or chronic illnesses like diabetes or peptic recommended foods must be within the means of the patient.

- **Food Habits :** These must be known so that the diets can be recommended keeping in mind the food preferences of the patient. Whether a person is a vegetarian or not, must be known. If vegetarian, then the degree of vegetarianism should be assessed. For instance, ovo-vegetarians eat egg but no flesh: egg and fish vegetarians eat just egg and fish but not animal flesh. While home-vegetarians prefer to remain vegetarian at home but consume meat/chicken at a restaurant or a party.
- **Food Intolerances :** The intolerances of the patient for specific food items must be assessed. For example, milk may lead to diarrhoea in some people while constipation in others. Those with colonic disorders are likely to get flatulence with whole pulses. Hence, while prescribing the diet, the food intolerances must be clearly indicated and known to the dietitian.
- **Allergy :** Food allergies manifest themselves as urticaria, abdominal cramps or bleeding asthma and angioedema. Many are found to be allergic to milk or egg and these foods may have to be excluded from the diet of the patient as you will learn later in Unit 6 in this course. Gluten enteropathy (celiac disease) may result as a consequence to gluten (a protein fraction in cereals and millets) sensitivity and colitis in young children due to milk consumption.
- **Occupation and Meal Timings :** The occupation and the time at which the meal is consumed daily must be considered. A factory worker who works on different shifts requires more detailed information for a peptic ulcer diet than a manager whose hours of work are fixed.

Next, let us, get to know how assessment of nutrient intake is done based on the patient's state of health.

Energy Allowance : The patient's requirement for energy varies with the physical activity and physiological condition. For instance, consider a person confined to bed. He tends to consume less than the one undergoing physical exertion. An example of a physiological state that leads to an increase in caloric needs is fever. A diet high in calories is indicated for undernourished patients. They are advised to take more of energy-dense foods such as starchy foods, sweets, cereals, butter and oils. While a low-calorie diet is indicated for all obese patients. The patient is encouraged to eat three meals a day, not to eat in-between the meals and to avoid energy-dense foods. These consist of raw and cooked vegetables, fruits, egg, meat, fish, chicken and skimmed milk with a low intake of cereals. An individual's energy requirement can be determined calculating either:

- required number of Kcal/kg/day OR
- percentage increase over basal metabolic demands.

You can estimate the basal energy expenditure (BEE) from anthropometric

data using the following Harris-Benedict formula:

$$\text{For men: BEE} = 66 + (13.7 \times W) + (5 \times H) - (6.8 \times A)$$

$$\text{For women: BEE} = 65.5 + (9.6 \times W) + (1.85 \times H) - (4.7 \times A)$$

where, W = kg body weight, H = height in cms and A = age in years.

An additional factor is added depending on the activity level of the patient. Another factor may also have to be added if the patient is under physiologic stress.

Mild stress - over BEE

Acute infections or burns — may require 100% over basal.

You should determine the actual energy requirements based on the assessment of the individual, his/her activity and his/her medical condition.

Carbohydrates: Carbohydrates provide bulk to the diet and along with fats, form the chief source of calories. The comparatively inexpensive form of carbohydrates in a high calorie diet can include chapaties, bread and biscuits. In a low-calorie diet these must be used sparingly.

Protein: Once the energy requirements have been estimated, protein requirements can be addressed. The aim is to achieve nitrogen balance. There are several factors influencing protein requirements and these include total energy intake, the metabolic state of the patient and protein losses. However, it is important to keep in mind that protein synthesis requires energy. The RDA for protein is 0.8 g to 1.0 g/kg body weight for adults. The actual minimum amount of protein needed to maintain nitrogen balance in healthy adults is 0.5g/kg, The requirement varies with specific disease states or protein needs related to specific conditions or illnesses, For instance, a larger amount of proteins may be needed during severe protein wasting, such as enteropathy or extensive drainage from wounds and fistulas. Protein restriction may be needed in acute renal failure or hepatic insufficiency..

Patients who require high protein diets are encouraged to drink 600-800 mL of milk a day. Now, you must be wondering how one can consume such a large portion of milk daily.

Can you suggest a few ways by which the patients get the required amount of protein comfortably?

Well, this can be done by a number of methods. Of these, one could be giving a different flavour to the milk by addition of coffee, ovaltine, chocolate or consuming milk as milk shakes, ice-creams, yoghurt. Another way could be consuming proteins from a different source such as egg, cheese, sausages etc.

Patients on protein-restricted or low protein diets include the ones with portal systemic encephalopathy, CRF etc. In such cases, the diet is based upon a daily allowance of protein foods with an emphasis on high class proteins.

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A few examples of low protein foods include beetroot, carrots, cabbage, mushrooms, tomatoes, turnips and most of the fruits.

Fats: As you are already aware, fats are reservoir of calories. In addition to carbohydrates, fats can also be an important source of needed calories. In certain therapeutic conditions, fat is necessary to prevent essential fatty acid deficiency. A high calorie diet should contain fatty foods (such as cream, butter, ghee and oil) while a low calorie diet contains a little or no fat.

Patients requiring low animal fat diets must restrict their total fat consumption to less than 30% of their energy needs and ideally 21-31% of this must be in the form of polyunsaturated fatty acids (PUFA) and monosaturated fatty acids (MUFA). This can be achieved by avoiding foods that are rich in saturated fatty acids (SFAs) such as all fried meats and fish, whole milk, fried eggs, cream, cheese, and nuts (peanuts, coconut) chocolates, butter etc. While food products such as poultry, white fish, egg whites, cottage cheese, skimmed milk, wholemeal cereals, fruits and vegetables, wholemeal bread, meringues, plain biscuits and fatless sponge require no restriction.

Mineral and Vitamins: The requirements for vitamins such as ascorbic acid and B-complex vitamins and minerals such as zinc may need to be increased to promote wound healing. Also in cases of long-term nutrition support, a careful assessment of vitamin and mineral status is essential to prevent the development of deficit or toxicities. You should consider the following factors to determine an appropriate vitamin and mineral intake:

- i) the requirements for healthy individuals,
- ii) nature of disease and injury,
- iii) body stores of specific nutrients,
- iv) normal and abnormal losses through the skin, urine or intestinal tract, and
- v) drug - nutrient interactions.

Next, let us have a look at sodium and potassium.

- **Sodium (Na) :** In sodium-restricted diets, no salt is added to the diet which still provides approximately 50 mmol Na. Foods containing high Na content must be avoided and the examples include processed or cured meats, tinned or smoked fish, tinned vegetables and soups, dehydrated and pre-packed meals, salted biscuits, nuts and crisps. There are very low Na diets as well which contain 20 mmol Na. These are much less palatable since no added salt is used at the table or during cooking. Unsalted butter is used and milk is restricted to 250 mL.
- **Potassium (K) :** Potassium restricted diets are important for patients with advanced renal failure undergoing conservative treatment or haemodialysis. The high potassium foods such as wholegrain breakfast cereals, vegetables e.g. beetroot, beans, broccoli, leeks, mushrooms, spinach, tomatoes, dry and split

peas, lentils, fruits e.g. prunes, dates, currants, grapefruit, oranges, banana etc. must be avoided. Vegetables should not be eaten raw rather they require leaching before consumption. The patients must also be aware of and warned against using salt substitutes.

- **Fluids:** Fluid diets are given to patients with more advanced dysphagia or fractured jaws. The diet may include fruit juices, thin strained porridge with milk, egg in milk, strained soups, thin milk pudding, ice-cream or yoghurt. Also, whole protein polymeric liquid feeds can be given. Since such diets lack bulk and can cause colonic dysfunction, these are available with fibre supplements.

A normal healthy adult at rest needs 1800 to 2500 mL/ fluids day (or approximately 1mL/Kcal consumed). If sufficient water is not consumed, it can lead to constipation.

Optimal convalescence requires adequate tissue hydration. The water intake must be liberal to ensure passage of light coloured urine. Additional fluids must be added to replace water lost by excessive perspiration, vomiting, diarrhoea, tube drainage or other conditions marked by increased water loss.

If sufficient water is not obtained through fluid intake and food, it must be supplied parenterally, usually along with electrolytes. Fluid restriction is needed in cases when excretion is impaired as in acute nephritis and kidney failure. Fluid requirements per day are calculated as 500 ml a day to replace the insensible loss in perspiration and sweating plus the volume of urine passed during the previous 24 hours.

During certain clinical conditions such as renal failure when the fluid intake can be detrimental to the prognosis of the disease; the fluid allowance is calculated by using the formula:

Fluid allowance: 500 ml + urine output in previous 24 hours + Fluid lost due to
(24 hrs.) (insensible losses) diarrhoea/
vomiting
(if any)

- **High fibre diets:** The patients are advised to eat high fibre cereals as whole grain flour and bread, whole grain breakfast cereals, whole wheat pasta and brown rice, all kinds of fruits and vegetables (with their-edible peels). Unprocessed bran can also be added to cereals or soups to give more fibre. .
- **Gluten-free diet:** It is a diet recommended for the patients with gluten enteropathy. Gluten is present in wheat, rye, barley and oats. Thus, foods containing these should not be eaten. A number of gluten free products are available on prescription and these include gluten-free flour, bread and biscuits.
- **Elimination diets:** This type of diet is used in a patient with suspected food intolerance, food allergy or Crohn 's disease. .
- **Exclusion diets:** Specific dietary exclusion becomes a necessity in case of

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food allergy or food intolerance. The therapeutic use of such diets requires a detailed discussion between the patient and the dietitian.

Each patient is provided with a list of foods that are permissible and avoided. Also, the need of scrutinize the ingredient lists in all convenience and manufactured foods is emphasized.

The examples of these diets include: meat free diet: Here, foods to be avoided are ordinary bread, biscuits, cakes, pastries, pasta and spaghetti and all wheat-containing breakfast cereals.

- **Milk free diet :** As the name implies, all foods containing milk protein must be avoided such as cheese, yoghurt, cream, ice-cream and butter.
- **Egg free diet:** In this, eggs and all products containing eggs are excluded from the diet such beef burgers, pies, cakes, meringues as well Bournvita and oval tine.
- **Additives free diets:** Additives here include permitted food colours such as tartrazine, sunset yellow, ponceus 4R and preservatives such as benzoic acil salicylates etc.
- **Ketogenic diet:** It is occasionally used to facilitate the control of epilepsy. Here, the patient is initially fasted for 48 hours and thereafter, half the energy requirement is provided as MCT (medium chain trigl yeeride) oil. Energy intake from ordinary food must be restricted to prevent the suppression of ketones. .
- **Diabetic diets:** These are therapeutic modifications in the quantity/ quality of various macronutrients particularly carbohydrates.

2.7 CONSTRUCTING THERAPEUTIC DIETS

You could do this by either using qualitative methods or quantitative methods. At times a combination of both may also be required.

What do we mean by these methods? How are these carried out?

Let us read the following section and find out. We shall begin with the qualitative methods.

- a) **Qualitative Methods:** This is where you give the individual choices, clear guidelines, menu guidance and supporting infomation such as advice on suitable manufactured products. You should encourage the patient to understand the key relationship between a food and a diet. The value of a food depends on the amount of nutrient in the food and the frequencywith which the food is consumed. The various qualitative methods include:

Guidelines issued for healthy eating
The Food Guide Pyramid
List of Desirable Food Choices, and
Elimination diets

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b) **Quantitative Methods:** These are often essential for constructing therapeutic diets. The two ways by which this could be done are as follows:

- i) Using an exchange system which delivers a fixed amount of nutrient per food portion. An example of this is the carbohydrate exchange system used in planning diets for insulin dependent diabetics. The desired level of intake is specified and the diet is constructed from an exchange list.
- ii) Quantifying the portion size of foods and the frequency of their consumption. This diet is constructed from normal sized portions of foods but those foods which have the highest content of a particular nutrient per portion are excluded from the diet. We learnt about this aspect earlier also, where we got to know about fat, Na and K restricted diets. We also had a look at the permitted and excluded food items based on their nutrient content.

Frequency of consumption of the various types of foods should also be considered. This method is used typically when a diet is a key component of a multifactorial condition e.g. coronary heart disease.

2.8 ROUTINE HOSPITAL DIETS

The most common diets that are prescribed or ordered in hospital situations are enumerated in this section and in Figure 2.1 Let us review them one by one.

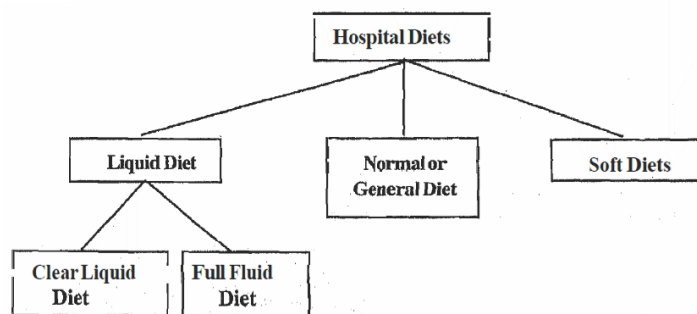


Figure 2.1: Routine hospital diets

2.8.1 Normal or General Diet

This diet is planned to be consistent with the Recommended Dietary Allowances (RDAs) of nutrients and is based on the food groups. It is usually based on cyclic menus planned according to the region, type of hospital and clientele. Nutritional adequacy depends on the patient's selection of food,

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as well as, the patient's intake of food. It is the responsibility of the clinical dietitian to monitor food selection and food intake to ensure adequate nutritional intake. The general diet is intended for the hospitalized patient whose medical condition does not warrant a therapeutic modification. .

Table 2.1: A Day's Normal Diet for an Adult

Foods	Quantity (g)	Carbohydrates (g)	Proteins (g)	Fats (g)
Cereals and cereal products	275	187	30.8	4
Milk and milk products (3% fat)	500	22	16.0	16
Pulses and grams	50	30	12.0	
Green vegetables	125	6	4.0	1
Root vegetables	125	9	3.0	
Other vegetables	50	12	1.0	-
Fruits	125	12	1.0	
Cooking oil	15			15
*Sugar	20	20		-
		298	67.8	36

2.8.2 Liquid Diets

A liquid diet is the one which consists of foods that can be served in liquid or strained form at room temperature. These are usually prescribed after certain kinds of surgery. The two major types of liquid diets include — Clear liquids and Full liquids.

Clear Liquid : It is composed of foods with low residue content which help to minimize the load of food needing digestion in the intestines. The clear liquid diet provides foods and fluids that are clear and liquid at room temperature. The type of liquid provided may vary depending upon the clinical condition of the patient, the diagnostic test or procedure, or specific surgery a patient is undergoing. The purpose of the clear liquid diet is to provide fluids and electrolytes to prevent dehydration. The diet is inadequate in calories and in essential nutrients. The clear liquid diet should not be the sole source of nourishment for more than 1 to 3 days without protein, calorie, vitamin and mineral supplementation. The clear liquid diet leaves minimal residue in the gastrointestinal tract. It also minimizes stimulation of the gastrointestinal tract.

The diet is used as an initial feeding progression between intravenous feeding and a full liquid or solid diet that follows surgery. It could be used as a dietary preparation for bowel examination or for surgery. It is also useful at times

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of acute disturbance of gastrointestinal function. It has application in many illnesses characterized by a high fever.

Recommended food items include:

- clear, fat free soups/broths
- light coffee, tea (without milk or cream)
- strained fruit juices
- tender coconut water, whey water, barley water
- gelatin, fruit ice, popsicle.
- sugar and salt added to liquids
- carbonated beverages as tolerated
- commercial high protein high calorie supplements (to be dissolved in a beverage or water), and
- honey.
- ice
- do not use any other food

Small amounts of fluids are offered at frequent intervals (50-100 mL every hour or two). The nutrient composition of the clear liquid diet will vary depending upon the types and amount of liquids provided and consumed by the patient. Do not use any solid food.

Full Liquid Diet : This diet provides foods and fluids that are liquid or semiliquid at room temperature. The type of food provided may vary depending upon the clinical condition of the patient. It is used as a step between a clear liquid diet and a regular diet.

The purpose of the diet is to provide an oral source of fluids for individuals who are incapable of chewing, swallowing or digesting solid food. It is used as an intermediate progression to solid foods following surgery, in conjunction with parenteral nutrition or in the presence of chewing or swallowing disorders or certain procedures such as jaw wiring. It is also used in the presence of oesophageal or gastrointestinal strictures, during moderate gastrointestinal inflammations and for acutely ill patients. Do not use any solid food.

Recommended food items include:

- soups and broths
- cereal porridges (refined cereals)
- milk and milk beverages, yoghurt
- coffee, tea, fruit juices, carbonated beverages
- butter, cream and oil added to foods
- plain puddings, custard, ice-cream, jelly, and
- sugar, honey, salt and mild flavourings,

The nutrient composition of the diet will depend upon the type(s) and

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amount(s) of liquids the patient can consume. The diet is low in iron, vitamin B and vitamin A and thiamine. By careful planning the diet can be made adequate for maintenance requirements, except for fibre. Liquid nutritional supplements or blenderized foods could be added to improve nutritional adequacy. The feeds are usually given at 2-4 hour intervals. Because this diet generally is inadequate in fibre, constipation may result from prolonged use. If it has to be used for long periods, vitamins, iron or liquid nutritional supplements must be added.

2.8.3 Soft Diets

The soft diet provides soft whole food that is lightly seasoned and moderately low in fibre. The foods have a soft texture and are easy to digest. Small volume meals are offered until the patient's tolerance to solid food is established.

The soft diet provides a transition between a liquid and a normal diet. It may be ordered for post operative cases, for patients with acute infections, gastrointestinal conditions or chewing problems. The soft diet should be individualized according to the clinical diagnosis, surgery, the patient's appetite, food tolerances, previous nutritional status, and chewing and swallowing ability.

The soft diet can be nutritionally adequate provided the patient is able to consume adequate amounts of food. Supplements or between meal feedings could be used to increase nutrient intake.

Foods allowed in soft diet include:

Soups	- mildly flavoured - broths and cream soups.
Beverages	- all
Meat	- moist, tender meat, fish or chicken, cottage cheese, eggs (except fried)
Fat	- butter, cream, oil, salad dressing.
Milk	- milk, milk beverages, yoghurt
Cereals	- soft cooked refined cereals - rice, pasta, bread, porridges.
Vegetables	- soft, cooked vegetables.
Fruits	- cooked and soft fruits, fruit juices
Desserts	- custard, ice-cream, jelly, cake (sponge), puddings without nuts
Sweets	- sugar, honey, plain candies

Foods to avoid include:

- fried foods and nuts,
- rich pastries and desserts,
- raw vegetables,

- heavily spiced foods,
- gas-forming vegetables,
- skin and seeds of vegetables and fruits.

Avoid rich gravies, sauces, pickles, fried foods, rich cakes and nuts.

The Inceclzanicl soft diet is a normal diet that is modified only in texture for ease of mastication. This is used when a patient cannot chew or use the facial muscles, for a variety of dental, medical or surgical conditions. The foods in the diet may be liquid, chopped, pureed or regular foods with a very soft consistency. Having reviewed the various hospital diets, we shall finally look at the different modes of feeding used for therapeutic purpose.

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2.9 MODE OF FEEDING

As a clinical dietitian you may also have to decide the method of feeding to be adopted. The method used will depend upon the patient's condition. The diet of an individual could be managed by using oral, tube, peripheral vein or total parenteral feeding. Sometimes, the patient may require assistance in feeding. The dietitian should understand the limitations of the patients and enlist the help of the nurse or patient's relative. The challenge is to be innovative and responsive. It is the clinical dietitian's responsibility to provide a combination of emotional support and technical nutrition advice on how to best achieve each patient's goals. Individuals who are hospitalized are sick and often have to be motivated or encouraged to eat. The food should be hygienically and attractively served. The food should be at the proper temperature and served in portions appropriate for the patient. The server should be pleasant. A correctly planned diet is successful only if it is eaten. There should also be effective communication between the physician, dietitian and nurse.

The different feeding methods include:

2.9.1 Oral Feeding

This is the preferred and most palatable method of feeding for meeting the increased nutritional demands of catabolism, it should be used as long as possible. If needed, nutrient supplements could be added to the oral diets.

2.9.2 Tube or Enteral Feeding

Ideally the patient must be fed orally, but in cases where the patient is unable to take solid foods, a part or all of intake is usually given by the tube. These are the cases where the gastrointestinal tract is functioning and can be used. Here, an alternate form of enteral feeding by tube provides nutritional support. Enteral nutrition can be provided by supplying intact, semi or

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completely hydrolyzed formulas through nasogastric/ duodenal/ jejunal routes or by the help of gastrostomies/ jejunostomies. These conditions include oral surgery, gastrointestinal surgery, dysphagia, unconsciousness, anorexia or oesophageal obstruction. Various commercial formulas are available for enteral tube feeding. Special formulas can also be calculated and blends prepared but these have a greater risk of contamination.

2.9.3 Peripheral Vein Feeding

Intravenous feeding is a method of providing parenteral nutrition when a patient cannot take in food or formula through the gastrointestinal tract. Various solutions of dextrose, aminoacids, vitamins, minerals and lipids can be fed through peripheral veins. But in this method the nutrient and kilocalorie intake is limited. It is used only when the nutritional need is not extensive or long term, where it is provided peripherally as a mixture of 5-10% glucose, a 3.5-5% amino acid solution and 10-20% lipid emulsion. The total fat intake should not exceed 2.5 g/kg/day. Vitamins, minerals and electrolytes are added as necessary, based on requirements and intake. The osmolarity of the solution should not be greater than 600 mOsm/L, What do you understand by this? Well, it simply means that large amounts of solution are needed to meet nutritional requirements. It is also used as a supplement to oral feeding in patients who cannot meet nutritional requirements completely by the oral or enteral route.

2.9.4 Total Parenteral Nutrition (TPN)

It is a method of providing, complete nutritional support in which the gastrointestinal (GI) tract is bypassed by introducing assimilable nutrients into a central vein. This is done in cases where a patient's nutritional need is great and assisted feeding is required for a longer time. You might wonder why specifically a central vein (mostly superior vena cava) is used. Well, this is because it is the central vein which can tolerate a hyperosmolar solution and hence nutritional support can be provided in a form that will meet all nutritional needs. Total Parenteral Nutrition (TPN) is a special surgical procedure in which special nutrient solutions are administered by a nutrition support team which includes the physician, dietitian, pharmacist and nurse. The patient needs special care and support. We shall learn more about parenteral and enteral nutrition later in Unit 4 of this course. Let us now attempt the check your progress exercise mentioned below.

2.10 LET US SUM UP

Therapeutic nutrition refers to the role of food and nutrition in the treatment of various diseases and disorders. In this unit, we learnt what are therapeutic

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diets, and the different types of dietary modification done in a normal diet to meet the therapeutic needs of a patient. We also learned that all therapeutic diets are modifications of the normal diet. Then we discussed the purposes for dietary modifications and the various types of therapeutic adaptations possible. These included liquid diets, soft diets and various modes of feeding such as oral feeding, tube feeding, peripheral vein feeding and total parenteral nutrition. Nutritional support is an integral part of medical therapy. A clinical dietitian accurate perception and sensitivity is essential to translate nutrition knowledge into a language appropriate for the individual client's need. Adequate knowledge, skills and proper attitudes are required to achieve or maintain optimal nutrition status.

2.11 GLOSSARY

Acute Renal failure	: renal failure associated with burns or other trauma or with acute infection or obstruction of the urinary tract.
Angio-edema	: swelling of the mucous membranes, tissues beneath the skin or an internal organ due to an allergic reaction.
Crohn's disease	: a chronic, recurrent disease characterized by patchy inflammation of any portion of digestive tract from the mouth to anus.
Portal Systemic Encephalopathy	: a syndrome associated with advanced liver disease.
Fistulas	: an abnormal opening between an internal cavity and another cavity or the surface.
Haemodialysis	: removal of chemical waste from the blood using blood flow through an artificial kidney.
Urticaria	: a skin condition characterized by the development of itchy, raised white lumps surrounded by an area of red' inflammation.
Osmolarity	: The osmotic concentration of a solution expressed as osmoles of solute per unit of solution.

2.12 CHECK YOUR PROGRES

- 1). What is a therapeutic diet? Discuss the purpose behind modifying a

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- normal or regular diet.
- 2). List the types of dietary adaptations to meet therapeutic needs.
 - 3). Normal nutrition is a basis for a therapeutic diet. Discuss.
 - 4). How is a clear liquid diet different from a full liquid diet?
 - 5). What is a mechanical soft diet? List any five foods to be avoided in a soft diet.

NUTRITIONAL MANAGEMENT OF INFECTIONS AND FEVERS

STRUCTURE

- 3.1 Learning Objective
- 3.2 Introduction
- 3.3 Defense Mechanism in the Body
- 3.4 Nutrition and Infection
- 3.5 Metabolic Changes during Infection
- 3.6 Classification and Etiology of Fever and Infection
- 3.7 Typhoid
- 3.8 Chronic Fever/Infection
- 3.9 Let Us Sum Up
- 3.10 Glossary
- 3.11 Check Your Progress

3.1 LEARNING OBJECTIVE

After studying this unit you will be able to:

- discuss the various defense mechanisms in the body which protect us from infections,
- explain the relationship between nutrition and infection,
- differentiate between acute and chronic infections,
- identify the symptoms associated with some common acute and chronic infections and their physiological significance, and
- describe the dietary management of acute and chronic infections.

3.2 INTRODUCTION

In this unit, we shall deal with defense mechanisms and the role of nutrition in managing fevers and infections caused due to impairment in the immune system. Go through these two examples given below to have a brief idea of the kind of issues with which we shall deal in this unit.

Case I : Shalini is a easy going college student, 19 years of age; extremely fond

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of snacking and trying out all varieties of food ranging from the "dhabas" to the "branded" ones. This summer she took ill... She had fever which she never monitored to begin with, but it kept on rising day by day. She was anorexic, felt bloated, complained of abdominal cramps and episodes of diarrhoea. She lost considerable weight. What do you think is the problem with Shalini?

Case II : Ram Singh is a coolie. He stays in the adjoining hutments of the railway station which has bad drainage, poor lighting and ventilation. In the recent past, he has been suffering frequently from upper respiratory infection (cold, cough and mild fever). This has been continuing for the past 1 year. Now he has started feeling weak and his cough has worsened with thick sputum and sometimes blood in sputum. What do you think Ram Singh is suffering from?

What are the factors/conditions which have led to this state for Shalini and Ram Singh?

What is the basic difference between the two cases presented?

How does the management of the two cases differ?

Infection, as you are already aware of, brings about many undesirable physiological changes which must be taken care of by proper nutrition care. How are these infections / fevers classified? What metabolic changes occur during such conditions? These are the issues which we will review in this. Finally, we will focus on the etiology, clinical manifestations and dietic management of different types of infections, including typhoid and HIV-AIDS.

3.3 DEFENSE MECHANISM IN THE BODY

What is meant by defense mechanism? What is its role? How does it function? Let us review once again.

Defense mechanisms relate to the body's ways to protect itself from any infection or a disease. These mechanisms can be:

- A) **Non specific:** These relate to the physical barriers like skin and mucous membrane. They form the first line of defense against entry of microorganisms or foreign substances in your body. Various body secretions, intestinal flora, iron binding proteins, certain nutrients like vitamin A, vitamin C, protein and zinc, hormonal influences and leukocytes (WBC) act against infections.
- B) **Specific:** These relate to specialized cells located throughout the body which respond to invasion of foreign materials/microorganisms such as bacteria, virus, fungi or unwanted substances produced or entering the body. Some specific cells are fixed in tissues such as thymus, lymph nodes, bone marrow, spleen, lymphoid tissue of the respiratory, gastrointestinal and genitourinary tract, Kupffer cells of

the liver and Peyer's patches of the small intestine.

- C) **Others:** These include the mobile cells such as leukocytes and lymphocytes. They are released in the blood and are carried to the site where they are needed when the microorganisms invade the tissue.

The specific immune (defense mechanisms) response can be broadly classified into two types:

- a) **Cell mediated immune response:** This is achieved through large number of activated lymphocytes that are specially designed to destroy foreign agents e.g. the T lymphocytes produced by the thymus.
- b) **Humoral immune response:** The body develops circulating antibodies which are globulin molecules that are capable of attacking the invading agents. B lymphocytes are produced by lymphoid tissue.

3.4 NUTRITION AND INFECTION

The interaction between nutrition and infection is synergistic — that is nutritional deficiency lowers resistance to infection and infection aggravates existing malnutrition. Individuals who are chronically undernourished not only succumb to infection more readily but take a longer period to recover than do the well nourished.

There are series of studies to show the specific roles of nutrients like vitamin A, vitamin C, vitamin E , omega 3 fatty acids, trace minerals like iron, zinc which have a role either in terms of maturation, number and/ or activity of T cells and B cells. These have an influence on the cellular and humoral immunity.

If you wish to go into the details of specific role of individual nutrients in the immune process, you could refer to some additional references listed, at the end of this course.

Malnutrition, impaired immunity and infection can form a triad or vicious cycle, which works synergistically and thus worsens the condition of an individual.

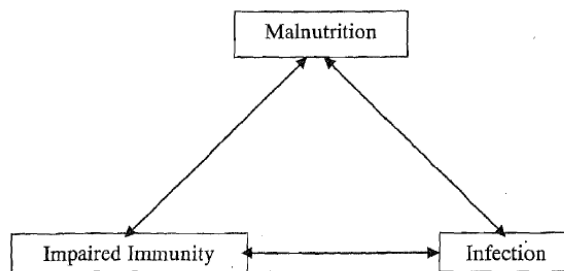


Figure 3.1: Synergism of malnutrition and Infection

So the synergism of malnutrition and infection must be clear. Next, let us

see what metabolic changes occur during infection which impact on our nutritional needs.

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3.5 METABOLIC CHANGES DURING INFECTION

With the rise in body temperature above normal (98.4°F or 37°C) due to infection several metabolic changes occur in the body that increase nutritional needs. Some important changes are discussed below:

- 1) The Basal Metabolic Rate (BMR) increases in severe infection with fever. There is a 7% increase in BMR with every 1°F increase in body temperature or 13% increase in BMR with every 1°C rise in body temperature. The energy needs are estimated to be 20% above BMR for mild infections and 70% above BMR for severe infections. Injury factors can also be taken into consideration to work out the exact calorie expenditure.
- 2) Nutrient losses may be increased due to increased perspiration, vomiting and diarrhoea. Minerals like sodium, potassium, zinc, magnesium and phosphorus are lost by the body.
- 3) Infection and fever sets in an environment of catabolism (breakdown) in the body. Infection can be related to a state of physiological stress. The body responds to this stress by altering the levels of several hormones to meet the increased requirements (Acute Phase Response). Increased levels of glucocorticoids, growth hormone, catecholamines, glucagon work synergistically to increase glycogenolysis (breakdown of glycogen) and gluconeogenesis (formation of glucose from non-carbohydrate sources). In addition, increased insulin levels and insulin resistance at the cellular level decreases the ability of the body to utilize carbohydrates as substrate for energy. Thus, body favours protein breakdown (catabolism) followed by fat breakdown (lipolysis) as the major substrate for meeting the energy requirements. Branched chain amino acids (leucine, isoleucine and valine) are used as major substrate for energy by the liver through the process of transamination (transfer of amine group), deamination (removal of NH_3 , from amino acid) followed by gluconeogenesis. The muscle breakdown favours nitrogen excretion and sets in a state of negative nitrogen balance (Excretion > Intake). The rate of protein breakdown (catabolism) increases depending upon the severity of the infection and the duration of fever. There are increased losses in long continuous fevers than in short duration fevers. Protein breakdown is especially marked in fevers such as typhoid, malaria, poliomyelitis and tuberculosis. This leads to increased nitrogen wastes and places an additional burden on the kidneys.

- 4) Infection and fever is invariably accompanied by anorexia (lack of appetite) which limits the food intake causing decreased energy intake which is not sufficient to meet the increased energy requirements. Thus leading to weight loss which particularly marked in severe infections.
- 5) The absorption of nutrients also decreases if there are intestinal infections.
- 6) Excessive sweat and urination causes further loss of body fluids and electrolytes. This can be an outcome of severe infection with high fever. The sweating occurs in response to high fever while the volume of urine is increased for elimination of nitrogen wastes produced due to catabolic activities.
- 7) Glycogen and adipose tissue stores decrease significantly due to increased energy expenditure. The above changes accompanied by loss of appetite, increase in energy needs, losses of protein due to catabolic activities, loss of fluid or electrolytes due to perspiration, vomiting and diarrhoea or poor absorption of some nutrients leads to loss of weight. Poor eating precipitates loss of weight particularly if the infection is prolonged. Thus infection may have a significant effect on nutritional status. The extent would depend on the following factors:
 - a) Nature of infection
 - b) Severity of the infection
 - c) Duration of the infection
 - d) Presence or absence of fever, and
 - e) Nutritional state of the individual before the infection sets in.Next, let us review how fevers are classified.

3.6 CLASSIFICATION AND ETIOLOGY OF FEVER/ INFECTION

Infection and fevers can be classified into two broad categories.

- a) **Acute Infections/Fever** : Acute fevers are of short duration with acute symptoms, the body temperature may rise to above 104°F. Examples of such infections are chickenpox, tonsillitis, influenza, pneumonia, typhoid and malaria.
- b) **Chronic Infections/Fever** : These are generally of longer and sustained duration. The patients have a past history of repeated episodes or continued spells of infection, Such infection may be characterized with a low-grade fever, which continues for even several months as in the case of tuberculosis. Thus chronic fever is the one, which has a slow, gradual onset and may be low grade in nature.

Examples of chronic fever infections include tuberculosis, HIV infection and AIDS.

Fevers can also be intermittent or cyclic in pattern and they are associated with infections like Malaria.

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INFECTION AND FEVERS ARE COEXISTENT. FEVER IS AN OUTCOME OF THE INFECTION.

You must have had fever sometime in your life. Do you remember? What caused it? In other words, what was the etiology of the fever? A number of internal (endogenous) and external (exogenous) factors play a role. Let us see what they are.

Etiology

Fevers can be caused due to

1. Internal (endogenous) factors: This could be caused within the body. Examples are antigen-antibody reactions, malignant cancer, graft rejections.
2. External (exogenous) factors: These are caused by bacteria, fungi, virus etc. which invades the body. The cause is from a source outside the body.

BACTERIA, FUNGUS, VIRUS

Infection due to bacteria, fungi or any antigenic input

Activation of phagocytes in the bone marrow

Release of pyrogen (fever inducing hormone)

Synthesis of prostaglandins (hormone like substance)

Thermo regulatory center in the anterior hypothalamus

Increase in temperature

Figure 3.2: Development of fever exogenous factor

So far in this section we have discussed about the basic concepts and inter-relationship between fever, infection and their association with nutrition. In the subsequent sections we shall be discussing about some common infections such as typhoid, tuberculosis and HIV-AIDS to help you in gaining an in-depth understanding regarding the care of patients with acute or chronic infections and fever. However, let us first perform the check your progress exercise before proceeding further.

3.7 TYPHOID

Typhoid is often called enteric fever because the infection or bacteria is found in

the intestines and attaches itself to the epithelium of the intestinal wall where it multiplies or finally reaches the blood causing damage and increase in the body temperature.

Typhoid is an enteric fever, which relates to acute infection of short duration. The mode of spread of this infection is through the fecal-oral route. The source of infection is the drinking water, milk and food contaminated by intestinal contents (through feces and urine) of the patients or "carriers" or by flies which transmit the disease.

It may affect all age groups but is commonly observed in children. The incidence and magnitude of typhoid fever has greatly reduced in the present context due to improved sanitation and vaccines and effective drugs available.

It is important for you to understand that this fever has a number of adverse effects on the body as it is extremely catabolic in nature, thus in no time it causes weakness, and compromised state of nutrition causing tremendous weight loss.

Some observations seen in this fever are:

1. Massive loss of lean body mass or muscle due to tissue breakdown (250-500 g muscle tissue is lost/day) leading to excessive nitrogen losses.
2. Body stores of glycogen are quickly depleted because of increased energy requirements.
3. The gastrointestinal tract is highly inflamed and irritable as the seat of typhoid infection is the Peyer's patches of the intestine. The bacteria attaches to the epithelium of the intestinal wall, penetrates and multiplies in the mesenteric lymph nodes, eventually reaching the blood stream which in turn leads to secondary infection (of the intestines).
4. Excessive diarrhoea, vomiting and perspiration can cause a lot of fluid and electrolyte losses.
5. Inflammation of GI tract can lead to intestinal ulceration and bleeding.

Clinical symptoms of typhoid are:

1. Graded fever which follows an upward ladder pattern.
2. Abdominal pain, cramps and diarrhoea.
3. Anorexia and vomiting.
4. Internal haemorrhage and melaena (gastrointestinal bleeding and black tarry stools).

Management of the typhoid patient includes:

1. Bed rest
2. Antibiotic therapy
3. Modification of diet

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Dietary Management

The golden rule in the dietary management of any fever is "feed the fever". Considering that enteric (typhoid) fever is accompanied by anorexia, vomiting and high grade temperature, the diet has to be modified as per the patients' tolerance. The patient needs to be encouraged to eat. Feeding several times a day improves tolerance. The texture of foods given would depend on the severity of infection. Bland, low fibre and soft foods are beneficial.

The dietary principles underlying the enteric diet include:

- High calorie
- High protein
- High carbohydrate
- Moderate fat
- High fluid
- Low fibre and soft diet

Now let us go further in details on the specifications of each nutrient and the basis for working the requirement of the same for the given condition.

- **Energy:** Fever is characterized by elevation of BMR, thus caloric requirements are increased. The increase in caloric needs is dependent on severity of infection and degree of rise of temperature. During fevers there is a decrease in appetite, as well as, a decreased tolerance due to enteric infection, thus a desired increase in calories is 10-20% above the normal recommended requirements. The actual intake can be adjusted and given as per patients' tolerance.
- **Protein:** The requirement of protein is increased in typhoid, as there is a massive tissue loss. Thus, the protein intake should be increased above the normal of 1g/kg/day to 1.5-2g of protein/kg/day. A high protein diet should be supported with a high carbohydrate intake to favour efficient protein utilization for anabolic or tissue building purposes. Foods providing appreciable and good quality protein (high biological value) should be incorporated in liberal amounts. Use of protein supplements is recommended to add on to the nutrient density without increasing the bulk of the diet, Carbohydrates: Carbohydrate intake should be liberal. This can be attributed to:
 1. Repletion of glycogen stores.
 2. Protein sparing action.
- **Carbohydrates:** Carbohydrate intake should be liberal. This can be attributed to:
 1. Repletion of glycogen stores.
 2. Protein sparing action.

Well cooked, easily digestible carbohydrates like simple starches, glucose, honey, jam should be included as they require much less digestion and are well assimilated. Glucose can be supplemented in a variety of beverages/light desserts as it is less sweet than sucrose and adds on to the total calorie intake.

- **Dietary fibre:** Typhoid patient has an inflamed intestinal mucosa, which can be easily perforated and ulcerated leading to internal haemorrhage. Thus foods high in fibre such as certain green leafy vegetables, whole pulses or cereals, thick skins or fruits or vegetables must be avoided. (These are rich in insoluble fibres). Soluble fibres can be given.
- **Fats:** Use of fats should be in moderation. This is because the typhoid patient has a compromised ability to digest and assimilate due to Peyer's patches (elongated thickening of the intestinal epithelium) which result in repeated episodes of diarrhoea. Fats help in increasing the energy density of the food without increasing the bulk of the diet, but the aspect which needs to be emphasized is the type of fat. Use of dairy fats like butter, cream, fats in milk products, egg yolk etc. help in easy digestion as they contain medium chain triglycerides. Excessive use of fat in cooking, eating fried foods can aggravate nausea, impair digestion and lead to severe diarrhoea. These should be avoided.
- **Minerals:** Loss of electrolytes and water is observed due to diarrhoea. Thus liberalizing on sodium intake through salty soups, beverages are desired. Potassium intake can be increased by emphasizing cooked fruits, low fibre vegetables, washed and dehusked pulses. Food preparations in forms like juices, stews, soups and dal water are beneficial. The other minerals, which are of importance, include iron particularly if blood is lost due to haemorrhage in the intestines. Losses of zinc and chromium have also been observed.
- **Vitamins:** Vitamins which need to be emphasized include B complex, considering the increase in the energy requirement and a decreased ability of the intestine to assimilate and synthesize some of the B complex vitamin due to compromised digestive processes and altered microbial flora. As a result, antibiotics are prescribed. In addition, vitamins A and C are also needed to boost immunity, favour wound healing and maintenance of the integrity of epithelial membrane (gut mucosa). Vitamin supplementation may be given in the early stages of the infection when the patient is anorexic and has low food tolerance. **Fluid:** Liberal fluid intake is desired to compensate for the fluid losses from the body.

A daily fluid intake of 2.5 to 3.5 litres may be recommended. Fluid intake can be accomplished through a variety of beverages, soups, juices, broths, dal besides plain water. Adequate fluid intake helps in eliminating wastes and maintaining water balance in the body.

So remember the foods to be included are:

- Juices, soups, dal water, broths.
- Refined cereals and their products (e.g. idli, dosa, rava, bread, rice, noodles,

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washed dals, pureed vegetables, stewed fruits). These foods contain low insoluble fibre.

- Eggs, cottage cheese, tender steamed or baked chicken, fish. These contain high biological value proteins.
- Fruit juices, gelatin, honey, sugar and milk products. Calorie and protein-rich desserts could be prepared.

Foods to be restricted include:

- Excessive milk and milk products and dairy fats such as cream and butter.

Foods to be avoided are:

- High fibre foods like whole grain cereals and their products (e.g. whole wheat flour, cracked wheat, whole pulses)
- Raw vegetables and fruits
- Fried fatty foods
- Chemical irritants like spices, pickles, papad, ketchups etc.

Now answer the questions given in check your progress exercise 1 and recapitulate what you have learnt so far.

3.8 CHRONIC FEVER/INFECTION

As mentioned earlier, chronic fever and infection are always supported with a long standing history of symptoms or repeated episodes of infection. One of the commonly observed chronic infections is tuberculosis. Let us understand the treatment and management of this chronic infection.

3.8.1 Tuberculosis

Tuberculosis is a chronic infectious disease which is caused by a bacterium - *Mycobacterium tuberculosis*. It affects the lungs most commonly but can get localized in other organs also, like lymph nodes, kidney, bone etc. The most commonly observed form of tuberculosis in India is pulmonary tuberculosis.

The prevalence of tuberculosis earlier was restricted to lower socioeconomic strata being attributed to poor hygiene, sanitation and poor quality of food intake. The present scenario reveals an increasing incidence of tuberculosis in people from higher socio-economic strata as well. A strong genetic history gets highlighted in most of the cases. This disease presents itself in an acute and a chronic phase.

In the acute stage, the disease is quite similar to that of acute fever and the chronic phase to that of chronic fever. The chronic phase is accompanied by low grade fever and therefore increase in metabolic rate is not so marked. The long duration of illness in turn leads to wasting of body tissues.

The symptoms, treatment and dietary management are enumerated next.

A. Salient Features of Pulmonary Tuberculosis :

The salient features tuberculosis include:

Wasting of tissues
Exhaustion
Cough
Expectoration, and
Fever

The acute phase, resembles pneumonia with high fever. The chronic phase presents itself with low grade fever, accompanied with exhaustion, cough, expectoration and loss of weight.

The progression of the disease may be slow with gradual worsening of the cough. This can lead to erosion of the blood vessel of lungs. The tubercle bacteria may thus subsequently get access to other body organs, thereby, establishing numerous secondary foci of infection.

B. Treatment

The disease can be very effectively treated with the help of antibiotic therapy, rest and nourishing food. The key to the treatment is early detection of the disease.

The antibiotic therapy given should be continued for the stipulated period of time (6 months to 1 year), in spite of the waning of the symptoms. A clinically recovered tuberculosis case can still be a carrier and thus a relapse of the disease is likely if the antibiotic therapy is not followed the right way.

C. Dietary Management

It must be evident to you that majority of the tuberculosis patients are emaciated and malnourished. Proper dietary management during and after the infection is essential to ensure complete treatment, proper rehabilitation and prevention of relapse. Let us review the energy and nutrient requirements of the patient.

- **Energy :** The criteria to establish the energy intake is the body weight status coupled with the rise in body temperature. Considering that the BMR is not highly elevated in the chronic stage of the disease the energy intake may be increased by 300-500 Kcal/day above the normal recommended intake.
- **Protein :** Chronic infection is marked by a prolonged duration of fever. This leads to wasting of muscles, increased nitrogenous loss and a subsequent decrease in serum albumin levels. Thus, the chronic stage of the disease needs to be supported with a high protein intake. A level of 1.2 to 1.5 g protein/kg body weight/day should be given. Emphasis should be on energy and protein dense foods coupled with high biological value of protein rich sources to favour its effective utilization. If the disease is observed in the low socioeconomic strata, selection of cheaper options of protein dense foods like pulses, soya, nuts in

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- combination with coarse cereals for mutual supplementation may be opted for.
- **Carbohydrates** : Adequacy of carbohydrate will also favour the optimal utilization of proteins. To favour the process of anabolism a total calories to nitrogen ratio of 150:1 should be achieved in a high calorie high protein diet. Carbohydrate being the most preferred substrate of energy by the body has a protein sparing effect.
 - **Fats**: Fats add to the calorie density of the diet. Digestibility of fats (medium chain triglycerides and emulsified fats should be preferred) and fat based preparations should be considered.
 - **Vitamins** : The vitamins of significance for a tuberculosis patient include:
 - a) **Vitamin A** : The preformed vitamin A or the retinol form of vitamin A needs to be emphasized in the diet of TB patient as the metabolism of vitamin A is adversely affected. Carotene appears to be poorly converted to vitamin A. Considering that the retinol form is restricted to milk, milk products, dietary fats and animal foods, vitamin A supplement may be recommended.
 - b) **Vitamin B complex**: The requirement of the B-complex vitamin increases with an increase in the energy requirement of the tuberculosis patient. The salient ones out of the B complex group are pyridoxine, folic acid and vitamin B₁₂. Neuritis (inflammation of peripheral nerves) can be prevented by treatment with 50 to 100 mg/day dose of pyridoxine. Folic acid and vitamin B₁₂ is also supplemented.
 - c) **Vitamin C**: Vitamin C helps in collagen synthesis and helps healing of the tubercle lesions. Rich vitamin C food sources like amla, guava, drumsticks, cabbage, capsicum and citrus juice should be included liberally in a tuberculosis patients diet.
 - **Minerals**: The minerals to be emphasized in tlic diet of a tuberculosis patient include:
 - 1) **Calcium**: Calcium intake needs to be increased since it is essential for healing the tuberculosis lesions. Intake of half a litre to 1 litre of milk is recommended in different forms which can be well tolerated by the patient. Calcium supplementation (500 mg/day) with active form of vitamin D may also be prescribed.
 - 2) **Iron**: The need of iron may be a concern in case of blood loss associated with expectoration or haemorrhage. The patient's haemoglobin levels should be monitored. The diet should be tailored as per the needs. Supplementation with iron is recommended in case blood haemoglobin levels are low.
 - 3) The other minerals of significance are zinc and chromium. These minerals help in boosting the immune system. Losses, if these are seen in the patients having tuberculosis.

Other considerations

- i) The diet should have good amount of fluid and electrolytes especially potassium.
- ii) Fibre need not be restricted but the food options should be easy to digest and well tolerated.
- iii) Frequent nutrient-dense feeds are recommended. Force feeding is not desired.
- iv) Anormal dietary pattern needs to be followed with a wise, balanced and nutritious selection of foods.

Let us conclude the discussion by highlighting the foods to be included or restricted in the diet of a tuberculosis patient

Foods to be included:

- Cereals (Ragi, jowar, bajra).
- Pulses (black channa, chawli, moth, rajrnah).
- Nuts and oilseeds.
- Green leafy vegetables like methi, chaulai, mint, spinach, cabbage, drumstick leaves, colocasia and cauliflower greens.
- Citrus fruits (guava, amla, capsicum).
- Milk and milk products.
- Jaggery, sugar.

Foods to be restricted

Excess fat, fried preparations, organ meats (liver, kidney, brain), red meat and refined sugars.

3.8.2 HIV (Human Immuno Deficiency Virus) Infection and AIDS (Acquired Immune Deficiency Syndrome)

HIV/AIDS is another example of chronic infections. Its existence was discovered recently in 1981. The spread of HIV infection is widespread and can progress to AIDS. It has been shown to be caused by a retrovirus since it is one of the most dreaded infections and is growing at a rapid rate. Let us gain some more knowledge about it.

Manifestations of HIV infection

Four stages of HIV disease have been recognized. These are:

- Acute HIV infection
- Asymptomatic HIV infection
- Symptomatic HIV infection

AIDS

A brief review on these stages follows:

- **Acute HIV Infection**

This period is 4-7 weeks after the primary infection. During this period, there is a rapid viral replication and the infected person develops an acute syndrome, characterized by fever, malaise, pharyngitis, headache, lymphadenopathy syndrome (swollen, firm and sometimes tender lymph nodes), influenza or cancer of lymph nodes; myalgia (muscle pain/weakness).

- **Asymptomatic HIV**

In asymptomatic HIV, very few symptoms may appear which are noticeable. This stage could even last for 10 years. Sub-clinical symptoms have been reported such as decrease in lean body mass without apparent change in weight. There is an increased susceptibility to food and water-borne pathogens.

- **Symptomatic HIV**

In this, the AIDS defining symptoms appear. A decline in nutritional status and body composition may occur. Fevers, sweating, skin problems and fatigue may be seen.

- **AIDS**

The term AIDS encompasses life threatening clinical conditions linked to HIV induced immune suppression. A small portion of persons infected with HIV may develop AIDS within months following primary infection. AIDS is the terminal stage of HIV. Infections with bacteria, virus, fungi and protozoa are common. They are often the cause of diarrhoea, malabsorption, fever and weight loss. Malignant disease such as Kaposi's Sarcoma, neurological disorders (HIV encephalopathy) myelopathy and peripheral neuropathy and myopathy lipodystrophy or lymphadenopathy may be seen.

Many other organs can be affected such as the gastrointestinal tract, liver, kidney and pancreas. If bacterium avium complex can be seen in the lymph nodes, liver, bone marrow, blood and urine of patients. Most cases are affected by tuberculosis of the lungs. Acute renal failure is seen in many subjects. Chronic diarrhoea may persist in the absence of identifiable enteric pathogens as a result of AIDS enteropathy.

Malnutrition is an important complication of AIDS. Protein energy malnutrition is seen as weight loss, body cell mass depletion, decreased skinfold thickness, decreased midarm circumference, decreased iron binding capacity and hypoalbuminemia. AIDS wasting syndrome is commonly reported.

So you could see for yourself the myriads of complications and disease conditions which an AIDS patient suffers from. We would like you to understand a little more about some of the symptoms/complications that have been mentioned above.

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- **Kaposi's sarcoma:** This is a malignant disease of the peripheral blood mononuclear cells which manifests itself as purple nodules on the skin, mucous membranes, lymph nodes and throughout the gastrointestinal tract. These purple nodules in the oral cavity or oesophagus lead to pain and difficulty in chewing or swallowing.

If in the intestinal tract, they can cause diarrhoea and even intestinal obstruction. Lymphomas, including non-Hodgkin's lymphoma can involve the small intestines and cause malabsorption, diarrhoea and intestinal obstruction. If the lymphoma is in the brain, it can cause alterations in personality (psychosis, depression and withdrawal) and motor and cognitive abilities, carcinomas of colour and tongue are also seen.

Neurologic Diseases

If the HIV enters the brain, it may result in AIDS dementia. Myelopathy (disease of the spinal cord) can result in paralysis of the lower extremities. Myelopathy affects the motor and sensory functions leading to spasticity, weakness in legs. Peripheral neuropathy is characterized by sensory loss, pain, weakness and wasting of muscles in the hands, legs and feet. The first signs are tingling, burning, numbness in toes and fingers.

AIDS wasting disease. It is a diagnose for AIDS in the HIV positive individuals for whom no other cause of the symptoms can be identified. It is characterized by persistent fever, chronic fatigue, malaise and diarrhoea of unknown etiology. Weight loss of 10%-15% is common.

Lypodystrophy: This is a disturbance of fat metabolism that involves the loss of the thin layer of fat making veins visible and protruding. Wasting of the face and limbs and accumulation of fat in the abdomen and between the shoulders is an important feature. Lymphadenopathy syndrome : The lymph nodes are swollen, firm and sometimes tender causing a range of infections such as HIV, influenza or even lymphoma (cancer of lymph nodes).

Today several retroviral drugs are available to destroy HIV or suppress its replication. Management after a viral load test which measures the quantity of free virus circulating in the blood stream as CD-4 and T-cells can make the treatment comprehensive and focussed on likelihood of developing AIDS. CD-4 cells, also known as T-cells are essential components of the defense mechanism against infectious diseases.

Table 3.1 gives some idea of the CD-4 cell count, the conditions, common problems and symptoms associated with it.

Result of CD-4 tests are used to plan appropriate treatment measures and medical intervention.

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Table 3.1: Condition associated with CD-4 + cell count in HIV infectious

CD-4 Cell Count	Conditions and Symptoms	Common Physical Problems
200-500/mm ³	Oral thrush, Kaposi's sarcoma, tuberculosis reactivation, Herpes zoster, Bacterial sinusitis or pneumonia.	Loss of appetite, white plaques, taste changes, mouth discomfort. Raised purplish lesions on the skin, mucous membranes, or lymph nodes (usually painless). Cough, blood-stained sputum, fever, night sweats, weight loss, chest pain and fatigue. Herpes causes painful vascular lesions on skin, inflammation of the nasal cavity and congestion due to fever,
100-200/mm ³	Herpes simplex or pneumonia	Weeping skin lesions (oral and perirectal), bleeding, rectal discharge, pain, fever, chills, night sweats, cough, shortness of breath, antibiotic side effects, weight loss and weakness. .
50-100/mm ³	Systemic fungal infections, meningitis, tuberculosis, infection (protozoal) of small and large bowels. Progressive multifocal leukoencephalopathy, peripheral neuropathy and cervical carcinoma	Headache, fever, malaise, nausea, fatigue and loss of appetite. Weight loss, skin lesions, difficulty in breathing, anaemia, pneumonia, cough, blood in sputum, night sweats, weight loss, chest pain, fatigue and anorexia. Severe chronic watery diarrhoea (up to 15-20 times a day), severe weight loss, electrolyte imbalance, abdominal cramping, fever, nausea, vomiting and enlarged lymph nodes. Progressive weakness and dementia, speech problems, forgetfulness, visual problems, incontinence. Painful burning feet, numbness in feet and/or hands and vaginal discharge.
0-50/mm ³	Cytomegalovirus disease bacterial (mycobacteria), Non-Hodgkin's lymphoma, AIDS, dementia	Blindness or visual loss, fever, fatigue, malaise, weight loss, facial oedema, enteritis or colitis, fever, severe weight loss, cachexia, diarrhoea and malabsorption. Mumps, fatigue and pain. Loss of coordination, mood swings, loss of inhibitions, cognitive dysfunction.

A. Prevalence, Etiology and Pathophysiology

More than 21 million people throughout the world suffer from HI V. The conservative forecast of WHO (World Health Organization) is that a minimum of 30-40 million people worldwide will be infected with AIDS by the end of the decade. Currently the infected umbrella spreads in a descending order to sub saharan Africa, South -Latin America (Caribbean) North America, Middle East, Western Europe, Eastern central Asia, China, Japan etc. The overall incidence in the country is about 0.1 percent.

Primary infection with HIV is the underlying cause of AIDS, This invades the genetic code of CD-4 cells or T helper lymphocyte cells. Infection occurs from an infected person through body fluids such as blood, semen, vaginal secretions and lymph system and the central nervous system. The virus (HIV) causes progressive depletion of CD- 4 cells. This leads to immune deficiency and other complications

finally leading to AIDS. Even breast milk, saliva, tears and urine contain enough virus to infect human beings. Unprotected sex, sharing of contaminated needles, across the placenta to the baby are common ways of getting in touch with infected body fluids.

Workers must take precautions for themselves or others when working with body fluids. The virus is not transmitted by causal contact, touching, hugging, kissing or sharing utensils, glasses etc.

It is uncertain whether all HIV infected persons develop AIDS. Reports indicate about 5% of HIV infected individuals exhibit no signs of progression to AIDS.

B. Diagnosis of HIV Infection

The first step is ELISA (Enzyme Linked Immunosorbent Assay) Screening. The second step involves western Blot Testing for confirmation of suspected positive samples. Both the tests are highly sensitive and specific. This is associated with the clinical symptoms that may vary with the stages of disease.

C. HIV Medical Management

The goals of medical management of HIV are to:

- prolong life and improve the quality of life,
- suppress the virus to as low a level as possible,
- optimize and extend the usefulness of currently available therapies, and
- minimize drug toxicity and manage side effects.

Disease progression differs from an individual to individual therefore treatment decisions must be individualized keeping in mind the stages.

NOTE : AIDS IS THE FINAL STAGE OF HIV. IT OFTEN CAUSES SEVERE PROTEIN ENERGY MALNUTRITION AND IS OFTEN KNOWN AS AIDS WASTING SYNDROME.

D. Malnutrition and AIDS

Malnutrition is an important and complicated consequence of HIV infection. It normally manifests as:

1. Protein Energy Malnutrition: associated with weight loss, body cell mass depletion, decreased skinfold thickness and mid-arm circumference, decreased iron binding capacity and hypoalbuminemia.
2. AIDS wasting syndrome: associated with the involuntary weight loss of 10% baseline body weight plus either chronic diarrhoea (2 loose stools (day for more than 20 days) or chronic weakness and documented fever (for 30 days or more, intermittent or constant) in the absence of an associated

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illness or condition that would explain the findings. It may be associated with the following:

- a) Inadequate food intake: Associated with disorders of mouth and oesophagus such as candidiasis, herpes simplex, severe anorexia, secondary to medication, depression, infections. In addition, other contributory factors would be nausea, vomiting, dyspnoea, fatigue or neurologic disease.
- b) Malabsorption of nutrients: The malabsorptive symptoms have been related to both drug diet interactions and the progressive effects of HIV infection. An AIDS enteropathy in the early stages of infection is characterized by blunting of the intestinal villi, abnormal intestinal enzymes that cause clinical malabsorption. HIV infection infiltrates lymphocytes, as well as, enterocytes which make the gut more prone to infections, leading to severe diarrhoea and malabsorption.
- c) Disordered metabolism : Relates to hypermetabolism or "Altered" energy metabolism - usually associated with end stage effects of the HIV infections, as well as, increased spread of several infections. There is a progressive depletion of lean body mass; as well as, increase in REE (Resting Energy Expenditure). Energy metabolism is altered as cycling of fatty acids is associated with increased serum triglycerides levels, the catabolism of skeletal muscle as an endogenous sources of energy and elevated levels of the cytokine alpha interferon produced by the cells of the immune system. The metabolic picture is unlike simple starvation in which body fat is oxidized for energy.

Studies indicate that as the wasting of lean body mass nears 55% normal for age, sex and height in persons with AIDS death is imminent regardless of the cause of malnutrition. Body fat is not a predictable marker of wasting in individuals with AIDS, especially men tend to lose body cell mass with little loss of fat in contrast to uncomplicated starvation in which fat stores are depleted.

Malnutrition may contribute to the frequency and severity of infection seen in AIDS by compromising immune function. Deficiencies of protein, calories, copper, zinc, selenium, iron, essential fatty acids, pyridoxine, folate, vitamin A, vitamin C and vitamin E all interfere with immune function. Severe weight loss can also result in organ damage which may increase the risk for a fatal outcome from infections.

Now let us understand the medical nutrition therapy for AIDS.

E. Medical Nutrition Therapy

The general goals of nutrition intervention are to:

- preserve optimal protein status,

- prevent nutrient deficiencies or excesses known to compromise immune function,
- minimize nutrition related complications that interfere with either intake or absorption of nutrients,
- support optimal therapeutic drug levels,
- enhance the quality of life, and
- educate patients on importance balanced diet or how to improve nutritional status.

Table 3.2: Screening for medical nutrition therapy in HIV-infected adults and adolescents older than 18 years of age.

<i>Referral is automatic when any one of the following conditions exist :</i>	
1.	Newly diagnosed HIV infection or never been seen by a registered dietitian
2.	If asymptomatic: not seen by a registered dietitian in the past 6 months to 1 year
3.	Newly diagnosed HIV with symptoms or AIDS
4.	If HIV with symptoms or AIDS: not seen by a dietitian in the past 6 months or more
5.	>5% unintentional weight loss from usual body weight within the last 6 months, or since the last visit (% weight loss formula: usual body weight– current body weight × 100)
6.	Poor oral intake of food or fluid
7.	Change in stools (colour, consistency, frequency, smell)
8.	Persistent gas, bloating, heartburn
9.	Persistent diarrhoea or constipation
10.	Persistent nausea or vomiting
11.	Difficulty chewing, swallowing. Mouth sores, thrush or herpes simplex type 1
12.	Severe dental caries
13.	Changes in perception of taste or smell
14.	Food allergies/food intolerance (fat, lactose, wheat, etc.)
15.	Economically unable to meet caloric and nutrient needs
16.	Concomitant diabetes mellitus , hypertension, hepatic or renal insufficiency, heart disease, cancer, pregnancy , or other nutrition-related condition.
17.	Visible wasting <90% ideal body weight
18.	Albumin 0.2 mg/dL
19.	Cholesterol < 120 mg/dL or >200 mg/dL
20.	Triglycerides > 450 mg/dL
21.	Scheduled chemotherapy or radiation therapy
22.	Medication regimen that involves food or meal modification
23.	Need for enteral or parenteral nutrition
24.	Patient- or MD-initiated weight management , obesity, vitamin-mineral supplementation, vegetarianism or complementary/alternative diet therapies.

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Once the patients have been screened for medical nutrition therapy, the dietary guidelines include:

- **Energy :** Energy needs vary depending on the health status of the individual at the time of HIV infection, the progression of the disease and the development of complications that impair nutrient intake and utilization. Activity can also affect energy requirements. Depending on the activity, the energy is increased. In case of fever, the energy intake should be further increased so that there is no loss of weight.
- **Protein:** High protein diets might safely promote positive nitrogen balance and lean body mass repletion. Studies are still needed to clarify the ability of high protein diets to reverse HIV associated malnutrition and body composition changes. Considering that the requirements of protein are increased, an intake of 1.0 to 1.4 g/kg for maintenance and 1.5 to 2.0 g/kg for repletion has been recommended. Protein restriction is recommended only in persons with severe hepatic or renal diseases.
- **Fats:** A moderate fat intake is recommended for an HIV patient to add on to the caloric density and palatability of the food given. Tolerance to fat varies from person to person. Studies suggest that the use of medium chain triglyceride (MCT) oil is better than long chain triglycerides based supplements for decreasing stool fat and stool nitrogen content and in reducing the number of bowel movements and abdominal symptoms. MCTs are more readily absorbed than long chain triglycerides. Fish oil (omega 3 fatty acids) when given with MCT oil may improve immune function because this combination is less inflammation promoting than the usual omega 6 fatty acids.
- **Fluids and Electrolytes:** Fluids needs in HIV infected individuals are similar to those of well individuals and are calculated to be 30-35 mL/kg (8-12 cups for adults) with additional amounts to compensate for losses from diarrhoea, nausea and vomiting, night sweats and prolonged fever. Replacement of electrolyte loss (Na, K and Chloride) in the presence of vomiting and diarrhoea is also recommended.
- **Vitamin and Minerals :** The exact requirements for vitamins and minerals are still unknown, increased intake of the following micronutrients: β -carotene, vitamin E, ascorbic acid, vitamin B₁₂, vitamin B₆ and folic acid is recommended. Use of vitamin mineral supplement providing 100% RDA is also recommended.

Water Safety: HIV/AIDS subjects are vulnerable because of immune suppression. Hence water borne pathogens are a concern. Water safety is important. Boil water, filter tap water, use boiled water as far as possible.

HIV and AIDS infected patients are known to have symptoms associated with medications, malnutrition, malabsorption.

Table 3.3: Practical eating suggestions for symptom management

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Symptom/problem	Management
Nausea	Small, frequent meals, Avoidance of high-fat, greasy foods, Cool or room-temperature foods, Avoidance of lying down flat after eating.
Sore mouth/throat	Soft, moist foods, Avoidance of spicy or acidic foods, Experimentation with temperature of foods (avoidance of very hot or very cold foods; cool or room-temperature foods are best). Use of nutrient-and energy-dense foods to maximize oral intake.
Xerostomia (dry mouth)	Use of foods that are moist or served with a sauce or gravy. Consumption of liquids at mealtimes and extra fluids between meals. Emphasis on good oral hygiene: flossing, brushing, and rinsing, regular dental care. Use of fluoride gels or mouthwashes. Consideration of prophylactic antifungal therapy. Chewing of sugarless gum or sucking of mints.
Difficulty with breathing.	Use of easy to eat foods, Use of nutrient-and energy-dense foods.
Diarrhoea	Fluid and electrolyte replacement, Low-insoluble, high-soluble fibre diet. Possible benefits from low-lactose diet, Low-fat diet (may be indicated). Avoidance of gas-causing foods, beverages and caffeine.
Constipation	Increased fluid and dietary fiber intake. Use of nutrient-and energy-dense foods, including nutritional supplements. Use of small, frequent meals and snacks. Consideration of alternative nutrition support.
Fatigue	Adequate sleep, relaxation, exercise, adequate diet, especially foods rich in vitamins, B ₁₂ , A, C, folate, and carotene or zinc, as inadequate levels may cause fatigue. Avoidance of caffeine, alcohol, cigarette smoking, and recreational drug use. Avoidance of stress and treatment of anxiety or depression. Identification and management of possible causes for anaemia. Medications: AZT, bactrim, interferon, pyrimethamine Other causes: alcohol abuse, bleeding, <i>Mycobacterium avium</i> complex, tuberculosis, . fungal infections, cytomegalovirus

Table 3.4: Screening for Paediatric patients (< 18 years of age) with HIV/AIDS for medical nutrition therapy

<i>In addition to the conditions listed in Table 3.3 referral to a dietitian should be automatic when any one of the following conditions exist:</i>	
1.	Weight for age <10 percentile (National Center for Health Statistics [NCHS])
2.	Height for age <10th percentile if weight for age is also <10th percentile for age (NCHS)
3.	Downward crossing of one major weight-for-age percentile
4.	Poor appetite; food or fluid refusals
5.	Prolonged bottle-feeding and/or severe dental caries
6.	Change in stools (colour, consistency, frequency, smell)
7.	Children 0-12 months having low birth weight
8.	Children 0-12 months old: no weight gain for 1 month
9.	Children 0-12 months old: diarrhoea or vomiting for >2 days
10.	Children 0-12 months old unable to suck properly
11.	Children 1-3 years old: no weight gain for 2 consecutive months
12.	Children 1-3 years old: diarrhoea or vomiting for >3 days
13.	Children 4-16 years old: no weight gain for 2 consecutive months
14.	Children 4-16 years old: diarrhoea or vomiting for >4 days
15.	Albumin <3.5 mg/dl
16.	Cholesterol < 65 mg/dl or > 175 mg/dl
17.	Triglycerides < 40 mg/dl or > 160 mg/dl

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So far we have discussed about the details of dietary management for patients irrespective of age or physiological status. However, it is known that management of pediatric patients is difficult and a challenging task. We shall now throw some light on this aspect also.

Nutritional Considerations for Children Suffering from HIV

Young children are among the most vulnerable segment to HIV infection. This is particularly important as nearly one-fifth of the world's population is between 10 to 19 years of age and also because HIV can get transmitted from the mother to the foetus.

Table 3.5: Symptoms of Paediatric HIV Infection

Clinical Category	Symptom Severity
N	No symptom
A	Mildly symptomatic - ≤ 2 symptoms (Lymphadenopathy, hepatosplenomegaly, dermatitis, parotitis, respiratory tract infection etc.)
B	Moderately symptomatic (anaemia, neutropenia, cardiomyopathy, diarrhoea, herpes stomatitis, nephropathy, fever >1 month etc.)
C	Severely symptomatic (opportunistic infections, severe failure to thrive, wasting syndrome, HIV encephalopathy, malignancy etc.)

You will find it difficult to understand some of the technical terms.

Nutritional problems among infants and children infected with HIV are related to:

- Poor growth weight gain
- Impaired immune function and recurrent infections
- Depressed gastrointestinal tract function (malabsorption of fat, protein and carbohydrates even without diarrhoea).
- Malnutrition with multiple nutritional deficiencies
- Dysregulation of lipid metabolism and the fat redistribution syndrome
- Poor appetite, limited food preferences and taking a long time to eat.

Nutrition assessment is very important before developing a diet plan for HIV infected children. Since developmental delays may occur, therefore the infant/child's developmental age rather than the actual age should be used to assess the most appropriate textures, feeding position and utensils. As a dietician, you must make an attempt to identify the timing of meals which can be managed by the child caregiver, in particular the time (duration) the child takes to eat a particular meal.

The nutrient requirement for paediatric patients can be much higher than for adults. The major changes in macro and micro nutrients being:

- The requirements for energy increase by 100% in view of their mobility, fever and catch-up growth. The height for age data may be used as a guidance to estimate energy requirements among children with impaired growth.
- The protein requirements increase by 150% to 200% above the recommended dietary allowance to counteract nutritional demands imposed by HIV infection and also to promote adequate growth/development of the infant/child.
- The and mineral requirements are not known for paediatric patients. However, it is recommended to increase the intake by 100% through the incorporation of natural foods as micronutrient supplements are generally not recommended.

Other Special Considerations

- Alternative modes of feeding such as gastrostomy may be required. The patient's diet may be supplemented or substituted with semi-elemental or elemental formulas or age-appropriate sip-feed supplements may be given.
- Paediatric patients may have a poor appetite wherein the child should be served small frequent meals with palatable snacks in-between. Prevent children from consuming too much of water/beverages before meals.
- At times HIV infected children may take a long time to eat meals. In such situations, limit meal timings to not more than 30 minutes. Encourage praise on completion and offer extra food if desired,
- In case of infants, care must be taken while introducing new foods. Monitor the child closely for any signs of allergic reactions.

Proper nutritional care of HIV infected paediatric patients can help them in achieving timely milestones and delay the onset of AIDS and its complications, We

end our discussions on HIV infection here. Every symptom and complication of AIDS requires careful dietary care. We have high lighted the key aspects in this section. However, it would be a good diesase in detail from other knowledge sources.

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3.9 LET US SUM UP

In this unit, we studied about the defense mechanism of our body. The defense mechanism, as you would recall, comprises of the first line of defense which are generalized and specific ones, comprising of cell mediated and humoral response. Then, we moved on to the infections and fevers. Here we learnt that infection and fevers are coexistent. Fevers, as you had seen, vary depending on the nature of an infection. An infection can be either acute or chronic.

Acute infections/fevers include typhoid, malaria etc. while chronic infections include tuberculosis, HIV/AIDS. We learnt the etiology, medical and nutritional therapy for each of these. Finally, we focussed on the metabolic aberrations, the associated symptoms, dietary modifications(due to drug and nutrient interaction) and a brief list of foods to be included and excluded among other considerations.

3.10 GLOSSARY

Acute HIV infection	: patients with transient signs and symptoms of HIV infection.
AIDS (Acquired Immuse Deficiency Syndrome)	: a state of HIV infection along with a CD-4 cell count of 200 or less (or < than 14%) or dementia, wasting syndrome or malignant disease or one of 26 opportunistic infections.
AIDS Wasting Syndrome (AWS)	: involuntary weight loss of 10% of baseline body weight plus either chronic diarrhoea (2 loose stools /day Ior more than 30 days) or chronic weakness and documented fever (for 30 days or more, intermittent, or constant) in the absence of concurrent illness or other condition.
Asymptoinatic HIV infection	: patients without previous signs or symptoms leading to classification in group 111 or IV.
Basal Metabolic Rate	: a measurement of energy required to keep + the body functioning at rest.
Cachexia	: general weight loss, wasting and reduction in vitality of body and mind.

Candidiasis	: a disease caused by a species of the yeast-like fungus; affects the skin, nails.
Carriers	: a typhoid patient who is asymptomatic but continues to excrete the bacteria for weeks.
CMV	: cytomegalovirus or Lymphoma.
Expectoration	: the act of spitting out saliva or mucus from the air passages via the mouth.
Graft Rejection	: a rejection of a donated bone marrow by the patient's body.
HIV Infection	: an acute syndrome characterized by fever, malaise, lymphadenopathy, pharyngitis headache, myalgia and sometimes rash.
Kaposi's sarcoma	: malignant disease of the peripheral blood mononuclear cells which manifests itself as purple nodules on the skin, mucous membranes, lymph nodes and throughout the GI tract,
Lymphadenopathy	: swollen, firm and some times tender lymph nodes secondary to any number of causes ranging from infections to cancer.
Peripheral neuropathy	: Neurological disorder causing sensory loss in toes and fingers leading to numbness and weakness felt in hands, feet and legs,
Persistent Generalized	: patients with lymphnodes > 1cm in diameter Lymphadenopathy (PGL) that persisted for 23 months at two or more extrainguinal sites.
MAI	: Mycobacterium avium intra cellulare.
Malignant Cancer	: a tumor that has a tendency to spread to other parts of the body. diffuse musclepain usually accompanied by malaise, vague feeling of discomfort or weakness.
Opportunistic Infection	: infection by an organism that does not ordinarily cause disease but which becomes pathogenic under certain circumstances such as impaired immune response.
Peyer's Patches	: flat patches of lymphatic tissue situated in

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Pharyngitis

the small intestine mainly in the ileum and are the seat of infection in typhoid fever.

: an inflammation of the pharynx resulting from a debilitating chronic disease.

Retrovirus

: species of a family of single strand RNA virus having an envelope and containing a reverse coding enzyme that allows for a reversal of genetic transcription from RNA to DNA rather than the usual DNA to RNA, the newly transcribed viral DNA then being incorporated into the host cells DNA strand for the production of a new RNA retroviruses.

3.11 CHECK YOUR PROGRESS

- 1). Enumerate any five metabolic changes occurring in the body as a consequence of infection.
- 2). Discuss the etiology of fevers.
- 3). What is a defense mechanism? What are its two major types?
- 4). Why is typhoid called an enteric fever ?
- 5). Discuss the adverse effects of typhoid fever on the body.
- 6). List the salient features of the diet given to a typhoid patient?

4

MEDICAL NUTRITION THERAPY IN CRITICAL CARE

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STRUCTURE

- 4.1 Learning Objective
- 4.2 Introduction
- 4.3 Nutritional Management of the Critically ill
- 4.4 Special Feeding Methods in Nutritional Support
- 4.5 Let Us Sum Up
- 4.6 Glossary
- 4.7 Check Your Progress

4.1 LEARNING OBJECTIVE

After studying this unit you will be able to:

- describe the nutritional management of critically ill individuals,
- enumerate the special feeding methods for nutritional support to the critically ill individuals, and
- explain the principle and protocol for prescribing these nutritional support.

4.2 INTRODUCTION.

The prevalence of malnutrition is a common problem in critically ill patients i.e. patients who have prolonged starvation for more than 2 weeks or intensive care unit (ICU) patients, or hospitalized patients with malnutrition. Malnutrition, we know, leads to poor outcomes and therefore should be avoided or treated promptly. In this context, nutritional support has become a routine part of the care of critically ill patients and it is now widely accepted for the treatment and prevention of malnutrition and other specific conditions of nutrient deficiencies.

There is growing evidence that early and appropriate goal oriented nutritional support in the ill individual aids recovery. What is the nutritional support recommended for the critically ill patients? What is the principle and protocol for prescribing these nutritional supports? These are a few issues highlighted in this

unit.

4.3 NUTRITIONAL MANAGEMENT OF THE CRITICALLY ILL

Evidence suggest that critically ill patients or hospitalized patients with malnutrition (macronutrient and/or micronutrient deficiency) suffer from increased infectious morbidity, prolonged hospital stays, and increased mortality. Moreover, even those hospitalized medical and surgical patients without antecedent malnutrition are typically subjected to stress, infection and impaired organ function, resulting in a hypercatabolic state. Often these patients are unable to meet their caloric needs, as they are either too sick or physically unable to ingest food. In fact, critically ill patients present with extreme degrees of metabolic disarrangement in protein and energy metabolism characterized by increased protein breakdown which is not entirely suppressed by protein or energy intake. In addition there are also extreme degrees of glucose and lipid intolerance.

Thus providing adequate and optimal nutrition support to the critically ill under these conditions constitute a challenging endeavour. Nutritional support has become a routine part of the care of critically ill patient and is now widely accepted for treatment and prevention of malnutrition and specific nutrient deficiencies. The main goal of nutrition support is to provide an optimum amount of nutrients and calories to prevent malnutrition from becoming the main cause for morbidity and mortality in the disease process. The other goals of nutrition support include:

- improve nutritional assessment indices,
- prevent single and multiple nutrient deficiencies,
- promote organ integrity and function,
- ameliorate clinical manifestations of the disease,
- favourably affect the disease process, and
- positively influence the patient outcome.

In fact, what proportion of the nutrients to be delivered to critically ill has been debated for years now. Interestingly, earlier concepts in critical care nutrition has undergone considerable changes in the past decade or so. For example, few years ago, 50- 70 Kcal/kg per day was routinely being delivered during critical illness and currently, a pragmatic approach is to attempt administration of 25 kilocalories per kilogram ideal body weight per day for most patients. The total calorie daily requirement should be administered in a fluid volume consistent with the patient's needs (usually 1 ml/Kcal).

Protein sources should comprise 15-20% of the total daily calorie requirement administered as protein or amino acid depending on the route of administration. The generally accepted amount of protein is between 1.2 and 1.5 g/kg per day, except in severe losses such as burns. 30-70 % the total calories can be given as carbohydrate. This is usually given as glucose but fructose and sorbitol

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can also be used. Insulin may be required to maintain blood glucose concentration within normal limits, especially since insulin resistance is often seen as part of the response to stress. 15-30% of the total calories can be given as fat. Critically ill patients often utilize fat better than carbohydrates as an energy source and although our normal diets contain around 30% fat, it is often advantageous to provide more than this to the patients in ICU (Intensive Care Unit) or in HDU (High Dependency Unit). at least 7% of total calories should be provided as Omega-6-polyunsaturated fatty acids (PUPA) and triglycerides to prevent essential fatty acid deficiency.

Regarding micronutrient requirements, approximately mmol/kg of both sodium and potassium are usually given but this figure will need to be altered when there are excessive losses, particularly common from excess sweating and gastrointestinal losses. An often forgotten electrolyte is phosphate and this is important since it is required for normal metabolic processes resulting in the formation of ATP. Other micronutrients, e.g. magnesium, iron, copper, zinc and selenium are also necessary, but in much smaller amounts. Fat soluble vitamins (vitamin A, carotene) and water- soluble vitamins are also important, but the precise requirement for specific vitamins remain unclear. Further, there is emerging data that increased antioxidant vitamins (vitamin A, E, C) may be beneficial in various high risk populations in ICU.

The Harris-Benedict equation can be used to calculate resting energy expenditure (REE), for men and women, along with the usual multiplication factor to provide adequate calorie intake as given herewith:

Calorie requirements/day : $1.25 \times \text{REE}$ (for each IOC above 37 add 10% extra allowance)

Women REE = $655 + (9.6 \times \text{weight in kg}) + (1.85 \times \text{height in cm}) - (4.7 \times \text{age in years})$

Men REE = $66 + (13.7 \times \text{weight in kg}) + (5.0 \times \text{height in cm}) - (6.8 \times \text{age in years})$

The significant reduction in calorie intake, as suggested above, has occurred for a number of reasons. One being the recent realization that critically ill individuals during hypermetabolic stress are unable to utilize excess calories and that despite delivery of adequate nutrients, endogenous glucose production is not reversed and in fact continues. Thus, the excess calorie delivery, in fact, has been shown to result in numerous metabolic complications such as hyperglycemia (presence of high concentration of glucose in blood), hyperinsulinemia (excessive level of insulin in blood) and hepatic steatosis (accumulation of fat in the liver). Severe hyperglycemia is associated with glycosuria (excess glucose in urine) and hyperosmolar dehydration leading to grave disturbances of fluid and electrolyte homeostasis in the critically ill.

In addition hyperglycemia has also been shown to result in significant reduction in neutrophil cell function. Hyperinsulinemia, on the other hand, leads to

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increased sodium and water retention with resultant greatly increased ventilatory requirements due to impaired lung compliance as a result of increased lung and body water. Hyperinsulinemia also leads to inhibition of endogeneous lipolysis (hydrolysis of lipids) leading to greater carbohydrate utilization, increased carbon dioxide (CO₂) production and impairment of respiratory function leading to increased morbidity.

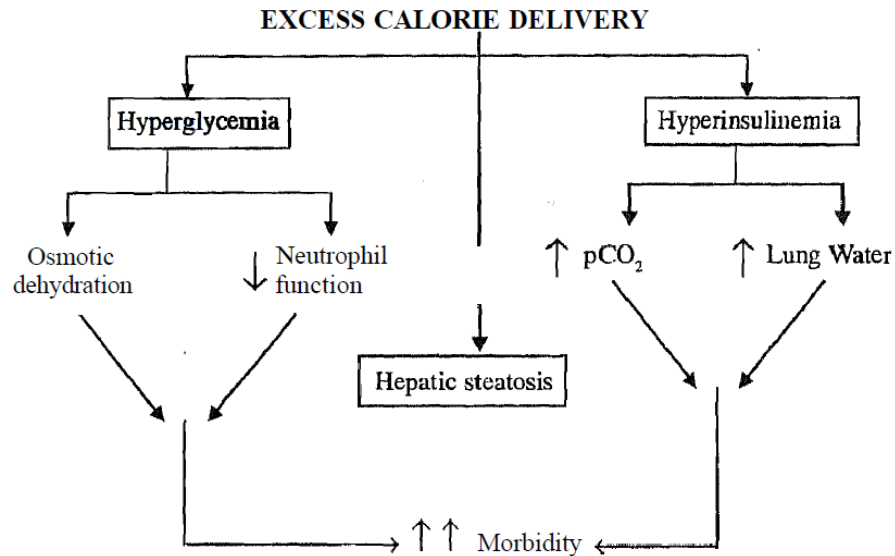


Figure 4.1: Effect of excess calorie delivery ia the critically ill

The proportion of nutrients delivered to critically ill children has also undergone considerable change over the last decade or so. As a broad outline, calorie delivery amounts to 20-30 calories / kg / day during the unstable Ebb phase followed by 50-3.00 calories / kg / day during the recovery phase (depending on individual tolerance). What do we mean by the Ebb and the recovery phase?

Protein intake of 1.5-3.0 g/kg per day depending on renal and hepatic functional status is optimal has been recommended that carbohydrate in the form of glucose should not exceed 4-6 mg/kg/minute. Hyperglycemia is often encountered in the sick stressed infant and older child. Blood sugars therefore should be retained ideally below 200 mg/dL in the critically ill stressed children. Regarding, lipid delivery, it is being recognized that long chain fatty acids (LCT) are potentially immunosuppressive when either administered rapidly or in large quantities, hence increased utilization of alternate lipid sources is becoming popular. Lipid emulsions which include medium chain triglycerides (MCT) and 0-3-fatty acids are being increasingly used. The amount of lipid administered in the critically ill child has been reduced to 15-20%of total caloric intake,

Besides the nutrients discussed above, several other specific nutrients have been reported to improve some body functions or even outcomes of hospitalized patients. The nutrients and drugs that have been reported to have beneficial nutritional effects on specific body functions and/or clinical outcomes are listed.

Table 4.1: Nutrients and drugs that have been reported to have beneficial nutritional effects on specific body functions and/or clinical outcomes

Branched Chain Amino Acids (isoleucine, leucine and valine) Medium chain triglycerides Structured lipids o-3-fatty acids	Alpha-ketoglutarate Arginine Glutamine Nucleotides Growth hormones
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4.4 SPECIAL FEEDING METHODS IN NUTRITIONAL SUPPORT

From our discussion above, it is clear that an appropriate goal oriented nutritional support is of paramount importance in both decreasing morbidity, as well as, mortality in the hypermetabolic and stressed critically ill patient. Initial attempts to achieve nutritional goals in critically ill patients should be via oral route. However, this may not be possible always. Sometimes a person cannot eat any or enough food because of an illness. The stomach or bowel may not be working quite right, or a person may have had surgery to remove part or all of these organs. Under those conditions, nutrition must be supplied in a different way. PEN is one such way. What does PEN stands for? PEN stands for parenteral and enteral nutrition.

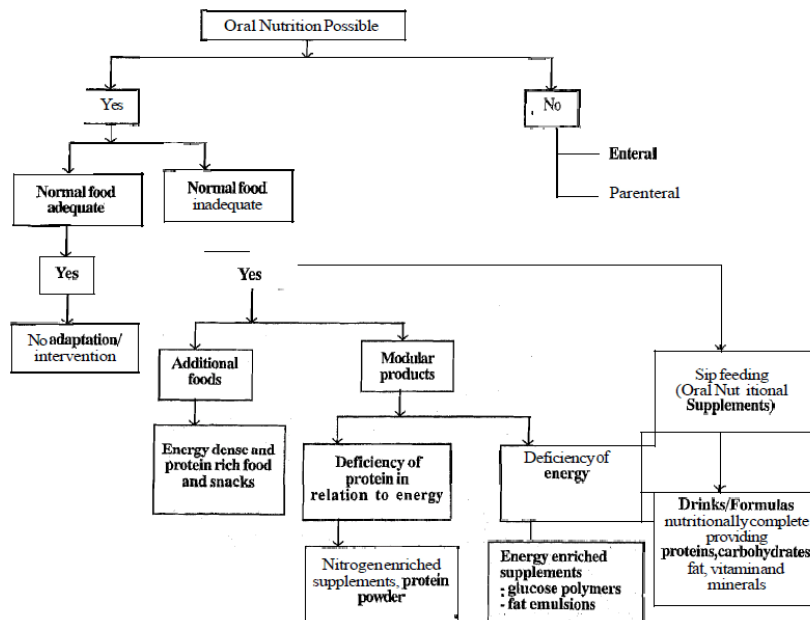


Figure 4.2: Oral feeding option

Both parenteral (pa-REN-te-rul) and enteral (EN-ta-rul) nutrition are in the form of a liquid. Enteral, is used when the gut is still partially working, but the patient cannot eat or absorb enough nutrients to stay healthy. Enteral is delivered directly into the stomach or intestine through a feeding tube. In parenteral, nutrients are delivered intravenously and the GI tract is bypassed entirely. Parenteral is given

through a catheter, which carries the liquid directly into the bloodstream.

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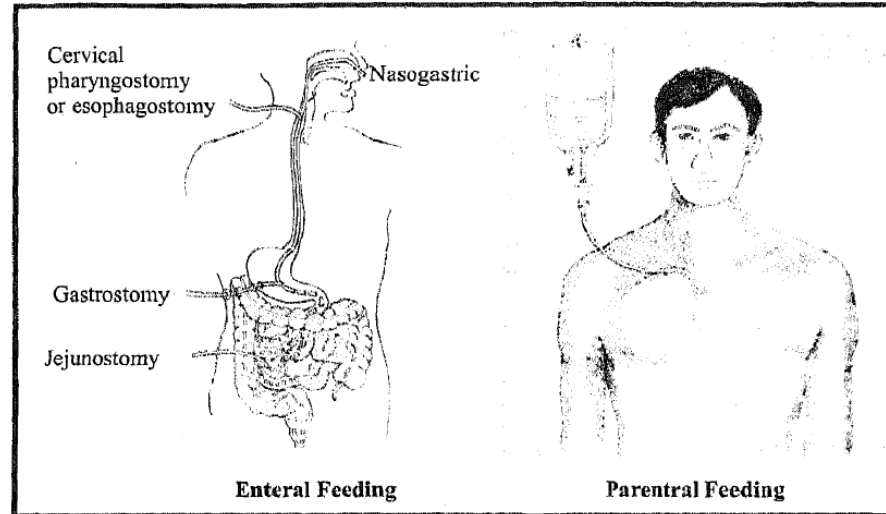


Figure 4.3: Enteral and parenteral nutrition support

The goal of nutrition intervention is to supply adequate nutrients to meet the patient's nutrient requirement by the most physiologic, safety and cost effective route. Let us get to know these two means of nutritional support better.

4.4.1 Enteral Nutrition (EN)

By definition, the term enteral means "within or by the way of the gastrointestinal (GI) tract." As described above, enteral is defined as provision of nutrition support through the gastro-intestinal (GI) tract or by accessing the gut. It also refers to feeding into the GI tract through a feeding tube. Enteral nutrition (EN) can be administered via transoral (oral ingestion of food), transnasal (administration of liquid feeds through feeding tube through the nose), or percutaneous transgastric routes (through stomach), or by a tube into the small intestine called a jejunostomy or percutaneous endoscopic jejunostomy (PEJ). Hence, enteral nutrition is often called tube feeding.

EN is a method of providing adequate nutrition that is expected to prevent, improve, or reverse malnutrition in patients who are not receiving adequate nutrition orally. Enteral is used when the gut is still partially working, but the patient cannot eat or absorb enough nutrients to stay healthy.

Some of the benefits/advantages of EN include:

- it provides nutrition when oral intake is not possible or adequate,
- it is easier to administer, present fewer metabolic and infectious complications (as compared to parenteral route),
- the intake is easily/accurately monitored,

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- enteral access is easy, gut integrity and motility are preserved and the stress response is attenuated,
- it reduces the incidence of pathogen entry or bacterial translocation into the stomach cavity or circulation,
- it provides more complete nutrients, trace elements and short chain fatty acids, as well as, fibre.
- it provides atrophic effect on the gut by promoting pancreatic and biliary secretion, as well as, endocrine, pancreine and neural factors that help promote the physiological and immunologic integrity of the GI tract.
- the supplies are readily available, and
- it is cost effective as compared to parenteral nutrition.

In animal studies, EN has shown to promote gut motility, it reduces bacterial translocation, prevents mucosal atrophy and stimulates the secretion of IgA that helps to reduce infectious complications. There is also evidence that EN improves nutritional- outcomes and results in greater wound healing.

Clinically, EN shouldnot be considered an 'all or none therapy. For patients unable to take adequate nutrition by mouth despite an appropriate modified oral diet, EN can provide the remaining calories and proteins to meet estimated requirements.

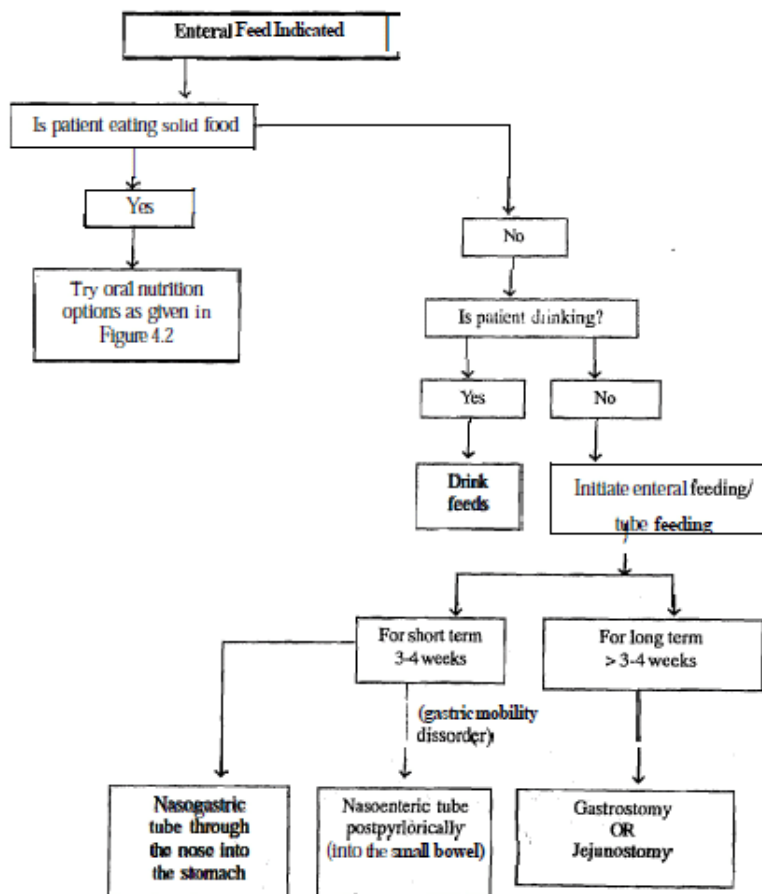


Figure 44: Indication for enteral feed

Table 4.2: Indications for enteric tube feeding for adults and children

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Indications	Conditions
For Adults Neurological indications	Severe head injuries Cerebrovascular accidents Coma Neoplasms: advanced primary and secondary intracranial tumors Dysphagia associated with neurological disorders
Hypermetabolism	Postoperative major surgery Sepsis Trauma, burns, organ transplant, acquired immune deficiency syndrome
Surgical indications	Facial and jaw surgeries Head and neck surgeries Oropharyngeal surgeries Pharyngoesophageal surgeries Polytrauma associated with extensive abdominal surgeries Patients with burns for surgeries unable to take oral nutrition Surgery complicated with sepsis
Gastrointestinal (GI) disease	Short-bowel syndrome (if absorptive capacity of remaining bowel is sufficient e.g. approximately a minimum of 100 cm jejunal and 150 cm of ileal length of functioning small bowel with ileocecal valve intact) Inflammatory bowel disease Minimal GI tract fistula output (less than 500 ml/d) Pancreatitis Oesophageal obstruction Malabsorption Fistulas
Cancer Resistance to oral intake	Oral malignancies Oropharyngeal malignancies Nasopharyngeal malignancies Head and neck malignancies Oesophageal malignancies Gastric malignancies Chemotherapy Radiotherapy Anorexia Dysphagia Severe depression
Malnutrition	Protein energy malnutrition with inadequate oral intake for at least 5 days Malnutrition preoperatively and postoperatively Malnutrition in cancer patients Malnutrition in patients with Acquired Immune Deficiency Syndrome (AIDS), who are unable to take oral nutrition Malnutrition in debilitated aged patients
Organ system failure	Respiratory failure Renal failure Cardiac failure Central nervous system failure Hepatic failure Multiple organ system failure
For Children	Malnutrition, malabsorption, hypermetabolism, failure to thrive, prematurity, disorders of absorption, digestion, excretion, utilization, or storage of nutrients

Once the indication for enteral nutrition is established, the next issue that confronts us is what are the types of enteral feeds/formulas that are available and can be delivered? The Drug and Food Administration (FDA), USA recognizes enteral formulas as a category of product independent from regular foods, dietary supplements or drugs. Multitudes of enteral formulas are available for infusion. The formulas have been traditionally divided into polymeric, oligomeric and modular. However, there are feeds, which can be home made or prepared with natural food items, or feeds, which are based on polymeric enteral diets such as disease specific feeds or opportunistic feeds.

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Table 4.3: Enteral feeds and their specific characteristics

Enteral Feeds/Formula	Specific Characteristics
<p>Polymeric formulas (also called defined formula diets)</p> <p>This is the general purpose, most widely prescribed feed. It is the sole source of nutrition intake for critically ill individuals with or near normal GI function.</p>	<ul style="list-style-type: none"> - Provide nitrogen as whole protein, often casein, egg white solids or soy protein. - Carbohydrate is provided as corn syrup, maltodextrins or glucose oligosaccharides, with sucrose added for sweetness in oral formulas. - Fat is usually provided as soy oil, although corn oil and safflower oil may be used. Medium-chain triglycerides (MCT oil) are rarely used.
<p>Oligomeric formulas (also called elemental or semi-elemental diets)</p> <p>Oligomeric diets are predigested and formulated to require minimal digestion by the gastrointestinal tract. In other words, these diets are "complete."</p>	<ul style="list-style-type: none"> - Most of these formulas provide enough protein, calories, water, electrolytes, minerals, vitamins and trace elements in 2 L/day for most "nonstressed" patients - Provide nitrogen as oligopeptides from partially hydrolyzed whole protein or as crystalline amino acids. - Carbohydrate tends to be provided as glucose oligosaccharides or glucose. - Fat is usually present in small quantities, enough to meet the requirement for linoleic acid (an essential fatty acid), which is about 2-4% of total calories. MCT oil is added to some formulas. - Oligomeric diets have been commercially promoted as ideal for patients with decreased bile output (cholestasis), pancreatic insufficiency and short bowel.
<p>Modular formulas/feeds (used when a particular component of the diet requires an increased intake or if a patient requires a special blend of diets)</p> <p>These modules are not required for the majority of patients, and are rarely used</p>	<ul style="list-style-type: none"> - Modular formulas are those that contain or predominantly contain one kind of nutrient. - There are commercially available modules for protein, fat, carbohydrates, vitamins, electrolytes and trace elements. Examples of this might include burns or protein-losing enteropathy, if more protein is to be given; or liver disease, if less protein is to be given.
<p>Blenderized feeds (For chronically ill patients with normal GI functions)</p>	<ul style="list-style-type: none"> - Prepared by mixing the ingredients and delivered in an easily digestible form. - Provide carbohydrates, proteins and fat in the amount as in the balanced diet. - For long-term nutritional management. - Natural food items are used to preparing the feed.
<p>Disease specific feeds (these are specially formulated polymeric enteral feeds)</p>	<ul style="list-style-type: none"> - For renal patients - For liver disease patients (specialized amino acid solutions have been made for use in special circumstances. For example, liver disease, renal disease and "stress," such as trauma and sepsis. For liver disease, these solutions are composed mostly or exclusively of branched-chain amino acids, whereas for renal disease the solutions are predominantly essential amino acids.
<p>Opportunistic feeds (with nutritional addition and substitution which are suggested to improve various aspects of organ function)</p>	<p>Addition and substitution include:</p> <ul style="list-style-type: none"> - more middle chain triglycerides (MCT) - increased level of n-3 fatty acids, carnitine, beta carotene, RNA, arginine, glutamine etc.
<p>Drink feeds (for those who cannot eat solid foods but can ingest liquid diets)</p>	<p>Nutritionally complete enteral feeds based on polymeric enteral diets Palatable</p>

Therefore, it is evident that various enteral formulas are available for infusion. So, which one to select? Well, the factors to consider when choosing an enteral formula include:

- the type of protein, fat, carbohydrate and fibre in the formula as related to the patients digestive and absorptive capacity,
- calorie and protein density of the formula (i.e. Kcal/ml, g protein/ml and Kcal: nitrogen ratio),
- sodium, potassium, magnesium and phosphorous content of the formula, especially for patients with cardiopulmonary, renal or hepatic failure, and
- viscosity of the formula related to tube size and method of feeding.

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Box 1	Enteral Formula Composition
<ul style="list-style-type: none"> - Most of the formula provide 1.0-1.2 Kcal/ml. In high concentrations, they may provide 1.5-2.0 Kcal/ml. - <i>Proteins</i> in enteral formulas provide 4% to 32% of total calories. Those formulas providing 18 to 32% of calories are considered high-protein solutions. - <i>Carbohydrates</i> contribute 40% to 90% of total calories in enteral formulas. Carbohydrate sources used in formulas are pureed fruits and vegetables, corn syrup solids, corn and tapioca starch hydrolysates, maltodextrins, sucrose, fructose and glucose. - Lipid provides 1.5% to 55% of the total calories of enteral formulas. - <i>Water</i> recommended. <ul style="list-style-type: none"> • Healthy adult : 1 ml/Kcal or 35 ml/kg. • Healthy infant: 1.5 ml/Kcal or 150 ml/kg. • Elderly: consider 25 ml/kg with renal, liver, or cardiac failure; or consider 35 ml/kg if history of dehydration • Normal tube feeding: 1 Kcal/ml; 80% to 85% water 	

Table 4.4 : Enteral nutrition commercial formulas for paediatric use

Product	Company	Calories/100 g
NOURISH	Claris	518
SIMYL MCT	FDC	460
PEDIA SURE (powder, reconstitute with water, ideal for both oral and tube feeding)	Abbott	496
PROSOYAL	FDC	506
IMPACT (IB)	Novartis	484
NOVASURE	Novartis	400
RESOURCE JUNIOR (contain lactose, fibre and gluten free)	Novartis	200 (per sachet of 42 g)
LACTODEX (complete low lactose, low fat nutrition during diarrhoea)	Raptakos	55 (each 100 ml of reconstitution)
LACTODEX-LBW (feeding of preterm/LBW infants until sufficient mother's milk is available)	Raptakos	80.3 (each 100 ml of reconstitution)
MILK CARE LBW (life saving formula for premature/LBW infants)	Dalmia	501
ZEROLAC (for lactose intolerance, acute chronic diarrhoea)	Raptakos	64 (each 100 ml of reconstitution)
ENERGEX (lactose intolerance to cow's milk)	Indon	506
TROPHOX (protein supplement)	Rapatakos	

Table 4.5: Enteral formulas for children over 4 years of age

Complete Balanced Nutrition Formula	Balanced Formula with Additives	Semi- elemental, Partially Hydrolyzed	Disease Specific
PEDIASURE (Powder) FRESUBIN- liquid: lactose, cholesterol and gluten free NUTREN 1.0- powder RESOURCE - powder: lactose free HORLICKS PLUS: Powder	Fresubin Isofibre Nutren fibre	PERATIVE- partially hydrolyzed protein SURVIMED OPD - oligopeptide PEPTAMEN - exceptional peptide formula	DIABETIC Glucerna Fresubin Diabetic Resource Diabetic Nutrocal DM HEPATIC Fresubin Hepa Resource Hepa RENAL <i>Nephro</i> - post dialysis, 475 Kcal, can be given orally <i>Suplena</i> - pre dialysis, 475 Kcal, can be given orally <i>Nutrenal CRF</i> - pre dialysis, 518 Kcal, can be given orally <i>Nutrenal TM</i> - post dialysis, 518 Kcal, can be given orally

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In the formulas listed above, you would have noticed the calorie content given for each formula. How is the energy and protein content of the formula determined? The formula used includes:

- Kcal/ml x ml given = Kcal
- % protein x Kcal = Kcal as protein
- Kcal as protein x 1 g / 4 Kcal = g protein

Example: Patient drinks 100 ml of a 18.2% protein product that has 1 Kcal/ml. Therefore the calorie, protein content would be:

- 1 Kcal/ml x 100 ml = 100 Kcal
- 0.182 % protein x 100 Kcal = 18.2 Kcal
- 18.2 Kcal x 1g protein / 4 Kcal = 4.55 g protein

The common methods of administering the enteral formulas include:

- Continuous method = slow rate of 50 to 150 ml/hr for 12 to 24 hours,
- Intermittent method = 250 to 400 ml of feeding given in 5 to 8 feedings per 24 hours, and

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Bolus method = may give 300 to 400 ml several times a day.

With a detail review of the nutrient composition and method of administering the enteral formulas, we shall end our study on enteral nutrition by reviewing the disadvantages and complications, if any linked with enteral feeding.

Disadvantages and Complications of Enteral Feeding

There are a few common complications linked with the use of enteral feeding. These complications include:

Access problems (tube obstruction)

Administration problems (aspiration, tube migration)

Gastrointestinal complications (diarrhoea)

Metabolic complications (overhydration)

Besides the complications mentioned above, use of enteral feeding is associated with increased risk of bacterial contamination especially in case of home made blendrized foimula. Further, they may be labour intensive and may require site care and monitoring. Thky are 'less palatabley and do cost inore than oral diets.

4.4.2 Parenteral Nutrition

Enteral nutrition, we learnt, above means within or by the way of gastrointestinal tract. Parenteral nutrition, on the other hand, refers to nutrients delivered to the patient in a manner other than through the gastrointestinal (GI) tract — usually delivered intravenously (bypassing the digestive tract)

Parenteral nutrition is one of the ways people receive food when they cannot eat and there is a dysfunctioning of the digestive tract. It is a special liquid food mixture administered into the blood through a vein. The mixture contains all the protein, sugars, fat, vitamins, minerals, and other nutrients needed. It is sometimes called "total parenteral nutrition," "TPN," or "hyperalimentation." Parenteral nutrition, in fact, can be of two types - total parenteral nutrition (TPN) and partial parenteral nutrition (PPN). TPN supplies all of the patient's daily nutritional requirements. Partial parenteral nutrition, on the other hand, supplies only part of the patient's daily nutritional requirements, supplementing oral intake. Many hospitalized patients receive dextrose or amino acid solutions by this method as part of their routine care.

When is the use of parenteral nutrition indicated? Parenteral nutrition support is indicated in the presence of compromised nutritional status when adequate nutrients (protein and calories) cannot be provided by oral or enteral route or when oral or enteral feeding is insufficient (as in burns or polytrauma), undesirable (as in the case of fistulas), ineffective (short bowel syndrome, severe malabsorption) or impossible (intestinal obstruction or pseudo obstruction). In other words, parenteral nutrition is generally used when the enteral route is either inaccessible or its use is contraindicated. It is also used as a supplement to enteral

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feeding if adequate nutrition is not possible via the enteral route alone. This type of nutrition is used in the most critical patients, which may have one or more of the following symptoms:

- Intestinal obstruction or ileus,
- Inadequate digestive or absorptive capacity,
- Uncontrollable vomiting (this is particularly life threatening to a diabetic patient),
- Inability to tolerate food for any reason (e.g. head trauma, burns to mouth/face/oesophagus),
- High risk of aspiration because patient is unconscious or has a neurologic problem, and
- Need for complete GI tract rest due to digestive disease, healing time needed for GI tract lesions or surgical repairs, acute pancreatitis or hepatitis.

In infants and children parenteral nutrition is indicated during intestinal failure (short gut, protracted diarrhoea, post-operative abdominal or cardio-thoracic surgery, radiation etc.), organ failure (acute renal or liver failure), and hyper-catabolism (as in extensive burns, severe trauma etc.).

The advantages of using parenteral nutrition is that it:

- provides nutrients when less than 2 to 3 feet of small intestine remains, when surgical procedures are carried out and
- allows nutrition support when GI intolerance prevents oral or enteral support

Parenteral support is generally given for a short period (two weeks or so), at which point the patient has hopefully begun to recover from the symptoms that caused the need for this type of support in the first place. PN may include a combination of sugar and carbohydrates (for energy), proteins (for muscle strength), lipids (fat), vitamins, electrolytes, and trace elements. Electrolytes include sodium, potassium, chloride, phosphate, calcium, and magnesium.

Trace elements include zinc, copper, manganese, and chromium. Vitamins include vitamins - A, C, D, E, K, B₁ (thiamine), B₂ (riboflavin), niacin, pantothenic acid, B₆ (pyridoxine), B₁₂, biotin, choline (cotactor for enzymatic reactions), and folic acid.

Let us take a closer look at the nutrient composition of parenteral nutrition.

Carbohydrates are principally provided through glucose and dextrose. Parenteral nutrition is initiated with a glucose infusion at a dosage of 5 mg/kg/minute and increased in daily increments to a maximum of 25 gm/kg/day.

Protein requirements can be met by providing the appropriate amounts of amino acids. The range in adults is from 0.5 to 3.5 g/kg/day. Protein requirement of 1.2 to 1.5 g protein/kg ideal body weight (IBW) is recommended in case of mild

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or moderate stress and 2.5 g protein/kg IBW in case of burns or severe trauma as you would read later in Unit 5. Proteins are generally provided as crystalline amino-acid solution in standard 500 ml bottles. The solutions vary in amino-acid concentration (3, 3.5, 5, 7, 8.5, 10% solutions) and composition. They can be selected based on underlying disease condition.

Lipids in parenteral nutrition are used as a source of essential fatty acids and energy. Fat emulsion solutions are available as 10% (1.1 Kcal/ml) or 20% (2 Kcal/ml) preparations, and are derived from soybean, safflower, or cotton-seed oil, with the fat mainly present as triglyceride. The ultimate total daily dose of parenteral lipid emulsion should not exceed 4 g/kg. 500 ml of 10% lipids given once or twice a week is enough to prevent the deficiency of essential fatty acids.

The fluids should be customized and prepared on a daily basis as per requirement and tolerance. 30 to 50 ml per kg is the fluid recommendation. The calculated quantities of amino acids, glucose and electrolytes are mixed in a bottle to which calcium gluconate, phosphates, multivitamin and heparin is added. The nutrient mixing should be carried out under aseptic conditions.

An example of the daily intravenous requirements, particularly for infants and children, for few nutrients..

Table 4.6: Daily intravenous nutritional requirements in infants and children

Body weight	Kcal/ kg	Fluid (ml/kg)	Carbohydrate (g/kg)	Protein (g/kg)	Fat (g/kg)	Sodium (mmol/kg)	Potassium (mmol/kg)
<10 kg	100	100-120	14	2.5	3	3	2.5
10 - 30 kg	75-100	60-90	7.5	2.0	2	3	2.5
>30 kg	45-75	40-90	5	1.5	2	3	2.5

Table 4.7: PN products available in Indian market

Now that we have a basic understanding about parenteral nutrition, it is also important for us to realize that there are certain precautions and complications linked with the use of parenteral nutrition. These are enumerated herewith:

Blood parameters needs daily check.

This type of nutritional support requires administration within the hospital due to the need to use sterile technique in handling the catheter and solution.

Also patient's requiring this support are generally very critical and need the supervision of 24 hour intensive care support, and

The risk of infection is the greatest worry with this type of nutrition requiring catheter. Aseptic (sterile) technique must be used at all times.

Sepsis is the most frequent serious complication during PN, resulting in increased morbidity (due to technical and metabolic complications), mortality and healthcare costs. Common sources of infection include *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Candida* species, *Pseudomonas* species, and *Escherichia coli*.

4.5 LET US SUM UP

In this unit, the focus was on the nutrient requirements of the critically ill individuals and on the types of nutrition support to be provided to such individuals.

We studied that the earlier concepts in critical care nutrition have undergone considerable changes in the past decade or so. Currently, a pragmatic approach is to provide 25 kilocalories per kilogram ideal body weight per day for most patients. The total caloric daily requirements should be administered in a fluid volume consistent with the patient's needs (usually 1ml[Kcal]). Protein sources should comprise 15-20% of the total daily calorie requirement. The generally accepted amount of protein is between 1.2 and 1.5 g/kg per day, except in severe losses such as burns. Glucose should comprise 30-70% of the total calories and fats 15-30%.

Both parenteral and enteral nutrition are the types of nutrition support administered to the critically ill patients. Both are in the form of a liquid. Enteral is delivered directly into the stomach or intestine through a feeding tube. In parenteral, nutrients are delivered intravenously and the GI tract is bypassed entirely. The indication for administration of these feeds varies and different types of products are available in the market. Some of the enteral feeds can be homemade as well. Nutritional support, therefore, does influence the outcome of critically ill patients and evidence suggests that the consistent achievement of nutritional goals is important and this should be feasible through the enteral or parenteral route.

4.6 GLOSSARY

- Catheter** : a flexible tube used to deliver fluids into or withdraw fluids from the body.
- Hepatic steatosis** : refers to simple fatty liver, i.e. the accumulation of fat in the liver cells with no inflammation or scarring.
- Hyperglycemia** : a condition characterized by the presence of an abnormally high concentration of glucose in the blood.
- Hyperinsulinemia** : a condition, present in people with Diabetes Mellitus (type 2) or insulin resistance where excess levels of circulating insulin is present in blood.

Jejunostomy : a way to provide food through a tube placed into the small intestine

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4.7 CHECK YOUR PROGRESS

- 1). Enumerate the nutrient requirements of the critically ill adult.
- 2). What are the types of nutrition support we can provide to a critically ill individual?
- 3). What is parenteral nutrition? How does it differ from enteral nutrition?
- 4). What are the indications for providing parenteral support to patients?

5**NUTRITION DURING STRESS****NOTES****STRUCTURE**

- 5.1 Learning Objective
- 5.2 Introduction
- 5.3 The Stress Response
- 5.4 Surgery
- 5.5 Burns
- 5.6 Trauma
- 5.7 Sepsis
- 5.8 Let Us Sum Up
- 5.9 Glossary
- 5.10 Check Your Progress

5.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- define a stress response,
- enlist the various phases of a stress response,
- discuss the physiological, hormonal and metabolic changes during situations of stress such as surgery, burns, trauma and sepsis, and
- describe the nutritional support required for these stress conditions.

5.2 INTRODUCTION

We all experience stress at some time or the other in life. Stress is the condition or stimulus that threatens the body's homeostasis. Stress may be physical and/or mental and may develop due to a number of reasons. Emotional stress results from feelings of ambition, divine and desire but is perceived as positive. However strain, tension and anxiety due to death of a dear one, financial problems, divorce, unemployment, sickness and injury, etc. are negative forms of emotional stress. Physical stress may occur in the form of starvation, illness, surgery, infection, injury, burns or trauma. We must also remember here that following a major stress, patients often exhibit a characteristic behaviour. These include immobility, when

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patients are fearful of moving or interacting; withdrawal, when patients may cease being aware of their environment and become incommunicative; and antagonism, when patients may resist interaction and display hostility to those around them. Altered cerebral blood flow may also be a reason for altered mental state.

In this unit, we shall be discussing the metabolic alterations and the importance of good nutrition in combating the ill effects of stress such as surgery, burns, trauma and sepsis. We shall first however, brief ourselves regarding the major physical/metabolic changes which may develop once the stress response has been activated.

5.3 THE STRESS RESPONSE

The terms trauma, stress, shock are very often used interchangeably and encompass a variety of conditions such as sepsis (infection), burns, injury (accidents, wounds), surgery (elective/emergency) etc. Before we proceed towards understanding the physical and metabolic responses of the human system towards stress, it is important to remember that whether the event is in the form of sepsis (infection), trauma (including burns), or surgery, once the systemic response is activated, the physiologic and metabolic changes that follow are similar and may lead to shock and other outcomes. Variable responses relate to patient's age, previous state of health, pre-existing disease, type of infection and presence/absence of multiple organ dysfunction syndrome (MODS).

It has long been recognized that the physiological response of the patient to a stress or disease process will very largely determine the outcome. To an extent this will depend on the extent of the shock and injury; this tends to be minimal for minor surgery or injury and extensive for major accidental or surgical trauma. However, the physiological reserve of the individual is also important. Signals that are initiated in injured or ischaemic tissues communicate the extent of the injury systemically. These stress responses are necessary for the process of recovery from injury. However, when trauma is severe, the resultant physiological responses are extensive and sustained, such that the same responses may be detrimental and contribute to the progression to critical illness and even death. Stress response means major changes in metabolism that occur after severe injury, illness or infection. The nutrient needs of the body are greatly altered as a result of this response. There is accelerated catabolism of lean body or skeletal mass resulting in muscular wasting and a negative nitrogen balance. The metabolic responses to critical illness have been studied in a variety of critically ill patients, especially those with trauma, burns, or sepsis. The responses are often grouped into phases on the basis of their temporal relation to the injury or insult. The stress response is therefore referred to as a dynamic process that has an ebb phase, a flow phase and an anabolic phase. Let us discuss these phases one by one.

Ebb-Phase : This occurs immediately following injury and lasts for

approximately 24-hours. The so-called ebb phase, which is the early phase of the injury response, is characterized by:

- an elevated blood glucose level,
- normal glucose production,
- elevated free fatty acid levels,
- low insulin concentration,
- elevated levels of catecholamines and glucagon,
- an elevated blood lactate level,
- depressed oxygen consumption,
- below-normal cardiac output, and
- below-normal core temperature.

The ebb phase is dominated cardiovascular instability, alterations in circulating blood volume, impairment of oxygen transport, and heightened autonomic activity. Emergency support of cardiopulmonary performance is of paramount therapeutic concern. Shock is the clinical manifestation of the ebb phase. After effective resuscitation has been accomplished and restoration of satisfactory oxygen transport has been achieved, the next phase i.e flow phase comes into play.

Flow Phase: This is a neuro-endocrine response to physiological stress following the ebb phase. This phase is characterized by:

- nonnal or slightly elevated blood glucose level,
- increased glucose production,
- normal or slightly elevated free fatty acid levels, with flux increased,
- a normal or elevated insulin concentration,
- normal or elevated levels of catecholamine and an elevated glucagon level,
- a normal blood lactate level,
- elevated oxygen consumption,
- increased cardiac output, and
- elevated core temperature.

It is characterized by hyper metabolism/catabolism. Increased cardiac output, urinary nitrogen losses, oxygen consumption, body temperature and energy expenditure occurs during the flow phase. There are also profound hormonal changes. As a result, there is breakdown of body protein stores to provide glucose and hence a rapid loss of nitrogen in the urine. Blood flow to the gastrointestinal tract is of en reduced during this phase. This decreases, the sllpply of oxygen and nutrients to the gastrointestinal tract. The secretion of mucus is decreased, whereas, gastric acid secretion is increased. This leads to wasting of the cells lining the gastrointestinal tract resulting in diarrhoea and bloating.

Recovery or Anabolic Phase: When wounds are closed and infection has resolved, repletion of lean tissue and fat stores along with restoration of strength

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and stamina can begin. This final, anabolic phase often begins near the time of hospital discharge' and may persist for months before the patient fully recovers and is characterized by building up of body tissue and nutrient stores (anabolism). This phase is also marked by hormonal changes. There is an increase in the release of insulin and growth hormones. The patient's progress to the anabolic phase is important and depends on a number of factors. Age, severity and duration of the stress, as well as, the individual's prior nutritional status influence tissue growth and anabolism. Attempts to restore body mass and nutritional status rapidly may induce adverse metabolic consequences. Underweight and overweight patients and the elderly are particularly vulnerable to overfeeding, because of the difficulties in assessing true requirements.

Patients with ample nutrient stores to draw on during stress are better able to tolerate the negative effects of the stress, especially in case of emergency or unexpected stress. Having learnt about the stress response and its three phases, next let us move on to the study of the stress conditions, namely surgery, burns, trauma and infections, which have a great impact on metabolism

We shall first begin with surgery. We will learn about the changes in the metabolism of the body before and after surgery and how can an appropriate nutrition support minimize the stress/contraindications of surgery thereby promoting a speedy recovery.

5.4 SURGERY

Surgery ! Does the word itself not create a feeling of anxiety and bring our thoughts towards a debilitating state of health which would be accompanied by pain, inability to move and a high susceptibility for severe morbidity/mortality! Well, surgery, in fact, is one of the most stressful situations encountered in life. But have you ever focused your thoughts as to what exactly does the term surgery mean? or during which situations is surgery performed? What is the impact of a surgical procedure on our health and how can judicious utilization of medical and dietary services improve the ultimate outcome? Let us discuss these aspects one by one.

Well, surgery is that branch of medical science which has for its object the cure of local injuries or diseases, as wounds or fractures, traumas etc., whether by manual operation or by medicines along with constitutional treatment. A surgical procedure may be conducted in response to a sudden injury/trauma as you may have witnessed in case of road accidents/crush injuries. This is referred, to 'Emergency Surgery' during which the patient is in a variable state of resuscitation and the objective of treatment is to preserve as many organs and bodily functions as possible with minimum further trauma. Surgery however may not always be an outcome of an emergency. It may be undertaken as part of a well planned patient care process and involves removal or reconstruction of

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organs/body parts to promote treatment or for cosmetic purposes. Such surgical procedures are referred to as 'Elective Surgery'. By-pass surgery of the heart, removal of organs, limb amputations, laproscopies are some common examples of elective surgery. The difference between elective and emergency surgery lies in the ability to prepare the patient for the injury and to control homeostasis/stress response in the elective surgical patient, while this is not possible in the traumatized patient. We must however remember here that whatever may be the form of surgery or its subsequent prognosis, the response of the human body to both forms of surgeries is quite similar. In our subsequent discussions we will help you in understanding some of the key physiological/metabolic responses to surgery which in-turn affect the nutritional requirements of the patient.

5.4.1 Physiological Response to Surgery

Although advances in medicine and nutrition support have greatly reduced the morbidity and mortality associated with surgery, debility commonly accompanies surgical illness. It occurs in varying degrees after elective/emergency operations and other critical illnesses. Debility is caused by a variety of factors, including specific biochemical and physiologic alterations that usually occur in response to injury and disease, especially those that persist for a long time. Virtually all surgical patients experience some pain. Pain usually occurs in association with an incision or with a wound resulting from fracture, burn, contusion, or any other type of injury. In addition to creating an unpleasant subjective experience, pain often limits physical activities, such as turning in bed, deep breathing, coughing, and walking, and thereby directly interferes with recovery. Elevation of body temperature above normal, leukocytosis, and other signs of inflammation are common features of critical surgical illness and should be expected. The extent of temperature elevation is generally proportional to the severity of illness. In a patient with a major burn — an extreme example of critical surgical illness — body temperature may be as high as 39°C (102°F). The leukocyte count is also typically elevated and may be as high as 20,000 cells/mm³ during satisfactory recovery.

Food is commonly withheld from the patient before and during various diagnostic and therapeutic procedures, as well as, before operations or after injury. Starvation for several days appears to be well tolerated by patients who were relatively well nourished before their critical illness. However, if food deprivation is prolonged, the complications of starvation will compound the effects of critical surgical illness. It is generally recommended that total starvation should usually be limited to a period no longer than 3 or 4 days.

Several metabolic changes also occur as a consequence of injury/surgery. For instance, injury caused by the operation initiates an inflammatory response resulting in the release of cytokines and acute phase proteins, along with the activation of stress hormones. The release of these mediators causes a change of metabolism into a catabolic state. There is a rise in the levels of circulating cortisol due to increased production of the adreno-corticotrophic hormone from the

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pituitary gland. Cortisol mobilizes amino acids from skeletal muscle to provide the substrate for wound healing and for hepatic synthesis of new glucose. The excitement, pain, fear and hypovolemia that accompany surgery stimulate the sympathetic nervous system which leads to increased production of epinephrine by the adrenal medulla. Injury also initiates the release of aldosterone, (a corticosteroid that causes renal sodium retention, and of vasopressin (anti-diuretic hormone), which stimulates renal tubular water resorption. The action of these hormones results in conservation of water and electrolytes. Weight gain secondary to salt and water retention occurs due to changes in the response of pancreas. There is diminished secretion of insulin, whereas, glucagon production is increased. These responses are also a result of increased sympathetic nervous system activity. A rise in glucagon and fall in insulin levels are signals to accelerate glucose production and maintain gluconeogenesis. The postoperative hormonal responses are beneficial to the patient. Salt and water conservation support the circulating blood volume. Increased glucose production provides adequate fuel for the nervous system. Skeletal muscle protein breaks down at an accelerated rate after surgery resulting in the release of a variety of substances into the circulation, including creatinine, creatine, 3 methyl histidine, potassium, magnesium and amino-acids. The amino acids serve as precursors for protein synthesis in wound healing and in the liver. Lipid metabolism is also affected by critical illness. There is almost a two fold increase in glycerol turnover which is indicative of an accelerated rate of triglyceride hydrolysis to form free fatty acids and glycerol. There is also a high rate of free-fatty acid recycling.

Finally, a word about the convalescence stage.

Stages Convalescence : This period of catabolism and alteration of the hormonal environment is known as the 'adrenergic — corticoid phase'. This is followed by a set of anabolism, which, in the absence of any postoperative complications, starts 3 to 5 days after a surgery. This 'turning point' from catabolism to anabolism is termed as 'corticoid — withdrawal phase' as there is spontaneous sodium and water diversion, and a reduction in nitrogen excretion, This phase lasts for 1 to 3 days. The patient then enters a period of early anabolism characterized by positive nitrogen balance and weight gain. Sustained feedings lead to protein synthesis and there is formation of lean mass and return of muscular strength. Then there is a final phase of late anabolism when there is much slower weight gain. There is deposition of body fat and nitrogen equilibrium is achieved.

Now that we are aware of the physiological response to surgery, let us in this context also understand dietary management of surgery.

5.4.2 Dietary Management during Surgery

Surgery increases the nutritional demands of the body; and can lead to the elicitation of several nutritional deficiencies/imbalance. Malnutrition compounds the severity of complications as far as surgery is concerned and is associated with

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a high incidence of postoperative morbidity and mortality. The cellular processes involved in wound healing are critically dependent on adequate perfusion (delivery) of oxygen, glucose, and other essential nutrients. Inadequate perfusion may result in relative tissue ischemia (flow or flood restricted) and delayed wound healing. Nutritional support of critically ill patients is important both for promoting protein synthesis and other anabolic processes essential to recovery and for reducing the net drain on the patient's fuel and protein stores. Enteral nutrition is preferred, but the availability of effective intravenous techniques allows the clinician to provide appropriate nutrition to virtually all patients as you may recall reading in the last unit. Exercise and mobility have clear anti-catabolic effects and should be initiated as early as is practicable. A principal responsibility of the dietician is therefore to ensure adequate tissue perfusion during the entire period of wound healing. Thus, a complete nutrition and health assessment of the patient is essential to determine the macro- and micronutrient requirements before and after surgery. A complete assessment must include:

Physical examination (anthropometric measurements such as ideal/usual body weight, skinfold thickness etc.);

Clinical examination (presence/absence of oedema; abnormal changes in the skin, eyes, hair etc. cardio-pulmonary function, functional status of vital organs- kidney, pancreas, brain etc.);

Bio-chemical examination (all important blood, urine, faecal components, as well as, enzymes/hormone levels that may be of immediate metabolic consequence)

Medical and diet history.

A comprehensive dietary management regime of the patient should be based on the patient profile as gathered pre and post operatively and should be able to minimize the physiological and metabolic alterations associated with surgery.

To minimize the reasons of weight loss and depletion of tissue reserves

To maintain an optimum energy and nitrogen ratio

To achieve and maintain a healthy body weight

To promote anabolism and hence wound healing

To help in replenishing the depleted nutrient reserves

In view of the above mentioned objectives, we shall now discuss the nutritional management of patients before and after surgery. Let us start with the pre-operative nutritional care of patient preparation for an elective surgery.

A. Preoperative Nutritional Care

It can be provided only to prospective candidates of elective surgery and is not feasible for emergency cases. Preoperative malnutrition is often a cause of poor postoperative outcome. Nutritional support should be given for 7-14 days to moderately or severely malnourished patients undergoing a major surgery.

any nutritional deficiency should be corrected and nutritional reserves must be maintained for the surgery period as well as, for the immediate postoperative period to ensure proper rehabilitation.

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Energy: The energy requirements of the patient should be based upon his present body weight which should be followed by a comparative assessment with his usual body weight (if data is available) or his ideal body weight. Adequate energy intake is important to build up weight deficit and build glycogen/adipose tissue reserves which are essential to provide energy to the body immediately after surgery when the nutrient intake may not be adequate enough to meet the increased requirements. Depending upon their nutritional status the energy requirements of the patients would be as follows:

Underweight: - 35 Kcal/kg ideal body weight per day

Normal weight: - 30 Kcal/kg ideal body weight per day

Overweight: - 20-25 Kcal/kg ideal body weight per day

- **Carbohydrates:** Carbohydrates are needed to build up glycogen stores and spare the proteins for tissue synthesis, Around 60% of the total energy should be provided from carbohydrates. Maintaining blood glucose levels around 4.5-6.0 mmol/litre (with/without insulin) helps in marked reduction in septic episodes, renal failure, time on the ventilator, polyneuropathy and mortality.
- **Protein:** Negative nitrogen balance is the most common nutritional deficiency related to surgery. Reserves of protein in the tissues and plasma help to overcome blood losses during surgery and tissue catabolism in the postoperative period. The optimal protein requirements for critically ill patients in the absence of end stage renal/liver disease is 1.5-2.0 g/kg/day. Emphasis should be laid on the inclusion of foods rich in high biological value proteins such as eggs, milk, yoghurt, curd, cheese, flesh foods, legumes and pulses. Foods should be selected according to the age and pathophysiological condition of the patient. We must also keep a close watch on the liver and renal function tests while giving a high protein diet to terminally ill patients.
- **Immediate Preoperative Period :** Nothing should be given by mouth for at least 8 hours before a general surgery so that the stomach has no left over food at the time of surgery. In case of emergency surgery, gastric suction is used to remove the food if the patient has recently eaten a meal. This is important because food in the stomach may be vomited or aspirated during the surgery or during recovery soon after the surgery. It may also interfere with the surgery itself if it is related to the abdomen or gastrointestinal tract. In such cases a low residue or residue free diet is given several days before the surgery to clear the operative site. Elemental or chemically defined formulas are given either orally or through tube feeds.

B. Postoperative Nutritional Care

The therapeutic goal for the post-operative patient is rapid recovery to normal

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function and well being, minimum complications and early discharge. Nutrition plays an important role in minimizing the development of catabolism and returning the patient from the catabolic state to one of anabolism. As the nutrient losses due to catabolism during surgery are great, nutritional support is extremely important to aid recovery. In the post-operative period, nutrition support is used to reduce nutrition deficits that ordinarily develop in untreated patients during the period of NPO (nil orally) after surgery. We will now discuss the calorie and nutrient requirements during post-operative period.

Energy : Adequate calorie intake is critical for successful outcome of surgery and should be provided in the form of carbohydrates and fat. Energy requirements are generally high post operatively due to hyper-metabolism.

The energy requirement of the patient should be such as to be able to maintain an energy equilibrium or in other words stabilization of body weight at the pre-operative level. The energy requirement may be as high as 4000 to 6000 Kcal a day although such a high intake is usually not practically feasible.

Protein : Increased catabolism leads to protein deficiency and hence negative nitrogen balance which amounts to loss of tissue proteins which can be as high as 1 lb per day. In addition, plasma proteins are lost through haemorrhage, wound bleeding and exudates. Metabolic losses also result from tissue inflammation, infection, trauma, immobilization and poor calorie intake. The protein deficit may cause serious complications, especially if the patient has some degree of previous malnutrition. Adequate intake of protein, is therefore, required to replace losses and meet the increased demand for the following reasons:

Tissue Synthesis Wound Healing: Tissue proteins are synthesized only by Nutrition During Stress amino acids brought to the tissues by circulating blood. The necessary amino acids are provided either by ingested proteins or intravenous feeding. Concentrated liquid diets or commercial formulas may be used to overcome a poor appetite. The protein intake should be slowly increased according to the patients' tolerance.

Avoidance of Shock: Loss of plasma proteins lead to decrease in blood volume (hypovolemia) and lowered red blood cell volume causing a potential danger of shock. Adequate intake of protein is, therefore, required as protein deficiency enhances the danger.

Control of Oedema : Low levels of circulating proteins lead to oedema due to loss of colloidal osmotic pressure to maintain the normal fluid shift mechanism between the capillaries and the surrounding tissues prior to clinical oedema, considerable fluid collects in the interstitial spaces affecting heart and lung action. Local oedema at the site of surgery also delays closure of the wound and the healing process.

Bone Healing : Protein is essential for callus formation, calcification and bone healing especially in cases of orthopaedic surgery. The protein matrix is required for mineral deposition in bone tissue.

Resistance to Infection : Amino acids help to build the body's defense

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mechanisms like antibodies, blood cells, hormones and enzymes so as to prevent infection. Tissue integrity is the first line of defense against infection.

Lipid Transport: Proteins provide the transport mechanism for lipids by forming lipoproteins. This helps to prevent fatty infiltration and hence protection of the liver which is the main site of fat metabolism.

Protein Deficiencies: A depleted amino acid pool leads to poor wound healing (dehiscence), delayed healing of fractures, anaemia, depressed pulmonary and cardiac function, reduced resistance to infection, weight loss, liver damage and hence increased risk of mortality.

Carbohydrates: Carbohydrates ensure the use of protein for tissue synthesis and energy required for increased metabolic demands. With a high protein intake in extensive surgery or burns, the energy requirement may be as high as 4000 to 6000 Kilocalorie a day to spare proteins for tissue regain and prevent them from being diverted to provide energy. Carbohydrates also provide glycogen reserves to avoid liver damage. Since a high carbohydrate infusion can elicit impaired glucose tolerance, infusion of insulin through intravenous route is carried out.

If oral intake can be resumed, emphasis should be laid on the inclusion of foods rich in simple carbohydrates which are easy to digest and can be used to prepare calorie dense dishes which have a small volume and hence facilitate an enhanced food intake. Glucose is the preferred form of energy in case of enteral or parenteral tube feeding.

Fat: Adequate amount of fat is needed to build up and maintain tissue fat reserves. Depending upon the existing health and nutrition status of the patient, fat may be incorporated in the diet to provide 15% to 30% of the total energy intake to prevent the deficiency of essential fatty acids and to meet the increased energy requirements particularly in the presence of glucose intolerance. Emulsified fats and medium chain triglycerides are generally well tolerated in the presence of sluggish gastrointestinal function.

Fluid: Extensive fluid losses may occur through vomiting, haemorrhage, diuresis, exudate, fever and sweating after a surgery. An adequate fluid intake is, therefore, required to meet the increased needs. Fluids may be provided by intravenous therapy initially but oral intake should begin as soon as possible. The fluid intake should be sufficient to avoid dehydration on one hand and intoxication on the other.

Vitamins and Minerals: Vitamins are required for wound healing after a surgery. Vitamin C provides the cementing material of connective tissue, capillary walls and for building up of new tissues. Supplements of vitamin C are needed for aiding extensive tissue regeneration.

With increase in energy and protein intake, intake of B group vitamins must also be increased as they are the coenzyme factors for energy and protein metabolism.

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Vitamin K helps in the blood clotting mechanism and is, therefore, essential for preventing blood loss. Potassium and phosphorus are lost during tissue catabolism. Electrolytes, accompany fluid losses. Overloading of patient with fluids and electrolytes can affect gastric function. Restricting postoperative maintenance fluids to 2000 ml and NaCl to 77 mmol/day has been shown to enhance substantially gastric motility and speed up the recovery. Iron deficiency occurs from blood loss or due to faulty iron absorption. Mineral intake, therefore, should be adequate to replace losses and correct deficiencies.

Aggressive nutritional support is important to avoid postoperative complications and mortality. Routine postoperative intravenous fluids are given to meet hydration needs and provide electrolytes but cannot meet the high nutritional needs. Majority of general surgery patients, therefore, should progress to oral feedings as soon as possible to provide adequate nutrition. In case of major tissue damage or trauma, or when a patient is unable to take sufficient oral feeds, parenteral feeding must be done. A high amount of glucose, amino acids, electrolytes, minerals and vitamins have to be provided.

As soon as intestinal peristalsis returns, water and clear fluids such as tea, coffee and juice may be given to supply fluids and electrolytes. These fluids help stimulate normal gastrointestinal function and return to a normal, nutritionally balanced diet. With improvement, milk and milk products, pudding, cream soups and high protein beverages should be started. Progression to solid feeds in the form of soft or regular diets should be done with improved tolerance.

Having learnt about surgery, its nutritional implications and dietary management, we shall now proceed towards learning about another form of stress viz., burns. But let us first check our learning and understanding about surgery by answering the questions given in check your progress exercise

5.5.1 Classification of Burns

Burns can be classified on the basis of the extent, depth, patient age and associated illness or injury. On the basis of depth, burns are usually classified by degree. First degree burns or erythema, i.e., redness of the skin produced by coagulation of the capillaries with cell destruction above the basal layer of epidermis. First degree burns are not blistered. Second degree burns is erythema and is characterized by blistering with necrosis within the dermis. Third degree burns lead to total loss of skin including the fat layer, hair follicles and sweat glands.

First- and second-degree burns heal in days to weeks without scarring. Deep second- degree and small (less than 1 inch) third-degree burns take weeks to heal and usually cause scarring. Larger third-degree burns require skin grafting. Burns that involve more than 90% of the body surface, or more than 60% in an older person, usually are fatal.

First degree or partial thickness burns regenerate new skin tissue from the epithelial cells of the skin or hair follicles, sweat glands and sebaceous glands,

Second and third degree or full thickness burns do not have sufficient skin for healing and therefore extensive burns require skin grafting.

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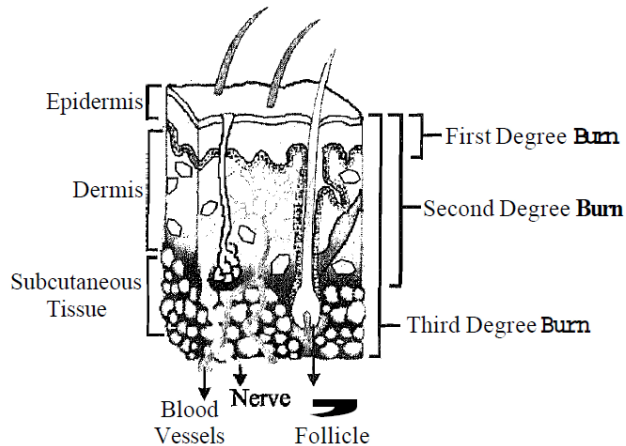


Figure 5.1: Diagrammatic representation- categorization according to depth of burns

You have often heard or read about description of an individual who has got burnt being ascribed as '40% burns', ' 90% burns' etc. What does this mean? This is a method of assessing the total body surface area that is burnt. It is often spoken of as "rule of nines". This information as you will learn later in this unit is utilized for computing the energy and other nutrient requirements of the patient. Data on age of patient can help in predicting the prognosis of a burn's injury. The most accurate rule of thumb for predicting mortality after severe burn injury is the Baux Score (age + percent burn, e.g. age 50 years + 20% burns = 50% mortality).

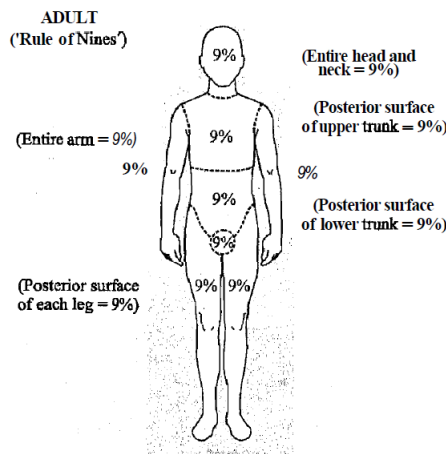


Figure 5.2: Diagrammatic assessment of the total body surface area burnt

5.5.2 Complications of Burns

Most minor burns are superficial and do not cause complications. However, deep second-degree and third-degree burns swell and take more time to heal. In addition, deeper burns can form a scar tissue. This scar tissue shrinks (contracts) as it heals. If the scarring occurs at a joint, the resulting contracture may restrict

movement.

Severe burns can result in serious complications due to extensive fluid loss and tissue damage. Complications from severe burns may take hours to develop. The longer the complication is present, the more severe are the problems it tends to cause. Young children and older adults tend to be more seriously affected by complications than other age groups.

Dehydration eventually develops in people with widespread burns, because fluid seeps from the blood to the burned tissues. Shock develops if dehydration is severe. Destruction of muscle tissue occurs in deep third-degree burns. The muscle tissue releases myoglobin, one of the muscle's proteins, into the blood. If present in high concentrations, myoglobin harms the kidneys.

Thick, crusty surfaces (eschars) are produced by deep third-degree burns. Eschars (a dry scab) can become too tight, cutting off blood supply to healthy tissues or impairing breathing.

Treatment of burned patient is incomplete and often unsuccessful if proper nutritional care is not provided. Through our subsequent discussions we will learn about the dietary management of patients suffering from burns.

5.5.3 Dietary Management for Burns

Nutritional support is a major part of therapy for a patient with burns in view of the large catabolic losses, essential anabolic demands and to meet personal support needs.

Nutritional care plan and outcome is dependent on factors, like

- Age — Elderly people, very young children pregnant women and lactating mothers are highly vulnerable.
- Health condition — Presence of diabetes, cardiovascular or renal disease complicates the care process.
- Severity of burns — The location and severity of the burns and time lapse before treatment.

Nutritional support needs are calculated on the basis of body surface area burnt. Second and third degree burns covering 15-20% or more of body surface, or even 10% in children and elderly persons cause extensive fluid loss and therefore require intravenous fluid therapy. Severe burns covering more than 50% of the body surface area are often fatal. Nutritional care for a patient in the burns is adjusted to individual needs and is given in three stages:

Ebb or shock period: During the initial burns after injury, the focus is on counteracting the stress induced neurohormonal and physiologic responses that accelerate the body's metabolism by a series of events. Loss of skin on the burn site and exposure of extra cellular fluids lead to immediate loss of water and

electrolytes, mainly sodium and also protein depletion. As a result, the body water shifts from extra cellular spaces in other parts of the body to the burn site adding to continuous loss of fluids and electrolytes.

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Due to this there are changes in the capillary fluid shift mechanism resulting in decreased volume hypotension, low haemo-concentration and diminished urine output. Intracellular water is also drawn out to balance extra cellular fluid losses leading to cellular dehydration. Patients with extensive burns need immediate fluid and electrolyte replacement during the first 12 to 24 hours after injury. A balanced salt solution such as lactated Ringer's solution is given to correct hypovolemia and prevent metabolic acidosis. Because the exact volume of fluid and infusion rate depend on the patient's response to fluid delivery, ongoing fluid replacement is based on close monitoring of the patient. The goal is to maintain an adequate blood pressure and haematocrit and a urine output of > 50 to 100 ml/hr (0.5 to 1 ml/kg/hr) in an adult or 1 ml/kg/hr in a child while avoiding circulatory overload. A general formula for the first 24 hr is 0.5 ml/kg/% Body Surface Area (BSA) of colloid and ml/kg/hr BSA of lactated Ringer's solution along with 100 mL/hr maintenance of lactated Ringer's solution. One fourth of the fluid is given in the first 4 hr, 114 in the second 4 hr, 114 in the next 8 hr, and 114 in the last 8 hr— measured from the time of injury, not from the time of arrival at the emergency facility, because large amounts of intravascular fluid can move into tissues, leading to shock, which begins immediately after injury. A colloidal solution such as albumin or plasma is not effective at this stage because it passes into the extra vascular fluids due to the increased permeability of the vascular endothelium caused by the burn. Usually, vascular permeability returns to normal after the first day and colloidal solutions are then given to restore plasma volume. During this initial period, nutritional requirements of protein and energy are not attempted to be met as the entire focus is on rapid and effective fluid and electrolyte therapy so as to prevent shock.

Flow or Recovery Period : After 48 to 72 hours, fluids and electrolytes are reabsorbed into the general circulation and excess fluid is excreted. fluid balance is gradually reestablished and massive tissue loss is reversed. Fluid intake and output must be checked to prevent dehydration or over hydration. The patient usually returns to preinjury weight by the end of first week and adequate bowel function returns. At this time, increased nutritional needs must be met for the following reasons:

- Replace losses of protein and electrolytes due to burn injury.
- Replace lean body mass due to extensive tissue breakdown.
- Meet increased metabolic needs for energy due to sepsis or Fever, increasing the need for carbohydrates and B vitamins. Minerals and vitamins are also needed for tissue regeneration.
- Providing healthy tissue for subsequent successful skin grafting.

Anabolic Phase: During this period the patient is well hydrated and the reactions due to metabolic stress are under control. The patient may still be hypermetabolic and has depleted reserves of all nutrients. Rigorous nutrition support during this

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period is essential to promote fast recovery and proper rehabilitation. Proper nutritional care during this period can help in preparing patient physically For undergoing successful skin grafting/ any other surgery. Transplantation of organs or body parts is done much latter during the anabolic phase.

The nutrient requirement and dietary management during the flow and anabolic phase is discussed next.

- **Nutrient requirement and dietary management during the flow and anabolic phase**

The patient usually is well hydrated and has body weight close to the pre-injury weight. The return of bowel movements is an indication that the patient can now be slowly introduced to nutrition support in the form of oral intake, enteral feeds or parenteral nutrition. A combination of any two alterations in these routes is required on a day-to-day basis depending upon the changing clinical parameters and the severity of burns. Dietary management should be such as to fulfill the following objectives:

- providing enough calories to prevent subsequent weight loss
- maintaining fluid and electrolyte balance
- minimizing stress response to pain and anxiety
- maintaining a positive nitrogen balance
- replenishing the depleted reserves of vitamins and minerals
- preventing curling's ulcers, ileus and other complications

As always we shall first discuss about the energy requirements of the patient during the flow and anabolic phase. Thereafter we shall learn about the contribution of various macronutrients for providing the required amount of calories.

Energy: The energy needs of the burned patient vary according to the depth and size of the burn. The requirements of course would be highest in third degree burns; Although several formulas have been developed to determine the energy needs; Currie formula is most commonly utilized and is mentioned below:

It must be evident from these formulas that the energy requirement of burnt patients are much higher than those for their healthy counterparts.

Let us now learn about the contribution towards the total energy through various macronutrients viz; carbohydrates, proteins and fat.

Carbohydrates: Liberal amounts of carbohydrates should be given i.e. around 60% to 65% of the total energy. However, care must be taken regarding the maximum rate of administration feasible keeping in mind the fact that the maximum tolerance level is about 7 mg/kg/min above which glucose is not oxidized to release energy but is converted to fat. Blood glucose levels should be closely monitored to prevent hyperglycemia and its associated complications such as dehydration, coma, respiratory problems etc. During the anabolic phase when the patient can

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eat orally and has normal defecation process, a combination of simple and complex carbohydrates may be given. Providing good amounts of foods rich in mono and di saccharides, as well as, starches help in preparing meals which are nutrient dense, have small volume and are easy to digest. Thus, rice, refined wheat flour, semolina, sago, arrowroot, rice, rice flakes, murmura, pastas, dextrose, glucose, honey, potato should be incorporated liberally in the diet,

Fat: Enteral and parenteral feedings; administration of lipids should be carried out carefully in all critically ill patients. A careful monitoring of immune function, feeding tolerance and serum triglycerides required during lipid administration. Most of the patients are able to tolerate around 12-15% of the total calories in the form of lipids. Structured lipids and medium chain triglycerides are currently being preferred. A low fat diet is preferred during the initial phases of recovery in view of its association with improved respiratory function, reduced incidence of pneumonia, faster recovery. During the later phases of anabolism (near discharge) the fat intake may be normalized. In view of the impaired gastro-intestinal function among many patients it is advisable to lay emphasis on foods rich in emulsified fat and medium chain triglycerides (MCT's).

Protein: It is one of the most crucial nutrient which determines the ultimate outcome of burns. Amino acid requirements are high due to increased losses through wounds and urine, increased requirement for promoting synthesis of blood proteins and wounds. Fluid loss from a burn wound may be considerable and can contain 4-6 g protein/100 ml, representing 25-50% of total nitrogen loss. Nitrogen losses via faeces have been estimated to be around 1-3g N/day. Thus, adult patients should be given 20-25% of the increased energy from protein. Among children the requirements are still higher i.e. 2.5 to 3.0 g per kg usual body weight per day. Protein intake beyond this level is not recommended in view of the increased burden on the kidneys.

Blood urea nitrogen, serum creatinine and level of hydration must be monitored carefully. The protein intake may need to be curtailed if the burnt area has involved the kidney / excretory system. High biological value protein food sources such as eggs, milk, cheese, yogurt, marine food, meat, poultry, legumes and pulses should be included liberally but in an easy to digest form. Hospital based tube feeds may be prepared by using soya milk, milk proteins (casein, whey protein, lactalbumin), eggs and flour of pulses particularly soyabean.

Other considerations

- oral intake is generally feasible only during the anabolic phase wherein the patient should be given a high-energy, high protein, micronutrient rich diet. The diet should have a small volume, it should be nutrient dense and easy to digest. A two hourly feeding schedule or a 6-7 meals/ day pattern should be adopted.
- Oral feeding may be supplemented with enteral nutrition to be able to replenish the depleted nutrient reserves,

- Most of the patients are depressed and may need encouragement by family and medical team to promote adequate food consumption.
- Anabolic steroids such as oxandrolone are often combined with a high protein diet to promote weight gain and enhance anabolism at the site of wound

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5.5.4 Mode of Feeding/ Nutrition Support

These two methods form the common nutrition support strategy for burns patients, Let us review these methods in the context of burn patients.

Oral feeding is desirable if tolerated by the patient. Concentrated oral liquids with protein hydrolysates or amino acids must be given to ensure adequate intake. Solid food should be gradually introduced according to food preferences. Support and encouragement help the patient to eat better. Food should be attractive and appetizing and individual like and dislikes must be considered.

Enteral nutrition can be utilized judiciously alone or in combination with other forms of feeding during various stages and purposes during the course of treatment for instance some patients may initially require tube feeding, low bulk defined formula solutions may be given. Commercial high protein formulas may also be used.

Parenteral Feeding is required for some patients to provide extra nutritional support if oral intake and tube feedings are inadequate to meet the high nutritional needs. This form of feeding is more commonly used during the ebb and flow phase.

Continuous nutritional support is important to maintain tissue integrity for successful skin grafting or plastic reconstructive surgery. Persistent supportive care — medical, nutritional and nursing helps the patient to cope with the stress situation.

5.5.5 Non-Dietary Treatment of Burns

While good nutritional care should be provided to the patient as soon as feasible it is equally imperative and at times critical to provide efficient and appropriate physical care to the patient to minimize trauma, pain and ensure re-habilitation. Some important aspects of non-dietary treatment are being briefed below.

The most immediate step is removal of the burning agent from the patient to prevent further damage. For example, fires are extinguished. Clothing — especially any that is smoldering (such as melted synthetic shirts), covered with hot tar, or soaked with chemicals should be immediately removed.

Hospitalization is sometimes necessary for optimal care of injuries. For example, elevating a severely burned arm or leg above the level of the heart to prevent swelling is more easily accommodated in a hospital. In addition, burns that prevent a person from performing essential daily functions, such as walking or eating, make hospitalization necessary. Severe burns, deep second- and third-degree

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bums, burns occurring in the very young or the very old, and burns involving the hands, feet, face, or genitals are usually best treated at burn centers. Burn centers are hospitals or wards in a hospital that are specially equipped and staffed to care for burn victims.

Superficial Minor Burns: Superficial minor burns are immersed immediately in cool water if possible. The burn is carefully cleaned to prevent infection. If dirt is deeply embedded, a doctor can give analgesics or numb the area by injecting a local anesthetic and then scrub the burn with a brush. Often, the only treatment required is application of an antibiotic cream, such as silver sulfadiazine which prevents infection and forms a seal to prevent further bacteria from entering the wound. A sterile bandage is then applied to protect the burned area from dirt and further injury. A tetanus vaccination is given if needed. Care at home includes keeping the burn clean to prevent infection. The burn can be covered with a nonstick bandage or with sterile gauze. The gauze can be removed without sticking by first being soaked in water.

Severe Burns: Severe, life-threatening burns require immediate care. Dehydration is treated with large amounts of fluids given intravenously. A person who has gone into shock as a result of dehydration is also given oxygen through a face mask. Destruction of muscle tissue is also treated with large amounts of fluids given intravenously. The fluids dilute the myoglobin in the blood, preventing extensive damage to the kidneys. Sometimes a chemical, sodium bicarbonate, is given intravenously to help dissolve myoglobin and thus also prevent further damage to the kidneys.

Eschars that cut off blood supply to an extremity or that impair breathing are cut open in a surgical procedure called escharotomy. Escharotomy usually causes some bleeding, but because the burn causing the eschar has destroyed the nerve endings in the skin, there is little pain.

Keeping the burned area clean is important, because the damaged skin is easily infected. Cleaning may be accomplished by gently running water over the burns periodically. Wounds are cleaned and bandages are usually changed 1 to 3 times per day.

5.6 TRAUMA

The term "trauma" comes from a Greek word which means "a wound" (and or damage or defect). Trauma is a form of shock to the human body which may occur in the form of crush injuries, diving/ air compression or specific wounds on body part/ organs such as head/spinal cord. Accidental or crush injury — a form of acute trauma is a major cause of death and disability. Crush injuries generally result from serious road accidents, industrial mishaps, explosions etc.

It may involve several fractured bones, profuse multiple external bleeding,

internal bleeding, shock and deterioration into unconsciousness. Optimal care of the injured patient is often intensive and prolonged. Survival rate is low and may be followed by years of rehabilitation. Metabolic and nutritional support of the injured patient is a major component of overall care.

5.6.1 Physiological Response to Injury

The physiological events are related to the severity of injury that is, greater the insult, the more pronounced is the response. Two distinct periods of post-traumatic responses have been identified:

Early ebb or shock phase: This is usually brief in duration lasting 12 to 24 hours and occurs immediately following injury. Blood pressure, cardiac output, body temperature and oxygen consumption are reduced. These are often associated with haemorrhage and result in hypo perfusion and lactic acidosis. As the blood volume is restored, more accelerated responses occur.

Flow Phase: It is characterized by hyper metabolism, increased cardiac output, increased urinary nitrogen losses, altered glucose metabolism and accelerated tissue catabolism. These flow phase responses to injury are similar to those following surgery but are usually more intensive and extend over a longer period of time. This phase is characterized by hyper metabolism and alterations in the metabolism of glucose, protein and fat.

5.6.2 Metabolic Response to Injury

There is an increase in the basal metabolic rate above the normal. The degree of hyper metabolism is related to the severity of the injury. Long-time fracture usually result in a 15 to 25 % increase in metabolic rate, multiple injuries increase metabolic rates by 50% and severe burn patients have metabolic rates raised by 100%. The body temperature of a trauma patient rises by 1-2°C due to an upward shift in the thermoregulatory set point of the brain. The changes in glucose, protein and fat metabolism are being discussed below.

- **Altered Glucose Metabolism:** Following injury, hypoglycemia commonly occurs and is related to the severity of the stress. In the ebb phase, insulin levels are low and glucose production is slightly elevated. During the flow phase, hyperglycemia persists although insulin levels are normal or even high. Hepatic glucose production and gluconeogenesis are increased.
- **Altered Protein Metabolism:** Urinary nitrogen loss is extensive following injury. Trauma accelerates nitrogen turnover. In unfed patients, tissue breakdown rates exceed synthesis and a negative balance occurs. Providing exogenous calories and increase in nitrogen synthesis and thus helps in restoring the nitrogen balance.
- **Altered Fat Metabolism:** The stored fat deposits are mobilized and oxidized at a high rate in order to support hyper metabolism and increased gluconeogenesis. Severely injured patients, if unfed, deplete their fat and protein stores rapidly.

The resulting malnutrition increases their susceptibility to haemorrhage, infections, organ system failure, sepsis and death.

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5.6.3 Hormonal Responses to Injury

A number of hormonal changes take place in patients following injury. There is a marked rise in the counter regulatory hormones, viz., glucagon, glucocorticoids and catecholamine. Glucagon has glycogenolytic and gluconeogenic effects the liver, Cortical mobilizes amino acids from skeletal muscle, increases hepatic gluconeogenesis and maintains body fat stores. The catecholamine also stimulates hepatic gluconeogenesis and glycolysis and increase lactate production from skeletal muscles. They also increase metabolic rate and lipolysis. Growth hormone is elevated while thyroid levels are reduced.

Now that we are familiar with the physiological, metabolic and hormonal changes, specific to trauma, you will find yourself better equipped to understand dietary management of this stress response. Since the dietary considerations/requirements are quite similar to that in the post operative period in surgery. The dietary management of trauma1 injury dealt here in this section is brief.

5.6.4 Dietary Management— Trauma

As a result of metabolic responses to injury, there is an increase in the energy expenditure. Oxidation of body fat stores takes place causing loss of weight. Most injured patients can tolerate a loss of 10% of their pre-injury body weight prior to injury. If weight loss exceeds 10% body weight, under nutrition increases the morbidity and mortality rates. The patients are exposed to a variety of infectious agents in the hospital., due to use of catheters and nasogastric tubes. Under nutrition increases the likelihood of sepsis, multiple organ system failure and death. It also delays wound healing.

The purpose of nutrition support for a trauma patient is to aid in the defense mechanisms of the body. Adequate nutrition allows normal responses that optimize wound healing and recovery. Nutritional support should be provided before significant weight loss occurs. Intravenous administration of hypertonic nutrient solutions, of peripheral vein feedings with fat emulsions and use of specific diets provide effective nutrition support to injured patients.

Nutrient Requirements during Trauma

Nutritional assessment of the trauma patient is done to determine energy and protein requirements. Basal energy requirements are determined from standard tables based on age, sex and body surface area. These requirements are adjusted for increase in metabolic rate due to injury or disease. Dietary protein is required in greater amounts to achieve nitrogen balance. Approximately, 15 to 20% of calorie intake should be from protein.

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Carbohydrates (glucose) should provide 60% of caloric needs and the rest of energy needs should be met by fat. Multivitamins are given daily along with supplements of vitamin C, which is required in increased amounts after injury. Electrolytes may be added to feed formulas so as to maintain normal serum levels. Potassium, magnesium and phosphate supplements are added to parenteral fluids. Zinc supplements should be given to severely malnourished patients or those with a history of poor nutrient intake, e.g. alcoholics.

The routes of nutrition support are oral, enteral and parenteral. Oral and enteral routes are generally preferred over the parenteral (intravenous) administration. Oral liquid supplements should be administered to increase the nutrient intake. The patient's injuries may interfere with oral feedings. Patients with facial and head injuries, disorders of the jaw, mouth or oesophagus and those receiving artificial ventilation are not able to take feeds orally.

Such patients have to be fed by use of tubes. Enteral or parenteral tube feed formulas are usually balanced mixtures of fat, carbohydrate and protein. Intravenous or parenteral feedings may be necessary to supplement enteral feedings or when enteral feeds cannot be tolerated. With nutritional requirements, we end our study on trauma. Next, let us get to know about sepsis-a complication which may arise on its own or as a consequence of any long-term stress.

5.7 SEPSIS

Sepsis is defined as the presence of an infection due to an identifiable organism. Bacteria and their toxins lead to a strong inflammatory response. Viruses, fungi and parasites also cause infection and inflammation. The Systemic Inflammatory Response Syndrome (SIRS) is the term used to describe the inflammation that occurs in infections, burns, multiple trauma, shock and organ injury.

The inflammation is usually present in areas much away from the primary site of injury and affects healthy tissues. The association between the terms sepsis and SIRS can be better understood by the diagnostic criteria. SIRS commonly leads to development of Multiple Organ Dysfunction Syndrome (MODS). It generally begins with lung failure followed by failure of the liver, intestines and kidney.

Multiple hypothesis have been proposed to explain the development of SIRS or MODS. The progression of SIRS to MODS appears to be mediated by excessive production of pro-inflammatory cytokines and other mediators of inflammation. According to the "gut hypothesis" disruption of the gut barrier function results in translocation of enteric bacteria into the mesentery lymph nodes, liver and other organs.

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Box 5.1	Diagnostic Criteria for Sepsis/SIRS
<p>Sepsis can be diagnosed if infection is proven by means of a positive blood culture and two or more of the following:</p> <ul style="list-style-type: none">● Heart rate > 90 beats per minute● Body temperature < 36 (98.6°F) or > 38°C (100.4°F)● Hyperventilation (high respiratory rate) > 20 breaths per minute or, on blood gas, a PaCO₂ less than 32 mm Hg● White blood cell count < 4000 cells/mm³ or > 12000 cells/mm³ (< 4 × 10⁹ or > 12 × 10⁹ cells/L), or greater than 10% band forms (immature white blood cells). <p>When two or more of these clinical parameters are met without confirmation of infection it is called systemic inflammatory response syndrome.</p>	

Despite a number of advances in the treatment of infections and a better understanding of its path physiology, the mortality and morbidity rates from septicemia are high. Unlike elective surgery and trauma, the response patterns following major infection are unpredictable. The variability in metabolic and physiological response is relied partly to the patient's age, previous health status, preexisting disease, previous stress, site of infection and the infective agent. Moreover, the organ system failure may mask the manifestation of systemic infection. Based on cardiac output, two physiological responses have been described.

The first is characterized by an increased cardiac output and high systemic perfusion. The second response is characterized by cardiac decomposition, inadequate tissue perfusion and acidosis, and is described as low flow sepsis. Both these responses reflect the body's reaction to systemic infection and are modified by the underlying disease and physiologic reserves of the patient. The invasion of the body by infective agents initiates host responses. There is mobilization of phagocytes and inflammation at the local site. As the infection progresses, fever, tachycardia and other responses occur,

5.7.1 Systemic Metabolic Responses

Many of the metabolic responses to infection are similar to those following Injury. The key changes include:

- **Hyper metabolism:** Oxygen consumption is elevated in the infected patient. It may be 50-60% higher than normal and is related to the severity of the infection (PaCO₂ of < 32 mmHg-hyperventilation). In the pre-operative and post injury

period, such a response often occurs secondary to severe pneumonia, abdominal infection or wound infection. Increased metabolism is related to fever—being 10-13% for every 1°C elevation in temperature. The metabolic rate returns to normal as the infection resolves.

- **Altered Glucose Metabolism:** Blood glucose levels are generally elevated in the infected patient but plasma insulin levels are normal or even higher in previously healthy patients who develop infection. Increased glucose production in infected patients is in addition to the increased gluconeogenesis following injury. Glucose metabolism following infection is, however, complex as hypoglycemia and diminished hepatic glucose production has also been seen in patients.
- **Altered Protein Metabolism:** There is increased proteolysis and nitrogen excretion resulting in negative nitrogen balance following an infection. Amino acid flow from skeletal muscle is accelerated in patients with sepsis.
- **Altered Fat Metabolism:** Fat is the major fuel oxidized in infected patients. If nutrition support is inadequate, the peripheral fat stores are mobilized. Increase in the sympathetic nervous system activity mediates the increase in lipolysis.
- **Changes in Trace Minerals:** Changes in the balance of magnesium, phosphate, zinc and potassium follows alterations in nitrogen balance. Iron and zinc level in the blood are decreased. This is not only due to body losses of these minerals but due to accumulation of these within the liver as a part of the lost defense mechanism. We will now move on to the catabolic responses to sepsis

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5.7.2 Catabolic Responses

Hormonal responses during the hypermetabolic phase of infection are same as in case of injury. Serum cortisol levels are elevated, glycogen is incurred and insulin levels may be normal or higher. The levels of catecholamine, growth hormone, antidiuretic hormone (ADH) and aldosterone are also elevated. The growth hormone level remains elevated during convalescence, to promote anabolism.

Interleukin-1 is an endogenously produced pyrogen which produces fever and has direct effects on the liver; it promotes hepatic repletion of zinc and iron, increases plasma copper levels and stimulates hepatic synthesis of plasma amino acids.

The metabolic and hormonal changes discussed above can result in reversible or irreversible alteration in the structure and/or function of one or more organs over a period of time. This is often referred to as multiple system organ failure and is being discussed below.

Multiple Organ Dysfunction Syndrome (MODS)

Failure of essential organs is the most severe complication of sepsis and may result in death. The treatment of systemic infection, therefore, consists of use of antibiotics, support of cardiovascular and respiratory function, supportive

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therapy of specific organs and vigorous nutrition support. Septic shock may lead to a decrease in peripheral resistance and cause pulmonary insufficiency. Patients often require ventilator support. Inadequate cardiac output may lead to impairment and malfunction of the kidney. The resulting uremia superimposed on the sepsis further impairs the hyper catabolic infected host. Sepsis causes marked changes in the structure/ function of gastrointestinal tract and may lead to stress ulcers and bleeding. Septicemia also commonly leads to hepatic dysfunction causing jaundice, hyperbilirubinemia and liver failure. Multi-system organ failure or MODS is associated with a high incidence of death.

5.7.3 Dietary Management of Sepsis with or without MODS

Before we begin with the dietary management of patients suffering from sepsis with or without MODS let us read a case below.

Mr. Sunder a 71 years old man was admitted to the ICU of a multi-speciality hospital suffering from moderate urinary tract infection and difficulty in breathing due to aspiration pneumonia. He was immediately put on ventilator to facilitate breathing. His medical history indicated that he was an old case of non-insulin dependent diabetes mellitus.

Due to aspiration pneumonia and resultant intubations; the patient had to be fed through external tube feeds. Presence of infection however exacerbated hyperglycemia and there was a marked reduction in WBC count. Gradually, the patient had to be fed through the parenteral route. Due to persistent infection there was considerable wasting and under-nourishment. On one hand the infection entered the blood stream and affected other organs whereas on the other hand feeding through parenteral route resulted in atrophy of the small intestine. The patient ultimately expired after three months due to the septic shock, renal failure and diabetic coma.

This is an example of the most common pathways which develop in critically ill patients. Keeping these complications in mind let us briefly discuss the nutrient requirements and the various forms of nutritional support which can be provided to such patients.

Dietary Management of and MODS

Patients suffering from sepsis and/or resultant multiple system organ dysfunction are critically ill and admitted in the intensive care unit of the hospital. usually have an impaired immune function and compromised cardiopulmonary functional capacity. Such patients may also have reduced functional and regulatory capacities of renal and/or gastrointestinal tract and impaired immune function along with compromised cardiopulmonary function capacity. They generally have altered blood/ urine indices (abnormal serum albumin) and are hypermetabolic. The Urine Urea Nitrogen (UUN) excretion in grams per day has been used to evaluate the degree of hypermetabolism. The UUN can be used to interpret the level of

hypermetabolism as follows:

Urine Urea Nitrogen

≤ 5 gm / 24 hrs. = No stress

5 to 10 gm / 24 hr = mild hypermetabolism or level 1 stress

10 to 15 gm / 24 hrs = moderate hypermetabolism or level 2 stress

< 15 gm / 24 hrs = severe hypermetabolism or level 3 stress

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Meeting the nutritional requirements of such patients can be a challenging issue as they suffer from not one but several metabolic/physiological abnormalities. For example, a diabetic patient may be suffering from urinary tract infection and end-stage renal disease wherein; the dietary management of one may be contradictory for the other form of illness. Further, these patients may be on life-support system (such as ventilator, catheters, dialysis) and oral intake may not be feasible. Multiple abnormalities may appear in the metabolism of energy, protein, carbohydrates, fat and several vitamins/minerals.

While meeting the nutrient requirements may not be always feasible; our endeavour should be to help the patient in maintaining a good nutritional status and prevent the progression of the disease. It is important to remember here that the nutritional care process undergoes several modifications over a small period of time and may require immediate implementation. However, the major/broad objectives of nutritional care are:

- to minimize the development of nutrient imbalance.
- to maintain fluid and electrolyte homeostasis
- to promote energy equilibrium
- to help in achieving and maintaining normal/ safe levels of all macro - and micro-nutrients.

The nutrition care plan for meeting the above mentioned objectives can come into play only when the patient is haemodynamically stable. We shall now proceed towards learning about the dietary management during sepsis/MODS. It is important to note that over-enthusiastic feeding of the patient would only worsen his disease condition. Patients suffering from sepsis and / or MODS should not be expected to gain weight/ body mass or strength until the source of hypermetabolism is treated.

Energy: Patients suffering from septicemia with or without MODS are generally hypermetabolic which results in weight loss. Critically ill patients are generally able to tolerate around 25-30 per kg usual body weight. Although adequate energy is essential for metabolically stressed patients excess calories intake may elicit complications such as hyperglycemia, excess carbon-dioxide production, which can exacerbate respiratory insufficiency or prolong weaning from mechanical

ventilator. Whatever may be the amount of calories given to the patient, our objective should be to maintain blood glucose levels ≤ 100 mg/dl, if required by the help of insulin.

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Proper choice of enteral/ parenteral tube feeds along with insulin infusions is advocated. A combination of two or three types of feed formulas may be required to meet the individualistic requirements of a patient. However, in isolated cases if oral intake is feasible; it is usually in the form of full-fluid/bemi-liquid diets (mild sepsis/ MODS).

- **Protein:** Adequate amount of protein is required by these patients to improve immunity against infections, promote recovery, spare lean body mass and reduce the amount of endogenous protein catabolism for gluconeogenesis. The requirements may vary from 0.8 gm to 20 gm per kg usual body weight per day. During mild sepsis with adequate organ function, the protein intake can be maintained at 0.8 gm/kg usual body weight per day. Intact protein or protein rich foods can be included in the form of enteral tube feeds or as liquid or semi-soft diets. However, if the patient is having complications particularly of liver or kidneys, it is advisable to give specific amino-acids according to the underlying disease condition.
- **Carbohydrates and Fat:** Carbohydrates should constitute nearly 60% to 70% of the total energy. Glucose is the primary calorie substrate in a parenteral, nutrition formulation. Parenteral nutrition should be initiated with a low dextrose infusion rate. Fats may provide 15% to 40% of the total calories depending on the underlying complications. Fat helps in preventing the deficiency in the presence of hyperglycemia. However, intravenous fat emulsions may create problems in patients having severe infection, liver or gall bladder disorders.
- **Micro-nutrients:** The requirement of almost all vitamins and certain mineral increases due to infection and inflammation. In the absence of underlying complications adequate intake of all minerals and trace-elements like iron, calcium, zinc, sodium, potassium and magnesium is suggested. However, if the patient is suffering from complications of liver, kidney or oedema then the sodium and potassium intake should be regulated. Liberal amounts of foods rich in B-group vitamins, vitamin A and C should be included in the diet. Adequate amount of fluids should also be provided to prevent complications arising due to dehydration or hypovolemia.

Other Feeding Considerations/Nutrition Support

The preferred route for feeding the patient should be oral intake/ via the utilization of gastrointestinal tract. If oral intake is feasible, natural foods may be given in the form of semi-soft/ full-fluid diets. However, if oral intake is not possible then should opt for enteral feedings which can be prepared from natural foods (absence of MODS/ complications) Commercially available foods (intact, hydrolyzed or semi-hydrolyzed formulas) parenteral nutrition should be provided if other forms of feeding can not be provided.

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From the above discussion it must be clear to you that the dietary management of septic patients, especially those suffering from MODS is complex and needs to be altered after every few hours depending on the clinical parameters which are analyzed atleast 24 hourly.

In this section we learnt about sepsis and MODS which are among the most critical and life threatening conditions for human beings. Let us attempt the check your progress exercise 3 to recapitulate the concepts learnt so far.

5.8 LET US SUM UP

In this unit we studied about the physiological and metabolic consequences of stress in its various forms viz. surgery, burns, injuries, sepsis and multiple organ dysfunction syndrome. We learnt that stress is a psycho-physiological response to a non-conductive environment within or outside the body which results in excessive or inappropriate activation of the body's defense mechanism.

In the first section we briefed ourselves regarding the stress response in the form of ebb and flow phase which is followed by the anabolic phase. The ebb phase is the most critical period with respect to survival of the patient. Efficient and prompt emergency treatment (first-aid) during this stage can help in reducing the incidence of mortality to a great extent. The flow phase which develops thereafter is characterized by elevated O₂ requirements, increased cardiac output, and marked catabolism. The dietician plays an important role in providing judicious and prompt care to manage the fluctuations in the nutrition status during the three phases.

The second section discussed about elective and emergency surgeries. The importance of a pro-active approach both pre - and post-operatively was discussed with regards to nutritional care.

We also learnt about a critical form of stress viz. burns which can be described as injuries to the tissues due to heat, electricity, radiation or chemicals. A briefing on classification of burns as per the common methods employed in the hospitals (rule nines, degree/depth of burn etc.) was followed by overall treatment of superficial/ severe burns. The importance of adequate resuscitation during the ebb/ shock period was also explained. The nutrient requirements and mode of feeding during the flow and anabolic phase is also critical for ensuring proper treatment and rehabilitation the patient.

This unit finally dealt with the most critical forms of illness viz. trauma related to injuries due to cold, radiations, altitude, accidents etc. Sepsis may result on its own as a consequence to surgery, burns, injuries etc. Sepsis can result in multiple organ dysfunction syndrome which is often difficult to handle as it involves structural functional changes in not one but several organs.

Such patients are usually referred to as those suffering from terminal illness and their nutritional care generally involves utilization of specialized formula foods through enteral or parenteral route.

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5.9 GLOSSARY

Adreno-corticoid	: a hormone secreted by the adrenal cortex.
Cortisol	: a gluco-corticoid produced by the adrenal cortex.
Homeostasis	: A balanced, normal state of the body's metabolic and physiological functioning.
Hypermetabolism	: metabolism at an increased or excessive rate.
Hypovolemia	: decrease in volume of blood.
Hypoxia	: lack of oxygen.
Sepsis	: a systematic response typically to a serious usually localized infection (abdomen/lungs) especially of bacterial origin.
SIRS	: a severe systemic response to a condition that provokes an acute inflammatory reaction.

5.10 CHECK YOUR PROGRESS

- 1). What is the difference between the ebb and flow phase in a stress response?
- 2). Explain different types of surgeries?
- 3). Enlist any five physiological consequences of a surgery?
- 4). Discuss the importance of protein intake in postoperative nutritional care.
- 5). What is the significance of protein in the diet of burn patients during the anabolic phase?
- 6). What are the benefits of vitamin A and C for promoting recovery during anabolic phase?

NUTRITIONAL MANAGEMENT OF FOOD ALLERGIES AND FOOD INTOLERANCE

STRUCTURE

- 6.1 Learning Objective
- 6.2 Introduction
- 6.3 Adverse Food Reactions
- 6.4 Adverse Food Reactions-The Diagnosis Process
- 6.5 Treatment and Management of Adverse Food Reactions
- 6.6 Prevention of Adverse Food Reactions
- 6.7 Let Us Sum Up
- 6.8 Glossary
- 6.9 Check Your Progress

6.1 LEARNING OBJECTIVE

After studying this unit you will be able to:

- classify adverse food reactions,
- differentiate between food allergies and food intolerance,
- discuss the etiology, clinical manifestation, metabolic aberrations and complications, linked with adverse food reactions,
- explain the diagnosis of adverse food reactions, and
- describe the dietary management of patients with food allergies and food intolerance.

6.2 INTRODUCTION

In this unit we will be discussing the adverse reactions associated with food. You may have read or heard of cases, wherein an individual after consuming specific foods have reported immediate and often dramatic physical reactions such as vomiting, diarrhoea, cramps, wheezing, swelling of the airways, a severe drop in blood pressure etc. On the other hand, you may have also come across individuals,

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who cannot tolerate particular foods say milk, wheat products etc. For example, people with lactose intolerance cannot digest the sugar lactose, in milk. They develop gas, bloating, and abdominal pain when they consume milk products. These are, in fact, different conditions, which link food to adverse reactions. Food allergies and food intolerance can cause much more than annoying gastrointestinal symptoms. And in some cases they cause no obvious symptoms until a chronic disease shows up later in life. Therefore it is important to detect and treat food allergies and food intolerance as early as possible. In this unit we will learn about different types of adverse food reactions — their cause, effects and the dietary management.

6.3 ADVERSE FOOD REACTIONS

What do we mean by adverse food reactions? We shall try to understand this concept with the help of a few case studies.

Case 1 : eight year old boy presented with hyperactivity, and disruptive behaviour at school. He was very moody and suffered from chronic rhinitis and frequent colds. This child had always been difficult and irritable. As an infant on a cow's milk formula, he suffered colic, sleeplessness, and screaming fits.

Case 2: A 27-year old IT Consultant who lives in Bangalore and who suffers with loose stools, gas, irritable bowel syndrome, inability to gain weight, acne on back, poor endurance, and hypoglycemia/need to eat every 2-3 hours. Allergy testing demonstrated a severe allergy to gluten. Removal of gluten from his diet resulted in complete resolution of his irritable bowel within 2 days.

Case 3: A 15-year-old schoolgirl was admitted to hospital as an emergency whilst on holiday. Her parents believed her to be allergic to nuts. At the age of 5 years, she vomited for about 1 minute after eating a bar of chocolate containing nuts. Three years later, she developed marked angioedema (swelling of the blood vessels) of her face, lips and tongue, followed by tightness of her throat and vomiting: this occurred 2-3 minutes after her friends forced her to have peanuts. Less severe attacks had followed after inadvertent ingestion of hazelnuts and almonds. As a consequence, she avoided peanuts and other nuts wherever possible.

Case 4: Monu, aged 2.5 years, was referred to hospital with eczema. It started behind his knees at the age of 7 months when solids were first introduced into his diet and steadily worsened. Monu has a strong family history of atopic disease; his mother and maternal grandmother both suffered from asthma. Monu was put on a diet free of cow's milk, wheat, oats, peas, beans, nuts, food preservatives and food colourings. Over the following 3 months, there was partial improvement in the severity of his eczema.

Case 5: August 6th, 1982 a woman in Alaska reported that three days before, she and her husband had become ill about one and a half hours after consuming a meal of marinated raw salmon. Illness consisted of generalized (red, itchy

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skin patches), a brassy taste, flushing, abdominal cramps, nausea, and vomiting without diarrhoea. Symptoms persisted for four hours.

You may have read or heard of similar case studies linking consumption of food(s) with adverse reactions such as vomiting, diarrhoea, cramps, wheezing, swelling of the airways, a severe drop in blood pressure, developing gas, bloating, and abdominal pain etc. These are, in fact, different conditions, which link food to adverse reactions. Adverse reaction is a general term indicating a clinically abnormal response or reaction to food regardless mechanism. Foods can cause a wide variety of reactions. Some can be life threatening, however most reactions are less severe. These reactions can fall into several different categories. They can be toxic or non-toxic, they can involve the immune system (as could be in case study, 3 and 4) or stems problems with digestion or metabolism (as in case study 2) or by pharmacologic agents in foods (as in the case study 5). These adverse reactions by mechanism.

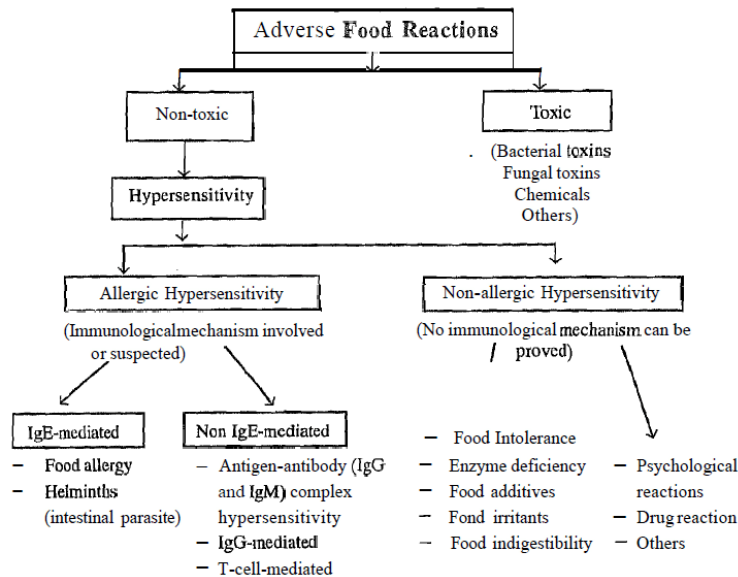


Figure 6.1: Adverse food reactions by mechanism

The toxic reactions from food toxicity or poisoning, can affect anyone. Toxins released from contaminated food or microorganism (bacterial or fungal) or parasites in food cause food-related illnesses. These toxic adverse reactions are not the focus of this unit. Non-toxic adverse food reaction that involves a response due to intake of specific foods classified as "hypersensitivity" or what is commonly known as food allergy and/or reactions caused by problems with digestion or metabolism, referred to as food intolerance are the focus of this unit. Food allergies and intolerances also are different from food poisoning, which generally results from spoiled or tainted food and affects more than one person eating the food.

6.3.1 Food Allergy (Hypersensitivity)

Case studies 1 , 3 and 4 presented above are typical examples of food allergy. What

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is. a food allergy? A food allergy or hypersensitivity is an abnormal response to a food by our immune system. This non-toxic allergichypersensitivity is immune mediated, i.e. immunological mechanism is involved or suspected. To understand this mechanism, we need refresh our understanding of the immune system. We learnt, are immunoglobulin produced by plasma cells in response to an antigen or allergen. Antigen, you may already be aware, is usually a foreign substance (i.e. protein, bacteria, virus, polysaccharide etc.) that stimulates antibody production. Allergens, on the otherhand, are substances foreign to the body that on interaction with the immune system causes an allergic reaction. Five classes of antibodies have been identified. Immunoglobulin A (IgA), Immunoglobulin D (IgD), Immunoglobulin E (IgE), Immunoglobulin G (IgG) and Immunoglobulin M (IgM).

Immunoglobulin E (IgE) helps to eliminate parasites (helminthes) and is responsible for classic allergic reactions commonly referred to as food hypersensitivity or food allergy (refer to Figure 6.1), a reaction that occurs when the immune system reacts to the normally harmless food protein that the body has erroneously identified as harmful. We classify this immune reaction, therefore, as the IgE-mediated reaction, In fact, the immune reactions are classified into four types: Type I, Type II, III, which are antibody dependent and Type IV which is T-cell dependent.

Table 6.1: Types of allergic reactions

<p>Type I: Immediate hypersensitivity, anaphylactic IgE-mediated</p>	<p>This is the quick-onset food allergy. The reaction occurs when an allergen reacts with sensitized IgE-antibody on mast cells. This leads to degranulation of the mast cells releasing mediators such as histamine, cytokines etc. which causes adverse reactions. In certain circumstances, IgG has also been involved in this type of reaction.</p>
<p>Type II: Cytotoxic hypersensitivity</p>	<p>In this reaction IgG antibody reacts with the cell membrane or an antigen associated with the cell membrane and fixes complement, which leads to complement-mediated cell death. It is not usually thought to play a role in food allergy.</p>
<p>Type III: Arthus reaction, antigen-antibody (IgG and IgM) complex hypersensitivity</p>	<p>In this type of reaction antigen and antibody (IgG and IgM) form complex to induce complement fixation and consequent local inflammatory response, several hours after exposure to antigen. This reaction may have a role in several food-related inflammatory diseases (such as colitis, enteritis with bleeding, malabsorptive disorders etc.).</p>
<p>Type IV: Cell-mediated immunity or delayed hypersensitivity</p>	<p>This reaction is mediated by T-cells. T-cells interact directly with antigens. This type of reaction may have a role in celiac disease, protein-losing enteropathies, inflammatory bowel disorder such as ulcerative colitis.</p>

It is important to note that it is the immediate hypersensitivity (Type I), involving IgE, which is responsible for classic allergic reactions. How does this immunopathologic IgE-mediated process leading to allergic reactions work? Let us find out.

Basically, there are three steps involved with the IgE-mediated allergic response.

These include:

Step 1: Sensitization

Step 2: Early phase reaction

Step 3: Late phase reaction

Let us understand this process.

The body's immune system normally reacts to the presence of toxins, bacteria or viruses by producing a chemical reaction to fight these invaders. However, sometimes the immune system reacts to ordinarily benign substances such as food or pollen, to which it has become sensitive. therefore is the first step in the IgE- mediated allergic response. If our immune system is inclined to form IgE to certain foods, our body must be exposed to the food before we can have an allergic reaction. In other words, people with food allergies have an unusually sensitive immune system. A true food allergy occurs when the body's immune system reacts to a protein from a particular food that has been swallowed. This protein is called an allergen. The allergen causes the person's own antibodies to attack the foreign substance. Thus initial exposure to allergen leads to production of allergen-specific IgE.

Production of allergen-specific IgE starts a chain reaction of chemical changes, which adverse reactions in the body. Reaction may occur immediately, in a few minutes, or several hours after the food is eaten (i.e. within minutes of subsequent exposure of Nutritional Management of the IgE antibody to the allergen). This is the second phase of the allergic reaction called the early phase reaction. As the food is digested, it triggers certain cells in our body to produce a food-specific IgE in large amounts. The food-specific IgE is then released and gets attached to the surfaces of mast cells

The next time we eat that food, it interacts with food-specific IgE on the surface of the mast cells and triggers the cells to release chemicals such as histamine, which is a key mediator of the early allergic response, producing smooth muscle constriction, mucus secretion, vascular permeability and sensory nerve stimulation. To illustrate, if you are allergic to a particular food, you may first feel itching in the mouth as you start to eat the food. After the food is digested in the stomach, you may have gastro intestinal disturbance such as vomiting, diarrhoea, or pain. When the food allergens enter and travel through the bloodstream, they may cause your blood pressure to drop, As the allergens reach the skin, they can cause hives or eczema. When the allergens reach the lungs, they may cause airway obstruction, asthma etc.

Allergen also stimulates immune cells (e.g., mast cells, T-cells) to produce inflammatory mediators (e.g., leukotrienes, cytokines). The newly formed mediators act at post- capillary endothelial cells, promoting outflow of plasma leading to localized oedema, adhesion of circulating leukocytes, infiltration of tissues by eosinophils, neutrophils, and basophils. Over the course of several hours, the

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infiltrating inflammatory cells become activated and release mediators stimulating and enhancing further inflammatory reactions. This is the late phase reaction.

Systemic anaphylaxis (life threatening allergic reaction) is the most acute and sometimes fatal response.

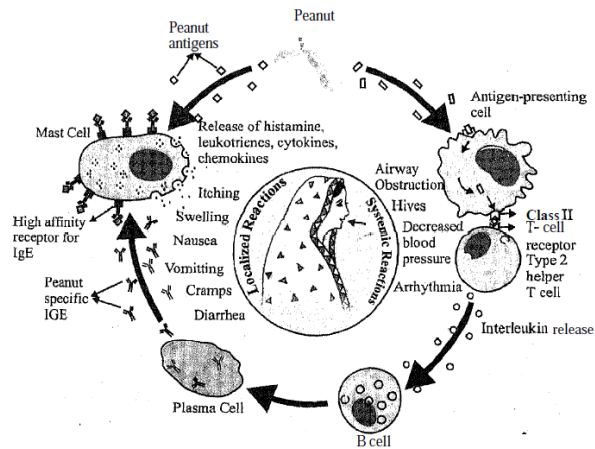


Figure 6.2: The allergy reaction process

From our discussion above, it emerges that a wide range of symptoms express during an allergic reaction ranging from mild abdominal discomfort to life-threatening anaphylaxis, Most frequently occurring symptoms are those linked to the skin, respiratory, cardiovascular and gastrointestinal system. Systemic anaphylaxis (severe allergic hypersensitivity) is the most dangerous allergic reaction and can include abdominal pain, nausea, vomiting, cyanosis, a drop in the blood pressure, chest pain, diarrhoea, shock and death.

Table 6.2: Symptoms of food allergy

Gastrointestinal Manifestations	Skin/Cutaneous Manifestations	Respiratory Manifestations	Systemic Manifestations	Neurological Behaviour
Abdominal pain, nausea, vomiting, diarrhoea, gastrointestinal bleeding, colitis, distention, protein- losing enteropathy	Itching, flushing, urticaria (hives), angioedema (swelling of the blood vessels) eczema, erythema (skin inflammation), redness	Running nose, cough, airway obstruction, airway tightening, wheezing, laryngeal oedema , asthma, rhinitis	Failure to thrive; anaphylaxis, hypotension, dysrhythmias	Headache, irritability, restlessness

The IgE-mediated adverse reaction, commonly referred to as food hypersensitivity or food allergy, though occurs within minutes and rarely takes more than a few hours. It is usually quite severe and, in cases of anaphylactic shock, can be fatal. Considering the pathophysiology of food allergy, it is therefore imperative that we try to understand the risk factors that are specific to development of food allergy.

What are the risk factors for the development food allergy?

Having gone through the discussion above, can you identify at least one important factor leading to food allergy?

Yes, a genuine food allergy occurs when a specific immune reaction occurs in the body in response to consuming a particular food. In other words, exposure to a food (antigen) or some element in the food (allergen, usually a protein) is a prerequisite for the development of food allergy. Excessive exposure to a particular food — for example, in Japan where rice is a staple, rice is a common food allergen; in Scandinavia the common allergen is codfish; while in India, it is chickpeas. Eggs, cow's milk, peanuts, wheat, soya and fish cause most of the allergic reactions in children. Peanuts, walnuts or almonds, fish, and shellfish (such as shrimps, crabfish, lobsters, crab) cause most of the allergic reactions in adults.

Besides food allergens, other risk factors include heredity, gastrointestinal permeability, and environmental factors. Heredity is thought to play a major role in the development of atopic disease. Atopy refers to an individual being prone to develop allergies because a genetic state of hyper responsiveness to allergens. In most cases, allergies occur when an individual who has a genetic sensitivity to certain allergens is exposed to the substance. Family history of allergies increases our risk of developing allergies, including food allergies.

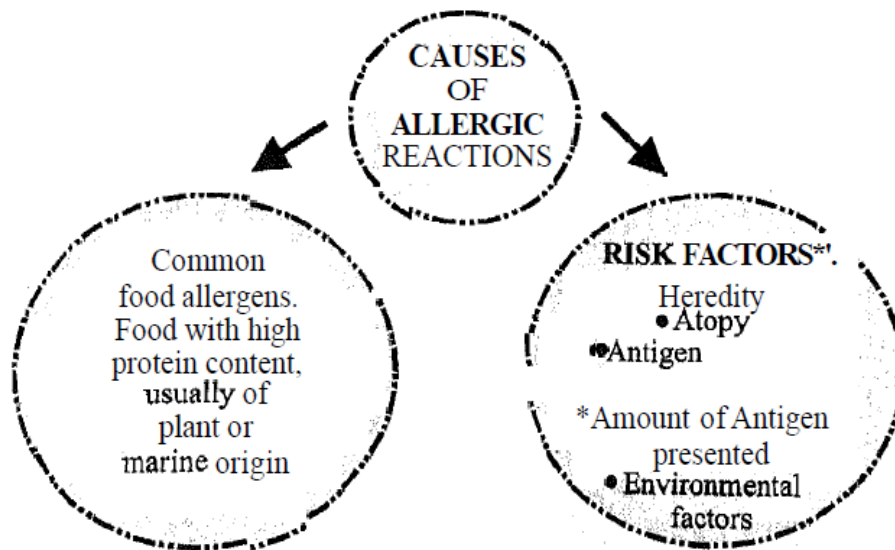


Figure 63: Causes of food allergies

Gastrointestinal permeability may allow antigen penetration and presentation to the lymphocytes. Gastrointestinal permeability is greatest in early infancy and declines with intestinal maturation. Other conditions such as gastrointestinal disease, malnutrition, prematurity and immunodeficiency states may also be associated with increased permeability and risk of developing food allergies. Environmental factors include early exposure to microbes, tobacco smoke, exercise and cold.

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It is important to note that allergies are most likely to affect babies and young children because of their underdeveloped immune system.

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6.3.2 Food Intolerance

What is food intolerance? How does it differ from food allergy? Food intolerance like food allergy is an adverse reaction to food. Food intolerance is different from food allergy in that it does not involve the body's immune system. Food intolerance is a digestive system response rather than an immune system response. It occurs when a food component irritates a person's digestive system or when a person is unable to properly digest or breakdown, the food. It is a non-allergic hypersensitivity, which can occur for variety of reasons. It maybe triggered by a physical reaction to a food or food additive or caused by a metabolic reaction to an enzyme deficiency such as the inability to digest milk properly (lactose intolerance), by phan-nacologicagents in foods, by food poisoning such as ingesting contaminated or spoiled fish, or a food idiosyncrasy such as sulphite-induced asthma. The situation is therefore rather different from a food allergy where a specific person's body for whatever reason reacts against a certain food. Here, the most likely causes are food intolerance or excessive consumption of a certain type of food.

Many factors may contribute to food intolerance. In some cases, as with lactose intolerance, the person lacks the chemicals, called enzymes, necessary to properly digest certain proteins found in food. Also common are intoleranceto some chemical ingredients added to food to provide colour, enhance taste and protect against the growth of bacteria. These ingredients include various dyes and monosodium glutamate (MSG), a flavour enhancer. Let us get to know about these factors in details.

- (a) **Enzyme deficiency or defects** : A food sensitivity or intolerance can occur when the body has difficulty in digesting a particular food and therefore reacts against it. Let us understand this mechanism in details. In normal digestion, the foods we eat are broken down (through chewing and the action of the acids etc. in our stomach and our intestines) into their component parts. The useful ones (the nutrients) are absorbed into the bloodstream through the digestive tract (or 'gut wall'), the redundant ones are flushed out through the bowel. However, if the foods are not properly broken down through some digestive malfunction the body either will not be able to absorb them properly or, since they have not been properly 'processed', may react against them. In the case of lactose intolerance, for example, the body fails to manufacture the enzyme lactase that is needed to digest the lactose sugar in milk. Without lactase, the digestion cannot process the lactose sugar in the milk. The digestion cannot cope with the raw lactose sugar so reacts against it in the form of cramps, vomiting, diarrhoea, abdominal pain etc. One other example is phenylketonuria (PKU), which is an inborn error of metabolism.In the Nutritional Biochemistry Course (MFN-002),

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you may recall studying, that in normal people the enzyme phenylalanine hydroxylase converts phenylalanine (an amino acid) to tyrosine (another amino acid), which is then utilized by the body. In PKU since phenylalanine cannot be hydroxylated to tyrosine, its metabolites accumulate and cause damage to the central nervous system and result in unusual irritability, eczema etc. Galactosemia due to deficiency of the enzyme galactose-1-phosphate uridylyl transferase which converts galactose-1-phosphate to glucose-1-phosphate leads to accumulation of galactose causing various symptoms such as vomiting, fever, jaundice etc.

- (b) **Naturally occurring food chemicals (pharmacologic effect):** Pharmacologically active substances include vasoactive amines such as histamine, tyramine, tryptamine, phenylethylamine, and serotonin. These substances are present naturally in many foods, and produced during fermentation, cooking, ageing and storage in other foods. These are substances, which cause a reaction like an allergic reaction. For example, histamine can reach high levels in cheese, some wines, and certain kinds of fish such as tuna and mackerel. These substances may also be consumed in foods such as fermented foods (i.e. sauerkraut, pork sausages, canned tuna), brewer's yeast, canned fish, avocados and bananas. In susceptible people, these foods can trigger urticaria, facial flushing, decreased blood pressure and headaches.

Further, certain foods, such as irritants, can trigger histamine release from intestinal immune cells, particularly if taken in large quantities. The most familiar of these substances is caffeine, found in tea, coffee, chocolate and cola drinks. A large intake of caffeine can cause tremor, migraine and palpitations. Other irritant substances include lecithin in legume, paprika, chilli, pepper, paprika, acidic foods, and alcohol.

- (c) **Reaction to food additives:** Food additives such as preservatives, flavour enhancers and colouring agents have been linked to adverse food reactions. Additives implicated include tartrazine (the yellow-orange colour dye used in foods, soft drinks, medicine), benzoic acid or sodium benzoate (added in soft drinks some cheeses, processed potato products) causing hives, rash and asthma. Sulphites are a group of sulphite-based compounds that may occur naturally or may be added to food as a flavour enhancer and preservative. Sulphites (including sodium sulphite, potassium sulphite, sodium metabisulphite, sodium bisulphite etc.) are added to many foods and beverages to prevent browning, control microbial growth, modify texture etc. have been well documented to cause adverse reactions such as acute asthma and anaphylaxis, loss of consciousness.

Monosodium glutamate (MSG) is thought to be safe for most people, but in some cases, adverse reaction to this flavouring agent includes headache, nausea, flushing, abdominal pain and asthma.

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- (d) **Reaction to microbial contamination:** The illness described in case 5 above appears to be scombroid fish poisoning, which results from the ingestion of histamine- like substances formed when bacteria degrade the flesh of certain marine fish. Ingestion of improperly handled and stored seafood (yellow- fin tuna, bonito and mackerel) you learnt causes scombroid poisoning. These types of fish contain bacteria in the intestine, which break down the protein (histidine) in the tissue (to histamine) if fish is not immediately gutted and cooled after being caught (i.e. improper storage). Histamine, at high concentrations, is risk a factor for food intoxication whereas moderate levels may lead to food intolerance. A host of symptoms, including skin flushing, facial swelling, dizziness, throbbing headache, oral burning, metallic, sharp or peppery taste in mouth, abdominal cramps, nausea, vomiting, diarrhoea, palpitations, and a sense of unease. Another example of microbial contamination is paralytic shellfish poisoning. The causative agent is saxitoxin. It is a powerful neurotoxin produced by *Gonyaulax catenella*. Initial symptoms of poisoning, which can be seen within 30 minutes of consuming shellfish, (mussels, clams) include headache, a floating feeling, dizziness, tingling, burning, numbness in the extremities, which spreads quickly throughout the body producing general lack of muscular co- ordination.
- (e) **Food indigestibility:** Certain food components, when ingested in large amounts cannot be digested properly and end up in the large intestine where bacteria feed on them. This can lead to bloating, flatulence and diarrhoea. Examples of such foods include fructose, xylitol, sorbitol, cellulose, hemicellulose, lignin, gums etc.
- (f) **Psychological reactions:** Some people may have a food intolerance that has a psychological trigger. For example an unpleasant event in one's life, often during childhood, tied to eating a particular food. Eating that food years later, even as an adult, is associated with a rush of unpleasant sensations and reactions. Symptoms may be precipitated by any food.

Having gone through the discussion above, now can you tell the difference between a food allergy and intolerance? Yes, food allergies can be triggered by even a small amount of the food and occurs every time the food is consumed. People with food allergies are generally advised to avoid the offending foods completely. On the other hand, food intolerances often are dose related. People with food intolerance may not have symptoms unless they eat a large portion of the food or eat the food frequently. For example, a person with lactose intolerance may be able to drink milk in coffee, but becomes sick if he or she drinks several glasses of milk.

Now that we can differentiate between food allergies and food intolerance, let us review the adverse reactions caused by food intolerance. Food intolerance reactions can be quite similar to those of food allergy (as described above), as well as, they may include: the skin (rashes, swelling), airways (asthma, stuffy or runny nose, frequent colds and infections), gastrointestinal tract (irritable bowel

symptoms, colic, bloating, diarrhoea, vomiting, frequent mouth ulcers, reflux, bedwetting), central nervous system (migraines, headaches, anxiety, depression, lethargy, impairment of memory and concentration, panic attacks, irritability, restlessness, inattention, sleep disturbance, restless legs, mood swings etc.). Symptoms of food intolerance can come and go and change throughout life.

Since, the symptoms of food intolerance can be mistaken for those of a food allergy; it is here that the role of the physician and a dietitian is crucial. Being able to determine what the client/patient is experiencing is (a food allergy or a food intolerance) is important for treatment. In the next section, we shall study about the diagnosis, prevention and treatment of these adverse reactions.

6.4 ADVERSE FOOD REACTIONS - THE DIAGNOSIS PROCESS

Diagnosis requires an initial screening, perhaps by a physician (a full physical examination) to rule out other diseases. Diagnostic test in food allergy also includes the biochemical, immunological testing {CAP-FETA, radioallergosorbent test (RAST) etc.) and skin tests. We shall not go into the details of these tests since they are not within the purview of this course. However, it is important to understand that these tests can be helpful only to diagnose the IgE-mediated allergic reactions. Biochemical testing can rule out non-allergenic causes of symptoms. One reliable proof for a food reaction is disappearing of symptoms on elimination and reoccurring on challenge. For food intolerance, particularly the diagnosis is via elimination and challenge with food substances/chemicals. Delayed reactions and non IgE-mediated reactions can, only be diagnosed an elimination diet. Diagnosis requires identification of the suspected food, proof that the food causes an adverse response, and verification of immunological involvement. Following questions about food reactions may be useful for this diagnosis:

- Whether the individual/patient can pinpoint a particular food
- The amount of food eaten
- The time it took from eating to the reaction developing
- Whether the same food has caused a reaction at some other time too
- Whether other foods have caused the same reaction
- How often the reactions occur
- Whether reactions are seasonal
- What is the usual food intake pattern?
- Whether a symptom and food intake diary is maintained, if not keep a symptom and food intake diary and record all events in chronological order.

The first diagnostic tool therefore is the clinical history. Information related to description of symptoms, the time of food ingested relative to the onset of symptoms, a description of the most recent reaction, a list of suspected foods, and

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an estimate of the quantity of food required to cause a reaction will be useful. The food and symptom diary is, therefore, a useful tool if there is a perceived general food reaction with chronic symptoms but no specific suspect food(s). A sample of the food and symptom diary. Asking the patients to maintain such a food and symptom diary, preferably for a week or 10 days, can be quite revealing.

Date:	MORNING Breakfast 6 am - 12 pm	AFTERNOON Lunch 12 pm - 3 pm	EVENING Tea 3 pm - 6 pm	NIGHT Dinner and bed time 6 pm - 6 am
Food				
Medications nutritional Supplements				
Activities				
Symptoms				

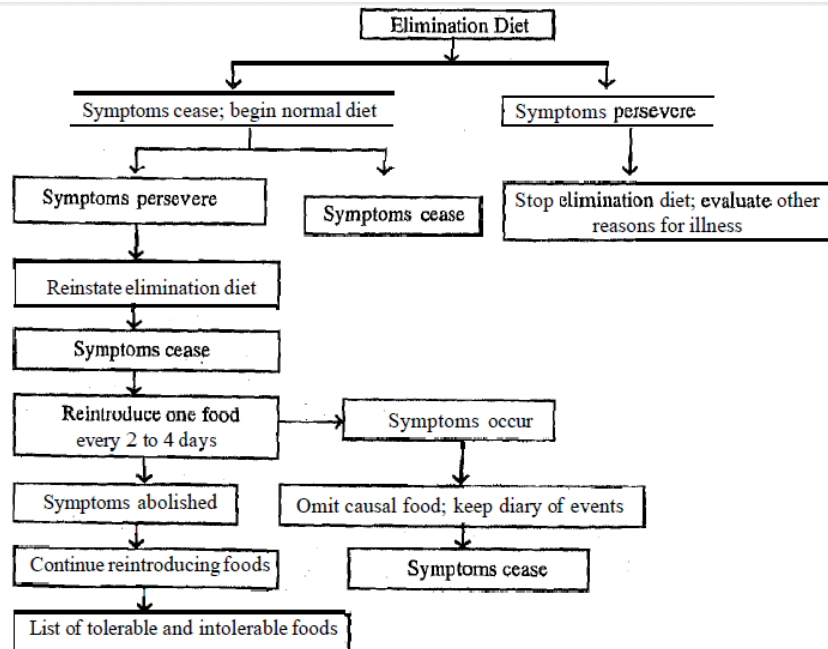
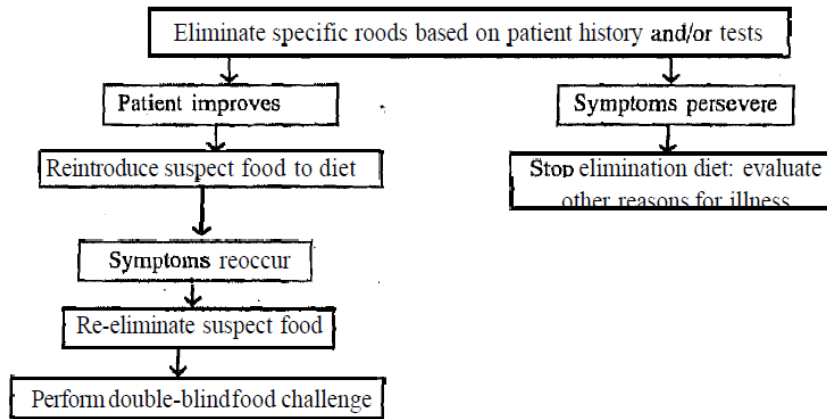
Figure 6.4: Food and symptom diary

Besides clinical history, the diagnosis of the adverse food reaction based on the response to a carefully designed elimination diet is also useful. Food elimination, therefore, is another tool in the diagnosis process. In the diagnostic food elimination, the elimination diets are prescribed for short term, under supervision and only good reason. Elimination diets are therapeutic trials. The elimination diet, as the name suggests, is a diet that eliminates a single or several foods depending on the medical and dietary history.

This diet eliminates foods and food additives considered to be common allergens, such as wheat, dairy products, eggs, corn, soy, citrus fruits, nuts, peanuts, tomatoes, food colouring agents and preservatives, coffee, chocolate etc. In immediate type food reaction the culprit is often known and only the offending food is eliminated. If the reactions are delayed, multiple foods may need to be eliminated. The type of elimination diet selected depends on the diet history, symptoms and severity of the reactions. The elimination diet may be basic, targeted or severe elimination diets.

The use of a basic elimination diet may be helpful in assessing the role of food allergy. In the targeted elimination diet foods based on patients history and/or the results of specific IgE tests to the foods in question is eliminated. The use of severe elimination diet is warranted when removal of suspected food(s) does not eliminate symptoms, multiple food sensitivities are suspected, and food is unlikely to be causing symptoms. The implementation of severe elimination diet. Thus using the elimination diets, foods most commonly implicated and foods most commonly eaten are usually excluded. In food intolerance, food chemicals, as well

as, additives are avoided using the elimination diet.



The diagnostic elimination diet is followed for a set period usually approximately for 4-6 weeks. As you may have noticed in Figure 6.5 and 6.6 above that if no response is seen within this period, a diet of different foods can be tried. If response is seen, the diet should be abandoned and alternate explanations for symptoms explored. If a marked improvement is seen, foods are re-introduced individually or in the case of food intolerance according to food chemical content. Food re-introduction is therefore yet another tool in diagnosis. Let us get to know this element better.

Food challenge or re-introduction is another crucial element in diagnosis. Challenge involves eating a test dose of suspected food or food chemical and noting down the response and the severity of the response. If an initial dose does not elicit a response, increasing doses are then used as challenges, sometimes over several days. If the response is negative, the next food is challenged after a stipulated time. If the response is positive, the food should be avoided. Positive challenge results require the avoidance of the food allergen. In food intolerance, the dose

of chemical tolerated also need to be established. Positive challenge should be repeated at intervals to establish, especially in children, whether they have outgrown the sensitivity.

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The food challenge can be conducted in three ways.

- a) Open Food Challenge (OFC), which allows the food to be given openly,
- b) Single-Blind Food Challenge (SBPCFC), in which the food is hidden from the patient with at least one placebo; and
- c) Double Blind, Placebo-Controlled Food Challenge (DBPCFC), in which the food is hidden from the patient and presented with at least one to three placebo. The ultimate 'gold standard' for the positive diagnosis is the DBPCFC, where the patient is challenged with the offending food in a disguised manner after an elimination period. Interestingly neither the patient nor the physician/dietitian knows whether they are consuming or introducing the allergen or not.

6.5 TREATMENT AND MANAGEMENT OF ADVERSE FOOD REACTIONS

Management of adverse food reactions involves diagnosing the problem followed by nutritional and medical care. We have already reviewed the diagnosis earlier in this unit. Here we shall focus on the nutritional management since medical management is not within the scope of our study.

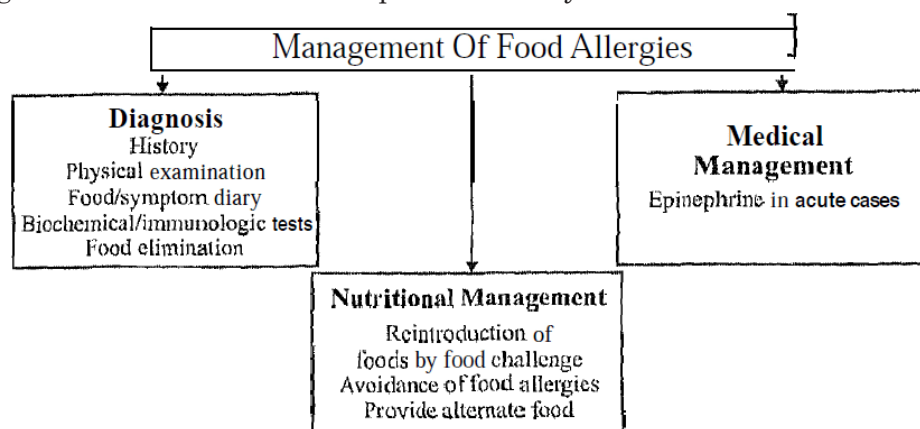


Figure 6.7: Management of food allergies

The four general principles of allergy management include:

1. Avoid factors that cause symptoms.
2. Use appropriate medications.
3. Evaluate for immunotherapy.

4. Educate and follow-up.

The primary treatment for managing food allergies is eliminating the offending food or foods. In fact, non-pharmacological treatment of food allergy requires complete elimination and strict avoidance of the allergen from the diet. A diet should be planned carefully so that the nutrient and calorie needs are met. If a major food group such as dairy products causes an allergy, a supplement or non-dairy food/formula may be added to the diet. In food intolerance, however, the aim should be to eat a diet with a tolerable dose of food chemicals including a wide variety of foods. Although many food intolerances may allow some ingestion of the offending food, food hypersensitivities or allergies do not. In fact, some types of food intolerance can be treated. Hence, we shall not dwell on their management here. We suggest you look up the relevant sections in the units mentioned above to learn about the management of adverse food reactions caused by these conditions.

Here, we would like to continue with our discussion on management of food allergies. Once all food challenges have been completed, a maintenance diet should be planned. A maintenance diet is based on avoidance of offending foods and substitution aiming at a nutritionally balanced diet. To help identify and avoid offending foods, allergy-specific list that describes foods to avoid may be useful. Table 6.3 presents allergy-specific list of foods for your reference. Besides avoidance of offending food, certain nutritional supplements may serve useful in management of adverse food reactions and maintenance of a good nutritional status during the course of treatment. A brief review on these supplements follows.

Improve digestion, helping the intestinal tract control the absorption of food allergens and/or by changing immune system responses to foods. It is well known that probiotics, like lactobacillus, assist in the digestion of lactose. One group of researchers has reported using probiotics to successfully treat infants with food allergies in two trials: a double-blind trial using Lactobacillus GG bacteria in infant formula, and a preliminary trial giving the same bacteria to nursing mothers. Probiotics may also be important in non-allergic food intolerance caused by imbalances in the normal intestinal flora. Many of the effects of allergic reactions, we learnt earlier, are caused by the release of histamine. Some natural substances, such as vitamin C and flavonoids, including quercetin, have demonstrated antihistamine effects in laboratory, animal, and other preliminary studies. However, no research has investigated whether these substances can specifically reduce allergic reactions in humans.

6.6 PREVENTION OF ADVERSE FOOD REACTIONS

Considering the increasing incidence, cost and morbidity associated with allergic reactions, it is perhaps useful to design preventive strategies geared towards minimizing the allergic responses. In fact, prevention strategies for allergic diseases are the key to minimizing the allergic response. In this section, therefore,

we will first focus on preventive strategy for food allergies. Having studied about the pathophysiology of the allergic responses, can you suggest measures means by which we can prevent these adverse reactions?

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Let us help you in this task. You may recall studying earlier about the three steps involved in the allergic reaction process i.e. first sensitization, followed by expression of the disease and symptoms in the second and third step. Based on these three stages of allergic sensitization/elicitation of disease, the preventive strategies too can be classified under three stages, namely:

Primary Prevention: Focuses on blocking sensitization and development of IgE-mediated response. These strategies would be useful for those individuals who have an atopic disposition but no sensitization.

Secondary Prevention: Attempts to inhibit expression of the disease despite sensitization. These are used for the patients who have been sensitized but do not express the disease or only expresses one type of disease (e.g., atopic dermatitis) but not other disorders (e.g., asthma)

Tertiary Prevention: Targets the control of factors that cause symptoms. This strategy would be appropriate for patients who have expressed the disease and needs to limit symptoms.

The first step for prevention is to identify what can and cannot be done. Many strategies, you would realize, in primary, secondary, and tertiary prevention may be similar but are utilized differently according to sensitization and expression of the disease. If a treatment works for allergy symptoms (tertiary), it may be used to prevent expression in a sensitized individual (secondary). If a treatment works for both tertiary and secondary prevention, it may then be utilized to prevent sensitization in primary prevention.

Let us further, move on measures we could adopt in primary, secondary and tertiary prevention. Current research in primary allergy prevention focuses on blocking IgE sensitization, e.g. environmental control. Some areas that are important and require investigation/emphasis at the primary level would include:

How do we identify the high-risk infant during the perinatal period?

How does avoiding exposure to food allergens affect expression of allergy?

Is there a role for maternal avoidance of allergens during the prenatal period and during lactation?

Is there a critical time during pregnancy and/or during the postnatal period when allergen exposure is most likely or least likely to sensitize?

Is there a critical dose required to cause or prevent sensitization?

What is the role of the infant diet, including breast-feeding, non-milk formula, delayed introduction of specific foods, delayed introduction of "at risk" foods (e.g., milk, egg, peanuts).

What are appropriate environmental control measures to limit infant exposures to allergens? (e.g., dust mites, animal dander)

Secondary allergic preventive measures will focus on:

initiating prospective surveillance of infants, young children and adults for expression of sensitization, including sensitization to certain foods (e.g., egg, peanut, milk) and aeroallergens (e.g., dust mites, cat dander, pollens, moulds etc.),

implementing indoor environmental control measures prior to disease expression,

using pharmacologic therapy and/or immunologic modulation in patients with allergic disorders (e.g., atopic dermatitis) to prevent subsequent expression of other disorders (e.g., asthma), and

avoiding environmental irritants (e.g., tobacco smoke, air pollutants).

The tertiary allergy prevention, as you already know, focus on patients with symptoms. Hence, the strategies will target the factors that increase symptoms. These would include environment control at home, school, workplace etc., avoidance of factors (exposure to allergens, environmental irritants) that cause symptoms and patient and family education. Among these measures, the patient and family education, is crucial. Let us get to know it better.

Family and Patient Education

Remember, involvement of the family and all other relevant caretakers along with the patient is crucial for prevention and management of the reactions. The goal of all patient education should be to help patients (and families) take the actions needed to control their symptoms and improve care. Educating the patient about strict avoidance of the foods to which the patient is sensitive is the only proven therapy for food allergy. A child with food allergy must not accept food from classmates or friends. The child, parents and ALL caregivers need to understand this. Just providing a list of "allowed foods" may not be beneficial as ingredients frequently change. Instead,

ENCOURAGE PATIENTS AND FAMILY MEMBERS TO READ LABELS.

Parents must learn to read labels carefully to avoid allergens. Often, the offending food can come in different forms or have a variety of names. For example, a child allergic to milk must avoid whey, a protein found in milk. By law, a food ingredient must be listed on the label, but allergenic components can accidentally get into foods. Children may also be taught how to read food labels and ingredient list. Parents will also need to be watchful when their child is in daycare, school, a restaurant, or at parties.

Further, with advancement in technology, individuals also need to be aware that a new method of growing produce, called biotechnology, may pose a threat to the allergic child or adult. For example, genetic material from peanuts is being transferred to tomatoes and other produce to develop better-looking (but not necessarily better tasting) produce. In this context, it would be required that new

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products be labeled to identify common allergens. Therefore, consumption of such genetically modified foods may need more caution.

Patient education, therefore, is critical and should include:

- Allergen identification (e.g., how to read food labels),
- Avoidance strategies and counseling,
- Symptom recognition,
- Cautions regarding the possibility of a life-threatening reaction,
- What to do in case of accidental ingestion,
- Development of a treatment plan, and.
- Consult a doctor immediately.

Tailoring the educational approach to the need (if the patient is crucial). Using simple language and clear-easy-to-follow instructions/steps and providing written and verbal information will be useful. Some suggestions to help prevent allergic diseases and promote better outcomes for the patient include:

- Promote breast-feeding. Although breastfeeding is one way to delay a child's exposure to allergens, certain allergens from foods in a mother's diet can be passed through breast milk and cause a reaction in an infant. Under this situation, particularly if a family has a history of food allergies and the mother is breastfeeding the child, it may be recommended that major allergenic foods, such as dairy products, eggs, peanuts and tree nuts, fish and shellfish, and soy, may be eliminated from the mother's diet,
- Discourage the early introduction of solid and "at risk" foods (e.g., milk products, eggs, peanuts etc.),
- Reduce dust mite levels in homes and avoid exposure to animal dander (tiny scales shed from skin or hair),
- Screen for allergy at all routine check-ups,
- Increase understanding of allergy and allergic disorders among patients and their families,
- Encourage the use of educational support groups for patients with allergic diseases,
- Encourage the awareness of allergen/environmental control measures at work, school, and home,
- Promote ways to reduce or eliminate environmental tobacco smoke, and.
- Promote legislative support for research, environmental control measures, and public education about allergic disorders.

The preventive measures discussed above so far focus mainly on allergic reactions. Let us now focus on food intolerance. Taking a few simple steps can help prevent the symptoms associated with food intolerance. These steps include:

- Help the patient/child learn which foods in what amounts cause symptoms and limit the intake to amounts the body can handle.

- Inform the patient that when dining out, ask the waiter/cook/server about ingredients of a recipe. Some meals may contain foods, which the individual cannot tolerate and that may not be evident from the description on the menu.
- Encourage the patient/family members to read food labels and check the ingredients for problem foods. Ensure that they do not forget to check condiments and seasonings. They may contain MSG or another additive that can lead to symptoms.

We hope the measures discussed above will go a long way in helping and guiding you to counsel patients suffering from food allergies and food intolerance. Let us now proceed over to the check your progress exercise 3 to reinforce our understanding of the concepts studied above.

6.7 LET US SUM UP

The term "hypersensitivity" is general and may include true allergies, reactions that do not affect the immune system (food intolerance), and reactions for which the cause has yet to be determined. It is unit focused on the immune-mediated and the non immune-mediated adverse food reactions, namely food allergy and food intolerance. Food allergy is a condition which is rarely curable in which specific foods cause an immediate and often dramatic physical reaction such as vomiting, diarrhoea, cramps, wheezing, swelling of the airways, a severe drop in blood pressure etc. In food allergy, food sets off what is known as an IgE mediated reaction in the body. Eggs, cow's milk, peanuts, wheat, soya and fish cause most of the allergic reactions in children. Peanuts, walnuts or almonds, fish, and shellfish (such as shrimps, crayfish, lobsters, crab) cause most of the allergic reactions in adults. Food intolerance, on the other hand, is often linked to other health problems, when the body has difficulty in digesting a particular food and therefore reacts against it, and its symptoms too are somewhat similar to food allergies, but far less defined. Examples of well-understood intolerances are lactose intolerance and phenylketonuria.

Diagnosis of these adverse reaction (food allergies and food intolerance), we learnt is based on clinical history and on the response to a carefully designed elimination diet and on food challenge. The primary treatment for managing food allergies is eliminating the offending food or foods. In food intolerance, however, the aim must be to eat a diet with a tolerable dose of food chemicals including a wide variety of foods. Preventive strategies may be classified as primary, secondary or tertiary level. It is the tertiary level that targets the control of factors that cause symptoms which is most crucial. Environmental control at home, school, workplace etc., avoidance of factors (exposure to allergens, environmental irritants) that cause symptoms and patient and family education are important components of tertiary preventive strategy.

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6.8 GLOSSARY

Anaphylaxis	: it refers to a rapidly developing and serious allergic reaction that affects a number of different areas of the body at one time. severe anaphylactic reactions can be fatal.
Atopic	: relating to, or caused by a hereditary predisposition toward developing certain hypersensitivity reactions, such as hay fever, asthma, or chronic urticaria, upon exposure to specific antigens.
Dermatitis	: dermatitis is a term literally meaning "inflammation of the skin"
Flavonoids	: the term flavonoid refers to a class of plant secondary metabolites most commonly known for their antioxidant activity.
Perinatal	: relates to the period around childbirth, especially the five months before and one month after birth activity.
Phenylketonuria	: a genetic disorder in which the body lacks the enzyme necessary to metabolize phenylalanine to tyrosine.
Rhinitis	: is an inflammation of the mucous membrane of the nose with symptoms of sneezing, itching, nasal discharge and congestion.
Urticaria	: it is defined as the appearance of hives caused by a specific stimulus. A hive, or wheal, is a circular, red, spongy lesion that evolves and changes over minutes to hours.

6.9 CHECK YOUR PROGRESS

- 1). What do you understand by adverse food reactions?
- 2). Food allergy is immune mediated. Elaborate on the statement.
- 3). Which organs are usually affected by food allergies? Give the common symptoms.
- 4). What are the common food allergies seen in adults and children?

- 5). Differentiate between food allergy and food intolerance, giving examples.
- 6). List the common causes of food intolerance.
- 7). What is elimination diet? Enumerate its significance in diagnosis of adverse food reaction.
- 8). What is food challenge? Discuss its relevance.

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UNIT

7

NUTRIENT AND DRUG INTERACTION

STRUCTURE

- 7.1 Learning Objective
- 7.2 Introduction
- 7.3 Nutrient and Drug Interaction: Basic Concept
- 7.4 Effect of Nutrition on Drugs
- 7.5 Drug Effects on Nutritional Status
- 7.6 Drug and Drug Interaction
- 7.7 Clinical Significance and Risk Factors for Drug-Nutrient Interaction
- 7.8 Guidelines to Lower Risk and Wise Use of Drugs
- 7.9 Let Us Sum Up
- 7.10 Glossary
- 7.11 Check Your Progress

7.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- define nutrient drug interactions,
- describe the effect of nutrients and food on drugs and the effect of drugs on the nutritional status,
- identify the clinical significance and risk factors associated with nutrient drug interaction, and
- list handy guidelines for safe and wise use of drug.

7.2 INTRODUCTION

Modern medicine has given us many useful drugs that not only prolong and save lives but in fact improve the quality of our lives. Have you ever thought that the beneficial effects of the drugs that we take can be affected by some of the foods in our diet? Many drugs have powerful ingredients that interact with the human body in different ways. Diet and lifestyle can sometimes have a significant impact on a drug's ability to work in the body. Certain foods, beverages, alcohol, caffeine

and even cigarettes can interact with drugs. These food and drug interactions can have dramatic, even dangerous effects on the way our bodies react to drugs. The purpose of this unit is to present the most common food and drug interactions, to see that we get the best results from the drugs that we need to take.

7.3 NUTRIENT AND DRUG INTERACTION: BASIC CONCEPT

Medicines can treat and cure many health problems. Nevertheless, do you recall your doctor advising or recommending certain medications to be taken with food whereas others on an empty stomach. Have you given a thought as to why this is being advised?

Well, this for the simple reason that medicines can be effected by the food we eat. Well, not all medications are affected by food, but many can be affected by what we eat and when we eat it. Sometimes, taking medications at the same time we eat may interfere with the way our stomach and intestines absorb medication. Other medications are recommended to be taken with food. A food-drug interaction can occur when the food we eat affects the ingredients in a medication we are taking, preventing the medicine from working the way it should. Some nutrients can affect the way we metabolize certain drugs by binding with drug ingredients, thus reducing their absorption or speeding their elimination. For example, the acidity of fruit juice may decrease the effectiveness of antibiotics such as penicillin. Dairy products may blunt the infection-fighting effects of tetracycline. Anti-depressants called MAO inhibitors are dangerous when mixed with foods or drinks that contain tyramine (beer, red wine, and some types of cheese).

So then, can you now define or explain what we mean by drug-nutrient interaction? Drug-nutrient interaction include specific changes in the process by which a drug is absorbed, distributed, metabolized, and eliminated by the body, caused by a nutrient or changes to the kinetics of a nutrient caused by a drug. In fact, nutrient-drug interaction is a broader term that also includes the effect of a medication on nutritional status. Nutritional status, you may already know, refers to the condition of health of an individual as influenced by the utilization of nutrients. Nutritional status may be impacted by the side effects of a medication, which could include an effect on appetite or the ability to eat.

Hence, a study of these interactions is important as it enables the health care professionals and patients to work together to avoid or minimize problems. The benefits of minimizing drug nutrient interaction, you would notice, would go a long way in ensuring:

- medications achieve their intended effect,
- continuity of the prescribed drug by the patient,
- fewer nutrient or caloric supplement is required,

optimal nutritional status is maintained,
disease complications are minimized, and
cost of health care services is reduced.

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The extent of the effects of any food and drug interaction can vary. Potential effects depend on the dose and the form in which the drug is taken (pill, liquid, etc.). It also varies with an individual's age, sex, body weight, nutritional status and specific medical condition. The number of potential food and drug interactions is almost limitless. Interaction problems most often occur with the use of diuretics, oral antibiotics, anticoagulant (blood-thinning) drugs, anti-hypertensive drugs, thyroid and sodium compounds, and alcohol.

Generally, administering oral medication along with the food or at a mealtime is a convenient manner of drug dosing. However, drug interactions can occur that modify the activity of the drug (decrease or increase drug effects) are referred to as drug-drug interactions or impair the nutritional benefit of certain food are called as a drug-nutrient interaction. While the effect of food or a nutrient on a drug or medication is a nutrient-drug interaction. The most commonly observed type of drug-food interaction affects the drug absorption.

Such interactions raise concerns that medications may lead to nutritional deficiencies or a poor diet may change how a medication works. This does not mean that if one is taking a medication, one needs to use a vitamin and or mineral supplement. There is a little chance that taking a medication for a short time (such as a ten day treatment), will affect the nutritional status, However, use of some medications for months or years may affect the nutritional health,

Drug and nutrient relationship can be categorized into two aspects:

1. Effect of nutrition on drugs: the influence of nutritional factors on drug absorption, action and effectiveness, and.
2. Effect of drugs on nutrients: the influence of drugs on nutritional intake, metabolism, excretion and requirements.

We will study about these interactions in the subsequent sections. We will also briefly review the drug and drug interactions. So let us get started. We shall begin first with effect of nutrition on drugs.

7.4 EFFECT OF NUTRITION ON DRUGS

Pharmacokinetics is the study of the time course of a drug in the body involving the absorption, distribution, metabolism and excretion of the drug. The movement of the drug through the body during absorption, distribution, metabolism etc. can be influenced by food or nutrients in the diet. These influences are reviewed next.

Food's Effect on Drug Absorption

The pharmacological effect of a drug depends on the rate and extent to which it is absorbed from the gastrointestinal tract. Food can decrease a drug's rate of absorption and/or increase the extent of absorption of numerous drugs. Examples include penicillin, tetracycline (TCN). The possible reasons for this may include:

- delayed gastric emptying,
- altered gastrointestinal pH,
- competition for binding sites with the nutrients,
- adsorption or the adhesion of food or a food component,
- chelation (combining) of drugs by food cations, and
- dietary fats impeding the absorption of hydrophilic drugs.

Gastric emptying may be delayed by the consumption of high-fibre meal and meals with high fat content. Chelation reactions occur between certain medications and divalent or trivalent cations (a positively charged ion), such as iron, calcium, magnesium etc. and the absorption of the drug may be reduced by chelation (combining) with one of these metal ions. To illustrate, the antibiotic tetracycline forms insoluble complexes with calcium in dairy products thus preventing or reducing the absorption of both drug and nutrient. Adsorption is another mechanism by which drug absorption is slowed or reduced. Example of this mechanism is the cardiovascular drug digoxin which should not be taken with high phytate food (such as wheat bran, oatmeal etc.).

The presence of food in the stomach enhances the absorption of some medication. Drugs whose absorption increases when taken with food include drugs such as spironolactone, griseofulvin and itraconazole. With some drugs, this food-drug interaction may be utilized to achieve higher serum drug levels or to use lesser amounts of drug per dose. For example, administration of the drug ketoconazole with acidic beverages such as colas, leads to increased and prolonged serum levels for the drug. This mechanism is based on changes in the gastrointestinal pH. Generally, these interactions have an insidious onset and may not be clinically evident except for the failure to achieve the therapeutic goals of therapy or loss of disease control. Continuous long-term monitoring of patients is needed when drugs and food must be taken together. An example to substantiate this aspect is as follows:

- The calcium in milk and milk products such as yoghurt and cheese decreases the absorption of certain antibiotics, including tetracycline. Therefore, these foods should not be eaten at the same time this drug is taken, so that the full dosage of the drug is available for adequate treatment of the infection.
- Tyramine is a vasoconstrictor that raises blood pressure. Significant ingestion of high-tyramine food, such as aged cheese and cured meats, by individuals while being treated with mono-amine oxidase inhibitors (MAOI) - an antidepressant, can cause a hypertensive crisis such as increased heart rate, flushing, headache,

- stroke and even death.
- Caffeine in foods or beverages increases the adverse effects of stimulant drugs such as amphetamines, methylphenidate, causing nervousness, tremor and insomnia.

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Box 7.1	Other Examples of Drug-Food Interactions
	<ol style="list-style-type: none"> 1) Vitamin K found in green leafy vegetables, tomatoes, coffee, beef liver, green tea etc., and some non prescription vitamin-mineral products can antagonize the anticoagulant effect of warfarin, resulting in decreased anticoagulant activity and lowered prothrombin time (PT); laboratory blood tests. 2) Vitamin B₆ (pyridoxine) found in avocados, beans, peas, sweet potatoes, bacon, pork, tuna, and some non prescription vitamin-mineral products, increases the metabolism of levodopa, producing decreased blood levels of dopamine and antiparkinsonism effects. We will learn more about this later in Unit 17. 3) Calcium, magnesium and aluminium found in food supplements or antacid compounds bind (chelate) with ciprofloxacin and tetracycline to form an insoluble complex resulting in significantly decreased absorption of these antibiotics and decreased antibiotic effect. 4) Calcium in vitamin-mineral products and liquid enteral nutritional supplements interact with some fluoroquinolone antibiotics and with phenytoin, reducing their bioavailability and resulting in decreased antibiotic activity and loss of seizure control, respectively.

Foods Effect on Drug Transport

Many drugs are transported in blood bound to plasma proteins. Severe malnutrition or diseases affecting the synthesis of plasma proteins (such as liver disease) may reduce the body's ability to transport drugs and hence impair their effectiveness. Albumin is the most important drug-binding protein in the blood. Low serum albumin levels (due to inadequate protein intake and poor nutrition), therefore, provides fewer binding sites for some highly protein-bound drugs such as warfarin and anticonvulsant phenytoin, which may lead to risk of excessive anticoagulation and bleeding or toxicity, respectively.

Food Effects on Drug Metabolism

The general tendency of the process of metabolism is to transform a drug from a lipidsoluble to a more water-soluble compound so that it can be handled more easily by the kidneys and excreted in the urine. Food can both inhibit and enhance the metabolism of drugs by altering the activity of the enzyme systems operating in the body. To illustrate, scientists discovered that grapefruit contains natural

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substances that can affect the way certain prescription medications are broken down (metabolized) by an enzyme, known as CYP3A4 (cytochrome P-450 3A4). This means that if a person drinks grapefruit juice and takes drugs (such as felodipine, Zocor) orally, more of the drug may enter the bloodstream than would have under normal circumstances resulting in a greater pharmacologic effect and possible toxicity. Factors, which affect the deactivation or conjugation of a drug, can thus alter its pharmacological or toxic effects.

Periods of short-term starvation or prolonged periods of nutritional inadequacy can influence the effectiveness or safety of drugs. The amount of a drug required to produce a certain pharmacological effect is determined by the body weight. Sudden reduction in weight or dehydration may therefore result in over dosage. Undernutrition also reduces the activity of microsomal drug metabolizing enzymes and this can diminish a drug's effectiveness (by reducing its rate of excretion). These are additional reasons for ensuring nutritional adequacy during illness, particularly since this is when pharmacological drug use is most likely to be needed.

Alcohol, which is also a drug in its own right often affects microsomal enzyme activity and can potentiate the action of some hypoglycemic drugs or central nervous system (CNS) depressants. The action of certain drugs such as propranolol may be reduced by alcohol.

Certain nutrients can also have a direct influence on drug metabolism. Vitamin K reduces the anticoagulant effect of warfarin and the dosage has to be sufficient to counteract the effects of habitual vitamin K intake. Any significant alteration may necessitate adjustment in warfarin dosage.

Sodium intake inversely affects serum levels of the mood stabilizer lithium carbonate and since this drug has a narrow range of therapeutic effectiveness, dietary sodium intake must be kept to a constant level in patients receiving this treatment.

Foods Effect on Drug Utilization

The following illustrations highlight the effect of food on drug utilization.

Liver and green leafy vegetables can decrease the effect of anticoagulants (blood-thinning drugs). These foods contain vitamin K, which helps promote blood clotting. On the other hand, aspirin and aspirin-containing compounds can enhance the effect of the blood-thinning drug and promote excessive bleeding.

- One of the most hazardous food and drug interactions is between monoamine oxidase (MAO) inhibitors and aged or fermented foods. MAO inhibitors are used to treat depression and high blood pressure. They decrease the metabolism in the body of compounds called monoamines. MAO inhibitors can react with a substance called tyramine (a monoamine) in foods such as aged cheese, fava beans and others. As a result blood pressure can rise to dangerous levels causing severe headaches, brain hemorrhage and, in extreme cases, death.

- Natural licorice contains a substance which can increase blood pressure when eaten in large amounts. Long-term use of licorice and licorice-flavoured candy or drugs can counteract the effect of medication used for treating high blood pressure.

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Food Effects on Drug Excretion

Food and nutrients can alter the reabsorption of drugs from the renal tubes. Urinary acidity affects drug reabsorption from the renal tubules. Hence, a change in urinary pH by food may change the amount of drug existing in the nonionic state, thus increasing or decreasing the amount of drug available for tubular reabsorption. Supplemental intakes of nutrients, which increase urinary acidity (e.g., large amounts of vitamin C intake), can decrease the excretion of salicylate drugs such as aspirin.

Effect of Nutrient Supplements on Drugs

A nutrient or nutrient supplement can alter the pharmacological action of a drug by enhancing the drug effect or by opposing it. These can result in drug-nutrient interactions. To illustrate, warfarin, is an oral anticoagulant that reduces the production of vitamin-K dependant clotting factors by inhibiting the conversion of vitamin K to a usable form. Because, this is a competitive interaction, the ingestion of vitamin K in the usable form (supplement) will oppose the action of warfarin and allow the production of more clotting factor. Therefore, to achieve an optimal level of coagulation, a balance must be maintained between the dose of the drug and the ingestion of vitamin K. On the other hand, ingestion of other nutrients like vitamin E in doses greater than 400 IU may enhance the anticoagulant effect of warfarin. Enhancement of the anticoagulation effects of warfarin may lead to serious bleeding events.

Further, in excessive amounts, vitamins and minerals act like drugs instead of nutrients. Nutrients in excessive amounts may interact with other nutrients or may even be toxic. For example, large amounts of zinc can interfere with copper and iron absorption. Similarly, large amounts of iron can interfere with zinc absorption.

Effect of feeding method on drug availability

The form in which a drug is administered or enters the body can influence its absorption, metabolism or excretion. This becomes more pronounced among critically ill patients who can not consume drugs orally and are on enteral food support. If the drug has to be administered through the enteral feeding tube it needs to be either crushed or dissolved in a solvent. Crushing oral preparations to pass down the tube may alter their absorption time/activity. Some of the drugs can not be added to the food infusion as it may alter their stability. Several drugs

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are available only in the form of tablets or capsules, dissolving these in feed, water, alcohol or sorbitol may not always be feasible. Drug such as cimetidine, aluminium hydroxide, metaclopramide are physically incompatible with enteral foods. Certain drugs like phenytoin form complexes with calcium and protein thereby resulting in markedly reduced absorption of this medication. Such drugs need to given only by stopping enteral feed infusions for 1-2 hours. Patients on total parenteral nutrition generally have a separate tube (catheter) for infusion of drugs. Toxicity/overdosage of drugs is frequently observed in case of certain drugs administered directly into the blood.

7.5 DRUG EFFECTS ON NUTRITIONAL STATUS

Drugs can affect nutritional status in a number & ways i.e. by enhancing excretion of certain nutrients, by interfering with nutrient absorption, or by decreasing the body's ability to change nutrients into usable forms. These effects are gradual so that the effects will be greater in persons taking drugs over a long period of time. For these people, vitamin and mineral deficiencies may result. Here are some examples of drug effects on nutrients in the body:

- Abuse of antacids can lead to phosphate depletion. This can lead to a vitamin D deficiency in severe cases. Some patients have developed osteomalacia or softening of the bones due to loss of calcium because of a vitamin D deficiency.
- The excessive use of diuretics ("water pills") may result in the loss of electrolytes, mainly potassium. This may put people with heart problems at higher risk for serious heart rhythm problems. People taking diuretics regularly should eat foods which are good sources of potassium: tomatoes, tomato juice, oranges, orange juice, bananas, raisins, pines, potatoes, sweet potatoes and winter squash.
- Women who take oral contraceptives over a long period of time may develop folic acid and vitamin C deficiencies if their diets are inadequate in these nutrients. The best sources of folic acid are spinach and other greens, asparagus, broccoli and lima beans. Excellent vitamin C sources include oranges, grapefruits, lemons and limes, strawberries, tomatoes, potatoes, cabbage and green peppers.
- Anticonvulsant drugs, prescribed to prevent seizures, can lead to vitamin D and folic acid deficiencies. The use of vitamin supplements by patients taking these drugs should be medically monitored.
- The anti-hypertension drug hydralazine can deplete the body's supply of vitamin B₁₂. This vitamin is widely distributed in foods. Some good sources are chicken, fish, liver, whole grain breads and cereals, egg yolks, bananas and potatoes. Consumption of these foods should be encouraged.
- Several drugs, including colchicine (used to treat acute gout), oral antidiabetic agents, and the antibiotic neomycin can impair absorption of vitamin B₁₂. Persons who do not eat any animal products (vegans) may have poorer vitamin B₁₂ status and may be at greater risk for a deficiency when taking one of these

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drugs.

It is, therefore, evident that use of certain drugs can lead to deficiency conditions and poor nutritional status. It is also common to find that certain drugs cause altered taste or dysgeusia, change in appetite, have gastrointestinal effects and some may lead to organ system toxicity. We shall review these changes briefly next.

Drug Effects on Food Intake

Food intake may be reduced because of drugs which:

- Have an anorexic effect, either as a direct effect of the drug on appetite e.g., some antibiotics or because of side-effects such as drowsiness or lethargy e.g., tranquilizers. Drugs can suppress appetite, leading to undesired weight changes, nutritional imbalances, and growth retardation. Most central nervous system stimulants, including amphetamine mixture and methylphenidate, suppress appetite or cause frank anorexia.
- Cause nausea and vomiting: This is a common side effect of many drugs, particularly the antineoplastic drugs, used to treat cancer.
- affect the gastrointestinal tract: Gastrointestinal irritation and ulceration are serious problems with many drugs. You may be aware that Non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin or ibuprofen often cause stomach irritation, indigestion, heartburn, gastritis, ulceration and sudden serious gastric bleeding. Other drugs may produce gastrointestinal side effects such as bloating or early satiety. Drugs can also cause changes in bowel function that can lead to constipation or diarrhoea. Narcotic agents such as codeine and morphine cause a nonproductive increase in smooth muscle tone of the intestinal muscle wall, thereby decreasing peristalsis and causing constipation. On the other hand use of certain drugs can lead to the destruction of intestinal bacteria leading to diarrhoea.
- Causes taste changes: Several drugs can cause an alteration in taste sensation, reduced acuity of taste sensation or leave an unpleasant after taste, any of which can affect food intake. Common drugs that cause alteration in taste sensation include the antihypertensive drug captopril, the anticonvulsant phenytoin. cause dry mouth (xerostomia) : Lack of saliva makes it difficult to masticate and swallow foods, especially those of a dry or fibrous consistency. Dry mouth immediately
- Causes loss of taste sensation: If dry mouth condition prevails for a long-term it can cause dental caries and loss of teeth, gum diseases and nutritional imbalance and undesired weight loss. The drugs to watch out in this case are anticholinergics, which include tricyclic antidepressants such as diphenhydramine, antihistamines (e.g. benadryl), and antispasmodic bladder control drugs such as oxybutynin (ditropan). These anticholinergic drugs compete with the neurotransmitters acetylcholine for its receptor sites, thereby inhibiting transmission of parasympathetic nerve impulses. This results in

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decreased secretions, including salivary secretion causing dry mouth.

- Cause sore or painful mouth : This is a common side effect of chemotherapy and can significantly affect food intake. Antineoplastic drugs, used in chemotherapy for cancer, affect the mucous membrane as well, causing inflammation or mucositis. This may manifest as stomatitis (mouth inflammation), glossitis (tongue inflammation) or cheilitis (lip inflammation and cracking).
- Confusion : Drugs, which impair memory or cause confusion can result in people forgetting to eat. Central nervous system side effects can interfere with the ability or desire to eat, Drugs that cause drowsiness, dizziness, ataxia, confusion, headache, weakness, and neuropathy can lead to nutritional compromise, particularly in older patients and chronically ill patients.

From our discussion above, it must be evident to you, that any of these problems, which are mentioned above from dry mouth to gastrointestinal irritation to constipation or diarrhoeas, can negatively affect food intake and absorption and thus have an impact on the nutritional status of the patient. This is one side of the coin. must understand that drugs may also increase food intake. This is because they can:

- stimulate appetite : This is a common side effect of corticosteroids, insulin and psychotropic drugs, and
- induce cravings for particular types of foods, particularly carbohydrates. Some psychotropic drugs have this effect.

Drug Effects on Absorption

Many drugs can impair, prevent or reduce absorption of nutrients due to:

- Formation of insoluble complexes : many drugs can chelate with minerals and trace elements e.g., antibiotics ciprofloxacin and tetracycline form insoluble complexes with calcium, magnesium, zinc or iron, thus preventing or reducing the absorption of these vital nutrients..
- Competition for binding sites : e.g., salicylate drugs such as aspirin competes with vitamin C.
- Damage to the absorptive surface of the intestinal mucosa : drugs used in chemotherapy can cause villous atrophy, resulting in malabsorption.
- Lack of bile acids : the absorption of fat-soluble vitamins such as vitamin A, D, E and K, will be impaired by bile salt binding drugs such as cholestyramine.
- Increased intestinal mobility : drugs, which cause diarrhoea or stimulate peristaltic activity may result in nutrient losses.

Drug effects on Metabolism

Drugs can affect the metabolism of various essential nutrients in the body. These impairments are highlighted here with:

- Carbohydrate metabolism: Hypoglycemic drugs such as insulin and

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sulphonylureas are prescribed because of their ability to increase carbohydrate utilization, and their action has to be balanced with carbohydrate intake in order to maintain glycemic control. Other drugs such as oral contraceptives and corticosteroids have adverse effects on carbohydrate metabolism and worsen glucose intolerance.

- Lipid metabolism: Some drugs are used to correct lipid metabolism, whilst others such as chlorpromazine and phenobarbitone can induce hyperlipidemia.
- Vitamin and mineral metabolism : are required as cofactors or coenzymes in many metabolic pathways, including those by which drugs are metabolized. Increased activity of these pathways because of drug metabolism may therefore increase micronutrient requirements.

Drugs can also compete with, or inhibit, the metabolic conversion of some micronutrients to their active metabolites, particularly folate. Methotrexate (used in the treatment of some cancers) directly antagonizes folic acid metabolism by inhibiting the activity of enzyme dihydrofolate reductase. Similarly, anticonvulsants impair vitamin D metabolism with consequent disturbances in calcium metabolism which adversely affects on bone.

- Dietary Components: Drugs may also affect the metabolism of dietary components such as MAOI.

Drugs Effects on Excretion

Use of certain drugs can influence the excretion of certain substances. For example, besides their intended increase in sodium excretion, diuretic drugs can also result in - enhanced losses of other elements such as potassium calcium, magnesium and zinc. Tetracycline increases the urinary excretion of vitamin C.

So far, we have covered the relationship or the interaction of drug with nutrients and the effect of food on drug absorption, metabolism, utilization and excretion. Let us now move on to the study of the effect of a drug on the other drug i.e. the drug and drug interaction.

7.6 DRUG AND DRUG INTERACTION

In the discussions above, we reviewed the effect of food on drug metabolism. Interestingly, not only can drugs interact with food and alcohol, they can also interact with each other. Some drugs are prescribed together on purpose for an added effect, like codeine and acetaminophen for pain relief. However, other drug-to-drug interactions may be unintended and harmful. Prescription drugs can interact with each other or over-the-counter (OTC) drugs, such as acetaminophen, aspirin, etc.

Sometimes, the effect of one drug may be increased or decreased. For example, tricyclic antidepressants can decrease the ability of a hypotensive to lower blood

pressure. In other cases, the effects of a drug can increase the risk of serious side effects. For example, some antifungal medications can interfere with the way some cholesterol-lowering medications are broken down by the body. This can increase the risk of a serious side effect.

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7.7 CLINICAL SIGNIFICANCE AND RISK FACTORS FOR DRUG-NUTRIENT INTERACTIONS

We are already aware of the fact that poor nutritional status can impair drug metabolism and the drug treatment can have a detrimental effect on the nutritional status. Not all drug-nutrient interactions are clinically significant. In many instances, any losses in nutrient availability or drug action will be small in scale and may be of short duration. Drugs, which are most likely to have diuretic implications, are those which:

- have a narrow range between therapeutic effect and toxicity,
- need to be taken for a prolonged period,
- have implications in terms of the timing of food intake,
- necessitate dietary restrictions or regulation,
- have side-effects which influence appetite or gastro-intestinal function, and
- compete directly with a nutrient.

People who are at risk from drug-nutrient interactions are the:

- Persons who have a poor diet or in other words have a poor nutritional status. Existing malnutrition places patients at greater risk. Protein alteration, particularly low albumin level, as you may recall studying earlier, can effect drug disposition,
- Persons who have serious health problems. Patients with active neoplastic diseases (cancer) or active acquired immunodeficiency syndrome (AIDS) with significant anorexia and muscle wasting are at special risk.
- Body composition: This is an important consideration in determining drug response. In obese or older patients, for instance, the proportion of adipose tissue to lean body mass is decreased. Accumulation of a drug and its metabolite in adipose tissue is greater, and may result in prolonged clearance and increased toxicity.
- Foetus, growing children, pregnant women: These individuals are at high risk for drug nutrient interaction.
- The physiological changes that occur with age, such as a decrease in lean body mass and body water, fall in plasma protein concentration, and a general decline in renal and liver function, mean that the risk of adverse drug reactions is much higher. Elderly people are also more likely to be given the types of drugs with powerful effects and which are most likely to have an impact on nutrition

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e.g., cytotoxic drugs, anti-Parkinson's drugs and antidiabetic drugs. Diminished salivation may make it more difficult to swallow tablets and oesophageal motility disorders may lead to bulky drugs sticking in the oesophageal mucosa. Other problems such as failing memory, poor hearing and vision, and difficulty with opening containers may mean that drug regimens are not followed correctly, particularly if they are complex. Many of these factors are likely to coexist in elderly people.

Persons taking two or more medications at the same time.

Persons using prescription and over-the counter medications together.

Persons not following medication directions.

Persons taking medications for long periods of time.

Persons who drink alcohol or smoke excessively.

7.8 GUIDELINES TO LOWER RISK AND WISE USE OF DRUGS

There can be no two views regarding the fact that the management of many diseases require drug therapy. However, they must be taken properly to ensure that they are safe and effective. Many medicines, as we have learnt so far, have powerful ingredients that interact with the human body in different ways, and diet and lifestyle can sometimes have a significant impact on a drug's ability to work in the body.

To help you in this task and equip you to take wise decisions here are a few handy guidelines. In fact, you may want to advocate these guidelines to the patients as well who shall come to you for dietetic advice.

Guidelines to Help Use Drugs Wisely

By now, you are aware that the interaction of foods and drugs is a complex problem. Researchers cannot always predict whether a new drug will react with a food or if a drug that has been in use for some time will react with a new food. Moreover, you, as the consumer, cannot be expected to know everything about the drug.

Then, what can we do to get the greatest benefit from drugs with the least risk? The following guidelines will assist us in preventing problems and getting the most from the medicines that one must take:

- When the doctor prescribes a medicine, be sure to mention every other drug taken including alcohol and over-the-counter agents such as aspirin, antacids and laxatives. If you don't know the amount and types of drugs you are taking, take the bottles with you when you visit the doctor.
- Be sure you understand when and how the drug/supplement/product should be taken and then follow directions (for example, with meals, before meals, or after meals. You could perhaps paste labels on the bottle(s) to remind you

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when and how the drug should be taken.

- Tell the doctor about any changes or ill effects you have after taking the drug and any unusual symptoms that occur after eating certain foods. Use the telephone, Do not wait until your next visit.
- Taking drugs with a full glass of water is generally the safest way and in many cases, it may help prevent irritation of the stomach lining.
- Do not mix medication into hot drinks, because the heat from the drink may destroy the effectiveness of the drug.
- Do not take vitamin pills at the same time as taking the medication; vitamins and minerals can interact with some drugs.
- Do not stir medicine into your food or take capsules apart (unless directed by your physician. This may change the way the drug works.
- If you take any drug, do not use alcohol without checking with the doctor first to see if it will be safe.
- If you have been taking a drug for a long time, ask the doctor if you should be concerned about any vitamin or mineral deficiencies.
- Read directions, warnings and interaction precautions printed on all medicine labels and package inserts.
- When buying any over-the-counter medicine, be sure to read the label and the package insert for directions and warnings. If in doubt about the product, ask the pharmacist.
- Finally, use the least number of drugs possible and take them as directed to reduce the chances of developing a drug/drug or food/drug interaction.

How to Lower the Risk of Drug-Nutrient Interactions

- Eat a healthy diet using the food guide pyramid.
- Follow directions on how to take medication (prescription and over-the-counter)
- Read warning labels on both prescription and over-the-counter medications.
- Do not take over-the-counter medications frequently on your own.
- Tell the physician about any other medications being taken, including over-the-counter medications and alcohol.
- Tell the physician about any new or intensified symptoms that develop when taking a medication.
- Keep a list of all medications (prescription and over-the-counter) being used.
- If you have questions, ask your physician for answers.

Besides the handy tips listed above, you will find some useful tips regarding the usage of certain drugs along with food and alcohol

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Table 7.1: Nutrient and drug interaction - handy tips

S. No.	Drugs	Interaction with	
		Food	Alcohol
1.	Antihistamines	Best on empty stomach	Increase drowsiness and slow mental and motor performance.
2.	Analgesics/ Antipyretics	Best on empty stomach	Increase risk of liver damage or stomach bleeding
3.	Non-steroidal Anti-inflammatory Drugs (NSAIDS)	Best with food or milk	Increase risk of liver damage or stomach bleeding
4.	Corticosteroids	Best with food or milk	---
5.	Narcotic Analgesics	---	Increases sedative effects of medication
6.	Bronchodilators	Can result in side effects such as nausea, vomiting, headache and irritability. Caffeine containing foods or beverages can stimulate central nervous system	Increase risk of side effects such as nausea, vomiting/headache and irritability.
7.	Diuretics	Cause loss of K, Ca and Mg. Also can lead to hyperkalemia resulting in irregular heart beat and heart palpitations	---
8.	Beta Blockers	---	Lowers the blood pressure
9.	Nitrates	---	Lowers the blood pressure
10.	Angiotensin Converting Enzyme (ACE) inhibitors	Decreases absorption Avoid foods rich in potassium	---
11.	HMG CoA Reductase Inhibitors	Absorption enhances with food intake	Increase the risk of liver damage
12.	Anticoagulants	Vitamin K reduces effectiveness	---
13.	Antibiotics	---	---
14.	Antifungals	Depends on an individual's tolerance. Avoid Ca-containing foods. Caffeine leads to excitability and nervousness	---
15.	MAO inhibitors	Avoid foods rich in tyramine	Avoid Alcohol
16.	Anti-anxiety drugs	Caffeine-rich foods cause excitability, nervousness and hyperactivity	Impair mental and motor performance
17.	Anti-depressant drugs	With or without food	Affect mental or motor skills
18.	Histamine blockers	With or without food caffeine-rich foods irritate the stomach	Irritate the stomach and delays the healing process.

7.9 LET US SUM UP

Certain foods, beverages, alcohol, caffeine, and even 'cigarettes can interact with medicines. This may make them less effective or may cause dangerous side effects or other problems. On the other hand, certain medicines and drugs have powerful ingredients that interact with the human body in different ways, and these can sometimes significantly influence the absorption, metabolism and utilization of nutrients in the body leading to poor nutritional status. These nutrient drug interactions, therefore, were the focus of this unit.

We learnt about the different ways in which the food affects drug absorption, metabolism. Further, the relationship between drugs and the nutritional status was also explored. The factors by which the drug influences food intake and the effect of drug on nutrient absorption, utilization and excretion were highlighted. The unit finally dwelt upon the risk factors associated with nutrient drug interaction(s) and presented handy guidelines for wise use of drugs and measures to adopt to reduce the risk from nutrient drug interactions.

7.10 GLOSSARY

Anti convulsants	: drugs used to prevent or treat convulsions (seizures) such as in case of epilepsy.
Anti fungals	: drugs that treat fungal infection.
Anti histamines	: drugs that treat symptoms of allergies.
Bronchodilators	: drugs that widen the airways of the lungs to ease breathing difficulties.
Dysgeusia	: an impairment or dysfunction of the sense of taste.
MAO Inhibitors	: mono-amine oxidase inhibitors are medicines that relieve certain types of mental depression.
Neuropathy	: a problem in peripheral nerve function (any part of the nervous system except the brain and spinal cord) that causes pain, numbness, tingling, swelling, and muscle weakness in various parts of the body.
Osteomalacia	: disease occurring mostly in adult women that results from a deficiency in vitamin D or calcium and is characterized by a softening of the bones with accompanying pain and weakness.
Prothrombin	: a plasma protein that is converted into thrombin

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7.11 CHECK YOUR PROGRESS

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- 1). What is drug nutrient interaction? Why is the study of these interactions important?
- 2). What are the different categories into which nutrient drug relationships can be categorized?
- 3). Food can decrease or increase a drug's rate of absorption. Comment on the statement giving suitable examples.
- 4). Give two examples of the effect of food on drug utilization.
- 5). How is drug nutrient interaction different from a nutrient drug interaction and a drug-drug interaction?
- 6). What are drug and drug interactions? Explain giving example.

8

NUTRITIONAL DIET AND CANCER

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STRUCTURE

- 8.1 Learning Objective
- 8.2 Introduction
- 8.3 Cancer
- 8.4 Etiological Risk Factors in Cancer
- 8.5 Metabolic Alterations and the Resultant Nutritional Problems/Clinical Manifestations Associated with Cancer
- 8.6 Nutritional Requirements of Cancer Patients
- 8.7 Dietary Management of Cancer Patients and Feeding Problems Related to Cancer Therapy
- 8.8 Cancer Prevention
- 8.9 Let Us Sum Up
- 8.10 Glossary
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8.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- elaborate on how cancer develops,
- enumerate the etiological (risk) factors in the development of cancer,
- describe the metabolic changes, clinical manifestations and complications in cancer,
- classify the different types of cancer depending upon the site of development,
- discuss the different modes of treatment,
- manage the cancer patients in relation to the diet therapy and feeding problems, and
- explain the preventive measures.

8.2 INTRODUCTION

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In this unit, we will discuss the common forms of cancer, how they develop, relevant etiological factors, pathological/metabolic changes and other complications related to cancer. We will also discuss the type of nutrition and diet counseling given to the patients with cancer. In the case of cancer patients there will be a lot of feeding problems related to cancer treatment.

So, we are going to learn more about how to handle these patients. We should remember that each patient will be different and the dietary modifications should be based on individual needs, likes and dislikes, treatment and so on. We should have real patience in handling patients with cancer,

8.3 CANCER

The word 'cancer' comes from the Latin for crab. It refers to many malignant growth or tumor caused by abnormal and uncontrolled cell division.

Body cells, we know, are the basic units of life — each of us has trillions of them. Our cells help us to carry out all functions of life — from the beating of the heart to the throwing of a football. Cancers are new growths of cells in our bodies. Through expression of these properties, it can cause destruction of major organs, and in some cases, life threatening disturbances in body function. Let us see why it happens.

Every cell in the body has the potential to form a new growth. Indeed, this is not a problem just of humans, but, in fact, all living organisms (plants and animals) are susceptible to cancer, simply because all living organisms are made up of cells. Cells, are dynamic — they are constantly in the process of making decisions about what they want to do next. The decision to grow is one such major decision. Cells grow by dividing in half, such that one cell will become two, and two become four (these new cells are called daughter cells). Normally, there are very strict rules as to when a cell can grow or not. These rules are set down by a variety of factors, including all cells around it, various hormones in the body and various external factors to which the cell may respond. One example is growth of bones from infancy to early adulthood.

The cell basically is set loose to divide without its normal control. These genetic events are not inherited through the gametes. There are changes in the somatic cells (other than sperm cells and ova). When this happens, the cell continues to divide, eventually forming a new growth that is what we know as a tumor or neoplasia. This growth is detectable only when this division reaches the point where the number of daughter cells is 1,000,000,000 (one billion).

When a cell is set loose from normal control, it becomes what is known as transformed. Basically, the cell no longer looks like its neighbours in terms of its shape, size, and its internal components. This transformed property is conferred upon all of the daughter cells. That is, all subsequent cells that arise from that initially transformed cell will also look different and grow in an uncontrolled manner. This

is the transmissible nature of cancer — once one cell becomes cancerous, all cells that arise from this abnormal cell also take on this characteristic.

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There are different forms of cancer with different characteristics, requiring different types of treatment. The causes (etiological factors) are also found to be different. So to make it simpler, cancer is a tumor or new growth which has a high growth factor. The new growth may be benign or malignant. A malignant growth can kill a patient if left untreated. A malignant tumor can invade the surrounding tissue and release cells that can be carried to other parts of the body and set up metastasis (growth of malignant tissue that spreads to the surrounding tissues). A benign growth is non- malignant.

Let us learn about development and characteristics of cancer next.

8.3.1 Development of Cancer

Are you interested in knowing how cancer develops? Well here it is. The cancer development is a process involving initiation, promotion and progression.. The first step is initiation when the exposure to a carcinogen allows the carcinogen to enter the cell. This carcinogen then alters the cellular DNA (de-oxyribo nucleic acid). The second step is promotion when there is enhancement of cancer development and the cell begins to multiply uncontrolled. The third step is known as progression when a tumor formation takes place. It may spread to other tissues or organs. Thus the cells released and carried to other parts of the body are called as metastasis.

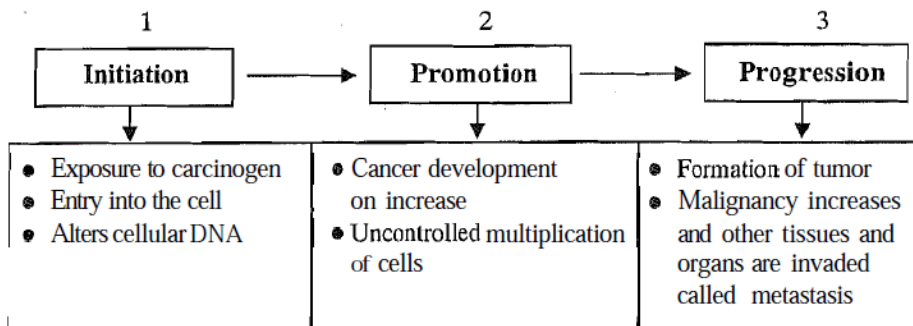


Figure 8.1: Steps in development of cancer

We should remember that cancer development is a process and not a single event. The initiating event may be either chemical or physical. The chemical event may occur when a carcinogen intrudes into the cell and alters the genetic material. The physical event may occur when radiation bombards the cell and alters the genetic material. Whatever is the reason, the protein-making machinery of the cell changes so that the DNA produces an odd structural protein. Then the cells begin to multiply out of control forming a tumor.

What do you understand by carcinogen?

Carcinogen is an agent or a process, which significantly triggers the cell to grow in an uncontrolled manner producing malignant neoplasm (new growth) in a tissue.

There are three main groups of carcinogens. They are ionizing radiation, virus and chemicals/ non- nutritive substances.

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8.3.2 Characteristics of Cancer

The important characteristics of cancer are excessive cellular multiplication, invasiveness and autonomy. The active process of invasion is known as metastasis. Metastasis requires specific surface receptors, enzymes, protein synthesis and use of energy. The process of invasion is described briefly below:

Metastatic cell penetrates the extra cellular matrix that surrounds the tumor and travels through the tissue until it reaches a blood vessel or a lymphatic vessel wall. It dissolves a portion of the wall and then propels itself through the circulating blood. At distant site, the tumor cell again re-attaches to blood vessel wall and repeats the process until it settles down and begins to form a new tumor. Tumor cells gain growth autonomy by either activation of growth promoting antigens or loss of growth inhibitory cancer suppressor genes. The cancer suppressor genes are called antioncogenes.

You may be little confused about the usage of the terms tumor and cancer. Let us be clear about what is tumor and what is cancer? Tumor is a swelling or growth because of an abnormal growth of tissue. Tumors can either be benign or malignant. The benign tumor remains highly localized. On the other hand, the malignant tumor known as cancer is characterized by invasiveness and can form distant colonies elsewhere in the body. Cancer cells are very irregular in shape and their arrangement in tumor tissue is very unruly. Cancer is painless if it does not compress the adjacent organs. Later, it causes pain by invading or pressing the adjacent vital organs.

Another aspect of malignancy is the ability of tumor cells to elude the immune system. These cells may cover up antigens that would otherwise mark them for destruction or they may rid themselves of the cell surface molecules that lymphocytes use to recognize foreign cells. The immune system is largely ineffective.

8.3.3 Identification of Cancer Cells

Cancer cells can be distinguished from normal cells by examining them under a microscope. In a specific tissue cancer cells are usually recognized by the characteristics of rapidly growing cells, a high nuclear to cytoplasm ratio, prominent nucleoli, many mitoses and relatively little specialized structure. The presence of invading cells in an otherwise normal tissue section is the most diagnostic indication of malignancy. Malignant tumors can be classified as:

Carcinomas: these tumors arise from the epithelial lining (the squamous cells) e.g. skin, tongue, breast, stomach, large intestine (the latter three arise from the glandular tissues).

Sarcomas: these arise from the connective tissue e.g. bone, cartilage and

fat.

Melanoma : arise from pigmented layers of skin e.g. pigmented moles.

Gliomas : these arise from the central nervous system e.g. brain and spinal cord.

Reticulo —endothelial tumors: these involve the lymphatic system.

Adenoma : the growth arises from a gland e.g. thyroid gland.

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Unlike carcinogens, which initiate cancer, some dietary components promote cancers. That is, once the initiating step has taken place, these components may accelerate tumor development. Studies suggest that dietary fats eaten in excess may promote cancer development. Specially, linoleic acid, the omega-6 fatty acid of vegetable oil, has been implicated in enhancing cancer development in rats. In contrast, omega-3 fatty acids appear to delay cancer development. We shall learn about this and other factors contributing to cancer next.

8.4 ETIOLOGICAL RISK FACTORS IN CANCER

Cancer risks are climbing due to increasingly sedentary lifestyles and diets that are high in fat and sugar but low in fruits, vegetables, legumes and whole grains. In developing nations, the risk is mounting due to rapid growth of the urban poor who move from rural areas into vast cities. That migration results in a loss of traditional agriculture and dietary habits, plus an increased use of processed foods and drinks has long been known. Obesity is yet another major part of the growing cancer threat.

People are being constantly exposed to many risk factors. First of all we have to remember that etiological or risk factors will be different for different types of cancer. In general, the basic cause of cancers is the loss of control over normal reproduction of cells. There are several factors contributing to this loss of cell control. They are genetic factors, environmental factors, dietary factors, carcinogens, radiation, oncogenic viruses, and others including stress factors.

8.4.1 Genetic Factors

Some cancers are programmed by genes to develop. Gene mutations result from one or more regulatory genes in the cell nucleus. It might be an inherited one but environmental factors also contribute to its expression. Person with a family history of cancer have a greater risk of developing cancers than a person without such a genetic pre-disposing factor.

8.4.2 Environmental Factors

Among environmental factors, smoking, water and air pollution and sun light exposure are known to cause cancer.

8.4.3 Carcinogenic : Dietary Factors

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Dietary constituents can also be carcinogenic. But to what extent diet is one of the contributing factors to cancer development is not known. The incidence of cancers, especially stomach cancers is high in parts of the world where people eat a lot of heavily smoked, pickled or salt-cured foods that produce carcinogenic nitrosamines. Alcohol has also been associated with a high incidence of some cancers, especially cancers of the mouth and throat. Beverages such as beer and scotch may contain damaging nitrosamines, as well as, alcohol. Other beverages such as wine and brandy may contain the carcinogen urethane, which is produced during fermentation.

Nitrosamines have been implicated in the etiology of cancer. Nitrosamines are a broad class of compounds formed from the nitrosation of substituted amides, ureas and guanidines. Nitrosamides are direct acting carcinogens meaning that the activation is non-enzymatic occurring by spontaneous hydrolysis.

A number of laboratory and epidemiological studies have shown the correlation between dietary factor and etiology of specific cancers. Severe calorie restriction in animals has shown to inhibit the growth of most types of tumors. But calorie restriction is not a means to prevent tumor formation. There is a lot of epidemiological evidence to show that there is an association between the high intake of calories by humans and increased risk for endometrial and gall bladder cancer.

Some epidemiological studies suggest that risk for cancer increased with high protein intakes. For instance, cancers of breast and colon occur with greater frequency in the industrialized nations. Some researchers have suggested a possible association between high intakes of total protein or animal protein and the risk of these specific cancers. Both the type and amount of fat are believed to influence tumor formation in animals. A high fat intake in humans has been linked to increased risk for breast and colon cancers. The possible mechanism that has been given is a high fat intake increased intestinal anaerobic bacteria and biliary steroid secretion. These anaerobic bacteria are capable of synthesizing estrogens. The estrogens are believed to be potential carcinogens in mammary tissues. In addition, bile acids are degraded by intestinal bacteria to the secondary bile acids such as deoxycholate and lithocholate. These may act as carcinogens in the colon. Another theory is that trans-fatty acids are more carcinogenic than cis fatty acids.

Next, let us learn about the non-dietary carcinogenic factors.

8.4.4 Carcinogenic: Non-dietary Factors

A large number of agents cause genetic damage and induce neoplastic transformation of cells, they fall into the following categories.

1. Oncogenic viruses
2. Chemical carcinogens

3. Radiant energy

Let us review these factors.

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1. Oncogenic Viruses: Certain viruses that interfere with the functions of the regulatory genes have been identified. These viruses are called oncogenic viruses. Several studies indicate that these viruses are the second most important risk factor. A large number of DNA and RNA viruses have been proved to be oncogenic in animals. Let us learn about these viruses.

DNA viruses : The three DNA viruses found to cause human cancers are EBV, HBV and HPV. What are these? Let us find out.

Epstein-Barr virus (EBV) : EBV belongs to herpes family. It causes Burkitt's lymphoma. It is a tumor of B-lymphocytes. EBV virus alone cannot cause the tumor. In patients with immune dysregulation, EBV causes sustained B cell proliferation. EBV is found to be closely associated with nasopharyngeal carcinoma.

Hepatitis B Virus (HBV) : Hepatitis B virus infection is found to be closely associated with formation of liver cancer.

Human Papilloma Virus (HPV) : HPV gives rise to multiple warts, which are benign squamous papillomas. Some of the warts undergo malignant transformation. Squamous cell carcinoma of cervix has been found to be associated with HPV.

RNA Viruses: All oncogenic RNA viruses are retroviruses. They are of 2 types. They are acute transforming retroviruses and slow transforming retroviruses. Acute transforming viruses include type C viruses and cause rapid induction of tumors in animals. They contain viral oncogenes (virus). The slow transforming retroviruses do not contain V-oncs and are replication competent and cause transformation of the cells slowly.

2. Chemical Carcinogens: Chemicals have been shown to be carcinogenic. Some are naturally occurring components of plants and microbial organisms. Some are synthetic products created by industry. Chemical carcinogens can be classified into two general categories based on the ability of compounds to bind to DNA. Compounds that bind to DNA are genotoxic, whereas compounds that are carcinogenic, but have no evidence of DNA binding are termed epigenetic. Some of the major chemical carcinogens are alkylating agents, acylating agents, and aromatic amines. Aflatoxin B₁, Betel nuts, nitrosamines and amides, vinyl chloride, nickel, chromium insecticide and fungicide are also some of the chemical carcinogens. Tobacco, smoking, drug abuse are also known to cause cancers.

3. Radiant Energy : Radiant energy whether in the form of the ultraviolet rays of sunlight or as ionizing electromagnetic and particulate radiation can transform all cell types in vitro and induce neoplasm in vivo in both human and experimental animals.

Ultraviolet rays: There is ample evidence from epidemiological studies

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that ultra violet rays derived from the sun induce an increased incidence of squamous cell carcinoma, basal cell carcinoma and melanocarcinoma of the skin.

Ionizing Radiation : Electromagnetic (X-rays, gamma rays) and particulate (α- particles, β - particles, protons, neutrons) radiations are all carcinogenic. Even therapeutic radiation has been documented to be carcinogenic.

8.4.5 Stress Factors

Emotions playing a part in malignancy are not a new idea. But these relationships are extremely difficult to measure. The interesting fact is more observations are being made of relationships between cancer and measurable factors of stress. Clinicians and researchers have reported that psychic trauma, seems to carry strong correlations with cancer. Two important physiological causes are assured for this correlation. One is damage to the thymus gland and the immune system. Second is the neuroendocrine effects mediated through the hypothalamus, pituitary and adrenal cortex. Specific studies need to be carried out in this area to confirm the association between stress factors and cancer.

We sum up our discussion on risk factors by highlighting the factors that cause cancer and others that reduce the risk of cancer. Table 8.1 presents this summary.

Table 8.1: Dietary and non-dietary factors in some cancer: Factors that prevent risk of cancers

S. No.	Type of Cancer	Dietary Factors	Non-dietary Factors	Risk Factors
1.	Mouth and pharynx	Alcohol	Smoking and tobacco	Lack of Vegetables and fruits (vitamin C)
2.	Nasopharynx	Salted fish	Tobacco smoking and virus infection	—
3.	Larynx	Alcohol	Tobacco smoking	Lack of Vegetables and fruit (vitamin C and β-carotene)
4.	Oesophagus	Alcohol, very hot drinks, nitrosamines	Tobacco smoking	Lack of Vegetables and fruit (vitamin C and β-carotene)
5.	Stomach	Grilled meats and nitrosamines	Bacterial infection <i>H. Pylori</i>	Lack of Vegetables and fruits (vitamin C and β-carotene) green tea, garlic, selenium
6.	Pancreas	High energy intake, cholesterol, meat, smoked meat and fish	Tobacco smoking	Lack of Vegetables and fruits (vitamin C fibre)

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7.	Gall Bladder	High energy and fat intake		—
8.	Liver	Alcohol, aflatoxins, iron	Viruses: hepatitis B and C	Lack of Vegetables (Selenium)
9	Colon and rectum	Red meat, alcohol high fat and sugar, eggs and iron	Genetic, smoking, ulcerative colitis	Lack of Vegetable and Fruits (vitamin C, β -carotene, fiber)
10.	Lung	Alcohol, saturated fat, animal fat and cholesterol	Smoking tobacco, asbestos, nickel and chromium	Lack of Vegetables and fruits (fibre and vitamin C) vitamin E, selenium
11.	Breast	Excess energy, alcohol, saturated fat.	Late menopause and pregnancy	Lack of Vegetables and fruits, fibre, vitamin C and carotene
12.	Ovary	Total fat, eggs and saturated fat	—	Lack of Vegetable and fruits (vitamin C and carotene)

8.5 METABOLIC ALTERATIONS AND THE RESULTANT NUTRITIONAL PROBLEMS/ CLINICAL MANIFESTATIONS ASSOCIATED WITH CANCER

Several research studies have shown that malignant growth (cancer) is responsible for numerous metabolic abnormalities which are associated with changes in body composition and nutritional status of the patient. These changes can be observed in the form of several clinical complications which are commonly grouped under the broad term of Cancer Cachexia. Let us then discuss in detail about these metabolic changes.

8.5.1 Metabolic Alterations during Cancer

As we have studied earlier, patients with advanced cancer experience profound anorexia, early satiety, changes in the structure/function of organs/glands/body parts, several nutritional deficiencies and weight loss. Although the cause for these symptoms is not clear but they have definitely been found to be associated with the metabolic status of the patient. There are alterations in the energy expenditure, carbohydrate, protein and fat metabolism, acid—base balance, enzyme activities,

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and endocrine functions. Generally, there is an increased metabolic rate. For example, it may be 10 percent greater than the normal level. But, there are variations among patients with gastrointestinal cancers. Some patients may be hyper metabolic, whereas patients with colon and rectal cancer do not show any difference. Therefore, it is evident that there can be variations among patients.

Cancer patients have been shown to have glucose intolerance. This is due to an increased insulin resistance and also reduced insulin secretion. There are also many reports to show that there is an increased rate of endogenous glucose production in cancer patients. This increased production combined with other carbohydrate changes is associated with weight loss. An increased rate of Cori cycling has been reported to occur in cancer patients.

What do you mean by Cori Cycling?

In this cycle, glucose released by peripheral tissues is metabolized to lactate, which is then resynthesized to glucose in the liver. This process is energy consuming because 6 AIP are required for synthesizing only 2 ATP. Therefore, if the tumor cells release more lactate, more energy will be wasted on the resynthesis of glucose. So, it is understood that the Cori Cycling could be one of the significant factors in the development of weight loss.

We should always remember that one of the important and significant concerns in cancer patients is weight loss.

Next, we shall discuss about the abnormalities caused in lipid and protein metabolism. Many research reports have stated that the major portion of weight loss in cancer patients is mainly due to body fat depletion. These include: increased lipolytic (break down of fats) rates caused by decreased food intake, stimulation of lipolysis due to the stress response to illness and release of lipolytic factors produced by the tumor itself. Loss of body fat occurs when both lipolysis and fatty acid oxidation are increased.

Elevated levels of lipid not significant in cancer patients, but may occur in association with certain tumors. The rate of fat oxidation is found to be greater than the rate of carbohydrate oxidation in cancer patients who had lost weight significantly. With regard to protein metabolism the following changes are observed.

1. The rates of whole body turnover increase.
2. Catabolic (breakdown) rates of muscle protein increase with advancing stages of disease leading to weight loss.
3. Decreased plasma branched — chain amino acids.
4. Skeletal muscle mass is reduced.

5. Albumin is the principal secretory protein of the liver. Its depletion is common in cancer and results in hypoalbuminemia.
6. Negative nitrogen balance occurs inspite of sufficient intake.

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Severe metabolic changes can cause progressive weight loss, protein energy malnutrition anaemia and other abnormalities in protein, fat and carbohydrate metabolism. This syndrome is known as cancer cachexia. Apart from these metabolic changes there are other changes, which are believed to be endogenous host responses. Fluid and electrolyte imbalances are seen in advanced cancer patients. Severe vomiting/ diarrhoea and changes affected in the enzyme system could elicit many symptoms. Immunologic functions of the host may be affected leading to progressive malnutrition. These are attributed to the release of mediators derived from cells of the immune system, These mediators are called cytokines.

What are Cytokines?

Cytokines are polypeptides, which influence the proliferation, differentiation, metabolism and activation of cells. There are three areas in cancer that have special relations to these regulatory polypeptides. One relates to the inhibiting cytokines with activated oncogenes, loss of tumor suppressor genes, emergence of drug resistance, and loss of intimate cell to cell contact. Secondly, the role of these regulators in tumor growth and in the development of various cancer metabolic abnormalities. Thirdly, the use of certain cytokines in anti tumor therapy.

Many cancer patients have diminished taste and appetite. Factor affecting taste and smell are quite complicated. There are few studies, which indicate that there is no abnormality in taste perception. Though these variations are existing in the research findings, we should not disregard the loss of taste and appetite in cancer patients. These are also contributory factors for weight loss in them.

Hypercalcemia is one of the most common metabolic complications. The common symptoms are nausea, muscle weakness, excess urine, elevated blood pressure, anorexia, lethargy, confusion and stupor progressing to coma. On the other hand, certain type of tumors reduces calcitriol concentration in conjunction with hypophosphatemia, thereby inducing an oncogenic osteomalacia. Muscle weakness of varying degree and back pain have been the frequent complaint. Having studied about the metabolic aberrations next, let us review the clinical manifestations and nutritional problems associated with cancer.

8.4.2 Clinical Manifestations and Nutritional Problems Associated with Cancer

In the previous section we learnt that cancer results in several changes in the metabolism of carbohydrates, protein, fat, fluids and several micronutrients. Changes in metabolism along with altered structural/functional capacity get

elicited in the form of cancer cachexia, increased morbidity and mortality. Major clinical signs and symptoms which are associated with the nutritional status of cancer patient includes:

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1. Anorexia with progressive weight loss and undernutrition.
2. Taste changes causing depressed or altered food intake.
3. Alterations in protein, carbohydrate and fat metabolism.
4. Increased energy expenditure despite weight loss.
5. Impaired food intake and malnutrition secondary to mechanical bowel obstruction at any level, intestinal dysmotility induced by various cancerous tumors.
6. Malabsorption associated with deficiency or inactivation of pancreatic enzymes, bile salts, failure of food to mix with digestive enzymes; fistulous bypass of small bowel, infiltration of small bowel wall or lymphatics and mesentery by malignant cells. Blind loop syndrome (blockage of small intestine resulting in stasis of the movement of food or digestive secretions) occurring with depressed gastric secretion or partial upper small bowel obstruction leading to bacterial overgrowth; malnutrition induced villous hypoplasia.
7. Protein losing enteropathy.
8. Metabolic abnormalities induced by tumor.
9. Anaemia of chronic blood loss and bone marrow suppression.
10. Electrolyte and fluid problems with persistent vomiting associated with intestinal obstruction or intracranial tumors, intestinal fluid losses through fistulas or diarrhoea.

8.6 NUTRITIONAL REQUIREMENTS OF CANCER PATIENTS – GENERAL GUIDELINES

Cancer we know is a chronic degenerative disease characterized by cancer cachexia which is a stage of marked body dysfunction, general ill health, malnutrition, anorexia and anaemia. Certain other associated symptoms includes xerostomia, nausea, vomiting cheilosis, glossitis which impair food intake. Whatever may be the type of cancer, the nutritional requirements are governed by:

impact of the cancer

impact of the drugs/ treatment on the overall health status of the patient.

In view of the pathophysiology, signs and symptoms, as well as, the impact of

various forms of cancer on the health and nutritional status of the patient the dietary management should aim at meeting the following objectives.

To prevent further tissue catabolism

To meet the increased metabolic demands of the body

To provide relief from the symptoms

To prevent progression and promote recovery from cancer cachexia.

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We will now discuss the nutrient requirements of cancer patients (in general) to fulfill the above mentioned objectives. Our discussions would begin with the calorie needs and proceed to the requirements of various micro and macronutrients. So let us get started with individual nutrients requirements.

Energy: It must be clear to you that cancer imposes increased energy demands because of the hypermetabolic state of the disease process and increased energy requirement to spare proteins for tissue healing and promote weight gain. In view of the inhibitory factors associated with food intake (cancer cachexia); it may not be feasible to promote an intake beyond 2000 Kcal/day. However, by the help of appetite stimulants and / or nutrition support systems (enteral tube feeding) malnourished patients can be inactivated to consume around 30-35 Kcal/kg body weight / day (3000-4000 Kcal/day.). A high energy diet is helpful in inhibiting the side-effects of chemotherapy and cancer cachexia.

Protein: Both the metabolic stress of cancer, as well as, chemotherapy result in increased tissue catabolism. Hypoalbuminemia and anaemia are also very common. Tissue protein synthesis, a necessary component of healing and rehabilitation, requires essential amino-acids and nitrogen. Efficient protein utilization which depends upon protein: energy ratio help to promote tissue anabolism, prevent catabolism and help build up body reserves. An adult patient with average nutritional status will require 80-100 grams protein per day to meet maintenance needs and ensure anabolism. However, a malnourished patient must consume 100-150 gm protein per day to replenish reserves and restore a positive nitrogen balance. Emphasis of course should be laid on the inclusion of high biological value protein rich food sources as milk, eggs, marine foods, and poultry. Renal and liver function tests must be closely monitored under such conditions.

Fat: You may wish to recapitulate from section 8.4 of this unit that during cancer there is enhanced mobilization of free fatty acids from adipose tissues resulting in subsequent depletion of total body fat. Around 15-20% of the modified energy requirements should be provided from fat as they help in making the meals calorie dense and improve palatability. Emphasis should be laid on the incorporation of emulsified fats and vegetable oils particularly those which are rich in medium chain triglycerides. Visible sources of animal fat (pure ghee, lard etc.) and flesh food (red meat) should be restricted in diet. A combination of vegetable oils (olive, coconut, safflower etc.) cream, butter etc. can help in improving taste and providing variety in terms of flavour in different meals.

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Carbohydrate: Adequate amount (60% of total energy) of carbohydrates should be provided. If a very high calorie diet is being given, emphasis may be required on the incorporation of easy-to-digest carbohydrates (mono/disaccharides and starches) so as to make the meals small in volume and energy dense. The fibre intake may need to be curtailed if the patient is suffering from cancer of the gastrointestinal tract or digestive disturbances. However, some patients may experience hyperglycemia. In such situations inclusion of food particularly those which are rich in soluble fibre (pulses and legumes) would be helpful.

Vitamins, Minerals and Phytochemicals : Several vitamins particularly those of the B-group are essential to promote adequate metabolism of energy and protein. Vitamin A, C and E should be provided liberally as they help in reducing the morbidity and mortality due to cancer, (required for tissue synthesis, cell differentiation and for maintaining cell integrity). Among the minerals, zinc and selenium are particularly important and their intake should be slightly increased by giving supplements, The role of phytochemicals (carotenoids, flavonoids, plant sterols, allium compounds, indols, phenols etc.) is gaining importance over the past few years. Incorporation of good amount of fresh fruits/ vegetables preferably with their edible peels, soyabean and certain Indian condiments/ herbs such as turmeric can help in promoting the dietary intake of phytochemicals. The role of epigallocatechin gallate in green tea, curcumin in turmeric, genistein in soya and Colic acid in reducing the morbidity associated with cancer is under investigation.

Fluids: Adequate fluid intake is imperative to replace losses due to gastrointestinal disturbances, infection/fever can also help the kidney's dispose off the metabolic breakdown products from the destroyed cancer cells, as well as, from the toxic drugs used in the treatment. Certain drugs such as cyclophosphamide requires 2-3 litres fluids to prevent cystitis. Adequate intake of fluids/beverages helps in providing relief from xerostomia and other swallowing problems. Menu's should be planned such that they include dishes rich in moisture/ water along with a beverage. Dry meals may not be preferred by most patients

Meal Pattern and Feeding Considerations

The meal pattern and the feeding considerations include the following:

- Meal timings play an important role in ensuring adequate food intake. Cancer patients often complain of decreased ability to eat as the day progresses. This might be because of delayed gastric emptying, decreased production of gastric secretions and mucosal atrophy of the gastrointestinal tract. Small frequent meals with greater emphasis in the morning is recommended.
- Patients with altered taste sensations may benefit by increased use of flavourings and seasonings particularly those which are rich in antioxidants (mint, coriander, turmeric etc.)
- Some patients may experience meat aversions due to chemotherapy. This may necessitate the elimination of red meats (lamb, pork, buffalo, cow etc.) which are stronger in flavour as compared to lean meats (marine foods, chicken

etc.). For such patients alternative sources of protein should be incorporated in the diet. Semi-soft/ full fluid diets should be given to those who experience dysphagia, have lesions in the mouth or oesophagus.

- High energy nutrient dense dishes (cream soups, creamed vegetables, puddings/ soufflé, honey/ jam toppings on fruit, milk shake, custard, sandwiches with cheese/ egg, addition of dextrose, sugar etc. to fruit juice, milk shakes etc.) should be served to facilitate a high energy intake.
- Although oral route is always the preferred form of feeding, it is recommended to use alternative methods of feeding (enteral, parenteral) as a supplement/ substitutes according to the feasibility. Soy-based or milk-based formulas are very popular for long-term enteral feeding particularly for home-based patients. TPN solutions comprising of glucose and a mixture of amino-acids which provide 25-35 Kcal/kg/ day and 1.2 to 2.0 gm protein per kg per day are also frequently utilized to help in maintaining a good nutritional status.

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With a basic understanding on the nutrient requirements, in our subsequent discussions we will learn about the various factors related to different modes of feeding and specialized requirements most suitable for a particular type of cancer/ treatment that would be helpful in the dietary management of patients.

8.7 DIETARY MANAGEMENT OF CANCER PATIENTS AND FEEDING PROBLEMS RELATED TO CANCER THERAPY

We took an overview about the general nutritional requirements of cancer patients. However, in order to be successful in maintaining an optimum nutritional status of the patient it is equally important to feed the right type of diet through an appropriate method and route by keeping in mind the details of the disease and mode of treatment.

We shall now discuss about the different types or modes of cancer treatment and the feeding problems associated with the treatments. While reading the details mentioned below we must also remember that the line of treatment for the cancer patients will depend on the site of cancer development. Currently the cancer therapy includes three major types of interventions namely surgery, radiation therapy and chemotherapy.

Differentiation therapy and adoptive immunotherapy are other cancer therapy strategies. Cancer therapy often involves combined significant nutritional problems, as well as, feeding problems which may arise not only from the malignant condition but also from specific treatment undertaken to control the neoplastic process. So our discussion will now begin with a review on different cancer therapies, the feeding problems related to these therapies and the dietary management.

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8.7.1 Surgery

Surgery is generally conducted in the absence of metastasis i.e when a tumor is localized. Nutrition support would vary depending on the area being operated and its association with the digestion/absorption of food. For instance, surgical removal of the tumor of appendix may not call for any major demands on the nutritional support whereas, surgical removal of a part of liver/pancreas may require specialized feeds and feeding support techniques (enteral/total parenteral). We shall now discuss methods of feeding required during surgical resections of different tissues.

a) Head and Neck Tumor

Treatment mostly involves combination of surgery and radiation. Chemotherapy is also used in some cases, We will learn about these therapies in a little while from now. But, remember radiation induces loss of taste (mouth blindness) and dry mouth (xerostomia). Injury to teeth may also occur. Surgery may include partial or total glossectomy (partial or total surgical excision of the tongue) and mandibulectomy (re-section of the lower jaw). Sometimes they may do a surgery of the hard or soft palate, or of the soft tissues of the lower face and neck. These procedures add to the difficulties in chewing and swallowing. Aspiration of food on swallowing may be another serious problems.

Nutritional Support Management

Tube feeding is usually started. If tube feeding is not possible, parenteral nutrition through peripheral vein or through the central vein can be given.

Before treatment, foods that are attractive with pleasant aroma can be given. Foods should be of high energy value. We should try our best to increase their total food intake. After treatment, nutritious food formulas can be administered by mouth if the patients are able to swallow. If they find it difficult, nasoesophageal tube feeding can be started. For long-term maintenance of patients requiring such support, gastrostomy tubes should be inserted. Some patients are at serious risk of aspiration of regurgitated food (tendency to vomit). This danger is reduced by placing the tip of the tube in the small bowel and infusing formula by slow drip. Care should be taken to regulate the flow rate.

Discharged patients who require long-term liquid feeding at home should be given information about both commercial and homemade formulas. Whatever be the type of feed, it should be nutritionally adequate and have sufficient bulk-forming materials to prevent constipation.

b) Oesophageal Carcinoma

Management of patients with oesophageal carcinoma includes surgery, radiation and combination chemotherapy. Radiation to the lower neck can induce

oesophagitis, fistulas and haemorrhage may also occur due to re-growth of the cancer. Chemotherapy may induce nausea, anorexia, sore mouth, and odynophagia. All these inhibit food intake and decrease the acceptance of tube feeding.

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Surgical treatment usually involves total or distal oesophagectomy (procedure to remove a portion of the tongue). Easy regurgitation, rapid satiety, decreased rate of gastric emptying of solid food, diarrhoea and steatorrhoea are common results of this surgery. Weight loss is another great problem. Preoperative parenteral feeding is indicated to improve nutritional status. This is found to reduce postoperative complications. Oral or tube feeding is often inadequate to meet the nutritional needs in the period of radiation and chemotherapy because of interference with the feeding programme, nausea, pain or combination of all these. Once the oral intake by the patient is normal, the dietary prescription should provide for frequent meals high in carbohydrate and adequate in protein and fat. In some cases, steatorrhoea occurs with increased frequency and foul smelling stools along with abdominal discomfort. For these patients partial substitution of long-chain triglycerides (LCT) by medium chain triglycerides (MCT) can be tested and may be helpful.

Postoperative stricture (narrowing of a passage due to scar tissue or tumor) may occur and requires dilation. Oral or tube fed liquid formulas can be given to assure adequate intake until the stricture is overcome.

Carcinoma of the oesophagogastric junction creates similar problems like those described above. Production of gastric juice may be reduced there by resulting in decrease of vitamin B₁₂ absorption.

c) Gastric Cancer

Surgical treatment is a very common mode of treatment in gastric cancer. Radiation and/or chemotherapy are given for patients with resected but residual localized disease.

Removal of most of the residues of the stomach reduces its functions such as secretory, diluting and digestive. This will definitely result in physiological and nutritional problems. These problems may vary from mild to severe depending on the extent of resection, the individual patient response, the appropriateness of the intervention and the postoperative care.

During the post-operative period, when food is ingested some patients show various signs and symptoms known as 'dumping syndrome'. This syndrome develops with varying severity. Usually the signs and symptoms occur within 15 to 30 minutes following ingestion of a meal. The gastrointestinal signs and symptoms include abdominal bloating, cramping and diarrhoea. These symptoms are more pronounced shortly after a meal. Another set of symptoms that usually occurs two hours after eating is also characterized by sweating, tachycardia (heart rate above 100 per minute) and faintness. Mental confusion may also occur. These symptoms

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are related to the discharge of catecholamine. This catecholamine discharge is mediated by hypoglycemia induced by the insulin response to the rapid entry of the meal into the upper small bowel. Malabsorption of fat occurs. Deficiencies of iron, calcium and fat-soluble vitamins may also occur due to malabsorption. The beneficial effect of somatostatin, especially its analogue — 'Octreotide' has been reported in the treatment of dumping syndrome. Some patients benefit from long-term use but many are unable to tolerate the drug due to diarrhoea. Let us learn about the nutritional management of dumping syndrome next.

Nutritional Support Management

The 'dumping syndrome' can be greatly minimized or prevented by adhering strictly to an antidumping diet. In general, such a diet is high in protein, has adequate fat, is low in total carbohydrate, particularly simple carbohydrates restricted in fluids at meal time. Small frequent meals, say six times per day should be served. Patient should be discouraged to lie down immediately after the meal, instead, encourage them to be in a reclining position for a short period of time. The use of soluble fibre such as pectin derivative has been reported to prolong gastric emptying, to decrease dumping and to minimize the fall in blood sugar.

If steatorrhoea (loss of fat in the stools) is significant, replacement of a portion of LCT (long-chain triglycerides) with MCT (medium chain triglycerides) will be helpful, The patient will be able to tolerate this better. Pancreatic extract can also be tried to rule out luminal pancreatic enzyme insufficiency. Insufficiency of pancreatic enzyme(s) may result from rapid entry of food and fluid into the upper small bowel or from a pancreatic secretory defect or from both.

Deficiencies of vitamins and minerals can be prevented by adequate oral administration of iron with ascorbic acid and by supplementing both water-soluble and fat-soluble vitamins. Monthly injections of 100 micrograms of vitamin B12 are required because the extensive gastric resection will eventually result in vitamin deficiency. Milk is found to be poorly tolerated by these patients. They can be asked to drink milk in small amounts frequently over the day (or to drink lactase treated milk if available) or to use yoghurt as tolerated. In case these approaches are of no use, the more soluble calcium salts should be given in divided doses. At least one gram of calcium should be given for a day.

Antiemetics (drugs to prevent nausea or vomiting) are used in treatment of chemotherapy induced nausea and vomiting. Antiemetics become absolutely necessary to help better adherence to therapeutic programmes and better intake of food and fluids.

Weight loss seen in these patients is mainly due to poor food intake. In addition to this, discomfort associated with eating may result from esophagitis secondary to bile regurgitation, anorexia associated with depression or the side effects of drugs and/or radiation. Hence, a careful diet history, conform an adequate basis for dietary modifications.

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If the above prescribed dietary management does not prevent the dumping syndrome or there is no adequate food intake to maintain gain body weight, slow-drip tube feedings of a complete formula is recommended. Because of the slow entry of food into the upper intestine by this technique, dumping is not likely to occur. Such feedings may need 10 be given only during the period of chemotherapy. This will help to improve the appetite. When patients remain seriously anorexia following chemotherapy, tube feedings at night are helpful.

d) Pancreatic Cancer

This is a condition often associated with abdominal pain, anorexia, nausea, vomiting and weight loss. Eating may aggravate pain. There may be digestive enzyme deficiency. Malabsorption combined with anorexia contributes to progressive weight loss. Bile insufficiency can occur if there is any obstruction in the common bile duct. This may reduce the intestinal absorption of vitamin K. Pancreatic carcinoma is an aggressive disease, and by the time it is diagnosed, most patients are at a stage in which curative treatment is not possible.

Surgical resection is the only chance of cure at present. There are numerous problems interfering with normal food intake. Fat malabsorption, decreased glucose tolerance and hyperglycemia are common. Under such circumstances, the nutritional management of pancreatic cancer patient is very important which is explained next.

Nutritional Support Management

When there is deficiency of exocrine pancreatic secretions, adequate amounts of pancreatic extract are helpful. It should be administered with all meals and snacks specially when there is severe fat malabsorption syndrome. MCI are more efficiently absorbed than the LCT in the absence of pancreatic enzymes and decreased bile salts. Glucose oligosaccharide may also help to increase the calorie intake and absorption among patients with pancreatic insufficiency. These are relatively short-chain glucose polymers and can be hydrolyzed to glucose by the brush border enzyme sucrase x - dextrinase. This white powdery material is not sweet and may be used in variety of ways to supplement intake.

e) Other Surgical Procedures

Major resection of the small bowel is not common. Resection of the ileum leads to certain physiological and nutritional problems. Colectomy (partial, total and diverting) etc. resection of the right colon with the ileocecal valve and a portion of the distal ileum may result in watery diarrhoea. Since only a small segment of distal ileum usually is sacrificed, vitamin B₁₂ deficiency is not likely to occur.

In total proctocolectomy (surgical removal of the rectum together with part or all of the colon), an ileostomy with stool collected in an external pouch is used for the patients. Loss of water and sodium through the ileostomy is significant

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during the first 10 days of postoperative period. These patients usually lose 300 to 600 ml of water, 40 to 100 mEq of sodium and 2.5 to 10 mEq of potassium daily. Patients can be managed with increased fluids and salt administration. Various other procedures have been designed and advocated depending on the conditions of individual patient. Eating patterns and dietary recommendations also depend on the patient's condition.

Sometimes a major portion of the large bowel is taken out of continuity by a diverting procedure. As a result of this, an inflammatory process termed diversion colitis can occur in this area. The common symptoms associated with the diversion colitis are abdominal cramping with mucoid and blood discharge. Studies have shown that infusion of a salt solution containing short-chain fatty acids (SCFA) into the rectal remnant results in healing.

The discussion so far focussed on surgery as a therapy and the nutritional support management after surgery for cancer patients. Next, we shall look at the radiation therapy and its nutritional support management.

8.6.2 Radiation Therapy

Radiation therapy is one of the options for the treatment of various tumors. During the administration of radiation patients experience acute radiation toxicity. This toxicity is manifested as nausea, vomiting and diarrhoea. This type of toxicity will subside usually within weeks of ending the radiation therapy (RT). Chronic, late gastrointestinal complications occur and may cause major morbidity and mortality. After 2 to 3 weeks of RT, abdominal cramping and watery diarrhoea may occur. Weight loss is common. Malabsorption of water, fats, bile salts, carbohydrates, calcium, magnesium, iron and vitamin B₁₂ occur during RT. Several factors contribute to the malabsorption that occurs in radiation damage. These include.

1. Decreased available absorptive surface area due to radiation damage.
2. Chronic lymphatic obstruction causing steatorrhoea and protein loss,
3. Secondary disaccharides deficiency
4. Bile salts malabsorption leading to choleric diarrhea.
5. Rapid intestinal transit.

Nutritional Support Management

For managing these patients on radiation therapy, the following measures can be under taken:

1. Administration of broad spectrum of antibiotics for bacterial overgrowth.
2. Diet low in fat and lactose.

3. Anti diarrhoea medications and anticholinergic and antispasmodic preparations,

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Diet therapy can play a major role in controlling symptoms and assuring adequate nutrition. In addition, enteral and parenteral nutrition can be used in severe cases. Enteral feedings with solutions containing amino acids or partially digested protein and very low fat content can be given. Studies have indicated that those patients who received only such feeding during RT show less diarrhoea and weight loss. Though this type of feedings is useful, there are some practical difficulties. Therefore, it is advisable to use such enteral feedings for only those patients who develop severe/ acute toxicity. Total parenteral feedings should be reserved only for those patients malnourished before starting a course of RT.

Some patient may also suffer from chronic radiation enteritis

Chronic Radiation Enteritis

First of all the nutritional status of the patient should be assessed. Assessment of selected biochemical parameters, radiographic studies of the intestinal tract, and absorption studies should be carried out. Dietary management includes restriction of fat, fiber, lactose and glutens. This may help in the symptomatic relief and improved nutrition. In patients with severe radiation enteritis who are unable to maintain their weight with oral or enteral diets, TPN is absolutely essential. It helps them to gain weight and improve overall nutrition.

8.7.3 Chemotherapy

Chemotherapy results in lot of side effects. This is because the drug effects are not specific to cancer cells alone. Even the host cells will be affected by chemotherapy. The severity of these side effects depends on factors such as the type of drug, dosage, duration of treatment, patient's nutritional status and individual susceptibility. Major areas affected by side effects are alimentary tract and bone marrow. In some instances, major effects occur on renal tubules and also in hepatic, cardiac, pulmonary and nerve cells.

Nausea and vomiting may occur acutely and in some cases it may be delayed for 24 hours or more after receiving chemotherapy. Factors influencing emeses — 'vomiting' include patient sensitivity, type of drug, dosage and frequency as well as route of administration.

The nitrogen equilibrium present before chemotherapy changes to negative nitrogen balance. Protein turnover, synthesis and catabolism decrease with the drug therapy despite continuing intravenous nutrition support.

A thorough study of the more commonly used chemotherapeutic agents, their mechanisms of action and potential side effects that influence nutritional status should be undertaken. Let us review the nutritional support in chemotherapy next.

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Nutritional Support Management

Antiemetics are used in the treatment of chemotherapy induced nausea and vomiting. Antiemetics become absolutely necessary to promote enhanced adherence to therapeutic programmes and better intake of food and fluids.

8.8 CANCER PREVENTION

A continuing and urgent need is there to take all steps to prevent cancer. Cancer rates are set to increase another 50 percent by the year 2020. Based on the current knowledge and research evidence, certain guidelines are recommended for cancer prevention. These are highlighted next.

8.8.1 Guidelines for Cancer Prevention

Tile guidelines for cancer prevention focus on tlie following:

1. Include plant-based diet, limiting red meat in particular.
2. Limiting fat consumption, especially saturated fat. Total fat consumption should not exceed 30% of total energy intake.
3. Avoiding or limiting alcohol.
4. Reducing intake of energy rich foods.
5. Include more of vegetables and cut down cereals.
6. Increase fiber intake to 20 to 30 grams per day.
7. Include a variety of vegetables and fruits daily in your diet.
8. Minimize consumption of salt-cured, salt-pickled and smoked foods.
9. Control body weight and prevent obesity.

And other additional recommendation is varying food choices. Instead of eating the same type of food, try and include a variety of other foods. This will help to dilute whatever is in one food with what is in the other foods.

Next, we shall review research finding related to specific foods and nutrients which play an importants role in cancer prevention.

8.8.2 Research Findings Related to Cancer Prevention

In this section we shall discuss some of the research findings related to cancer prevention. Many of the natural, cancer-fighting phytochemicals are found in citrus fruits, cruciferous vegetables such as broccoli, brussels sprouts and cauliflower.

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Alpha Lipoic Acid (ALA), a potent antioxidant, has been used in the prevention and treatment of cancer. It is found in foods such as potatoes, carrots, yam and red meat, and is synthesized in the body. Astaxanthin, a fat-soluble carotenoid has been found to have the ability to fight cancer. Flavonoid, a colour pigment is found to possess anti-inflammatory compounds and protect against cancer. The rich food sources of this flavonoid are garlic, carrots, brinjal, grape fruit, onions, oranges, red cabbage, tea and tomatoes. Low blood levels of glutathione are associated with problems such as heart disease and cancer. Foods rich in this, such as asparagus and avocado can be included more often in our diet.

Vitamin C is another well researched water soluble vitamin, which is found to destroy cancer-causing agents. Food sources include broccoli, brussels sprouts. Consumption of these vegetables has been found to increase in the Indian dietary. Capsicum, lime, orange, papaya and amla are other rich sources. Vitamin E, a fat-soluble vitamin is another antioxidant which can be used for cancer prevention. Food sources of this vitamin are almonds, soybeans, spinach, sunflower seeds, sweet potatoes, walnut, wheat germ, and whole-wheat flour. Laboratory studies suggest that omega - 3 - fatty acids found in fish, walnuts, soybean oils many help protect against cancer. Dietary fiber is found to exert a protective effect against colon cancer by several mechanisms.

It may bind with some of the potential carcinogens thereby reducing the exposure of epithelial surfaces to potential carcinogens. It influences in decreased formation or enhanced excretion of potential carcinogens. It influences intestinal flora with decreased degradation of bile acids and neutral sterols and diluting potential carcinogen in the bowel. Unrefined carbohydrates such as whole wheat, brown rice contain dietary fiber, which is linked to lower colon cancer risk. The impact of nuts on the cancer is less certain. The studies link regular consumption of nuts, seeds and beans with lower risk of prostate cancer. Laboratory research has identified at least five natural phytochemicals in nuts that seem to offer protection against cancer development although lots remain to be learned about this process.

The goal is a balanced, mostly plant-based diet with plenty of fruits, vegetables, whole grains, beans and nuts in the prevention of cancer. Antioxidants play a very important role in treating people with different types of cancers.

8.8.3 Role of Antioxidants in Cancer Prevention

What are antioxidants? To put it simply, antioxidants are important naturally occurring nutrients, (vitamins, minerals) which help to protect body from certain types of cancers. Vitamin A, vitamin C and vitamin E are well proved antioxidants in treating cancers such as gastrointestinal, cervical and breast cancers. Also, antioxidants decrease the risk of cancer mortality.

These free radicals, we learnt are highly unstable and steal components from other cellular molecules, such as fat, protein, or DNA, thereby spreading the damage. This damage continues in a chain reaction, and entire cells soon become

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damaged and die. This process is called peroxidation. Peroxidation is useful because it helps the body destroy cells that have outlived their usefulness and kills germs and parasites. However, peroxidation, when left unchecked, also destroys or damages healthy cells. Antioxidants help prevent widespread cellular destruction by willingly donating components to stabilize free radicals. More importantly, antioxidants return to the surface of the cell to stabilize rather than damage other cellular components.

Lycopene is a carotenoid which colours fruits and vegetables and is most abundantly present in the prostate gland. Studies have shown that this carotenoid reduces the risk of various deadly cancers including cancers of the prostate, colon and rectum. High intake of lycopene by patients with prostate cancer has caused a regression in the disease and decreased the malignancy.

Fat-soluble vitamin A compounds include retinol, retinal and retinoic acid. This group is vital for eye and retinal function, protects the mucous membrane and reduces the risk of infection. Therefore, it is called an immune enhancer and reduces the risk of cancer. Apart from reducing the cancer mortality, it helps in treating cancer patients who have had surgery to remove primary tumors.

Vitamin A and carotenoids antioxidant and immune stimulatory property have developed synergistic cancer treatment application. Vitamin A levels decrease during chemotherapy. Hence, additional intake of vitamin A is recommended during chemotherapy.

Vitamin C based on research evidence plays an important role in the prevention and treatment of cancer. Its anti cancer properties are:

- Scavenging cancer causing free radicals such as hydrogen peroxide to prevent lipid peroxidation,
- Neutralizing carcinogenic chemicals,
- Generating potent antioxidant vitamin E,
- Enhancing lymphocyte function and rapid mobilization of phagocytes,
- Potent antiviral and antibacterial activity,
- Enhancement of immunoglobulins IgA, IgM,
- Modulation of interferon synthesis, and
- Increasing synthesis of prostaglandin.

Research studies reveal that, higher the vitamin C intake, lower the level of mortality for all cancer patients. Vitamin C reduces the risk of gastrointestinal cancer, breast cancer and liver cancer.

Various other studies have proved vitamin E to be effective in decreasing the risk of colon cancer, inhibiting breast tumors, reducing the severity of liver cancer and also restoring the cellular immune function in patients treated with radiotherapy. Mentioned below is a list of cancer preventive nutrients/food

compounds and their food sources.

Foods Related to Cancer Prevention

Active ingredient	Food sources	NOTES
Phytochemicals	Fruits, broccoli, cauliflower, etc.	
Alpha Lipoic Acid (ALA)	Potatoes, Spinach	
Anthaxanthin & Flavonoids	Fruits, vegetables, grains	
Lycopene	Fruits and vegetables (especially tomatoes)	
Flavonoids	Garlic, Carrots, Onions, Brinjal, Red cabbage, tomatoes grapefruit, oranges, tea.	
Glutathione	Asparagus and avocado	
Fiber	Whole wheat, Brown Rice, Nuts, Fruits and Vegetables.	
Vitamin A	Butter, dairy products, fish oils, carrots, egg, yolk.	
Vitamin E	Almonds, soybeans, spinach, sunflower seeds, sweet potatoes, walnuts, wheat germ, whole wheat flour.	
Selenium	Seafoods	

To sum up, low plasma levels of all major essential antioxidants (Vitamin A, C, E and beta carotene) is associated with an increased risk of cancer mortality. Hence, the plasma levels of these antioxidants have to be increased to reduce the risk of cancer by providing diets rich in these antioxidants. Selenium is an essential micronutrient and the best source of selenium is seafood.

It is toxic in extremely high doses (2.5 to 3.0 g/ day), but normal level of 50-200 micrograms per day is safe. It is a potent antioxidant. Selenium reduces the risk of breast cancer and inhibits colon cancer due to changes in prostaglandin synthesis. The antioxidant property protects the body against environmental and chemical sensitivities and their immune functions enhance the body's antibacterial and antiviral defenses.

With a review on antioxidants and their role in cancer prevention we end our study on nutrition, diet and cancer.

8.9 LET US SUM UP

In this unit, we studied about what is cancer, how it develops and how cancer cells differ from normal cells. We also learnt about the characteristics and types of cancer. Then we focused on the major etiological (risk) factors associated with causation of cancer.

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These, as you would recall, include genetic factors, environmental factors, dietary and non-dietary factors, as well as, the stress factors. After this, we moved on to the discussion on various metabolic abnormalities associated with cancer; referred to as cancer cachexia.

Finally, we looked at the nutritional problems and clinical manifestations associated with cancer, Our last section focused on the therapies for different types of cancers along with their dietary management and feeding problems.

Lastly, we dealt with a few guidelines pertaining to cancer prevention, the research findings and the role of antioxidants in preventing cancer.

8.10 GLOSSARY

Anastomosis	: the intercommunication between two or more vessels or nerves, as the cross communication between arteries or veins.
Anti-emetics	: drugs that prevent or relieve nausea and vomiting.
Anorexia	: loss or deficiency of appetite for food.
Benign tumor	: mass of abnormal tissue that is encapsulated and does not infiltrate adjacent tissue.
Cancer cachexia	: a stage of marked body dysfunction, general ill health, malnutrition, anorexia and anaemia.
Carcinogen	: any cancer producing substance/agent.
Chemotherapy	: the use of certain drugs to treat a disease as distinct from other form of treatment, such as surgery.
Cheilosis	: inflammation and cracking of the lips.
Coma	: a sleep-like state in which a person is not conscious.
Cori Cycle	: the conversion of glucose to lactate in the skeletal muscle on exertion and then lactate to glucose in the liver.
Cytokines	: non-antibody proteins secreted by inflammatory leukocytes and some non-leukocytic cells that act as intercellular mediator.

Dumping syndrome	: a syndrome that occurs when food moves too fast from the stomach into the small intestine.
Dyshagia	: difficulty in swallowing.
Enteral feeding	: a way to provide food through a tube placed in the nose, stomach, or the small intestine.
Ectopic hormones	: a peptide hormone released from its usual site or from a neoplasm of that tissue.
Fistula	: an abnormal connection between two organs, or between an organ and outside of the body.
Gametes	: the reproductive cells in multicellular organisms.
Glossitis	: inflammation of the tongue.
Glossectomy	: the surgical removal of all or part of the tongue.
Hypocalcemia	: excessive calcium in the blood.
Ileostomy	: a surgically made fistula between ileum and anterior abdominal wall as a permanent artificial anus when whole of large bowel has to be removed.
Interferon	: a protein produced naturally by the cells of our bodies and increases the resistance of surrounding cells to attacks by viruses.
Malignant	: mass of abnormal tissues that is not encapsulated and infiltrates adjacent tissue.
Melanocarcinoma	: a malignant tumour of melanocytes predominantly occurring in the skin.
Neoplasm	: an abnormal growth of tissue which may be benign or malignant.
Odynophagia	: severe pain on swallowing due to a disorder of the oesophagus.
Oesophagitis	: an inflammation of the oesophagus.
Oncogenes	: genes that promote cell growth and duplication. They may undergo changes that activate them, causing cells to grow too quickly and form tumors.
Oncogenic osteomalacia	: tumour-induced osteomalacia.

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8.11 CHECK YOUR PROGRESS

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- 1). Define the following terms:
 - a) Carcinogen:
 - b) Tumor:
 - c) Metastasis:
 - d) Oncogenic virus:
 - e) Anti oncogenes:
- 2). What is cancer? Briefly discuss the steps involved in cancer development.
- 3). How can cancer cells be distinguished from normal cells?
- 4). Enumerate the risk factors associated with the etiology of cancer. Briefly discuss dietary factor known to cause cancer.
- 5). Discuss the guidelines for cancer prevention.
- 6). What are antioxidants? Discuss the role of antioxidants in cancer prevention.
- 7). Why vitamin A is called an immune enhancer? Why additional intake of vitamin A is recommended during chemotherapy?
- 8). Discuss the anti-cancer properties of vitamin C.

9

**NUTRITIONAL CARE IN WEIGHT
MANAGEMENT**

NOTES

STRUCTURE

- 9.1 Learning Objective
- 9.2 Introduction
- 9.3 Weight Imbalance — Prevalence and Classification
- 9.4 Guidelines for Calculating Ideal Body Weight
- 9.5 Obesity
- 9.6 Management of Obesity
- 9.7 Underweight
- 9.8 Let Us Sum Up
- 9.9 Glossary
- 9.10 Check Your Progress

9.1 LEARNING OBJECTIVE

After studying this unit, you will be able to

- explain the importance of maintaining a desirable weight throughout the life,
- enumerate the guidelines for calculating the ideal body weight, and
- describe the causative factors, prevention and treatment of various conditions related to weight management (such as obesity, underweight).

9.2 INTRODUCTION

Weight management has assumed a lot of significance in the present scenario with increasing affluence, abundance of convenience foods and lack of physical activity. There is nothing mysterious about what causes people to be overweight. Excess weight is the result (in long term, consistent consumption of much more calories than you are able to expend, irrespective of the etiology. The emphasis in treating obesity currently has shifted from mere 'weight loss' to 'weight management' which implies that efforts should be directed towards attaining the best possible weight

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(desirable body weight) in relation to overall health.

We have already learnt in the previous unit that obesity is one of the important factors in the causation of certain types of cancers and many other diseases like arthritis and cardiovascular disease. In this unit you will come across the significance of maintaining appropriate weight for preventing certain other types of chronic degenerative diseases. We will also learn about the various approaches the overweight, the obese and the morbidly obese individuals need to consider for attaining desirable weight and more importantly, how you can prevent putting on weight in the first place.

Too much either side from the appropriate range of body weight increases our risk of health problems. Just as overweight as the result of positive energy balance, underweight results when the energy balance is negative. Obsession with slimming, especially in the adolescent age group may result in eating disorders like anorexia nervosa and bulimia nervosa. How to cope with problems of underweight? This is the focus of the second part of the unit.

9.3 WEIGHT IMBALANCE - PREVALENCE AND CLASSIFICATION

You are aware that obesity is one of the major public health problems of the world, Earlier a problem of the developed nations, it is now increasingly afflicting our country. Maintenance of a fairly constant body weight is of vital importance in increasing the life expectancy, as well as, quality of life of individuals and communities. It is a fact that exaggerated weight fluctuations on either side (underweight or overweight) of a desirable range of weight lead to an increase in the Inorbidity/mortality rate.

Prevalence

WHO (1998) estimates that in developing countries about 245 million adults are moderately underweight and 93 million severely underweight. At the same time, there are over 200 million adults worldwide who are moderately or severely overweight, of whom 58 million are in developing countries. Overall it appears that in any country — developed or developing — prevalence of malnutrition (underweight and overweight) is about 50%. The WHO report states that the growth in the number of severely overweight adults is expected to double that of underweight adults during 1995-2025.

Now, let us have a look at the situation in developed countries. As per the report of National Health and Nutrition Examination Surveys (NHANES) conducted by the Centers for Disease Control and Prevention, 2002, currently 64.5% of U.S. adults age 20 years and older are overweight and 30.5% are obese. These figures stood at 46.0% and 14.4%, respectively during 1976-1980 implying thereby that there has

been a consistent increase in prevalence of obesity.

The increase in prevalence of obesity among children also is a cause of great concern. Estimating true prevalence is difficult because of the lack of agreement of different bodies in defining obesity in children and adolescents. However, data from 79 developing countries and a number of industrialized countries suggests that, by WHO standards, about 22 million children under 5 years old are overweight worldwide (WHO, 1998). In the USA, the percentage of overweight children (aged 5-14 years) has risen from 15% to 32% during the last 30 years.

Obesity in India

The results of a recently concluded study on the prevalence of obesity in urban Delhi by the Nutrition Foundation of India has projected that nearly one third of the males and more than half of females belonging to the 'upper middle class' in India are currently overweight with even higher prevalence of abdominal obesity. Converting these fractions to numbers, approximately 40-50 million subjects belonging to the upper middle class are overweight today in India. If present trends continue, the situation can get worse even within a decade and overweight can emerge as the single most important public health problem in adults. This is despite the fact that one fourth of our country's population still falls below the poverty line. So we have learnt that

338 million adults in developing countries are underweight while 58 million are overweight.

During the past 30 years there is 18.5% rise in overweight and 16.1 % rise in obese Americans while there is 17% rise in percentage of overweight children (5-14 years).

Roughly 40-50 million Indians belonging to the upper middle class are over weight. With increasing numbers every years, obesity could become a public health problem in adults.

Classification

Obesity is defined as a condition with accumulation of excess body fat. Do you think that a measure of how much fat a person has in its body would serve as a tool for classification of obesity? No, because the measurement of direct body fat is difficult, so we use an indirect method, a ratio called the Body Mass Index (BMI) also termed Quetelet's index. This ratio estimates dependence on frame size and provides the most useful method of measuring obesity in populations. BMI can be calculated from the following equation:

$$\text{BMI} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

where kg = kilogram, m = metre

By this method, various grades of obesity, normal and underweight can be known. Our BMI value near 18.5 to 24.9 is the ideal value for us to remain healthy and enjoy a quality life. Table 9.1 presents the weight status according to the BMI range.

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Table 9.1: Weight status according to BMI range

Weight Status	BMI Range
Underweight	< 18.5
Normal	18.5 to 24.0
Preobese	25 to 29.9
Obesity (Grade I)	30 to 34.9
Obesity (Grade II)	35 to 39.9
Obesity (Grade III)	40 and above

Interestingly it has been found that for a given BMI, Indians have more body fat than other ethnic groups, both within and outside Asia. This relative increase in adiposity in Indians has led to the suggestion that the BMI cut-off for non-communicable diseases such as obesity should be reduced for Indians to about 23 kg/m² or lower. In other words refer to it as a public health action point at a BMI of 23 kg/m².

So then adiposity can be inferred from the BMI, however, this may not be sufficient to fully explore relationship between body fat and alterations in human health. Besides, the BMI, the location of the fat in the body is also important. In this context the measurement of waist and hip circumference and their ratio is crucial. The waist to hip ratio is described next.

Waist to Hip Ratio (WHR)

Two individuals who have the same BMI and the same total body fat may have different abdominal fat mass. Abdominal fat accumulation increases the risk of a number of chronic degenerative diseases.

The waist-hip ratio (waist circumference divided by hip circumference) therefore is a simple method for distinguishing between fatness in the lower trunk (hip and buttocks) and fatness in the upper trunk (waist and abdomen area). A WHR of >1.0 for men and >0.85 for women is an indicator of abdominal obesity. Lower trunk fatness (i.e. lower waist to hip ratio) is often referred to as 'gynoid obesity'. Upper trunk or central fatness (higher waist to hip ratio) is called 'android obesity'.

How do you make these measurements? Waist and hip measurements are taken on standing posture and the unit used is centimeter. Waist is measured around the navel and hip is measured around its broadest part. You may have realize that in addition to having a normal BMI value, it is also important for us to have

a normal WHR to have a healthy, as well as, attractive body.

Obesity in Children

It is difficult to measure overweight or obesity in children and adolescents because they grow and mature at different rates. Weight status in children can be classified based on percentile curves for BMI for age as per the WHO Technical Report (1995). presents the weight status based on percentile curves of BMI for age.

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Weight Status	BMI for age
Underweight	< 5 th percentile
At risk of overweight	> 85 th to <95 th percentile
Overweight	> 95 th percentile
Overweight or at risk	> 85 th percentile

The latest BMI for age percentiles for boys and girls 2 to 20 years have been published by the United States National Center for Health Statistics (NCHS) in collaboration with the National Center for Chronic Diseases Prevention and Health Promotion in the year 2000 which may be applied to affluent Indian children also.

Now that we are clear about how to classify obesity and underweight, we will further look at the guidelines for calculating the ideal body weight.

9.4 GUIDELINES FOR CALCULATING IDEAL BODY WEIGHT (IBW)

What is the ideal body weight for me? Am I obese? Am I underweight? These are the questions that must have come to your mind as you stepped into your teens. The three main factors that determine your ideal weight are your age, sex and height. You have already learnt in the previous section about two important indices of body weight that help in the evaluation of your current weight status, i.e., calculation of BMI and the measurement of WHR. In addition, you have standard height and weight charts for adult males and females that help you to determine the range of weight which is appropriate or desirable for you at a given height.

You must appreciate that the best weight for a given individual's height, age, bone structure and muscular development is not known exactly. A lot of people continue to gain weight till the fourth or fifth decade of their life which is neither inevitable nor physiologically necessary. In general, the best weight is the weight at which you both look and feel your best. The life insurance statistics, on which the height weight standards are normally based, tell us that the most nearly ideal weight to maintain throughout life is that which is proper at the age 25 for your height and body build. Age, of course, is an important factor in determination of body weight

in the growing stage, i.e., for children. The standard height and weight charts for Indian adult males and females and also for children at different ages. You can also use these charts to assess whether for your height the weight is ideal or not.

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Table 9.3: Standard height and weight For indian men and women

Height	Men		Women	
	Weight (kg.)	Weight (lb.)	Weight (kg.)	Weight (lb.)
1.52 M (5'0")	-	-	50-54	112-120
1.54 M (5'1")	-	-	51-55	114-122
1.57 M (5'2")	56-60	124-133	53-56	117-125
1.59 M (5'3")	57-61	127-136	54-58	120-128
1.62 M (5'4")	59-63	130-140	56-60	124-132
1.65 M (5'5")	61-65	134-144	58-61	127-135
1.67 M (5'6")	62-67	137-147	59-64	130-140
1.70 M (5'7")	64-68	141-151	61-65	134-144
1.72 M (5'8")	66-71	145-156	62-67	137-147
1.75 M (5'9")	68-73	149-160	64-69	141-151
1.77 M (5'10")	69-74	153-164	66-70	145-155
1.80 M (5'11")	71-76	157-168	67-72	148-158
1.82 M (6'0")	73-78	161-173	69-74	151-163
1.85 M (6'1")	75-81	166-178	-	-
1.87 M (6'2")	77-84	171-184	-	-

Table 9.4: Height and weight of Indian children

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Age	Height		Weight	
	Male (cm)	Female (cm)	Male (kg)	Female (kg)
3 months	56.8	56.0	5.2	5.1
6 months	62.8	61.7	7.0	6.6
9 months	68.9	66.8	8.0	7.7
12 months	72.8	70.7	8.6	8.3
1-2 years	82.61	79.89	10.94	10.21
2-3 years	91.14	89.63	12.79	12.11
3-4 years	98.36	96.21	14.78	13.79
4-5 years	104.70	104.19	16.12	15.85
6 years	118.9	117.3	22.1	21.4
7 years	123.3	122.7	24.5	24.3
8 years	127.9	126.8	26.4	26.1
9 years	133.6	132.3	30.0	29.7
10 years	138.5	138.5	32.4	33.5
11 years	143.4	144.1	35.3	36.5
12 years	148.9	150.3	38.8	42.6
13 years	154.9	153.0	42.9	44.4
14 years	161.7	155.1	48.3	46.7
15 years	165.3	155.3	52.2	48.2
16 years	168.4	155.4	55.4	49.8
17 years	168.9	156.4	59.0	49.9
18 years	169.4	157.2	62.0	50.0

9.5 OBESITY

Obesity is a condition resulting from accumulation of excess body fat. The fat deposition takes place because over a period of time, people consume diets which provided much more energy than they were able to expend for their metabolism, physical activity and growth.

The prevalence of obesity in developing countries has increased. This is because communities have emerged from a life style of subsistence towards a life style of affluence. In India, there has been an increased migration of the rural population to urban areas. This shift also has been a contributing factor to life style changes including significant reduction in physical activity leading to changes in weight.

9.5.1 Etiology

What are the causes of obesity? However simple the question may sound, the answer to it is not all that simple. We cannot deny that excess weight results from positive energy balance or consistent consumption of excess calories than the body is able to expend. This means that obesity can be corrected by balancing the

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intake and output of calories consumed and expended. Thus, it is not an easy task to accomplish because obesity is the net result of a complex interplay of genetic predisposition towards fat storage and a number of environmental factors that determine the weight status of an individual.

We cannot change our heredity but we can, to a certain extent, exercise control over environmental factors by carrying out suitable modifications in our life style. Indians as an ethnic group are at a disadvantage. It is a fact that for a given degree of obesity or BMI, Indians have higher body fat percent and visceral fat (fat around internal organs) than other populations which increases the risk of chronic degenerative diseases in later life. Let's enumerate the various etiological factors for obesity. We will learn about each of them in detail also.

Genetic susceptibility

Dietary habits

Physical activity

Affluence and abundant availability of food

Psychological factors

Hormonal imbalance

Birth weight and childhood growth pattern

Genetic Susceptibility: Some people inherit a tendency to become fat. Earlier, it was thought that genetic inheritance ranged from 66% to 80% but now it is believed that our chances of inheriting our parent's BMI is about 33%. Obesity or thinness of an individual is inherited, basically from the biological mother. If our biological mother has been overweight as an adult, the likelihood of our being heavy is about 75%. A number of genes are implicated in pathogenesis of obesity. The two genes which recently have received much attention are the *Ob* gene and the *A* - adrenoreceptor gene. The *Ob* gene produces leptin (a hormone) which is normally secreted from fat cells. Mutations in the *Ob* gene cause obesity. Treatment of obese mice with leptin has shown to reduce food intake and body fat. Some scientists are of the opinion that the *Ob* gene may not have a major role to play in human obesity.

The β_3 - adrenoreceptor gene is basically located in adipose tissue. It regulates Resting Metabolic Rate and oxidation of fat in human beings. A mutation in this gene may lead to weight gain. In some individuals, it may increase the body's ability to store fat when food is limited and cause an increased risk of obesity when plenty of food is available and energy expenditure is reduced. However, this does not mean that it is inevitable for a person to be obese because of genetic mutations. Expression of the genetic tendency may be controlled by appropriate life style modifications also. Dietary habits: You are aware that a positive energy balance is one of the major contributory factors for obesity. Some people are in the habit of eating too much food.

They may also be ignorant about the caloric values of common foods like butter, cheese, jam or rich baked snacks and desserts, the extra helpings consumed

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rapidly increase the amount of calories ingested. Sometimes maintaining social relationships also contributes to intake of excess calories. As you advance in age, your metabolic rate slows down and you require less energy to carry out the same set of activities than that needed 20 years ago. On the same diet and eating habits you will definitely put on weight. It is believed that women are more likely to be obese in the age group 40-60 years across all geographical regions.

Besides the diet per se, there are certain eating habits which may lead people to obesity, e.g., those who eat food at a very fast rate tend to chew food less and end up eating more food. Similarly, nibbling between meals may contribute significantly extra calories to the total intake than is normally realized. Also, those who tend to eat whenever food of their liking is available or those who just follow meal times even if they are not hungry tend to put on weight. Mothers generally eat leftovers of children because they want to avoid wastage of food, adding on more calories to their own calorie intake.

Physical activity; Life style with lack of an exercise schedule tends to make one obese. As we approach middle age, our physical activity generally decreases without a corresponding decrease in food consumption leading to obesity. Activity may be decreased because of a debilitating illness like arthritis or cardiac disease. A change of occupation or simply because of the fact that period of quiet, rest and relaxation have increased, may lead to decreased activity. In addition, when we are more active, the body prefers to metabolize fat as an energy source leading to a decrease in the adipose tissue.

Affluence and abundant availability of food: With increasing affluence, increase in purchasing power and abundance of food, people tend to eat more. Intake is more when people are offered a variety of foods than when a single food is available. Eating out has become fashionable leading to an increased consumption of junk food which is rich in calories and short on essential nutrients. The ready availability of and preference for high fat and/or fast foods also contributes to obesity. Psychological factors: Lonely, bored and depressed individuals may find solace in eating. When there is nothing else to do, eating provides diversion resulting in increased consumption of calories.

Hormonal imbalance: Certain diseases associated with secretion of hormones, e.g., hypothyroidism, hypogonadism and Cushing's syndrome exhibit obesity as one of the characteristic features. A large number of persons who are unsuccessful in reducing their weight tend to cite hormonal imbalance as causative factor for their obesity but the fact is that only a very small percentage actually suffers from it. Diagnostic tests are available which help in finding out if a person is actually suffering from hormonal imbalance.

Birth weight and childhood growth pattern: It has been shown that slow growth of the foetus in utero and during infancy is followed by accelerated weight gain in childhood. This combination of small size at birth and accelerated childhood weight gain has been found to be associated with exaggeration of adiposity, as

well as, insulin resistance in later life. So can we say that small size at birth and accelerated childhood weight gain is a predictor of later obesity?

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9.5.2 Energy Balance

Obesity is a state of positive energy balance created by consumption of calories in amount excessive to the total energy expenditure (TEE) by the body. TEE comprises the following:

- a) Resting Energy Expenditure (REE) — 60-75% of TEE
- b) Thermic Energy of Food (TEF) — of TEE
- c) Energy Expended on Physical Activity (EEPA) — 15-30% of TEE

REE is the energy required to sustain normal body functions like circulation, respiration, pumping of ions across membranes, synthesis of various compounds, maintenance of body temperature etc. The extent of this expenditure depends upon body size and composition.

TEF is the energy expended to digest, absorb and metabolize food including synthesis and storage of various nutrients.

EEPA is the most variable component of total energy expenditure and includes energy expended in voluntary exercises like in walking, cycling, swimming etc. as also that expended involuntarily e.g., in shivering and fidgeting. The sum total of REE, TEF and EEPA gives us the value for total energy expenditure(TEE).

Total Energy Expenditure	=	Resting Energy Expenditure	+	Thermic Energy of Food	+	Energy Expended in Physical Activity
(TEE)	=	(REE)	+	(TEF)	+	(EEPA)

You are aware that the total energy derived from the food that we consume can be calculated from the energy provided by protein, fat and carbohydrates present in the food. Energy provided by 1gram of protein = 4 Kcals, 1 gram of fat = 9 Kcals and 1grams of carbohydrate = 4 Kcals.

Weight status is maintained when the total energy derived from food intake equals the total energy expended by the body. We tend to lose weight when less energy is derived from food than is expended. Let us see what happens when an individual is consuming daily, say, 100 Kcal over and above the amount he is able to expend.

Extra calories ingested/day = 100 Kcal

Extra calories ingested/month = 3000 Kcal

Now 1kg adipose tissue represents = 7700 Kcal (1 gm adipose tissue — - 7.7 Kcal)

Weight gain/month = 3000 + 7700 = 0.38 kg approximately

Weight gain/year = 4.56 kg

You will appreciate that if this continues for a period of, say, five years, theoretically, even before the person realizes, he is transformed into a grossly obese individual. It is hard to believe that as little as one extra chapatti or two teaspoons of butter everyday will result in about 20 kg weight gain over a period of five years.

Even though in effect, weight is not deposited in as direct proportion as this. Let us see why. With the increase in energy intake, energy output is affected in a number of ways. Firstly, as the quantity of food ingested is increased, thermogenic effect of food would also increase amounting to about 10% of the excess intake. Secondly, the energy stored would increase both the fat and the fat-free mass resulting in an increase in metabolic rate. This adaptation of metabolic rate which tends to oppose fluctuation in weight does not permit weight gain in direct proportion to increase in caloric intake. The thermodynamics of weight loss is a bit less complicated. As opposed to the great metabolic cost involved in storage of excess dietary calories as fat, protein or glycogen, hardly any metabolic cost is involved in mobilization of these stores.

Plateau effect: You must have noticed that when people start following weight reducing diets, they lose weight rapidly in the beginning, then a little slowly and finally a plateau is reached when they no longer lose weight. Initially, glycogen stores (sugar stored in liver) are mobilized which is accompanied by a corresponding loss of water. Then, as weight is lost, it results in loss of extra muscle which was developed to support the extra adipose tissue. Loss of lean body mass reduces the RMR rapidly so that on a given diet, the energy deficit is reduced and the rate of weight loss slows down. Weight loss stops at this point unless a change is made either in nutritional intake or physical activity. This fact has been hypothesized as "set-point theory".

Weight cycling: There are a number of obese people who keep losing and gaining weight a number of times in their lives. This is called the Yo-yo effect. Every time they regain lost weight, it takes longer to lose the same amount of weight and also less time to regain it. This frequent losing and gaining of weight is associated with health risks related to normal functioning of the heart. Psychologically also repeated weight gain is quite demoralizing for the obese individual. Withstanding, any amount of intentional weight loss results in significant reduction in all cause, cardiovascular and cancer mortality.

Adipose tissue: At this point, it will not be irrelevant to consider how exactly does an increase in the fat depot take place. For understanding obesity better, it is important for you to know that fat is stored as triglyceride in fat depots made up of adipose tissue. A normal adult woman has about 20% to 25% of her body weight as fat while in men appropriate body fatness is 12% to 15% of body weight. When we put on weight, there is an increase in the adipose tissue. This may either be a result of hypertrophy or hyperplasia of adipocytes (fat cells) or a combination of the two processes. Hypertrophy means increase in the size of adipocytes already

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present in the body while an increase in their number is known as hyperplasia. As an adult we put on weight mostly by hypertrophy of fat cells although in some forms of obesity hyperplasia may also be there. Hyperplasia basically occurs during infancy and adolescence as a part of growth process. Fat cell size decreases when we lose weight for any reason but weight loss does not involve a decrease in the number of adipocytes, Brown fat and white adipose tissue (WAT): There are two kinds of adipose tissue.

Brown Fat is located around the shoulder blades and kidneys, constituting 1-2% of body weight. It is highly vascular which is the reason for its brown colour. It is capable of producing a large amount of heat for cold adaptation by burning of excess energy. It is a site for conversion of thyroid hormone, thyroxine, to its biologically active form.. White adipose tissue acts as a cushion to protect abdominal organs and is the fat that accumulates under the skin. Earlier, it was thought that WAT is passive and acts only as a fat storage depot. WAT, in fact, is a smart tissue and has a number of functions to perform. It has now been realized that WAT is an endocrine organ, which besides some other factors, secretes a hormone leptin. Leptin seems to have a role to play in reducing appetite or increasing satiety and also in regulation of the energy balance. A deficiency of leptin, therefore, is conducive to obesity. Adipocytes in WAT also have number of hormone receptors on their cell surfaces. That is why individuals with abdominal obesity are prone to developing insulin resistance which initially causes impaired glucose tolerance and ultimately may cause Diabetes mellitus.

9.5.3 Metabolic Aberrations and Clinical Manifestations

The state of obesity brings about certain alterations in the normal body processes .

Deranged lipid profile: Lipids, as you are already aware, are important dietary constituents that include fats, steroids, phospholipids and glycolipids. A number of vitamins and essential fatty acids are associated with them. In obese individuals, the lipid profile is usually deranged. The triglyceride values are generally high and HDL cholesterol is low. Both triglycerides and HDL cholesterol are synthesized from products of digestion of dietary fats. With weight reduction, both these levels come back to normal.

Insulin resistance: Insulin resistance is a condition in which your body cells cannot utilize insulin efficiently although sufficient amounts are secreted by the pancreas. Obesity is a contributing factor towards insulin resistance. Because sufficient insulin is being produced but the body cells are not able to use it, the blood insulin levels become high (hyperinsulinaemia). This affects the utilization of glucose leading to high fasting blood sugar levels and abnormal glucose tolerance. In addition, levels of plasma glucagon (a hormone produced by pancreas having an effect opposite to that of insulin), free fatty acids and uric acid also are found to be elevated in obese individuals. All these altered biochemical parameters get back to normal as weight loss is affected.

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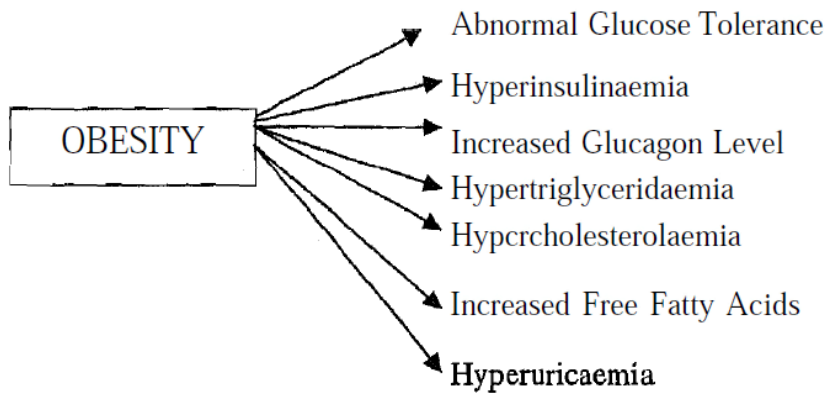


Figure 9.1: Altered biochemical parameters in obesity

Clinical manifestations:

You must have observed that your overweight friends and colleagues seem to have less energy which makes them an easy prey for fatigue. They are also less agile and more likely to fall because of imbalance, They have a tendency to have high blood pressure and dyspnoea (breathlessness on exertion).

Many of them may have increased susceptibility to developing skin disorders such as heat rash, intenrigo (superficial inflammation of two skin surfaces that are in contact with each other such as between thighs), candidiasis (a fungal infection) and acanthosis nigricans (dark, warty growths in skin folds like groin, armpits and mouth).

9.5.4 Consequences

Obesity has a number of adverse effects and is a risk factor for several problems. It is a risk factor for all causes of mortality and morbidity.

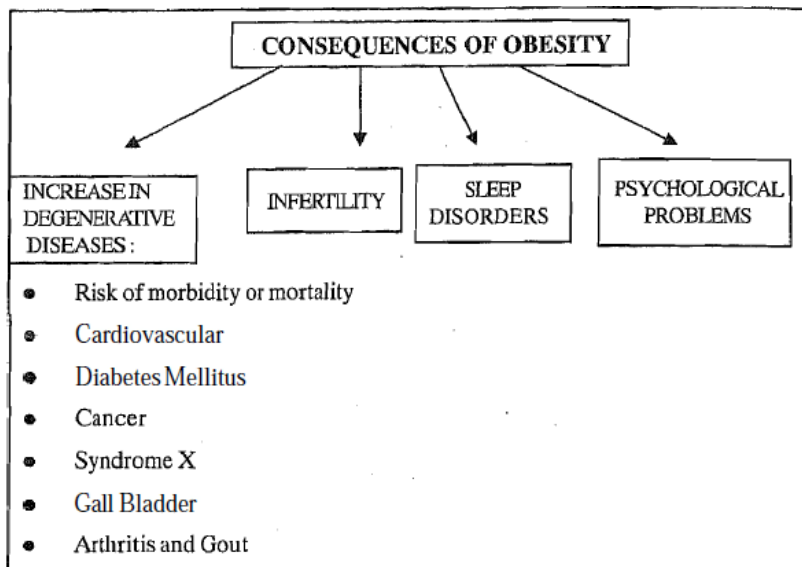


Figure 9.2: Consequences of obesity

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General mortality and morbidity risk: Obesity increases the risk of morbidity and mortality. The obese are more prone to developing morbidities or other chronic diseases like, cardiovascular disease including hypertension and dyslipidaemia, non-insulin dependent diabetes mellitus, gall bladder disease and gout. The risk of developing some non-fatal conditions like arthritis, back pain, infertility, sleep disorders and other respiratory conditions leads to increased morbidity among the obese. Let's discuss these conditions in slightly more detail.

Cardiovascular disease and stroke: Obesity may be an independent risk factor for coronary heart disease (CHD) with the degree of obesity being directly proportional to the rate of development of CHD. Even moderate overweight has been shown to increase the risk of CHD. A reduction in weight leads to improvement in cardiovascular risk factors like hypertension and abnormal lipid levels. The blood pressure returns to normal and the lipid profile improves.

When the blood vessels of the brain are diseased, they may rupture or there may be inadequate blood supply to brain resulting in a stroke. This may be due to hypertension or fatty deposits in blood vessels of the obese.

Type - 1 Diabetes: In people with normal weight, Type 1 Diabetes is not a major cause of death but it is an important contributor to morbidity and mortality in obese people. It is associated with insulin resistance and hyperinsulinaemia (increased level of circulating insulin in blood). Fortunately, reasonable control in blood sugar levels may be achieved by modification in the lifestyle. A balanced diet, physical activity and drugs can control blood sugars and an obese can lead a near normal life.

Syndrome X : People with intra-abdominal obesity with high waist- to- hip ratio are more prone to develop the metabolic syndrome X. This is characterized by the collective presence of chronic disorders that include glucose intolerance, insulin resistance, hyperlipidaemia and hypertension. The syndrome X is one of the major public health problems associated with obesity.

Gall bladder disease: Obesity is one of the risk factors for formation of gallstones.

The supersaturation of bile with cholesterol in obese individuals makes them prone to having gallstones as you will learn later in Unit 15. The excess adipose tissue is also known to contain a large amount of cholesterol. Weight loss does not reduce the risk of gallstone formation because the mobilization of adipose tissue may cause the bile to become even more saturated with cholesterol in obese people. Cancer: Risk of cancers of the colon, rectum and prostate increases greatly in obese men while obese women are more likely to develop cancer of breast, ovary, endometrium and cervix.

Backpain, arthritis and gout: Abdominal obesity increases the risk of back pain because of the extra load on the spinal column. This, in turn, reduces physical activity leading again to an increase in adiposity.

Obesity is also associated with the development of osteoarthritis and gout. The extra stress on the weight bearing joints is a contributing factor. Obese are prone to developing hyperuricaemia (excess uric acid in blood) resulting in gout.

Infertility: Obese women are reported to suffer more from menstrual disorder, infertility and polycystic ovary syndrome all of which tend to improve on reduction of weight.

Sleep disorder: One of the common problems that obese males and females suffer from is sleep disorder, commonly known as sleep apnoea. Obesity causes narrowing of the upper airway when the person is in supine position. This can result in sudden death in severe cases.

Psychological problems: Obese people may be exposed to ridicule and discrimination in areas like employment, promotions and social interactions. This may result in low self-esteem and depression leading to overeating for consolation. This aggravates the existing problem further. Although it is increasingly being understood that obesity is a complex interaction of metabolic, physiological, and genetic factors, obese people are still viewed as being weak-willed and self-indulgent.

After a detailed study of various factors related to obesity, let us now move on to the management and prevention of this multidimensional public health problem. In the forthcoming section we shall discuss the strategies for achieving a negative energy balance, as well as, the steps that must be considered for prevention of obesity. However, let us first make an effort to check our understanding on the issues discussed above.

9.6 MANAGEMENT OF OBESITY

Management of obesity should be taken up with a clear understanding of the harsh realities of the problem and its outcome. It may be a frustrating experience for the physician and the nutritionist because of the frequent failures encountered during the treatment.

You have already read about multiple etiological factors causing the chronic condition, the cause of which is difficult to pin-point. This makes the treatment even more difficult. The lost weight is frequently regained by the obese which may be demoralizing for continuing the necessary changes in the diet and physical activity,

Goals of treatment: As said in the beginning, the goal of treatment of obesity today has shifted from mere 'weight loss' to 'weight management'. Each weight loss programme has to have its separate set of goals keeping in view the overall health of the individual. A loss of as little as 5-10% of the original body weight by the obese results in significant improvement of health and helps in reducing the severity of the comorbidities or the risk factors associated with obesity. Studies

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have shown that even with a 5-10% weight reduction, an obese has better glycemic control, and lowered blood pressure and serum cholesterol levels. Hence it may not be realistic for the obese to always have singular focus of coming down to the desirable weight. Obsession with desirable weight may actually be inappropriate in some cases under certain circumstances. So let us see what is the best approach to manage obesity.

9.6.1 Dietary and Lifestyle Modifications

The management of obesity basically comprises the following three-pronged approach.

- a) Dietary modifications
- b) Physical activity
- c) Behaviour and lifestyle modifications

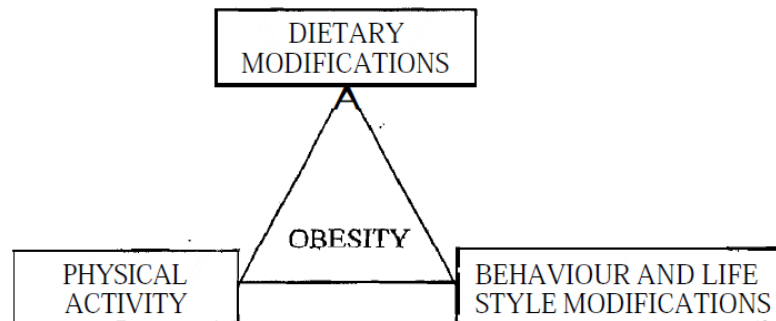


Figure 3.3: Management of obesity

Some cases where obesity is accompanied by certain comorbidities at higher BMI values, the use of drugs and/or surgery may need to be considered. Pharmacological and surgical interventions are required in relatively few cases and should not be construed upon as substitute for necessary changes in diet and physical activity.

a) Dietary Modifications

The dietary modifications serve as a guide for the obese to make healthy food choices. The first step towards prescribing a diet for weight reduction is to take a careful dietary history of the obese person. You need to know the routine eating pattern, the diet he/she is accustomed to, availability of foods and the likes and dislikes. Determine the ideal weight from the height-weight tables. The daily diet plan should have an energy deficit of 500-1000 Kcal in general. It is also important for us to know whether the person has tried to lose weight earlier too and what advice was given then and why was the outcome unsatisfactory. All this information can be gathered in a few minutes and this could form the basis of providing appropriate advice to the obese for losing weight. The following dietetic principles must be considered while planning diets for weight reduction. Of course, you will be learning more about this aspect in your practicals too. So let us learn about the dietary guidelines.

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Energy : Energy or calorie intake is the key factor which will determine the outcome of dietary management for overweight/obese individuals, You will appreciate the fact that to effect any degree of weight loss, the energy has to be restricted to the level that enables mobilization of fat stores for carrying out the daily activities of the body. The energy requirements can be determined on the basis of ideal body weight. Three main categories, depending upon the individual's size and level of activities have been determined on the basis of ideal body weight

Table 9.5: Energy requirements based on activity levels for obese, normal and underweight subjects

Energy Requirements* (Kcal/kg IBW/day)			
Activity	Obese	Normal	Underweight
Sedentary	20-25	30	35
Moderate	30	35	40
Heavy	35	40	45-50

The energy requirements for obese, normal and under weight adults for various levels of activity. Since the basal metabolic rate is affected by the type of build, it is imperative to increase or decrease the energy intake by 10% depending on the build (exomorphs and endomorphs, diets are often prescribed.

1. Moderate Deficit Diet (For pre obese) : 1400 Kcal/day and above for males
1200 Kcal/day for females is safe for use
2. Low Calorie Diet (For obese) : 800 to 1400 Kcal/day for males 800 to 1200 Kcal/day for females use under medical supervision
3. Very Low Calorie Diet (For very obese) : Less than 800 Kcal/day use under (VLCD) medical supervision

Despite calorie restriction, all the above diets must be nutritionally adequate. In general, it is safe to use the moderate deficit diets providing 1200-1400 Kcal and low calorie diets that provide minimum of 1000 calories/day. They can be planned to provide optimum nutrition and offer sustainable weight loss. The VLCDs providing 400-800 Kcal/day on the other hand promote rapid weight reduction but must be followed under close supervision of physician and dietician and that too, for a limited period of 12 to 16 weeks to minimize the risk of body protein losses and cardiac problems. They may only be considered for the obese with a minimum BMI of 32.

REMEMBER : IT IS SAFE TO USE MODERATE DEFICIT DIETS PROVIDING 1200--1400 KCAI/ DAY. LOW CALORIE AND VERY LOW CALORIE DIETS MUST BE USED UNDER STRICT MEDICAL CARE FOR LIMITED PERIODS.

NOTES

Proteins: Adequate amount of proteins should be included in the diet to ensure proper metabolism and prevent weakness which is usually experienced by patients after weight loss which is achieved by consuming an unbalanced diet. Protein rich foods provide a higher satiety as compared to those rich in carbohydrates (other than non-starch polysaccharides). Proteins also have a high specific dynamic action which implies that their ingestion produces a greater increase in metabolism than ingestion of carbohydrates or fats — an important aspect when you are trying to lose weight. Include about 1g protein per kg body weight. Emphasis should be laid on the inclusion protein rich foods from plant origin rather than from animal sources as the former are low in fat but high in dietary fibre.

Fats: Fats, being a concentrated source of energy need to be restricted. Excess dietary fat promotes much more weight gain than carbohydrate or protein of the same amount. Further, the gain in weight due to excess intake of fat is in the form of adipose tissues which is not conducive to good health. Include fat in the form of vegetable oils (rich in MUFA's and PUFA's) so that sufficient essential fatty acids are supplied in the diet and at the same time the risk of developing coronary artery disease can be minimized. Not more than 20% of the total energy should come from fat. Foods rich in saturated fatty acids such as red meats, whole milk/its products should be strictly avoided.

Carbohydrates: Carbohydrates in the form of non-starch poly-saccharides provide bulk and satiety value to the reducing diet. They are also important for regular bowel movements; constipation being a common problem among obese. About 50-55% of total calories may be from complex carbohydrates and 100/0 from simple carbohydrates. Include liberal amounts of fresh high fibre vegetables and fruits preferably raw and with their edible peels in the diet.

Vitamins: If adequate amount of fresh fruits and vegetables are included in the diet, the body stores of water soluble vitamins are usually not depleted. However when we restrict fats for prolonged periods, the diet may be deficient in fat-soluble vitamins A and D. They may need to be supplemented for the chronic cases.

Minerals: A diet high in sodium may promote retention of fluid in the body. Moderate restriction in the use of common/table salt may be helpful in a weight reducing diet, particularly if the patient is also hypertensive.

Fluids: Liberal amounts of water and zero/low calorie fluids may be included in the diet. It may be helpful to have a glass of water before meal to reduce food intake. Some patients benefit by taking a spoon of guar-gum/pectin/xanthum gum or finely ground husk/bran of cereals and pulses in glass of water before meals as it gives a feeling of satiety.

Mentioned below is an example of a weight reduction diet (1200 Kcal) along with a sample menu to give you an idea regarding the applied aspects of the parameters discussed so far.

1200 KCAL DIET

Nutritional
Care In Weight
Management

Food	Amount (approx.) per exchange (g)	CHO (g)	Protein (g)	Fat (g)
Milk (double toned)	250	24	16	7.5
Vegetables	100	28	8	—
Fruit	100	10	—	—
Cereal	25	75	10	—
Pulse	25	51	21	—
Fat	5	—	—	20
	—	188	55	27.5

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This is 'just an idea given to you about a 1200 Kcal diet.

Sample Menu for 1200 Kcal Diet

Early Morning : Tealcoffee, Plain

Breakfast : I slice cracked wheat bread
2 slices tomato
1 egg white (boiled)
1 tsp. green chutney
2 tbsp. cornflakes
1 glass double toned milk
OR
1 spinach missi roti
1 medium bowl of curd
1 guava

Mid-morning : 1 orange

Lunch : 2 chapatties
1 bowl moong whole pulse
1 bowl cabbage vegetable

1 plate carrot and tomato
salad with lemon dressing

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		1 bowl curd
Evening	:	Tealcoffee plain
Dinner	:	1 cup clear vegetable soup
		2 chapatties (wheat flour + soya flour)
		1 bowl nutrinugget and pea vegetable
		1 cucumber
		1 bowl curd

You may or would experience several times that overweight/obese patients attain their lost body weight again and again particularly after leaving a weight reduction programme. This generally happens due to inadequate counseling of the patient regarding appropriate dietary habits. Proper dietary counseling gives the patient a clearer understanding regarding the association of food with weight gain/loss. Behaviour modification can result only through repeated counseling sessions and has therefore been identified as a long-term approach for management of the achieved weight loss.

Diet Counseling

As discussed above diet counseling is a very important aspect of a successful weight reduction programme. The person who attempts weight loss should be suitably motivated and should be armed with facts related to the whole exercise. Counseling can be given in person or to a group as you would recall studying in Unit 1. Individual counseling is of prime importance because that is required to establish realistic goals for the treatment so that you can relate to the diet and comply with the same. It is also important to take the dietary history of the patient and to know about his food habits and pattern of living for prescribing the diet schedule.

Group sessions have an importance of their own in the sense that they provide a platform to people having similar problems to share their experiences and exchange ways and means to bring about changes in their diets. The individuals are also likely to be motivated better when they compare their progress with others in the group. Both individual and group counseling are associated with motivation and psychological support. There is no point in handing over a diet schedule to the patient unless he has some motivation for losing weight. To bring about a change in dietary habits is not easy because their foundation is laid in early infancy and childhood. You are not likely to change unless you are strongly motivated to do so. Improvement or maintenance of health is a very strong factor

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which the physician or dietician can use for motivating the patient to bring about the necessary changes. With their guidance at initial and follow up visits, this motivation can come from within the individual himself which will see him/her through the programme successfully.

The patient should be very clear about the fact that excess calorie intake has to be brought down to effect weight reduction. He/she also needs to understand the reasons of overeating and how to control the factors leading to the same.

The counseling sessions help in increasing knowledge regarding food facts. The obese may feel disheartened after a few weeks when they realize that the rate of weight loss has decreased. The individual must understand that there is a reduction in metabolic rate after some weight is lost. Despite careful adherence to the prescribed schedule, the rate of weight loss will decrease. For weight loss to progress further calorie restriction or increase in activity will be required, the latter is a better alternative. Knowledge about calorie values of foods is another area which requires emphasis. Food exchange lists which group different food items having approximately the same calorie values are helpful in this regard. The individuals should know about portion control to enable them to stick to the prescribed diet. Many foods have low calories but when eaten in large portions contribute substantial calories in the diet. So how much should the patient eat is also important.

Instructions about eating out are important. An obese must select judiciously from options available. For example any recipe that says cream of, creamy, buttered or fried is bound to be high in calories. A clear soup, broiled or roasted non-vegetarian dish or vegetables without sauces or thick gravies, salads without oily dressings and fruit instead of a rich dessert are better options for them. Excess socialization hinders the weight reduction programme. Eating light meals at home may be a good idea so that the obese could minimize on extra calories.

b) Physical Activity

You are already aware that exercise plays an important role in initiating and sustaining weight loss along with dietary and lifestyle modifications. Exercise promotes a sense of well being and increases bone density, as well as, cardiovascular strength. It helps in increasing the lean body mass in proportion to fat. Exercise burns glycogen stores paving the way for fat to be used as fuel.

What the effect of physical activity on health?

The Surgeon General's report (1996) summarizes the effects of physical activity on health as follows.

Overall Mortality: Higher levels of regular activity are associated with lower mortality rates among adults and even moderate activity on a regular basis results in lower mortality rates than those who are least active. The risk of several degenerative diseases is also reduced.

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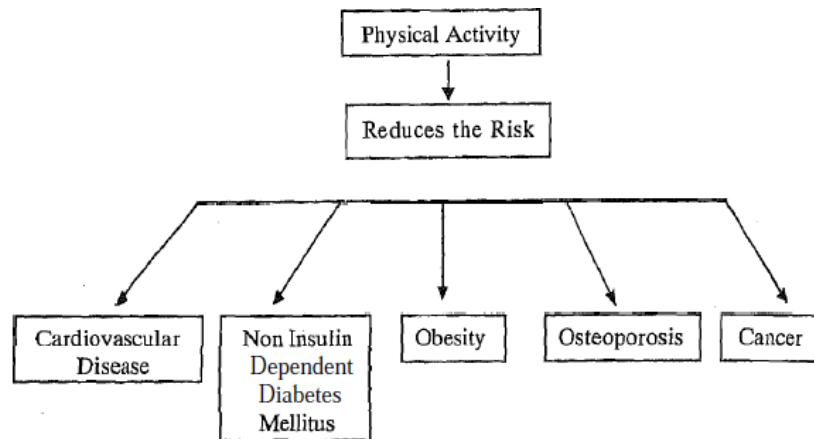


Figure 9.4: Effect of physical activity on health

How much physical activity is enough?

Although it is difficult to prescribe the optimum amount of physical activity, it is important to note that any exercise programme has to be consistent for affecting some degree of weight loss. It is recommended that 30 minutes or more of moderate intensity physical activity, even if accumulated in intermittent short spells at least five days a week (preferably everyday) should form a daily routine of all adults. A single 30 minute stretch may have the first 5 minutes for warming up, 20 minutes of moderate intensity exercise and 5 minutes of cooling down to prevent muscle injury. In any exercise programme, intensity should be increased only gradually with professional advice, especially for those who are above 40 years of age or have any health problems. In general, it helps to take stairs instead of lift, to take the glass of water yourself instead of asking somebody, walking to short distances instead of taking the car and in general being a little more active than before.

The exercise selected by an individual should be pleasant, enjoyable, affordable and easy to do. Practically speaking, the benefits of exercise besides its role in weight management can be summed up as follows.

-
- ```
graph TD; A[Exercise] --> B["* Reduces blood pressure"]; A --> C["* Helps lessen angina pains"]; A --> D["* Decreases body fats"]; A --> E["* Increases HDL cholesterol"]; A --> F["* Makes the heart stronger and more efficient"]; A --> G["* Help in increasing bone density"]; A --> H["* Reduces risk of cancers"]; A --> I["* Increases longevity"]; A --> J["* Offsets the immunity slump that accompanies aging"];
```
- \* Reduces blood pressure
  - \* Helps lessen angina pains
  - \* Decreases body fats
  - \* Increases HDL cholesterol
  - \* Makes the heart stronger and more efficient
  - \* Help in increasing bone density
  - \* Reduces risk of cancers
  - \* Increases longevity
  - \* Offsets the immunity slump that accompanies aging

**DAILY 30 MINUTES OR MORE OF MODERATE EXERCISE IS BEST FOR MAINTAINING IDEAL WEIGHT, HEALTHY FEELING, LONGEVITY AND GOOD IMMUNITY.**

### ***c) Behaviour and Life Style Modifications***

Behaviour and life style modifications are an integral part of the weight reduction plan. They are based on analysis of behaviour associated with appropriate, as well as, inappropriate thinking and eating habits. The obese tend to overeat in certain situations which if controlled may help towards keeping the weight in check. Keeping a food diary, the act itself is associated with weight loss. This means that if an individual pays attention to when and what he/she eats, they tend to eat less. It should not be inferred from here that behaviour therapy avoids the need for restricting energy intake. That still remains the mainstay of the treatment. The individual must learn to correct the negative thoughts that accompany a dietary lapse, e.g., instead of thinking that 'I have wasted all my efforts, I ate a piece of cake today', they should think 'One slice of cake is not going to increase my weight'. This shift of thought process helps tremendously in continuing the effort to lose weight. The following strategies related to lifestyle modifications are helpful. You may advocate these to obese individuals.

Remember:

Have regular mealtimes. Irregular eating habits put a lot of strain on the body.

Do not read or watch television while eating, you will land up eating More than you do otherwise.

Try to keep healthy snacks at home like fruits, vegetables and sprouts instead of biscuits, cakes, fried snacks and other fast foods.

Do not keep nibbling between meals. You will benefit by planning three main meals with one mid-morning and an evening snack.

Eat slowly, chewing the food properly.

Serve smaller portions so that another helping can be taken. —

Avoid drinking of alcohol and smoking. —

Incorporate some amount of exercise in your daily routine.

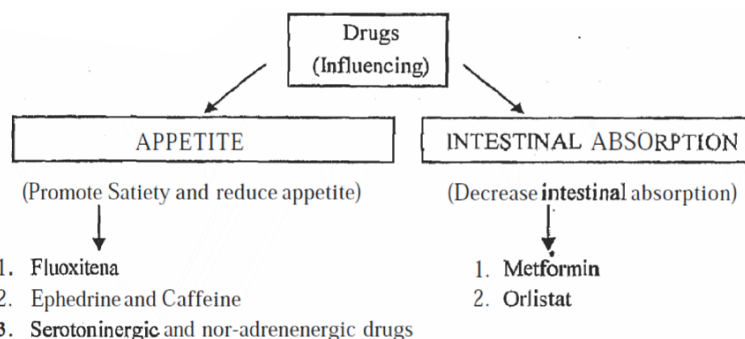
Handle stress in a positive manner through exercise, yoga and meditation.

### **9.6.2 Pharmaceutical Management**

A person with BMI 30 and above may require pharmaceutical management in addition to dietary and lifestyle modifications. It may also need to be considered when the obese person has associated problems such as impaired glucose tolerance, dyslipidaemia and hypertension. Complications like severe osteoarthritis, obstructive sleep dyspnoea etc. may also necessitate use of drugs. Let us get to know about these things.

Anti-obesity Drugs: The anti-obesity drugs can be classified into two broad groups

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**Figure 9.5: Anti-obesity drugs**

Drugs must be taken only under Doctor's advice as some can lead to side effects such as cardiac and liver problems. Herbal preparations must not be used as they lack clinical evidence. Laxatives and diuretics are ineffective and liberal use of these can affect the water and electrolyte balance of the patients body.

Caution should be practised in giving antiobesity drugs to patients undergoing psychiatric treatment or those having any drug allergy. Their use is contraindicated for children and pregnant and lactating women.

### 9.6.3 Surgical Management

Surgical procedures are generally restricted for the morbidly obese persons. If an individual has a BMI of 40 or higher, or a BMI of 35 or higher with associated comorbidities he/she may benefit by one of the surgical procedures. This specialized area is known as Bariatric Surgery and includes the following procedures :

- a) Gastric restrictive surgery
- b) Jejunioleal Bypass
- c) Jaw Wiring
- d) Liposuction

Post-operative evaluation by the team of surgeons, dietician and psychologist at regular intervals throughout life is of prime importance. Let us review the procedures.

Gastric Bypass Surgery' is the current 'gold standard' for bariatric surgical procedures. It involves use of a stapling device to create a tiny stomach 'pouch' by partitioning the stomach near its upper end to reduce the capacity of the stomach. On an average, the patient loses 30-40% of weight by this procedure.

The stomach size can also be reduced by using stainless steel staples across the upper portion of the stomach. Only about 1 cm opening is left into the distal stomach. This method is known as gastroplasty. This is found to be



quite successful.

**Jejuno-ileal Bypass:** Absorptive surface of the small intestines can also be reduced through surgery called the jejuno-ileal bypass. Some complications may arise by this method. **Jaw Wiring:**

Wiring the jaws closed has been effective in reducing weight because wiring permits the intake of only liquid that can be taken through a straw. Liquids and supplements that will provide adequate nutrition are given.

**Liposuction:** Liposuction is a cosmetic surgical procedure different from bariatric surgery. It involves aspiration of subcutaneous fat using thin cannulas inserted through very small incisions. The cannulas are attached to a high vacuum source and fat is aspirated with a collection device. Contour is diminished as the overlying skin shrinks to the reduced fat volume. Only 5 lb. of fat can be removed at a time.

You have learnt how surgical methods are used in case of morbid obesity. Once the treatment is done maintenance of appropriate weight is of prime importance. Let us see how this can be done. The preventive aspects are discussed next.

#### 9.6.4 Preventive Aspects

**Maintenance of Weight Loss:** Once an individual has managed to lose weight to a desirable level, it must not be assumed that the weight loss will be maintained automatically. The person will have to make a conscientious effort to prevent gain in weight. Energy requirements are reduced after weight is lost. After the intense dietary effort is over and the person reverts to the so called pre-dieting eating pattern, he/she is likely to put all the weight back before equilibrium is re-established.

To avoid this weight cycling, one must add extra food items to the diet only gradually and with extreme caution. The person still needs to avoid high calorie recipes. He/she must keep a record of weight every week. Any extra weight gained during this period, however small, must be lost immediately either by reducing food intake or increasing energy expenditure the following week. The person must not reduce the physical activity once he/she attains the goal.

You must remember that there is a reduction in metabolic rate after weight loss which is also conducive to subsequent weight gain on the same energy diet. To remain at the target weight the person will need to, in fact, reduce energy intake by 10-15% which is the maintenance energy cost of the weight lost.

**Prevention in the prevalence of overweight/obesity:** It is believed that the increase in prevalence of obesity worldwide is more due to the environment that has become conducive to weight gain rather than genetic mutations within individuals. As pointed out earlier, Asian population is more susceptible to developing comorbidities even at quite modest weight gains. You must have fully understood by now that obesity is a major risk factor for several chronic degenerative diseases.

That is why it is all the more important to employ strategies that aim at creating environments facilitating behavioural changes in general populations regarding diet and physical activity to prevent this enormous public health problem.

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The first and most important step for the rapidly progressing developing countries like India is to collect and organize data from various regions about the prevalence of obesity. Once the true prevalence is known, the goals or targets to reduce the same can be set. The prevention strategies need to be targeted basically at two sub-groups in the population.

- 1) Those who are already obese and need advice regarding reducing weight and maintaining it. These persons, if not careful about their diet are liable to gain weight. They need to be guided appropriately regarding a maintenance diet because after a period of controlled eating, they tend to go back to their original diet or favourite foods which may be high in energy and short on important nutrients. All weight reducing clinics/community slimming centers must offer appropriate guidance and support to people who have achieved weight loss successfully to prevent regain in weight.
- 2) Those who are at increased risk of becoming obese and require help to avoid putting on weight. This is the major population group towards which public health measures need to be targeted. It is believed that fitness at the age of 13 years is quite a strong predictor of adult fitness. Children between the age of 7 and 12 years of age, therefore, may be a very important group that falls into this category.

In addition, it is important to develop strategies to prevent the population in general from becoming obese. The major approaches of any public health strategy to reduce obesity shall be firstly, to reduce calorie intake from fat and secondly, to increase the level of physical activity.

### ***How to reduce calorie intake from fat?***

Efforts should be made to increase the nutrition knowledge of the general public through mass media.

The foods with lower fat content should be made easily available and popularized.

People should be motivated to make healthier food choices, especially when eating out.

Sincere efforts should be made by health professionals/systems to promote dietary changes.

### ***How to increase the levels of physical activity?***

The benefits of physical fitness should be spread among the public through mass media.

Physical activity should be encouraged in educational and other

institutions.

Opportunities for physical activity should be provided at work places and industry.

Public facilities for physical activity and exercise should be increased.

### ***National Approach -A n Example***

A number of countries have adopted a national approach to deal with the prevention of obesity and other non-communicable diseases. At a symposium on 'Obesity' at the IX Asian Congress of Nutrition, in 2003, Mabel Deurenberg-Yap of National University of Singapore discussed the health promotion strategies to reduce obesity in her small and highly urbanized country, Singapore. Singapore had initiated National Healthy Lifestyle Programmes and School Health Promotion Programmes twenty years ago to promote healthy eating and active life styles with a view to reduce the risk factors associated with lifestyle related non-communicable diseases.

Strong governmental support, as well as, consistent effort by the organizers has resulted in reducing obesity rates today, particularly among children. Adults have also been highly motivated to engage in physical activity. The government has developed a number of parks and swimming pools at short distances near residential areas to facilitate participation in aerobic exercises and games. Schools besides classroom teaching have special emphasis on sports and outdoor exercises.

Evaluation of these programmes to reduce obesity is regularly being carried out in order that they may be improved upon and may become more effective. Special emphasis is also given to health promotion research and evaluation. The country has been successful in setting an example which other nations must tly to emulate.

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## **9.7 UNDERWEIGHT**

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Just as overweight is the result of a positive energy balance irrespective of the etiology, underweight results when the energy balance is negative. Failure to consume sufficient calories to meet the energy requirement of the body for whatever reasons is responsible for not maintaining optimum weight. You have learnt that too much deviation on either side from the appropriate body weight increases the risk of health problems.

In addition, it is relevant to note that as per the report of WHO (1998), an estimated 50 million adult women are classified as being severely underweight in developing countries.

It also states that the consequences of poor health in childhood and adolescence including malnutrition, become apparent in adulthood, particularly during the childbearing years. At the IX Asian Congress of Nutrition (2003), Z.A. Bhutta, Pakistan reported that adult women who suffer from malnutrition had a much

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higher risk of giving birth to low birth weight (LBW) infants. LBW infants are at a higher risk of mortality. Those who survive are poorly breastfed and weaned, resulting in stunted, malnourished children. Additionally, LBW females developed into malnourished mothers who in turn gave birth to LBW infants. He stressed that this cycle could only be broken by optimizing nutrition throughout the life cycle. So then the adverse consequences of underweight are obvious. But, what is the cause for this condition? Let us read the next section and find out.

### 9.6.1 Etiology

There are a number of factors causing underweight. These are:

- Poor selection of food
- Physical activity
- Mother's health status
- Pathological condition
- Genetic predisposition

Let us learn a little about each of these factors.

**Poor Selection of Food:** Poor selection of food along with irregular eating habits

may be responsible for insufficient food intake and hence calorie intake. It may be due to ignorance or a lack of purchasing power of the family.

**Physical Activity and Psychological Factor:** Individuals who are tense, nervous and extremely active and who do not rest sufficiently tend to expend more energy than what they are able to eat. This can cause undernutrition.

**Mother's Health Status:** Poor nutritional status of the girl child coupled with under nutrition during pregnancy results in LBW infant being born. These children born are at a disadvantage right from infancy and may fail to reach optimum weight in adulthood.

**Pathologic Conditions:** Illness can affect weight status in a number of ways. For example, fevers and infections, increase the demand for energy, which if not met because of poor appetite, lead to loss of weight. Food intake may be severely limited by nausea, vomiting or diarrhoea in gastrointestinal disturbances. Metabolic rate may be greatly increased in hyperthyroidism resulting in underweight. Drug therapy may also alter taste or reduce appetite, leading to weight loss.

**Genetic Predisposition:** As explained in section 9.2 of this unit, the weight of an individual is inherited basically from his biological mother. In the event of the biological mother being thin, there is 75% likelihood of the individual being thin also.

## 9.7.2 Metabolic Aberrations and Clinical Manifestations

**Metabolic Aberrations:** When energy intake falls below the minimal requirements, the body responds with an orderly physiologic adaptation involving the hormones of energy metabolism. This causes mobilization of free fatty acids from adipose tissues and of amino acids from muscle to provide energy. Protein synthesis is cut down because proteins are burnt up for providing energy to the body. The metabolic rate of the body is reduced and lean body mass and adipose tissue contract resulting in weight loss.

Undernutrition is generally accompanied by protein deficiency in the body. Fortunately, the condition is reversible with proper nutritional support.

**Changes in Body Tissue Compartments:** The severity of nutritional deprivation determines the extent of changes in the body tissue compartments. The first casualties in moderate undernutrition are mainly the visceral proteins and muscle cell mass without any change in body fat. In severe undernutrition, losses of both muscle cell mass and body fat occur to a significant degree. Anthropometric measures and laboratory determination (of protein status can predict the extent of changes in the body tissue compartments.

A number of micronutrient deficiencies may occur in individuals who are underweight because of the less quantity of food ingested. A starving patient has inelastic skin, slow pulse, low blood pressure, marked emaciation and progressive loss of weight. **clinical manifestations:** Underweight may predispose to fatigue, lethargy and breathlessness. Iron-deficiency anaemia is usually seen because the diet is bound to be deficient in iron at a low intake of food. The accompanying protein deficiency, if severe, may manifest itself in the form of oedema. Underweight individuals are likely to suffer repeatedly from infection because of low immunity. Hip fracture is often preceded by weight loss. Metabolic aberrations occur during starvation and these may cause bradycardia (slow pulse), hypotension (low blood pressure), constipation, dry skin and hair, abnormalities of nervous system, depression and ultimately death. So what can we do to prevent these manifestations. The dietary management is highlighted next.

## 9.7.3 Dietary Management

We just read about the etiological factors that may lead to undernutrition and weight loss. Whatever may be the cause, all underweight individuals are usually in a negative energy balance and have depleted reserves of most nutrients. The diet prescribed for effecting weight gain should be high in calories, proteins, fat and carbohydrates. Since the capacity of the intestines to digest and absorb food is considerably reduced with undernutrition, the addition of foods above the usual intake has to be slow and gradual. We shall now discuss some of the salient features of a weight gain diet for individuals not suffering from any form of chronic disease that requires restrictions in the nutrient intake. So, let us start with the calorie intake which is most significant to weight gain.

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- **Energy:** The total calorie intake should be 500 to 1000 Kcal in excess of the daily needs in order to result a gain in weight by half to one kilogram in a week. Thus, if you need 2000 Kcal for your normal activity, you require 2500-3000 Kcal per day for weight gain. We can also compute the energy requirements on the basis of ideal body weight (as discussed in subsection 9.5.1 of this unit). The patient may be given 30-35 Kcal per Kg ideal body weight per day. The calories should be increased gradually over a period of one or two weeks to avoid digestive disturbances.
- **Proteins:** Proteins are required for tissue building, as well as, to take care of the daily wear and tear. Under weight individuals generally have depleted lean body mass and poor reserves of amino acids/blood proteins. Thus, the patient may benefit by consuming around 1.2 g per kg body weight of proteins per day. A combination of both animal and plant proteins should be incorporated but emphasis should be laid on the inclusion of easy to digest forms of protein such as half boiled egg, steamed/ boiled/sautéed flesh food etc.
- **Fats:** We know that fats are concentrated source of energy (1g = 9 Kcals). Fats are capable of increasing the energy value of the diet without adding much bulk to it. Add extra fat gradually, a sudden increase in fatty foods like butter, cream and oil may produce diarrhoea. About 30% of calories should come from unsaturated sources of fat.
- **Carbohydrates:** Liberal amounts of easy to digest carbohydrates should be included in the diet. The intake of dietary fibre should be minimized so as to prepare meals which are nutrient dense and have a small volume. Include more of high calorie vegetable like potatoes, colocasia and yam instead of radish, cucumber, leafy vegetables which are low in the carbohydrate content. All cereals provide high calories at low cost and should provide about 55-65% of total kilocalories.
- **Vitamins and Minerals:** If the diet provides good amounts of fresh fruits and vegetables, vitamin or mineral supplements are usually not required. However, if the patient indicates clinical signs of a severe nutritional deficiency, it may be imperative to use supplements or employ other essential medical measures.
- **Fluids:** Take fluids only after a meal instead of with or before meals so that food intake is not reduced. High calorie nourishing beverages such as milk shakes, egg nog should be preferred over low nutrient beverages such as cold-drinks, barley water, plain soda etc.

### *Planning the Daily Diet*

As mentioned above you need to add calories gradually to the diet. A practical way of doing so is to take the present intake of the patient and to improve upon it both qualitatively and quantitatively day by day till you reach the prescribed level. Try to add foods from most of the food groups. You can add 500 Kcal to the diet by including any of the following combinations

Whole milk : 1 glass Boiled egg : one

Bread : one slice  
Banana : one

OR

Chapatties : 2  
Dal : 1 bowl (30 g raw)  
Cottage cheese : 40g  
Sapota : One  
Fried Rice : 1 bowl(30 g raw)  
Curd(whole milk) : 1 bowl (100 g)  
Ice cream with fruits : 1 cup

**NOTES**

The patient should be advised to take small, frequent, easy to digest meals. The person improves in weight without having any gastrointestinal problems, he/she could take calorie-rich foods. You will learn how to select high calorie foods and plan diets and hospitalized patients to promote weight gain in the Practical Manual

**SELECT CALORIE-RICH FOODS. INCREASE THE SERVINGS OF FOODS GRADUALLY. EAT FREQUENTLY. ENJOY THE FOODS YOU LIKE IN A HAPPY ENVIRONMENT.**

The subject of weight management is vast and has unending diverse applications in the management of individuals with/without an underlying disease condition. We end our discussion here within the parameters of this but strongly recommend additional reading to clear your views on various food fads and misbeliefs. Let us now attempt the questions mentioned in check your progress exercise 4 to recapitulate the contents of this section.

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## 9.8 LET US SUM UP

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It must have been an interesting unit to read because we all are interested in maintaining an ideal body weight and also because this topic has a wide applied aspect. For this reason; we have also designed a partical (Manual-005) for you to learn the use/planning of diet(s) based upon maintenance of an optimum body weight especially with respect to various diseases.

In this unit we learnt about weight imbalance and the difference between different grades of under/excess body weight. The metabolic and clinical manifestations of both under and overweight were also discussed (impaired glucose tolerance, hyperinsulinemia, insulin resistance, hyper-lipidemia etc.). In the section 8.5 you

## NOTES

must have learnt about the dietary and life-style management for over weight/underweight individuals. Read this carefully as the fundamentals of these are utilized for effective and accurate planning of diet(s) for such individuals with or without a disease (diabetes, coronary artery disease(s), cancer; gout; Fever etc.). The physiological effects of increased physical activity were also briefed in this unit. Nutrition supports and non- dietary (surgical, pharmaceutical), measures with respect to weight management me gaining in roads for the treatment of secure obesity and a dietician's help is often required for ensuring optimum nutritional care of the patient. Reading this unit must have helped you in gaining an insight/better understanding on the various aspects of weight management.

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## 9.9 GLOSSARY

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|                              |                                                                                                                                                     |
|------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Arthritis</b>             | : a disease that involves an inflammation of a joint or joints.                                                                                     |
| <b>Bariatric Surgery</b>     | : surgical procedures for treatment of obesity.                                                                                                     |
| <b>Baria trics</b>           | : a scientific study of obesity and its related disorders.                                                                                          |
| <b>Binge eating</b>          | : an episode of excessive eating accompanied by a sense of loss of control over the eating process.                                                 |
| <b>Borborygmi</b>            | : abdominal gurgles due to movement of excessive fluid and gas in the intestines.                                                                   |
| <b>Brown Fat</b>             | : a dark-coloured, mitochondrion-rich adipose tissue in many mammals that generates heat to regulate body temperature.                              |
| <b>Carotenemia</b>           | : presence in the blood of yellow pigment carotene from excessive intake of carotene rich vegetables and fruits.                                    |
| <b>Comorbidity</b>           | : any condition that worsens as the degree of obesity increases and iinproves as obesity is successfully treated.                                   |
| <b>Cushing's syndrome</b>    | : a glandular disorder caused by excessive steroid hormone resulting in greater than normal functioning of adrenal gland; characterized by obesity. |
| <b>Hirsutism</b>             | : an excessive growth of coarse hair particularly in women.                                                                                         |
| <b>Nypercholesterolaemia</b> | : elevated blood cholesterol levels.                                                                                                                |
| <b>Hyperplasia</b>           | : an increase in tissue size by an increase in number                                                                                               |



**NOTES**

|                               |                                                                                                                                                                                 |
|-------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|                               | of cells.                                                                                                                                                                       |
| <b>Hypertriglyceridaemia</b>  | : elevated level of serum triglycerides.                                                                                                                                        |
| <b>Hypertrophy</b>            | : an increase in tissue size by an increase in cell size.                                                                                                                       |
| <b>Hyperuricaemia</b>         | : elevated serum uric acid levels.                                                                                                                                              |
| <b>Life expectancy</b>        | : a statistical measure of the average of the remaining life time of an individual in the given group.                                                                          |
| <b>Lipogenesis</b>            | : fat formation.                                                                                                                                                                |
| <b>Liposuction</b>            | : the removal of excess body fat by suction with specialized surgical equipment.                                                                                                |
| <b>Morbidly obese</b>         | : patient's who are 0-100 % above their ideal body weight; a BX'II value greater than 39.                                                                                       |
| <b>Obesity</b>                | : a condition describing excess body weight in the form of fat.                                                                                                                 |
| <b>Osteopaenia</b>            | : a decrease in the bone mass due to a decreased rate of osteoid (organic matrix bone) synthesis.                                                                               |
| <b>Osteoporosis</b>           | : loss of bony tissue resulting in bones that are brittle and liable to fracture.                                                                                               |
| <b>Overweight</b>             | : being too heavy for one's height; a BMI of 25 to 30 kg/m <sup>2</sup> Quality of life the level of well being of life style and the physical conditions in which people live. |
| <b>Resting Metabolic Rate</b> | : the minimum number of calories needed by the body to support its basic physiologic functions.                                                                                 |
| <b>Syndrome X</b>             | : a condition associated with glucose intolerance, insulin resistance, hyperlipidemia and hypertension, strongly linked to fat accumulation in the intra-abdominal cavity.      |

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## 9.10 CHECK YOUR PROGRESS

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- 1). What method is used for classifying a person over weight? How is it calculated?
- 2). What are the factors that determine one's ideal body weight?

**NOTES**

- 3). What is obesity? Enumerate the various etiological factors.
- 4). Give reasons for the following:
  - a) An increase in weight gain is not directly proportional to an increase in calorie intake.
  - b) A deficiency of leptin is conducive to obesity.
- 5). Briefly discuss the metabolic aberrations of obesity?
- 6). List the fatal, as well as, non-fatal conditions for which obesity is a risk factor.

# 10

## NUTRITIONAL MANAGEMENT OF EATING DISORDERS

NOTES

### STRUCTURE

- 10.1 Learning Objective
- 10.2 Introduction
- 10.3 Eating Disorders — A Review
- 10.4 Anorexia Nervosa
- 10.5 Bulimia Nervosa
- 10.6 Eating Disorder not Otherwise Specified (EDNOS)
- 10.7 Binge Eating Disorder
- 10.8 Management of Eating Disorders
- 10.9 Nutritional Management of Eating Disorders
- 10.10 Let Us Sum up
- 10.11 Glossary
- 10.12 Check Your Progress

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### 10.1 LEARNING OBJECTIVE

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After studying this unit, you will be able to:

- define the eating disorders,
- differentiate between the different eating disorders,
- diagnose patients suffering from different eating disorders such as anorexia nervosa, bulimia nervosa, binge eating disorder etc.,
- describe the management of these eating disorders, and
- plan for the nutritional management of these disorders.

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### 10.2 INTRODUCTION

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In the last unit, we focused on weight management. It is important to understand that the problems associated with too much deviation on either side from the appropriate range of body weight increases our risk of health problems. Obsession with slimming, especially in the adolescent age group may result in eating disorders

## NOTES

like anorexia nervosa, bulimia nervosa and other eating disorders. What are these eating disorders? What are the strategies employed for treating such problems? How to cope with the problems of refeeding individuals after acute starvation? These are some of the issues discussed in this unit.

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### 10.3 EATING DISORDER - A REVIEW

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If you talk to a group of young boys and girls in an informal setting about their physical appearance, you will find that a majority of them are dissatisfied with their weights, as well as, their physical appearance. Boys in general want to be taller and want to have more muscular bodies which in fact, is a healthy trend. Most girls, on the other hand, view themselves as fat and want to look slimmer the so-called 'Aishwarya Rai Syndrome'. Sometimes, the obsession with body weight may be so severe that it leads to eating disorders like anorexia nervosa and bulimia nervosa. These are psychiatric disorders, primarily affecting adolescent girls or young women, who had been previously healthy but start developing a paralyzing fear of becoming fat.

In addition to these two disorders mentioned above, overlap syndromes also exist. Some emaciated patients who fulfill the criteria of true anorexia nervosa may show bulimic behaviour at some time. Similarly, subjects with bulimia often pass through a phase of anorexia. In our discussion, we will assume that the two disorders are different expressions of a psychological obsession with body weight.

Other eating disorders such as eating disorder not otherwise specified or binge eating disorder or childhood eating disorder and eating disorder in athletes have also been observed. We will study about some of these disorders, with particular focus on anorexia and bulimia in this unit. Before we move further, it would be useful to define some common terms that would appear in this unit as given in Table 10.1 in order to facilitate their understanding at this stage.

**Table 10.1: Definitions of some common eating disorders**

|                  |                                                                                                                                                                                  |
|------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Anorexia         | It refers to loss of appetite, especially as a result of disease.                                                                                                                |
| Anorexia Nervosa | A disease characterized by refusal to maintain a minimally normal body weight, intense fear of gaining weight, body image distortion and amenorrhoea in post menarcheal females. |

**NOTES**

|                                                 |                                                                                                                                                                                                                                                                                                                     |
|-------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Binge                                           | An episode of eating marked by three particular features: 1) the amount of food eaten is larger than most persons would eat under similar circumstances, 2) the excessive eating occurs in a discreet period, usually less than 2 hours, and 3) the eating is accompanied by a subjective sense of loss of control. |
| Binge Eating Disorder                           | A disorder characterized by the occurrence of binge eating episodes <b>atleast</b> twice a week for a 6-month period.                                                                                                                                                                                               |
| Bulimia Nervosa                                 | A disorder characterized by repeated episodes of binge eating followed by inappropriate compensatory methods such as purging, including self-induced vomiting or misuse of laxatives, diuretics or non-purging including fasting or engaging in excessive exercise.                                                 |
| Eating Disorder not Otherwise Specified (EDNOS) | A diagnostic criteria for eating disorders that fail to meet full criteria for either anorexia nervosa or bulimia nervosa.                                                                                                                                                                                          |
| Purging                                         | It is a method intending to reverse the effect of binge eating. This may involve <b>self-induced</b> vomiting, which is the most common purging method. Additional methods may include laxatives, enema and diuretic abuse.                                                                                         |

Now that you are aware of the different disorders and the episodes, methods associated with them, we will move further and study about the different disorders-their prevalence, incident, etiology and the diagnostic criteria in detail. We begin with anorexia

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## 10.4 EATING DISORDER - A REVIEW

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## NOTES

the obsession with body weight may be so severe that it leads to eating disorders like anorexia nervosa and bulimia nervosa. These are psychiatric disorders, primarily affecting adolescent girls or young women, who had been previously healthy but start developing a paralyzing fear of becoming fat.

In addition to these two disorders mentioned above, overlap syndromes also exist. Some emaciated patients who fulfill the criteria of true anorexia nervosa may show bulimic behaviour at some time. Similarly, subjects with bulimia often pass through a phase of anorexia. In our discussion, we will assume that the two disorders are different expressions of a psychological obsession with body weight.

Other eating disorders such as eating disorder not otherwise specified or binge eating disorder or childhood eating disorder and eating disorder in athletes have also been observed. We will study about some of these disorders, with particular focus on anorexia and bulimia in this unit. Before we move further, it would be useful to define some common terms that would appear in this unit as given in Table 10.1 in order to facilitate their understanding at this stage.

### Definitions of some common eating disorders

1. Refusal to maintain body weight at or above a minimally normal weight for age and height, (i.e. weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected) .
2. Intense fear of gaining weight or becoming fat even though underweight.
3. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self evaluation, or denial of seriousness of the current low body weight.
4. In postmenarchal females, amenorrhoea i.e. absence of **at least** three consecutive menstrual cycles.

A restricting type or the binge/purging subtype of anorexia nervosa also exist. What is the difference between these two subtypes? Let us consider. During a current episode of anorexia nervosa, if the person has not regularly engaged in binge eating or purging behaviour, the individual may be diagnosed as the restricting type

On the other hand, during the current episode of anorexia nervosa, if the person has regularly been engaged in binge eating and purging behaviour, the anorexia nervosa patient is binge/purge subtype. Though binge eating and purging are characteristic features of bulimia nervosa, as you will soon find out, low body weight is the major factor that differentiates bulimia nervosa from this subtype of anorexia nervosa. Thus according to the established diagnostic criteria, patients who are 15 percent below normal body weight and binge eat or purge are considered to have anorexia nervosa.

Having looked at the diagnostic criteria, let us next review how common is this problem? What is the cause for this disorder and what are the clinical manifestations/ characteristics of anorexia nervosa?

### ***Prevalence/Incidence***

The disorder occurs most commonly in adolescent girls and young women, but adolescent boys and young men may be affected more rarely, as may children approaching puberty and older women up to the menopause. Although the prevalence/ incidence of the disorder is not known with certainty, about 0.5% to 1% of adolescent girls and young women are reported to suffer from anorexia nervosa that meets the full criteria. Its incidence (appearance of new cases) among the 10-19 year old girls has definitely increased during the past decade. Anorexia nervosa appears to be more prevalent in industrialized countries that embrace and idealize a thin body type, but recent reviews suggest that it is increasing even in developing third world countries, such as ours.

What then is the cause for this disorder? Read and find out next.

### ***Etiology***

The exact cause of eating disorders is not known. It is multifactorial in origin in which the personality of the patient, family relationship, socio-cultural factors and may be genetic factors play an important role.

Although the fundamental causes of anorexia nervosa remain elusive, there is growing evidence that interacting socio-cultural and biological factors contribute to its causation, as do less specific psychological mechanism and a vulnerability of personality.

It is possible that the disorders begin when there are disturbed family relationships, e.g., when the parents pretend to be getting along well with each other but are actually highly dissatisfied with their marriage. Such a family may be overprotective, rigid and too goal oriented. Some may have unusual interest in weight, food or shape of the body. The eating disorder for the child in such a family serves as a focus in order to bring control into an otherwise chaotic life. It is not clear how these factors lead to intense fear of being fat that is central to both anorexia and other eating disorders like bulimia about which we shall learn later in this unit.

Psychiatric illnesses like depression and obsessive compulsive behaviour very frequently are found in those with eating disorders, especially bulimia. These abnormalities may predispose to the development of eating disorders. Cultural factors are important. Today everyone wants to be healthy and fit. This may reinforce the fear of fatness in an emotionally unstable person; and may tip the borderline case into frank' disorder.

Occupation may also play a role. Dancers have a prevalence of anorexia

### **NOTES**

## NOTES

nervosa 10 times that of the general population. Some studies show that a genetic component may be involved as well. However, such involvement in the causation of these disorders is considered only minor, if at all it exists. Apart from these, other multidimensional causative factors that lead to anorexia nervosa are: vulnerable personality;

psychological conflicts — individual and family; socio-cultural factors — cult of thinness, hazardous dieting, social class and race and finally genetic and constitutional factors. Next, a review on the clinical manifestations and medical complications follows.

### *Clinical Features and Medical Complications*

Anorexia nervosa, as we have learnt above, is a disorder characterized by deliberate weight loss, induced and/or sustained by the patient. You will notice that the individuals suffering from anorexia nervosa have a typical and distinctive appearance. Their body appearance makes them look younger than their age. The clinical characteristics and the medical complications associated with this disorder are included herewith:

The patients deny hunger, thinness or fatigue despite profound weight loss.

They may be preoccupied with food and may take pleasure in cooking and serving meals for others.

They generally have constipation and are intolerant to cold. Patients are hypothermic and often wear more clothing than is environmentally appropriate.

In severe cases, the bones protrude through the skin, as there is hardly any body fat.

The skin may be dry and scaly.

Palms may be yellow because of carotenaemia (high level of yellow pigment carotene in blood).

Body hair is increased. Frank hirsutism (excessive growth of coarse hair in women).

Oedema may be present.

Parotid glands may be enlarged.

Anorexia nervosa, in fact, constitutes an independent syndrome in the following sense:

- a) the clinical features of the syndrome are easily recognized, so that diagnosis is reliable with a high level of agreement between clinicians, and
- b) follow-up studies have shown that, among patients who do not recover, a considerable number continue to show the same main features of anorexia nervosa, in a chronic form.



**NOTES**

Common physical characteristics include lanugo (covering of fine, soft hair), brittle listless hair; cyanosis (abnormal, bluish colour) of the extremities. Bradycardia below 60 beats per minute and hypotension below 70 mm Hg (systolic) are frequently present. Further, laboratory findings include anaemia, and low serum potassium and albumin levels. Plasma cholesterol is occasionally high but triglyceride levels are normal. Glucose tolerance is abnormal.

A number of hormonal disturbances are present. The disorder is associated with undernutrition of varying severity, with resulting secondary endocrine and metabolic changes and disturbances of bodily function. There remains some doubt as to whether the characteristic endocrine disorder is entirely due to the undernutrition and the direct effect of various behaviours that have brought it about (e.g. restricted dietary choice, excessive exercise, alterations in body composition, induced vomiting and purgation and the consequent electrolyte disturbances), or whether uncertain factors are also involved.

Complications include sudden death because of a cardiac problem. A decline in weight to 35% below the ideal increases the risk of death.

**Table 10.3 : Physical complications of anorexia nervosa**

|                               |                                    |
|-------------------------------|------------------------------------|
| • Amenorrhoea/ loss of libido | • Gastric dilatation               |
| • Hypothermia                 | • Myopathy and neuropathy          |
| • Peripheral oedema           | • Impaired liver function          |
| • Congestive heart failure    | • Delayed puberty (if early onset) |
| • Hypokalaemia                | a Osteoporosis                     |
| • Bone marrow hypoplasia      |                                    |

---

## 10.5 BULIMIA NERVOSA

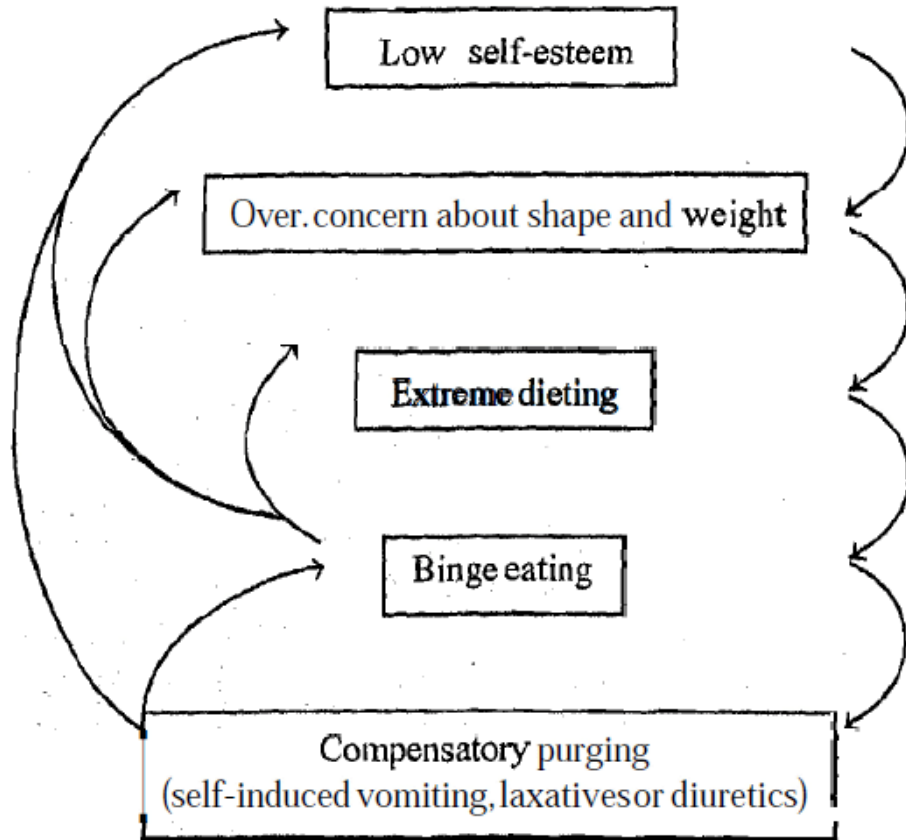
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Bulimia Nervosa, as you may recall studying earlier, is a disorder characterized by episodes of binge eating or very rapid intake of large amounts of high calorie food accompanied by self-induced vomiting. Use of laxatives and diuretics is also practiced to lose weight. Patients fear that they will start gaining weight if they stop purging. It occurs in those who want to eat more but at the same time want to remain thin.

So, what is typical or characteristic feature of bulimia nervosa patient? It is a recurring episode of binge eating followed by one or more inappropriate behaviours to prevent weight gain. These behaviours may include self-induced vomiting, laxative abuse, diuretic abuse, excessive fasting or compulsive exercise.

**NOTES**

The combination of heightened anxiety, low self-esteem, overconcern about body shape, physical discomfort and intense guilt provokes the drive to purge the food self-induced vomiting, excessive exercise or the misuse of laxatives or diuretics, the vicious cycle that maintains binge eating. This perspective on the perpetuation of bulimia nervosa is a cognitive one.



**Figure 10.1: The cognitive view of the maintenance of bulimia nervosa**

***Bulimia Nervosa - Diagnostic Criteria***

Bulimia Nervosa patients, unlike those of anorexia nervosa with binge and purge subtype, are typically within the normal weight range, although some may be slightly underweight or overweight. The common complaints are bloating and flatulence (due to excessive gas production), abdominal pain, constipation and nausea.

The patients are secretive about the eating-vomiting episodes so that family and friends do not know about it. Generally, one episode occurs daily. Calorie dense and high carbohydrate foods like ice cream, cold drinks, bread, jam etc. are eaten in large amounts. Bulimia literally means 'ox-hunger' and the term given to the eating pattern is 'dietary chaos'. Patients tend to suffer from dental caries.

## NOTES

1. **Recurrent** episodes of binge eating. An episode of binge eating is characterized by both of the following
  - a) Eating, in a discrete **period** of time (e.g., within any 2 hour period), an amount of food that is definitely larger than **most** people would eat during **similar** period of time under similar circumstances.
  - b) A sense of lack of **control** over eating during an **episode** e.g., a feeling that one cannot stop eating or control what or how much one is eating.
2. Recurrent inappropriate compensatory behaviour to prevent weight gain such as self-induced vomiting, misuse of laxatives diuretics or other medications; fasting; or excessive exercise.
3. The binge eating and inappropriate compensatory behaviours both occur, on an average, at least twice a week for **3 months**.
4. Self-evaluation is unduly influenced by body shape and weight.
5. The disturbance does not occur exclusively during episodes of **anorexia nervosa**.

Bulimia nervosa, is further characterized as the purging type or the non-purging type. In the purging type, during a current episode of bulimia nervosa, the person regularly engages in self-induced vomiting or the misuse of laxatives, diuretics or enemas.

In the non-purging type, during an episode of bulimia nervosa, the person uses other inappropriate compensatory behaviours, such as fasting or excessive exercise as mentioned earlier, but does not regularly engage in self-induced vomiting or use of laxatives, diuretics etc.

Diagnostic criteria for bulimia nervosa require that at least twice a week for 3 months patient must have episode of consuming a very large amount of food, which a normal person is not capable of eating. There must be evidence that binge eating, which is central to the diagnosis, is followed by self-induced vomiting which is seen in 80% to 90% of persons with bulimia nervosa. Disturbed body image is also an essential feature.

Further, in bulimia, weight fluctuations are common. Patients even though thin, may be well within 15% of the ideal weight, some may even be overweight as informed earlier. About 50% have normal menstrual cycle. Physical findings are generally minimal. It is important to look for scars from self-mutilation in every suspected case.

### ***Etiology***

Bulimia nervosa is a multifaceted disorder with psychologic, physiologic, developmental and cultural components. There may be a genetic predisposition for the disorder. Other predisposing factors include psychologic and personality

## NOTES

factors, such as perfectionism, impaired self-concept, affective instability, poor impulse control and an absence of adaptive functioning to maturational tasks and developmental stressors (e.g., puberty, peer and parental relationships, sexuality, marriage and pregnancy).

Researchers suggest that abnormalities of central nervous system neurotransmitters may also play a role in bulimia nervosa. Furthermore, several familial factors may increase the risk of developing this disorder. For example, researchers have discovered that first- and second-degree relatives of individuals with bulimia nervosa have an increased incidence of depression and manic-depressive illnesses, eating disorders, and alcohol and substance abuse problems.

### *Prevalence/Incidence*

Bulimia nervosa appears to have become more prevalent during the past 30 years. We do not have much data on Indian population related to this disorder, however, as per the American Psychiatric Association, the prevalence of bulimia nervosa in women is 1% to 3%. It is 10 times more common in females than in males. The condition usually becomes symptomatic between the ages of 13 and 20 years, and it has a chronic, sometimes episodic course.

The clinical manifestations and medical complications linked with bulimia nervosa are discussed next.

### *Clinical Features and Medical Complications*

Unlike, anorexia nervosa, in bulimia you will find that symptoms are more difficult to detect because patients are usually of normal weight and are secretive in behaviour. Comorbid mood, anxiety, personality disorders and substance-abuse related disorders, as highlighted in Table 10.5, may also be commonly noted, although it is not clear if the mood disturbances is a function of bulimia nervosa or a separate phenomena. Common sign/symptoms typical of bulimia may include:

#### *Symptoms of Bulimia*

Binges, minimum twice a week for three months

Purging

Menstrual irregularities

Swollen glands

Frequent fluctuations in weight

Inability to voluntarily stop eating/feeling guilty or ashamed about eating

Depressive moods

Persistent over concern with body shape and weight

Overeating in reaction to emotional stress.

**Table 10.5: Psychiatric conditions commonly coexisting with bulimia nervosa**

|                                    |                                   |
|------------------------------------|-----------------------------------|
| <b>Mood disorders</b>              | <b>Anxiety disorders</b>          |
| Major depression                   | Panic disorder                    |
| Bipolar disorder                   | Obsessive-compulsive disorder     |
|                                    | Generalized anxiety disorder      |
|                                    | Post-traumatic stress disorder    |
| <b>Substance-related disorders</b> | <b>Personality disorders</b>      |
| Alcohol abuse                      | Borderline personality disorder   |
| Stimulant abuse                    | Histrionic personality disorder   |
| Polysubstance abuse                | Narcissistic personality disorder |
|                                    | Antisocial personality disorder   |

**NOTES**

The medical complications of bulimia nervosa range from fairly benign, transient symptoms, such as fatigue, bloating and constipation, to chronic or life-threatening conditions, including hypokalemia (low potassium levels), cathartic colon, impaired renal function and cardiac arrest. Serious medical complications of bulimia nervosa are uncommon, but patients may suffer from dental erosion, swollen salivary glands, oral and hand trauma, gastrointestinal irritation and electrolyte imbalances (especially of potassium, calcium, sodium and hydrogen chloride).

Laboratory tests reveal low potassium levels (hypokalaemia) because of vomiting and laxative use. Hormonal changes are less prominent than in anorexia nervosa and may be absent altogether. Complications include aspiration, oesophageal or gastric rupture, or inflammation of pancreas.

Physical complications arising due to inappropriate compensatory behaviour such as self-induced vomiting or use of laxatives etc. may include:

- Cardiac arrhythmias
- Renal impairment from hypokalaemia
- Muscular paralysis
- Urinary infection epileptic seizures
- Tetany (from hypokalaemic alkalosis)
- Swollen salivary glands
- Eroded dental enamel
- Injury to myenteric plexuses of large bowel

Having gone through the clinical features specific to both anorexia and bulimia nervosa, you would have realized that certain characteristics are specific to each one of these disorders, which can help us in diagnosing and differentiating these specific disease conditions.

**NOTES**

**Table 10.6: Anorexia nervosa (AN) and bulimia nervosa (BN) - clinical features**

| <b>Clinical Features</b>         | <b>AN</b> | <b>BN</b>    |
|----------------------------------|-----------|--------------|
| Food avoidance                   | Constant  | Intermittent |
| Overeating                       | -         | +++          |
| Self-induced vomiting or purging | ±         | +++          |
| Weight loss                      | +++       | +            |
| Amenorrhoea                      | +         | +            |
| Dread of weight gain             | ++        | +++          |

Where AN = Anorexia nervosa, BN = Bulimia nervosa

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## **10.6 EATING DISORDER NOT OTHERWISE SPECIFIED (EDNOS)**

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A diagnostic category for eating disorders that fail to meet full criteria for anorexia nervosa or bulimia nervosa is termed as eating disorder not otherwise specified. In fact, according to the American Psychiatric Association, this category is for disorders of eating that do not meet criteria for any specific eating disorder.

It is interesting to note that patients who fall under eating disorder not otherwise specified constitute about 50% of the population suffering with eating disorders. It is equally important to understand that if left untreated, patients with an eating disorder not otherwise specified may develop full-fledged anorexia nervosa or bulimia nervosa.

Besides EDNOS, another specific eating disorder identified is the binge eating disorder. Let us get to understand it and see how it differs from other eating disorders.

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## **10.7 BINGE EATING DISORDER**

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Binge eating disorder is probably the most common eating disorder. In Bulimia, however, episodes of binge eating are followed by inappropriate behaviour such as purging, periods of fasting, or performance of strenuous exercise. People with binge eating disorder, by contrast, do not purge, fast or engage in strenuous exercise after binge eating. Additionally, people with bulimia are typically of normal weight or may be slightly overweight (the purging, etc., have little to no effect on the subject's body fat), whereas people with binge eating disorder are typically overweight or obese. Binge eating disorder is a psychiatric disorder in which a subject:

periodically does not exercise control over consumption of food,

## NOTES

eats an unusually large amount of food at one time,  
eats much more quickly during binge episodes than during normal eating episodes,  
eats until physically uncomfortable,  
eats large amounts of food, even when they are not really hungry,  
always eats alone during binge eating episodes, in order to avoid discovery of the disorder,  
often eats alone during periods of normal eating, owing to feelings Of embarrassment about food, and  
feels disgusted, depressed, or guilty after binge eating.

So can we say that binge eating disorder is compulsive eating? Binge eating disorder is similar to, but it is distinct from, compulsive eating. People with binge eating disorder do not have a compulsion to overeat and do not spend a great deal of time fantasizing about food. On the contrary, some people with binge eating disorder have very negative feelings about food. .

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## 10.8 MANAGEMENT OF EATING DISORDERS

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We shall consider the components of the management of anorexia nervosa and bulimia nervosa together, since the nutritional consequences and nutritional management for both these conditions are on similar lines. However, before that, consideration needs to be given to symptoms of the starvation syndrome i.e. (starvation caused either by food restriction/dieting as in anorexia nervosa or problems related to food absorption as in purging or excessive exercise may mean that insufficient energy is consumed for weight maintenance in bulimia nervosa).

Having looked at the symptoms specific to starvation let us proceed further, The treatment of patients suffering from eating disorders, you would realize, has to be done by a multidisciplinary team comprising of a physician, nutritionist and psychotherapists. Under no circumstances a nutritionist alone, however experienced she/he may be, should try to manage such cases on his own, without consulting experts from the other two disciplines. The increased psychological stress and the medical complications that accompany refeeding necessitate the presence of the team.

The treatment of eating disorders can be said to have three components.

- A) Psychological Management
- B) Medical and Biochemical Management, and
- C) Nutritional Management

### ***Psychological Management***

All anorexia nervosa or bulimia nervosa patients are resistant to any kind of

## NOTES

therapy and hospitalization may be a life saving measure, especially with anorexia nervosa patients. The intense fear of becoming fat and the disturbed perception of their weight status is what make treatment difficult. The supportive care by an understanding physician/ psychiatrist is most important. The patient needs to be repeatedly assured by the doctors that the treatment will not make her fat. The patient may be given some reward, may be in terms of freedom/ home-visit as she shows some gain in weight or even when she consumes extra food.

The patient needs to be told in a calm but realistic manner about the risks that accompany starvation including sudden death. You will recall reading earlier that sudden death is a real danger if the patient is below 35% of the ideal weight and if the weight loss has been rapid. For bulimic patients hospitalization may be required only for treatment of medical complications like aspiration or electrolyte imbalance. The short-term goal for treatment of bulimics may be stoppage of gorging-regurgitation cycles so that the load of food ingested may be limited. This also minimizes the chance of aspiration or gastric rupture. Psychiatric treatment is required because depression and anti social behaviour are common in bulimia.

The use of drugs remains controversial. The use of antidepressant therapy in both the disorders needs to be considered on individual merits of the case.

### **Biochemical and metabolic problems and their management**

Hypokalaemia (low concentration of potassium ion in the blood), we learnt earlier, is a problem caused due to self-induced vomiting and/or laxative misuse. For this potassium supplementation is often required. Hyponatraemia (deficiency of sodium) may result from diarrhoea and vomiting, misuse of diuretics or excessive intake of water. Rapid correction of hyponatraemia and the use of hypertonic fluids are hazardous. Hence, medical treatment becomes important.

Further, in patients with eating disorder, iron, folic acid deficiency may occur and those who have been avoiding animal foods may be deficient in vitamin B12 as well. However, supplemental iron may be dangerous in the early stages of refeeding. Some people with eating disorders are also deficient in zinc. Zinc deficiency may cause altered taste, smell, appetite, as well as, a variety of neuropsychiatric symptoms. Taking zinc supplements may help. Recent studies with bulimia patients indicate that zinc supplements seem to reduce their obsession with weight and concern with body image.

A significant proportion of patients are deficient in thiamin and the increase in carbohydrate metabolism which occurs during refeeding may exhaust inadequate thiamin reserves. The early stages of refeeding are a high-risk period for biochemical, fluid balance and cardiovascular abnormalities and patients should be monitored closely. Patients at particular risk include those whose weight is very low, those who have had previous biochemical abnormalities or purge, and those with concurrent medical conditions such as diabetes, infection and major organ failure. Electrolyte disturbances are most likely to occur during the first 1-2 weeks



of refeeding. There is a risk of hypophosphataemia (deficiency of phosphate) and acute thiamin deficiency when beginning refeeding. Abnormal liver function tests can occur at presentation or during refeeding. This appears to be self-limiting but other causes of liver dysfunction should be excluded. Delayed gastric emptying results in early satiety and sensations of abdominal fullness or bloating

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## **10.9 NUTRITIONAL MANAGEMENT OF EATING DISORDERS**

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Good nutritional management of patients with eating disorders requires attention to a number of areas. It is important to have a nutritional assessment of the patient through diet history, weight history, as well as, assessment of biochemical, metabolic and anthropometric measurements. A nutritional assessment form for eating disorders. Initial dietary assessment should be part of the overall assessment of patients with eating disorder. initial assessment should cover only those aspects essential to immediate management decisions. may include:

- recent change in eating habit
- rate of weight loss
- binge eating episodes
- vomiting and laxative misuse
- gastrointestinal and bowel function
- fluid intake
- restrictions on the variety of acceptable food
- other conditions that may require dietary management (such as diabetes, dehydration, hypophosphataemia etc.)
- presence of conditions that may affect nutritional requirements (such as infection or any trauma, growth)
- exercise schedule
- menstrual history
- use of vitamin/mineral supplements
- use of alcohol.

The aims of dietetic treatment should be to:

- establish a normal pattern of food intake,
- encourage a nutritional intake appropriate to the individual's needs, and
- maintain a body weight within a normal range.

Let us review the nutritional management of anorexia nervosa and bulimia nervosa, separately next.

NOTES

Eating Disorder Assessment Form

|                                                                                                                                |                                                                                                                                               |                                                       |
|--------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------|
| Date of Birth .....                                                                                                            |                                                                                                                                               | OTHER SUPPLEMENTS .....                               |
| DIAGNOSIS                                                                                                                      | Hospitalizations for Eating disorder:                                                                                                         | SUGAR AND FAT SUBSTITUTES: .....                      |
| <ul style="list-style-type: none"> <li>● Anorexia Nervosa</li> <li>● Bulimia Nervosa</li> <li>● Eating Disorder NOS</li> </ul> | <ul style="list-style-type: none"> <li>● In-patient</li> <li>● Day Patient</li> <li>● Out-Patient</li> <li>● Intensive Out Patient</li> </ul> | MISCELLANEOUS: Chewing gum: .....                     |
| WEIGHT HISTORY                                                                                                                 |                                                                                                                                               | Hard candy: .....                                     |
| Wt Loss # lb ..... from ..... to .....                                                                                         |                                                                                                                                               | Condiments: .....                                     |
| Minimum weight at current height .....                                                                                         |                                                                                                                                               | BINGES: # per day # per week                          |
| Maximum weight at current height .....                                                                                         |                                                                                                                                               | Duration per episode .....                            |
| IBW: ..... %IBW: ..... %Wt loss: ..... BMI% .....                                                                              |                                                                                                                                               | Binge foods: .....                                    |
| ANTHROPOMETRIC PROFILE                                                                                                         |                                                                                                                                               | Approximate kcal/binge: .....                         |
| Skinfolds(mm): .....                                                                                                           |                                                                                                                                               | SELF-INDUCED VOMITING:                                |
| Triceps..... Biceps..... Subscapular...                                                                                        |                                                                                                                                               | Times per day..... Method: .....                      |
| Suprailliac: .....                                                                                                             |                                                                                                                                               | LAXATIVES:                                            |
| Sum of sites (mm): ..... %Body fat .....                                                                                       |                                                                                                                                               | Type/brand: ..... Amount: .....                       |
| TST/o.....                                                                                                                     |                                                                                                                                               | Duration of use..... Frequency of use.....            |
| MAC(cm): ..... MAMC(cm): .....                                                                                                 |                                                                                                                                               | DIURETICS: Type..... amount.....                      |
| MAMC% .....                                                                                                                    |                                                                                                                                               | Duration of use..... Frequency of use.....            |
| BODY IMAGE .....                                                                                                               |                                                                                                                                               | EXERCISE:                                             |
| FULL ALLERGIES .....                                                                                                           |                                                                                                                                               | Type.....                                             |
| 24-HOUR RECALL: .....                                                                                                          |                                                                                                                                               | Minutes/day ..... times/week .....                    |
| .....                                                                                                                          |                                                                                                                                               | Purpose of exercise.....                              |
| .....                                                                                                                          |                                                                                                                                               | MENSTRUAL HISTORY:                                    |
| .....                                                                                                                          |                                                                                                                                               | Age of menarche.....                                  |
| .....                                                                                                                          |                                                                                                                                               | Last menstrual period.....                            |
| FLUID INTAKE .....                                                                                                             |                                                                                                                                               | MEDICATIONS (Prescript on and over-the-counter) ..... |
| VITAMIN/MINERAL SUPPLEMENTS: .....                                                                                             |                                                                                                                                               | BOWEL FUNCTION : .....                                |
| .....                                                                                                                          |                                                                                                                                               | .....                                                 |
| .....                                                                                                                          |                                                                                                                                               | .....                                                 |
| .....                                                                                                                          |                                                                                                                                               | .....                                                 |

Figure 10.2 : Eating disorder assessment form

10.9.1 Nutritional Management of Anorexia Nervosa

The overall goal of nutritional rehabilitation of anorexia nervosa patients is to restore weight, normalize eating pattern, achieve normal perception of hunger and satiety, and correct biological and psychological sequelae of malnutrition.

To achieve this goal, we need to understand that in the severely malnourished patient, two separate but linked processes occur. First, inadequate food consumption leads to wasting and functional changes in all tissues. Second, the general metabolic response to infection, trauma or other stress results in further specific nutrient losses and cellular damage. Now starting to eat again after a period of prolonged starvation, what we call as the 'refeeding syndrome' can precipitate problems and complications in an anorexic patient. Therefore, the first step has to be to repair the machinery, with tissue repletion being a secondary consideration during the early phase of treatment. The nutritional management of severe anorexia nervosa is therefore, considered in terms of three consecutive phases:

**NOTES**

Resuscitation: First, identify and correct medical emergencies such as hypothermia, hypoglycaemia, electrolyte disturbance, dehydration and cardiovascular function as far as possible. Infections may also be treated, Repair: the tissue/organ functions cannot be restored unless the cellular activity has been repaired. In this context, the correction of multiple specific nutrient deficiencies, needs to be corrected, and Repletion: the ultimate objective of treatment is to return body composition to normal. But, abnormal body composition can only be corrected safely when the cellular machinery has been adequately repaired, Any aggressive attempts to drive weight gain at an early stage of treatment or correction of abnormal blood biochemistry are potentially dangerous. Hence, slow and systematic repletion and treatment regimen needs to be considered.

Clinical experience suggests that many of the untoward consequences of refeeding can be minimized or avoided by starting the patient on relatively small amounts of food and increasing the quantities progressively. Initial intake should be sufficient at least to prevent further weight loss. Thereafter, once the body is repleted, we need to provide for initial weight gain phase followed by controlled weight gain and finally weight maintenance phase. The guideline for nutrient/diet therapy and nutrient intake for anorexia nervosa patients therefore include:

***A. Calorie Intake***

Initial Intake: For refeeding and for all but the most severely ill patients intake levels should usually start at 30-40 Kcal/kg/per day i.e. approximately 1000 Kcal-1600 Kcal/ day. This level of intake should be continued until it can be confirmed that gut function is normal (i.e. bowel sounds are present) and that water overload, if present, is beginning to resolve. The latter is indicated by weight stabilization and normally occurs within 7—10 days. Thereafter, food intake should be increased as discussed next.

Weight gain phase: The food intake can be increased as quickly as the level of supervision and support will allow. Intake may have to be increased to as high as 70-100 Kcal/kg per day for some patients during the weight gain phase. A weekly weight gain of 0.5 -1.0 kg is generally regarded as optimum. There is some preliminary research evidence that a minimum weight gain of 0.5 kg per week results in greater weight gain at discharge than use of a higher minimum. A gain of 1 kg per week requires an energy intake of 1000 Kcal daily above the maintenance requirement.

An intake of 2200-2500 Kcal daily will promote weight gain of 0.5-1.0 kg per week in most patients. The rate of gain will slow down as weight increases, owing to an increase in metabolic rate and physical activity. It may be appropriate to increase energy intake to compensate for this or to allow a slower rate of weight gain in order to facilitate stopping at the agreed maintenance figure. This is followed by the weight maintenance. phase.

Weight maintenance phase: The intake level during weight maintenance for adults

and as needed in children and adolescents for further growth and maintenance should be set at 40-60 Kcal/kg per day.

### ***B. Proteins***

To ensure adequacy, minimum protein intake should equal the recommended dietary intake for age and sex in g,4Kg ideal body weight. A protein intake in the range of 15-20% of total calories is recommended. Protein sources of high biological value need to be included in the diet of the patient.

### ***C. Carbohydrates***

Carbohydrate intake in the range of 50-55%of calories is well tolerated. It is important to include sources of insoluble fibre for optimal health and for relief from constipation.

### ***D. Fats***

A dietary fat intake in the range of 25-30% of calories is recommended. Patients may have an aversion to fat, which rnakes weight gain difficult. Fat may therefore be included in the diet in a disguised form. For example, giving whole milk instead of toned li-lk will help..

### ***E. Micronutrients***

It is advisable to include one 100% RDA multivitamin tablet with minerals. The use of prophylactic thiamine supplements in oral form is recommended for in-patients and those undergoing rapid weight gain. In the absence of data on the appropriate dose of thiamine, it is recommend to give 25 mg per day; in cases of confirmed deficiency, higher dosages may be required. Riboflavin deficiency may cause angular stomatitis and iron deficiency causes anaemia. Vitamin C deficiency can cause bleeding gums. These problems may need specific nutritional supplementation. Vitamin D requirements are higher than average in anorexia nervosa, owing to the risk of osteoporosis, and there is an argument for giving vitamin D supplements as part of refeeding.

Calcium rich foods should also be included because of risk of osteoporosis and osteopaenia. As discussed above, the patient must be encouraged to increase food intake gradually and decrease energy output to achieve a positive balance. A nasogastric tube may be needed but is not the preferred method and majority of patients can be fed orally. To ensure intake of sufficient calories, the feedings should start early in the day. Initially, the intake is generally low and patient can tolerate three meals per day, may be without abdominal distention and discomfort. But as the calorie prescription increases (>3000 Kcals may be required) the number of feedings must be increased. This may result in guilt feedings in patients because they feel that they are snacking between meals. It is wise to introduce calorie dense, may be commercial liquid supplements between meals which are easier

## **NOTES**

**NOTES**

to discontinue when the desired weight is reached, During treatment daily food record and caloric intake should be recorded along with eating behaviour, time of the meal and the meal consumed, food/drink eaten and their amounts etc.

In addition, all the foods eaten at a binge and the time and method of purging also needs to be noted down whenever they occur to further plan the treatment and monitor the course of the disorder. In addition to the food diary, record of exercise undertaken should also be maintained to evaluate energy balance. Body weight must be checked daily when patient is hospitalized and later once every one or two weeks after discharge.

Having gone through the nutritional rehabilitation described above you would realize that management of anorexia nervosa is complex and require different treatment modalities at different stages of illness and recovery. Next, let us learn about nutritional rehabilitation of bulimia nervosa.

### 10.9. 2 Nutritional Management of Bulimia Nervosa

Bulimia nervosa, we know, is characterized by the recurring episode of binge eating followed by one or more inappropriate behaviour to prevent weight gain. Therefore, the initial attempts in the treatment of bulimia are aimed at correcting the dietary chaos by breaking the gorging-regurgitation cycles. Instead of weight loss which is the ultimate goal, stabilization of weight should be aimed at in the beginning. Generally 1200-1500 Kcals are prescribed at first with the same nutrient prescription as for anorexia nervosa. A standard multivitamin and mineral supplement is given initially. In general, a balanced diet providing 50-55% of the calories from carbohydrate, 15-20% calories from proteins and 25-30% from fat is reasonable. Small amount of dietary fat may be encouraged at each meal. Fat may be better tolerated when not visible as in foods like whole milk and cheese. Further, it would be beneficial to include more sources of essential fatty acids in the diet. It is important to remember that patients suffering from bulimia nervosa are likely to remain on low-calorie intake for longer period as compared to anorexic counterparts.

Although most patients with bulimia are normal to overweight, they may be hypermetabolic. This aspect must be considered when prescribing the calorie intake. [If a low metabolism is suspected, initial calorie prescription may be equal to 100% of the Harris-Benedict predicted REE. Typically, this is around 1500 Kcal for adults., You may recall studying about the Harris-Benedict equation in Unit 4 earlier. We learnt that this equation can be used to calculate resting energy expenditure (REE), for men and women, along with the usual multiplication factor to provide adequate calorie intake as given herewith:

*Calorie requirements/day*:  $1.25 \times \text{REE}$  (for each  $1^{\circ}\text{C}$  above  $37^{\circ}\text{C}$  add 10% extra allowance)

*Women REE* =  $655 + (9.6 \times \text{weight in kg}) + (1.85 \times \text{height in cm}) - (4.7 \times \text{age in years})$

*Men REE* =  $66 + (13.7 \times \text{weight in kg}) + (5.0 \times \text{height in cm}) - (6.8 \times \text{age in years})$

This equation can be used to prescribe the calorie intake for weight maintenance in bulimia nervosa patients.

The patients of bulimia, because of the binge eating and purging behaviour in the past fail to recognize hunger and satiety signals. These biological cues get strengthened by regular meals and prescribed in-between snacks at a reasonable calorie level. Patients tend to digress from the prescribed pattern when a binge episode occurs during treatment. An important part of breaking this cycle is to get the individual to monitor their intake through completing a food diary.

**NOTES**

**Table 10.7: Example of a food diary**

| Time/Meal         | Food/Drink Eaten and Amount                                                                        | Binge/Vomit/Laxatives/Eating Behaviour/Food Tolerated | Comment/Feelings                                                                                                            |
|-------------------|----------------------------------------------------------------------------------------------------|-------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------|
| Breakfast         | Nothing                                                                                            | -                                                     | Not hungry                                                                                                                  |
| Mid Morning       | Tea - 2 cups                                                                                       | -                                                     | Need something to fill my stomach.<br>Really busy at work so no time to eat.                                                |
| Lunch (1.30 p.m.) | 2 dry chapatti,<br>1 katori dal, small cube of cottage cheese,<br>1 tomato, can of diet cola       |                                                       | Very hungry, feel as if I could eat more but must not.                                                                      |
| Tea (5.00 p.m.)   | Two gulabjamun                                                                                     | Vomited                                               | Someone's birthday in the office so couldn't refuse. Feel really guilty and had to be sick.                                 |
| Dinner            | 4 pieces of pizza,<br>2 packets of crisps,<br>2 bowls of ice cream,<br>6 snack size chocolate bars | Binge!! Vomited and took 10 laxatives                 | Couldn't decide what to have for dinner, so started on pizza.<br>Could not stop this binge at any cost.<br>I feel terrible. |

Completing a food diary as mentioned above is a valuable aspect of treatment by identifying areas of difficulty and allowing progress to be monitored, as well as, enabling the individual to reveal problematic thoughts and feelings at the time they are consuming food. This can then be used as the basis for planning meals to lessen anxiety around eating. Patience and support from the dietitian and family members are crucial for helping the patients in retaining the positive changes regarding attitudes and eating habits. An important goal for nutritional management, therefore, is to establish the individual on a regular pattern of eating. Often, normal cues for hunger and satiety are disrupted through repeated cycles of binge and restrictive eating so encouraging a regular meal pattern also helps the sufferer to begin to identify hunger and fullness again. They should be encouraged to eat regular meals and snacks and to maintain this pattern of eating even after a binge.

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Each meal or snack should be based around carbohydrate, with moderate amounts of protein Foods and vegetables and fruit. They should be encouraged to include non-diet foods and to include foods containing fat. It is also worth getting them to compile a list of foods normally avoided or associated only with binges and to encourage them to include these within their meal pattern, when they feel able to do so. The amount of food needed to meet energy needs is greater than that needed to consume sufficient nutrients. Thus, consumption of some energy dense, less nutritious food should be encouraged. A minimum intake of 1500 Kcal, as discussed earlier, is usually an appropriate level to begin with, increasing 10 an intake corresponding to the estimated average requirement for the particular patient as recovery proceeds.

Further, a detailed weight history should be carried out to include current, highest, lowest and ideal weights, and it should be stressed that recovery cannot be accomplished if the sufferer is trying to maintain a weight below normal. Thus, those with a pre- morbid history of obesity may have to accept that they will need to reach a weight that is higher than they would like to be. Weight stabilization should be an initial emphasis, particularly for those experiencing weight fluctuations. Even among patients of normal weight, nutritional counseling can be used to accomplish the desired results. There is some evidence that treatment/ management programmes that include dietary counseling as part of the programme are more effective than those that do not. Let us see how.

### **Nutrition counseling/education — an important component of management of eating disorders**

Nutrition counseling can be used to accomplish a variety of goals, such as reducing behaviours related to the eating disorder, minimizing food restrictions, correcting nutritional deficiencies, increasing the variety of foods eaten and encouraging healthy and not excessive exercise pattern. Nutrition Education is an important aspect of the treatment. The patients report an extremely good knowledge of foods and nutrition. For example, most anorexia nervosa patients tend to argue against adding calorie rich foods to their diet because they have known their energy values. However, their interpretation of facts may be faulty because of the nature of their illness.

They need to be educated about the impact of malnutrition on growth, development and behaviour. They should be made aware of the importance of balanced diet, food guide pyramid and also the ineffectiveness of vomiting, laxatives and diuretics in long-term weight control. Education may be imparted individually or in a group setting. Treatment of anorexia-bulimia syndromes is a long-term affair. Many a time's failures are experienced. It requires great perseverance and consistent effort by the patient, the family and the dietitian/physician for effective outcome. The prognosis of these eating disorders is highlighted next.

Prognosis: About 50% of the patients recover fully from anorexia nervosa and achieve normal weight, 30% improve but have a partial recovery and 20% will have

## NOTES

lifelong problems with eating pattenis. Older age of onset, long duration of illness, extreme weight loss and significant depression result in poor prognosis. Bulimia has even poorer prognosis because of medical dangers of gorging and severe psychiatric disturbances. The suicide rate also is high in bulimicpatients. Almost 40% of treated patients remain bulimic after one and a half year of treatment. About 2/3 experience relapse within a year of recovery. The outcome criteria e.g., weight, food intake, proper body image, menstruation and social, psychological and sexual adjustments must be assessed for a number of years after recovery. Early intervention and better treatment strategies today have helped in reducing the mortality from eating disorders.

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### 10.10 LET US SUM UP

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In this unit we learnt about the prevalence, classification, causative factors, metabolic changes, clinical manifestations and management of various problems related to eating disorders. Anorexia nervosa and bulimia nervosa are the two eating disorders, which may have life threatening consequences. Patients with eating disorders, we learnt, display a broad range of symptoms that frequently occur along a continuum between those of anorexia nervosa and bulimia nervosa. The care of patients with eating disorder involves a comprehensive array of approaches including, psychiatric, medical and nutritional management. They necessitate treatment by a multidisciplinary team and not by nutritionist alone. The firm approach by the dietitian/treating doctors and regular follow up monitoring for many years afterwards helps in the recovery of patients,

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### 10.11 GLOSSARY

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|                     |                                                                                                                  |
|---------------------|------------------------------------------------------------------------------------------------------------------|
| <b>Binge eating</b> | : an episode of excessive eating accompanied by a sense of loss of control over the eating process.              |
| <b>Bradycardia</b>  | : refers to the slowness of the heart rate, usually fewer than 60 pulse beats per minute in an adult human.      |
| <b>Carotenemia</b>  | : presence in the blood of yellow pigment carotene from excessive intake of carotene rich vegetables and fruits. |
| <b>Cyanosis</b>     | : an abnormal bluish colour of the skin or mucous membranes.                                                     |
| <b>Hirsutism</b>    | : an excessive growth of coarse hair particularly in women.                                                      |
| <b>Lanugo</b>       | : a covering of fine, soft hair, as on a leaf, an insect, or a newborn child.                                    |



|                        |                                                                                                                            |
|------------------------|----------------------------------------------------------------------------------------------------------------------------|
| <b>Life expectancy</b> | : a statistical measure of the average of the remaining life time of an individual in the given group.                     |
| <b>Overweight</b>      | : being too heavy for one's height; a BMI of 25 to 30 kg/m <sup>2</sup>                                                    |
| <b>Purging</b>         | : methods intended to reverse the effects (f binge eating like self-induced vomiting, use of laxative, enema or diuretics. |

## NOTES

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### 10.12 CHECK YOUR PROGRESS

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- 1).What are the two main eating disorders? Discuss their etiology.
- 2). Enumerate any five clinical features of anorexia nervosa.
- 3). List the signs/symptoms associated with Bulimia. Also mention any two physical complications.
- 4). What is the diagnostic criterion for eating disorders not otherwise specified?
- 5). How is binge eating disorder different than bulimia nervosa?
- 6). What is the starvation syndrome?
- 7). List the three components of the treatment of eating disorders. Why is medical management important?

## NUTRITIONAL MANAGEMENT OF CORONARY HEART DISEASES

### STRUCTURE

- 11.1 Learning Objective
- 11.2 Introduction
- 11.3 Coronary Heart Diseases (CHD)
- 11.4 Common Disorders of Coronary Heart Diseases and their Management
- 11.5 Prevention of Coronary Heart Diseases
- 11.6 Let Us Sum Up
- 11.7 Glossary
- 11.8 Check Your Progress

### 11.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- 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 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 overweight can be one of the risk factors The cardiovascular disease (C V D). Risk factors as

we know are conditions and habits that have been demonstrated 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.

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### 11.3 CORONARY HEART DISEASES (CHI)

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Coronary heart disease is a broad term comprising of a spectrum of diseases associated with disorders 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 Coronary Heart Diseases (CHI)**

| <b>Disorders/Complications of CHD</b> | <b>Definition</b>                                                                                                                  |
|---------------------------------------|------------------------------------------------------------------------------------------------------------------------------------|
| 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.                                                                                                 |

## NOTES

|                                       |                                                                                                                                  |
|---------------------------------------|----------------------------------------------------------------------------------------------------------------------------------|
| <b>Angina Pectoris</b>                | A characteristic pain or discomfort in the chest.                                                                                |
| <b>Myocardial Infarction</b>          | An area of necrosis (dying/dead cells of the myocardium) in a tissue.                                                            |
| <b>Congestive Cardiac Failure</b>     | A clinical syndrome caused by heart disease characterized by breathlessness, chest pain and abnormal sodium and water retention. |
| <b>Rheumatoid Heart Disease (RHD)</b> | 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.

### 11.3.1 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 prosperous, 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.

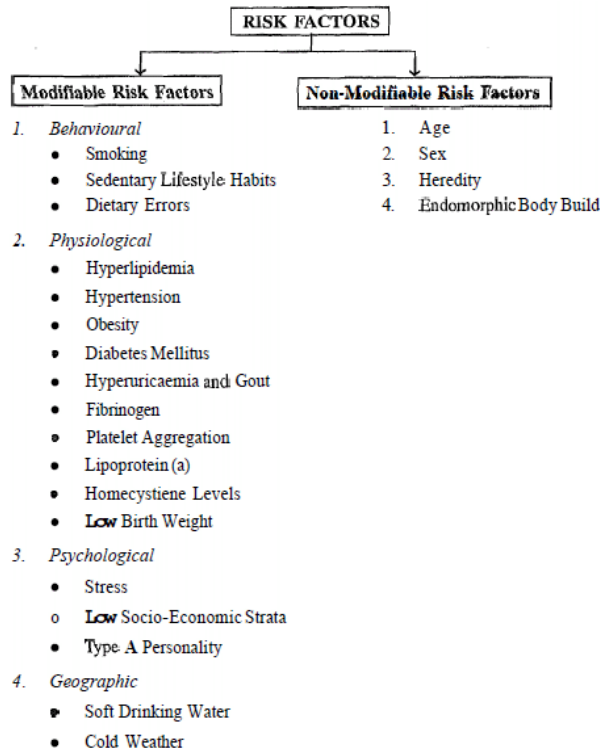
But why is the incidence of coronary heart diseases increasing at a rapid rate every year? Are there any factors, which may predispose Asians particularly Indians to an early onset of heart diseases? Let us read further to learn about the causative factors associated with the development of heart diseases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

### 11.3.2 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 a role in causing or increasing the risk of getting the cardiovascular disease are called risk factors.

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.



**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 is 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

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- 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 cardio- myopathies. You shall study about diabetes and its association 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-cholesterolemic 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 abnormalities all 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 hypercholesterolemic individuals.
  11. Psychological, social, cultural and factors indirectly influence the risk of cardiovascular diseases by their effects on kind of food and quantity of food consumed, cigarette and alcohol consumed. Highly 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 homocysteine 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.

### **11.3.3 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.

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## **11.4 COMMON DISORDERS OF CORONARY HEART DISEASES AND THEIR MANAGEMENT**

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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.

## NOTES

### 11.4.1 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 et al (1967).

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 Lipoproteins (LDL):** This type of lipoprotein is most atherogenic 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 total 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 A1, AII, B48, C1, CII, CIII, D and E. The apoproteins A1 and AII 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 above-mentioned parameters. As a dietician, you should be able to interpret and utilize this information for planning the dietary regime of the patient.

| Box 11.1 Classification of Lipid/Lipoprotein Levels |                |
|-----------------------------------------------------|----------------|
| <b>Classification of Total Cholesterol</b>          |                |
| Total Cholesterol (mg/dl)                           | Interpretation |
| < 200                                               | Desirable      |
| 200 - 239                                           | Borderline     |
| ≥ 240                                               | High           |
| <b>Classification of Triglyceride levels</b>        |                |
| Triglyceride (mg/dl)                                | Interpretation |
| < 150                                               | Normal         |
| 150 - 199                                           | Borderline     |
| 200 - 499                                           | High           |
| ≥ 500                                               | Very High      |
| <b>Classification of HDL-c *</b>                    |                |
| HDL-c (mg/dl)                                       | Interpretation |
| < 40                                                | Low            |
| 40 - 60                                             | Normal         |
| ≥ 60                                                | High           |
| <b>Classification of LDL-c**</b>                    |                |
| LDL-c (mg/dl)                                       | Interpretation |
| < 100                                               | Optimal        |
| 100 - 129                                           | Near Optimal   |
| 130 - 159                                           | Borderline     |
| 160 - 189                                           | High           |
| ≥ 190                                               | Very High      |
| <b>Classification of cholesterol: HDL-c*</b>        |                |
| 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      |

## 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.

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1. **Environmental factors:** These include diets high in saturated fats, excessive calorie intake, alcohol intake and sedentary 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 xanthoma 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 cholesterol (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

-Alcohol

-Uncontrolled diabetes

-Hypothyroidism

-Genetic

-Liver disease

-Drugs

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 Discascs 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

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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 (f 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 (f 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 atherogenic 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: 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, <b>bajra</b> , green leafy vegetables, <b>methi</b> , mustard (rai), <b>almonds</b> , Black gram, cow pea (lobia), <b>rajmah</b> 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 IVocalories 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. > 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 (&100 g)**

|                              | Saturated | 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       |
| Olive oil                    | 13        | 76               | 10                  | < 0.5 | Monounsaturated                  |
| Groundnut                    | 24        | 50               | 25                  | < 0.5 | 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 Polyunsaturated         |
| 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                  |

**NOTES**

***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 carbohydrates in 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 get 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

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

| <b>Type of Fibre</b>    | <b>Water Insoluble<br/>(Cellulose)</b>                                                       | <b>Water Soluble<br/>(Gums, pectins, mucilages)</b>                                                                                                                                                                                        |
|-------------------------|----------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Benefits reduces</b> | 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. |
| <b>Source</b>           | Whole wheat products, Bajra, Ragi, Maize, Green leafy vegetables, fruits with skin and seeds | Oats, whole pulses, whole grains, apples, pears, citrus fruits and vegetables like potato, bhindi, Soya 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

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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. Soybeans are a good source of fibre and soya proteins have estrogenic effect, which causes lipid lowering. A recent analysis of 38 completed trials showed the beneficial effects 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 fact that plant origin foods, which are good sources of protein, are generally rich sources of dietary fibre, have low amounts of saturated fat 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. Nutritional Management of Coronary Heart Diseases (vitamins) that scavenge cell-damaging free radicals and act as antioxidants. 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 soyabean (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 soyabean 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 great variety of antioxidants and free radical scavengers for different purposes and to protect 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.



### ***Dietary guidelines for hyperlipidemic patients.***

- Calories: to maintain ideal body weight.
- Carbohydrates should constitute 55-65% of calories with emphasis on
- polysaccharides (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 to 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 hyperlipidemic 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, soybean, 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)
- reducing portion size

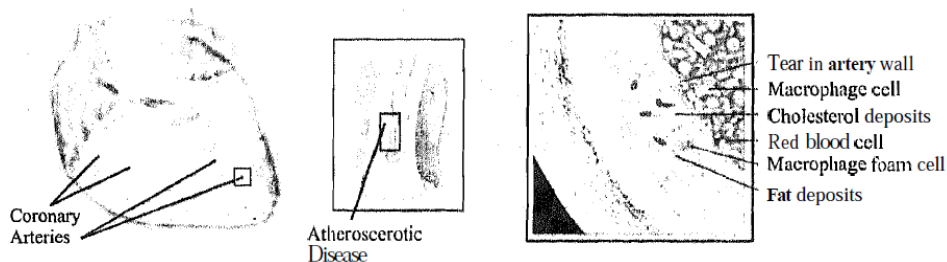
## NOTES

### 11.4.2 Atherosclerosis: A Coronary Artery Disease

Atherosclerosis is an arterial lesion characterized by patchy thickening of the intima (innermost coat 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

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 continuum of as fatty streaks, intermediate lesions, fibrous plaques and complicated lesions. There are 5 phases to atherosclerosis:

- Phase 1 : Asymptomatic phase, consists of fatty streaks which are non-obstructive, lipid filled 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/myocardial infarction and even sudden death.
- Phase 5: Fibrotic or occlusive lesion. Large thrombi can cause serious acute defects.



**Figurem 1.2: Progression of plaque in coronely artery**

## ***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 are 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 the rate of atherosclerosis, increased CAD risk, On the other hand, regular exercise is seen to be protective.
6. **Factors causing endothelial damage:** Elevated blood homocysteine (genetically determined) and viral infections of lungs could damage the endothelial and cause injury and hence lead to atherosclerosis.

## ***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 the 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

**NOTES**

**11.4.3 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-OOI), in Unit 4, is the systolic sound, which occurs when the heart contracts 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 idea about normal and high blood pressure range. Table 11.5 presents the classification given 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**

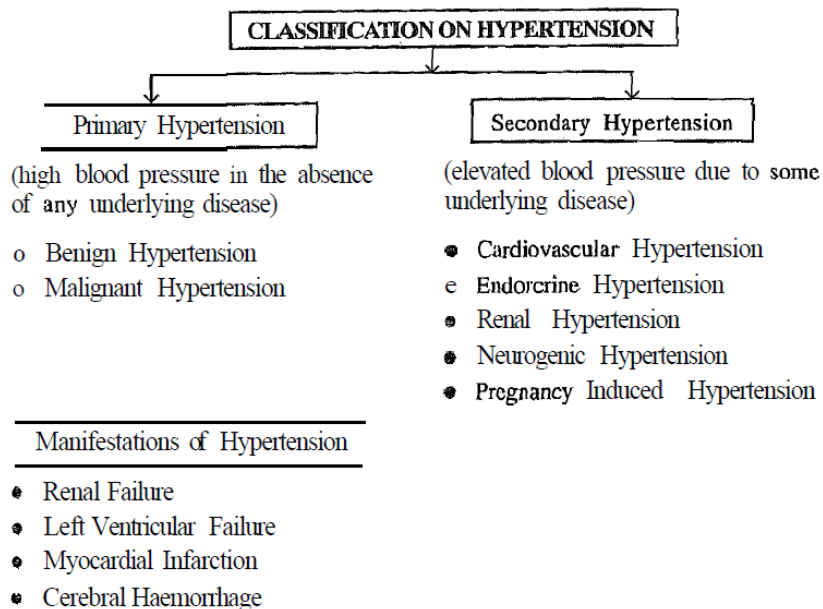
| Blood pressure range SBP/DBP | Classification                  |
|------------------------------|---------------------------------|
| 120/80                       | Normal (optimal)                |
| 120-129/80-84                | Prehypertension (normal)        |
| 130-139/85-89                | Prehypertension (Borderline HT) |
| > 140/90                     | Hypertension                    |
| Stage I 140-159/90-99        | Hypertension (Stage I)          |
| Stage II 160-179/100-109     | Hypertension (Stage II)         |
| Stage III > 180/110          | Hypertension (Stage III)        |

High blood pressure is one of the leading causes of kidney failure, also commonly called end-stage renal disease (ESRD). Major complications of hypertension includes:

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

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

cause and 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: Classification and manifestation of hypertension**

Hypertension can be damaging due to hvo effects:

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

## NOTES

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 dueno increase in muscle formation). As hypertension becomes severe, the heart becomes weaker,

High bloodpressure 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 tie 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 believed that there is polygenic inheritance and when environmental factors are not healthy, hypertension is precipitated.
2. Body weight and height: Hypertension inc • ases with increase in the weight and height. Hence those who are obese have higher blood pressure values. Increase in BNdI increases hypeffension.
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.  
Nutritional Management of Coronary Heart Diseases
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 nutritional 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:** A normal 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 dyslipidemia for details). **Carbohydrates:** About 60-65% energy should be provided from carbohydrates which are polysaccharides (complex carbohydrates) rather than simple sugars (monosaccharides and disaccharides),

**NOTES**

**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                                                                                                                                                                                                   | 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**

| <b>Modification</b>                                | <b>Recommendation</b>                                                                                                 |
|----------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------|
| 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                                  | <b>Engage</b> in regular aerobic physical activity such as brisk walk (at least <b>30</b> minutes per day, most days) |
| Alcohol Consumption                                | Limit to no more than two drinks per day                                                                              |

#### 11.4.4 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.

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### *Symptoms*

The pain of angina is usually over the center of the chest (below the sternum) but can be felt from epigastrium 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.

### *Treatment and dietary management*

Proper and careful treatment of the underlying cause (usually dyslipidemia, advanced atherosclerosis or severe 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]

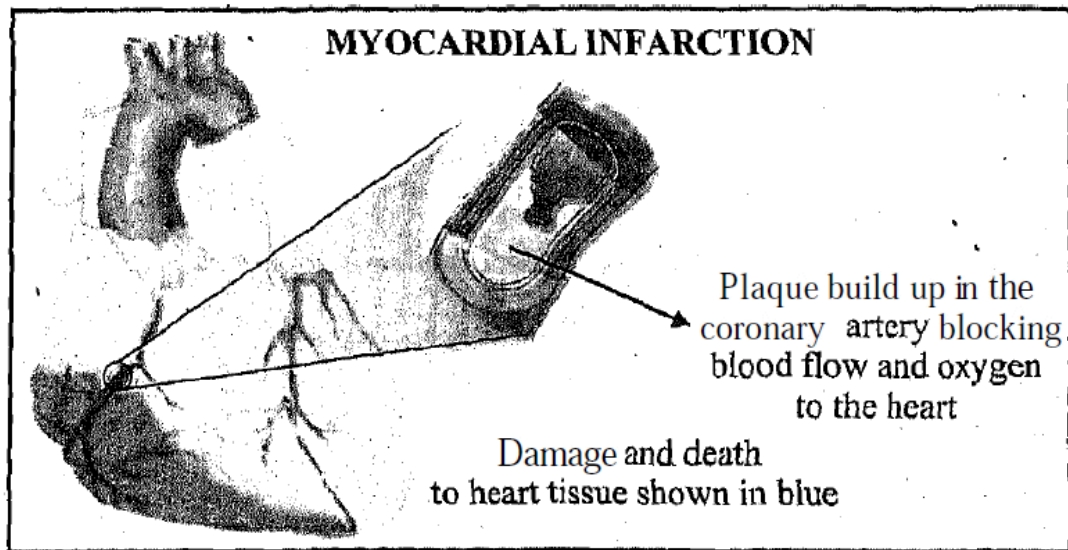
The nutrient requirements here are the same as discussed earlier, however, to sum up it can be said that we need to restrict calories from total fats (particularly saturated fats) and from simple carbohydrates; avoid glandular meats (rain, liver, kidneys etc.); use less salt in cooking and avoid salt 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.

### **11.4.5 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 myocardial 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 h to 1 hour after eating is essential to rule out the elicitation of angina pectoris or another attack of myocardial infarction.

## NOTES

### **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 the 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 which 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 which is preferably 1 to 2 kg below IBW.

**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 necrotic 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.

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.).

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

|      | <b>Avoid</b>                     | <b>Restrict</b>                              | <b>Use</b>           |
|------|----------------------------------|----------------------------------------------|----------------------|
| 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 |

**NOTES**

**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<sub>12</sub>.

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 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 10 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 eat 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 patient 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.

## NOTES

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.

### 11.4.6 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 affects 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 later 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:

|                                 |                                                      |
|---------------------------------|------------------------------------------------------|
| o Weight gain (due to oedema)   | o Swelling of feet and ankles                        |
| • Swelling of the abdomen       | o 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).

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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 congestive cardiac failure 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 nutritional support that can help in the treatment of delaying the progression of congestive cardiac failure.

### ***Dietary Management***

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 lose 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 to 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.

**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 on 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 flour, rice, dehusked pulses, papaya, mango, brinjal, pumpkin, gourd etc.). Whole cereals and pulses, legumes, lotus- stem, cabbage, soya flour should be avoided.

**Fat:** The quantity and quality of fat would be governed by the severity of hyper-Nutritional Management of lipidemia and adiposity. Emphasis, as always should

## NOTES

be laid on oils rich in MUFA's Coronary Heart Diseases 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:

$$\text{Fluid allowance} = \text{urine output (Previous 24 hrs.)} + 500\text{rnl (basal losses)} + \text{losses of fluids due to diarrhoea vomiting (if any)}$$

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 sparingly 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.

### **11.4.7 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 infection) 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.

#### ***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 symptoms 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 rash 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, arrhythmia, 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).

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## **11.5 PREVENTION OF CORONARY HEART DISEASES**

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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.

**NOTES**

**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%<br>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  | —   |

**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<br>(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.

## NOTES

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### 11.6 LET US SUM UP

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In this unit, we learnt about their etiological 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).

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### 11.7 GLOSSARY

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|                                |                                                                                                                                                      |
|--------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Aneurism</b>                | : a localized, pathological, blood-filled dilation of a blood vessel caused by a disease or weakening of the vessel's wall                           |
| <b>Angina pectoris</b>         | : a recurring pain or discomfort in the chest that happens when some part of the heart does not receive enough blood.                                |
| <b>Antioxidant</b>             | : a group of vitamins including vitamin C, E, selenium and carotenoids.                                                                              |
| <b>Arteriosclerosis</b>        | : sclerosis or thickening of the walls of the smaller arteries                                                                                       |
| <b>Atherosclerosis</b>         | : 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. |
| <b>Carditis</b>                | : inflammation of the heart tissues.                                                                                                                 |
| <b>Coronary Heart Disease</b>  | : disease involving the network of blood vessels that surround the heart and serves the myocardium.                                                  |
| <b>Diuretics</b>               | : a substance or drug that tends to increase the urine discharge.                                                                                    |
| <b>Embolt/Embolus</b>          | : abnormal particle (such as a blood clot or air bubble) circulating in the bloodstream.                                                             |
| <b>End Stage Renal Disease</b> | : severe kidney dysfunction reached when kidney function is reduced to 10% or less of normal function.                                               |
| <b>Epitopes</b>                | : the part of a foreign organism (or its proteins) that                                                                                              |

**NOTES**

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 0-6 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 inueased blood cliolesterol.
- Non-starch Polysaccharides** : the main components of dietary fibre and includes cellulose, 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.

|                      |                                                                                                                                           |
|----------------------|-------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Xanthacalasma</b> | : xanthoma of the eyelids.                                                                                                                |
| <b>Xanthoma</b>      | : yellowish coloured lesions on the skin containing cholesterol and fats, often associated with ilfferited disorders of lipid metabolism. |

**NOTES**

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## 11.8 CHECK YOUR PROGRESS

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- 1). What do you understand by the term CHD? Enlist some major forms of acquired heart diseases.
- 2). Enumerate the modifiable risk factors that increase the risk for developing heart diseases.
- 3). Enlist the major stages of CHD development.
- 4). What do you understand by the term Syndrome X?
- 5). Why is it essential to have water-soluble fibre in the diet?
- 6). Discuss the dietary guidelines for carbohydrates during atherosclerosis.



# 12

## NUTRITIONAL MANAGEMENT IN DIABETES MELLITUS

### NOTES

### STRUCTURE

- 12.1 Learning Objective
- 12.2 Introduction
- 12.3 Diabetes Mellitus
- 12.4 Management of Diabetes
- 12.5 Exercise and Drugs
- 12.6 Education
- 12.7 Prevention
- 12.8 Let Us Sum Up
- 12.9 Glossary
- 12.10 Check Your Progress

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### 12.1 LEARNING OBJECTIVE

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After studying this unit, you will be able to:

- describe the various forms of diabetes
- factors causing diabetes
- prevention methods for diabetes
- management of diabetes

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### 12.2 INTRODUCTION

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You have already learnt about nutrition care for weight management (Unit 8) and nutritional management of cardiovascular diseases (Unit 9). In the Unit 12, we will learn about the nutritional management of diabetes mellitus which is an important metabolic disorder of public health significance.

Metabolism is a term that we must understand. It means the sum of physical and chemical changes taking place in living cells. It includes all reactions by which the

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body obtains energy from food and spends it. So when we say metabolic diseases, these diseases refer to those disorders in which the various reactions in the cells are affected (production of energy' or utilization of energy) due to abnormal production of one or more hormones, or a deficiency of an enzyme.

The metabolic diseases that we will deal with are diabetes mellitus, gout and disorders due to inborn errors of metabolism such as phenylketonuria, tyrosinemia, maple syrup urine disease, homocystinuria and galactosemia. This unit will focus on diabetes mellitus while gout and disorders due to inborn errors of metabolism .

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### 12.3 DIABETES MELLITUS

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Diabetes mellitus (DM), commonly known as diabetes, is a group of metabolic disorders characterized by a high blood sugar level over a prolonged period of time. Symptoms often include frequent urination, increased thirst, and increased appetite. If left untreated, diabetes can cause many complications. Acute complications can include diabetic ketoacidosis, hyperosmolar hyperglycemic state, or death. Serious long-term complications include cardiovascular disease, stroke, chronic kidney disease, foot ulcers, damage to the nerves, damage to the eyes and cognitive impairment.

Diabetes is due to either the pancreas not producing enough insulin, or the cells of the body not responding properly to the insulin produced. There are three main types of diabetes mellitus.

Diabetes mellitus, also called diabetes, is a term for several conditions involving how your body turns food into energy.

When you eat a carbohydrate, your body turns it into a sugar called glucose and sends that to your bloodstream. Your pancreas releases insulin, a hormone that helps move glucose from your blood into your cells, which use it for energy.

When you have diabetes and don't get treatment, your body doesn't use insulin like it should. Too much glucose stays in your blood, a condition usually called high blood sugar. This can cause health problems that may be serious or even life-threatening.

There's no cure for diabetes. But with treatment and lifestyle changes, you can live a long, healthy life.

Diabetes comes in different forms, depending on the cause.

#### **Prediabetes**

Prediabetes is when your blood sugar is higher than it should be but not high enough for your doctor to diagnose diabetes. More than a third of people in the United States have it, but most of them don't know it.

## NOTES

Prediabetes can make you more likely to get type 2 diabetes and heart disease. Exercising more and losing extra pounds, even as little as 5% to 7% of your body weight, can lower those risks. Diabetes is due to either the pancreas not producing enough insulin, or the cells of the body not responding properly to the insulin produced. There are three main types of diabetes mellitus.

Several forms of diabetes have been identified as a result of research and survey conducted world-wide. These forms of diabetes include:

Type 1 - Insulin Dependent Diabetes Mellitus (IDDM)

Type 2 - Non Insulin Dependent Diabetes Mellitus (NIDDM)

Type 3 - Malnutrition Related Diabetes Mellitus (MRDM)

Impaired Glucose Tolerance (IGT)

Gestational Diabetes

### **Type 1 - Insulin Dependent Diabetes Mellitus (IDDM)**

Mostly children and adolescents suffer from this type of diabetes however, it may appear in adults and elderly too. In this, there is little or no production of insulin by the cells of the pancreas. Hence the young individuals require daily insulin injections. If daily injection is not taken, there could be life threatening metabolic complications and the symptoms could be very severe. Since various types of insulins are available (short term and long term), the carbohydrate content of the diet has to be adjusted accordingly. More people are underweight in this type of diabetes.

### **Type 2 - Non Insulin Dependent Diabetes Mellitus (NIDDM)**

Overweight and obese adults are generally afflicted by this type of diabetes. The insulin produced by the pancreas is normal or even high. The symptoms of the disease are gradual. The problem is caused by insulin resistance. Obesity is the main cause of insulin resistance. Weight reduction, diet and exercise can be helpful to decrease the insulin resistance. Anti-diabetic drugs can also be useful.

### **Type 3 - Malnutrition Related Diabetes Mellitus (MRDM)**

This type of diabetes has been categorized as a separate entity. It is often seen commonly in developing countries, India being one of them. It occurs in the young mostly between the ages of 15-30 years. People look thin, lean and malnourished. The reason for this type of diabetes is that the pancreas does not produce enough insulin (necrotic tissues on the pancreas) and hence these individuals require insulin. Very often hormonal disorders may occur. It has also been seen that when insulin is discontinued, the complications are lesser than type 1 diabetes.

### **Impaired Glucose Tolerance (IGT)**

Glucose tolerance is assessed by taking the fasting blood sugar value. An oral

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glucose load of 75 grams glucose is administered and blood sugar value checked again after | 1/2 - 2 hrs. The value of sugar obtained is checked against the normal or fasting value. If values are above normal, then we describe the condition as impaired glucose tolerance. In this type, the individuals are free from the symptoms of diabetes but they could develop diabetes at a later stage if they are unable to control the diet and avoid obesity. Regular exercise also helps in maintaining the blood sugar levels.

**Gestational Diabetes**

When a pregnant woman develops diabetes, it is known as gestational diabetes. It occurs in only 10/oof the pregnant women. Pregnant women who have a risk of diabetes because of family history or bad obstetrics history should be screened for diabetes. Pregnant mothers develop diabetes related complications and after delivery can also continue with the diabetic conditions.

Besides the types of diabetes discussed above, another form of diabetes namely maturity onset diabetes of the young (MODY) is being identified. Maturity onset diabetes of the young (MODY) refers to any of several rare hereditary forms of diabetes mellitus due to dominantly inherited defects of insulin secretion. As of now, six types havp been enumerated, but more are likely to be added. MOD Y 2 and MODY 3 are the most common forms. The severity of the different types varies considerably, but most commonly MODY acts like a very mild version of type 1 diabetes, with continued partial insulin production and normal insulin sensitivity. Age of onset for MOD Y is usually 25 years or it may develop earlier.

Having reviewed the diabetes types, next we shall move on to the etiology of the disorder.

***Etiology***

The precise etiology of diabetes is not known but multiple factors contribute to the disorder. These are reviewed herewith.

**Type-I Diabetes:** Factors contributing to the disease are both genetic and environmental.

|    |                 |                                                                                                                                                                                                                                                                                                   |
|----|-----------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. | Genetic         | There is, a familial tendency to develop Type-I diabetes. Altered frequency of certain (HLA) human lymphocyte antigen (usually a protein, foreign substance that produces <b>antibodies</b> ) on chromosome 6, abnormal immune response, autoimmunity and islet cell <b>antibodies</b> are noted. |
| 2. | Age             | The disease can occur at younger age.                                                                                                                                                                                                                                                             |
| 3. | Sex             | In younger group males are prone, but <b>females</b> are <b>afflicted</b> in greater numbers.                                                                                                                                                                                                     |
| 4. | Dietary factors | Low <b>intake</b> of fibre in the diet is associated with diabetes, Excess food ( <b>especially</b> refined food), causes an energy imbalance. May lead to obesity - a <b>risk factor</b> for diabetes. Over <b>and</b> under nutrition are important etiological factors.                        |

|    |                      |                                                                                                                                                         |
|----|----------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------|
| 5. | Infections           | Viral infections such as measles and mumps can trigger abnormal auto immune response that destroy the $\beta$ -cells of pancreas which produce insulin. |
| 6. | Increased catabolism | Excess breakdown of liver sugar (glycogen). Tissue protein and fat breakdown cause metabolic changes causing diabetes.                                  |
| 7. | Stress               | Stress is a contributing factor in precipitating this disorder.                                                                                         |

**Table 12.2: Etiological factors**

**Type II diabetes:** Besides some of the above genetic and environmental factors, imbalance of hormones can cause this type of diabetes many of these hormones may be insulin antagonist. These hormones include:

Growth Hormone: About 1/3rd of diabetics have been shown to have excess growth hormone.

Adrenocortical Hormone: Cortisol and corticosteroids lead to an increased protein breakdown and inhibit sugar utilization by the tissues, thus increasing blood sugar levels.

Adrenaline: This hormone increases the breakdown of glycogen (the storage form of glucose) in the liver. It also suppresses the insulin secretion thus increasing the blood sugar level.

Thyroid hormone: Excess thyroid hormone aggravates diabetes.

Besides imbalance of hormones, diseases too could be an etiological factor,

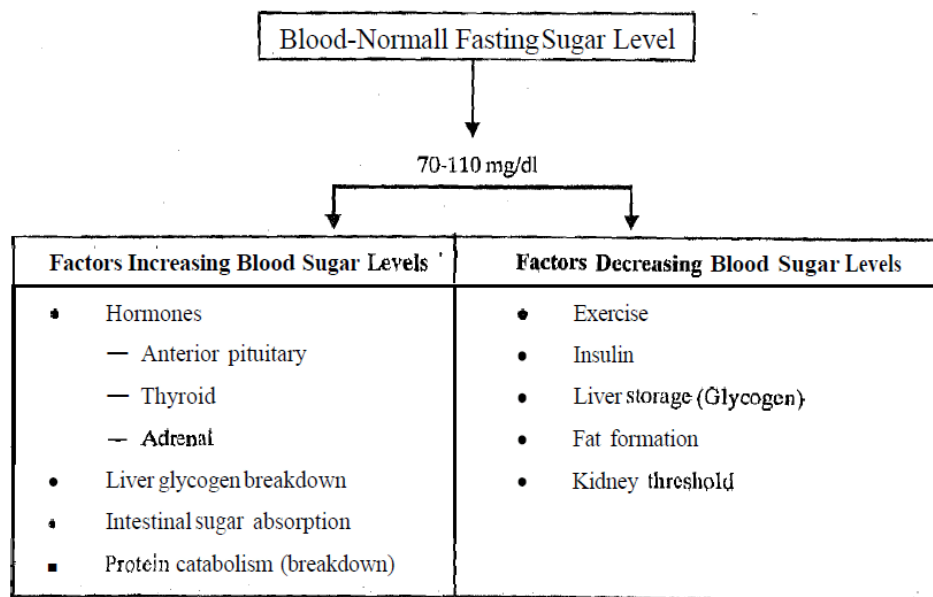
Diseases: In aging, a very important cause of diabetes could be an underlying disease. Diabetes occurs secondary to some other disorders, which may lead to insulin insensitivity or impaired secretion of insulin. These diseases are pancreatitis (inflammation of pancreas), carcinoma of pancreas, pancreatectomy (surgery of pancreatic duct). Mostly type II diabetes shows insulin resistance. Gestational Diabetes is the temporary diabetes seen in pregnancy. This is due to increased production of hormones which are antagonistic to insulin production.

**EXCESS FOOD, LACK OF EXERCISE AND AGING ARE ENVIRONMENTAL FACTORS FOR DIABETES BESIDES THE GENERIC FACTOR.**

### 12.3.3 Factors Affecting Normal Blood Sugar Levels

Earlier in our discussion, we learnt that insulin is the most important factor responsible for normal blood sugar levels. Now we will learn about the various factors that can increase blood sugar levels and those that can reduce it. First of all, let us know what is the normal sugar level (glucose) in the blood? The normal sugar level ranges from 70-110 mg/100 ml. This is the fasting level also. shows some factors that increase the sugar levels and others that decrease the sugar levels.

**NOTES**



**Figure 12.1: Factors affecting glucose levels in blood**

So we have learnt that — insufficient production or insulin resistance— can diabetes, commonly known as primary diabetes. Factors that increase sugar levels like hormones, intestinal disorders, diseases in which there is heavy breakdown of proteins (stress and trauma), dietary habits, lack of exercise, metabolic disorders, can also cause disturbances in the normal sugar levels, leading to abnormal or impaired glucose tolerance (IGT). Diabetes due to factors other than insulin is known as secondary diabetes. We can conclude therefore that the etiology of diabetes is multiactorial. Most common form of diabetes of course is primary diabetes.

### 12.3.4 Metabolic Aberrations and Symptoms

We all know that insulin exerts an important role in the maintenance of normal blood glucose levels through its effect on the metabolism of carbohydrate, fat and proteins. Three major effects of insulin on the metabolism include:

1. Decreased utilization of glucose by the body cells with a resultant increase in the blood sugar levels to ranges between 300 to 1200 mg/dl. Thus we can say that the carbohydrate metabolism gets affected and the body cells do not get the fuel for energy purposes.
2. Markedly increased mobilization of fats from the fat storage areas, causes abnormal fat metabolism, as well as, deposition of lipids in vascular walls that cause atherosclerosis. Thus, the body depends more on energy derived from fat than that derived from carbohydrates. When this happens, the level of acetone, acetoacetic acid and hydroxybutyric acid in the body fluids rises thus causing a condition known as acidosis.

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A second effect which is even niore important is the direct increase of ketoacids. These ketoacids have a low threshold for excretionby the kidneys. As much as 100-200 grams of ketoacids can be excreted in the urine each day. Because these are strong acids, they combine with sodium derived from the extracellular fluid replacing the extracellular fluid sodium with hydrogen ions, thereby making the urine more acidic. This causes rapid and deep breathing. These extreme effects occur in severe or poorly managed diabetes, leading to acidic coma and even death.

- The third effect is on the depletion of protein in tissues of the body causing changes in the protein metabolism. The catabolic activity of muscle protein is accelerated in diabetes leading to increase in nitrogen that must be excreted after deamination. Also the cellular potassium in the blood is increased which needs to be excreted in the urine.

**UNCONTROLLED DIABETES AFFECTS NOT ONLY CARBOHYDRATE METABOLISM BUT ALSO TI-IE FAT AND PROTEIN METABOLISM.**

***Clinical Symptoms***

In mild cases of diabetes mellitus, no symptoms may be seen. The diagnosis could be incidental during a blood or urine investigation. In most severe cases of diabetes, the clinical symptoms are more pronounced.

**Table 12.3: Clinical symptoms of diabetes mellitus**

|    |                                             |                                                                                                                                                                            |
|----|---------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. | Polyurea                                    | Excessive urinary output especially at night. In very small children bed wetting may be commonly seen.                                                                     |
| 2. | Polydipsea                                  | Excessive thirst due to loss of water from the body (polyurea)                                                                                                             |
| 3. | Polyphagia                                  | Increased appetite, urge for sweet items of food due to heavy loss of sugar in urine.                                                                                      |
| 4. | Loss of weight                              | Inspite of eating more (polyphagia) there is loss in weight.                                                                                                               |
| 5. | Lassitude and lack of energy                | Untreated diabetes can cause easy tiredness, drowsiness and even coma at later stages.                                                                                     |
| 6. | Pruritis vulvae                             | Irritation in the genitalia caused by local deposition of sugar from urine. It can also cause disturbed sleep.                                                             |
| 7. | Paraesthesia                                | A tingling sensation felt in the hands and feet.                                                                                                                           |
| 8. | Blurring of vision                          | Excess sugar deposits on the eye lens causing refraction changes resulting in the blurring of vision.                                                                      |
| 9. | Delay in wound healing and minor infections | Lack of nutrients to the wound delays wound healing. Minor recurrent infections occur (boils, foot, skin and urinary infections). If major infections, consult the doctor. |

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Polyurea, polydipsia, polyphagia, loss of weight, paraesthesia are earliest symptoms of diabetes. Polyurea is due to the osmotic diuretic effect of glucose in the kidney tubules. In turn, the polydipsia is due to dehydration resulting from polyuria. The failure of glucose (and protein) utilization by the body causes loss of weight and tendency towards polyphagia. The paraesthesia apparently is also caused by loss of body protein.

### 12.3.5 Diagnosis

Timely and proper diagnosis plays a key role not only in identifying new cases but also in managing old cases with or without diabetic complications. Certain diagnostic tests help in understanding the previous history of a diabetic and also in preventing the onset of future complications.

Let us review some of these.

When sugar is suspected in the blood or urine then tests need to be carried out to confirm that an individual has diabetes. We will briefly talk about these tests. These are:

### 12.3.5 Diagnosis

Timely and proper diagnosis plays a key role not only in identifying new cases but also in managing old cases with or without diabetic complications. Certain diagnostic tests help in understanding the previous history of a diabetic and also in preventing the onset of future complications. Let us review some of these. When sugar is suspected in the blood or urine then tests need to be carried out to confirm that an individual has diabetes. We will briefly talk about these tests. These are:

The Oral Glucose Tolerance Test (OGTT)

Urinary Sugar Test (Benedict's Test)

Glycosylated Haemoglobin Test

Let us discuss each of these.

#### **1. Oral Glucose Tolerance Test (OGTT)**

This is most commonly used diagnostic test particularly for identifying and 'at risk' individuals. The test is carried out after fasting overnight (12 hours). Glucose, 75 g in adults and 1.75 g/kg body weight in children, is administered orally (dissolve in glass of water). Before the glucose load and 2 hours after the blood samples are taken and estimated for sugar levels.

In normal individuals free from diabetes and with no impaired glucose tolerance, the fasting sugar levels are between 80-100 mg/100 dl. The blood sugar levels increase after glucose load and come down to basal level after 2 hours.



**Table 12.4: Diagnostic criteria for IGT and diabetes**

| Parameter                                                   | Blood Sugar Levels mg/dl |          |
|-------------------------------------------------------------|--------------------------|----------|
|                                                             | IGT                      | Diabetes |
| Fasting                                                     | <120                     | >120     |
| Glucose load 2 hours after<br>(also known as post prandial) | 120-180                  | >180     |

**NOTES**

**2. Urinary' Sugar (Uristix Benedict's test)**

No sugar/glucose is excreted in the urine in normal individuals. In diabetics, glucose is excreted by the kidneys when the blood sugar levels are more than 180 mg /dl. This is known as the renal threshold. Varying amounts of glucose is found in the urine depending on the severity of diabetes and also the intake of carbohydrates. This test is not as reliable as the blood sugar test since reducing sugars such as lactose (in lactating women) can give a positive test which is not related to diabetes.

Today, commercially diagnostic strips (uristix) are available with the chemists. The strip is dipped in the urine and the colour change on the strip indicates the sugar level in the urine. Another method of checking sugar in the urine is called the Benedict's test. In this test, eight drops of urine and 5 ml of Benedict's solution are taken in a test tube, mixed and heated for 5 minutes. Change of colour indicates sugar in the urine. The interpretation of Benedict's test is given in Table 12.5. Self monitoring by this method can be done several times if diabetes is severe (blood sugar levels at 180 mg ,or more per 100 dl). It is better to carry the test in the morning in the second urine sample. Urinary sample will more or less reflect the blood sugar level.

**Table 12.5: Interpretation of Benedict's test**

| Colour               | Report     | Urine g% | Blood mg% |
|----------------------|------------|----------|-----------|
| Green discolouration | 0 to trace | -        | < 200     |
| Green precipitate    | +          | 0.25     | 200-500   |
| Greenish yellow ppt  | N/A        | 0.5      | 250-300   |
| Yellowish orange ppt | +++        | 1.0      | 300-350   |
| Brick red ppt        | ++++       | >2.0     | >350      |

Source: ICMR 1993. Diet and Diabetes

**3. Glycosylated Haemoglobin**

Glycosylated haemoglobin values give important diagnostic inferences regarding

the recent past of a diabetic i.e. how well did he/she manage the hyperglycemia over the past few months. It is currently one of the best ways to check whether diabetes is under control or not.

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The higher the value the poorer has been the management. It is based upon the concept that excess blood sugar level circulating in the blood gets attached to the pigment (haemoglobin) present in the red blood cell (RBC) to make a glycosylated haemoglobin called Hb A1C. This deposit increases with the degree of diabetes. The combined glucose and haemoglobin in RBC can be estimated. This is high in diabetics (8-18% range) as compared to normal individuals (4-7% range). The glycosylated haemoglobin reflects the trend of sugar levels in the blood during the past 2-3 months. Patients having high values should be identified as the target groups for rigorous counseling regarding management of hyperglycemia.

It must be clear by the discussion above that these tests need not only be used for identification of new cases but also for the management of old/chronic patients. Regular assessment of vital parameters such as renal, blood and liver function tests along with blood glucose and glycosylated haemoglobin can prevent or delay the onset of several complications which arise due to sustained elevated blood glucose levels. These complications may be acute or chronic in nature and need to be handled as of utmost urgency by the entire medical team. Let us brief ourselves on some of the common complications.

### 12.3.6 Complications of Diabetes

Diabetes, as you know, is a lifelong disease. We have said earlier also that it can be controlled but not cured. Control of diabetes by ensuring normal blood level is important for preventing the complications to develop, but sometimes they do develop as acute or chronic complications. Let us read further and see what we mean by these complications.

#### ***A. Acute Complications (of Diabetes)***

Acute complications of diabetes include:

- **Hypoglycemia or low blood sugar:** The most frequent cause of low blood sugar is poor timing of meals and snacks. As you know, the treatment programme is based on proper balance of insulin, food consumption and exercise. Hence by reducing the food intake, by skipping or delaying a meal or snack, the amount of sugar in the blood will be less than if one had followed the normal meal. This creates a situation in which there is more insulin than needed for the sugar in the blood. The insulin will work on whatever sugar it gets and lower the existing blood glucose levels further. The second cause could be exercising more than usual without adding an extra meal or snack in the diet. So the usual amount of insulin and increased exercise can lower the blood sugar level further. Low blood sugar can also be caused by accidentally taking too much (of) insulin. When this occurs, more insulin is present in the

body than needed. The extra insulin works on glucose (sugar) already in the blood resulting in abnormally low blood sugar. The symptoms of low blood sugar are called 'insulin reactions' or 'hypoglycemia'. These reactions appear suddenly and must be treated immediately.

Symptoms appearing first are shakiness, nervousness, sweating, dizziness, weakness, irritability and hunger. Symptoms that develop further but slowly are crying, anger, drowsiness, confusion, staggered gait, inability to complete work, blurred vision and headache.

If the above are not treated immediately, more serious symptoms may eventually develop. These are increased confusion, delirium, convulsions and unconsciousness. If the individual is conscious, encourage him/her to eat a fast acting carbohydrate, such as sugar, honey, sweet or a chocolate and coca cola. Stop all activity and allow to rest for 10-15 minutes.

The glucose in the food should raise the blood sugar levels quickly. If not, try the second time. If unconscious, a friend, a family member should come to the rescue. The individual may need an injection of glucagon (glucagon is a hormone like insulin but produced by the alpha cells of islets of Langerhans and the pancreas). It has an action opposite to that of insulin as it increases blood sugar,

- **Hyperglycemia (abnormally high blood sugar) :** Maintenance of blood glucose levels within or close to the normal range has been stressed by all medical authorities worldwide. Even with conscientious effort there may be times when the blood sugar is high or abnormally low. Either of these two conditions left untreated can lead to serious problems. We have already learnt about low blood sugar (hypoglycemia) and now let us see what can happen during hyperglycemia, ketoacidosis.

This condition occurs when there is not enough insulin to meet the body needs and the deficiency is allowed to continue uncorrected. With high sugar levels in the blood, the sugar in the urine is excreted at high levels. In addition, the body breaks down fat for energy and ketones are made: Increased production of ketones is known as ketosis and their elevated levels in blood is referred to as ketonemia. Their increased excretion in urine is referred to as ketonuria and the term ketoacidosis includes all the above mentioned disorders associated with enhanced fat break down. Ketoacidosis is an extremely serious condition which can cause "Diabetic coma" or even death. The onset of ketoacidosis is gradual but when ketones are present, it is important to test for both glucose or ketones in the urine.

### **Do you know how ketoacidosis develops?**

The chain of reaction begins with insufficient insulin to meet the needs of the body. Several factors then work together. These include:

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1. Illness, infection, injury or emotional stress could increase the body's need for insulin because of the extra energy required. Effectiveness of available insulin would also require release of additional glucose from liver (already stored as glycogen) or from fat sources.
2. Omitting insulin doses, which reduce the amount of insulin available to the body,
3. Reducing exercise thereby upsetting the food, insulin and exercise balance. This factor and other factors given above may aggravate the tendency towards ketoacidosis.
4. Excessive amounts of carbohydrate food may also aggravate the ketoacidosis.

What will happen? Without enough insulin two things can happen. Firstly, the cells of the body will be unable to use the glucose in the blood for energy. Secondly, glucose cannot be converted to glycogen in the liver for future use. Thus blood sugar levels will rise and the sugar levels reach above 180 mg/100 ml. The extra sugar will spill into the urine causing high levels of sugar in the urine. So to make energy available the fat sources will be used for getting energy as a result of which ketoacids in the blood and urine will increase.

The onset of ketoacidosis is gradual but in the young diabetics this development is faster. Diabetic coma can develop in 12-24 hours. Many symptoms are similar to hypoglycemia but additional symptoms can appear. These are excessive urination, excessive thirst, increased hunger, drowsiness, unexplained weight loss, slow healing of cuts and wounds, dry itching skin, vaginal itching, a abdominal pain and rapid shallow breathing with acetone smell.

Can we prevent ketoacidosis?

Normally, while maintaining a good sugar control, there is a very little danger of ketoacidosis. One should test for ketones under the following conditions:

- Blood glucose levels are about 240 mg/100 ml
- fever is present
- nausea and vomiting
- stress, and
- if insulin dosages is being adjusted.

We will now proceed towards the long-term complications which are generally observed among old cases of diabetics i.e. after the initial diagnosis.

### ***B. Chronic complications***

These occur gradually when the diabetics do not monitor the blood sugar and they are careless about eating, exercise and the medications as a result of which the blood sugar levels are high. Uncontrolled diabetes with circulating high sugar

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and lipids precipitate vascular disease. The chronic complications arising due to uncontrolled diabetes are highlighted herewith.

- **Atherosclerosis:** Degeneration of walls of the arteries due to fatty plaques - deposition on arterial walls as you may recall studying in the Figure 11.2 in Unit II. Diabetics are more prone to myocardial infarction, stroke and deep artery blockages in extremities. Atherosclerosis is a common complication in the diabetics. Lipoprotein abnormalities are common in diabetics and responsible for this condition.
- **Nephropathy:** Changes occur in the nephrons of the kidney due to thickening of capillary basement membrane, leading to glomerulonephrosis (disease of kidneys). These changes lead to defects in filtration increasing the proteins in urine (proteinuria) and causing uraemia and finally renal failure about which we will learn later in Unit 16.
- **Retinopathy:** Long duration diabetes with uncontrolled sugar may affect the small blood vessels of the eyes (microangiopathy). This can result in rapid deterioration of the eyesight (retinopathy). **Neuropathy:** Lesions of peripheral nervous system (neuropathy) could cause tingling, burning or numbness in the sensation of the upper and lower limbs.
- **Infections:** Diabetics are also prone to various bacterial, viral and fungal infections. In diabetics, cuts and wounds heal slowly. Individuals are prone to tuberculosis, infections of the skin, urinary tract and foot. The doctor must be consulted and appropriate antibiotics taken. Foot care is most important as the common problems related to the foot are lack of circulation and neuropathy. In 5% diabetics, amputation is necessary due to negligence.

So far we have learnt about the increasing prevalence of diabetes, types of diabetes, etiology, diagnosis and a briefly on the salient complications. In our next section we shall learn about the management of diabetes wherein a dietician plays a key role.

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## 12.4 MANAGEMENT OF DIABETES

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By now you know that diabetes can not be cured but can be treated so that an individual leads a normal life. Patients who maintain their blood glucose levels within the normal range suffer from lesser complications as compared to those frequently experience fluctuations in the blood glucose levels. A good synchronization between diet, life style and drugs can help in preventing/delaying the onset of complications.

As a dietitian, our key objective involves bringing about life-long adoptable quantitative and qualitative changes in the nutrient intake, dietary habits and food choices of the patient(s) as per their insulin/ drug dosage and life-style. The dietician should work in close co-ordination with the doctor, and other members of the patient care team. We will now discuss the details for dietary management

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for diabetic which will be followed by a briefing on life style modifications and drug/ insulin. While reading the subsequent concepts one must keep in mind that successful management of diabetes involves a holistic approach with coordination between diet, lifestyle and hypoglycemic drugs/ insulin. Let us begin our discussions with dietary management.

### 12.4.1 Management of Diet

Diet plays a very important role in management of diabetes as it exerts a direct influence on the blood glucose levels. It is one of the vital components in diabetes control besides the medical, exercise and behavioural aspects of the treatment. Let us talk of the diet first. The goals of diet therapy are to maintain and prolong a healthy, productive and a happy life. This means nutritional assessment is important for setting the practical and acceptable goals for the patient. Goal that the patient can comprehend, relate to, and can be followed easily. Only after this can we plan the diet and execute it. Clinical parameters should be monitored from time to time in order to improve adherence to the programme and make the necessary changes if required. Dietary changes can be made in the plans which are more acceptable and can easily be followed. This also means that:

DIABETIC DIETS SHOULD BE INDIVIDUALIZED, BASED ON THE NUTRITIONAL STATUS OF THE PATIENT. IT SHOULD BE PRACTICAL, SUITED TO THE NEEDS AND CAN BE FOLLOWED TO MEET THE DIETARY GOALS.

What are the dietary goals? Well here they are. These are to:

1. supply optimum nutrition to maintain good health,
2. provide calories for maintaining ideal weight and allowing normal growth and development (in case of children),
3. maintain blood sugar control (glycemic control),
4. achieve optimum blood lipid levels, and Diabetes Mellitus
5. minimize acute and chronic complications of diabetes mellitus.

The above objectives can be met by adhering to some of the basic principles of planning diets which include the consideration of factors like:

1. body weight, age, sex, activity,
2. economic, social and cultural factors,
3. type of diabetes, mode of treatment and control of diabetes, and
4. other factors like pregnancy, obesity, cardiovascular and renal disorders or even gastrointestinal problems and other infections.

Keeping the above principles in mind, the diet planned should be such as to help in

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maintaining an optimum nutritional status and achieving desirable body weight. As a thumb rule if the patient is undernourished, feed more calories, and if over-nourished reduce the calories. Calories are mainly obtained from carbohydrates, protein and fat. You already know that one gram of carbohydrate and proteins provide 4 Kcal and one gam of fat supplies 9 kilocalories. Let us now understand the contribution of these macronutrients in a diabetic diet.

**A. Calories, Proteins, Fats and Carbohydrates**

We have learnt earlier that sufficient calories should be given to maintain an ideal weight for adults and to provide for normal growth and developinent for children and adolescents and also provide for the increased needs during pregnancy, lactation and illness.

Calories: The energy requirements (f adult patients is governed by their present body weight and the need to maintain a desirable or ideal body weight. Remember, we learnt about computations of energy requirements on the basis of ideal body weight earlier in Unit 9. Table 12.6 gives details of the energy requirements for excess, normal and underweight patients. We can calculate the energy requirements using the values.

**Table 12.6: Calorie requirements according to weight and activity**

|    | <b>Weight</b>     | <b>Activity Levels</b>     | <b>Calories Required/kg<br/>Ideal Body Weight</b> |
|----|-------------------|----------------------------|---------------------------------------------------|
| 1. | Ideal body weight | Sedentary activity         | 25                                                |
| 2. | Ideal body weight | Moderate activity          | 30                                                |
| 3. | Overweight        | Sedentary activity         | 15-20                                             |
| 4. | Underweight       | <b>S</b> edentary activity | 30                                                |
| 5. | Underweight       | Moderate activity          | 35                                                |

**Proteins:** Proteins should be provided in adequate amounts to maintain a normal body composition and prevent depletion of lean tissue mass. Adult diabetics without any complications ale able to maintain good health when given LO gm protein/ kg ideal body weight per day. During childhood, adolescence, pregnancy and laclation the requiremenls are higher than the RDI and patients usually benefit by increasing the protein by 10 to 15%. However, in case of renal complications protein intake needs be restricted in accordance with the clinical parameters of renal function test. In such situations the patient should not be given more than 0.8 gm per kg IBW per day.

The total fat recommended by WHO is less than 30% of the total calories.

However in view of the widely prevalent Asian paradox in India, it is generally recommended not to provide more than 15-20% of the total energy from dietary fat. This is particularly important in case of obese diabetics having hypertension or

cardiac disorders. Vegetable oils rich in mono/poly unsaturated fatty acids should be preferred over animal fats which are generally rich in saturated fatty acids. regarding quantity and quality of fat.

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**Table 12.7 : Recommendations regarding quantity and quality of fat**

| Type of Fat                                            | Recommended % |
|--------------------------------------------------------|---------------|
| Polyunsaturated fats<br>(vegetable oil)                | 10            |
| Saturated fats<br>(ghee, butter, vanaspati, margarine) | 7             |
| Monosaturated<br>(Olive, palm, ground nut)             | 10-13         |

The dietary cholesterol intake should be kept below 300 mg/day for diabetics without any complications. However, if the patient is at risk or is suffering from hyperlipidemia or CAD, the cholesterol intake should not exceed 200 mg/day. Mé hope that you do remember the food sources of cholesterol we discussed in the previous unit. Well, a quick recapitulation would have taken your thoughts to sources of cholesterol such as whole milk and its products, egg yolk, red meat, organ meats (liver, kidney, brain).

**Carbohydrates:** These to a large extent depend on the food habits. Complex carbohydrates with more fibre are recommended to simple carbohydrates like sugars.

The amount should provide 55-65% of the day's caloric intake. The percentage and distribution of carbohydrate will vary with the insulin regimens and the treatment goals and also individual habits. Remember diabetics need not restrict their carbohydrate intake, but they can alter the type of carbohydrates in their diets, eat complex carbohydrates (whole cereals, pulses and vegetables) and avoid foods rich in simple carbohydrates (honey, jaggery, sugar and jams).

SIMPLE CARBOHYDRATES ARE NOT RECOMMENDED FOR DIABETICS, COMPLEX CARBOHYDRATES ARE BENEFICIAL.

We just read about laying emphasis on complex carbohydrates. What is the benefit of complex carbohydrates over simple ones? We will now learn about the significance of incorporating complex carbohydrates, particularly the non-starch polysaccharides (dietary fibre) with respect to maintaining blood glucose within normal range.

### ***B. Dietary Fibre***

Dietary fibre is that part of food which is not digested by the intestines. High fibre meals have shown to give the best glycemic control in diabetics. It not only reduces blood sugar but lowers blood cholesterol and hence is good for cardiovascular diseases, constipation and some forms of cancer. Fibre is found in a wide range of



foods such as whole cereals, pulses, fruits, green leafy vegetables (insoluble fibres). Many soluble fibres such as those present in beans, fenugreek seeds are found to be more effective in controlling blood sugar and serum lipid levels. We will talk about these later. Intake of 25 g of dietary fibre per 1000 calories/day is considered optimum for a diabetic. Remember high fiber diets are of low calorie value and also have a low glycemic index. So diabetics must consume more of these foods. We will tell you more about glycemic index

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**Table 12.8: Fibre content of some common Indian foods**

| <b>Foods</b>                   | <b>Dietary Fibre (g/100 g)</b> |
|--------------------------------|--------------------------------|
| <b><i>Cereals</i></b>          |                                |
| Rice                           | 7.6                            |
| Wheat                          | 17.6                           |
| Ragi                           | 18.6                           |
| Oatmeal                        | 3.5                            |
| Bajra                          | 20.3                           |
| <b><i>Pulses</i></b>           |                                |
| Soyabean                       | 3.7                            |
| Whole green gram               | 13.5                           |
| Black gram                     | 14.3                           |
| Bengal gram                    | 14.1                           |
| <b><i>Nuts and Oils</i></b>    |                                |
| Ground nut                     | 6.1                            |
| Coconut (dry)                  | 8.9                            |
| <b><i>Roots and Tubers</i></b> |                                |
| Sweet Potato                   | 7.3                            |
| Potato                         | 4.0                            |
| Yam                            | 5.3                            |
| <b><i>Fruits</i></b>           |                                |
| Pomegranate                    | 5.1                            |
| Banana                         | 2.5                            |
| Guava                          | 5.2                            |
| Mango                          | 2.3                            |
| <b><i>Vegetables</i></b>       |                                |
| Fenugreek Leaves               | 1.1                            |
| Palak                          | 5.0                            |
| Peas (green)                   | 4.0                            |
| French Beans                   | 1.8                            |

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**C. Vitamins and Minerals**

The recommendation of vitamins and minerals is the same as for general population. However, lower levels of magnesium are associated with risk of diabetes. Magnesium depletion has been associated with insulin insensitivity, which may improve with oral supplements.

Chromium supplementation has been shown to exhibit beneficial effects in diabetic patients. Mineral and other vitamins supplementation must be considered during infections and complications or situations such as extreme weight reducing diets, strict vegetarians, pregnant, lactating and elderly or those individuals who are on drugs and have malabsorption disorders and other ailments.

Sodium restriction is suggested for hypertensive diabetics or those who are suffering from renal complications/oedema. In our discussion above we learnt about the nutrient requirement, In short, we have learnt that the diabetics should avoid simple carbohydrates, use fat in limited amounts, consume less saturated fats and more polyunsaturated fats, take whole cereals and pulses in right amount, use fiber rich food in large amount, take vegetables as desired and fruits in limited amounts.

Some handy guidelines for selection of foods for diabetic patients is presented next.

You may be aware that some foods need to be avoided, other used in moderate amount and yet use some foods freely. Let us get to know which these foods are:

| <b>Foods to be used freely</b> | <b>Foods to be used in moderate amounts</b> | <b>Foods to be avoided</b>            |
|--------------------------------|---------------------------------------------|---------------------------------------|
| - Vegetables (low starch)      | - Flesh food especially red meats           | - Sugar                               |
| - Green leafy vegetables       | - Fats                                      | - Sweets                              |
| - Spices and condiments        | - Nuts                                      | - Honey                               |
| - High fibre foods             | - Cereals/Roots/Tubers                      | - Jams                                |
| - Coffee/tea (without sugar)   | - Pulses                                    | - Jellies                             |
|                                | - Milk Products                             | - Cakes and Pastries                  |
|                                | - Eggs                                      | - Pizzas                              |
|                                |                                             | - Aerated drinks and sweetened juices |
|                                |                                             | - Sweetened yoghurt                   |

By now you must be well versed with the dietary guidelines of diabetics. In brief avoid sweets, use less fat, take whole cereals and pulses in right amount. Consume vegetables as desired. Take permitted fruits in moderate amounts and include as much fibre rich foods as you can. An important aspect about dietary management of diabetics involves the concept of substituting food-stuffs having similar (not same) nutritive value or at least similar contribution to the post-prandial blood glucose. The use of food exchanges is a very convenient method, which is essentially adopted by dieticians and patients for maintaining blood glucose levels within a safe range. In our subsequent discussion we shall learn about food exchanges — their characteristic features and how to use them.

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**12.4.2 Food Exchange System**

In a diabetic's day to day diet, the calorie intake and the quantity of food consumed should not have wide fluctuations. Also the diet should not get monotonous and rigid pressurizing the patient to take the same foods day in and day out. Variety in foods makes life more interesting. Don't you think so? For this reason we have the food exchange system which provides almost the same amount of calories, carbohydrates, proteins and fats grouped together. By this we keep the total intake of nutrients constant but at the same time provide variety in foods. Thus there may be difference in the weight of the food which provides the constant nutrients.

**Table 12.9: Milk exchange (Provides 100 calories and 5 g protein)**

| Food                | Quantity |
|---------------------|----------|
| Butter milk         | 750 ml   |
| Cheese              | 30 g     |
| Curd                | 210 g    |
| Khoa                | 30 g     |
| Buffalo Milk        | 90 ml    |
| Cow's Milk          | 180 ml   |
| Skimmed Milk        | 260 ml*  |
| Skimmed Milk Powder | 30 g*    |

Is this not interesting?

If we know the total exchanges of milk we have to take in a day, we can have a number of choices of milk alternatives which we can substitute for milk giving us the same nutritive content. Occasionally, we could have curds, buttermilk, cow's milk, even cheese, khoa QI' skimmed milk, if we want to. So if we can educate the diabetics regarding all exchanges of food groups they can play with their diet themselves. Exchanges for vegetables, fruits, cereals, pulses, meat or fats have similarly been worked out. Each exchange list will provide a number of items that can be exchanged within the group or with each food group. You will use these exchange lists while planning your diets in the practicals and therefore will be more familiar with them.

**12.4.3 Glycemic Index (GI)**

Although the use of exchange lists is still popular for planning diabetic diets, it has been realized in recent years that in exchange lists though different foods contain the same amount of nutrients, the rise in blood sugar after the meal (post-prandial) varies and is not the same. This has given rise to a new concept that different carbohydrates raise the blood sugar to a variable extent. diabetic diets, it is important to know that how much would the blood sugar rise using different foods or combination of foods. Therefore, the glycemic index concept was born.

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What is this glycemic index? Glycemic index is the numerical index given to a carbohydrate-rich food that is based on the average increase in blood glucose level occurring in blood after the food is eaten. The higher the number, the greater the blood sugar response.

If you take a certain food and measure the rise in blood sugar in response to the food consumed in comparison with the response to an equal amount of glucose and know the glycemic index in percentage, then it would be a better method of planning diabetic diets. We do have glycemic index of individual food items such as wheat, rice, pulses and vegetables. For example, cereals like wheat, rice, vegetables (potato and carrots) have a high glycemic index (65-75%), fruits have 45-55%, lentils (peas, beans, green gram, Bengal gram) have a low glycemic index (30-40%). Glucose has a glycemic index 100%. So diets with lower glycemic index are more beneficial for diabetics.

These foods generally have a more fibre content. Since we do not take individual foods but two or more foods cooked and prepared, we would be interested in glycemic index of common mixed foods consumed by us in our main meals and also some common snacks. Work is being conducted in various institutions on this area. Some day we will have this data and would be able to switch from food exchange system to glycemic index of cooked food for planning diabetic diets. We can calculate the glycemic index of food by using this formula:

$$GI = \frac{\text{Area under 2 hours blood response curve to test food}}{\text{Area under 2 hours response for equivalent glucose}} \times 100$$

**Table 12.10: Glycemic Index of some common food items**

| Food Items      | Glycemic Index |
|-----------------|----------------|
| Rice            | 72             |
| Parantha, Wheat | 70             |
| Bread           | 70             |
| Upma            | 75             |
| Idli            | 80             |
| Chole           | 65             |
| Sprouted gram   | 60             |
| Milk            | 33             |
| Ice Cream       | 36             |
| Curds           | 36             |
| Tomato soup     | 38             |
| Groundnuts      | 13             |
| Apple           | 39             |
| Orange          | 40             |
| Banana          | 69             |
| Potato          | 70             |
| Sweet potato    | 48             |
| Beetroot        | 64             |
| Soyabeans       | 43             |
| Rajmah          | 29             |
| Bengal gram     | 47             |

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A GI of 70 or more is high, a GI of 56 to 69 is medium and a GI of 55 or less is low.

Thus From our discussion above it is clear that the glycemic index tells us how rapidly a particular carbohydrate turns into sugar. It certainly does not tell us how much of carbohydrate is actually present in a serving of a particular food. This information too is important if we want to assess the time impact of carbohydrate consumption. To know this we need to understand another concept called the glycemic load. The glycemic load of a food is the glycemic index divided by hundred and multiplied by its available carbohydrate content (i.e. carbohydrate minus fibre) in grams. The glycemic load is a relatively new way to assess the impact of carbohydrate consumption that takes the glycemic index into account, but gives a full picture than does glycemic index alone.

For example, if we consider watermelon. Watermelon has a high glycemic index (about 72). However, a serving of 200 g of watermelon has only about 6 grams of available carbohydrate per serving. So its glycemic load is pretty low i.e.  $72/100 \times 6 = 4.32$ . Perhaps suitable for diabetics. We all know that sugar, honey, jaggery i.e. all sweeteners, which are rich in mono and disaccharides, are completely omitted from the diet of diabetics. However, craving to eat sweet meats is always high among several patients. It is for this reason that diabetics are suggested to use artificial (nutritive/non-nutritive) sweeteners, which usually do not provide any significant amount of calories or other nutrients.

### 12.4.4 Sweeteners: Nutritive and Non-Nutritive

**Nutritive Sweeteners:** We know some sweeteners like glucose, honey, molasses, fruit juice, dextrose, maltose, mannitol, sorbitol, xylitol and hydrogenated starch hydrolysates have shown no advantage or disadvantage over sucrose. Fructose which occurs in fruits, vegetables and honey provides 4 Kcals/g. Fructose through natural foods in the form of fruits and vegetables can be given in moderate amount to diabetics. Restriction of these nutritive sweeteners is because of its caloric content.

**Non-Nutritive Sweeteners:** These are characterized by an intense sweet taste. They are needed in small quantities and do not make any nutritive contribution which is significant. Three non nutritive sweeteners available are saccharin, aspartame and acesulfame K. In some countries, cyclamate and stevioside is available. From the data available on clinical studies it is seen that these sweeteners have no adverse effect on diabetes control. The recommended use of aspartame has been approved by the American Diabetic Association.

The recommended safe limit for aspartame is 50 mg/kg of body weight per day. Aspartame is commercially known as 'Equal', Nutrasweet' and 'sugarfree'. In America, Acesulfame is the newest non nutritive sweetener available. It is available commercially as 'Sunette' or 'Sweet one'. The safety limit given by FDA is 15 mg/kg body weight per day. Let us read a little more about commercially available processed foods which have been developed particularly for diabetics.

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### **12.4.5 Dietetic Foods**

With advances in food technology, foods for special use by diabetics are now available in the global market, they are emerging in India also. These foods have reduced calories or sugar content. Diet pepsi/cola, high fibre flour, breakfast cereals or even ice-cream is available in the Indian market currently.

A dietetic food product must contain less than 20 calories per serving and may be used as a 'free food' at meals or as snacks. In a day, not more than 60 calories should be obtained by dietetic foods. All diabetics should be counseled to read the food label carefully and then only select the food for consumption. What about alcohol? Can diabetic patients be allowed to take a peg or two? Let us find out.

### **12.4.6 Alcohol**

Intake of alcohol should be limited. It is high in calories, lacks essential nutrients and may therefore promote ketoacidosis, hypertriglyceridemia (high triglycerides) and alcohol induced hypoglycemia (low sugar levels in the blood). Hypoglycemic drugs should never be consumed with alcohol. Patients on insulin should take not more than 2 drinks per day (one drink equals to 12 oz beer, 5 oz wine, 1 1/2 oz whisky).

The calorie content of drinks consumed can be accordingly calculated. Total food intake should not be reduced when the patient is consuming alcohol. When calorie intake is restricted alcohol consumption should preferably be avoided. Women diabetics should abstain from alcohol during pregnancy and lactation. Some guidelines regarding alcohol consumption for insulin users:

1. Avoid drinking alcoholic beverages
2. Take not more than 2 small drinks a day.
3. Drink only with food.
4. Do not reduce food intake.
5. Restrain yourself if pregnant or lactating.

**For non-insulin users:**

1. Substitute for fat calories. A drink providing 100 calories should be substituted by 2 fat exchanges.
2. Avoid if your triglycerides are high,
3. Limit if calorie intake is restricted.
4. Restrain if pregnant or lactating.

**Table 12.11: Composition of common alcoholic beverages**

| Alcoholic Beverages | Quantity (ml) | Kilocalories |
|---------------------|---------------|--------------|
| Whisky              | 30            | 91           |
| Rum                 | 30            | 98           |
| Gin                 | 30            | 84           |
| Brandy              | 30            | 98           |
| Wine                | 100           | 78           |
| Beer (regular)      | 250           | 122          |

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These foods help in improving taste and increasing variety in the diet of diabetics. In our subsequent discussion let us learn about certain foods or food components which can help in creating a plateau affect in the post-prandial blood glucose thereby reducing the risk for developing acute complications (hyper/hypoglycemia) and at times perhaps also reduce the insulin /drug dosage.

### 12.4.7 Beneficial Effect of Some Foods: Supportive Therapy

Certain foods, part of food or food components have been found to be beneficial in managing hyperglycemia. Most of these have been identified due to the presence of soluble and /or insoluble fibre in them. Research has well documented evidence that soluble fibre i s particularly are useful for managing hyperglycemiaand hyperlipidemia (particularly elevated cholesterol seen among diabetics). Some of the common and effective sources include:

**Fenugreek seeds:** Commonly known as methi seeds in Hindi. They are commonly used in Indian cuisine in chutney and even in pickles and several dishes. These seeds are rich in fiber which is mucilaginous (20-50% fiber content). It also contains an alkaloid substance called Trigonelline known to reduce blood sugar levels. Fenugreek powder reduces blood and urine sugar levels and improves glucose tolerance. It has an action on blood lipids, lowering triglycerides or blood cholesterol. Fenugreek seeds are beneficial for use in diabetics. The same beneficial effects are not seen with fenugreek leaves. About 25-50 g of fenugreek seeds can be soaked in water or powdered form can be mixed in a drink of buttermilk or water, It should be taken in 15 minutes before meals 2-3 times a day. The seeds are bitter but benefits are great. You could use the powdered form in rice, dal and vegetable preparatian. Other foods/ isolated fibre: It is generally recommended that diabetics should substitute whole wheat four with soya flour, whole Bengal flour or stalks of green leafy vegetables in the ratio 3:1 while preparing dough for chapattis etc. as it helps in increasing the fibre content of meals and preventing peak rise post-prandially. Wheat bran, bengal gram husk can be given in doses

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around 15 g/day. These show a hypoglycemic effect in diabetics. Wheat bran is best incorporated into biscuits 'bran biscuits' and are available in the market as high/rich in the insoluble fibers. Guar gum (a soluble fiber) can also be incorporated in the form of biscuits or laddoos (20% level). Both these fibers have shown beneficial effect on blood sugar of diabetics and 298 in type 2 diabetics have demonstrated a reduction in hypoglycemic drugs.

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## 12.5 EXERCISE AND DRUGS

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In this section, we shall review the role of exercise and drug in the management of diabetes. We shall begin with exercise.

### 12.5.1 Exercise

Regular controlled exercise helps to increase glucose utilization. It helps attain ideal weight by burning calories. It also builds stamina and provides a sense of well being.

A MODERATE EXERCISE SCHEDULE SHOULD BE PART OF  
MANAGEMENT PROGRAMME OF DIABETES.

Aerobic exercise for at least 20-30 minutes four or more times a week is recommended. Exercise after meals is preferred. There are many benefits of exercise particularly for type 2 diabetes. It:

- lowers or eliminates the need for insulin drugs,
- decreases insulin resistance,
- helps in weight loss and maintenance of decreased weight,
- lowers the triglyceride and increases the HDL levels,
- improves circulation throughout the body,
- reduces stress, and
- reduces high blood pressure.

What are the moderate intensity exercises?

- Walking for 30 min or jogging
- Biking leisurely for 1/2 hr.
- Playing tennis, swimming, gardening, golfing
- Vacuuming for 1/2 hr.

Walking is the best form of exercise especially for the elderly.

What precautions should be taken while exercising?

Being a diabetic the patient has to take a few precautions. These are:

- In case you get hypoglycemia, keep sugar/sweet ready for use.



If your blood sugar values are high and diabetes is not under control, stop exercising.

## NOTES

It has been discussed earlier in section 12.3 that the meal pattern and the quantity of food served for each meal should be according to the dosage, timing and peak action of drugs/ insulin. We will now learn about drug and insulin commonly used for the management of diabetes.

It has been discussed earlier in this unit that hypoglycemic drugs and or insulin are used for the management of hyperglycemia among NIDDM and IDDM patients respectively. You must be remembering that earlier in this section 12.3, it was mentioned that the meal pattern and the quantity of food served for each meal should be in accordance with the dosage, timing and peak action of drugs/ insulin. We will now learn about the commonly employed drugs and insulin used during diabetes mellitus.

### 12.5.2 Drugs and Insulin

When diet, exercise or even weight reduction do not improve the diabetic symptoms and blood sugar levels, the use of hypoglycemia drugs become necessary. Oral hypoglycemia drugs are generally recommended to NIDDM, patients. Several types of oral drugs are available. They work by stimulating the pancreas to release additional insulin or to help the cells of the body to utilize the insulin properly. Some commonly used hypoglycemia drugs include:

- **Sulphonylureas:** Tolbutarnide is the mildest and its effect remains for 8 hours hence 2-3 doses need to be taken. Another drug, chlorpropamide, needs only a single dosage as it is a stronger and a long acting medicine. They stimulate the pancreas to release more insulin. Tolbutamide and talzamide are first generation sulphonylureas while glyburide, glopizide, glimeperide belong to the category of second generation sulphonylureas.
- **Bigunnides:** They are anti-diabetic drugs which do not affect the output of insulin. These are preferred to sulphonylureas because they do not cause weight increase. Metformin is from this group and generally prescribed to obese subjects while sulphonylureas are given to non- obese individuals.

Are there any side effects of oral hypoglycemic drugs? These are rare but may occur such as skin rashes, itching, loss of appetite and stomach upset on weight increase. These, if present, must be reported to your doctor.

Depending on the diabetic condition, the doctor can prescribe a combination of oral drugs or insulin. Let us get to know more about insulin and its types.

### **INSULIN**

The discovery of insulin has dramatically changed the lives of people having type 1 diabetes. With this wonder drug diabetics can lead a normal, enjoyable and a productive life.

## NOTES

A dietician should be well versed with the salient characteristics of different types of insulin in order to plan an appropriate diet and meal pattern for diabetics. Let us see what these facts are:

1. Insulin is measured in units. A unit of insulin lowers the blood glucose by a certain amount. The doctor will decide the number of units of insulin a diabetic will need per day.
2. Insulin is generally produced in two different strengths —U100 and U40. This means that if the diabetic takes U100, there are 100 units of insulin in each cubic centimeter. Similarly U40 has 40 units of insulin per cubic centimeter.
3. Three types of Insulin is available. The type varies in how quickly it starts working (lowering blood glucose), time of peak activity (when they work the hardest) and how long it works. The three types are short acting, intermediate acting and long acting.
  - **Short acting:** This type of insulin begins working quickly, works hardest 2- 3 hours after injection but is completely gone after 4-6 hrs. So if a diabetic takes this type of insulin, they need a shot every 4-6 hrs. This is also known as Regular insulin. Sometimes, a small amount of zinc is added to the regular insulin because it prolongs its action. This is known as the 'Semilente'.
  - **Intermediate acting:** This type works more slowly than short acting. It works hardest 8-12 hours after injection and still keeps on working to some extent 24 hours later. One form of this type of insulin is known a Neutral Protaminc Hagedorn (NPH). Hagedorn is the name of the developer and protamine is a simple protein added to it. Intermediate acting insulin is called Lente.
  - **Long acting:** Long acting insulin does not work until 4 10 8 hours after injecting. Its peak activity occurs 18 to 24 hours after injection and it continues to work to some extent after 36 hours. This is used less commonly because it is easier to work with 4 hours or 24 hours rather than 36 hours. Sometimes the doctor uses a combination of short and intermediate insulin.

The carbohydrate distribution varies with the type of insulin prescribed. For example, in case of regular insulin 1/3rd each carbohydrate in three meals can be distributed but not in the intermediate or long-acting insulin. In intermediate it could be 1/7th for breakfast, 2/ 7th for noon, 1/ 7th mid-afternoon, 2/7th for evening and 1/7th at bed time. For long acting insulin; it could be 1/5th for breakfast, 2/5th noon and 2/5th evening and remaining for bed time.

You will understand this in your practical classes. A diabetic patient need not always be hospitalized and can refer to a dietician on a day-to-day basis. The food intake and activity level are ever changing variables that affect blood glucose levels. Festivals, religious occasions, professional and social responsibilities along with common illness such as constipation, fever, cold etc. can affect the insulin and food requirements markedly.

It has, therefore, been strongly advocated that diabetics must be rigorously

**NOTES**

counseled so that they can manage their blood glucose levels themselves. Patient education through counseling sessions and mass media can help reduce the morbidities associated with diabetes. The subsequent discussion will highlight some of the points which should be kept in mind by the dietician while planning or implementing a counseling session.

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## 12.6 EDUCATION

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Education is very important for diabetics because it is a lifelong disorder. In order to enable the patient to lead a healthy and a good quality life free from complications, the diabetics must be educated sufficiently in the areas given below:

The nature disease.

Its acute and chronic complications, (consequences of negligence).

Self monitoring at regular interval for sugar (urine and blood) and serum lipids or any weight alterations and importance of weight maintenance.

Adequate information on the diet (foods restricted, foods given in the moderation and free foods, concept of food exchanges glycemic index and planning meals).

Knowledge about insulin and drugs in relation to carbohydrates.

Importance of management by Diet, drugs and exercise.

Sick day guidelines

Clarification of common food fads and misbeliefs,

| <b>Box 12.1</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             | <b>Sick Day Guidelines</b> |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------|
| <p>A sickness, even minor, can <b>increase</b> the body's <b>need</b> for insulin. Extra insulin may be required because of the extra <b>energy</b> required by the body to overcome the illness. This can result in energy needs being fulfilled from other sources of energy in the body like fats causing acidosis. On any sick day, one must consider the need and follow the sick day guidelines given below:</p> <ol style="list-style-type: none"> <li>1. Never omit the insulin dose, even if the person is unable to eat.</li> <li>2. Test the blood glucose level before each mealtime and at bedtime.</li> <li>3. If blood glucose levels are 240 mg/100 ml or higher, one should also test for urine ketones. <b>DO THIS WHEN ILL FROM ANY CAUSE.</b></li> <li>4. Take liquids every hour.</li> <li>5. If the patient is too ill to follow the meal plan, advice to eat and drink whatever they can tolerate. Use fluids, water, fruit juice, tea etc. If they are unable to take fluids because of vomiting, call the DOCTOR. Soft foods like baked custard, cooked soft cereal, creamed soups, milk shake, jelly, toast and even an ice-cream can be consumed.</li> <li>6. Rest or keep warm, do not exercise. Ask someone to care for the patient. If too unwell call the Doctor.</li> </ol> |                            |

## NOTES

Suitable information needs to be distributed to diabetics. This could be self care materials, food guides or even simple innovative recipes. Audiotapes and videotapes in local languages may be developed so as to facilitate wide spread dissemination of knowledge throughout the country.

| Box 12.2                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            | Food Fads and Misbeliefs |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------|
| <p>Some common food fads and fallacies linked with feeding diabetic patients are highlighted herewith.</p> <ol style="list-style-type: none"><li data-bbox="423 520 976 552">1. <i>You cannot eat rice if you are a diabetic.</i><br/>Both wheat and rice have a similar glycemic index which means that they raise the blood sugar level to the same extent. As long as you restrict the quantity (see food exchange), it does not matter what you eat.</li><li data-bbox="423 695 889 726">2. <i>You can fast if you are a diabetic.</i><br/>Total fasting is not good especially for those who are on insulin or hypoglycemic drugs. It causes acute complications of hypoglycemia which can be life threatening.</li><li data-bbox="423 869 1065 900">3. <i>Carbohydrates should be restricted in diabetics.</i><br/>It is now known that carbohydrates need not be restricted and it could provide 60-65% of the day's calories. Complex carbohydrates (high fiber content) found in cereals, pulses or green vegetables are known to improve the insulin action but simple carbohydrates present in sugar, sweets, honey and jams can increase blood sugar levels. Type of carbohydrate consumed is more important,</li></ol> |                          |

Initial and time to time counseling regarding prevention and control or management of diabetes must be obtained by individuals suffering from diabetes. In case of doubt seek the help of a dietitian. What are the preventive measures which a diabetic patient needs to practice to ensure a healthy life? These measures are discussed finally.

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## 12.7 PREVENTION

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We now know that diabetes is the outcome of many genetic and environmental factors. The former is not in our control but the latter can be modified. The most important rule in the prevention strategy is to maintain ideal weight. We have already learnt in Unit 9 about this concept. We can use the information given in the unit to calculate ideal weight for a diabetic patient depending on the height, gender and activity pattern. Can you not?

Yes you can. So you can prevent diabetes by encouraging the diabetic

**NOTES**

patient to:

1. Maintaining ideal body weight.
2. Avoid high sugar, salt and fat foods.
3. Take small bites and chew foods thoroughly. Eat and drink slowly.
4. Eat protein rich food at each meal.
5. Eat wisely at social gatherings and restaurants.
6. Do physical activity. Walk daily.
7. Avoid aerated and excessive alcoholic drinks.
8. Minimize use of tobacco and other harmful drugs.
9. Use artificial sweeteners if you have a 'sweet tooth'.
10. Read and educate yourself on various aspects of diabetes.
11. Avoid stress; enjoy good music, meditate and have positive attitude towards life. In other words make life-style and behavioural changes for better health.

The section above must have helped you in understanding about the overall management of diabetes. Next, the check your progress exercise 3 given herewith will help you in recapitulating your concepts.

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## 12.8 LET US SUM UP

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Diabetes as we all know is a disease with ever increasing prevalence in our country. It can be described as a disease characterized by disordered metabolism and Inappropriately hyperglycemia due to a deficiency either of insulin secretion or to a combination of insulin resistance and inadequate insulin secretion.

In this we have learnt about the various types of diabetes and impaired glucose tolerance in different age groups. A brief note on the symptoms and complications of diabetes, as well as, the common diagnostic tests to identify and monitor diabetes are also given. The unit further elaborated on the overall management of diabetes with emphasis on diet related aspects.

We learnt that the management of diabetes involves a co-ordination between the diet, drugs and activity pattern of the patient, The nutrient requirements are elaborated and the use of alternative food components/ supportive therapy is also discussed. This unit also dealt with the significance and advantages of regular exercise for maintaining blood glucose within normal range. The use of different types of drugs/ insulin, their characteristic features and duration of action were also discussed thereafter.

A dietitian is very often involved with counseling sessions which are aimed to help the patient in becoming self-reliant regarding the day-to-day management

of variations in blood glucose. Some useful tips regarding an effective education programme were also elucidated.

## NOTES

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### 12.9 GLOSSARY

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|                           |                                                                                                                                     |
|---------------------------|-------------------------------------------------------------------------------------------------------------------------------------|
| <b>Acidosis</b>           | : depletion of the body's alkali reserve with resulting disturbance of acid-base balance. Excess acid in the blood and body fluids. |
| <b>Ascites</b>            | : a type of oedema characterized by the accumulation of fluid in the abdominal cavity.                                              |
| <b>Insulin resistance</b> | : the condition in which the cells fail to respond to insulin as they do in healthy people.                                         |
| <b>Polyuria</b>           | : excessive urination due to glucose in urine.                                                                                      |
| <b>Polyphagia</b>         | : increased appetite due to inadequate utilization of glucose.                                                                      |
| <b>Somogyi</b>            | : effect hyperglycemia in the morning as a feed back response of counter- regulatory hormones to nocturnal hypoglycemia.            |
| <b>Dawn phenomenon</b>    | : reduced tissue sensitivity early morning resulting in mild hyperglycemia.                                                         |

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### 12.10 CHECK YOUR PROGRESS

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- 1). Explain the following terms:
  - a) Polyurea
  - b) Paraesthesia
- 2). Explain the following terms:
  - a) Retinopathy
  - b) Neuropathy
- 3). What foods can be given daily in moderate amounts for a diabetic?
- 4). Describe the following briefly:
  - a) Food exchanges
  - b) Glycemic Index of foods

**NOTES**

## GOUT AND INBORN ERRORS OF METABOLISM

### STRUCTURE

- 13.1 Learning Objective
- 13.2 Introduction
- 13.3 Gout
- 13.4 Inborn Errors of Metabolism
- 13.5 Let Us Sum Up
- 13.6 Glossary
- 13.7 Check Your Progress

### 13.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- enumerate the etiological factors and symptoms of gout and few inborn errors of metabolism,
- describe the metabolic changes in these disease conditions and the complications, diagnose the condition(s),
- discuss management of the diseases, and
- provide guidelines for diet counseling, prevention and control.

### 13.2 INTRODUCTION

Metabolic diseases, as you already know, refer to those disorders in which the various reactions in the cells are effected (production of energy or utilization of energy) due to abnormal production one or more hormones, or a deficiency of an enzyme. Most metabolic disorders are genetic, though a few are "acquired" as a result of diet, toxins, infections, etc. Metabolic disorders or inborn errors of metabolism are inherited traits that result in the absence or reduced activity of a specific enzyme or cofactor. In general, these genetic metabolic disorders are caused by genetic defects I-hat result in missing or improperly constructed enzymes necessary for some step in the metabolic process of the cell. Phenylketonuria,



tyrosinemia, maple syrup urine disease, homocystinuria and galactosemia are some common metabolic diseases caused by genetic defects.

Gout, is yet another metabolic disease caused due to a disturbance of uric acid metabolism occurring chiefly in males, characterized by painful inflammation of the joints, especially of the feet and hands, and arthritic attacks resulting from elevated levels of uric acid in the blood and the deposition of urate crystals around the joints. The condition can become chronic and result in deformity. What is the cause of these metabolic disorders? How to diagnose and prevent them? What is the treatment? These are a few aspects discussed in this unit.

## NOTES

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### 13.3 GOUT

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#### Role of Protein and Purines

Cellular materials of plants, grams and legumes and animal glandular organ meats (liver, pancreas brain, kidneys) contain nucleoproteins. The nucleoproteins are digested and converted to purines, which are finally oxidized to uric acid. Uric acid, therefore, is a substance that results from the breakdown of purines, which are part of all human tissue and are found in many foods. You may also recall studying that the body can also synthesize proteins from carbon and nitrogen compounds (acetic acid, glycine) from carbohydrates, protein or fat and give rise to uric acid.

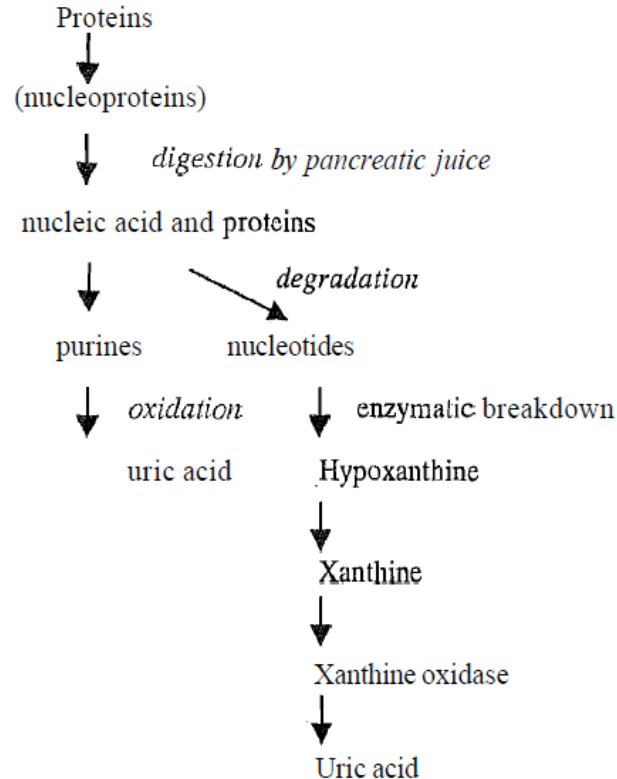


Figure 13.1: Degradation of purine to uric acid

## NOTES

It is important to understand that uric acid is normally excreted in the urine. However, excess of uric acid produced by the body from protein we eat and/or internal cellular or N<sub>2</sub> utilization can increase the uric acid levels of the blood. The range of uric acid is 2-7 mg/100 ml. If the level of uric acid in our body is above 7 mg/100 ml, we have a risk of developing the disorder called gout. In severe gout, the levels of uric acid may go up to 20 mg/100 ml.

Normally, uric acid is dissolved in the blood and passed through the kidneys into the urine, where it is eliminated. If the body increases its production of uric acid or if the kidneys do not eliminate enough uric acid from the body, levels of it build up in the blood resulting in a condition called hyperuricemia. Hyperuricemia also may result when a person eats too many high-purine foods, such as liver, dried beans and peas and gravies. Hyperuricemia is not a disease and by itself is not dangerous. However, if excess uric acid crystals form because of hyperuricemia, gout can develop. The excess crystals build up in the joints spaces, causing inflammation. Deposits of uric acid, can appear as lumps under the skin around the joints and at the rim of the ear. In addition, uric acid crystals can collect in the kidneys and cause kidney stones.

### 13.3.1 Etiopathology

Who is likely to develop gout? What are the risk factors? Let us find out.

Gout is caused when there is over production of uric acid in normal purine metabolism in the body. In fact, a number of risk factors are related to the development of hyperuricemia and gout. These factors include:

- Hereditary : Genetics may play a role in determining a person's risk, since up to 18 percent of people with gout have a family history of the disease.
- Gender and age are related to the risk of developing gout; it is more common in men than in women and more common in adults than in children.
- Being overweight increases the risk of developing hyperuricemia and gout because there is more tissue available for turnover or breakdown, which leads to excess uric acid production.
- Drinking too much alcohol can lead to hyperuricemia because it interferes with the removal of uric acid from the body.
- Eating too many foods rich in purines can cause or aggravate gout in some people.
- An enzyme defect that interferes with the way the body breaks down purines causes gout in a small number of people, many of whom have a family history of gout.
- Exposure to lead in the environment can cause gout.,

The disease predominantly affects males after the age of 35 years. Gout starts suddenly with an arthritic pain in the big toe and may continue up to the leg. Small injury or excessive exertion can precipitate the attack. Sometimes exposure

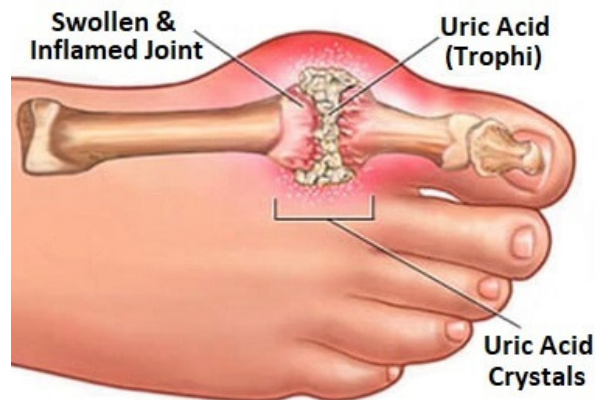
to cold, surgery, minor trauma can trigger the attack.

Secondary gout could be due to genetic abnormality of uric acid metabolism.

### 13.3.2 Clinical Features and Complications

You may be enjoying good health but may suddenly get a severe attack. You wake up in the middle of the night, and your big toe feels as if it is on fire. It is hot, swollen and so tender that even the weight of a blanket on it seems intolerable. These problems could indicate an acute attack of gout characterized by sudden, severe attacks of pain, redness and tenderness in joints.

The big toe gets affected mostly but in chronic conditions, it could be the elbow or the helix (outer fleshy ridge of the ear) can also be the site. The uric acid crystals (called tophi) deposit as urate in the joint causing swelling and tenderness of the joint with severe pain. Presence of uric acid crystals in the joint fluid, therefore, is an important diagnostic test for the disease. The doctor will take a sample of the joint fluid and look for the presence of uric acid crystals. Gout is also easily identifiable through a physical examination. The skin is tense, red and shiny and may be associated with fever, anorexia (lack of appetite) and malaise (unwell feeling)



**Figure 13.2: Uric acid crystals in the big toe**

The main complication of chronic gout is, however, chronic renal failure. That is a big problem.

### 13.3.3 Management of Gout

The goals of therapy or management of gout is based on the following aspects:

- early resolution of inflammation,
- prevention of recurrent attacks, and
- reversal of complications arising from deposition of urate crystals in joints, kidneys.

To help meet these goals the treatment is based on drug and diet therapy. Let us

### NOTES

consider these one by one.

### ***Dietary Management***

#### **NOTES**

Treatment for gout often include a diet of lower purine intake. Indeed, about one third of the body's uric acid can be attributed to diet. Changing the diet to foods with lower purines can help relieve the symptoms, as well as, address the actual problem of hyperuricemia (elevated levels of uric acid in the blood).

Since diet is an important factor, exclusion of foods extremely high in purines may be helpful. All meats, fish, poultry contain moderate to high amounts of purine and pulses and lentils need to be avoided. Some vegetables contain low to moderate amounts of purine. Efforts to greatly restrict these foods are generally unnecessary because of their insignificant effect as compared to medications. In fact, drugs are so effective in lowering the serum urate concentration that rigid restriction of dietary purines is rarely necessary. When purine is restricted, as in case of severe gout, it should be restricted to 100-150 mg/day. This list can serve as a handy guide for the patients in selection of food items. The thumb rule for dietary management is to advice the patient to try to cut down or avoid:

Red meats.

Organ meats such as brains, kidneys, liver and heart.

such as mussels, oysters, sea eggs etc.

Peas and beans.

Alcohol, especially beer and wine.

**Table 13.1: Dietary recommendations for gout**

| <i>Avoid foods highest in purines (150 - 825 mg / 100 gm)</i> | <i>Limit foods containing moderate amount of purines (50 -150 mg/ 100 gm)</i> | <i>Consume foods lowest in purines (0-50 mg / 100 gm)</i> |
|---------------------------------------------------------------|-------------------------------------------------------------------------------|-----------------------------------------------------------|
| Brain                                                         | Whole grain bread or cereals                                                  | Beverages (coffee, tea and soda)                          |
| Kidney                                                        | Cauliflower                                                                   | Refined cereals                                           |
| Liver                                                         | Spinach                                                                       | Cheese                                                    |
| Gravies                                                       | Fresh saltwater fish                                                          | Eggs                                                      |
| Herring                                                       | <b>Legumes</b> (beans, peas and lentils)                                      | <b>Fat</b>                                                |
| Sardines                                                      | Meat soups and <b>broth</b>                                                   | Fruits and fruit juices                                   |
| Broth                                                         | Mushrooms                                                                     | Milk                                                      |
| Meat Extracts                                                 | Asparagus                                                                     | Nuts                                                      |
| Minced meat                                                   | Oatmeal                                                                       | Sugar syrup                                               |
| Sweet Breads                                                  | Chicken                                                                       | Vegetable                                                 |
|                                                               | Spinach                                                                       | <b>creamed soups</b>                                      |
|                                                               | Wheat germ and bran                                                           | <b>Macaroni/Noodles</b>                                   |

**NOTES**

| Permitted                                                                                                                      | Excluded                                                                                |
|--------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------|
| Refined cereals and cereal products-<br><b>cornflakes</b> , white bread, pasta, flour, arrow-<br>root, sago, tapioca and cakes | Beans, peas, lentils, spinach,<br>oatmeal, <b>asparagus</b> ,<br>cauliflower, mushrooms |
| Milk, <b>milk</b> products and eggs                                                                                            | Fish, seafood                                                                           |
| Lettuce, tomatoes and green vegetables                                                                                         | Meats, poultry or other flesh;<br>meat extract, gravies,<br><b>marmite</b>              |
| Vegetables and cream soups made from<br>vegetables                                                                             | Liver, kidney,                                                                          |
| Sugar and sweets, gelatin                                                                                                      | Yeast and beer products, beer<br>alcohol                                                |
| Butter, polyunsaturated margarine, and fats<br>of any <b>kind</b>                                                              |                                                                                         |
| Fruit, nuts, peanut butter                                                                                                     |                                                                                         |
| Beverages – water, fruit juice, cordials,<br><b>carbonated</b> drinks, tea, coffee and cocoa                                   |                                                                                         |

**Table 13.2: Food items for a patient with gout**

Intake of fluids should be encouraged to assist with the excretion of uric acid and to minimize the possibility of renal calculi formation. Fluid intake  $\geq 3$  L/day is desirable for all gouty patients, especially for those who chronically pass uric acid or have calcium oxalate renal calculi. Because urate excretion tends to be reduced by fats and enhanced by carbohydrates; the diet should be relatively high in carbohydrates (providing 50% to 55% of calories) and low in fat (30% of calories), modified in cholesterol (300 mg/day) and protein intake should be moderate.

Further, since being overweight increases the risk of hyperuricemia, patients should be encouraged to lose weight during a quiescent phase of the disease. Finally, avoidance of alcohol should be recommended.

***Nutritional care: Summary***

Advise the patient to:

- **Maintain** ideal weight.
- Avoid high purine content foods.
- Take moderate protein, use low fat dairy products, eggs and cheese.
- Take liberal carbohydrates, refined cereals, beverages, fruits and **fruit** juices, vegetables.
- Take a low fat diet.
- **Restrict/eliminate** alcohol.
- Take liberal amounts of fluid.

### SAMPLE MENU FOR A PATENT WITH GOUT

#### NOTES

| Western Diet                                                                                                 | Vegetarian Diet                                                                   |
|--------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|
| <b>Breakfast</b>                                                                                             |                                                                                   |
| Breakfast cereal<br>Bread/toast with butter and fat/jam<br>Tea/Coffee                                        | Breakfast cereal<br>Toast/khakhra/Dosa<br>Tea/coffee                              |
| <b>Mid-morning</b>                                                                                           |                                                                                   |
| Tea/coffee<br>Biscuits                                                                                       | Tea/coffee<br>Cream crackers                                                      |
| <b>Lunch</b>                                                                                                 |                                                                                   |
| Shepherd's pie with gravy<br>Parsley potatoes<br>Steamed green beans<br>Bread and butter<br>Pudding          | Chapatties/Rice<br>Tomato paneer curry<br>AlooMethi<br>Butter                     |
| <b>Mid-afternoon</b>                                                                                         |                                                                                   |
| Tea/coffee<br>Biscuits                                                                                       | Tea/coffee<br>Biscuits                                                            |
| <b>Dinner</b>                                                                                                |                                                                                   |
| Smoked haddock with egg sauce<br>Mashed potatoes<br>Shredded cabbage<br>Tomato and cucumber salad<br>Pudding | Vegetable pulao<br>Kadhi (Spinach pakodi)<br>Salad (cucumber and tomato)<br>Kheer |

#### *Drug Management*

Drugs are so effective in lowering the serum urate concentration that rigid restriction of dietary purines is rarely necessary. Uricosuric drugs are used to increase uric acid excretion, Allopurinol inhibits the action of enzyme xanthine oxidase, which then produces less uric acid. These drugs must be taken close to the meals as prolonged use could damage the intestinal mucosa resulting in reduced absorption of several nutrients and large quantities of water. The person will lose weight gradually and must avoid excessive intake of alcohol.

### 13.4 INBORN ERRORS OF METABOLISM

We are now going to learn about some of the disorders caused by inborn error of metabolism namely phenylketonuria, tyrosinemia, maple syrup urine disease, homocystinuria and galactosemia. You may recall studying about these metabolic disorders earlier in your Nutritional Biochemistry Course (MFN-002) in Unit 12. We hope you remember the information presented there. If not, we suggest you look up this unit now and read the information given there along with the matter presented here in this section. This will help you consolidate your understanding on this important topic.

## NOTES

An inborn error of metabolism is a genetic error that alters the production of a protein. In many cases, the protein is an enzyme. When the enzyme is absent, the functions that depend on that enzyme can not proceed. Incompletely metabolized products accumulate in the body. This leads to a variety of problems and in many cases it becomes fatal. Further, this imbalance creates problems in other metabolic pathways that adds to existing problem. The goal of therapy is to prevent the accumulation of toxic metabolites and to replace essential nutrients that are deficient as a result of the defective metabolic pathway.

In these metabolic disorders, since there is an inability to metabolize a specific amino acid the basis of dietary management is to restrict the offending amino acid(s) by means of a special low protein diet. In addition, to meet the requirement for growth and development it is essential to supplement the diet with the appropriate amino acid mixture, which does not contain the offending amino acid.

Low-protein food products are recommended to persons with particular types of metabolic disorders such as homocystinuria (HCU), phenylketonuria (PKU), tyrosinemia etc. Each of these disorders requires food products, which are low, in particular type of amino acid i.e. methionine in the case of HCU, phenylalanine in the case of PKU. However, it is not easy to find high protein foods with low content of these amino acids. Hence, the patients are given low-protein foods as you would realize while studying these disorders.

### 13.4.1 Phenylketonuria

The essential amino acid phenylketonuria is utilized for tissue protein synthesis and hydroxylated to form tyrosine. The hydroxylation reaction requires phenylalanine hydroxylase. Phenylketonuria (PKU) is a group of inherited disorders of phenylalanine metabolism caused by impaired phenylalanine hydroxylase activity. PKU cannot break down phenylalanine into another amino acid, tyrosine. Phenylalanine then builds up in the bloodstream and causes brain damage.

Phenylketonuria, commonly referred to as PKU, occurs at the age of 3 to 6 months and is characterized by developmental delay, microcephaly (abnormally small head), abnormal electro-encephalogram, eczema, musty odour and hyper activity. If untreated before three weeks of age, the metabolic imbalance produces irreversible mental retardation. The defect in metabolism in classic PKU is associated with less than 2% activity of normal phenylalanine hydroxylase.

Accumulation of phenylalanine and its catabolic products leads to central nervous system damage. The extent of damage caused to the brain depends on the time at which the insult occurs. Deficient myelination and abnormalities in brain proteolipids and proteins occur in late gestation and during first 6 to 9 months of life. In the fully matured brain, the synthesis of neurotransmitters is affected. This might cause nerve degeneration, behavioural difficulties and delayed development.

## NOTES

Appreciate diagnosis is necessary to establish the mode of therapy. Patients with initial blood phenylketonuria level of  $121 \text{ m mol/l}$  ( $> 2 \text{ mg/dl}$ ) should repeat the test. There are several laboratory methods to confirm the disorder. These include ion exchange chromatography for quantification of amino acid concentration, determination of genotype of parents, and assays of bioprotein and dihydropteridine reductase.

Phenylketonuria can now be well controlled by special diet therapy. A low-phenylalanine diet effectively controls the serum phenylalanine levels. This will help to prevent clinical symptoms and promote normal growth and development. Remember that phenylalanine is an essential amino acid and therefore can not be totally avoided. Based on many studies, the following guidelines for dietary management of PKU are being used effectively.

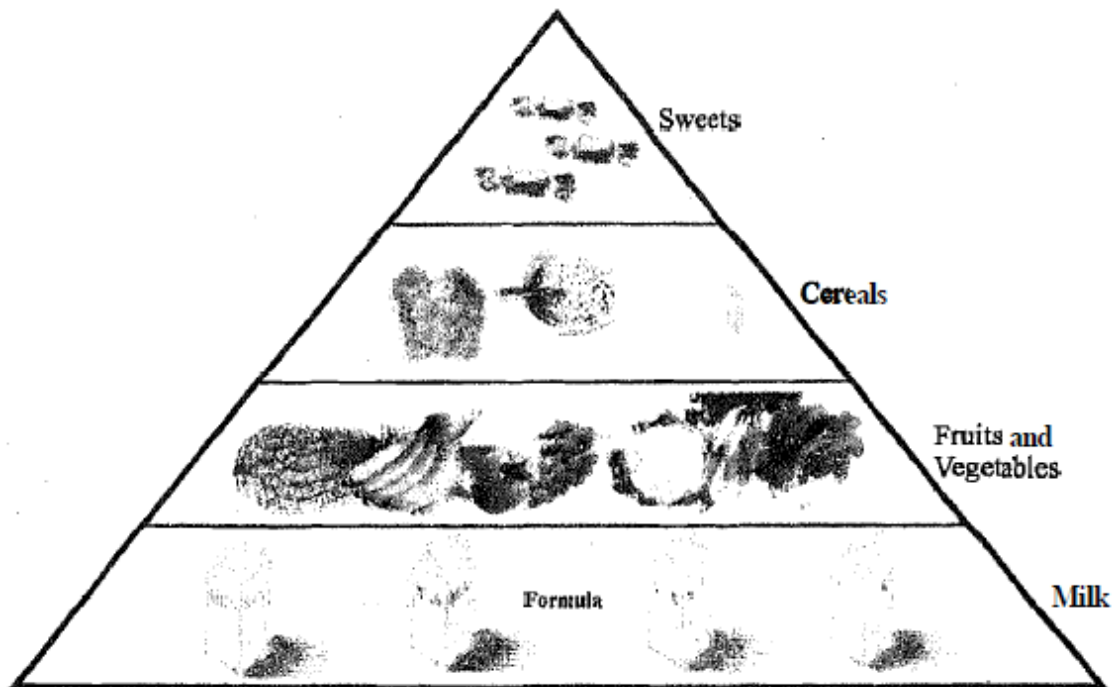
1. Estimate the daily energy, protein and phenylalanine requirements (child's age and weight dependent).
2. Calculate the amount of special formula to be given to provide the recommended allowances.
3. Assess the amounts of other foods to be used. Lofenalac is a formula which is nutritionally complete except for phenylalanine. This formula is the main diet for the infants suffering from PKU. The diet should be progressed as for a normal infant and child. Utmost care should be taken while introducing new foods to them. Effort should be taken to develop a variety of recipes using foods low in phenylalanine. Phenylalanine is found in milk, cheese, eggs, fish, meat, beans, nuts, and infant formulas (both regular and soy), and to a lesser extent in cereals, vegetables and fruit. People with PKU cannot break down phenylalanine into another amino acid, tyrosine. Phenylalanine then builds up in the bloodstream and causes brain damage as you learnt earlier. To avoid this, a person can eat a healthy diet that includes a low-phenylalanine formula, fruits, vegetables, sugars and other low-protein foods.

The majority of foods for a PKU patient should come from the bottom half of the pyramid. This contains the phenylalanine free formula. Lofenalac in one such formula. This bottom layer provides the major food needs. The next layer of the PKU food pyramid is built of fruits and vegetables which contain low levels of phenylalanine. Eating a variety of foods (pineapple, banana, grapes, apple, cauliflower, carrots and greens) will help the patient grow strong and healthy. The next layer of the PKU food pyramid is built of low protein foods such as breads, pasta, cereal, and rice. This layer does not contain as much food as the first two layers. Eating low protein foods from this layer will help the patient remain healthy. The top of the pyramid is built of candies and treats. These foods should be eaten just on special days. The foods outside the target are not included in the low-phenylalanine meal plan. These are high protein foods, such as milk, dairy products, meat, fish, chicken, eggs, beans, and nuts. These foods cause high blood



phenylalanine levels for people with PKU.

**Table 13.3: Food list for PKU patients**



**NOTES**

The optimal age for discontinuing the diet is not known. Phenylalanine restriction is recommended during pregnancy of the phenylketonuric woman. It will be better if these women restrict the diet at the time of conception, in order to prevent the risk of mental retardation, microcephaly and congenital heart disease in infants.

***Box 13.1 Important Facts Related to PKU***

- The brain of a fetus with classic PKU develops normally in intrauterine stage.
- The critical period of human brain growth and development extends over the first 6 months of neonatal life requiring that dietary therapy to be instituted right after birth.
- Myelination may not be completed until 5 or 6 years of age and hence dietary restriction must be rigidly followed.
- The proportion of dietary phenylalanine that is utilized for protein synthesis varies with age — 50-60% during early growth and only about 10% for normal

## NOTES

adult.

- Blood phenylalanine levels must be maintained between 3-15 mg/dl.
- For a phenylalanine- restricted diet 50-80% of the natural protein must be replaced by a protein preparation that contains little or no phenylalanine.
- Most natural proteins contain about 50 mg phenylalanine/g protein.
- The composition of the preparation should meet all nutrient requirements.
- Tyrosine must be supplemented in the diet.
- Usually one-third to one-tenth of normal phenylalanine content is recommended.
- Infections in the infant should be avoided to prevent tissue catabolism and increased phenylalanine levels in blood.
- Higher dietary phenylalanine intakes may be allowed after 6-10 years of age along with frequent clinical and biochemical supervision.
- Strict dietary restrictions should be adhered to by phenylketonuric women during pregnancy to prevent damage to the foetus.

With these guidelines, we end our study of PKU. Next, we move on to tyrosinemia.

### 13.4.2 Tyrosinemia

In the Nutritional Biochemistry Course in Unit 12, sub section 12.3.3, you may recall studying about tyrosinemia. There are two forms of hereditary tyrosinemia. They are tyrosinemia Type I and tyrosinemia Type II. Type I was thought to be due to a deficiency of parahydroxy phenylpyruvic acid oxidase. But recently secondary impairment in this enzyme has been attributed to a primary defect of hepatic fumaryl acetoacetate hydrolase. Patients with deficiency show renal tubular impairment, hypophosphatemic rickets, liver failure and hypertension. The plasma concentrations of phenylalanine and tyrosine are elevated.

In tyrosinemia type II, there is a very elevated concentration of blood and urine tyrosine. Increase in urinary phenolic acids, N-acetyltyrosine and tyramine is seen.

Other symptoms include corneal erosions, hyperkeratosis on the fingers and palms and sole of the feet. Mental retardation may occur.

Dietary modification includes nutritional support that allows normal growth and development. Plasma phenylalanine concentration between the range of 40 and 80  $\mu\text{mol/L}$  and tyrosine concentration between 50 and 150  $\mu\text{mol/L}$ , should be maintained. Phenylalanine - tyrosine restricted diet is advocated. Initially a hydrolysate low in phenylalanine and tyrosine is used with small amounts of milk. If there is an elevation in methionine a synthetic mixture without tyrosine, phenylalanine and methionine should be used. Individuals with tyrosinemia have food patterns very similar to the one for PKU discussed above. PKU is more common than tyrosinemia. Foods that are high in tyrosine and phenylalanine are high in protein. Thus, children with tyrosinemia should eat foods that are low in

## NOTES

protein. Each child can "tolerate" a different amount of tyrosine/phenylalanine, so each child will have a slightly different food pattern.

The most important food for a child with tyrosinemia, however, is his/her formula. Formula provides energy (calories), protein, and the vitamins and minerals (including those that are found in high protein foods). Foods that all children with tyrosinemia should avoid are foods rich in protein i.e. meat, chicken, fish, milk, cheese, beans and legumes, peanut butter, and eggs. These foods are often called "no" foods and are those foods outside the target as illustrated in Figure 13.4 above.

Foods with moderate amounts of protein can be eaten in limited amounts. These foods include grains, bread, pasta, rice, potatoes, corn, and peas. Foods with little or no protein are the mainstay (in addition to formula). These foods include most fruits and vegetables. Low protein products, including bread, pasta, noodles, rice etc. may also be used. Therefore, we may conclude by saying that bread, cereals, fruits and vegetables and fats are allowed. A high carbohydrate feed, providing 65-75% of calories may be recommended.

### 13.4.3 Maple Syrup Urine Disease

Maple Syrup Urine Disease (MSUD) is a group of inherited metabolic disorders of three branched chain amino acids (BCAA) namely leucine, isoleucine and valine. These three amino acids are normally metabolised to ketoacids and then decarboxylated to simple acids. In MSUD disorder an oxidative decarboxylase in the white blood cells is missing. Since the carboxyl groups can not be removed there is an accumulation of branched chain ketoacids and their amino acid precursors. The branched chain ketoacids are relatively acute neurotoxins and probably interfere with oxygen consumption and ATP production in the medullary reticular substance of the brain. There is a progressive neurologic dysfunction and production of fragrant urine with the odour of burnt sugar or maple syrup.

Infants with MSUD appear normal at birth. Once they are fed on protein containing feed, they start showing the classic symptoms. Neurologic impairment in the new born is manifested by poor sucking, irregular respiration, and intermittent periods of rigidity and flaccidity.

Clinical manifestations are expressed upon protein loading or with febrile illness. In most severely impaired enzyme deficiency, seizures, apnea (brief pause in breathing) and death may occur. In untreated patients with classic MSUD who survive beyond early infancy, there is retardation of physical and mental development. Early diagnosis and therapy lead to normal growth and development.

Most infants with classic MSUD have greater than 8 mg/dl leucine at 72 hours of diagnosis. Diagnosis is confirmed using ion exchange chromatography to quantify Inborn Errors of Metabolism plasma isoleucine, leucine, valine and alloisoleucine, and urinary

## NOTES

branched chain keto acids.

Special feeds containing energy and proteins without the branched chain amino acids can be given to the infants. In some infants if the orogastric feeding is not acceptable, gastrostomy is initiated for initial care during the neonatal period. Long-term therapy for MSUD is by means of diet. Patients with classic MSUD are unable to terminate diet. Plasma concentration of isoleucine, leucine and valine should be maintained in the range of 40 to 90 m mol; 80 to 200 m mol, 200 to 425 m mol, respectively.

Long-term treatment of MSUD involves a carefully controlled diet which strictly limits dietary protein in order to prevent the accumulation of BCAAs in the blood. The major component of the diet is a special formula designed for children with MSUD. These MSUD formulas do not contain any leucine, isoleucine or valine but are otherwise nutritionally complete. They contain all the necessary vitamins, minerals, calories and the other amino acids needed for growth. The diet of a MSUD patient (child) should therefore involve:

Measured quantities of natural protein or leucine from foods.

A BCAA free protein, vitamin and mineral supplement.

The natural protein and vitamin/mineral supplement should ideally be evenly distributed through the day to allow maximum utilization of the amino acids for protein anabolism, and for tolerance of the supplement.

Free foods that are low in BCAA including sugars, fats and oils, as well as, foods specifically produced for a low protein diet (breads, biscuits, pastas etc.) and supplements be given.

Supplements of valine and/or isoleucine are helpful.

### 13.4.4 Homocystinuria

Homocystinuria may result from errors of methionine metabolism. This produces defects in the function of cystathionine b synthase or 5-methyltetrahydrofolate homocysteine methyltransferase. The most common form of homocystinuria is caused by a deficiency of the enzyme cystathionine b synthase. This enzyme is essential for the conversion of homocysteine to cystathionine. Severely impaired enzyme function produces accumulation of plasma homocysteine and methionine and decreased cysteine in cells.

Large amounts of homocystine are excreted in the urine. If this is unattended early in life, skeletal changes, dislocated lenses, intravascular thrombosis, osteoporosis and mental retardation might occur.

Therapeutic dose of pyridoxine (1g/day) is usually tried in all patients with hypermethioninemia and homocystinemia. For patients who do not respond to pyridoxine, methionine restricted diet supplemented with L-cysteine is used. If plasma folate concentrations are below normal, folate should be given as a supplement. What are the special preparations and foods low in or free of

methionine? This list can be referred to while planning diet plans for patients suffering from homocystinuria. While planning the diet for homocystinuria one must consider energy, protein, methionine, cysteine, folate, vitamins B<sub>6</sub> and B<sub>12</sub> and fluid needs.

**NOTES**

**Table 13.4: Foods and food items containing varying levels of methionine**

|                                                                                                                       |                                                                                                                                                                                                                   |
|-----------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Vegetables that are <b>high</b> in methionine/ per 100 gm (more than 25 mg-50 mg of methionine/100 gm of the product) | Broccoli, Mushroom, Cauliflower, Avocado, Bean sprouts, Potatoes,                                                                                                                                                 |
| Vegetables that are <b>high</b> in methionine/ per 100 gm (more than 50 mg of methionine/100 gm of the product)       | Spinach, Green Peas, Corn (boiled),                                                                                                                                                                               |
| Vegetables that are <b>low</b> in methionine/ per 100 gm (less than 25 mg / 100 gm of the product)                    | Carrot ,Beetroot (boiled), Tomatoes, Green Pepper, Red Pepper, Yellow Pepper, Eggplant, Green Cabbage and Red Cabbage, Kale (boiled), Lettuce, Cucumber, Green Beans (boiled), Red and Brown Onions, Celery, Okra |
| Fruits that are <b>medium high</b> in methionine 25-50 mg of methionine/100 gm of the product)                        | Mandarin Oranges                                                                                                                                                                                                  |
| Fruits that are <b>low</b> in methionine (less than 25mg of methionine/ 100 gm of the product)                        | Watermelon, Cantaloupe, Honeydew melon, Apples, Pears, Cranberries, Raspberries, Blueberries, Strawberries, Mango, Plums, Dates (dried), Peaches, Nectarines, Banana, Pineapple, Apricots                         |
| Snacks that are moderate in methionine (25-50 mg of methionine/ 28 gm of the product)                                 | Potato chips (28 gm), French Fries(10 pieces)                                                                                                                                                                     |
| Snacks that are <b>high</b> in methionine (more than 50 mg of methionine/ 28 gm of the product)                       | All nuts, like Peanuts, Pistachio and Popcorn                                                                                                                                                                     |
| Other foods that are <b>high</b> in methionine (more than 100 mg of methionine/100 gm of the product)                 | Tofu, dried beans like Kidney beans, Black beans, Tempeh                                                                                                                                                          |

The discussion so far focused on the disorders linked to protein metabolism. Disorders of carbohydrate metabolism also exist. One such disorder is galactosemia which is also an inherited disorder characterized by an inability of the body to utilize galactose. Let us get to know more about this disorder.

**13.4.5 Galactosemia**

Galactosemia is a genetic disorder caused by deficient functioning of any of these three enzymes namely galactokinase, galactose -1 - phosphate uridyl transferase, or UDP galactose - 4- epimerase. Galactose derived from the hydrolysis of lactose in the intestine is absorbed normally. But in the absence of gal-I-P-transferase it leads to the accumulation of galactose, galactose-I-phosphate, and galactitol in the blood and tissues. Accumulation of gal-I-P reduces the intracellular phosphate for high energy phosphate bonds. Thus ATP, GTP and CTP are reduced.

**NOTES**

Patients with galactokinase deficiency suffer only from cataract, Galactitol accumulates in the lens of the eye creating an osmotic gradient that allows the glutathione from the lens to efflux. Due to this the concentration of glutathione in the lens is decreased. Glutathione peroxidase and hydrogenperoxidase are inactivated. As a result hydrogen peroxide accumulates in the lens denaturing the proteins of the lens.

Clinical symptoms of gal-1-P-transferase deficiency is found to appear early in infancy. Some infants are born with cataract, jaundice and cirrhosis. The reason may be due to maternal ingestion of lactose. In untreated patients, development of hepatomegaly is very common. Liver is damaged leading to decreased synthesis of prothrombin and albumin. Glomeruli and tubules of the kidney are affected due to the accumulation of galactose and its metabolites. In addition, active tubular transport is impaired because of deficient AIP. Aminoaciduria occurs. Decreased albumin synthesis, proteinuria, ascites and generalized oedema is seen. Without treatment death usually results. Even if the untreated patients survive, there is retardation of physical and mental growth.

Diagnosis of galactosemia is by measuring the activity of gal-I-P-transferase in erythrocytes. Galactose should be restricted if the gal-I -P-transferase is elevated above 2 mg/dl.

Objectives of diet therapy in galactosemia are to prevent symptoms and to provide nutrients for normal growth and development. Treatment should begin in the first few days of life itself. The goal of dietary treatment for galactosemia is to minimize galactose intake which in turn minimizes galactose-1-phosphate production. Therefore, dietary treatment of galactosemia is to remove any foods containing galactose from the diet. Because zilk and milk products are the most common food source of galactose, persons with galactosemia should avoid these foods. The diet allows most protein-containing foods other than milk and milk products. Fruits, vegetables, grains, breads, fats and sugars are acceptable, as long as they do not have ingredients that contain galactose. Some fruits and vegetables do contain small amounts of galactose. However, the form of galactose (bound galactose) found in fruits and vegetables is not usable by the body, and may not contribute to elevated blood gal-1-p.

Table 13.5: Galactose content of some common foods

| Item                   | Galactose/100 mg/ml |
|------------------------|---------------------|
| Cow's milk             | 227                 |
| Human milk             | 350                 |
| <b>Infant formula:</b> |                     |
| Soy protein isolate    | 1.5                 |
| Casein hydrolysate     | 6-7.5               |

**NOTES**

|                           |         |
|---------------------------|---------|
| Fruits and vegetables     | <0.5-35 |
| <b>Legumes, (cooked):</b> |         |
| Garbanzo beans            | 444     |
| <b>Baby lima</b>          | 175     |
| Kidney                    | 153     |
| Lentil                    | 116     |
| Soybean                   | 44      |

All sources of lactose and galactose should be totally excluded from the diet. Nutritional requirements for these infants and children are the same as the normal children. Foods that are excluded from the diet therefore, include:

Milk of any species.

All products containing milk such as curds, cheese, ice-cream, milk shake, butter, cream, milk sweets.

Soups containing milk or cream.

Fruits containing galactose.

Any processed foods with lactose.

Salad dressings containing milk.

Baked products containing milk.

Patient or the family members of the patients must be educated to read food labels while purchasing food. The labels of all processed foods must be read carefully for ingredients it contains. The milk products to be avoided in processed foods are milk, casein, dry milk solids, lactose, curds and whey.

The milk proteins casein and caseinate must be limited in the diet. They can provide large amounts of galactose if many foods or large amounts of any food containing casein are eaten. The following products may be used because they do not contain lactose: lactate, lactic acid, lactylates and calcium compounds.

Milk and milk products are the usual dietary source of calcium. Because persons with galactosemia remove milk products from their diet, they need to add calcium back into their diet through supplements. All people with galactosemia should be given a regular daily supplement of calcium. A soy-based formula can be used or tablets can be given.

Fermented soy products (fermented soy sauce, miso etc.) are not recommended as galactose can be released in the fermentation process. Non-fermented soy products (tofu, textured vegetable protein, and soybean extract) are acceptable.

We hope having gone through the discussion presented above you would now find yourself better equipped to deal with patients coming to you for dietary management of the metabolic diseases.

## 13.5 LET US SUM UP

In this unit, we studied about the nutritional management of metabolic diseases such as gout and a few inborn errors of metabolism such as phenylketonuria, tyrosinemia, MSUD, homocystinuria and galactosernia.

Gout, we learnt, is a chronic disease due to an inherited abnormality of purine metabolism. Dietary treatment for gout often include a diet of lower purine intake. Drugs are so effective in lowering the serum urate concentration that rigid restriction of dietary purines is rarely necessary.

Finally, in our last section, we dealt with the major diseases of inborn errors of metabolism and their nutritional management. An overview of the disorders of Inborn Errors of Metabolism is given below:

| Disorder        | Caused due to the Defects in the Function of | Characteristics                                                                                                                                        | Diet Therapy                                                                                          |
|-----------------|----------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------|
| Phenylketonuria | Phenylalanine hydroxylase                    | Developmental delay, microcephaly, nerve degenerations                                                                                                 | Low phenylalanine diet, Lofenalac formula                                                             |
| Tyrosinemia     | Hepatic fumaryl acetoacetate hydrolase       | Corneal erosion, hyperkeratosis on the fingers and palms, sole of the feet, mental retardation may occur                                               | Phenylalanine and tyrosine restricted diet.                                                           |
| MSUD            | Oxidative decarboxylase                      | Accumulation of branched chain amino acids. Production of fragrant urine with the odour of burnt sugar. Retardation of physical and mental development | Special feeds containing energy and protein without branched chain amino acids.                       |
| Homocystinuria  | Cystathionine $\beta$ -synthase              | Accumulation of homocysteine. Skeletal changes, dislocated lenses, intravascular thrombosis, osteoporosis, mental retardation may occur.               | Pyridoxine(1g/day) is usually given methionine restricted diet supplemented with L-Cysteine is given. |

## 13.6 GLOSSARY

**Arthritis** : inflammation of a joint, usually accompanied by pain, swelling, and structural changes.

**Congenital** : existing from birth or before

**Deamination** : removal of amino group (NH<sub>2</sub>) amino acid.

**Metatarsal** : the five bones of the foot between the ankle and the toes.

**Microcephaly** : pertaining to an abnormally small head.



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## 13.7 CHECK YOUR PROGRESS EXERCISES

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Gout And  
Inborn Errors Of  
Metabolism

### NOTES

- 1). Give five important aspects of the nutritional care summary.
- 2). What is galactosemia? Also define its symptoms and diagnosis methods.
- 3). What is Maple Syrup Urine Disease?
- 4). Give brief details about inborn error of metabolism.

## NUTRITIONAL MANAGEMENT OF GASTROINTESTINAL DISEASES AND DISORDERS

### STRUCTURE

- 14.1 Learning Objective
- 14.2 Introduction
- 14.3 Gastrointestinal Diseases and Disorders
- 14.4 Let Us Sum Up
- 14.5 Glossary
- 14.6 Check Your Progress

### 14.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- explain the disease conditions, causes, complications of the disorders of the gastrointestinal tract,
- discuss the effect of diseases on normal functioning of the gastrointestinal tract,
- describe the modification of the regular or normal diet to suit these disease conditions, and
- enumerate the principles of nutritional management in different disorders and diseases of the gastrointestinal tract

### 14.2 INTRODUCTION

In our previous units, we dealt with major metabolic disorders. In this unit, we shall study about the diseases and disorders of the gastrointestinal (GI) tract. Have you ever suffered from abnormal symptoms of the gastrointestinal tract? The symptoms could be as simple as nausea, anorexia, weakness to more severe ones like abdominal pain, abdominal gas and flatulence, delayed gastric emptying, diarrhoea or very severe ones such as the dumping syndrome, malabsorption syndromes and many others that we will learn about in this unit. In order to prevent the development of these disorders, the gut must remain healthy so that the GI tract functions can be carried out normally or else the site and the extent

of the disease process can affect the nutrient absorption and cause malnutrition. In this unit, we will touch upon the common gastrointestinal disorders and diseases to highlight the causes, important signs and symptoms and the dietary management of the problems.

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## 14.3 GASTROINTESTINAL DISEASES AND DISORDERS

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Before discussing the many gastrointestinal problems, it is useful to understand the basic physiology of the gastrointestinal tract. The gastrointestinal system performs the following four important functions. It helps to:

- store food,
- mix the food with enzymes produced in different parts of the gastrointestinal tract to break the complex foods to simpler forms of food (digestion),
- propel the food mixture through mouth, oesophagus, stomach, duodenum, small and large intestines to the anus, and
- absorb the various nutrients into the blood especially from small intestine and outer parts.

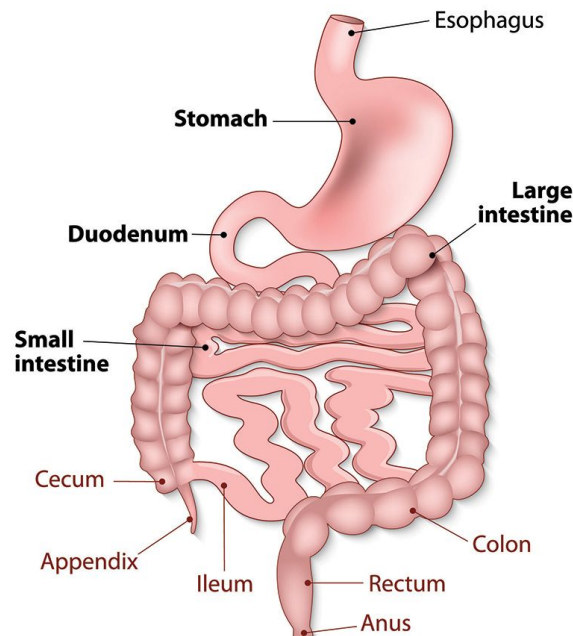


Figure 14.1: Various parts of the GI tract

Looking at the functions, you can understand that any disorder or diseases of a particular part can affect the storage, propulsion, digestion and result in nutrient deficiencies. We shall discuss the same in this unit and will begin with diarrhoea — one of the most common and fatal GI tract problems.

### 14.3.1 Diarrhoea

**NOTES**

What is diarrhoea? Diarrhoea is characterized by the frequent evacuation (f liquid stools, usually exceeding 300 ml, accompanied by an excessive loss fluids and electrolytes, especially sodium and potassium. It occurs when there is excessively rapid transit of intestinal contents through the small intestine, decreased enzymatic digestion of foods , decreased absorption of fluids and nutrients or increased secretion of fluids into the GI tract. It is important to note here that diarrhoea is a symptom and not a disease.

An episode of diarrhoea can be acute (recent origin) or chronic (extended duration and repeated episodes) in nature. You may recall reading in the Food Microbiology and Safety Course about microbial infections and toxins, which are a major cause of diarrhoea among individuals. However, there are several other causes of diarrhoea such as metal poisoning, deficiency of enzymes, side effects of drugs, structural/ functional abnormalities in the organs etc.

| Acute Diarrhoea                                                                                          | Chronic Diarrhoea                                                 |
|----------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------|
| Heavy metal poisoning e.g lead, mercury, arsenic.                                                        | Malabsorption, lesions of anatomic, mucosal or enzymatic origin.  |
| Viral infection (rotavirus)                                                                              |                                                                   |
| Bacterial toxin (Salmonella, related to food poisoning), Bacterial infection (E. Coli; <i>Shigella</i> ) | Metabolic disease such as diabetic neuropathy, Addison's Disease. |
| Drugs (Ncomycin, colichine, antibiotics, antacids, chemotherapy, digoxin, sorbitol)                      | Carcinoma of small intestine and colon.                           |
| Psychogenic factors                                                                                      | Cirrhosis of liver                                                |
| Protozoa infection (giardia, lamblia, entamoeba histolytica)                                             | Allergy and food sensitivity                                      |

**Table 14.1: Causes of diarrhoea**

It must be evident from the table above that acute diarrhoea generally occurs in association with infections, poisons and drugs. Chronic diarrhoea on the other hand are the result of long-term diseases such as malabsorption syndromes, deficiency of GI secretions, chronic deficiencies/allergies etc. Some common forms of chronic diarrhoea which you may come across while managing other disease conditions include:

- **Osmotic diarrhoea:** This kind of diarrhoea is caused by the presence of osmotically active substances in the intestinal tract, which in turn, favour the drawing of large volumes of water in the gut e.g. diarrhoea associated with lactose intolerance (sugar lactose is not digested due to lack of enzyme lactase in the intestine), dumping syndrome (multiple symptoms related to removal of part of stomach).
- **Secretory diarrhoea:** It is a result of active secretion of electrolytes and water by the intestinal epithelium caused by bacterial and viral infections. These,

in turn, lead to the production of exotoxins and increased intestinal hormone secretion.

- **Exudative diarrhoea:** It is associated with the mucosal damage leading to out pouring of mucus, blood and plasma proteins with a net accumulation of water and electrolytes in the gut.
- **Limited mucosal contact diarrhoea:** It results from situations of inadequate mixing of chyme (semi-liquid mass of food passing through intestine) and inadequate exposure of chyme to intestinal epithelium because of destruction and decreased mucosa due to surgical procedure. This type of diarrhoea is usually complicated by steatorrhoea (increased amount of fat in feces).

## Consequences of Diarrhoea

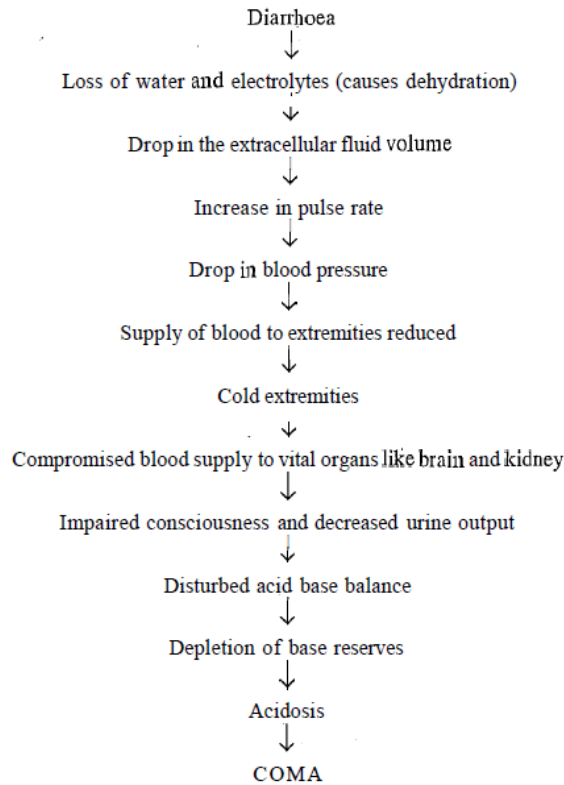
All of us must have suffered from diarrhoea at least once in our lifetime. How do you feel thereafter? Well most of us must have experienced weakness, dizziness, dryness of mouth and anorexia. Our skin also becomes dry and loose. During diarrhoea the stools are loose and have a high water content — an indicator that water is being lost in higher than normal amounts. The stools also contain a high amount of electrolytes due to enhanced peristaltic movements i.e. increased movements of the stomach and intestines. This results in the deficiency of water and electrolytes in the body which is referred to as dehydration. Dehydration results in reduction in the extracellular blood volume and hence a reduction in the total blood volume which is often referred to as hypovolemia.

Low blood volume is associated with hypotension and a low cardiac output. In response to hypotension, the heart tries to compensate by increasing the number of cardiac cycles per minute which is indicated by a high pulse rate. You will often find that during diarrhoea the patients have low blood pressure but usually a higher than normal pulse rate. As the severity of dehydration increases, despite enhanced cardiac cycles, adequate amount of blood and nutrients do not reach all body parts/organs. The patient is therefore cold to touch at the extremities. Another complication that may arise if severe dehydration does not receive prompt medical care is ischemic damage to the tissues of various organs due to reduced supply of oxygen and nutrients. Of most significance is damage to the kidneys and brain. Reduced blood volume results in reduced glomerular filtration rate and hence a low urine output.

This in turn can result in accumulation of toxic waste products in the blood which can be measured by the level of blood urea nitrogen levels. Other associated changes can be observed in the form of azotemia — abnormal high level of urea and creatinine and metabolic acidosis which develop during acute renal failure. Acute renal failure is particularly observed during severe diarrhoea coupled with delayed or absence of adequate fluid management. Another consequence, which we were discussing, involved changes in the nervous system. They can be as mild as dizziness due to less supply of oxygen, glucose and other nutrients to the brain cells or as severe as resulting in coma due to excessive accumulation of nitrogenous waste products and other toxic metabolites in the blood. It would thus be evident

that maintenance of adequate blood volume is imperative to prevent dehydration and its consequences some of which can be life threatening. A basic outline of the consequences of diarrhoea has been indicated in the Figure 14.2

## NOTES



**Figure 14.2: Consequences of diarrhoea**

By now, you must have understood the seriousness of diarrhoea and can understand that it is the highest cause of illness and death especially in children.

## Treatment and management of diarrhoea

You must have realized by now that diarrhoea should not be neglected and must receive prompt medical care to minimize the frequency of morbidity and mortalities. In light of the complications discussed above, let us now examine what should be the objectives in the management of this disease.

### Objectives

The major objectives in the management of diarrhoea include:

1. Fluid and electrolyte replacement
2. Removal of cause (especially if infection)
3. Nutrition concerns (chronic diarrhoea)

Prompt replacement of fluids and electrolytes is of most significance to prevent morbidities and mortality associated with dehydration. This is followed by removal of cause which is a medical aspect and shall not be discussed in detail here. Removal of cause may require antibiotics, gastric lavage or operative procedures to name

a few. We will now discuss the management of diarrhoea in detail.

The first step should be to determine the status of dehydration. We have already talked about the mild, moderate and severe dehydration. In mild to moderate cases fluid, electrolyte and acid base homeostasis should be preserved. Nutritional status should be restored and anti-microbial agents should be given. Associated problems like persistent vomiting, abdominal distension and convulsions should be managed.

The therapy for diarrhoea thus consists of:

**1. Determining the status of dehydration**

**2. Fluid management**

- Oral Rehydration Therapy (ORT) — home made/commercial Oral
- Rehydration Salts (ORS)
- Emergency treatment and drug management

**3. Nutritional management**

Now, let us understand these points.

1. Determining the status of dehydration has been explained to you earlier. The child in a severe dehydration state must be hospitalized,
2. Fluid management: The key to diarrhoea management is the early replacement of fluid lost in the stools through intravenous or oral route. While severe cases need administration of dextrose and electrolyte solutions intravenously; mild to moderate cases can be managed at home. The patient can be easily managed by giving any fluid at home e.g. coconut water, buttermilk, salted rice kanji, lemon sugar salt beverage or weak tea. This is commonly referred to as the Oral rehydration therapy (ORT). Let us read more about ORT.

Oral Rehydration Therapy (ORT) refers to providing fluids and/or oral rehydration salt solutions to the patient. An oral rehydration solution can easily be prepared at home by taking a teaspoon of salt, 3 tablespoon of sugar with or without lemon juice mixed in a liter of potable water. Oral Rehydration Salt formulations as suggested by WHO are freely available commercially in small packets.

Composition of Oral Rehydration Salt (WHO Standard Formulation)

1. Sodium Chloride (common salt) = 3.5 g
2. Glucose = 20 g
3. Trisodium Citrate = 2.9 g or sodium bicarbonate = 2.5 g
4. Potassium chloride = 1.5 g.
5. Dissolved in a litre of potable water.

This solution provides (g per litre of water):

1. Glucose (g/100 ml) = 2 (provides energy)
2. Sodium (mEq/L) = 90 (favours rehydration)
3. Potassium (mEq/L) = 20 (prevents acidosis)
4. Chloride (mEq/L) = 80 (favours rehydration)

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5. Bicarbonate (mEq/L) = 30 (builds base reserves)

6. Osmolality (mOsm/L) = 330 (maintains osmotic balance and favours early rehydration).

Emergency treatment and drug treatment: Severe dehydration is fatal and requires intravenous fluids and hence hospitalization. You have read about causes of diarrhoea and know that several types of protozoas, viruses and bacterias cause diarrhoea. Many toxins are produced by some varieties of bacilli, which are harmful for the mucosal lining and hence drug therapy is required.

Next, we shall discuss about the dietary management of diarrhoea.

### 3. Nutritional management

The conservative concept of treatment for diarrhoea was not in favour of feeding adequate amount of food.

However, with the identification of varied underlying causes and not so positive outcomes of the starvation therapy, it has become evident that adequate nutritional care is pertinent to ensure enhanced recovery and proper rehabilitation. Dietary management of diarrhoea has changed completely over the years and it is now advocated that the patient should be prescribed a diet most suitable for the underlying etiology of diarrhoea.

Today we know that the nutrient requirements and or the quality (consistency) of diet may not necessarily be the same for all forms of diarrhoea. While the demand for fluids and electrolytes are particularly high during an acute episode; that of all macro- and micronutrients increases during chronic diarrhoeas. In our subsequent section, we shall discuss in detail the nutrient requirements during diarrhoea.

### Dietary Recommendations during Diarrhoea

The diet should take into account the normal RDI and various adjustments made to the quantity and quality of the foods to be given. The following information will help you to understand these concepts.

- **Energy:** During the acute phase of diarrhoea, the caloric intake can be increased gradually as per the tolerance of the patient. An increment of 200-300 Kcal is a feasible target. Patients suffering from diarrhoea should never be starved as even in acute diarrhoea digestive enzymes are functional and almost 60% digestion can take place. Resting the gut can be most damaging as it can bring about structural changes in the gut membrane, which can predispose an individual to associated complications,

Recent studies indicate that children who are fed with appropriate type and amounts of food through the acute phase of diarrhoea show absorption of substantial amounts of nutrients, and are therefore at lesser risk to nutritional deficiency. These children show better weight gain, have shorter duration of diarrhoeal episode and a quicker recovery. Calories can be provided through easily digestible carbohydrates.



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Excess sugar may be avoided to prevent fermentative effect, which may aggravate the diarrhoea.

- **Protein:** Requirements are increased only in chronic diarrhoea because of associated tissue depletion. An additional 10 g of protein may be recommended above the normal requirements. Milk a source of good quality protein is restricted as it is a high residue food or if it is anticipated that diarrhoea may have developed due to relative deficiency of lactase in the gastrointestinal tract. Milk in the fermented form like curds is better tolerated, as it is easy to digest and helps in maintaining the gut health. Other cooked and diluted milk products like a light porridge; paneer etc can also be tolerated in small amounts. Apart from these, easily assimilated protein-rich foods like minced meat, egg, skimmed milk and its preparations can be given,
- **Fats:** Total amount of fat may be restricted as its digestion and absorption is compromised. In order to increase on the calorie density of the diet, emulsified fats or those, which are rich in medium chain triglycerides, may be added in restricted amounts. Fats like butter, ghee and cream are easily digested. Fried food must be avoided. Invisible form of fat i.e. fat present inherently in the food (egg yolk, whole milk, paneer, curd, flesh food etc.) is tolerated more as compared to visible form of fat.
- **Carbohydrates:** Adequate amount of carbohydrates i.e 60-65% of the total energy should be given to the patient. Easily assimilated carbohydrates i.e. principally starches should be preferred. Glucose, sugar, honey, jaggery, potato, yam, colocasia, rice, sago, semolina, refined flour, pastas can be incorporated. 10 prepare dishes such as khichdi, vegetable/pulse puree, fruit juices, soufflé, shakes, custard and kanji. The fibre content of the diet should be kept minimum and insoluble fibre should particularly be avoided. A low-residue/low fiber diet limits the amount of food waste that has to move through the large intestine. These diets may help control diarrhoea and abdominal cramping and make eating more enjoyable.

### Box 14.1 Residue in Foods

Residue is defined medically as the solid contents that have reached the lower intestine. A low residue diet is composed of foods, which are easily digested and readily absorbed, resulting in a minimum of residue in the intestinal tract. Thus, a low-residue diet contains limited amounts of undigested or only partially digested ingredients. Foods, which are high in residue, are those, which are high in roughage, or fiber. The main source of residue is fiber in foods like whole-grain breads and cereals, seeds and nuts, dried fruits, and the stalks and skins of fruits and vegetables. Milk should be consumed in moderation.

- **Fibre:** Insoluble fibre in the form of skins, seeds and structural plant materials should be strictly avoided to minimize on the irritation of the GI tract. Soluble fibre in the form of stewed fruits and vegetables like apple juice, stew, guava nectar and pomegranate juice help in binding the stool and favour good environment in the gut. Fruits like papaya and banana have an astringent property and are beneficial.

**NOTES**

**Table 14.2: Low fibre foods**

|               |                                                               |
|---------------|---------------------------------------------------------------|
| Milk products | Paneer, curds, toned milk                                     |
| Cereals       | Refined cereals: rice, white bread, noodles, maida, suji      |
| Pulses        | Dehusked pulses                                               |
| Vegetables    | Potato, bottle gourd, tomato (without skin or seeds), spinach |
| Fruits        | Papaya, banana and fruit juices                               |

**Table 14.3: Low residue foods**

|                       |                                                                                 |
|-----------------------|---------------------------------------------------------------------------------|
| Cereals               | Rice, refined cereals such as maida, suji, white bread sweet biscuit, cornflour |
| Vegetables            | Tender, well-cooked, pureed low fibre vegetables                                |
| Fruits                | Fruit juices or pureed fruits                                                   |
| Meat and its products | Chicken and fish                                                                |
| Pasta                 | plain macaroni, noodles, sphagetti etc.                                         |
| Sweets                | White sugar, brown sugar, honey, clear jelly                                    |

**Vitamins and minerals:** Loss of vitamins is related to the degree of mucosal damage in chronic diarrhoea, which in turn impair absorption and synthesis of various essential substances in the body. The vitamins of importance are B complex vitamins especially folic acid, vitamin B12 and vitamin C. Fat soluble vitamins (A, D, E and K) can be lost if fat is not digested and lost in stools. Minerals which are of importance include iron especially if there is an associated bleeding. Sodium and potassium may need to be replaced. Potassium supplementation may favour bowel motility and build up appetite,

- **Fluids:** Intake should be liberal to minimize the risk of dehydration. Remember we read about fluid management in this section before. Preference must be given to diluted drinks as concentrated ones may favour osmotic diarrhoea.

Lastly, a few simple tips which should be given to the patient.

- Boiling, steaming, baking, pressure cooking should be encouraged

- Consume small and light meals frequently instead of 3 big meals a day to replenish the lost nutrients.
- Have plenty of fluids like lemon juice, fruit juices, vegetable soups, watery dals, lassi, coconut water etc. to make up for the losses of fluids.
- Have fruits like banana and apple as they are rich in potassium which helps to maintain fluid balance.
- Try to restrict the consumption of milk and dairy products, as they are difficult to digest,
- Avoid fried foods.

### **Avoid raw vegetables like salads.**

While the above mentioned principles are applicable for patients of all age groups and gender, we shall discuss some important aspects of management among infants and young children which are the most vulnerable segments especially with respect to developing complications. It has also been observed that myths regarding breast feeding and food consumption are rampant due to ignorance. Efficient and aggressive counseling of the parents/caregivers is equally important for preventing dehydration and malnutrition which may affect the growth and development of the child in the long run especially in cases of chronic diarrhoea. Let us now move on to the management of diarrhoea in children — which is one of the leading causative factors of infant deaths in our country.

### **Dietary Management of Children Diarrhoea**

Diarrhoeas are more common in children and malnutrition often leads to the elicitation of this symptom as undernourished are more prone. Poverty, ignorance, poor sanitation are often the underlying risk factors. The magnitude of the incidence of diarrhoea in India is majorly observed in children. It has been estimated that almost 250 million episodes of diarrhoea annually are observed in children below 5 years and nearly 1.5 million children die of diarrhoea annually. Thus, managing diarrhoea in children is of great concern. The guidelines for the same are as follows:

1. The first objective is to rehydrate the child. Thus using ORS would be the ideal modality. The WHO-ORS standard preparation is useful but recent studies have suggested that the osmolarity of the solution should be reduced from 311mmol/litre to 200-250 mmol/litre by reducing the concentration of glucose. Sodium has a beneficial effect on the stool output and duration especially in non-cholera diarrhoea. This can be achieved by diluting the ORS salt in 1.5 litres instead of the usual 1 litre for infants under age 2 months.
2. Breast feeding should be continued in young infants during diarrhoea. This helps in decreasing the number and volume of stools and the duration of diarrhoea. Starving the child during diarrhoea may deteriorate the nutritional status.
3. Children 4-6 months of age or older should receive energy rich mixture of soft weaning foods in addition to breast milk. The energy rich mixture can be a mix

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of cereal, pulse, roots, green leafy vegetables, and fats like ghee, butter that are easy to digest. The caloric density of the feeds can be increased by using amylase rich flour (ARF), i.e the flour obtained from germinated grain which is rich in amylase and can be prepared as a soft and thin porridge without taking away its nutritional value. Fermented foods like dhokla, bread, idli, dosa may be included in the diet.

4. Foods to be avoided include: spicy and oily foods, confectionary, mithai and chocolates, as well as, uncooked fruits and vegetables.
5. Supplementation with B-complex vitamins especially folic acid, vitamin B 12 and minerals like zinc help in normalizing the intestine.
6. The criteria for monitoring the state of hydration and nutritional status are: good urine output, appearance of the eyes, skin, buccal mucosa and weight gain.
7. Consult the doctor, if required.

REMEMBER THE GOLDEN RULE: FEED IN DIARRHOEA, DON'T STARVE THE CHILD.

THERE ARE MORE LIVES LOST DUE TO STARVATION THAN FEEDING.

- INFANT: CONTINUE BREAST FEEDING
- OLDER CHILDREN; MAKE NECESSARY MODIFICATIONS IN THE NORMAL DAILY DIET. GIVE FREQUENT LIQUIDS OR/AND LOW RESIDUE SOFT DIGESTIVE FEEDINGS. GIVE BLAND AND LOW FIBRE DIETS.

### 14.3.2 Constipation

Constipation is irregular, infrequent or difficult passage of faeces. It is the most common physiological disorder of the alimentary tract. It is characterized by incomplete evacuation of hard, dried stools. It occurs commonly in children, adolescents, adults on low fibre diets, patients confined to bed, in invalids and in elderly persons. It is a condition in which:

- fewer than 3 stools per week are passed while a person is eating a high residue diet,
- more than 3 days go by without the passage of a stool, and
- stools passed in one day amounting to less than 35 grams.

There are three main types of constipation. These are:

1. Atonic constipation: This type is most common, often it is called the "lazy bowel". There is a loss of muscle tone causing weak peristalsis, the causes are:
  - a) lack of fluids, roughage and potassium
  - b) vitamin B Complex deficiency

- c) irregular defeaction habit and poor personel hygiene.
- d) excessive purgation or use of enema.

**NOTES**

**sedentary lifestyle or lack of exercise**

1. Spastic constipation: It results from excessive tone of the colonic muscle.
2. Obstructive constipation:It occurs usually due to obstruction in the colon, cancer, or any other obstruction due to inflammation or narrowing of the lumen.

Let us see what are the causes and symptoms of constipation.

**Etiology**

The most common causes of constipation are poor elimination habits, a lack of fibre in the diet, insufficient fluid intake, lack of exercise and a loss of lone in the intestinal musculature. Apart from these, chronic overuse of laxatives, nervous strain and wony are also some common causes. The causes can be classified under two heads — systemic and gastrointestinal —as highlighted in Table 14.4.

| <b>Systemic</b>                                              | <b>Gastrointestinal</b>      |
|--------------------------------------------------------------|------------------------------|
| Side effects of medications                                  | Celiac disease               |
| Metabolic or endocrinal abnormalities such as hypothyroidism | Duodenal ulcer               |
| Lack of exercise                                             | Gastric cancer               |
| Ignoring the urge to defecate                                | Cystic fibrosis              |
| Vascular disease of the large bowel                          | Diseases of the large bowel  |
| Diet low in fibre                                            | Irritable bowel syndrome     |
| Pregnancy                                                    | Anal fissures & haemorrhoids |
|                                                              | Laxative abuse               |

**Table 14.4: General causes of constipation**

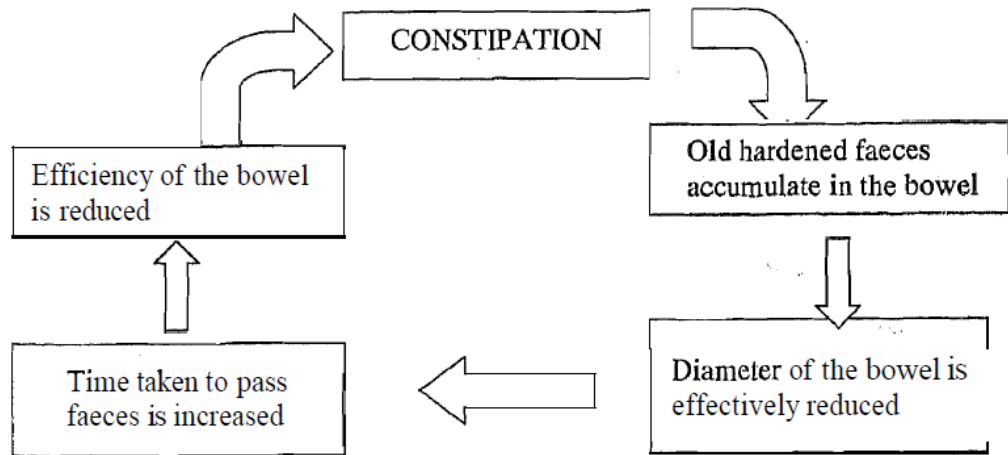
**Symptoms**

Have you ever suffered from constipation? Do you recall the symptoms associated with the problem? Yes, the symptoms were specific to having a bloated stomach, stomach pains/cramps, inability to evacuate, a feeling of fullness in the lower abdomen, lethargy, irritability, a sensation of dullness or even moderate pain in the head. These are the symptoms of constipation.

Let us now move on to the major complications involved in this disorder. If

constipation is suffered frequently, the problem worsens due to a vicious cycle of events.

**NOTES**



**Figure 14.3: Complications in constipation**

**Table 14.5: Complications associated with constipation**

|                                                      |                                                           |
|------------------------------------------------------|-----------------------------------------------------------|
| Haemorrhoids (swollen blood vessels around the anus) | Diarrhoea                                                 |
| Anal fissure (a tear in the anal region)             | Faecal incontinence (inability to control bowel movement) |
| Rectal bleeding (protruding rectum through the anus) | Rectal prolapse                                           |
| Rectal hernia                                        | Faecal impaction (hard stools in the bowel)               |
| Uterine hernia                                       | Uterine prolapse (downward displacement)                  |

**Management of Constipation**

You must have realized by now that proper dietary and lifestyle management can help in maintaining the normal bowel movements to a great extent. Medical interventions are required only when constipation arises because of some structural functional change in the gastrointestinal tract. In our subsequent discussions, we will deal with the dietary management of constipation. Let us first identify the objectives of the patient care process.

**Dietary and Life Style management Goals**

The dietary and life style management goals include:

- To develop regularity of habits in evacuation
- To follow a regular meal pattern
- Consume a high fibre diet
- Take adequate amounts of fluids
- Increase physical activity

Based on these objectives, the dietary management of constipation is highlighted next. Dietary Management of Constipation

Management of constipation lies in developing regularity of habit through a bowel-training programme and by establishing good healthy habits such as regular meals and elimination timings, adequate fibre and fluid intake, and sufficient exercise.

The mainstay of the treatment of constipation is however dietary in nature with a lot of emphasis on dietary fibre and fluid intake. So let us get to know about dietary fibre — its sources and potential benefits.

Dietary fibre is defined as plant polysaccharide resistant to hydrolysis by the digestive enzymes in the human intestinal tract. It includes:

- Structural polysaccharides (insoluble fibre) of the plant cell wall such as cellulose, hemicelluloses, non-carbohydrate material, lignin etc.
- Non-structural polysaccharides (soluble fibre) such as pectins, gums and mucilages.

### **What are the sources of dietary fibre in our diet?**

The sources of dietary fibre include whole grain cereals, legumes, whole pulses, leafy vegetables, vegetables like peas, beans, ladies finger, fruits like guava, apple, citrus fruits, nuts, oilseeds like flaxseeds, methi seeds etc. Remember we read about the sources of soluble and insoluble fibre in Unit 11 earlier. You may wish to go back and recapitulate.

Do you know what the recommendations for fibre are? Well, the crude fibre intake should be 14 g/1000 Kcal. For adult woman 25 g/day and for adult man 38 g/day is desirable.

Increase in fibre intake may lead to symptoms such as flatulence and abdominal distention. This can be relieved through use of inputs like sprouting, fermentation, proper distribution of high fibre foods through out the day and adequate fluids. Bran and powdered supplements may be of help in individuals who do not eat sufficient amounts of fibrous foods.

### **What about the fluid and other nutrient intake during constipation?**

The fluid intake should be at least 2 litres daily. This includes fluid as foods and beverage besides water. The intake of lemon juice, citrus fruit juices, coconut water, vegetable soups, watery dal, lassi and watermelon juice may have an added benefit of adding vital nutrients like potassium which improve the muscle tone.

As for the other nutrients i.e. calories, proteins, carbohydrates and fat the requirements would be the same as the RDA for a particular age, sex, occupation of the individual, weight status etc.

The nutritional management should aim at:

- developing regularity of habits of evacuation
- following a regular and balanced meal pattern
- consuming a high fibre and adequate fluid diet, and
- increase in physical activity and exercise

The requirements of various nutrients are not altered in constipation. It is essentially a normal balanced diet (normal RDA's) with modification in fibre and fluid intake. The intake of fibre should be increased. High fibre foods should be given freely. Some of the foods which can be given freely (avoided have been mentioned in Table 14.6 and 14.7 respectively).

|            |                                                                                     |
|------------|-------------------------------------------------------------------------------------|
| Cereals    | Whole-wheat, maize, millets.                                                        |
| Pulses     | Whole-pulses such as rajma, chole, whole green gram etc.                            |
| Vegetables | Green leafy vegetables, knoll khol, lotus stem, peas, beans                         |
| Fruits     | Guavas, pomegranate, apples with skin, chickoos, cherries, pear, peaches and plums. |

**Table 14.6: Foods to be given freely in constipation**

|                                                                                                       |
|-------------------------------------------------------------------------------------------------------|
| Refined foods: pasta, refined cereals like maida, suji, baked products, pizza, patties, biscuits etc. |
| Deep fried foods                                                                                      |
| Pureed fruits and vegetables, banana, mango etc.                                                      |

**Table 14.7: Foods to be avoided/ restricted during constipation**

When changes in diet and activity patterns do not improve constipation, further



evaluation is warranted and the need for drugs prescribed by the physician may be necessary.

### 14.3.3 Oesophagitis

We already know that oesophagus is a muscular tube 25 cm in length and basically helps in transporting the food from the mouth to stomach. As the bolus of food is moved voluntarily from the mouth to the pharynx, the upper oesophageal sphincter relaxes, the food enters oesophagus and subsequently the lower oesophageal sphincter (LES) relaxes to receive the food bolus. With the help of peristaltic waves, the bolus of food is moved into the stomach. Refer to Unit 6, section 6.6 in the Applied Physiology Course for more details on this aspect.

Oesophagitis occurs in the lower oesophagus as a result of the irritating effect of acidic gastric reflux on the oesophageal mucosa. It can be an acute/chronic inflammation of the oesophageal wall. It is associated with the common symptom of heartburn (burning epigastric substernal pain). Other symptoms are regurgitation and dysphagia (difficulty in swallowing). Difficulty in swallowing occurs due to pain associated with inflammation of the tissues of the oesophagus. Regurgitation of the acidic gastric contents can be a cause or result of oesophagitis. Let us learn about the two types of oesophagitis conditions:

- 1. Acute Oesophagitis** — It is characterized by substernal pain on swallowing. It is due to prolonged intubation, extensive burns, excessive vomiting, ingestion of a toxin/chemical or due to diphtheria.
- 2. Chronic or Reflux Oesophagitis** — It is characterized by intermittent heartburn, pain on taking hot or cold foods, spicy or acid foods. This is a result of recurrent gastroesophageal reflux due to hiatus hernia, reduced lower oesophageal sphincter (LES) pressure, increased abdominal pressure (obesity, pregnancy, hiatus) increased abdominal adiposity and recurrent vomiting.

The severity of the oesophagitis resulting from oesophageal reflux is determined by the content of gastric reflux, mucosal resistance, clearing rate of oesophagus and rate of gastric emptying. Content of gastric reflux may include partly digested food, pepsin, acid and possibly bile and at times pancreatic enzymes. It is probably this combination, which causes mucosal damage. Symptoms develop when reflux becomes frequent and mucosa of oesophagus becomes sensitive to the reflux contents.

Competency of LES (Lower Oesophageal Sphincter) is important. The pressure of this sphincter is controlled by many factors, one of which is hormonal. LES decreases during pregnancy, in women on oral contraceptive and late stage of menstrual cycle. Other factors may include overweight, hiatus hernia, tight clothing, H. pylori bacteria, and excessive use of drugs like aspirin and anti-inflammatory drugs.

Certain foods like fatty meals, chocolate, coffee, alcohol, spicy food, citrus juices lower the sphincter pressure (also hormone mediated).

The causes include tissue erosion by hydrochloric acid (HCl) and pepsin, with symptoms of substernal burning, cramping, pressure sensation or severe pain.

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These symptoms are related to:

- anincompetent gastroesophageal sphincter,
- frequency and duration of the acid reflux, and
- inability of the oesophagus to produce normal peristaltic waves to prevent prolonged contact of the oesophageal mucosa with the acid pepsin.

The patients usually present the following symptoms:

### Symptoms

The symptoms of oesophagitis include heartburn or pyrosis, iron-deficiency anaemia due to chronic tissue bleeding, aspiration, which may cause cough, dyspnea or pneumonia. The complications involved in the disorder are stenosis and oesophageal ulcer. Significant gastritis in the herniated portion of the stomach may cause occult (hidden or minute amounts) bleeding and anaemia.

Before we move on to the dietary factors, let us have a look at the following case. Meeta is a 49-year-old female working in a MNC. From past few days, she had been complaining of persistent abdominal pain, diarrhoea and weight loss. Her medical examination revealed the acute condition of oesophagitis. What dietary advice would you give to her so that her symptoms are relieved and she gains back her normal body weight? Let us read and find out.

### Nutrition Management Goals

The objectives of nutritional care include the following:

1. Prevention of irritation of the inflamed oesophageal mucosa (in the acute phase).
2. Prevention of oesophageal reflux
3. To decrease the acidity of the gastric juice.

It is evident from these objectives that there is no significant change in the nutrient requirements of the patient. We however need to make several qualitative changes in the diet and feeding pattern. Let us take an overview on these aspects now.

### Dietary Management

Providing adequate nutrition support may require emphasis of different aspects during acute and chronic oesophagitis.

In acute phase, the dietary factors to be kept in mind are:

- Liquid diet; small and frequent meals.
- Less abrasion to the oesophagus thus avoiding orange juice and other citrus and
- tomato products because of their acidity.
- Spices like chili powder, black pepper to be avoided.

While in chronic phase, following factors must be considered as well:

Efforts must be taken to increase LES pressure. Meals/foods high in protein increase sphincter pressure and reduce the likelihood of reflux and heartburn.

Avoiding foods that are known to cause heartburn and decrease LES pressure like

chocolate, alcohol, caffeine containing beverages, coffee, cold drink, fatty foods and increased fat intake.

Timing of the meals is very important especially before the afternoon nap and evening. The patients should consume nothing except water 3 hours before lying down. This in turn ensures an almost empty stomach with less likelihood of reflux on lying down.

Avoid lying down, bending or straining immediately after eating.

Reduce weight so that abdominal pressure is decreased.

### **Avoid tight fitting clothes.**

- Avoid smoking as nicotine decreases LES pressure. Other effects of smoking on the GI tract includes:
  1. A decrease in pyloric sphincter pressure which may predispose an individual to duodenal ulcer.
  2. An alteration in the nature of gastric contents— inhibition of bicarbonate secretion, decreased duodenal pH, acid secretory response to gastrin is increased.
  3. Impairs the ability of drugs to lower overnight acid secretion (cimetidine).
  4. Impairs healing of wounds — favours ulceration of wounds leading to haemorrhage necessitating surgery.

Let us now understand one of the most commonly observed conditions associated with chronic oesophagitis i.e. gastroesophageal reflux disease (GERD).

### **14.3.4 Gastro Oesophageal Reflux Disease (GERD)**

GERD refers to the regurgitation of acidic stomach contents into the oesophagus. It results in a spectrum of clinical manifestations, the most common being heartburn and acid regurgitation. The pathologic reflux can result in a wide variety of clinical presentations. GERD is typically chronic, and while it is generally non-progressive, some cases are associated with the development of complications of increasing severity and significance. Reflux occurs when the pressure inside the stomach is higher than that maintained by the muscles found where the stomach and oesophagus meet. This can happen for a number of reasons, outlined in the etiology below.

#### **Etiology**

GERD may develop due to any of the following reasons:

- decreased muscle tone or abnormal relaxation of the LES,
- reduced stomach motility, allowing food to remain too long in the stomach (gastric stasis), where hiatus hernia.

Episodes of reflux are triggered or worsened by a variety of factors. Symptoms may be aggravated by chocolate, caffeine, alcohol and spicy foods, which stimulate acid secretion or by fatty foods, which delay stomach emptying. Gravity works against normal digestion and can promote reflux when the patient bends over or

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lies horizontally.

In addition, pregnancy or constipation may worsen GERD by increasing intra-abdominal pressure. Secondary causes of GERD include reflux caused by acid hypersecretory states, connective tissue disorders (scleroderma), gastric outlet obstruction as caused by ulceration and stricture, and delayed gastric emptying due to conditions such as gastric stasis, neuromuscular disease, idiopathic gastroparesis, pyloric dysfunction, duodenal dysmotility, or duodenogastroesophageal bile reflux

Most patients with complicated GERD have a hiatal hernia, which, by displacing the LES segment of the distal oesophagus, both reduces LES pressure and impairs acid clearance. We will learn about hiatus hernia later in this section.

Let us move on to the study the signs and symptoms of GERD.

### Symptoms

Most commonly, people with GERD complain of heartburn, a painful or uncomfortable feeling in the chest, which may radiate to the back. Often the patient will recognize a pattern of symptoms related to timing, food or body position. Heartburn may be mistaken for angina pectoris, which is pain in the chest related to heart disease; a careful history and physical examination should distinguish between the two (although it is certainly possible for a patient to have both). Symptoms are most often present after meals, especially after eating certain foods, and at night, while lying in bed, and may be relieved temporarily by antacids or milk.

In addition to heartburn, difficult or painful swallowing, a sour taste in the mouth, and frequent belching are common. Less typical features include chronic cough, hoarseness, sore throat and a sensation of fullness. Important extra-oesophageal symptoms include laryngitis, pharyngitis, chronic sinusitis, dental erosions, asthma, and chronic cough. These symptoms can occur as a result of gastric acid reflux into the throat and vocal cords or down into the lungs. Acid reflux due to GERD can also erode teeth.

Some of the common complications which have been observed among patients with GERD are being discussed below.

### Complications

Apart from the symptoms, which you have just read above, there are a few extra-oesophageal manifestations of GERD. These include nausea and vomiting and erosive changes in dental enamel. Occasionally, patients present with occult upper gastrointestinal bleeding or with iron deficiency anaemia.

The main complications include:

- oesophagitis (inflammation of the oesophagus),
- bleeding, oesophageal erosions and ulcerations (frequent reflux of acid into the oesophagus), stricture (narrowing) of the intestines.

- Barrett's oesophagus (replacement of ulcerated squamous epithelium by a metaplastic intestinal-type epithelium), and
- adenocarcinoma of the oesophagus. Reflux-induced injury to oesophageal tissue can result in tissue destruction and the development of oesophageal erosions or ulcerations.

Several long/short term complications may arise due to GERD which may in turn increase the frequency or severity of this disease. One of the complications of clinical relevance is hiatus hernia. Box 14.2 presents a review on hernia.

It must be clear from our discussions on the etiology, symptoms and complications on GERD that the nutrient requirements during this condition do not vary much from that of the RDI for a normal individual. However, as in case of oesophagitis, dietary management particularly with respect to meal pattern is of greater significance. Let us brief ourselves on some important aspects.

### **Nutritional Management and other Factors**

As mentioned above the nutrient requirements remain the same as per the RDI for most patients. It would be important to note here that if GERD has developed due to obesity, it is essential to prescribe a weight reduction diet for the patient. Nutrient requirements may also change during certain complications such as bleeding thereby necessitating an increased intake of dietary proteins, iron, B-group vitamins and vitamin C. Dietary pattern on the other hand needs to be altered for all patients.

Meals are the major aggravating factor of GERD symptoms, since they stimulate the production of gastric acid available for reflux into the oesophagus. Meals eaten within 2 to 3 hours of bedtime (which increase acid availability at night time), or with alcohol, can predispose patients to nocturnal reflux. Dietary fat in the duodenum also appears to be a strong reflux trigger, in part impairing gastric emptying. Also, it is inappropriate to advise to reduce the fat content of their meals, at least with regard to GERD symptom relief.

Specific foods that have been identified as potentially aggravating factors in certain patients include raw onions, chocolate, caffeine, peppermint, citrus juices, alcoholic beverages, tomato products and spicy foods. Peppermint and chocolate are thought to lower LES tone, facilitating reflux. Citrus juice, tomato juice and probably pepper can irritate damaged oesophageal mucosa. Cola drinks, coffee, tea and beer can have an acidic pH, lowering LES to precipitate symptoms. Potential oesophageal irritants should be restricted.

Obesity is thought to be another potential predisposing factor to gastroesophageal reflux or GERD. Maintenance of ideal weight for age may help in reducing the symptoms.

Pregnancy is the most common condition predisposing to GERD and is generally associated with symptomatic GERD (typically heartburn) rather than oesophagitis. In most cases, symptoms occur for the first time during the pregnancy and subside soon after delivery. Recurrence is also a possibility with subsequent pregnancies.

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A hiatus hernia is frequently found among patients with GERD. You will read about it next. Viewed as part of a GERD continuum, a hiatal hernia is another factor disrupting the integrity of the gastroesophageal sphincter, resulting in increased oesophageal acid exposure. Smoking has been found to be related to an increased number reflux events in association with deep inspiration and coughing.

### Box 14.2 Hiatus Hernia

Several long/short term complications may arise due to GERD which may in turn increase the frequency or severity of this disease. One of the complications of clinical relevance is Hiatus Hernia. What is Hiatus Hernia and why does it occur? Well, hiatus hernia refers to out pouching of a part of the stomach through several openings of the diaphragm; the most common one being the hiatus. It occurs when a portion of the upper part of the stomach at this entry point of the oesophagus protrudes through the hiatus alongside the lower portion of the oesophagus. This is a major complication of GERD. Depending on the extent of herniation and placement of stomach, it could be of two types:

1. Para Oesophageal Hernia (oesophagus is in normal portion)
2. Oesophageal Hiatus Hernia (elevated oesophagus)

Food may be easily captured in the herniated area of the stomach and mixed with acid and pepsin. Then it is regurgitated back up to the lower portion of the oesophagus. Gastritis can occur in the herniated portion of the stomach and may cause occult bleeding (blood loss small to be detected under microscope) anaemia. The problem is found in stocky built overweight middle-aged persons, pregnant women and sometimes in persons having chronic constipation that weakens the hiatus.

### Symptoms

In most patients, no symptoms are seen. When symptoms do occur, they are:

1. discomfort after heavy meal,
2. difficulty in breathing while lying down and bending over,
3. sensation of heartburn and food sticking,
4. chronic reflux of acid into the oesophagus, causing injury and bleeding.
5. anaemia, or low RBC count can result, and
6. belching and hiccups

The dietary considerations for this condition are the same as in oesophagitis. The patient should be recommended to:

- eat smaller meals
- reduce weight
- avoid lying down immediately after meals
- use ant acids to relieve burning sensation
- large or sliding hernia may require surgical treatment

In this section, we learnt about oesophagitis and gastroesophageal reflux disease. We also read about hiatus hernia, which is a common complication and, at times a cause of GERD. Given herewith are a few questions as part of the check your progress exercise which will help you in quick recapitulation.

### **14.3.5 Dyspepsia**

Dyspepsia is the most frequent disorder which we all experience some time or the other. Dyspepsia is the term given to a group of gastrointestinal symptoms associated with the taking of food e.g., nausea, heartburn, epigastric pain, discomfort and distension. It is commonly known as indigestion.

Dyspepsia may be:

1. Functional
2. Organic

In functional there is no structural change in any part of the alimentary canal. The symptoms may be psychological and emotional in origin or due to intolerance of a particular food or faulty food habits. A disease or a disorder of the digestive tract or a chronic disease of the kidney or even of the heart generally causes the second type i.e. organic dyspepsia. Let us read about some of the common symptoms associated with dyspepsia.

### **Symptoms**

The symptoms are heartburn, upper abdominal discomfort (often food-related) indigestion, bloating, fullness, nausea and anorexia. Such symptoms can also be seen in gastroesophageal reflux, peptic ulcer disease and cancer of the stomach or pancreas and gallstones disease. With other organs associated, many other symptoms can be noted besides a stomach upset. These are bloating (fullness of stomach), burping, epigastric pain, gastrointestinal bleeding etc.

However, why does dyspepsia occur? Let us find out.

### **Etology**

The main etiological factor of dyspepsia is the failure of proper digestion and absorption of food in the alimentary tract and the consequences thereof. Often, dyspepsia is caused by a stomach ulcer or acid reflux disease. If an individual has acid reflux disease, stomach acid backs up into the oesophagus. This causes pain in the chest. Some medicines like anti-inflammatory medicines can cause dyspepsia. Rarely, dyspepsia is caused by stomach cancer, so you should take this problem seriously. Sometimes no cause of dyspepsia can be found.

### **Complications**

Some common complications of dyspepsia are listed below:

Weight loss: Since eating most often provokes the symptoms, patients restrict their food. Restriction of food and skipping of meals often causes weight loss. Specific foods are also associated with symptoms e.g. fats, vegetables, milk restriction

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which can result in calcium and energy deficiency.

- Altered social life: Most commonly, functional diseases interfere with the patients' comfort and daily activities leading to alteration in social life.

We will now move over to the dietary management of dyspepsia.

### Dietary Management

Keeping in mind the etiology, symptoms and complications of dyspepsia it must be clear that treatment and management of this disorder does not require any changes in the nutrient intake. All we need to take care is avoidance of a high fat diet. Modifications in the meal pattern and elimination of certain foods may however prove to be beneficial in most of the cases.

Usually, the bland diets are prescribed in such conditions. For excessive belching reduce the foods that are gaseous (whole pulses like rajmah, channa). Soaking/sprouting whole pulses may help in making the fibre softer and hence reduce the symptoms of belching. Vegetables like radish, turnip, cauliflower, broccoli, beans and peas should be avoided.

Intolerance to lactose (the sugar in milk) often is blamed for dyspepsia. Since dyspepsia and lactose intolerance both are common, the two conditions may coexist. In this situation, restricting lactose will improve the symptoms of lactose intolerance, but will not affect the symptoms of dyspepsia. If lactose is determined to be responsible for some or all of the symptoms, elimination of lactose-containing foods is appropriate.

The lifestyle modifications in terms of work schedules, eating behaviour like conquering meals when extremely tired, quitting alcohol and smoking would help to alleviate the symptoms of dyspepsia. The patient may be counseled with the following handy tips:

- If you smoke, stop smoking.
- If some foods bother your stomach, try to avoid eating them.
- Gulping of food should be avoided, slow eating should be encouraged;
- Eat food at least 2 hours before lying down.
- Try to reduce stress.

If you have acid reflux, don't eat right before bedtime. Raising the head of your bed could help.

Follow the advice of your doctor for taking antacids and anti-inflammatory drugs.

We will discuss another common disorder of the gastrointestinal tract that is commonly referred to as acidity by the general masses but actually means hyperacidity or gastritis. Gastritis may be as mild to get managed by the help of an acid neutralizer to as severe to result in the development of ulcers. Let us learn about this disorder in detail.



### 14.3.6 Gastritis

Gastritis is an inflammatory lesion of the gastric mucosa, (the inner lining of the stomach).

The problem is seen in two forms:

- a) Acute gastritis
- b) Chronic gastritis

Let us review them.

#### **A . Acute Gastritis**

It is a sudden inflammation of the lining of the stomach. It occurs mainly due to overeating, overuse of alcohol, tobacco, chronic or excessive dose of aspirin, anti-inflammatory drugs, increased production of bile acids, trauma, surgery, shock, fever, jaundice, renal failure, burns, radiation therapy, H. Pylori infection etc. The symptoms of the disorder are nausea, vomiting, malaise, anorexia, headache, haemorrhage and pain in the upper abdomen, dark stools, hiccups, tachycardia, rapid pulse and low blood pressure. Its complication involves severe blood loss, with blood suddenly flowing into the region known as hyperemia, inflammation and even exudation. In severe cases there may be erosion of localized areas and even bleeding. The major symptoms are mentioned below.

#### **Symptoms**

Anorexia, epigastric discomfort, heartburn and severe vomiting, pain in upper abdomen, headache and even bleeding.

#### **Etiology**

Some most frequently associated risk factors for gastritis include:

- Faulty dietary habits like overeating and taking highly seasoned foods.
- Bacterial toxins (Salmonella, Staphylococcus), metabolic toxins (uremia) and Helicobacter pylori infection.
- Excessive use of alcohol, drugs (aspirin, anti-inflammatory).
- Exposure of gastric mucosa to irradiation.
- Increased production of bile salts.
- Burns and renal failure.

We will now learn about another form of gastritis i.e., chronic gastritis.

#### **B. Chronic Gastritis**

It precedes development of organic gastric lesion, or tissue damage. Recurrent inflammation leads to changes in enzyme activity of gastric mucosal cells. Complete atrophy results in lack of absorption of vitamin B<sub>12</sub> (Pernicious anaemia). The chronic gastritis is clinically more important than the acute gastritis.

Gastroscopic observation shows 3 types of chronic gastritis:

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1. Superficial gastritis: gastric mucosa is red, oedematous, covered with adherent mucous, mucous haemorrhage and small erosions are frequently seen.
2. Atrophic gastritis: the mucous lining becomes thinner, gray or grayish green haemorrhage mucosa irregularly distributed.
3. Hypertrophicgastritis: presents a dull spongy nodular appearance of the rnucosa, the edges are irregular thickened with nodular haemorrhages or superficial haemorrhages.

### Symptoms

These include anorexia, chronic fatigue, and feeling of fullness, belching, vague epigastric pain, nausea and vomiting and passage of black tarry stools.

### Etiology

They are same as acute. Generally acute gastritis if well treated gets healed in 3-4 days, however if untreated can progress to chronic gastritis.

We will now discuss about the dietary management of gastritis.

### Dietary Management

Prompt medical care is successful in the management of an acute attack of gastritis only if it is accompanied by efficient and judicious nutritional care. During an acute attack, meeting the nutrient requirements is not of prime importance. Depending on the seriousness of the patient the food maybe with held for 24-48 hours. Fluids maybe given intravenously if needed. Liquid foods are given as per patients tolerance level. The amount of food and number of feedings are adjusted according to the patients tolerance, until a full regular diet is achieved. Always follow a progressive diet i.e. liquid to semi solid to solid as when the symptoms improve. The diet should contain less fat and must be bland. Many nutritional deficiencies occur in this disorder especially during chronic gastritis e.g. vitamin B<sub>12</sub>, Iron, and other vitamin deficiencies.

The nutritional treatment must follow general principles of soft diet. The diet should be adequate in,calories and nutrients. There must be small feedings at regular intervals. Avoid gastric initantsand highly seasoned foods (onion garlic, chilli, caffeine, cola and alcohol). Excess water or other liquids with meals may cause distention.

The dietary guidelines are enumerated herewith:

- **Energy:** Give adequate calories through frequent feedings or else proteins would be utilized for energy of repair work.
- **Proteins:** Give adequate proteins (1g/kg body weight) through skimmed milk, egg, steamed fish, chicken, minced meat etc.
- **Carbohydrates :** Simple easy to digest carbohydrates should be included in soft well cookedform. Thus, semolina, rice, maida, sago, arrowroot etc. may be included whereas whole cereals and millets should particularly be avoided if gastritis has caused damage to the mucosa.

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- **Fiber** : Eating a diet high in fibre reduces the risk of developing the ulcers and also speeds up the healing process. However, care must be taken that fibre rich foods (soluble fibre) are always included in a soft cooked form. Raw foods, seeds etc should be completely avoided in the diet. While soluble fibre is safer for the patient as compared to insoluble fibre (husk/bran of cereals and pulses, peels of fruits and vegetables).
- **Vitamin B<sub>12</sub>** : Supplementation with vitamin B<sub>12</sub> helps to treat pernicious anaemia and H. pylori infection. Its sources include fish, dairy products, organ meats, eggs, beef and pork.
- **Vitamin A** : A combination of vitamin A (found in many green and orange coloured fruits and vegetables) and antacids is helpful in healing ulcers.
- **Vitamin C** : A high dose of vitamin C treatment is effective in treating H. pylori infection.

It has been observed that diets high in soluble fibre, carotenoids, and antioxidants reduce the risk of developing gastritis.

A brief list of foods to be avoided is given below in Table 14.8.

|                                    |                          |
|------------------------------------|--------------------------|
| Coffee – with and without caffeine | Alcohol                  |
| Tobacco/Smoking                    | Carbonated beverages     |
| Fruit juices with citric acid      | High fat foods           |
| Mint and vinegar                   | MILK                     |
| Spices                             | Pepper, onion and garlic |

**Table 14.8: Foods/substances to be avoided**

### 14.3.7 Diverticular Disease

A common disorder of the large bowel, diverticulosis, is an early stage of the disease. It can be identified in 15% of the people over the age of 50 years. It is a condition of abnormal pouches in portions the colon (small mucosal sacs called diverticula protrude through the intestinal wall). It has a history of constipation, which results in an increased intracolonic pressure, straining to pass hard faeces and rupturing of the bowel wall at weak points to form small pockets, which are called diverticulitis. Inflammation and bacterial overgrowth in diverticuli may result in diarrhoea. When the pouches become inflamed (often as a result of bacterial infection), symptoms such as cramping pains, fever, and nausea can result. Such an infection, called as diverticulitis, is potentially life threatening and requires immediate medical intervention due to complications like ulceration or perforation or profuse bleeding.

Let us now discuss about the symptoms of diverticulosis.

## Symptoms

Depending on the site of diverticula the symptoms may appear. It occurs most often in sigmoid colon and frequency increases with age. It is more common in the western world where the fibre intake is significantly lower. Often diverticula (pouches) cause no symptoms, except the person may experience some irregularities in bowel habits. When there is an active infection, there may also be fever, chills, nausea, and vomiting, changes in bowel habits, rectal bleeding and constipation.

We will now understand the causes of this disease.

## Etiology

The causes of diverticular disease are not certain, but several factors may contribute to changes in the wall of the colon. These include:

- aging,
- 4 the movement of waste through the colon,
- o changes in intestinal pressure,
- a low-fiber diet, and
- anatomic defects.

The many complications of the disease include the following conditions:

- A perforation (hole) in the intestine leading to peritonitis, sepsis, and even shock
- An abscess (pocket of pus)
- Fistulas, which may also lead to sepsis
- Blacked intestine
- Rectal bleeding

It must be clear to you that diverticulosis occurs to a great extent due to faulty dietary habits and that several complications listed above may necessitate a surgical procedure. It is thus important to provide good nutritional care to the patient. We will mwhighlight some important aspects of the dietary management.

## Dietary Management

Most of the diseases which we have discussed so far do not require any nujorclmges in the nutrient intake. The patients generally benefit from a high fibre diet. Hare, a greater amount of bulk or fiber in the diet will promote soft, bulky stools dut pass more swiftly and are defecated more easily. Also, the intra luminal pressure generated in the lower colon would beless and the fewer diverticuli will be formed. An meased intake of fluid must be emphasized. A decreased intake of fat in the diel may be suggested.

For mild symptoms, a clear liquid diet is recommended. More serious cases may require hospitalization, intravenous feeding to rest the bowel, and intravenousantlbiotics. Eating a high-fiber diet and taking psyllium supplement

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are beneficial. Maintain nutrition. Some important aspects, which need to be taken care of, include:

1. **High-fiber based studies** suggest that eating a high-fiber diet helps prevent diverticular disease and other gastrointestinal disorders. A review of such studies reports that vegetarians are less likely to have diverticular disease, most likely because they tend to eat more fiber. Lower intake of protein such as red meat and milk products can reduce the risk of diverticulosis. Fibre supplements could improve constipation. One can give 1-2 tablespoons of wheat bran daily or isabgol. Also, remember that an amount of 15-20 g/day of crude fibre and 30-60 g/day of dietary fibre should be given.
2. **Glutamine:** While specific nutrients that may have an impact on diverticular disease have not been studied as thoroughly as the high-fiber diet, glutamine supplements are beneficial as they strengthen and protect the colon wall.
3. **Omega-3 Essential Fatty Acids:** Omega-3 essential fatty acids found in flax and cold-water fish help fight inflammation. For a condition such as diverticulitis, it may be wise to eat a diet rich in omega-3 fatty acids. This type of diet may also help prevent colon cancer.
4. **Lifestyle modifications:** Obesity may be associated with increased severity of diverticular disease. Hence, maintaining ideal weight for age is beneficial from health aspects. Physical activity like jogging and running are beneficial. Exercise also reduces the symptoms of this disorder.

The prevention strategy for the disease involves the following:

- Eat a high-fiber diet (more than 15 g/day of crude fiber). This helps the stools to move smoothly through the intestines maintaining proper pressure in the colon. Fibre should be included when the inflammation subsides.
- If diverticula are present, avoid foods such as seeds that may block the opening of a diverticulum and lead to inflammation and infection.
- Exercise regularly to decrease the occurrence of symptoms.

The management goals discussed above are basic to prevention of diverticulitis. However, diet therapy during diverticulitis may be limited to clear liquids progressing to full liquids to normal diet. Increase the fibre only when the inflammation subsides. So please note during a bout of diverticulitis the patient should be given a low fibre soft bland diet. Severe diverticulitis is treated by surgical methods. In such situations patient should be prepared for an elective surgery. You may recapitulate about dietary management pre and post operatively by reading the section on surgery in Unit 5 of this manual.

### 14.3.8 Peptic Ulcer

Peptic ulcers are one of the more serious forms of gastrointestinal disorders of the gastrointestinal tract particularly the stomach. The prevalence of ulcers has been increasing over the past few years owing to massive changes in the dietary habits and life style practices. We shall learn about these factors in detail in a short while from now. However, let us first understand about ulcers.

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The term peptic ulcer is used to describe any localized erosion or disintegration of the mucosal lining that comes in contact with gastric juice. Mostly, the oesophagus, stomach and duodenum lining is affected and cause stomach and duodenal ulcers (peptic ulcers). The disintegration of tissues can also result in necrosis (death of cells/ tissues). The mucosa of the stomach and the duodenum is normally protected from proteolytic action of gastric juice by the mucosal barrier. Thus, the areas affected in peptic ulcer (due to erosion) can be:

1. lower part of the oesophagus.
- 2' stomach (lesser curvature — antrum, where the food stays for a longer time).
3. first portion of the duodenum which is also called duodenal bulb.

We read a little while ago about the relation of dietary errors and lifestyle practices with respect to the development of ulcers. In the subsequent text we shall learn about the causative factors for the ulcers.

**Etiology**

Peptic ulcer results when the neural and hormonal abnormality disrupts the factors that normally maintain mucosal integrity and permit proteolytic and acidic erosion of the mucosal tissue.

Let us learn about the factors, which damage or protect the mucosa. These are summarized in Table 14.9.

| <b>Destructive Factors</b>                                                  | <b>Defensive Factors</b>                    |
|-----------------------------------------------------------------------------|---------------------------------------------|
| Hydrochloric acid                                                           | Epithelial cells barrier                    |
| Pepsin (Proteolytic enzyme)                                                 | Mucous                                      |
| Psychological factors (stress and anxiety)                                  | Gastric blood flow                          |
| Gastric irritants (alcohol, caffeine, excess tea, meat extracts and spices) | <b>Regulation of acid secretion</b>         |
| Nicotine and Tobacco                                                        | Ability of the body to regenerate the cells |
| Anti inflammatory drugs/ Analgesics                                         |                                             |

**Table 14.9: Factors affecting mucosa**

Duodenal ulcers occur in a 3 cm space between the duodenal bulb in an area immediately below the pylorus where the gastric juices are not neutralized. It can be attributed to a number of factors:

- H. pylori infection is strongly implicated which has a damaging effect on the mucosal defense thereby increasing the vulnerability to ulceration,
- Increased number of parietal cells or acid secretion.
- Increased gastric emptying rates.
- Reduced ability of the duodenum to handle an acid load
- Stress (physical and mental)

Excessive use of non steroidal anti inflammatory drugs (NSAID) and corticosteroids. Gastric ulcers occur in the lesser curvature the antrum of stomach. A gastric ulcer appears to be caused by reflux of bile and factors that disrupt the mucosal barrier permitting the hydrogen ions to diffuse into the mucosal tissue where they cause damage. The damage causes cell destruction and subsequent ulceration (due to defect in pyloric sphincter). NSAID dramatically increase the risk of ulcers and is related to the systemic inhibition of prostaglandin production. Thus results in impaired defense against acidity by the gastric mucosa. Another pathogenetic factor is *H. pylori* infection and the resulting impaired mucosal defense. Let us have a look at the pathogenetic factors in the development of peptic ulcer disease as given in Table 14.10.

| <b>Gastric Ulcer</b>                                                                            | <b>Duodenal Ulcer</b>                        |
|-------------------------------------------------------------------------------------------------|----------------------------------------------|
| Seen at the back of the stomach                                                                 | Appears within 3 cms of the pylorus          |
| Abnormal pyloric function                                                                       | Increased acid secretory capacity            |
| Duodeno-gastric reflux                                                                          | Increased basal acid secretion               |
| Defective gastric mucosal defenses                                                              | Increased parietal cell mass and sensitivity |
| Decreased mucosal blood flow,<br>Prostaglandin production,<br>Bicarbonate production, Gel layer | Prolonged meal secretory response            |
| <i>H. pylori</i> infection                                                                      | Abnormal gastric emptying                    |
|                                                                                                 | Abnormal duodenal mucosal defenses           |
|                                                                                                 | Decreased bicarbonate secretion              |

**Table 14.10: Pathogenic factors**

Peptic ulcers if not managed properly can result in serious consequences which can even be life threatening. The complications involved in this disease are:

- Gastrointestinal bleeding
- Intestinal perforation
- Peritonitis (inflammation of the lining of abdominal cavity)
- Anaemia
- Intestinal narrowing and obstruction
- Shock

Following symptoms present a peptic ulcer picture

### **Symptoms**

Increased gastric tone and painful hunger contraction when stomach is empty. Hunger contraction 1-3 hours after meals is the main complaint. Pain is often described as dull, piercing, burning and gnawing and is usually relieved by taking

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food. Frequent vomiting sometimes with blood leads to loss of weight and anaemia.

- Ulcers develop gradually, over a period of several months to several years. Majority of the patients are undernourished and have depleted reserves of several nutrients. Pain associated with consumption of food, vomiting and anaemia due to haemorrhages/ Bleeding result in reduced food intake partially due to anorexia. This is a major reason for weight loss. Maintenance of an optimum nutritional status to promote healing and proper rehabilitation is therefore the prime objective of the dietician. We shall now discuss some of the important treatment and management aspects in detail.

### Medical and Nutritional Management

To provide physiological rest and support tissue healing, treatment should be based on providing rest to the affected area. Judicious use of drug therapy and dietary modifications are the key to the management of peptic ulcers. Recent development of new drugs like Cimetidine and Ranitidine (H<sub>2</sub> blockers) have revolutionized the dietary regimes used earlier such as the Sippy's diet, Henhartz and Meulen Gratcht diet which were based on milk and contained inadequate protein and protective foods leading to nutritional deficiencies. Today the new drugs mentioned above are better than earlier drugs. Use of these drugs allow a liberal approach for food choices and the patient is able to restore good nutritional status. It is well known now that a good dietary regime and nutritional status can help in reducing the impact of the disease on the overall health and well being of the patient.

Thus, the objectives of the nutritional care process should include the following points:

1. Restoration of good nutritional status with dietary modifications and counseling.
2. Alleviate the symptoms.
3. Neutralize acids.
4. Reduce acid secretion.
5. Preservation of epithelial resistance to the destructive action of gastric juice.

Let us now move on to understand the dietary approach. We shall begin with nutrient requirements of the patients. However before we talk about the key nutrient intake let us brief ourselves on the energy intake.

- **Energy:** The energy intake should be such as to help the patient in achieving maintaining an ideal body weight. An adequate energy intake is a must to prevent subsequent weight loss and spare the proteins for healing of ulcers. You can calculate the energy requirements on the basis of patients height, activity and gender as has been discussed earlier in Unit 10 on Weight Management.

We shall now understand about the requirement for other nutrients.

- **Proteins:** Ulcer is a form of wound which if not healed on time can get perforated bleed. Adequate protein intake ensures synthesis of new tissues essential for healing. Normal milk protein is valued for their buffering action



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but it is supposed to provide temporary relief because the products of protein digestion (amino acid and polypeptides) reach the antrum and they stimulate the secretion of gastrin and gastric acid, Nifk neutralizes gastric acidity only for 20 and 60 minutes after its ingestion and the pH reverts back to the basal levels. Current studies indicate that a diet with high milk content has an adverse effect on the healing rate of ulcer because of high calcium content. This, in turn, may also stimulate excess acid production. Thus, the use of milk should be limited and used for symptomatic relief for a short duration only. Restricted intake of milk gives relief for a short period as it contains the prostaglandin PGE<sub>2</sub>-a protective agent against stress induced ulcers.

Proteins from cottage cheese, egg, chicken and fish in adequate amount is beneficial for regeneration of cell. Protein supplement may be used and the protein content can be increased by 10-15 g /day above the RDA.

- **Fats :** These delay the gastric emptying. The products of fat digestion in the small intestine stimulate enterogastrone, which inhibits gastric juice secretion. Recently poly-unsaturated fatty acids such as linoleic and eicosapentanoic acid have been found to be effective against duodenal ulcer by inhibiting in vitro growth of H. Pylori. Around 25-30 g of visible fat may be incorporated in the daily dietaries. Fats should preferably be emulsified for easier digestion. Fried foods must be avoided as they cause digestive problems.
- **Carbohydrates:** These should provide around 55 to 65% of the daily intake. Emphasis should be laid on the consumption of both simple and complex carbohydrates but in soft well cooked form. Soluble fibre is more beneficial as compared to insoluble fibre in view of its physical attributes. The presence of fibre in the diet is advocated because it delays gastric emptying time and hence prevents the mucosal damage by acidic gastric juice.

Other important factors which need to be considered have been discussed below:

- **pH of food:** It has a little therapeutic importance except for patients with lesions in the mouth or oesophagus. Most foods are considerably less acidic than the normal gastric pH of 1.6. The pH of both orange juice and grapefruit is 3.2 to 3.6. Thus on the basis of their immediate acidity, acidic fruit juices should be acceptable components of the diet for the patients with ulcers. Fruits, in general, are related to an alkaline ash diet. If they are not well tolerated by some individuals, avoid them.
- **Foods that damage GI mucosa:** A number of spices, herbs and other condiments have been found to have little or no irritating effect on the majority of persons with ulcers. The sight, smell and taste of most food normally initiates the cephalic phase of gastric secretion but no significant change in gastric pH was noted with any particular items except in case of alcohol, caffeine, black pepper and meat extracts.
- **Alcohol:** Alcohol is known to cause damage to intestinal mucosa independent of gastric acid content. Thus high amounts are not advised.
- **Cigarette smoking:** Smoking of cigarette causes an adverse effect because

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of the presence of nicotine which causes pyloric incompetence, increased reflux of duodenal juice into the stomach, increased gastric acid secretion by favouring gastrin secretion, decrease pancreatic bicarbonatesynthesis. Stopping smoking is highly recommended for peptic ulcer patients.

- **Food texture:** Recent studies indicate that strict omission of fibre is of no help on a peptic ulcer patient. The recurrence of peptic ulcer was observed to be much lower in individualson high soluble fibre diet. This has also been attributed to increased salivation due to increased chewing which has shown to have a buffering effect.
- **Gas formers :** Omitting a number of foods routinely because of their reputation of being gas formers has also been questioned. Clinical observations have shown that tolerancefor a variety of standard foods is highly individual. Pulses, soyabean, cabbage, cauliflower, onions, peas, apple, watermelon are some of the foods identified.

Current approach of liberal management in peptic ulcer medical nutrition therapy postulates: It is the individual patient who the focus of treatment thus treat the patient as a whole and not merely treat the hole. Remember the latest drug therapy is essential.

### **The basic principles involved are:**

The individual must be treated as such and for that careful initial history — daily living situation, attitudes; food reactions, tolerances must be kept in mind. This would serve as a basis for formulating the nutritional programme

The activity of the patient's ulcer will influence the dietary management. During period of active ulceration more acute modified treatment may be needed to control acidity and initiate healing. However when pain disappears feedings are liberalizedaccording to the individual's tolerance levels and desires using a variety of foods.

Let us move on to the different stages involved in feeding the patient.

**Stage I :** It is characterized by haematemesis (vomiting of blood which may be derived from mouth, stomach, oesophagus or duodenum or melena (passage of black tany stools indicativeof GI bleeding). Initially, for bleeding ulcer, if the patient is extremely nauseated or vomiting, he must be kept on NBM (nil by mouth). This is followed by an hourly feeding to begin with. Milk and cream 100 Bihour (especially in stages of acute pain) followed by small feedings of easily digestible foods like soft cooked eggs, custards, refined flour products, cottage chkeese, low fibre vegetables like gourds, clear soup with no seasoning and herbs, soft over ripe fillit whips and light desserts. The diets must be fed orally, and of liquid / semi liquid / soft consistency, which is easy to digest.

**Stage II :** The characteristics of this stage involves - marked recovery from pain.

- 6 meal pattern followed

- light, bland, low fibre diet

mechanical/thermal, chemical irritation of gastric mucosa to be avoided

a. late night feeding avoided, as the end products of digestion may cause the epigastric pain.

**Stage III :** In this stage, following characteristics are involved -

- number of feeds reduced to 3-4, as recent studies show no benefit in terms of
- gastric acid secretion
- discharged from hospital
- increased amount / feed

**Stage IV :** This stage involves -

- liberalizing the diet depending on the patient's individual tolerance and schedule.
- ensuring optimum intake of calorie, protein, fats, vitamins and minerals.
- relaxed atmosphere on eating.
- lifestyle change (stress, alcohol, caffeine, smoking )

**Remember to recommend:**

1. More than three regular meals to be eaten daily.
2. Eat small meals to avoid stomach distention.
3. Avoid drinking excess of coffee and alcohol.
4. Cut down on or quit smoking cigarettes
5. Avoid using large amount of aspirin, Non Steroidal Anti Inflammatory Drugs(NSAIDs) and other drugs known to damage the stomach lining.
6. Avoid foods or drinks that cause discomfort. Reduce spices especially black and red pepper. Increase n-3 and n-6 fatty acid consumption.
7. Eat meals in as relaxed atmosphere as much as possible.
8. Take antacids 1 and 3 hours after meals and before bedtime respectively.
9. Take adequate rest, relaxation and sleep.
10. Take the necessary drugs advised by your doctor, for neutralizing the acid, reducing acid secretion or preservation of the epithelial tissue or an antibiotic combination for eradication of the H.pylori infection.

### 14.3.9 Malabsorption Syndrome

Did you know that a major part of the absorption of nutrients takes place in the small intestine and the set of enzymes involved in this process are called disaccharidases. In some conditions either genetically, or due to some intestinal damage there appears to be a deficiency of some of these enzymes, which in turn, leads to the malabsorption of some of the nutrients precipitating symptoms of diarrhoea, distention and abdominal discomfort and steatorrhoea (fat in stools).

These conditions are together referred to as Malabsorption Syndromes. The term

**NOTES**

'malabsorption syndrome', as you have learnt earlier also, is used to describe deficient absorption to a variable degree of a number of substances such as fats, proteins, carbohydrates, vitamins, minerals and water.

Before we understand about this syndrome, let us look at the following case study. Anuradha, a teenager, presents to the physician's office with a two-year history of intermittent diarrhoea. Her reports reveal a past history of anaemia, anorexia and minor abdominal pain. Her weight has been the same for 2 years now. Her mother has attributed this to her having a "rough time in school". Her mother also questions whether the symptoms could be related to a recent move from their home. She has not yet reached menarche, A diet history suggests a normal diet with adequate iron intake. Can you guess what she suffers from and what could be the causative factors leading to such a condition?

Well, you have guessed it right. She suffers from 'Malabsorption syndrome'. Let us proceed further and get to know more about it. We shall begin with the etiology.

**Etiology**

The causes cited for malabsorption can be associated with a number of diseases. Many of these diseases you may not know presently but they will unfold as you read further.

|                        |                                                                                             |
|------------------------|---------------------------------------------------------------------------------------------|
| Anatomical or Surgical | : Surgical resection, Fistula, Gastric surgery, Blind loop and stricture and Diverticulosis |
| Enzyme deficiencies    | : Pancreatic disease, Biliary obstruction and Disaccharidase deficiency                     |
| Mucosal defects        | : Celiac disease, Tropical Sprue, Crohn's disease and Radiation                             |
| Systemic causes        | : Scleroderma, Diabetes, Lymphoma, Thyroid disease and Severe skin disorders                |
| Drugs                  | : Cholestyramine, Antibiotics, excess laxatives                                             |
| Infections             | : Giardia and parasitic infestation, tuberculosis and bacterial overgrowth.                 |

**Table 14.11: Principal causes of malabsorption**

Malabsorption can thus occur due to a host of reasons. However, what are the symptoms that would have an impact on the nutrition and health status of the patient and hence his dietary intake. Let us read and find out next.

**Symptoms**

The most common symptoms are weakness, lassitude and marked weight loss. Steatorrhoea (excess fat in stools), anaemia and chronic ill health. Diarrhoea is the most common GI tract disturbance along with flatulence, mild abdominal pain, anorexia, nausea and vomiting. Nutritional deficiency commonly occurs and may manifest itself as glossitis, tetany, bone pain and paraesthesia and convulsions. The objective evidence of these are scenas smooth tongue, oedema, dry skin, bleeding, pigmentation, dermatitis, peripheral neuropathy and proximal muscle

atrophy.

Let now discuss a few important conditions grouped collectively under the term of malabsorption syndrome. These are:

- Celiac disease
- Tropical sprue
- Steatorrhoea
- Lactose intolerance
- Inflammatory bowel syndrome,
- Ulcerative colitis
- Short bowel syndrome

We shall brief ourselves on some of these disorders. Let begin with celiac disease,

### **A). Celiac Disease**

Gluten-sensitive enteropathy or, as it is more commonly called, celiac disease, is an autoimmune inflammatory disease of the small intestine. It is precipitated by the ingestion of gluten, a component of wheat protein-gliadin, in genetically susceptible persons. A defect in the enzyme system that splits this protein fraction along with atrophy of jejunal mucosa may be the specific cause for celiac disease. It usually develops within the first three years of life.

### **Symptoms**

Child with celiac's disease fails to thrive, loses appetite and has a potbelly. Stools are large, pale and offensive due to the presence of fat in the form of fatty acids. Anaemia is present with symptoms of paleness, fatigue, tachycardia (fast pulse). The microscopic section of the villi show flattening of the villi. When gluten-free foods are given there is a dramatic recovery in the symptoms and the reversal of villi to normal growth. Celiac disease has also been noted to be associated with numerous neurologic disorders, including epilepsy, cerebral calcifications, and peripheral neuropathy. The list of symptoms as mentioned in various sources for celiac disease includes:

**Digestive symptoms** — diarrhoea, abdominal pain, abdominal bloating, pale stool, foul-smelling stool, loose stool, flatulence.

**Behavioural symptoms** — there are also several other symptoms such as **irritability** — especially in children, depression and behaviour changes in adults.

**Inadequate nutrition symptoms** — because celiac causes malabsorption, the body does not get enough nutrients, leading to symptoms such as weight loss, delayed growth, failure to thrive (infants), missed menstrual periods, anaemia and fatigue. Anaemia is the most common laboratory manifestation of celiac disease. Iron is absorbed in the proximal small intestine, where celiac manifestations are most prominent; hence, iron malabsorption is common. Less commonly, vitamin

## NOTES

B<sub>12</sub> deficiency, folate deficiency, or both may be present.

Gas formation, bone pain, joint pain, seizures and inuscle cramps.

**Non-specific symptoms** — some people get mild but unclear symptoms such as, tingling sensation, numbness (due to damage of nerves in the legs), painful skin rash, tooth discolouration and enamel loss. Some important complications are enumerated herewith:

### Complications

Patients with severe form of celiac's disease for long period are at risk for several complications mainly due to nutrient absorption problems leading to malnutrition.

|                                                       |                            |
|-------------------------------------------------------|----------------------------|
| Congenital defects — in babies born to celiac mothers | Miscarriage                |
| Osteoporosis (weak and brittle bones)                 | Stunted growth in children |
| Lymphoma (can develop in the intestine)               |                            |
| Seizures or convulsions                               | Anaemia                    |

**Table 14.12: Complications due to Celiac's disease**

Dietary management of celiac disease is of crucial significance as it is related not only to the frequency and severity of morbidities but also mortality of the patient, usually a child. We will brief ourselves on the dietary management next. Based on the cause, symptoms and complications the major objectives of dietary management include the following:

- Providing a nutritionally adequate diet
- Strict restriction of gluten foods
- Vitamins and mineral supplementation

In the subsequent text we will learn about the dietary management of celiac disease.

### Dietary Recommendations

The only dietary treatment for celiac disease is to follow a gluten-free diet. For most such a diet improves symptoms, heals intestinal damage, and prevents further damage.

**NOTES**

Improvements begin within days of starting the diet, intestines are healed within 3-6 months for children but in adults it could take upto 2 years. The gluten-free diet is a lifetime requirement. Eating any gluten, no matter how small an amount, can damage the intestine. A small percentage of people with celiac disease do not improve on the gluten-free diet because the intestines are severely damaged. Such patients must be evaluated for any complications. In such cases there is need for intravenous nutrition supplements.

What is a gluten-free diet? Let us read and find out. The Gluten-Free Diet, as we have learnt earlier also, is a diet that contains no gluten. Foods like wheat, rye, barley, and possibly oats must be avoided. The gluten-free diet is complicated. It requires a completely new approach to eating that affects a person's entire life. Products like bread biscuits, breakfast cereals, poories, paranthas, chapathis, macaroni, noodles and other pasta preparations have to be totally eliminated. Eating out can be a challenge as the person with celiac disease learns to scrutinize the menu for foods with gluten and question the waiter or chef about possible hidden sources of gluten. What are these hidden sources? Well, these hidden sources include additives, preservatives, and stabilizers found in processed food, medicines, and mouthwashes. Despite these restrictions, people with celiac disease can eat a well-balanced diet with a variety of foods.

**Table 14.13: Gluten and Non-gluten sources**

| Gluten Sources                                                                                                                | Non-gluten Sources                                                  |
|-------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------|
| <i>Beverages:</i> cereal beverages, .<br>ovaltine, beer and ale                                                               | Coffee, tea, chocolate drinks (pure<br>cocoa) wine                  |
| Milk beverages that contain malt                                                                                              | Whole, toned, skim milk and buttermilk                              |
| <i>Meat and meat products:</i><br>Breaded meats commercially available                                                        | Pure meats, fish, poultry, eggs, cottage<br>cheese, peanut butter   |
| <i>Fats and oils:</i><br>Commercial gravies, white sauce<br>and cream sauces                                                  | Butter fats and oils                                                |
| <i>Cereal and cereal products:</i><br>Bread, wheat, oats, rye, malt, pastry<br>flour (maida), bran, barley, wafers,<br>pasta. | Rice, potato flour and soya flour,<br>pure corn, popcorn.           |
| <i>Vegetables:</i><br>Breaded vegetables with any of the<br>sauces, white sauces etc.                                         | All fresh vegetables, canned and pureed.                            |
| <i>Fruits:</i><br>Any fillings e.g. pies etc.                                                                                 | All fresh fruits                                                    |
| Snacks:<br>Pastries, patties, pizzas, samosas,<br>mathris etc.                                                                | Milk base sweets (pure) without addition<br>of any cereal products. |

Let us have a look at few of the tips which patients can follow and enjoy their meals and at the same time, avoid any possible nutritional deficiency.

Iron deficiency should be treated with supplemental iron.

Osteoporosis should be treated with calcium and vitamin D supplements.

Depending on individual factors, patients with gluten-sensitive

## NOTES

enteropathy may

need to take a multivitamin supplement along with iron, calcium, magnesium,

zinc, selenium, vitamin D, or other nutrients,

Check for commercial gluten-free products, including breads, cookies, chips, and breakfast cereals that may be available in India.

Meats, vegetables, fruits, and most dairy products are free of gluten.

Another common malabsorption syndrome listed in the section 14.8 steatorrhoea which is discussed below.

### **B). Steatorrhoea**

Steatorrhoea is a symptom of the disorders of fat metabolism and malabsorption syndrome and can be defined as a condition of foul-smelling bulky faeces. It is suspected when the patient passes large, "greasy", and foul-smelling stools. Both digestive and absorptive disorders can cause steatorrhoea. Digestive disorders affect the production and release of the enzyme lipase from the pancreas, or bile from the liver, which are substances that aid digestion of fats. Absorptive disorders disturb the absorptive and enzyme functions of the intestine. Any condition that causes malabsorption or maldigestion is also associated, with increased faecal fat (steatorrhoea). As an example, children with cystic fibrosis (hereditary disease) have mucous plugs that block the pancreatic ducts. The absence or significant decrease of the pancreatic enzymes; amylase, lipase, trypsin, and chymotrypsin limits fat, protein and carbohydrate digestion, resulting in steatorrhoea due to fat malabsorption.

A predominant feature is delayed and defective absorption of fat, which results in bulky stools containing large quantities of fat (known as steatorrhoea). The severity of steatorrhoea depends on the quantity of fat in the diet. Besides the absorption of water, electrolytes, vitamins and minerals is also impaired. These defects are due to flattening of the villi in the jejunum (a part of the small intestine). Remission and relapses are common if proper medical and dietary care is not provided. The major reasons attributable to steatorrhoea have been enumerated below.

#### ***Etiology***

The list of possible underlying causes of steatorrhoea includes:

Malabsorption

Malabsorption of fats in small intestine

Pancreatitis

Celiac disease

Sprue

#### ***Symptoms***



The list of symptoms includes:

- Bulky, pale, loose, greasy and foul smelling stools.
- Anorexia, feeling of fullness, pain in abdomen.

The major points that we must remember while planning diets for patients suffering from steatorrhoea are highlighted below for a quick reference.

### ***Nutritional Management***

The nutritional management of steatorrhoea should focus on the following:

- Plenty of rest and relaxation and avoid stress
- Correction of water balance
- Correction of electrolyte problems (Na, K, Ca)
- Vitamin supplementation (A, D, E and K)
- Inclusion of low fat, carbohydrates and fibre diet
- High to moderate protein intake
- Give digestive enzyme supplements (if required)

The nutrient requirements do not change considerably and other principles of dietary management remain more or less the same as for chronic diarrhoea.

Next, we will discuss about lactose intolerance— a form of food allergy which has a widespread prevalence.

### **C). Lactose Intolerance**

We commonly hear from people of all age groups, particularly children and elderly to be complaining of abdominal discomfort after consuming milk. Some individuals are able to tolerate a small quantity while others are unable to tolerate even a small amount. Well, this could be due to lactose intolerance. But what is lactose intolerance?

Lactose intolerance relates to insufficiency of the disaccharidase enzyme 'lactase' which is found in the greatest quantity in the outer membrane of the mucosal cell of the jejunum. The degree of lactase deficiency may vary in individuals. Lack of lactase does not break down the disaccharide sugar— lactose present in milk, to glucose and galactose, it passes unchanged into the large intestines where it gets converted to lactic acid by the bacteria, which subsequently cause diarrhoea and other symptoms of discomfort, distension and abdominal pain. The problem is gene related and often seen in infants and young children commonly, but may also be present in adults, Major causative factors are being discussed below.

### ***Etiology***

The etiological factors contributing to lactose intolerance include:

- Genetic factor

Reduction in jejunal lactase activity due to infections in the gut.

Any structural damage to the jejunal mucosa in disease conditions like celiac, tropical sprue, colitis in which the jejunal villi are structurally damaged.

Surgical causes in which large part of jejunum is removed.

## NOTES

### *Symptoms*

Common symptoms linked to lactose intolerance include:

1. Anorexia and nausea.
2. Intestinal distension
3. Abdominal cramps
4. Gas and flatulence
5. Severe diarrhoea
6. Under-nutrition and loss of weight.

The dietary treatment is based upon the determination of lactase activity as the treatment depends on the level of activity of lactase enzyme. Let us see how.

### *Nutritional Management*

Diagnostic tests are available that can give information about the level and activity of the lactase enzyme. Depending on the level of activity (very low level, moderate level) the dietary treatment could be planned. Let us see how.

- Very low level of lactase activity: at very low level of lactase activity all milk products must be eliminated, substitutes of milk like soya milk, groundnut milk and their preparations could be given. Enzyme such as Lactaid and Maxilact are available in the market. Addition of these in the milk or milk products could digest 90% of lactose in milk and thus minimize the symptoms of lactose intolerance.
- Moderate level of Lactase activity : Intake of milk is restricted depending on the tolerance. Fermented and cooked form of milk should be preferred as it is better tolerated. Fermentation converts a major part of lactose to lactic acid and in cooked product lactose gets bound and the concentration reduces. It is better tolerated in the form of buttermilk, curds, custards, porridges and cottage cheese or when mixed with cereals, cocoa etc. These allow gradual lactose breakdown and decrease the symptoms of lactose intolerance. Curds are better tolerated possibly due to microbial culture that facilitates lactose digestion in the intestine. Small amount of milk can be taken with the meal.'

Some important points to remember are highlighted next:

## Remember

- Identify the level of lactase activities (diagnostic tests).
- Depending on the enzyme activity eliminate milk and milk products.
- Substitute milk and milk products by giving soya sources like —tofu, soymilk, soy curd and groundnut milk.
- Give a well balanced diet.
- If moderate lactase activity is present small amounts of lactose (within individual's tolerance level) can be given several times a day.
- Small amounts of milk in moderate lactase activity can be tolerated if taken with other foods e.g. after a meal or a snack.
- Curds is better tolerated than milk.
- Low lactose foods if available commercially like ice cream, cottage cheese, try them.
- Lactase enzymes are available these can be added in the milk.
- Deficiency of lactase and calcium could be supplemented by giving other foods.

The last malabsorption syndrome which will be discussed is the inflammatory bowel disease which is a broad term that refers to a number of disorders of the bowel.

### 14.4.9.4 Inflammatory Bowel Disease (IBD)

Inflammatory bowel disease is a general term used to refer to chronic inflammatory conditions of the intestine. It is applied to three conditions having similar symptoms but different underlying clinical problems. It includes:

1. Ulcerative colitis
2. Crohn's disease
3. Short bowel syndrome

When the inflammation is in the rectum with extension into the colon without affecting the right colon or small intestine, the disease is called ulcerative colitis. When an inflammatory process involves one or more lengthy segments of the small or large intestine with inflammation from the mucosa to serosa, the disease is called Crohn's disease.

What is the difference between ulcerative colitis and Crohn's disease? The differences between the two include:

- 1) Anatomic distribution of the inflammatory process: Crohn's disease can occur in any part of the GI tract — the small intestine, the colon and even the colorectal region. However, in ulcerative colitis, the inflammation is confined to the left colon and rectum.

- 2) In Crohn's disease, inflammatory process affects the entire thickness of the wall of the small intestine leading to strictures that can cause obstructions or formation of fistulas. In ulcerative colitis the inflammatory process is in mucosa and sub mucosal tissues of the intestine and lasts for a few weeks.

## NOTES

Having looked at the difference between ulcerative colitis and Crohn's disease, let us next review the etiology of these diseases.

### **Etiology**

These diseases are referred to as idiopathic (cause unknown) and though the possible mechanism suggested includes genetic factors, immune mechanism, bacterial or viral agents, sugar (excess) and low fibre intake has also been implicated especially in Crohn's disease.

The symptoms, complications of inflammatory bowel disease are summarized next.

### **Symptoms**

Inflammatory bowel diseases are associated with:

- Abdominal cramping, diarrhoea
- Steatorrhoea
- Obstruction caused due to bulky foods, and
- Malnutrition

What are the causes for malnutrition in these disease conditions? Let us find out.

#### **Causes of Malnutrition in Inflammatory Bowel Disease**

The causes of malnutrition include:

- Decreased oral intake, which can be disease induced due to abdominal pain, diarrhoea, nausea, anorexia.
- Malabsorption due to decreased absorptive surface (destruction of villi), bile salt deficiency, bacterial overgrowth and use of drugs.
- Increased secretion and nutrient losses due to GI blood losses, electrolyte, trace mineral losses.
- Drugs Increased requirements due to inflammation, fever, increased intestinal cell turnover, haemolysis.
- Drugs interference related to corticosteroids, (interferes in calcium absorption and protein metabolism), sulfasalazine (interferes in folate absorption), Cholestyramine (interferes in fat soluble vitamin absorption).
- Deficiency of folate, vitamins A, C, D, low serum levels of zinc, copper and iron of Gastrointestinal are observed in patients.

### ***Nutritional Management: Inflammatory Bowel Disease***

Adequacy of nutritional needs and minimizing stress on the inflamed or narrowed

segment of the bowel are the main principles of nutrition management.

To decrease eating associated symptoms and decreased bowel activity during healing, patients hospitalized for IBD (Inflammatory Bowel Disease) are placed on a "bowel rest" programme, which involves reduction in oral intake, clear liquids and low residue foods. This is normally done to achieve the following:

1. Decrease the absorptive work of the bowel and provide rest.
2. Minimize mechanical trauma caused by the passage of food.
3. Decreased diet associated secretions (acid, enzymes) that may aggravate inflammation.

The diet should be liberal in protein and calories and should be sufficient to maintain or restore weight/su ort growth of children and adolescents. Supplementa tion with multivitamin re arations (1-5 times above RDA) is necessary as this condition re ci itates deficiency of many nutrients, decreases absor tion and increases requirements.

Overemphasis on fibre may be avoided in patients with strictures as it may lead to blockage.

Steatorrhoea is more common in Crohn's disease due to ileal resection. This may favour calcium-fatty acid complex formation and increased excretion. It may be accompanied by magnesium (Mg) and zinc (Zn) losses. Steatorrhoea also favours increased absorption of oxalates. In addition, this state is marked with an increased binding of fatty acid to calcium and thus more oxalate is free in solution for colonic absorption. Fatty acid also increases the permeability of oxalate through colonic mucosa. Thus, a reduction in fat intake coupled with calcium, magnesium and zinc supplementation is suggested.

To help you understand the inflammatory bowel diseases better, we have a detail discussion on two of these diseases namely ulcerative colitis and small bowel syndrome. We begin with ulcerative colitis.

### ***A. Ulcerative Colitis***

Let us understand clearly about ulcerative colitis by reading the following case.

Varun, a 48-year-old male, had a very successful career in a computer company. His company was his life. He put in long hours when he was working on an important contract and seldom even took a Sunday off. He was delighted when a deal came together, and he celebrated his success at his favourite Chinese restaurant. When he worked 10-12 hours at a stretch, he just ordered his favorite Chinese food, which frequently gave him bouts of diarrhoea. But the latest episode was really bad.

He felt nauseated and had cramps for 2 nights and developed a fever. On the second night, he noticed blood in the stools and he resolved to call the doctor.

What do you think he might be suffering from? Well, this is the case of Ulcerative Colitis. What is it? Let us find out.

## NOTES

Ulcerative Colitis is a diffuse inflammatory and ulcerative disease of unknown etiology involving the mucosa and sub-mucosa of the large intestine. It occurs at any age but predominates in young adults. Onset is insidious in the majority of cases.

### ***Etiology***

No single etiologic factor has been identified although genetic auto-immune factors are thought to be involved. Although exacerbations are more likely during the conditions of mental conflict and emotional stress. Allergy to certain foods especially milk may be a factor in precipitating the disease.

What are the disease symptoms? Let us find out.

### ***Symptoms***

As discussed in the case study above, the common symptoms are:

1. Mild abdominal discomfort, an urgent need to defecate several times a day.
2. Diarrhoea accompanied by rectal bleeding.
3. Weight loss, dehydration, fever, anaemia and general debility.
4. Edematous and hyperemic mucosa seen in early stages.
5. In more severe disease, necrosis and frank ulceration of the mucosa occurs.

So how to manage this chronic condition? The dietary management is described next.

### ***Dietary Management***

Proper dietary management is important for maintaining a good nutritional status of the patient. Long-term management is generally required as the disease develops gradually and it takes a long time to cure this disorder. We will now learn in detail the important aspects of dietary management.

The dietary management and nutrient recommendations need individual attention depending on the extent of disease and problems of malnutrition exhibited, There is a wide range of tolerance for various foods observed from one patient to another. Let us first identify the calorie needs of the patient.

**Energy:** The calorie requirements must be increased to:

- 1) restore weight status and maintain ideal weight.
- 2) compensate for the elevated BMR.

- 3) support growth especially if the age group is adolescents. A caloric intake of 40- 50 Kcal/kg IBW/day is recommended.

**Proteins:** Patients with ulcerative colitis lose about 4-8 g fecal N<sub>2</sub> as compared to the normal excretion of 2 g. In severe ulcerative colitis, 20 g N<sub>2</sub> (equivalent to 125 g of protein) may be lost daily. The serum albumin is low. Proteins are necessary for tissue synthesis, tissue healing and to compensate for the increased losses in stools. Thus, liberal amounts of high quality protein i.e. 1.5 g / 1B W are needed to make up for the losses. Emphasis should be on tender meats, fish, poultry and eggs for those patients who are allergic to milk.

**Fats:** Usual foods, which contain fats (invisible or inherent fat), are permitted but not fried foods, as they are not easily digested due to liver dysfunction. Thus fats rich in medium chain triglycerides should be consumed as steatorrhoea is predominant in ulcerative colitis. Total fat intake can be kept close to 55-60 g with visible fat intake less than 25-30 g/day.

**Carbohydrates:** They form the easily absorbable source of energy. Bulk-producing vegetables are restricted so as to allow better intake of nourishing foods. Sugars and starches can make the increased caloric intake.

**Fibre :** Eliminating roughage seems to have a better effect on preventing relapses of the disease. A low residue diet may be given during an acute attack to prevent severe bleeding during diarrhoea. Thereafter some degree of fibre restriction is generally needed as many ulcerative colitis patients do not tolerate raw vegetables. It may cause further damage to the already inflamed mucosa. The forms, in which vegetables are given, can be changed. All kinds of irritant and spicy foods should be strictly avoided. Raw vegetables, spicy and irritant foods may be avoided.

**Vitamins:** Commercial multivitamin preparation should be administered orally especially the ones needed for the healing process and the utilization of calories and proteins.

**Minerals:** Mineral losses may be marked and unless replaced may contribute to a fatal outcome. A patient with moderately advanced ulcerative colitis passes a large volume over 400 ml of faeces per day and thus may lose considerable amount of sodium (6 g NaCl/litre of stool). Thus oral sodium intake is increased by added salt, sprinkling additional salt in foods. Potassium loss can be estimated as 30 mEq / 22 g of potassium chloride / litre. Usually high excretion of potassium even 167 mEq / day may sometimes be encountered. Manifestations of potassium deficiency such as weakness, hypotonia; abdominal distension and even electrocardiographic changes may occur. Oral administration of potassium salts as potassium citrate may may be helpful.

Elimination of milk from the diet may call upon calcium supplementation to the extent of 400-800 mg/day. Protein to Calcium ratio is to be maintained for optimum utilization. Iron by the oral route is usually not well tolerated. Daily about 30 mg of elemental iron is given. If anaemia is marked, then blood transfusions may be

## NOTES

given.

**Fluids** : A liberal intake of fluid should be given to prevent dehydration. The passage of at least 1200 ml of urine indicates that a patient is well hydrated.

### **B. Short Bowel Syndrome (SBS)**

Short bowel syndrome is a group of problems affecting people who have had half or more of their small intestine removed. The massive resection of the intestine decreases the transit time of the faeces. Besides any damage to the small intestine, especially that of the jejunum affects the nutrient uptake and absorption.

#### ***Etiology***

The etiological factors involved in this disease are:

- Anaemia
- Osteoporosis
- Stone formation
- Decreased susceptibility to infection
- Dehydration

The common symptoms elicited by this disease have been mentioned below.

#### ***Symptoms***

Diarrhoea is the main symptom of short bowel syndrome. Other symptoms include:

- Anorexia
- Steatorrhoea
- Heart burn and cramping
- Bloating and abdominal pain
- Extreme fatigue
- Weight loss
- Fluid retention
- Anaemia and osteomalacia

Many people with short bowel syndrome are malnourished because their remaining small intestine is unable to absorb enough water, vitamins, and other nutrients from food. They may also become dehydrated, which can be life threatening. Problems associated with dehydration and malnutrition include weakness, fatigue, depression, weight loss, bacterial infections, and food sensitivities. Complications

Complications of short bowel syndrome are generally related to malnutrition. What are the complications of malnutrition? A person with short bowel syndrome is likely to be deficient in a range of important nutrients such as calcium, folate, iron, magnesium, vitamin B12 and zinc.



## ***Dietary Management***

It must be evident from the symptoms listed above that the disease results in reduced food intake, impaired absorption and hence weight loss. The patient generally has depleted reserves for several nutrients. The Nutritional management goals should therefore include the following objectives:

- Relieve the symptoms
- Correct nutritional deficiencies,
- Control inflammation and relieve pain.

The dietary considerations should be aimed to give sufficient calories. Special feeding methods such as enteral and parenteral feeding may have to be incorporated. The following points should be kept in mind:

- A high calorie and low residue diet that also supplies the vitamins, minerals and other nutrients is necessary. The food should be bland.
- The food should be divided into several small meals of protein and complex carbohydrates. A minimum of concentrated sweets, fruit juices should be included.
- Nutritional supplements and dietary restrictions are used in treating SBS. The vitamin and mineral supplements may have to be several times greater than the standard recommended daily allowances in order to maintain adequate tissue functioning.
- Special feeding to be given when essential (enteral and parenteral feeding). Oral feedings are started using a basic soft diet, which can be digested without much work required in the bowel. The complexity of the diet is gradually increased over time, allowing the remaining digestive system to adapt. Finally, patients are weaned entirely off the enteral feeding and receive nutrition completely from oral intake of regular foods. If parenteral feeding is must, it could be given but enteral feeding should be preferred.

Dietary management is supported with drug therapy. Frequently used medications include anti-inflammatories, immunosuppressants, antibiotics, corticosteroids and antidiarrhoeals.

In this section, we learnt about a spectrum of malabsorption syndromes. You may have noticed that the dietary management of these disorders is quite varied. Attempt the questions given in check your progress exercise 5 to recapitulate your concepts.

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## **14.4 LET US SUM UP**

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In this unit, we learnt about a host of disorders associated with a part or entire of the gastrointestinal tract. The important and the most frequent occurring GI tract disorders such as constipation, diarrhoea, oesophagitis, GERD, hernia,

## NOTES

ulcers, dyspepsia, gastritis, various malabsorption syndromes, inflammatory bowel diseases were dealt in this unit. We first reviewed our knowledge on gastrointestinal diseases in general their etiology, symptoms and complications followed by nutritional management and goals, dietary modifications and foods to be avoided, restricted and those to be given freely.

Next, we studied about these disorders separately in a greater detail, discussing their etiology, symptoms, associated complications and clinical manifestations. The nutritional aspects of these disorders and their corresponding dietary management have also been emphasized.

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## 14.5 GLOSSARY

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|                            |                                                                                                                                                                              |
|----------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Acidosis</b>            | : Increase in concentration of acidic substances in the blood.                                                                                                               |
| <b>Crohn's disease</b>     | : a chronic inflammatory disease of unknown etiology involving small and large intestines that results in diarrhoea, strictures, fistula and malabsorption.                  |
| <b>Fistula</b>             | : an abnormal passage between two internal organs or from an internal organ to the surface of the body.                                                                      |
| <b>Helicobacter pylori</b> | : a type of bacteria that can chronically infect the stomach, thought to be the primary contributor to the development of gastritis, peptic ulcers and even gastric cancers. |

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## 14.6 CHECK YOUR PROGRESS

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- 1). List five food preparations which can be used in the rehydration of acute diarrhoea. What is the relationship between salt, sugar and water in ORS?
- 2). List a few common symptoms of oesophagitis. Write two lines about nutritional management of chronic oesophagitis.
- 3). Give the causes that lead to the development of GERD. Also highlight the points that you would keep in mind while counseling such patients.

# NUTRITIONAL MANAGEMENT IN LIVER, GALL BLADDER AND PANCREATIC DISEASES

## STRUCTURE

- 15.1 Learning Objective
- 15.2 Introduction
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### 15.1 LEARNING OBJECTIVE

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After studying this you will be able to:

- describe the numerous functions of the liver, gall bladder and pancreas,
- discuss the disease conditions of these organs and how the functioning of these
- organs are compromised in various disease conditions,
- explain the causes of the disease and symptoms produced,
- elaborate on the principles involved in the nutritional and dietary management of these disorders, and
- list the foods that can be given and those avoided in these disease conditions.

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### 15.2 INTRODUCTION

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The last dealt with the gastrointestinal disorders. Did you know that when we talk 'about the gastrointestinal system, we include the liver, gall bladder and

pancreas also because of their interrelated functioning? Thus to complete our understanding in this regard let us study the disorders of these vital components of the gastrointestinal system as well.

## NOTES

This section will deal with the disease, etiology, symptoms, complications and the nutritional management goals and dietary management including foods to be avoided, restricted and taken freely in liver, gall bladder and pancreatic disorders.

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### 15.3 LIVER DISEASES

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In our section on liver diseases, let us first get a brief input to understand the normal functioning of liver as an organ. This would help us to understand clearly the variety of conditions associated with the abnormal liver functioning namely infective hepatitis, liver cirrhosis and hepatic coma. From your understanding of the Applied Physiology Course you already know that liver is the largest and the most complex organ in the body.

#### Functions of Liver

The functions of the liver are most varied and extensive and any change in normal function can affect the nutritional status of an individual. The liver is considered one of the most important organs involved in the metabolism of each and every nutrient. Damage caused to liver can impair these and many vital processes in the body. The simple digested products of carbohydrates (glucose, fructose and galactose), fats (fatty acids), protein (amino acids), vitamins and minerals are taken to the liver. The liver stores many nutrients and also produces new compounds, which may contain fat and protein, products that help in clotting of blood. The liver also removes the nitrogen produced as a result of protein breakdown and converts it into urea, which is then excreted from the blood through the kidneys. It also removes several toxins. The liver has a very important role to play in the metabolism of carbohydrates fat and protein metabolism. Now, let us get to know about the various metabolisms.

- **Carbohydrate Metabolism:** Liver cells store energy in the form of glycogen and releases it as glucose when required. This conversion of sugar from carbohydrates is known as glycogenolysis. In the absence of carbohydrates the proteins can also be converted to glucose, which is known as gluconeogenesis in the liver cells. Both glycogenolysis and gluconeogenesis help to maintain normal blood sugar levels.
- **Fat metabolism :** It relates to the production (synthesis) of triglycerides and phospholipids. Liver synthesizes lipoproteins, which are required for the transport of lipids to peripheral tissues for use or storage. It synthesizes cholesterol and converts 80% into bile and conjugated bile salts and the remainder is transported in the form of lipoproteins. It is also involved in the oxidation of fatty acids to acetyl CoA to give energy and the synthesis of bile and conjugation of bile salts.

- **Protein Metabolism:** Liver removes the nitrogen from amino acids (known as deamination), which then could be used as an energy source or converted, to carbohydrates and fats. It also converts ammonia to urea (a waste product of protein breakdown). It is the site of most plasma protein synthesis. It acts as a reserve of these proteins to replenish serum proteins. It maintains the level of non-essential amino acids, which promote tissue synthesis.
- **Mineral and Vitamin Metabolism:** Liver is a storehouse of iron and is essential for haemoglobin formation and is stored in the form of ferritin. The liver destroys the RBC and recovers the iron from it. Other minerals like zinc and copper and many vitamins are also stored in the liver and play a role in the enzymatic reactions in metabolism. Liver is a storehouse of all fat-soluble vitamins. It is involved in the conversion of carotene to retinol. It is also needed for the activation of prothrombin and conversion of vitamin D to its active form 1,25 dihydroxy cholecalciferol.

**Other functions:** The liver converts carotene to retinol—a form of vitamin A. It synthesizes heparin (anticoagulant) which prevents intra-vascular coagulation of blood.

We have read about the numerous functions of the liver. We can also understand that in any infection, inflammation or damage to the liver the normal working of the liver would be affected, this could be related to the storage function of bile, protein, fat and carbohydrates and other compounds, problems with waste production and excretion and detoxication of poisons and toxins. An infected liver becomes sluggish and the patient shows signs of lack of appetite, symptoms of jaundice (yellow colour due to bile) and even increase in liver enzymes. Jaundice is not a disease by itself but a disease.

## Jaundice

Jaundice is a term given to the yellow discolouration of the skin, mucous membranes, sclera and body tissues because of accumulation of bile pigments, in the blood. It results due to an increase in bilirubin content of the blood above the normal range (0.2 to 0.8 mg/ 100 dl plasma). The red blood cells (RBC's) are broken down in the liver after a duration of 120 days. The haemoglobin gives a pigment known as bilirubin. Under normal conditions, the liver cells absorb bilirubin and secrete it along with other bile constituents. If the liver is infected or diseased or the flow of bile is obstructed or if excess bile is produced then it gets accumulated in the blood and eventually causes jaundice. Based on the cause of jaundice it can be of three types: haemolytic, hepatic and obstructive jaundice.

Let us get to know about these types of jaundice.

- **Haemolytic Jaundice:** It is also known as pre hepatic jaundice. This relates to excessive destruction of RBC resulting in an increased bilirubin formation and anaemia. There is an increased unconjugated plasma bilirubin, which is excreted through the urine (pigment known as urobilinogen). A healthy liver

## NOTES

can handle a bilirubin load 6 times greater than normal before unconjugated bilirubin accumulates in plasma. Thus, this kind of jaundice is seen normally in individuals with congenital defects like sickle cell anaemia, thalassemia, blood transfusion reactions and septicemia.

- **Hepatic Jaundice:** In this, there is a normal bilirubin production. The liver cannot convert fat soluble bilirubin to the water soluble form. Hence, there is a decreased conjugation leading to hepatocyte damage. An excessive amount of bilirubin is seen as a mixture of unconjugated and conjugated bilirubin. This condition leads to hepatocyte damage ( jaundice). Failure of about 80% or more of hepatic functions is observed. Its clinical features include liver disease, increased unconjugated plasma bilirubin and increased alanine aminotransferase (ALT)/ aspartate transaminase (AST) enzymes.
- **Obstructive Jaundice:** It is referred to as post-hepatic jaundice. This results from the interference of normal flow of bile into the duodenum due to stones, tumors or inflammation of mucosa of the duct. This results in a backflow of bile into the blood stream and is circulated in the body giving a yellow colour.

**Toxic Jaundice:** It is also known as hepatocellular jaundice. It originates from poisons, drugs or viral infections of the liver.

### 15.3.1 Viral Hepatitis

Hepatitis is a condition of inflammation of liver. which can result in damage of the liver cells. A virus causes viral hepatitis, as liver cells are particularly susceptible to such infections. It causes damage to the liver cells and interferes with the uptake of bilirubin by the cells, and its conjugation and excretion. It can be either in form of an acute or chronic condition and is caused due to different strains of viruses such as A, B, C, D and E.

The complications they can cause:

1. **Hepatitis A:** It is commonly called infectious hepatitis caused by a known virus Hepatitis A (HAV). It is common among children and young adults. It is contracted through contaminated water, food and sewage and transmitted by faecal-oral route.
2. **Hepatitis B and C :** It is caused by a virus hepatitis B (HBV) and hepatitis C (HCV). It is more severe and prolonged in nature, and can be fatal. It is transmitted by blood transfusion from a carrier, improperly sterilized medical instruments, dental drills, skin puncturing instruments that come in contact with contaminated blood, sexual contact and saliva of an infected person. Chronic active hepatitis can develop leading to cirrhosis and liver failure.
3. **Hepatitis D :** Hepatitis D virus (HDV) is dependent on the HBV for survival and propagation in humans. It may be a co-infection (occurring at the same time as HBV) or a super infection (superimposing itself on the HBV

carrier state). This form of hepatitis becomes chronic.

4. Hepatitis E : Hepatitis E virus is transmitted via oral fecal route. Contaminated water is the major factor. Overcrowded unsanitary areas are prone to acute form of this type of hepatitis.

Acute hepatitis settles within a few (usually six) weeks and patient becomes asymptomatic. Hepatitis A virus is an example. Chronic hepatitis a more complex form of disease is caused mostly when acute hepatitis is neglected.

The various symptoms of hepatitis are:

- itchy skin
- fatigue and fever
- lack of appetite
- nausea and vomiting
- weight loss
- jaundice
- enlarged liver and spleen
- mood swings
- pain in joints of the body (osteomalacia and osteoporosis)
- autoimmune problems
- associated with high risk of cancer.

These symptoms may be seen in both acute and chronic hepatitis, however, additional symptoms during severe chronic hepatitis may include Chronic inflammation of liver, fibrosis and finally death of liver cells (necrosis). Hepatitis virus B and C are known to elicit these symptoms.

### ***Etiology***

The various causes of acute and chronic liver disease are enumerated herewith.

- 1). Acute Liver Disease (recent origin)
  - Viral Infection (hepatitis)
  - Non Viral Infection (Coxiella burnetti)
  - Drugs (paracetamol)
  - Alcohol
  - Poisons (Aflatoxin).
  - Others e.g. complications of pregnancy.
- 2). Chronic Liver Disease (Hepatitis)
  - Drugs and Toxins (Alcohol, Isoniazid, Methotrexate).
  - Neglected/acute infections (Hepatitis B, C virus)
  - Auto Immune Disease

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Biliary Obstruction (Gall stones, narrowing of duct) Now you can understand the differences between acute and chronic hepatitis in terms of symptoms and causes. The latter is more serious and if unattended can lead to a more serious damage to the liver. The disease is called liver cirrhosis.

### 15.3.2 Liver Cirrhosis

Cirrhosis is a complication of many liver diseases that is characterized by abnormal structure and function of the liver. It is the final stage of liver injury and degeneration. We have already said that neglected chronic hepatitis can progress to liver cirrhosis. In this the liver cells get inflamed, fibrous septa get develop and the liver cells die and finally nodules develop which lead to obstructions and liver failure. In other words, the active liver tissue is replaced by inactive tissue incapable of normal functioning. Such cells get filled with fibrous tissue and fat.

Thus, cirrhosis develops when the repair that is associated with the dying liver cells causes scar tissue to form. The liver cells that do not die multiply in an attempt to replace the cells that have died. This results in clusters of newly formed liver cells (regenerative nodules) within the scar tissue.

#### *Symptoms*

The common symptoms include:

- GI disturbances (anorexia, nausea, vomiting, abdominal pain and distension)
- Electrolyte and fluid imbalance
- Weight loss and muscle wasting
- Abnormal serum amino acid levels
- Fatty infiltration of the liver
- Severe jaundice
- Hepatic encephalopathy (mental problems ranging from mild confusion to coma)
- Bleeding tendency
- Ascites (accumulation of fluid in the abdominal cavity)
- Osteomalacia and osteoporosis
- High drug sensitivity
- Chronic inflammation of the liver
- Fibrosis and fatty infiltration of the liver
- Necrosis (death of cells)



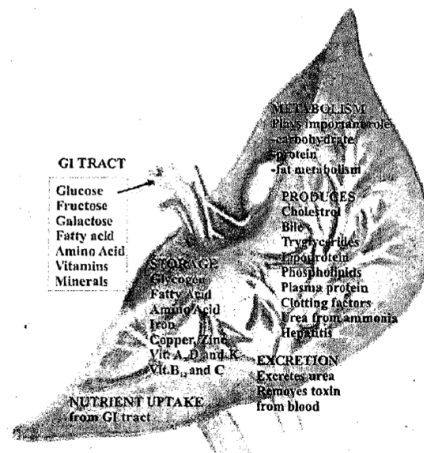
## Etiology

The etiology of cirrhosis can be enumerated as under:

- Neglected acute/chronic hepatitis
- Alcoholism associated with malnutrition
- Virus and toxins
- Metabolic disorders
- Prolonged biliary stasis.
- Altered immune response

Wilson disease is a rare autosomal recessive disorder, characterized by an abnormal copper transport and storage mechanism resulting in an excessive copper deposition in body tissues, mainly in brain, kidney, cornea including liver causing cirrhosis.

Majority of the cases of cirrhosis are, however, due to chronic abuse of alcohol, which has a hepatotoxic effect leading to malnutrition. Since excess alcohol is a major cause of liver disorder, let us understand the complications that arise from excess alcohol consumption.



**Figure 15.2: Complications of excessive alcohol consumptions**

Alcohol consumption can harm the liver by causing inflammation, necrosis due to fat accumulation in the cells which reduces the normal functioning of the liver causing serious vitamin and protein deficiency leading to malnutrition and changes in the metabolism of carbohydrate, protein and fat. For example, clotting defects could precipitate excess bleeding, protein deficiency could cause encephalopathy and result in coma. Portal hypertension could result in ruptured varices and ascites. Excess uric acid could precipitate gout and mineral deficiencies. Excess alcohol intake can cause multiple complications and malnutrition. It can also affect the kidney, heart and the vessels and may cause other problems such as oedema or ascites, bleeding, gout and acidosis.

The pathogenesis of alcoholic liver disease progresses in 3 stages.

## NOTES

### **Stage 1: Hepatic Steatosis or Fatty Liver**

During this stage, the fat infiltrates into the functioning liver cells and cause problems in normal functioning of liver. The excess fat could come from the fat stores of the body, increased production of fat in the liver. This stage is reversible with abstinence from alcohol and if abuse continues, it can lead to hepatitis and cirrhosis.

### **Stage 2: Alcoholic Hepatitis**

Alcoholic Hepatitis is characterized by hepatomegaly (enlargement of liver). Patients have abdominal pain, anorexia, nausea, vomiting, weakness, and diarrhoea, weight loss and fever. If patient discontinues the alcohol intake, hepatitis may resolve or else it progresses to the third stage of alcoholic cirrhosis.

### **Stage 3: Alcoholic Cirrhosis**

In this stage, patients develop further complications of ascites, gastrointestinal bleeding, portal hypertension, hepatic encephalopathy and other symptoms of liver disease.

### ***Complications***

Major complications of cirrhosis include:

- Ascites (accumulation of water in abdomen)
- Upper gastrointestinal bleeding (oesophageal varices)
- Hepatic coma or Hepatic Encephalopathy

A brief discussion on each of these follows.

**Ascites:** It is a characteristic symptom of advanced stage of liver cirrhosis. It relates to the accumulation of massive quantities of fluid in the peritoneal cavity of the abdomen. This may be due to:

- a) Portal hypertension (obstruction of portal blood vessels that increase intra hepatic pressure),
  - b) Hypoalbuminaemia (a fall in colloidal osmotic pressure due to inadequacy of serum albumin), and
  - c) Renal dysfunction (increased renal tubular sodium resorption; and water retention).
- **Oesophageal varices:** It relates to a state of varicose (distended or dilated) veins in the oesophagus and upper part of the stomach, which develops as a consequence of portal hypertension. Upper GI tract bleeding may be the risk associated with this state.
  - **Hepatic Coma:** It relates to a state of confusion, apathy, personality

changes, asterixis (tremor of the hands when extended in front of the chest) and spasticity. We shall deal with it in much more details later in this unit.

### Box 15.1 Malnutrition in Liver Disease

Malnutrition is predominant in liver disease and it can be related to a number of factors such as decreased intake of food, impaired digestion and malabsorption, increased energy needs, inefficient protein synthesis, accelerated protein breakdown and increased protein oxidation. These factors are reviewed herewith.

1. Decreased intake of food is generally due to:
  - Anorexia
  - Nausea and vomiting
  - Early satiety
  - Unpalatable diet
  - Drugs used
2. Impaired digestion and absorption could also result due to:
  - P'ancreatic insufficiency
  - Bile salt deficiency
  - Impaired absorption
  - Mucosal defect (portal hypertensive enteropathy), major defect is in fiit digestion and absorptiorl leading to steatorrhoea.
3. Increased energy requirements: the energy needs increase considerably and if not met worsens the condition of liver
4. Insufficient protein synthesis
5. Accelerated protein breakdown
6. Increased protein oxidation

All 4,5 and 6 factors related to protein metabolism, require an increased protein intake and if not met leads to malnutrition.

7. Alcohol consumption replaces food in the diet and supplies empty calories. Alcohol is a source of empty calories (7 Kcal/g) and does not contain any other vital nutrient. The energy supplied also is lost to a great extent due to the wasteful pathways which alcohol metabolism takes. Alcohol causes inflammation of the stomach, pancreas and intestine and interferes with the normal processes of digestion and absorption. It may lead to malabsorption of nutrients like thiamin, vitamin B125 folic acid and vitamin C, Alcohol gets converted to acetaldehyde, which in turn interferes with the activation of vitamins by the liver cells. So numerous dietary nutrients are not synthesized or activated which leads to malnutrition. Alcoholism and malnutritionforms a vicious circle that is difficult to break.

### 15.3.3 Hepatic Encephalopathy (HE) or Hepatic Coma

Hepatic encephalopathy is brain and nervous system damage that occurs as a complication of liver disorders that reduce liver functioning (as in hepatitis or cirrhosis). It is a complex syndrome characterized by neurological disturbances. The symptoms associated with it are: changes in mental state, consciousness, personality and behaviour changes characterized by the following signs —mild confusion, euphoria or depression, decreased attention, slowing of activity to perform mental tasks, irritability, and disorder of sleep pattern, drowsiness, lethargy, speech disorientation, incomprehensive speech and finally coma.

Besides the neurological changes the blood picture shows some abnormalities. These include:

1. Elevated blood ammonia levels (ammonia not converted to urea by liver)
2. High blood concentration of aromatic amino acids (AAA), especially phenylalanine, tyrosine and tryptophan, an increase in methionine, lysine, glutamine, asparagine and histidine, threonine, glycine and serine which are the ammoniogenic amino acids, In other words they can produce ammonia which worsen the condition.
3. Low levels of branched chain amino acid (BCAA), leucine, isoleucine and valine in plasma, due to a depression in the process of gluconeogenesis and ketogenesis — (the processes through which they are used as a source of energy by skeletal muscle, heart and brain).
4. Altered plasma amino acids composition (decreased ratio of BCAA to AAA).

There are four clinical stages of hepatic encephalopathy.

1. **Stage I** — Mild confusion, euphoria or depression, decreased attention, agitation, irritability, sleep disturbance, slowing of ability to perform mental tasks.
2. **Stage II** — Lethargy, disorientation, inappropriate behaviour, irritability in performing mental tasks.
3. **Stage III** — Somnolent but arousable, incomprehensible speech and confused aggressive behaviour when awake.
4. **Stage IV** — Coma.

#### ***Etiology***

The cause of encephalopathy is unknown, but there are three proposed mechanisms leading to it. These include:

1. Accumulation of increased toxins due to impaired liver functions. Excess ammonia being the major toxin.

2. Altered plasma amino acid composition. Decreased ratio of BCAA to AAA, which leads to false neurotransmitter impulses in the brain and hence neurological symptoms.
3. An increase in serum and brain neuro-inhibitory substances, like increased gamma-amino butyric acid (GABA) levels.

Before we move on the management of liver disease, let us review what we have learnt so far.

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## 15.4 NUTRITIONALMANAGEMENT OF LIVER DISEASES

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Having gone through the information presented in the section(s) above, it must be evident that malnutrition is predominant in liver diseases and it can be related to a number of factors such as decreased intake of food, impaired digestion and malabsorption, increased energy needs, inefficientprotein synthesis, accelerated protein breakdownand increased protein oxidation. The diet of the patient must be individualized. The main goals of dietary management for a. liver disease patient should be:

- Maintain adequate nutrition
- Prevent breakdown of body protein tissue
- Control of oederna and ascites, and
- Prevent symptoms of encephalopathy

| Nutrients                  | Hepatitis                                                             | Cirrhosis                                         | Encephalopathy                                       |
|----------------------------|-----------------------------------------------------------------------|---------------------------------------------------|------------------------------------------------------|
| Kilocalories (Kcal)        | 3000 and above                                                        | 2000-3000                                         | 1800                                                 |
| Carbohydrates (g)          | 300-400                                                               | 300-400                                           | 450                                                  |
| Protein (g)                | High 1.5-2 g/ kg<br>(100 g or more)                                   | Moderate<br>0.8-1g/kg (70 g)                      | Low 0.5 g/kg<br>(20-40 g)                            |
| Fat (g)*                   | Moderate 30%<br>total Kcal                                            | Low 25%<br>total Kcals                            | Initially restricted<br>slowly increase to<br>25-30% |
| Vitamins and<br>minerals** | Vitamin B and C,<br>Folic acid, calcium,<br><b>magnesium</b> and zinc | B group and<br>C, Fe if anaemia,<br>Ca, Mg and Zn | Vitamin B and<br>C, Fe if anaemia,<br>Ca, Mg and Zn  |

**Table 15.1: Dietary guidelines for liver patients**

Let us now review the dietary guidelines for each condition one by one. We begin with the dietary recommendations for viral hepatitis.

### 15.4.1 Dietary Recommendations for Viral Hepatitis

An increased carbohydrate, increased protein and moderate fat diet is advised in the case of viral hepatitis with vitamin and mineral supplementation. The requirements for individual nutrients are reviewed further.

## NOTES

**Carbohydrates:** Liberal intake of CHO is advised (300-400 g). This is to prevent endogenous breakdown of protein thus having a protein sparing effect, increase the (intra hepatic) glycogen stores to improve the functioning and protect the liver against infectious agents. The caloric intake advised for adults is 35-40 Kcal/kg IBW or as per the requirement to maintain a desirable body weight.

**Proteins:** Moderate protein intake in the diet is required for the following reasons: to prevent negative N<sub>2</sub> balance, which may lead to hypoproteinaemia, for adequate tissue regeneration especially of parenchymal cells; and prevent fatty infiltration of liver cells. Thus, 1.5 to 2.0 g/kg IBW protein is recommended. Supplements of high protein beverages are recommended in between the meals.

**Fats:** Fats should not be severely restricted as they can make the food unpalatable. About 20% of the total calories should be from MCT are preferred as they are easily digestible and assimilable (40-50 g). For examples dairy fat, cream and butter are preferable.

**Vitamins:** Supplementation of B complex vitamin and C should be given.

**Minerals:** Sodium restriction is required only if there is fluid retention. Potassium supplements are necessary with diuretic therapy. Iron supplementation is needed only if there is anaemia. Keeping these considerations in mind, the food items for a patient with viral hepatitis, showing symptoms of obstructive jaundice have been included in Table 15.2 for your reference.

Table 15.2: Food items for a patient with viral hepatitis or obstructive jaundice

| Freely given foods                                                                     | Foods to be avoided              |
|----------------------------------------------------------------------------------------|----------------------------------|
| Cereals - Bread or chapatties of wheat; rice, maida, suji, maize, jowar, bajra or ragi | Whole pulses (dal) or beans      |
| Breakfast cereal of broken wheat, rice, oatmeal or maize                               | Red Meat, high fat organ meats   |
| Milk or milk products                                                                  | Egg                              |
| Soups                                                                                  | Fried Foods, butter (restricted) |
| Vegetable salad                                                                        | Nuts and oilseeds, dry fruits    |
| Vegetables, cooked                                                                     | Condiments and spices            |
| Potato, sweet potato, or yam                                                           | Papad, chutney or pickles        |
| Fruits, fruit juices                                                                   | Strong tea or coffee             |
| Sugar, jaggery or honey                                                                |                                  |
| Jam or murabba, jellies and other sugar concentrates                                   | Alcoholic beverages              |
| Biscuits                                                                               |                                  |
| Desserts as light custard or ice-cream                                                 |                                  |
| Beverages, water (liberal), glucose water                                              |                                  |

### 15.4.2 Dietary Recommendations for Liver Cirrhosis

A high carbohydrate, moderate protein and low fat diet is advised to a patient with liver cirrhosis along with vitamin and mineral supplementation. Since anorexia is at its peak, the food should be given in several feedings with moderate portions (6-8

feedings). The diet needs progression from liquid, soft to normal diet, depending on the acute stage and recovery. Judicious use of spices and condiments to stimulate appetite is needed. High calorie and protein beverages are useful in between meals. In case of complications of encephalopathy low protein diet needs to be given. Fat also needs to be less and gradually increased as the subject improves. Nutrition care should maintain or improve the nutritional status. Individualized diets must be given depending on the degree of malnutrition and tolerance level of the patient. Counseling the patient on diet and food choices is helpful.

**Proteins:** Intake to be adjusted as per the individual requirement, depending on the pathological state. A protein intake high enough to maintain nitrogen balance and low enough to prevent hepatic coma in the initial stages is recommended. In uncomplicated hepatitis or cirrhosis without encephalopathy, a protein requirement 1 g/kg of dry weight/ day to achieve nitrogen balance is advised. To promote nitrogen accumulation or positive balance, at least 1.5 to 1.2 g/kg daily is needed. Protein intake is restricted to 0.5 g/day if there are signs of impending coma. In situations of stress such as alcoholic hepatitis or sepsis; infection, GI bleeding, severe ascites, at least 1.5 g of protein /kg/day should be provided.

**Carbohydrates:** To ascertain the carbohydrate need is challenging because of deteriorating functional state of liver, which favours preference for alternative fuels. Depending on the state of the liver, the carbohydrate content is kept adequate (300-400 g/day) for its protein sparing effect. It protects and supports the liver function. Adequacy of calories for maintaining weight needs to be emphasized. Emphasis should also be on improving the total intake of the patient. An intake of 25 to 35 Kcal/ kg estimated dry body weight should be used in calculations to prevent overfeeding. Intravenous glucose administration must be done only if there is severe nausea and vomiting.

**Fats:** Steatorrhea or fatty infiltration of liver may be seen in a cirrhosis patient, thus a moderate intake with the substitution of medium chain triglycerides (MCTs) may prove to be effective in reducing malabsorption of fat.

**Vitamins:** Supplementation with vitamins is desired to replenish liver stores and repair tissue damage especially if the patient has anorexia. This is due to the intimate role of liver in nutrient transport, storage and metabolism, in addition to the side effects of the drugs used. The vitamins of importance are water-soluble vitamins — pyridoxine, cyanocobalamin, folate, niacin and thiamin associated especially with alcoholic liver disease leading to Wernicke's encephalopathy. Deficiency of fat-soluble vitamin has been observed due to malabsorption and decreased storage capacity of diseased liver, Therefore supplementation is necessary using water-soluble forms.

**Minerals:** Calcium, Magnesium and Zinc are the important minerals as the serum levels tend to decrease in cirrhotics due to malabsorption associated with steatorrhea. Thus adequate doses as per the requirements should be

**NOTES**

supplemented. Sodium: Restriction of sodium is essential if oedema and ascites are present. Sodium restriction up to 500 mg/day is seen with ascites but generally relaxed to 2 g/day with diuretics. An extremely low sodium diet can affect the palatability, as well as, increase risk to hyponatremia,. Emphasis must be on low sodium foods and avoidance of table salt or salt in food preparation. Also protein intake must be adequate without increasing sodium intake. Serum sodium and potassium levels need to be closely monitored.

**Fluids:** These may need not be severely limited if sodium restriction is effective in correcting oedema and ascites. No more than 1500 ml of fluid/day is given. Fluid requirement is generally worked out as per the previous day urinary output coupled with the insensible losses (perspiration, breath, feces etc) which normally amounts to 500 ml/day as well as loses due to diarrhoea or vomiting (if any).

**Fibre:** Reduction in fibre content is necessary in advanced cirrhosis to prevent danger of haemorrhage from oesophageal varices. Hence, liquid and soft diet and small meals are emphasized. A sample menu for a cirrhotic patient providing roughly 2000 Kcal and 60 g proteins is given.

**Table 15.3: Permitted and excluded foods**

| <b>Permitted Foods</b>                                                                                             | <b>Excluded Foods</b>                                                                                     |
|--------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------|
| Bread (wheat), rice, maize, jowar, bajra, breakfast cereals, pasta and other refined cereals like maida, suji etc. | Fried foods                                                                                               |
| Toned milk and its products like paneer, curd etc.                                                                 | Organ meat, egg yolk                                                                                      |
| Washed and split pulses and beans                                                                                  | Whole pulses and fibre rich cereals like oats, barley                                                     |
| Sugar, jaggery, honey, jam or murabba,, jellies                                                                    | Extra salt and baking soda, preserved foods and foods containing salt like papads, chutneys, pickles etc. |
| Lean meat, egg white, fish or chicken                                                                              |                                                                                                           |
| Fat or butter, cream                                                                                               |                                                                                                           |
| Potato, sweet potato or yam                                                                                        |                                                                                                           |
| Pastries, dessert, sweetmeat                                                                                       |                                                                                                           |
| Beverages, lemonade, fruit juices                                                                                  |                                                                                                           |

For cirrhotic patients with Wilson disease, binding agents also known as chelating agents are often used for removing copper. Also a vegetarian diet which is low in copper is recommended. Inclusion of foods low in copper will help prevent symptoms



of Wilson disease.

**Table 15.4: Copper content of foods**

| Foods <b>High</b> in Copper (to be avoided)              | Foods <b>Low</b> in Copper ( <i>desirable</i> )          |
|----------------------------------------------------------|----------------------------------------------------------|
| Meat: organ meats, lamb, pork, fish, liver and shellfish | Eggs                                                     |
| Milk: chocolate, cocoa and soyamilk, tofu                | All other dairy products                                 |
| Pulses: Beans Peas and lentils                           | Bread and pasta from refined flour, rice, sweet potatoes |
| Cereals: Bran containing cereals, soyafLOUR              | All other vegetables including tomatoes                  |
| Vegetables: Mushrooms and juice                          | All other fruits (including jams, jellies)               |
| Fruits: Dried fruits, dates, raisins and prunes          | Lemonade and fruit flavoured beverages                   |
| Brewer's yeast                                           |                                                          |

### 15.4.3 Dietary Recommendations for Hepatic Encephalopathy (HE) or Hepatic Coma

The nutritional management goals for hepatic coma include:

- reduction in protein intake to a minimum in order to decrease amount of ammonia produced,
- correcting plasma amino acid profile, and
- prevention of catabolism of tissue protein.

There is no general treatment. The diet needs to be individualized. The dietary recommendations include:

**Calories:** A 1500 to 2000 Kcal diet is recommended to prevent breakdown of tissue protein for energy. It is provided chiefly in the form of carbohydrates. It can be given by parenteral or tube feeding if needed. Carbohydrates help to build up liver glycogen reserves and have a protective role in the healing process.

**Carbohydrates:** An increase in carbohydrates in the diet is recommended because it is the main source of energy and thus spares the protein. It promotes glycogen repletion, which improves with carbohydrate adequacy. It also prevents hypoglycemia.

**Proteins:** It has not yet been proved that severe protein restriction improves the mental state of the patient in hepatic encephalopathy. Unnecessary protein restriction may only worsen body protein losses and therefore must be avoided. More than 95% of cirrhotic patients can tolerate mixed protein diets. The protein intake may begin with 0.2 g/ kg IBW /day. If the patient remains asymptomatic for a week it may gradually be increased by 10-15 g per week, and then 20-40 g and gradually to 0.5 g/kg IBW per day as indicated in Table 15.1 above,

Research postulates that vegetable proteins and caesin may improve mental status compared to animal protein. Vegetable based diets are lower in AAAs and

higher in BCAAs than meat based diets. The potential advantage of vegetable protein is that it is low in methionine and ammoniogenic amino acids. The BCAA are desirable supplements in liver disease.

## NOTES

These amino acids are metabolized by the muscles independent of the liver to provide energy, other amino-acids or small nitrogenous compounds and help in obtaining a positive nitrogen balance. Vegetable proteins are rich in BCAA. The common food sources rich in BCAA include dairy products and red meat. Whey protein and egg protein supplements are among the other sources. Experts agree that BCAA enriched formulas should be indicated for patients with encephalopathy who do not tolerate standard proteins. Both enteral and parenteral BCAA supplement formulas are available commercially and could be used, if required.

An increase in BCAA helps in a number of ways. These include: enhances the uptake of AAA by muscles. increases protein synthesis in muscles. increases hepatic protein synthesis. reduces the cerebral AAA levels by competing for a common transport system across the blood brain barrier.

**Fats:** Fats require restriction, as diseased liver cannot metabolize fats. Substitution with MCT is recommended as they do not require bile salts and micelle formation for absorption and are readily taken up by the portal route.

**Vitamins:** Increase in intake of B-complex vitamins such as folate, thiamin, B<sub>12</sub> and vitamin C is recommended as these vitamins act as coenzymes in various metabolic reactions.

**Sodium:** Depending on the state of the patient, a restriction of 2 g/day along with use of diuretics is recommended.

**Fluid:** Hyperaldosteronism is associated with liver failure, which results in increased renal sodium exchange for potassium. This urinary potassium loss further gets aggravated by diuretic therapy. There is an evident fluid retention. Thus, depending on the patients' state of hydration, urine output, presence of oedema and diuretic therapy, the fluid intake should be decided and recommended.

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## 15.5 GALLBLADDER AND BILIARY TRACT DISEASES

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Gall Bladder, is an organ attached to the right side of the undersurface of the liver. Its main function is to concentrate and store the bile formed in the liver until the body needs to digest fat. At that time, the gall bladder contracts and pushes the bile into a tube, called the common bile duct that carries it to the small intestine, where it helps with digestion.

Bile contains water, cholesterol, fats, bile salts, proteins, and bilirubin. Bile salts break up fat, and bilirubin gives bile and stool a yellowish colour. If the liquid bile contains too much cholesterol, bile salts, or bilirubin, under certain

conditions it can harden into stones, The two types of gallstones are cholesterol stones and pigment stones. Cholesterol stones are usually yellow-green and are made primarily of hardened cholesterol. They account for about 80 percent of gallstones. Pigment stones are small, dark stones made of bilirubin.

The disorders of the biliary tract and gall bladder are closely associated with liver disorders. The common diseases of the biliary tract are cholelithiasis, choledocholithiasis and cholecystitis. Cholelithiasis: it is the formation of gallstones in the absence of infection of the gall bladder. These may cause no symptoms and the patient might be unaware of their presence.

**Choledocholithiasis:** when stones slip into the bile duct producing obstruction, pain and cramps, it is referred to as choledocholithiasis.

**Cholecystitis:** inflammation of gall bladder. It is usually caused by, gallstones obstructing the bile ducts causing a backflow of bile. The walls of the gall bladder become inflamed and distended and infection can occur. During such episodes, the patient experiences upper quadrant abdominal pain accompanied by nausea, vomiting and flatulence. Jaundice can also occur during this disease.

Cholecystitis can be either acute or chronic.

1. Acute: This can occur without stones mainly in critically ill patients or when the gall bladder and its bile are stagnant.
2. Chronic: This appears to be due to diminished spontaneous contractile activity of the gall bladder and decreased contractile responsiveness to the hormone cholecystokinin.

The risk factors associated with gall stone formation, particularly the cholesterol stones.

These can be listed as:

- Female Gender
- Pregnancy
- Older Age
- Family History
- Obesity
- Trunkal body fat distribution
- Diabetes Mellitus
- Inflammatory Bowel Disease (IBD)
- Drugs like lipid lowering medications, oral contraceptives
- Rapid weight loss through severe calorie restriction— biliary sludge
- Im grade chronic infections
- High dietary fat intake

Other factors include sickle cell anaemia, thalassemia, biliary tract infection, cirrhosis, alcoholism and long term parenteral nutrition.

### ***Treatment of gallstones***

Removal of gall bladder is called cholecystectomy. This can be done by the traditional laparotomy or non-invasive laparoscopic procedure. Conservative treatment is by using chemicals to dissolve the stones. Currently the shock wave lithotripsy is also being used extensively. After the removal of the gall bladder, the biliary tract dilates forming a simulated pouch over time to allow bile to be held in a manner similar to the original gall bladder.

### ***Nutritional Management***

The dietary considerations for management of gall bladder stones are enumerated herewith:

- The main aim of the treatment is to reduce discomfort by providing a diet restricted in fat. In an acute condition it is advisable to keep the gall bladder at rest and minimize contractions by excluding fat from the diet. In an acute attack 'nil by mouth diet', followed by an extremely low fat intake to prevent gall bladder stimulation is recommended. Fat content is gradually increased by providing 2 hourly liquid diet for a few days. In chronic phase 20-30 g of fat may be provided and later increased gradually to 40-45 g thus increasing the palatability of the diet. After the symptoms settle clear soups, toned milk, refined cereals could be given. The patient is put on a 30-45 g of fat intake per day, which can be achieved by incorporating a variety of low fat options.
- A calorie-restricted diet with a restriction on the intake of refined carbohydrates is beneficial. Refined sugars increase cholesterol saturation and lithogenicity of the bile. Control on fat intake also contributes to weight loss due to calorie restriction,
- A moderate fat restriction can be considered. Around 25% of total calories can come from fat with a prudent usage of Medium Chain Triglycerides and good quality fat (high MUFA and low N6: N3 ratio) from invisible sources. Use of MUFAs has shown a powerful effect on gall bladder emptying. This is because monounsaturated fats increase the ratio of HDL cholesterol to LDL cholesterol and it may therefore provide important protection against gallstone formation.
- An appropriate selection of low fat options like toned/ skim milk, paneer, low fat meat options, fish and egg in restricted quantity may help in getting the good quality proteins. Studies reveal that animal proteins in the diets are more lithogenic (stone forming) as compared to vegetable proteins.
- A high intake of soluble fibre through pulses/ sprouts/ dals, vegetables, fruits and oilseeds (flax seeds and methi seeds) can help the body to get rid of bile acid sterols out of the gut. This also gives a desirable micro flora to favour inhibiting endogenous production of cholesterol. It also provides less time for colonic bacteria to produce secondary bile acids like deoxycholic acid from cholic acid, hence less deoxycholic acid is absorbed.

## **NOTES**

- The degree of food intolerance needs to be individualized, as it may vary' from patient to patient.
- The distribution of meals throughout the day may be of use to give symptomatic relief and better tolerance to food.
- Administration of water-soluble forms of fat-soluble vitamins may be of benefit in chronic gall bladder disease patients, as fat malabsorption is suspected. Vitamin C is beneficial as it decreases the incidence of cholelithiasis.
- Coffee consumption has shown to be having a protective effect on gallstones formation as coffee and various constituents of coffee affect various metabolic processes that are involved in cholesterol lithogenicity. Coffee stimulates cholecystokinin release, increases gall bladder motility and possibly enhances large bowel motility. Caffeine inhibits biliary cholesterol crystallization, decreases gall bladder fluid absorption and increases hepatic bile flow.

**Table 15.5: Food items for a patient with gall bladder disease**

| Permitted                                                   | Excluded                                    |
|-------------------------------------------------------------|---------------------------------------------|
| Bread or chapatties from wheat, maize, jowar, bajra or ragi | Fried foods                                 |
| Breakfast cereal of wheat, rice, oatmeal or maize           | Fatty meats                                 |
| Rice, cooked                                                | Fruits, dried                               |
| Pulses (dal) or beans as thin dal, 1 cup                    | Nuts                                        |
| Fish or chicken                                             | Sweets or sweetmeats                        |
| Egg (if no discomfort)                                      | Condiments and spices                       |
| Milk or milk products (with cream removed)                  | Papad, chutney or pickles                   |
| Soup (thin soups)                                           | Thick soups and gravies<br>whole milk/khoya |
| Vegetable salad                                             |                                             |
| Vegetables, cooked                                          |                                             |
| Potato, sweet potato, or yam                                |                                             |
| Fat for cooking or butter if no symptoms                    |                                             |
| Sugar, jaggery or honey                                     |                                             |
| Fruits, fresh                                               |                                             |
| Fluid liberal                                               |                                             |
| Jam or murabba                                              |                                             |
| Pastry only as biscuits or light cakes                      |                                             |
| Desserts as light custard, jelly ice-cream                  |                                             |
| Beverages                                                   |                                             |

## 15.6 PANCREATIC DISEASES

## NOTES

The pancreas is located deep in the upper abdomen behind the stomach.

The main functions of pancreas include insulin manufacture and secretion of enzymes that participate in the digestion Of various nutrients. One of the conditions linked to the abnormal functioning of pancreas is pancreatitis.

### ***What is pancreatitis ?***

Pancreatitis relates to the inflammation of the pancreas and is characterized by oedema, cellular exudates and fat necrosis. The disease can range from mild and self-limiting to severe condition which results in auto digestion, necrosis and haemorrhage of pancreatic tissue. Pancreatitis can be classified as acute and chronic.

Bile, we learnt above, is synthesized by the liver, stored in the gall bladder and secreted in the intestine. It has a distinct role to play in fat metabolism and decreased production and secretion in the intestine can affect fat digestion and lead to fat malabsorption. A building up of back up bile pressure due to renal or post renal causes can lead to precipitation of jaundice and can be a cause of secondary biliary cirrhosis, cholelithiasis. Obstruction of distal common bile duct can lead to pancreatitis if the pancreatic duct is blocked, Thus, liver, gall bladder and pancreatic diseases can have an overlapping etiology.

### **15.6.1 Acute Pancreatitis**

The pancreas is protected against its own enzyme by their synthesis as proenzymes. Acute pancreatitis develops when activated pancreatic enzymes (activated prematurely in pancreas) are liberated within the pancreatic system. The clinical feature of pancreatitis results from auto digestion of tissue and toxic effects of digestion products. Elevated serum and urinary amylase concentration due to enzyme released by necrosed pancreatic cells remain one of the main diagnostic criteria in acute pancreatitis. The severity of pancreatitis can be seen by the Ranson's criteria to classify pancreatitis.

### ***Etiology***

The etiological factors involved in the disease are biliary tract disease, such as gallstones, alcohol abuse, trauma and hyperlipidemia (rarely).

### ***Symptoms***

- The symptoms of pancreatitis include:
- Continuous or intermittent pain of varying intensity in the upper abdominal region that radiates to the back. Symptoms worsen with ingestion of food.
- Swollen and tender abdomen.
- Nausea and vomiting—precipitated by large meal and alcohol consumption.
- Steatorrhea and malabsorption.

- Sweating, fever, mild jaundice and rapid pulse are also seen.

## ***Complications***

Some of its complications include low blood pressure, heart failure, kidney failure, diabetes, ascites and cysts in pancreas.

## ***Nutritional Management***

Acute pancreatitis often results in a catabolic state characterized by profound haemodynamic, metabolic, cardiovascular, pulmonary, haematological and renal aberrations. Parenteral nutrition and metabolic support becomes essential in order to minimize mortality. Since TPN feeding needs specialized set ups, increased costs and long term maintenance, it is difficult for patients to afford the treatment. Hence, enteral nutrition is a preferred mode. This type of feeding is safe, well tolerated with less infections/non-infectious complications. The nutritional management goals of acute pancreatitis include:

1. Conservative management involves resting pancreas and maintaining fluid balance.
2. Nil by mouth till the pain and fever subsides; as the oral intake further aggravates the symptoms caused by an increased secretory mechanism of pancreatic enzyme and bile.
3. The patient needs to be supported by early enteral nutrition with the formulation of nutrient in predigested forms and supplementation with low fat intake to prevent further precipitation of malnutrition. Sometimes TPN is required.
4. When oral feeding is resorted it should be a clear liquid diet with a waiting period to see the response of the patient in terms of undesirable symptoms.
5. A low fat diet with an intake of total fat as 30 g/day, which may gradually be increased as per the patient's tolerance. MCT may be incorporated for better digestibility and assimilation, as they do not require the pancreatic enzyme system for the same. They also help in increasing the total caloric intake of the patient.
6. A careful monitoring of all the biochemical parameters starting from enzymic assay to serum albumin concentration should be done on regular basis.
7. Decreased calcium levels are observed during acute pancreatitis. This can be due to (i) hypoalbuminemia (as calcium is bound to protein), and (ii) soap formation of calcium with fatty acids created by fat necrosis. Hence calcium supplementation may be required.

Next, we move on to chronic pancreatitis.

## NOTES

### 15.6.2 Chronic Pancreatitis

Chronic pancreatitis mainly results following the repeated attacks of acute pancreatitis or the effect of digestive enzymes on pancreas or may be associated with chronic inflammation of the biliary tract. There is also a strong relationship of alcohol abuse and development of chronic pancreatitis as it acts as an intestinal irritant and leads to reoccurrences.

What causes this chronic condition? Let us find out.

#### ***Etiology***

The etiological factors include:

- Neglect of acute pancreatitis
- Alcohol abuse
- Excessive iron in the blood
- Unknown factors

The symptoms of this acute condition are highlighted next.

#### ***Symptoms***

Common symptoms include:

1. Pain
2. Malabsorption
3. Weight loss
4. Malnutrition (could be due to alcohol abuse).
5. Steatorrhoea

Finally let us get to know about the nutritional management of chronic pancreatitis.

#### ***Nutritional Management of Chronic Pancreatitis***

The nutritional management goals for chronic pancreatitis include:

- Rest to pancreas
- Prevention of diabetes
- Enteral supplementation
- Diet control and special feeding

The nutritional management ranges from fundamental dietary modifications to administration of appropriate digestive enzymes to enteral supplementation. It however, depends on the stage, severity and manifestations of pancreatitis. Pancreatic enzyme supplementation is important in long term patient management and it helps to control and reduce malabsorption.



At times appropriate digestive enzymes help and other times it needs special feeding methods to be employed especially enteral feeding. In chronic cases with extensive pancreatic destruction, the insulin secretory capacity of the pancreas decrease and glucose intolerance develops. Treatment with insulin and nutritional care is similar to diabetes.

The dietary guidelines include:

- A caloric intake of 35 Kcal/kg IBW is ascertained keeping in mind the moderate stressful state. The patient needs to be kept on a low fat diet (40 to 60 g/day), the levels are ascertained with the tolerance of the patient.
- Calcium and vitamin B<sub>12</sub> are important as deficiency of pancreatic protease may prevent cleaving of vitamin B<sub>12</sub> from its carrier protein thereby leading to vitamin B<sub>12</sub> deficiency.
- Fat-soluble vitamin malabsorption may occur. Thus, parenteral administration of fat-soluble vitamin is necessary.

Some important guidelines for managing chronic pancreatitis. These include:

- Avoid alcohol
- Avoid meal with a high fat content. Use MCT's in severe steatorrhea
- Give adequate energy and protein intake
- Monitor blood glucose levels regularly
- Give vitamin and mineral supplementation (fat soluble vitamins, folic acid and calcium)
- Pancreatic enzyme supplementation should be taken adjusted to quantity of food and fat content of meals)

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## 15.7 LET US SUM UP

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In this unit, we learnt about the important liver, pancreatic and gall bladder disorders such as hepatitis, cirrhosis, pancreatitis, gall bladder disorders (stones), hepatic encephalopathy or coma. To have a better understanding of the diseases involved, we first reviewed our knowledge about the functions of these organs. Next, we studied about these disorders separately in a greater detail, discussing their etiology, symptoms, associated complications and clinical manifestations. The nutritional aspects of these disorders and their corresponding dietary management were also emphasized. The discussion also included the types of foods to be included, as well as, excluded, a few dietary tips to remember and some sample diets.

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## 15.8 GLOSSARY

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## NOTES

|                                |                                                                                                                                                                                                                                                        |
|--------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Atresia</b>                 | : inadequate development of an organ or a part of organ during pregnancy.                                                                                                                                                                              |
| <b>Cholangitis</b>             | : an inflammation of the bile duct; can be acute as an outcome of infection or liver failure.                                                                                                                                                          |
| <b>Cholestasis</b>             | : a condition of sludge like buildup in the gall bladder as a result of lack of stimulation or release of bile.                                                                                                                                        |
| <b>Chorea</b>                  | : rapid jerky, dance-like movements of the body especially extremities and head.                                                                                                                                                                       |
| <b>Cystic fibrosis</b>         | : an inherited autosomal recessive condition that causes the secretion of abnormal mucus in the lungs and problems with pancreas function and food absorption.                                                                                         |
| <b>Dystonic facies</b>         | : the appearance or expression of the face, especially when typical of a certain disorder or disease.                                                                                                                                                  |
| <b>Fecal-oral route</b>        | : Many diseases can be passed when the stool (or remnants thereof) of one host ends up in someone else's mouth. This is referred to as the fecal-oral route (or alternately, the oral-fecal route or oro-fecal route).                                 |
| <b>Fatty Liver</b>             | : a condition characterized by the accumulation of excess fat in the liver commonly caused by alcohol abuse but also associated with obesity, starvation, intestinal bypass etc.                                                                       |
| <b>Fulminate liver disease</b> | : absence of pre-existing liver disease with development of hepatic encephalopathy within 2 months of onset of illness.                                                                                                                                |
| <b>Hemochromatosis</b>         | : a disorder of iron metabolism characterized by excessive absorption of ingested iron, saturation of iron-binding protein and deposition of haemosiderin in tissue, liver cirrhosis, diabetes, pigmentation of the skin and eventually heart failure. |

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## 15.9 CHECK YOUR PROGRESS

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1) List the various liver functions and briefly give the role of liver in fat metabolism.

2) What do you understand by the term 'liver cirrhosis'? Enumerate the various etiological factors involved in its pathogenesis.

3) Discuss the pathogenesis and major complications of alcoholic liver disease.

Nutritional  
Management  
In Liver, Gall  
Bladder And  
Pancreatic  
Disease  
**NOTES**

## **NUTRITIONAL MANAGEMENT OF RENAL DISEASES**

### **STRUCTURE**

- 16.1 Learning Objective
- 16.2 Introduction
- 16.3 Physiology of the Kidney
- 16.4 Assessment of Kidney Function: Diagnostic Tests
- 16.5 Common Renal Diseases
- 16.6 General Principle of Dietary Management in Renal Diseases
- 16.7 Acute and Chronic Nephritis
- 16.8 Nephrotic Syndrome
- 16.9 Acute Renal Failure (ARF)
- 16.10 Chronic Renal Failure (CRF)
- 16.11 End Stage Renal Disease, (ESRD)
- 16.12 Renal Calculi
- 16.13 Commonly Available Commercial Enteral Nutrition Formulas for Renal Patients
- 16.14 Let Us Sum Up
- 16.15 Glossary
- 16.16 Check Your Progress

### **16.1 LEARNING OBJECTIVE**

After studying this unit, you will be able to:

- recapitulate and describe the physiology of the kidneys,
- discuss the renal function and diagnostic tests,
- identify different renal disorders, their etiology, clinical and metabolic manifestation, and
- rationalize the dietary modifications in renal disorders, especially proteins, minerals and fluids.

## 16.2 INTRODUCTION

In our previous units, we discussed about gastrointestinal tract disorders and their nutritional management. In this unit, we shall study about one of the most prevalent disorders, that is, renal disorders.

Next, in this unit, we will learn about various kidney function tests that involve both biochemical analysis and clinical examination. Later on, we shall get to know about renal diseases such as nephritis, nephrotic syndrome, acute renal failure (ARF), chronic renal failure (CRF), end stage renal disease (ESRD) etc. along with their dietary management. This would include the list of foods to be excluded dming these diseases.

## 16.3 PHYSIOLOGY OF THE KIDNEY

The kidneys are a major organ for excretion of metabolic wastes from the body. Through glomerular filtration, tubular secretion and selective reabsorption that takes place in the 'nephrons' (the functional units of the kidneys), the kidneys maintain the intemal balance of body fluids. This includes normal osmotic pressure, composition and volume of blood, acid base balance and fluid electrolyte balance. Let us elaboratea bit on the kidney ftmctions.

### 16.3.1 Kidney Functions

Figure 16.1 highlights the kidney functions. The primary functions of the kidney include:

- Excretion
- Endocrine
- Metabolic

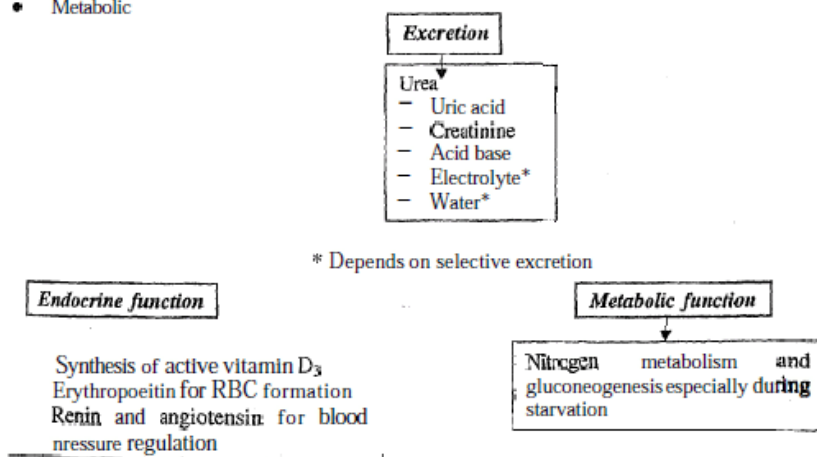


Figure 16.1 : Functions of the kidney

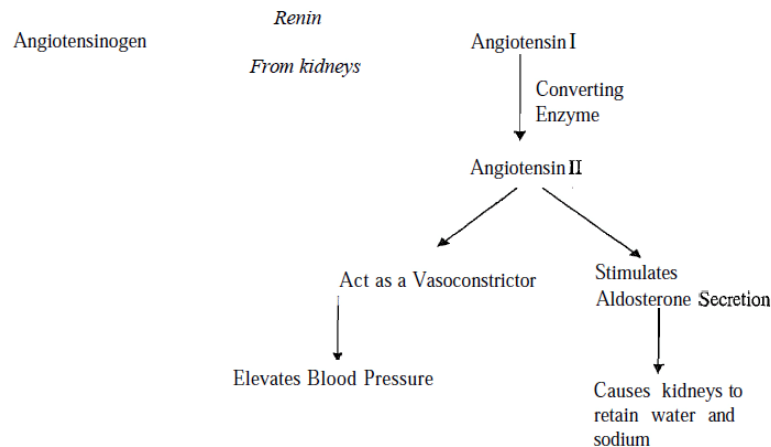
You may recall that we studied about the contribution of the kidneys in urine fomition and its role in maintaining the pH and performing other regulatory functions in the body.

## NOTES

### A. Endocrine Functions

The endocrine functions of kidneys include:

- Synthesis of active vitamin D: Kidneys maintain calcium-phosphorus homeostasis, which involves the complex interactions of PTH, calcitonin, vitamin D and three effector organs — gut, kidney and bone. Kidneys produce active vitamin D, as well as, eliminate both calcium and phosphorus. Vitamin D you might be aware promotes efficient absorption of calcium (Ca) and is one of the substances necessary for bone remodeling and maintenance.
- Erythropoietin for RBC formation: Erythropoietin, a critical determinant in RBC formation is produced by the kidneys.
- Renin and angiotensin for regulation of blood pressure: A lowered blood volume causes the cells of the glomerulus to react by secreting renin — a proteolytic enzyme. Renin acts in the plasma to form angiotensin I, which is converted to angiotensin II (a powerful vasoconstrictor) and a potent stimulus of aldosterone secretion by the adrenal gland. As a consequence, sodium is reabsorbed and blood pressure returns to normal.



**Figure 16.2: Renin-angiotensin aldosterone mechanism**

### B. Metabolic Function

The main metabolic role of kidney is linked to nitrogen metabolism. However, research indicates that kidneys may also play an important role in glucose counter regulation. The main purpose of gluconeogenesis, as you may already be aware, is to synthesize glucose from non-carbohydrate precursors. This process occurs mainly in the liver and kidney. The role of kidney in eliminating the end products of protein metabolism is highlighted next.

**Nitrogen Metabolism:** The majority of solute load consists of nitrogenous wastes, which are the end products of protein metabolism. These include urea, uric acid, creatinine and ammonia.

In case of inappropriate elimination of these waste products, they get collected in abnormal quantities in blood. The inability of the kidneys to excrete these

nitrogen wastes leads to renal failure.

### ***Important Outcomes of Functions***

It will be useful to recall, from Unit 7 in the Applied Physiology Course some of the important outcomes of kidney functions. These include:

- The kidneys have a large blood flow.
- About 180 L ultra filtrate is formed at the glomeruli / day but daily urine output is only 1.2-1.5 L/day.
- For greater solute load, a greater volume of fluid and urine output is required.
- Healthy kidneys have a capacity to produce urine with wide variability of volume, osmolality and pH.
- Healthy kidneys can regulate one substance at the expense of another, based on the need.
- In this context, control of blood volume has priority over other parameters.
- Healthy kidneys have considerable reserve capacity and health can be maintained even on half a functional kidney.
- With progressive impairment (when more than 60% nephrons are damaged), the urine output falls and products of metabolism start accumulating in the body. Gradually metabolic and endocrine functions also get impaired and renal failure can develop.

With this knowledge, we will be able to understand the diagnostic tests for kidney function and various kidney disorders, in relation to treatment. Let us then move on to learn about the methods/techniques used to assess kidney functions,

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## **16.4 ASSESSMENT OF FUNCTION: DIAGNOSTIC TESTS**

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The kidney functions can be assessed through various biochemical tests and clinical examination. These are described in this section.

### **Biochemical Tests**

The kidney function can be assessed through various biochemical tests. These mostly measure glomerular and / or tubular function of the kidneys. The functional capacity of the nephrons reduces in kidney disorders, changes occur in the blood and urine chemistry.

Common diagnostic tests to assess renal function include measurement of:

1. Plasma /serum concentration urea, creatinine and uric acid: Levels of these tend to increase in blood due to inadequate excretion in impaired kidney function.

## **NOTES**

**NOTES**

2. Serum electrolytes like sodium, potassium, chloride, inorganic phosphorus, and bicarbonate: Their levels may increase or decrease depending on the kidney disorder.
3. Glomerular Filtration Rate (GFR) is measured by the creatinine clearance: Clearance tests are a measure of the efficiency with which kidneys remove a substance from the blood. Normal creatinine clearance in adults is about 125 ml/ minute, which can be reduced to 30 ml/minute or less when kidneys fail.
4. Alkaline phosphatase in blood: This can be altered as vitamin D synthesis is affected in impaired kidney function.
5. Blood Haemoglobin: Its levels are dependent on erythropoietin synthesis.
6. Urine Volume and Extra Cellular Fluid (ECF) : Increase or decrease in urine volume and volume of body, based on excretory capacity.
7. Urine examination: Urine analysis for clarity, colour, microscopic examination, pH, specific gravity, osmolality and presence of abnormal constituents like blood, protein, casts etc.
8. Urine, electrolytes: Urine electrolytes like sodium, potassium, magnesium especially in tubular disorders, where excess loss may occur.

**Table 16.1: Normal blood parameters for diagnosis in kidney diseases.**

| Blood Parameters                 | Normal Range          |
|----------------------------------|-----------------------|
| <b>BUN (Blood Urea Nitrogen)</b> | <b>7-22 mg %</b>      |
| Creatinine                       | 0.8-1.8 mg%           |
| Uric acid                        | 2.4-7.0 mg %          |
| Sodium                           | 132-148 mEq/L         |
| Potassium                        | 3.5-5.5 mEq/L         |
| <b>Phosphorus</b>                | <b>3.0-4.5 mg %</b>   |
| Calcium                          | 8.1-10.4 mg %         |
| Chloride                         | 98-108 mEq/L          |
| Total protein                    | 6.6-8.7 gm %          |
| Albumin                          | 3.8-4.4 gm %          |
| Globulin                         | 1.8-3.6 gm %          |
| A/G ratio                        | 2:1                   |
| Alkaline phosphatase             | 30-150 unit per litre |
| Total cholesterol                | 150-250 mg %          |



Next let us review the normal urine parameters which are listed in Table 16.2.

**Table 16.2: Normal urine parameters Nutritional**

|                     |                                                                |
|---------------------|----------------------------------------------------------------|
| Volume              | 800-2000 ml/day                                                |
| Glucose             | 0-0.8 mmol/L(0-15 mg/dL)                                       |
| Protein             | Less than 150 mg/day                                           |
| Ketones             | negative is normal                                             |
| Presence of acetone | Small: < 20 mg/dl<br>Moderate 30-40 mg/dl<br>Large: > 80 mg/dl |
| Urine Electrolytes: |                                                                |
| Chloride            | 110-250 mEq                                                    |
| Sodium              | 15-250 mEq/L                                                   |
| Potassium           | 25-120mEq/L                                                    |
| Magnesium           | Less than 150 mg/day                                           |
| Calcium             | 100-300 mg                                                     |
| Creatinine          | 1.1-3.4 g                                                      |
| Creatine            | Little or none                                                 |
| Specific gravity    | 1.015-1.020                                                    |
| pH                  | 6.0                                                            |

**NOTES**

Abnormalities in the parameters listed above are an indication of kidney disorders.

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## 16.5 COMMON RENAL DISEASES

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Kidney disorders may be infective, inflammatory in origin, or degenerative in nature. With an increasing severity or long duration, these can result in renal failure. Diseases of the kidney may involve the nephrons, tubules or glomerulus. Inflammation of the nephrons is termed nephritis. Glomerulonephritis (GN) refers to involvement of specifically the glomeruli. With glomerular damage, usually tubules also get affected. Stones formation can also take place in the kidneys, this is known as renal calculi or nephrolithiasis. Degenerative or vascular disorders can lead to nephrosclerosis, where blood vessels of the kidneys become narrow. This leads to reduced blood and oxygen supply, and hence kidney damage.

Kidney diseases may be acute or chronic and have several underlying causes. The treatment is dependent on the disease origin, extent and type of damage and clinical and metabolic outcomes. As kidneys have a direct effect on nutritional status via homeostasis of fluid, electrolytes and nutrients in the body, diet plays an important role in treatment. Some of the common renal disorders are:

- Acute and chronic nephritis
- Nephrotic syndrome
- Acute renal failure

- Dialysis
- Renal transplantation
- Renal calculi

## NOTES

The etiological factors for each of these disorders are discussed later in this unit under each renal disorder covered separately. You will realize that the management of these disorders also varies, however, there are a few common aspects linked with the dietary management in renal diseases. These general principles are described here next, before we take up each disease separately.

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### 16.6 GENERAL PRINCIPLES OF DIETARY MANAGEMENT IN RENAL DISEASES

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Several common principles apply to the dietary management of various renal diseases. This is because various symptoms, clinical and biochemical manifestations may be common in different renal disorders as you will find out soon. The main objectives of dietary management in renal diseases are to:

- reduce the excretory work of the kidneys, while maintaining as near normal fluid, acid base and electrolyte balance.
- maintain satisfactory nutritional status.
- prevent progression of renal damage and development of uremia (accumulation of nitrogenous waste products in blood).

To meet these objectives, modifications in the diet are required mainly for

- a) Proteins
- b) Electrolytes, especially sodium and potassium
- c) Fluids

Let us now understand the general dietary principles.

**Energy:** It should be normal or increased in case of weight loss. A simple diet low in proteins, moderate to high in carbohydrates and unsaturated fats is preferred.

**Protein:** The amount of protein to be given is dependent on GFR. Proteins must be enough to prevent muscle wasting and malnutrition, but it needs adjustment to prevent accumulation of nitrogenous wastes in the blood and uremia. Good quality proteins from milk and eggs are recommended to meet the body's need of essential amino acids and yet not burden the body with excess non essential amino acids.

#### ***Electrolytes:***

1. Sodium intake mostly needs restriction depending on whether it is retained in the body or excessive loss in urine occurs. Retained sodium and water can result in oedema, hypertension and congestive cardiac failure (CCF).

2. Potassium (K), if retained, needs restriction to prevent hyperkalemia (excess K in blood), which can result in cardiac arrest. Both electrolytes, if lost in excess amounts in urine may need replacement.

### ***Vitamins and Minerals:***

1. Calcium may require supplements in chronic or severe cases along with vitamin D.
2. Phosphate mostly needs restriction as it tends to be retained in the body.
3. Iron supplements are needed with impaired erythropoietin synthesis,
4. Vitamin B and C supplements are required due to increased needs in renal patients.

Fluid: This mostly has to be restricted due to a fall in the GFR in most patients. If however, polyuria occurs, fluid needs may increase.

If you have understood these basic principles, you will be able to modify the diet in various renal disorders on a more rationale basis as you will find out while learning about the various renal disorders described next.

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## **16.7 ACUTE AND CHRONIC NEPHRITIS**

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As mentioned earlier, nephritis refers to the inflammatory disease of the nephrons due to infection, degenerative processes or vascular disease. In most cases, the inflammatory process affects the capillaries of the glomeruli, this disorder is termed as glomerulonephritis (GN).

There may be damage to the tubules also. The most common and well understood of the different types glomerulonephritis, is post streptococcal proliferative glomerulonephritis, about which we will discuss here in this section. GN may be self-limiting or progress to serious renal damage. Let us learn about the etiology, clinical and metabolic manifestations and dietary management of glomerulonephritis next.

### **16.7.1 Etiology**

Acute form of glomerulonephritis is commonly seen in 3-10 year old children, although in 5% or more cases the initial attack occurs in adults past the age of 50 years. Previous streptococcal infection, 7-20 days prior to onset, is a common cause of this disease and antigen-antibody reaction is mostly the basis of damage of nephrons. In acute glomerulonephritis, there is a usually sudden onset and the condition is usually completely cleared in a year or two.

In chronic cases, progressive renal damage occurs involving an increased amount

### **NOTES**

of renal tissue, eventually requiring dialysis and other support treatments.'

### 16.7.2 Clinical and Metabolic Manifestations

#### NOTES

Classic symptoms include gross haematuria (presence of blood in urine) and proteinuria (protein in the urine). Due to sodium and water retention and circulatory congestion, varying degree of oedema with shortness of breath may be observed. The patient generally is anorexic, which results in feeding problems. If the disease leads to renal insufficiency, oliguria (reduced urine output) or anuria (no urine output) occurs, which indicates chances of development of acute renal failure.

### 16.7.3 Dietary Management

Keeping in mind the general objectives of dietary management and principles of dietary modifications already discussed earlier in section 16.4, the following dietary guidelines is recommended.

**Calories:** A high-energy diet is recommended to spare the proteins for tissue repair rather than being used for energy. Adults may need 30-40 Kcal / kg dry weight and children about 100 Kcal/kg dry weight or more, based on age. Increased energy requirements are also recommended since elevation of body temperature i.e. fevers may be present in patients with glomerulonephritis. Elevation of body temperature results in increase in basal metabolic rate (BMR) and hence the energy intake may be increased to about 10% (from the level suggested by RDI).

**Proteins:** Blood urea nitrogen (BUN) and oliguria determine the restriction of protein in the diet. Initially, 0.5 to 0.6-g protein/kg Ideal Body Weight (IBW) is provided using principally high quality protein. Normal levels of protein (1 g/kg IBW) may be provided if BUN levels remain within the normal range.

Emphasis should be to include good quality protein or proteins of high biological value (milk and milk products, egg white, meat etc.) in the diet of the patients.

**Carbohydrates:** Liberal carbohydrate intake is important for protein sparing action, for reducing catabolism of protein, as well as, for preventing starvation ketosis. Both simple carbohydrates such as sugar, as well as, complex for1 such as starches can be included in the diet.

**Fats:** Based on tolerance levels, fat is included to provide non-protein calories for energy needs. Being energy dense, fat reduces the bulk of the diet and makes the diet more palatable.

**Sodium:** The restriction of sodium is dependent on the degree of oliguria and hence sodium retention. If renal function is impaired, sodium may be restricted to 500 to 1000 mndday. With recovery, the intake may be increased. A list of sodium-rich foods is included in Table 16.2 for your reference. You may need to avoid these. foods in the . diet of the patient suffering from nephritis.

**NOTES**

**Potassium:** In case of oliguria, renal clearance of potassium is impaired resulting in hyperkalemia (increased level of potassium in blood). In the initial stages, therefore potassium may have to be restricted to 1200-1500 mg/day. In addition, fruit, fruit juices, nuts and coconut water may be restricted or avoided, as they are rich in potassium.

**Fluid** Intake of fluids needs to be restricted in case of reduced GRF and oliguria to 500-700 ml/day plus the volume of urine output in previous 24 hours. Without oliguria fluid intake may be normal.

Dietary guidelines for acute chronic nephritis are summarized in Table 16.3.

**Table 16.3: Dietary guidelines in nephritis**

| Nutrients    | Comments                                                                                                                                                                                                         |
|--------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Protein      | BUN and oliguria determine the protein restriction. Give 0.5 to 0.6 g protein/kg of ideal body weight and then increase gradually depending on BUN values.                                                       |
| Kilocalories | Adults given 30-40 Kcal/kg dry weight and children about 100 Kcal/kg dry weight based on age. Sufficient simple and complex carbohydrates have a protein sparing action. Fat should be given based on tolerance. |
| Sodium       | Sodium depends on sodium retention and is restricted to 500-1000 mg/day.                                                                                                                                         |
| Potassium    | Potassium restriction to 1200-1500 mg/day especially if excess in blood (hyperkalemia).                                                                                                                          |
| Fluid        | If reduced GRF and oliguria, restrict to an amount equal to urine volume plus 500 ml.                                                                                                                            |

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## 16.8 NEPHROTIC SYNDROME

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Nephrotic syndrome is a disorder where the kidneys have been damaged, causing them to leak protein from the blood into the urine. It is a fairly benign disease when it occurs in childhood, but may lead on to chronic renal failure, especially in adults.

Nephrotic syndrome is also termed as nephrosis. This disorder is characterized by massive oedema and proteinuria resulting from degenerative lesions of the tubules, mesangium (central part of the renal glomerulus) or basement membrane of the glomeruli.

What is the cause for these degenerative lesions to occur? Let us find out.

**NOTES**

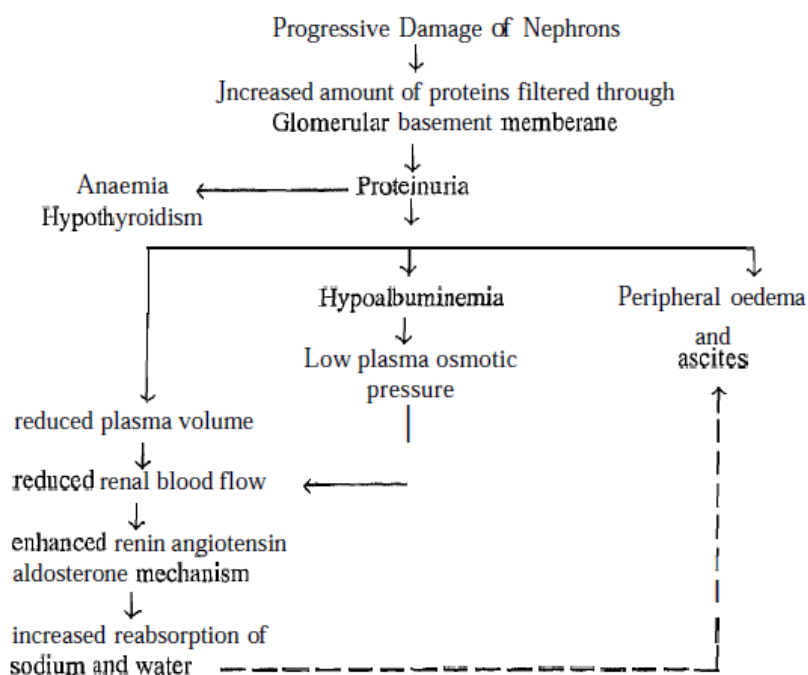
**16.8.1 Etiology**

There are many etiological factors that cause a nephrotic syndrome. Progressive glomerulonephritis, disease such as diabetes, collagen disease or drug reactions, from exposure to heavy metals, or even from a reaction to toxin venom following a bee sting can cause this syndrome.

**16.8.2 Clinical and Metabolic Manifestations**

Nephrotic syndrome is characterized by massive oedema and proteinuria, hypoalbuminemia, hypercholesterolemia and abnormal bone metabolism. Let us get to see how this happens.,Denegerative lesions of the capillary basement membrane of the glomeruli lead to loss of the glomerular barrier to protein. Large amount of proteins (upto 4-10 g/day) are thus lost in the urine as albumin. This proteinuria results in plasma proteins being substantially lowered and causing massive hypoproteinemic oedema to occur due to reduction in plasma albumin, which is mainly responsible for maintaining balance between tissue and circulating fluids. Pedal (foot) and periorbital (around the orbit of the eye) oedema and ascites (fluid in abdominal cavity) are common.

Other proteins lost include globulins, thyroid and iron binding protein. The continued loss of proteins results in tissue breakdown and malnutrition, often masked by the oedema. Development of fatty liver and sodium retention worsens the oedema. Another feature of this disorder, linked to hypoproteinemia is elevation of serum lipids, especially cholesterol to above 300 mg/dl. Nephrotic syndrome may be characterized by spontaneous period of remission and exacerbation.



**Figure 16.3 : Flow diagram for nephrotic syndrome**

As the disease progresses, calcium or phosphate levels in blood maybe altered due to altered vitamin D levels, resulting in renal osteodystrophy and osteomalacia.

From our discussion above it is evident that nephrotic syndrome occurs due to loss of proteins in the urine in large quantities which reduces the amount of protein in blood, So, then what is the dietary management for this disorder? The next section presents a detail review on this aspect.

### 16.8.3 Dietary Management

The major objectives of dietary management are 1) to control and correct protein deficiency, and 2) correct and prevent oedema and 3) maintain adequate nutrition to afford better resistance to infection.

To help meet this objective the dietary guidelines include:

**Calories:** High daily intake of 35 to 60 Kcal/kg of ideal body weight for adult and about 100 Kcal / kg or more for children is prescribed so as to conserve proteins. This ensures adequate amount of energy and optimal utilization of protein for tissue synthesis,

**Protein:** The major cause of nephrotic ascites and oedema is reduction of 20% or less than normal value in plasma albumin level.

Therefore, replacement of prolonged protein loss is most immediate and fundamental. A daily protein intake upto 1.25 g/kg/day in adults is advocated. As for children, since Indian children usually have a low intake of less than the RDA), adequate protein intake of upto 2 g/kg/day and not more than 3 g/kg/day in infants is advocated to replenish the depleted stores and to enhance synthesis of albumin and thereby reduce the oedema. However, please note, a very high protein diet may cause tubular damage the kidneys as the kidneys will have to filter more of the proteins.

Foodstuffs rich in protein are cow's milk, skimmed milk, eggs, fish, dry fish, chicken, lean meat, paneer made from cow's milk, cheese, sprouts, pulses and legumes. At least 60-70 % of this protein should be of good / high biological value (milk and milk products, egg whites and meats).

**Carbohydrates:** A high carbohydrate intake is recommended for the protein sparing action.

**Fat:** High amount of fats should be avoided as the cholesterol and triglyceride levels tend to be high in patients with nephrotic syndrome. The diet must be high in calories so as to conserve proteins, yet low in fats. Excess of oily food and saturated fats (ghee, margarine, etc) should be avoided. If patient has hyperlipoproteinemia and hypercholesterolemia, the total fat, as well as, cholesterol intake needs to be restricted to less than 30% energy from fat and < 300 mg cholesterol per day.

**NOTES**

**Sodium:** Reduction in sodium intake is required to reduce the oedema. Approximately 2-3 g of sodium/day may be recommended. Usually added salt is prohibited in these patients. Refer to Table 16.2 given earlier which presents the foodstuffs high in sodium. Avoid these foods in the diet of the nephrotic syndrome patient. Diuretics are usually used to prevent further oedema.

**Potassium:** If oliguria and anuria is not present, potassium restriction is not necessary. In fact adequate potassium is important as losses may occur due to tissue protein breakdown and diuretic use.

**Calcium:** If deficiency of calcium results leading to bone rarefaction, increased calcium intake or calcium supplementation is recommended along with moderate increase in protein.

**Fluid:** May be normal unless GFR is reduced.

**Table 16.4: Dietary guidelines for nephrotic syndrome**

| Nutrients     | Comments                                                                                                                                        |
|---------------|-------------------------------------------------------------------------------------------------------------------------------------------------|
| Calories      | For adults' – 35-60 Kcal/kg/IBW and children 100 Kcal/kg. Sufficient calories provide for optimum utilization of proteins for tissue synthesis. |
| Protein       | For adults 1.25g/kg IBW/day and children 2-3 g/kg IBW. Of this, 60-70% proteins must be of high BV to prevent ascites and oedema.               |
| Carbohydrates | High intake due to its protein sparing action.                                                                                                  |
| Fats          | Limit to 30% energy from fat and < 300 mg cholesterol, especially if the patient has hyperlipoproteinemia and hypercholesterolemia.             |
| Sodium        | 2-3 g/day and diuretics in order to reduce oedema.                                                                                              |
| Potassium     | Required in adequate amounts as its losses may occur due to tissue protein breakdown and diuretic use.                                          |
| Calcium       | Additional amounts needed in case of bone rarefaction.                                                                                          |
| Fluid         | Normal – unless GFR is reduced.                                                                                                                 |

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## 16.9 ACUTE RENAL FAILURE

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Acute renal failure (ARF) is a rapid loss of renal function due to damage to the kidneys, resulting in retention of nitrogenous (urea and creatinine) and non-nitrogenous waste products that are normally excreted by the kidney. Depending



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on the severity and duration of the renal dysfunction, this accumulation is accompanied by metabolic disturbances, such as metabolic acidosis (acidification of the blood) and hyperkalaemia (elevated potassium levels), changes in body fluid balance, and effects on many other organ systems. It can be characterized by oliguria or anuria (decrease or cessation of urine production), although nonoliguric ARF may occur. It is a serious disease and treated as a medical emergency.

Acute renal failure, in fact is a sudden loss of the ability of the kidneys to excrete waste, concentrate urine and conserve electrolytes. It is a serious condition characterized by a sudden shutdown of kidney function as mentioned above due to decreased renal flow, acute glomerular or a tubular damage. It results in a decline in glomerular filtration rate (GFR), usually associated with azotemia (accumulation of nitrogenous waste products in the blood) and a fall in urine output.

### 16.9.1 Etiology

Several conditions can lead to ARE These include:

- Circulatory shock, large blood loss and reduced renal blood flow as in traumatic injury, shock, severe burns, surgery, septicemia, dehydration and fluid loss,
- Mismatched blood transfusions,
- Nephrotoxins like carbon tetrachloride, certain poisonous mushrooms,
- Infections, snake bite, bee stings etc.
- Immunological reactions to drugs like certain antibiotics, and
- Renal disease like acute glomerulonephritis.

The most common cause is altered renal haemodynamics or ingestion of nephrotoxins.

What then are the clinical manifestations of this disorder? Let us find out.

### 16.9.2 Clinical and Metabolic Manifestations

The onset of ARF is sudden, with the course of the disorder having two phases, namely:

- a) oliguria or initial acute phase, and
- b) diuretic phase.

The latter indicates restoration of renal function, although it may still remain poor for several days. The major clinical features of ARF are oliguria or anuria (urine output 20-200 ml), due to drastic reduction of GFR to 1-2% of normal. Along with this, haematuria and proteinuria are usually present. There is an elevation of serum urea nitrogen and creatinine due to reduced GFR and tissue protein breakdown. Uremia may develop along with associated symptoms like disorientation, lethargy, nausea, vomiting and anorexia. Blood pressure elevation

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acid increased levels of potassium, phosphate and sulphate occurs with lowered levels of sodium and bicarbonate. Water balance is a crucial factor and unless controlled, the condition can prove fatal mostly due to potassium intoxication or excess fluid retention leading to cardiac failure. Return to renal function is characterized by an increase in urine output or diuresis.

When diuresis is established, the urine volume gradually increases to between 3 to 5 litres/day and the excretion of sodium, potassium, urea and other solutes also increase. The blood urea falls to normal in 7 to 10 days, indicating that glomerular filtration has effectively improved. Although the excretory function of kidney is restored, the recovery of regulatory function of the tubules is slower. The internal environment of the patient is still at risk because of excessive losses of water, sodium, potassium, bicarbonate and magnesium.

In majority of patients, the kidneys will recover with little or no residual damage if the patient can survive the oliguric phase. However, in a few cases, residual damage of tubular function may sometimes be detected even long after the blood urea has returned to normal levels.

### 16.9.3 Dietary Management

In ARF the common nutritional problems include: (1) Poor appetite, (2) Inability to take food or fluids orally due to intubation, and (3) Hypercatabolism (increased metabolism) due to underlying illnesses such as infection, postoperative healing.

reestablishment of fluid electrolyte balance,  
maintenance of acceptable levels of blood urea and creatinine while supporting  
tissue healing and making up catabolic losses, and  
preventing infection.

With conservative medical and diet treatment, recovery may occur within a few days or weeks. However, if oliguria continues with a rise in nitrogenous wastes and potassium, aggressive therapy including haemodialysis may be required with nutritional support. Thus dietary management is a challenge and plays an important role. Oral feeding is best, but if nutritional support is needed, caution is necessary to avoid fluid overload and uremia.

The dietary guidelines for acute renal failure include:

**Calories:** In most adults, energy requirement amounts to 30-40 Kcal/kg body weight with up to 40-45 Kcal/kg for hypercatabolic cases. The major source of energy is carbohydrates followed by fat. An intake of 100-200 g or more of sugar /glucose per 24 hours is administered when the oral intake is poor because of vomiting and diarrhoea. Children need enough calories to support growth. In some cases enteral or parental non-protein concentrated calories sources may have to be provided because of fluid restriction.

**NOTES**

**Protein:** This needs restriction and intake is dependent on GFR and extent of hypercatabolism. Initially the intake may range from 0.5 to 0.6 pkg IBW, subsequently, increased to 0.8-1.2 g/kg IBW/day. Therefore, depending on the degree of protein catabolism 0.5-1g/kg/day of protein may be given. With improvement, at least 60- 70% of good quality proteins are recommended to reduce unnecessary nitrogen load.

In case total parenteral nutrition (TPN) is required, a balanced amino acid solution containing both essential and nonessential amino acids should be administered. In addition to essential amino acids, arginine, histidine, serine, taurine and tyrosine may be recommended.

**Sodium:** During the oliguric phase, sodium may need to be restricted to 500-1000 mg (20-40 mEq) daily. It can be liberalized with onset of diuresis.

**Potassium:** Since, hyperkalaemia is a life-threatening complication of acute renal failure, it needs to be treated urgently. Potassium intake is restricted to 1000-2000 mg (25 to 50 mEq) and should be monitored strictly and regularly. As the renal function improves, the intake may be increased.

**Fluid:** Intake is based on fluid balance but is usually restricted to a basic allowance of 500 ml/day for an average adult with addition made for losses via other routes. The fluid allowance is usually regulated in accordance with urinary output and any additional losses from vomiting or diarrhoea. Strict monitoring of fluid balance is important, the patient's weight and blood sodium levels are good indicators of fluid balance, and the amount of fluid required. If fluid intake is not adequate for excretion of metabolic wastes, dialysis is usually recommended.

**Table 16.5: Dietary guidelines in acute renal failure**

| Nutrients | Comments                                                                                                                                                                                 |
|-----------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Protein   | If not on dialysis, 0.6 g/kg/day. If on dialysis, 1-1.2 g/kg from dietary or parenteral sources.                                                                                         |
| Calories  | Provide sufficient amount for weight maintenance or 30-40 Kcal. Increase to 40-45 Kcal/kg in hypercatabolic subjects. Encourage non-protein calories from fats and simple carbohydrates. |
| Sodium    | In oliguric phase, restrict to 500-1000 mg (20-40 mEq). On diuresis, the amount may be increased.                                                                                        |
| Potassium | If hyperkalemia is present restrict to 1000-2000 mg (25-50 mEq). On improvement, increase to 60-70 mEq.                                                                                  |
| Fluids    | Limit fluids to an amount equal to urine volume plus 500 ml.                                                                                                                             |

## NOTES

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### 16.10 CHRONIC RENAL FAILURE (CRF)

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Chronic renal failure is a slow progressive loss of renal function over a period of months or years and defined as an abnormally low glomerular filtration rate, which is usually determined indirectly by the creatinine level in blood serum.

CRF, therefore, is a condition that arises due to advanced and progressive damage of kidneys with impairment of renal function. Few functional nephrons remain and CRF results in what is usually termed uremia. Uremia, you may already know by now is a toxic condition resulting from renal failure, when kidney function is compromised and urea, a waste product normally excreted in the urine, is retained in the blood.

Unlike acute renal failure, with its sudden reversible failure of kidney function, chronic renal failure is a gradual and progressive loss of the ability of the kidneys to excrete wastes, concentrate urine, and conserve electrolytes. CRF can range from mild dysfunction to severe kidney failure. CRF that leads to severe illness and requires some form of renal replacement therapy (such as dialysis) is called end-stage renal disease (ESRD) about which we shall learn in the next section.

#### 16.10.1 Etiology

A number of diseases that involves the nephrons may result in CRF like primary glomerular diseases such as:

- immune complex glomerulonephritis,
- a metabolic disease with renal involvement such as diabetes mellitus, especially IDDM,
- hypertension,
- exposure to toxic substances,
- kidney stones and infections,
- renal vascular diseases,
- renal tubular diseases,
- chronic pyelonephritis, and
- congenital abnormalities of both kidneys.

These conditions can cause extensive changes in kidney structure and function. CRF results in accumulation of fluids and waste products which can lead to many clinical signs and complications which are enumerated next.

#### 16.10.2 Clinical and Metabolic Manifestations

Progressive loss of nephrons with a decreased renal blood flow and glomerular filtration results in a marked impairment of not only excretory but also metabolic and endocrine functions of the kidney. There is a decreased ability of the kidneys

## NOTES

10 maintain body water balance, concentrate solutes in body fluid (osmolality) and electrolyte and acid- base balance, Other clinical manifestations that develop may relate to almost every system of the body due to an overall pervasive metabolic derangement of the body.

Increased solute load of metabolic wastes results in osmotic diuresis initially, due to an impaired ability of the kidney to concentrate urine. This leads to loss of sodium and potassium. However, with continued renal damage and reduced GFR, sodium, potassium and nitrogenous wastes tend to be retained in the body. This contributes to oedema, hypertension, hyperkalemia and azotemia, respectively, Azotemia is the buildup of nitrogen waste products in the blood.

Retention of phosphate, sulphate and organic acids causes metabolic acidosis due to loss of bicarbonate. Impaired calcium and phosphorus balance due to decreased vitamin D 3 and consequent secondary hyperparathyroidism leads to renal osteodystrophy or renal bone disease, with bone and joint pains. Calcification of soft tissues is another complication that can develop.

Other clinical features include anaemia due to impaired RBC synthesis. Hypertension arises due to stimulation of the renin angiotensin system by the reduced renal blood flow, resulting in vasoconstriction. The resultant cardiovascular damage worsens renal function. Other related symptoms are shortness of breath and fatigue. Azotemia and other metabolic changes cause anorexia, weight loss, gastrointestinal irritability, nausea, vomiting and diarrhoea. Subcutaneous nasal or GI bleeding can occur due to increased capillary fragility. Mouth ulceration, taste changes, neurological symptoms, increased susceptibility to infection due to malnutrition also commonly observed.

Controlling the symptoms, minimizing complications, and slowing the progression of the disease, therefore is the main focus for the management of CRF, particularly the dietary management which is discussed next.

### 16.10.3 Dietary Management

Feeding is a challenge in CRF as anorexia and taste changes reduce food intake. The main focus of dietary management is on protein, sodium, potassium, phosphate, water and adequate non-protein calories. Individual modifications are required based on clinical profile, treatment and response of the patient. Keeping in mind the general objectives of diet treatment in renal disease, the following modifications are required.

Energy: About 2000-2500 Kcal/day are recommended or 30-40 Kcal/kg/day for adults and about 100-150 Kcal/kg/day for children. If the calorie intake is inadequate, endogenous protein catabolism and gluconeogenesis occur to supply energy and further aggravate uremia. Therefore, 300-400 g of carbohydrates are recommended.

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**Protein:** Protein needs to be restricted. However, enough has to be provided to minimize tissue catabolism. About 0.5 g/kg/day is recommended but has to be regulated depending on declining renal function. A protein intake of 35-40 g/day (60-70% of high biological value protein) with liberal calorie intake can maintain the nitrogen equilibrium for long periods while reducing azotemia. If BUN rises, the protein intake may need to be restricted to 20 g/day. High biological value proteins from milk and eggs are recommended to provide all the essential amino acids. To reduce the nitrogen load, in advanced cases, mixture of essential amino acids or nitrogen free precursors of the essential amino acids like a keto or a hydroxy analogs may be recommended.

The potential benefits of protein restriction include:

- Decreases glomerular hyperfiltration, which may slow progression of glomerulosclerosis.
- Protein restricted diets are phosphorus restricted, which delays onset of renal secondary hyperparathyroidism and may slow progression of glomerulosclerosis,
- Reduces proteinuria in glomerulopathies.
- Reduce net acid load.
- May reduce serum lipids.
- Improves the symptoms of uremia.

**Sodium:** Sodium intake will vary between 500 mg to 2.0 g/day. Weight loss and decreasing urine volume usually indicate a need for additional sodium, whereas if hypertension and oedema are present, the sodium intake needs to be restricted.

**Potassium:** The failing kidney cannot excrete potassium adequately and therefore intake is kept at about 1500 mg/day (35 to 40 mEq /day). The potassium intake has to be adjusted to maintain normal levels in blood. In severe vomiting and diarrhoea, significant losses of potassium can occur and in these conditions, careful potassium

supplementation may be needed. Potassium content of various vegetables and the methods of leaching potassium are given in subsection 16.11.4. Read this information Nutritional

**Calcium and phosphorus:** To maintain calcium and phosphorus balance and prevent or delay renal bone diseases, calcium supplements (1-2 g/day) are recommended and phosphate is restricted to 800-1200 mg/day. Phosphate binding agents may be used if required to reduce absorption. Calcium carbonate supplements can help buffer metabolic acidosis, if present. It is important to remember not to start calcium supplements, unless phosphate is restricted, to avoid soft tissue calcification.

**Vitamin:** Multivitamin supplements are recommended for a diet with < 40 g/day of protein. Supplements of vitamin D<sub>3</sub> may be recommended, based on need.

**NOTES**

Fluid: Intake is dependent on urine output and water balance. Fluid intake should be adequate to stimulate urine output for excretion of wastes but should avoid excess fluid retention at the same time.

Some nutritional supplements are available in the inarket that are very specific for this condition and can be recommended if the dietary intake is unsatisfactory. Details related to commonly available commercial enteral nutrition formulas for renal patients is given later in section 16.12. You may like to review these formulas now. So go ahead and review these formulas.

The summary of the dietary guidelines for CRF is given in Table 16.6 for your ready reference.

**Table 16.6: Dietary guidelines for CRF**

| Nutrients               | Comments                                                                                                                                    |
|-------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|
| Energy                  | 30-40 Kcal/kg/day for adults and 100-150 Kcal/kg/day for children.                                                                          |
| Carbohydrates           | 300-400 g/day to avoid endogenous protein catabolism, gluconeogenesis and subsequently uremia                                               |
| Proteins                | 0.5 g/kg/day, with 60-70% as high BV protein. To reduce N <sub>2</sub> load, a mixture of essential amino acids is recommended.             |
| Sodium                  | 500 mg-2.0g/day. Additional Na in case of weight loss and decreasing urine volume and restriction of Na in case of oedema and hypertension. |
| Potassium               | Intake must be kept at 1500 mg/day (3-0 mEq/day) and in case of significant losses, potassium supplements should be given,                  |
| Calcium and Phosphorous | Calcium supplementation – 1 to 2 g/day and phosphate to be restricted to 800-1200 mg/day                                                    |
| Vitamin                 | Multivitamin supplements, specially vitamin D <sub>3</sub>                                                                                  |
| Fluid                   | Intake is dependent on urine output and water balance.                                                                                      |

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## 16.11 END STAGE RENAL DISEASE (ESRD)

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Do you know that in India with a population of more than 1 billion an approximate 100,000 patients develop ESRD? Of these, 90% never see a nephrologist and 10% who do consult a specialist find it difficult to afford the treatment.

The condition when kidneys have lost all or most of their ability to function with GFR < 5 ml/minute is called end stage renal disease (ESRD). Today, there are new and better treatments for ESRD that replace the work of healthy kidneys. The treatment of choice includes dialysis — haemodialysis (HD) and peritoneal

dialysis (PD) — and kidney transplantation,

## Dialysis

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So what does the process of dialysis involve? Yes, the process of dialysis involves cleansing the blood of metabolic wastes, based on the principle of osmosis and diffusion. A semi permeable porous membrane is used in dialysis to separate the patient blood carrying excess fluid and metabolic wastes and the hypotonic "dialysis fluid" called dialysate. Through osmosis and diffusion, the metabolic waste and excess water move into the dialysate. The pores of the semi permeable membrane do not permit large particles like protein and RBC to pass through, but smaller water-soluble molecules can pass.

You may also recall studying that there are two types of dialysis. These include:

b) peritoneal dialysis

A brief review of these two types follows.

**Haemodialysis (HD)** — In this, patient's blood circulates outside the body through what is commonly referred to as an "artificial kidney machine". An opening is created to connect an artery and a vein. Blood leaves the body via the artery, into the dialyses and after cleansing, flow back to the body.

**Peritoneal Dalysis (PD)** — In this, the patient's peritoneum is used as the semi permeable membrane and excess water and metabolic wastes are removed by injecting the dialysis fluid into the peritoneal cavity as illustrated in Figure 16.5. After same time, the fluid with the metabolic waste is drained out from the peritoneum. Peritoneal dialysis is less effective than haemodialysis and can result in loss of intact large molecular proteins also.

For long-tenn use, continuous ambulatory peritoneal dialysis (CAPD) may be used based on facilities available. In this, the dialysis fluid is exchanged 4-5 times daily. It is also important to prevent/control infection. In some cases, continous cyclic peritoneal dialysis CCPD or Intermittent Peritoneal Dialysis (IPD) may be used.

### 16.11.2 Dietary Management during Dialysis

Once dialysis is started, the diet in ESRD can be liberalized, taking care that accumulation of metabolic wastes and water is prevented between treatments and biochemical balance is maintained.

The objectives of dietmy management thus are to maintain balance of protein, energy, fluid and electrolytes, calcium and phosphorus, while making up losses of water-soluble nutrients lost in the dialysate. The dietary guidelines include:

**Energy:** Up to 35-40 Kcal/kg/day for adults and 100 Kcal or more [kg/day for children is recommended to meet the body needs and minimize tissue protein breakdown. Fats and carbohydrates are the main energy sources used. Some



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restriction of total and saturated fats may be needed, as dialysis patients are prone to cardiovascular disease.

**Protein:** The requirement is increased due to losses in the dialysate. In haemodialysis, 1.2-1.5 g/kg/day is required. At least 70% of the protein given should be of high biological value from eggs, fish, chicken and milk, though milk may need to be limited being a rich source of potassium.

This protein intake helps to maintain positive nitrogen balance, replace losses and prevent undue accumulation of nitrogen wastes, between treatments. Amino acid replacement may also be required in case of large losses.

**Sodium:** A daily intake of 1500 to 2500 mg may be permitted to control fluid retention and hypertension. This restriction helps to prevent pulmonary oedema or congestive heart failure because of fluid overload. Regular assessment of the kidneys ability to handle sodium and water is important to determine the intake.

**Potassium:** A daily intake of 1500-2500 mg is prescribed to prevent hyperkalemia. Potassium accumulations easily cause cardiac arrhythmias or cardiac arrest.

**Phosphorus:** This may need some restriction.

**Vitamins and Minerals:** A daily supplement of water-soluble vitamins and minerals are usually given, as these are lost in the dialysate. Fat-soluble vitamins may be retained. Thus, their supplements are avoided except vitamin D. Supplements of minerals like calcium, iron and zinc are recommended.

**Fluid:** Usually 400-500 ml (basal losses) plus the urine output is recommended. The fluid intake must take into account all sources of fluid input and output to maintain balance. Mild fluid retention between treatments usually occurs.

Patient counseling and support is an important part of dietary management to help renal patients understand their dietary modifications, the foods permitted and those to be avoided. Counseling about the ways to increase the palatability of the restricted diets can encourage the patient to increase their dietary intake.

### 16.11.3 Kidney Transplantation

Kidney transplantation, you may recall studying, is a procedure that surgically places a healthy kidney from a donor into the recipient's body. This new kidney does the work of the failed kidneys. Donated kidneys may come preferably from blood relatives, after tissue and blood matching. Success of kidney transplant has improved with the use of immunosuppressive drugs and steroids to prevent organ rejection and infection. Post transplant nutritional support is required for this major surgical procedure. Optimal energy and protein intake are important for recovery, as you may recall studying earlier in Unit 5 dealing with surgery. Initially, while on medication, some restriction of sodium, simple sugars, total fat, cholesterol and saturated fat may be required. This is because of the side effects of

immunosuppressant and steroids. With recovery and reduction or withdrawal of medication, the diet can be normalized. Table 16.7 gives the nutrient requirements for adult with end stage renal disease based on the type of therapy.

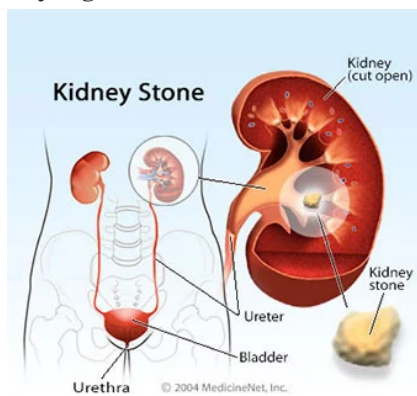
**NOTES**

**Table 16.7: Nutrient requirement for adult with end stage renal renal**

| Therapy                                          | Energy (Kcal/kg BW)      | Protein (g/kg IBW) | Fluid (ml/day)                                    | Sodium (g/day) | Potassium (g/day) | Phosphorus (g/day)     |
|--------------------------------------------------|--------------------------|--------------------|---------------------------------------------------|----------------|-------------------|------------------------|
| Haemodialysis                                    | 35                       | 1<br>output        | 500+urine<br>2-3                                  | Variable       | 2-3               | 1-1.2                  |
| Intermittent Peritoneal Dialysis (IPD)           | 30(40-50 for repletion)  | 1.2                | 500+urine<br>output                               | 2-3            | 2-3               | 1-1.2                  |
| Continuous Ambulatory Peritoneal Dialysis (CAPD) | 25 (40-50 for repletion) | 1.2                | Adlibitum (minimum or 2000 ml/day + urine output) | 6-8            | 3-4               | 1.5-2                  |
| Transplant (4 to 6 weeks after transplant)       | 30-35                    | 1.5-2              | Adlibitum                                         | Variable       | Variable          | 1.2, calcium 1-2 g/day |
| Transplant (six weeks or longer)                 | To achieve IBW           | 1                  | Adlibitum                                         | Variable       | Variable          | Calcium 1-2 g/day      |

**16.12 RENAL CALCULI**

Renal calculi or stones may be formed in the kidney, pelvis or ureter, when the concentration of components in the urine reaches a level in which crystallization is possible. The process of stone formation is also called nephrolithiasis or urolithiasis. Figure 16.6 illustrates the kidney stone. A kidney stone is a solid mass that consists of a collection of tiny crystals. There be one or more stones present at the same time in the kidney or in the ureter. They generally are composed of calcium salt, uric acid, cystic or struvite (triple salt of ammonium, magnesium and phosphorus). Crystals of these substances interspersed in an organic matrix or base can form stones of varying size.



**Figure 16.6: Kidney stone**

### 16.12.1 Etiology

Kidney stones may form when the urine becomes too concentrated with certain substances. These substances may create small crystals that become stones. Different types of stones form under different circumstances. Although, the exact cause of renal stones is not known, but multiple factors may play a role directly or indirectly, mostly related to urine composition and urinary tract environment. Some possible etiological factors in different types of calculi are enumerated herewith:

- Calcium stones (oxalate, phosphate and carbonate): Excess intake of calcium, oxalate, hypervitaminosis D, hyperparathyroidism, prolonged bed rest, renal tubular acidosis, idiopathic hypercalciuria.
- Struvite stones: Mostly due to urinary tract infection (UTI).
- Uric acid stones: Impaired purine metabolism with increased urinary excretion of uric acid.

Cystine stones: Hereditary metabolic defect in renal tubular reabsorption.

Hot climates leading to over concentrated urine, changes in pH of urine also predispose to stone formation. Although there is a high intake of animal protein, deficiency of vitamins B6 and magnesium are reported to play a role in the causation of these stones.

Some types of stones tend to run in families, Some types may be associated with bowel disease, ileal bypass for obesity, or renal tubule defects. So whatever may be the cause of the renal stones, it causes discomfort and typical symptoms.

### 16.12.2 Clinical Symptoms

The kidney stones may not produce symptoms until they begin to move down the ureter, causing pain. The pain is usually severe and often starts in the flank region, then moves down to the groin. The patient experiences blood in the urine, severe pain, weakness and in some cases fever. Laboratory examination and chemical analysis can help determine location, size and main constituent of stones to determine the treatment.

### 16.12.3 Dietary Management

The goal of treatment of renal calculi is to relieve symptoms and prevent further complications. Treatment, therefore, varies depending on the type of stone and the extent of symptoms or complications.

Kidney stones usually pass on their own. In acute stage with stones less than 5 mm in diameter, it may pass in the urine by drinking large quantities of fluid especially water and needs no specific treatment. Stones more than 7 mm in diameter may require surgical treatment or lithotripsy by which large stones are broken down and excreted in the urine.

**NOTES**

Although, role of diet in the formation urinary stones is not well established, it is advisable to have liberal fluid intake, a balanced diet and restrict foods based on the main constituent of the stones. Table 16.8 gives information related to different stones and their corresponding diet restriction.

**Table 16.8: Different stones and their corresponding diet restrictions**

| Main constituents                                                                                                                                                | Diet restriction                                      | Urine pH |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------|----------|
| <ul style="list-style-type: none"> <li>• Calcium stones</li> <li style="padding-left: 20px;">-phosphate</li> <li style="padding-left: 20px;">-oxalate</li> </ul> | Calcium – 400-600 mg<br><br>Phosphorus - 1000-1200 mg | Acid     |
| <ul style="list-style-type: none"> <li>• Struvite stones</li> </ul>                                                                                              | Low phosphorus diet                                   | Acid     |
| <ul style="list-style-type: none"> <li>• Uric acid</li> </ul>                                                                                                    | Low purine diet                                       | Alkaline |
| <ul style="list-style-type: none"> <li>e Cystine</li> </ul>                                                                                                      | Low methionine diet                                   | Alkaline |

Besides liberal fluid intake and some dietary restriction, urine pH control helps based on the chemical composition of the stone, mainly via acidifying or alkalinizing agents or diet. Binding agents to bind the stone constituent may also be used.

Let us now have a look at the dietary sources of various constituents of the renal stones.

**16.12.4 Dietary Sources of Various Constituents of the Renal Stones**

Dietary sources of potassium, sodium, calcium, oxalate and uric acid are given in this section. We begin our study with the dietary sources of potassium.

***Directions / Methods for leaching Potassium***

Method I — Wash, peel and cut vegetables into small pieces. Soak in warm water for 2-3 hours. Discard water. Add large volume of fresh water and cook the vegetables. Discard water.

Method II — Peel vegetables and cut into small pieces. Bring to boil in a large quantity of water. Discard excess water and cook in a large volume of fresh water. Discard excess water.

***B. Sources of Sodium***

Food items with a high sodium content (these food items should be avoided):

Salt

- Baking powder
- Bicarbonate of soda
- Canned, preserved and processed food items as processed cheese, sauce, margarine, etc.
- Bacon, ham and sausages
- Meat and yeast extracts like marmite
- Salted chips, nuts, popcorn and biscuits
- Commercial salad dressings and sauces
- Soup cubes
- Malted beverages like Boost, Bournvita and Proteinex.
- Flavour enhancers such as Monosodium glutamate (MSG)

## NOTES

### C. Sources of Calcium, Oxalate and Uric Acid

#### Calcium

Beans, cauliflower, egg yolk, figs, milk and milk products like cheese, curds, molasses and potatoes.

#### Oxalate

cashewnuts, chickoo, chocolate, cocoa, custard apple, groundnuts, spinach, strawberries, tomatoes and tea.

#### Uric Acid

Fish herring, fish roe, salmon, sardines, kidney, liver, meat extracts and soups, and sweet bread.

We just learnt about the dietary sources of various minerals which may or may not contribute to the formation of renal calculi. Lastly, we move on to a few commonly available commercial enteral nutrition formulas which could be of help to renal patients.

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### 16.13 COMMONLY AVAILABLE COMMERCIAL ENTERAL NUTRITION FORMULAS FOR RENAL PATIENTS

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Earlier in this course in Unit 4, you may recall studying about the enteral and parenteral feeding and the formulas commonly used for these feeding methods. Some common enteral nutrition formulas for renal patients are highlighted here in this section.

*Reno — care I (For pre-dialysis state) Sachet —51.5 g provide:*  
*Low protein (3.75 g/sachet)*  
*Low sodium (4.10 mEq/sachet)*

## NOTES

*Low potassium (2.40 mEq /Sachet)*

*Low phosphorus (80 mg/sachet)*

*Reno - care II (For dialysis state) Sachet —52.5 g provide:*

*Moderate protein (8.80 g/sachet)*

*Low sodium (4.10 mEq/sachet)*

*Im potassium (240 mEq /sachet)*

*Low phosphorus( 86.0 mg /sachet)*

*Nutrenal CRF*

*Composition P' 100 g*

*Protein 7.74 g*

*Sodium 202.72 mg*

*Potassium 288.83 mg*

*Phosphorus 188.86 mg*

*Nutrenal Dialysis*

*Compositionper100 g*

*Protein 18.09 g*

*Sodium214.71 mg*

*Potassium 27356*

*Phosphorus 177.65 mg*

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## 16.14 LET US SUM UP

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In this unit, we learnt that kidney performs a myriad of functions including excretory, endocrinal and metabolic. The kidney maintains the fluid, electrolyte balance and normal volume, pH and osmolality of blood. Metabolic waste products and toxins are excreted from the body while essential nutrients are reabsorbed by the kidneys.

Further, the unit focused on the diseases of the kidney. These diseases alter the capacity of the kidneys to perform the functions. The common diseases in which diet plays an important role are glomerulonephritis, nephrotic syndrome, acute renal failure, chronic renal failure and renal calculi. We learnt that these diseases have multiple etiological factors and the clinical manifestations and biochemical parameters govern the dietary management of these diseases.

**NOTES**

The main objectives of the dietary management are to reduce the excretory workload of the kidney and prevent progression of renal damage while maintaining satisfactory nutritional status and as near normal fluid, acid base and electrolyte balance. To meet these objectives, modifications in the diet are required mainly for protein, sodium, potassium, fluid, calcium and phosphate. The main objective is to try and maintain the internal milieu of the body. In case of poor oral intake, commercially available supplements in the market may have to be used. When the kidney function fails dialysis or transplantation is recommended.

Finally, the unit focused on renal calculi. Patients with kidney stones have to be treated according to the type of stones and diet therapy is recommended thereafter. Thus, nutritional care will depend largely on the type of renal disease and biochemical and clinical manifestations.

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**16.15 GLOSSARY**

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|                   |                                                                                                       |
|-------------------|-------------------------------------------------------------------------------------------------------|
| <b>Anuria</b>     | : complete lack of urine excretion.                                                                   |
| <b>Anorexia</b>   | : loss of appetite.                                                                                   |
| <b>Ascites</b>    | : an accumulation of fluid in the abdominal cavity.                                                   |
| <b>Azotemia</b>   | : an accumulation of abnormal quantities of urea, uric acid, creatinine and other nitrogenous wastes. |
| <b>Catabolism</b> | : The destructive phase of metabolism in which complex substances are converted into simpler ones.    |
| <b>Congenital</b> | : of genetic origin.                                                                                  |
| <b>Dialysate</b>  | : the solution used in dialysis to remove waste products and excess fluid from the blood.             |
| <b>Diuretic</b>   | : an agent that increases urine secretion.                                                            |

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**16.16 CHECK YOUR PROGRESS**

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1. Briefly review the important physiological functions of kidneys.
2. Mention three significant kidney function tests.
3. Enlist the diseases caused due to kidney dysfunction.
4. What are the causes of nephrotic syndrome? What dietary measures help to correct oedema in nephrotic syndrome?
5. What are the two major symptoms of glomerulonephritis? What

are the recommended protein and fluid intake of glomerulonephritis?

**NOTES**

6. What is the recommended protein intake for ARF and CRF patients?
7. Why proteins of high biological value are recommended in renal failure?
8. What sodium intake is recommended for CRF patient?



## NUTRITIONAL MANAGEMENT OF NEUROLOGICAL DISORDERS

### STRUCTURE

- 17.1 Learning Objective
- 17.2 Introduction
- 17.3 Common Neurological Disorders
- 17.4 The Central Nervous System (CNS) - Some Relevant Physiological Aspects
- 17.5 Neurological Diseases: Feeding and Nutritional Issues - General Goals of Nutritional Care
- 17.6 Dysphagia
- 17.7 Alzheimer's Disease
- 17.8 Parkinson's Disease
- 17.9 Epilepsy
- 17.10 Neuro Trauma
- 17.11 Spinal Trauma
- 17.12 Let Us Sun1 Up
- 17.13 Glossary
- 17.14 Check Your Progress

### 17.1 LEARNING OBJECTIVE

After studying this unit, you will be able to:

- identify some common neurological disorders, their etiology and clinical features,
- explain the consequences of these disorders on feeding and nutrition, and suggest feeding and dietary recommendations to meet the needs of these disorders,

### 17.2 INTRODUCTION

Your friend's aged father, over a period of time has become very confused, forgetful

## NOTES

and is unable to do many things he could do earlier. He has also developed eating problems. Laxmi's baby suffered from anoxia (lack of oxygen) at birth which has led to developmental impairment and difficulty in chewing and swallowing. Ramesh suffered spinal injury in a road accident. This has resulted in a partial paralysis with associated impaired feeding ability. As a nutrition expert, how do you manage these situations? First of all, you must understand that all of these occur as a consequence to neurological problems. In this unit, we will provide you with the salient guidelines for the better management of these disorders.

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### 17.3 COMMON NEUROLOGICAL DISORDERS

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Neurological disorders may be of two types from the nutritional view point. What are these? Let us read and find out.

Neurological disorders arising due to imbalanced nutritional intake (deficiency or excess) — Common examples are the neurological manifestations of beriberi, pellagra, pernicious anaemia, Wernicke Korsakoff syndrome due to nutrient deficits and stroke, hypertension and diabetes due to nutrient excesses, unbalanced diet leading to malnutrition. Alcoholism and malabsorption could also be other causative factors.

Neurological disorders of non-nutritional etiology — Some of the common disorders are Alzheimer's disease, Parkinson's disease, epilepsy, spinal and neuro trauma. Dysphagia (difficulty in swallowing), though not a disease, is a symptom which may occur in several neurological disorders.

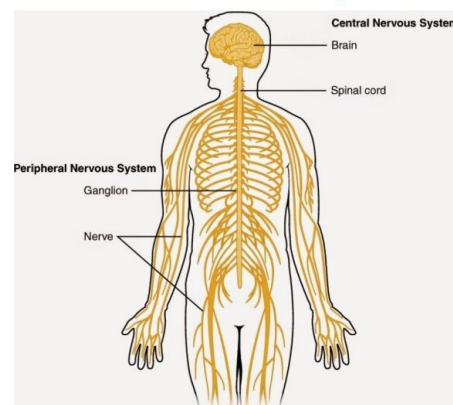
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### 17.4 THE CENTRAL NERVOUS SYSTEM (CNS) - SOME RELEVANT PHYSIOLOGICAL ASPECTS

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#### CENTRAL NERVOUS SYSTEM

#### Central Nervous System



**Figure 17.1: Structure and components of CNS**

Lesions of different parts Of the CNS can result in different dysfunctions with different nutritional significance. Signs of weakness are the most quantifiable clinical symptom of any neurological disease. Any damage to any part Of the CNS results in the inability of the body to meet its nutritional and metabolic needs,

**NOTES**



**Figure 17.2: Lobes of Cerebral Cortex**

**Table 17.1: Some consequences of CNS damage of nutritional significance**

| Area of Damage                            | Consequences                                                                                                                                                     |
|-------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Frontal lobe-base                         | Loss of sense of smell, visual changes                                                                                                                           |
| Frontal lobe-central                      | Motor apraxia or inability to carry out a complex activity, <b>inspite</b> of understanding it                                                                   |
| Frontal lobe-posterior                    | Seizures(convulsions)                                                                                                                                            |
| Temporal lobe                             | Memory and speech impairment                                                                                                                                     |
| Occipital lobe                            | Vision deficits                                                                                                                                                  |
| Brain stem                                | Damage of cranial nerves which innervate face and head, including eyes, ears, jaws, tongue, pharynx and facial muscles. Dysphagia and aspiration risk can occur. |
| Hypothalamus                              | Damage of center for hunger and satiety, leading to overeating or anorexia problems.                                                                             |
| Spinal Cord                               | Motor <b>impairment</b> / paralysis based on location of injury with resultant feeding difficulties.                                                             |
| Peripheral nerves and neuromotor junction | Impaired nutritional and metabolic balance.                                                                                                                      |

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## 17.4 NEUROLOGICAL DISEASES: FEEDNG AND NUTRITIONALISSUES-GENERAL GOALS OF NUTRITIONAL CARE

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**NOTES**

Nutritional management of the patients with neurological disease is complex, as mechanisms and abilities needed for adequate nourishment get impaired. Reduced functional capacity would impair the ability to procure and prepare food. Self feeding impairment may arise due to limb weakness, poor positioning of the body due to hemiparesis or partial paralysis. Hemianopsia or half sided blindness, apraxia (inability to perform), mental confusion, fatigue and early satiety can affect feeding. Weakness of the tongue, facial and masticator muscles can lead to prolonged feeding time and coughing or choking while eating. Chewing and swallowing difficulties could also arise. Emotional and metabolic stress and trauma can compound the eating and nutritional problems as these can have an effect on the nutritional requirements.

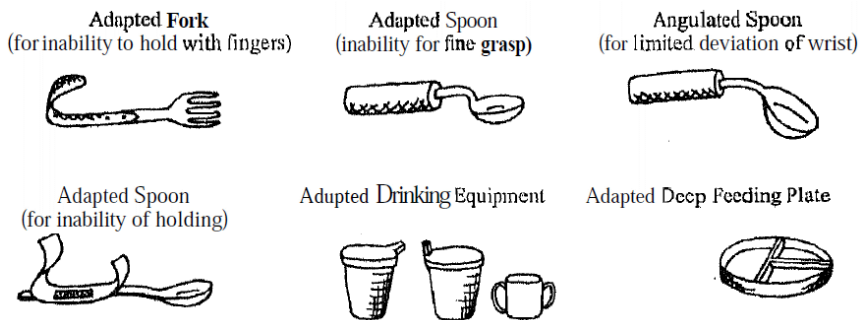
In view of the long term disabling consequences, most neurological disorders require Nutritional Management of special rehabilitative care. Personalized nutrition care plays a vital role in this rehabilitation, for healing and recovery, along with physical, mental and social support and involvement of the patient and the family.

The important goals of nutritional care are to:

- restore or achieve optimal potential of the patients physical, mental and social abilities, and
- improve the quality of life of the patient.

In order to meet these goals, the following are necessary:

- Assessing the nutritional, physical and other parameters of the individual and monitoring the same at intervals to assess the improvement.
- Regular evaluation to improve patient outcome in term of quality and quantity of food consumed, weight changes, clinical assessment.
- Dysphagia assessment for textural and other food or feeding modifications.
- Nutritional therapy recommendations based on individual needs. For example, nutritional deficiencies like anemia may have to be identified as iron is required by the neurotransmitters serotonin and dopamine.
- Nutrition counseling of the patient, family and caregiver.



**Figure 173: Examples of modified eating equipment**

A few additional points related to the patient care that need to be kept in mind include paying attention to the patients living situation, facilities, help, economic status, psychosocial aspects and emotional adjustments. Neglect or overprotection should be avoided to help develop a positive sense of self and the will to fight against a difficult situation. A combined effort of the rehabilitation team including the occupational and speech therapists is desirable, besides the nutrition professional. Next, let us move on to the most common neurological disorder, that is dysphagia. But before that, let us quickly review what we have learnt so far.

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## **17.6 DYSPHAGIA**

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Dysphagia is the inability to swallow or difficulty in swallowing. It is a common problem in those with neurological disorders and can occur in any of the three phases of swallowing: oral, pharyngeal and oesophageal. Many patients may not be able to consume adequate intake of food and hence special feeding methods such as enteral feeding may have to be adopted.

### **17.6.1 Etiology and Clinical Features**

The cause of this neurological disorder can be mechanical or paralytic. The mechanical cause is primarily due to surgical resection or to alteration of one or more organs of swallowing owing to trauma, obstruction, cancer or other disease. The paralytic type results from a lesion in the cerebral cortex or cranial nerves of the brain stem in particular the medulla oblongata. The most common cause of the paralytic type is accident, head injury, brain diseases which may affect the nerves causing dysphagia. If we understand the swallowing process, we can understand the problems that arise in neurological damage better. So let us get to know more about the swallowing process.

Swallowing has three phases:

- i) **Oral Phase:** In this, food is placed in the mouth, mixed with saliva, chewed if required and formed into a bolus by the tongue. The tongue pushes the food to the rear of the oral cavity against the hard and soft palate.
- ii) **Pharyngeal Phase:** This is initiated when the bolus is propelled into the oesophagus. The soft palate gets raised at this stage to close off the nasopharynx to prevent nasopharyngeal regurgitation.
- iii) **Oesophageal Phase:** This is the final phase in which the bolus continues through the oesophagus into the stomach. This phase is involuntary.

In neurological disorders, damage to cranial nerves can lead to weak and poorly co-ordinated tongue movements, which results in problems in completing the oral phase of swallowing. Weakened lip muscles cause an incomplete mouth closure with sucking difficulties. There may be a difficulty in forming a cohesive bolus and moving it through the oral cavity. Loss of sensation and facial weakness can result

## NOTES

in food being pocketed in the buccal recesses of the mouth, without the patient being aware of it. Poorly co-ordinated pharyngeal phase can cause gagging, choking and nasopharyngeal regurgitation. Aspiration of food into the lungs is a complication of dysphagia, unless care is taken.

Thus, some symptoms commonly associated with dysphagia are drooling, coughing, and choking while eating, oral food retention, gurgling voice quality and feeling of lump in the throat. There is an increased risk of aspiration and pneumonia.

Having studied the etiology and symptoms, let us next learn about the feeding and nutritional management of dysphagia,

### 17.6.2 Feeding and Nutritional Management

A multidisciplinary approach is essential for successful management of dysphagia. Patient, nurse, physician, dietitian and swallowing therapist need to coordinate in this endeavour. For feeding, patient should be upright at 90 degree angle with hips flexed, feet flat on the floor and head slightly forward. Food of liquid consistency often causes the greatest problem. Hence, attention has to be paid to the consistency and texture of food as enumerated herewith.

**Consistency and Texture:** Aspiration of liquids, including water, into the lungs can result in complications. To avoid this problem and yet fulfill the fluid needs, liquids could be thickened with starch or milk powder. Intake of caffeine beverages is best limited as these have a diuretic effect and may lead to mild dehydration and fatigue. Foods that form a cohesive bolus within the mouth must be selected. Those that break apart such as plain rice, chopped meat should not be given. Sticky foods that adhere to the mouth should be avoided as they cause oral manipulation problems and fatigue.

Dual texture must not be given such as canned fruit with juice, soup with noodles, and dry cereal with milk. One could moisten food with gravy to facilitate a bolus.

A variety of food items should be served in an appetizing manner with as many characteristics to a normal food. Patients should be gradual progressive transitions in the texture and consistency of food viz., pureed to ground to soft textured foods and eventually to all textured foods.

Hence, paying attention to improved and appropriate taste, texture and temperature of food facilitates swallowing. Cool temperature, sauces and gravies which lubricate food and prevent its fragmentation in the oral cavity, makes swallowing easier.

In acute cases of dysphagia, if oral intake is not possible or inadequate, or if there are increased metabolic demands for nutrients, then nutritional support may be required. Enteral nutrition needs to be given.

**NOTES**

**Table 17.2: Some dietary tips for dysphagia**

| Dysfunction                                                                                | Dietary Tips                                                                                                                                                                                                                                                                                                                  |
|--------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>● Slow / weak / uncoordinated swallowing</li> </ul> | <ul style="list-style-type: none"> <li>- Semisolid consistency to form a cohesive bolus</li> <li>- Textured foods as diced cooked vegetables</li> <li>- Small frequent meals</li> <li>- Well seasoned, flavoured, aromatic, sweet foods.</li> <li>- Cold temperatures</li> </ul> <p>Avoid: sticky, bulky and thin liquids</p> |
| <ul style="list-style-type: none"> <li>● Poor oro-motor Control</li> </ul>                 | <ul style="list-style-type: none"> <li>- Semisolid foods that form a cohesive bolus</li> <li>- Small frequent meals</li> </ul> <p>Avoid: slippery and sticky foods and purees and thin liquids</p>                                                                                                                            |
| <ul style="list-style-type: none"> <li>● Reduced oral Sensation</li> </ul>                 | <ul style="list-style-type: none"> <li>- Food placed in most sensitive area of mouth</li> <li>- Different textures, not mixed, to maximize sensation</li> <li>- Highly seasoned foods</li> <li>- Cold temperatures</li> </ul> <p>Avoid: Hot foods and mixing of different textures</p>                                        |

**Table 17.3: Food consistency and textures**

| Desirable Foods                                                                                                                                                                                                                                                    | Undesirable Foods                                                                                                                                                                                                                             |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>○ <i>Foods forming a cohesive bolus:</i><br/>Egg dishes, soft cheese and paneer, pasta dishes, rice with gravy, ground meats with gravy, gelatin based desserts, hot cereals and vegetables in a gravy or sauce.</li> </ul> | <ul style="list-style-type: none"> <li>● <i>Foods that fall apart:</i><br/>Dry crumbly bread, dry cracker biscuits, thin pureed foods, plain chopped raw vegetables and fruit, plain ground meat, thin cereals and plain dry rice.</li> </ul> |
| <ul style="list-style-type: none"> <li>● <i>Medium thick liquids:</i><br/>Vegetable juice, blenderized cream soups, blenderized fruit juices, milk shakes and soft custard.</li> </ul>                                                                             | <ul style="list-style-type: none"> <li>● <i>Sticky and Bulky Foods:</i><br/>Fresh white bread, plain mashed potato, banana, bran enriched cereals, large pieces of meat, raw vegetables and fruit.</li> </ul>                                 |
| <ul style="list-style-type: none"> <li>● <i>Semi soft/thick liquids:</i><br/>Curd/yoghurt, pureed fruit, ice cream, soft smooth desserts, frozen shakes and frozen juices.</li> </ul>                                                                              | <ul style="list-style-type: none"> <li>● <i>Thin Liquids:</i><br/>Apple, orange and other citrus juices, milk, tea, coffee, water and soda.</li> </ul>                                                                                        |

## NOTES

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### 17.7 ALZHEIMER'S DISEASE

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Named after the German neurologist who first described it, Alzheimer's disease is the most common cause of progressive dementia, due to the degeneration of nerve cells in the brain and shrinkage of brain matter. Extra cellular deposits of amyloid forming protein or amyloid plaques are reported in the cerebral vessels. Let us now find out what factors lead to this disorder and what its clinical features are.

#### 17.7.1 Etiology and Clinical Features

The probable risk factors include a genetic basis, head injury, low education level, down syndrome and mother's age at birth. However, no single factor has been proven to be responsible for this disease.

The clinical manifestations of Alzheimer's disease along with the nutrition related changes may be divided into three stages. Impairment of a wide range of neurological functions is involved, being a disease of the cortical neurons. The three stages are:

Stage I— There is an increased forgetfulness, anxiety and depression. Associated nutrition related changes include difficulty in food preparation, forgetting to eat, taste and smell changes, altered food choices and impaired appetite regulation.

Stage II — There is a memory loss, especially for the recent events. There is disorientation and personality changes occur. Dietary manifestations include an increase in energy requirements as a result of agitation, holding food in the mouth, forgetting to eat and swallow, forgetting the use of eating equipment except perhaps a spoon and eating with hand.

Stage III— This is characterized by severe mental confusion, psychosis, memory loss, personal neglect and distinct feeding problems. There may be no recognition of food with refusal to open the mouth for eating,

Persons with Alzheimer's disease thus have impaired ability to recognize hunger, thirst, or satiety. They are prone to dehydration. As the disease progresses, their attention span reduce, they are easily distracted and forgetful and may stop eating or not eat enough or try and eat inedible items. Eventually nutritional support may be required to sustain them.

The nutritional management of such patients is discussed next.

#### 17.7.2 Feeding and Nutritional Management

Keeping the clinical manifestations of Alzheimer's disease in mind, treatment involves personalized care, keeping the patient well nourished, reducing anxiety and stress and improving the quality of life. The main objectives of nutritional management, hence, are to:



provide adequate nutrition,  
prevent malnutrition, and  
devise methods to tackle feeding problems.

Several strategies may have to be used to achieve these objectives, keeping the functional impairments in mind. Some of these, based on the stage of the disease and individual needs are:

- supervising meal times with minimal distractions,
- assessing chewing and swallowing ability and providing foods of appropriate consistency,
- initiating the activity of eating, by making the person touch or taste the food,
- giving one food at a time in small bowls so as to avoid stress of food choices,
- supervising to avoid eating of spill food or inedible items,
- giving only a spoon or finger foods, in case of inability to use other eating equipment,
- encouraging individual appropriate feeding techniques,
- permitting adequate feeding time to increase intake,
- use of nutrient dense foods, frequent snacks and nutritional supplements to avoid malnutrition ,
- avoiding finger foods and using only a small spoon in case of tendency to take a large bolus, and
- guarding against aspiration, in case of dysphagia.

To ensure adequate food and nutrient intake, continuous assessment of nutritional status is desirable, supported by behaviour modification, if required. Patient guidance and supervision including continuous verbal instructions during each step of feeding may also be needed, like to eat, chew and stop chewing.

Let us move on to the discussion of the next disorder i.e. Parkinson's disease.

To ensure adequate food and nutrient intake, continuous assessment of nutritional status is desirable, supported by behaviour modification, if required. Patient guidance and supervision including continuous verbal instructions during each step of feeding may also be needed, like to eat, chew and stop chewing.

Let us move on to the discussion of the next disorder i.e. Parkinson's disease.

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## **17.8 PARKINSON'S DISEASE**

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Parkinson's disease is a degenerative central nervous system (CNS) condition characterized by progressive loss of cells within substantia nigra. Substantia

## NOTES

nigra is a portion of the midbrain, as illustrated in Figure 17.4, which is thought to be involved in certain aspects of movement and attention. It consists of two subdivisions, the pars compacta and the pars reticulata. The cells within the substantia nigra release the neurotransmitter dopamine and it is the loss of dopamine that is primarily responsible for the motor defects.

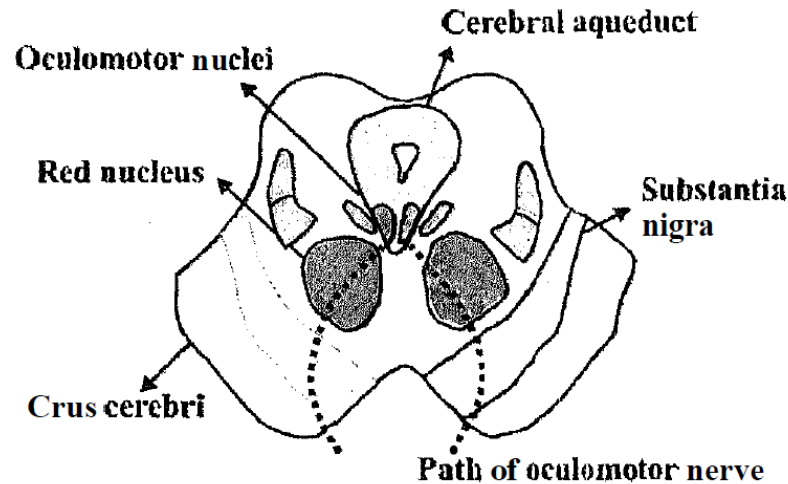


Figure 17.k The substantia nigra

The disease is much more common in senior citizens and is slightly more prevalent in men than women. What are the causative factors surrounding the clinical features of this disease? Read the next section and find out.

### 17.8.1 Etiology and Clinical Features

The cause of Parkinson's is unknown. Genetic predisposition (in most cases the reason for the death of these dopamine neurons is unknown), and exposure to neurotoxins and industrial toxins are said to be important risk factors. Viral infection such as encephalitis can also produce the disease condition.

The common clinical features of the disease include:

- slowness of movement
- inability to initiate movements
- muscular rigidity
- resting tremor
- postural instability
- weight loss.

Parkinsonism describes the common symptoms of Parkinson's disease such as tremor, rigidity, akinesia (inability to initiate movements) or bradykinesia (slow movement) and postural instability.

Considering the pathology of Parkinson's disease, the feeding and nutritional care

is a crucial aspect in the management of the patients suffering from this disease. The next section focuses on the nutritional management of Parkinson's disease.

### **17.8.2 Management: Drug, Feeding and Nutritional Care**

There is no cure yet for Parkinson's disease, but its symptoms can be minimized with drug therapy. Levodopa or a precursor of dopamine is used mostly. Once levodopa enters the brain it can be decarboxylated to dopamine thus replenishing the depleted neurotransmitter dopamine. Levodopa may produce gastric symptoms and nausea, which can interfere with food intake. For many patients these symptoms are mild and tolerance to nausea does develop. The large amino acids generated from metabolic breakdown of proteins can inhibit the absorption of levodopa and hence is best to have it 1 hour before meals. Very large protein meals can reduce the effect of levodopa, hence managing/manipulating the proteins (intake) well can give a better performance in patients. For example patient wishing to remain in an optimum state of activity could benefit from redistributing the protein. Day time restriction of dietary protein- 10 g or less upto 5 pm has been shown to improve the efficacy of levodopa. After 5 pm the remaining day's protein requirement can be consumed. This way the patient can have adequate performance of day time activities.

Hence from our discussion above, it is evident that nutrient-drug interaction is an important aspect that we need to consider in the nutritional management of Parkinson's disease. We have already touched on this aspect earlier in Unit 7.

Weight loss is also an occasional problem with patients. This could be due to increased calorie needs resulting from involuntary movements, difficulty in feeding, nausea, medicine related factors, dementia, depression and dysphagia could be causative factors.

Constipation is also a problem in Parkinson's disease patients due to low grade autonomic function or medication that may contribute to constipation. Difficulty in swallowing too can reduce fluid and fibre intake leading to constipation.

As the disease progresses some food related difficulties appear. These are:

- difficulty in food preparation and eating due to tremors,
- gradual development of chewing and swallowing difficulties and risk of aspiration,
- tendency for constipation, and
- prolonged meal times, up to 1 hour, due to muscle rigidity leading to an impaired head and neck control and hence feeding difficulties.

These problems have to be taken into consideration while planning meals. For example:

- Foods rich in fibre and which can be cut into pieces and made into cohesive

## NOTES

bites could be given.

- Very liquidy foods may be difficult to handle, but care should be taken to ensure fluid intake is adequate to prevent constipation and hypertension (low blood pressure).
- Small frequent meals with more carbohydrates and less fat may be better tolerated, in view of the gastric side effects and delayed gastric emptying.
- Diets given should be balanced and nutritionally adequate.
- Frequent use of high-protein snacks has deleterious effects upon Parkinson disease control. Hence this should be avoided.
- Supplementation of vitamin B6 (pyridoxine) should be avoided as this vitamin can facilitate the premature conversion of levodopa to dopamine thus reducing the potency of the drug.

Thus, the dietary goals in Parkinson disease can be highlighted as under.

### *Dietary Goals in Parkinson's Disease*

The main goals include:

maintain desirable weight

promote absorption of anti-parkinson drug levodopa

lessen swallowing difficulties as a result of disease or medication,

alter food consistency or texture,

drink sufficient fluids or have good source of fibre for effective bowel function,

and prevent constipation, and

redistribute the protein

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## 17.9 EPILEPSY

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Epilepsy is a neuromuscular disorder in which transient seizures recur, due to an abnormal brain activity. The brain, through an orderly electric excitation of its nerve cells, controls all activities of the body. When however, due to some reason, the discharge is unregulated and chaotic, an epileptic seizure can occur. Seizures may occur spontaneously or may be triggered by a stimulus. What factors lead to these and what are its clinical features? Let us read and find out in our next section.

### 17.9.1 Etiology and Clinical Features

This disorder usually starts in childhood, with the peak incidence between birth and two years. Etiological factors include birth trauma, head injury, brain infection, and metabolic imbalance in the body, neurotoxins or a genetic basis. It may also be idiopathic or of unknown origin. About 1/3rd persons outgrow this

condition and do not require medication. In 1/3rd drugs can control the seizures. In the remaining, the condition usually gets worse.

Any of the etiological factors in epilepsy, can result in intermittent derangement of the nervous system due to a sudden excessive disorderly discharge of cerebral neurons.

Different types of seizures may occur:

- 1) Generalized or tonic-clonic seizures, where the entire brain cortex is involved and post seizure disorientation may last for a few minutes to few hours,
- 2) Petit nzal or absence seizures which involve no post seizure fatigue or disorientation, and
- 3) Partial seizures, where there is an epileptogenic focus in the brain tissue, but electrical activity may spread across the entire brain.

The type of seizure determines the drug therapy and nutrient requirement.

Anticonvulsant drug reactions are of relevance in nutritional management.

The general symptoms of epilepsy include weakness, fainting, uncoordinated musclemovement. Based on the drug, common side effects include nausea, vomiting, anorexia or increased appetite, diarrhoea or constipation, decreased taste sensation, increased vitamin D and K catabolism, low levels of serum calcium, vitamins B, and B12 and folate. Long-term usage may lead to rickets in children and cause liver damage.

The management of epileptic patients is described next.

### **17.9.2 Management: Drug, Feeding and Nutritional Care**

The primary treatment of epilepsy is anticonvulsant drugs. The focus of nutritional management is a diet, which is appropriate for growth during childhood and maintenance in adults. Nutrient drug interactions also must be considered and remedial steps must be taken. Whereas anticonvulsant drugs may cause side effects of nutritional significance, folic acid supplements can interfere with the action of one of the drugs— Phenytoin. Food intake can delay the absorption of phenobarbital. Low serum albumin due to a state of malnutrition can result in a higher drug concentration in the blood and thus toxicity.

In mild/moderate epilepsy, 'ketogenic diets' are sometimes recommended. A ketogenic diet is a high fat diet, with a of or 3:1 of fat to carbohydrate and protein calories. Usually about 75% of the recommended energy intake for weight and height is given. Protein given is about 1g/ kg for growth. This leaves a minimal amount of carbohydrate to make up the calories. This diet may be used for upto three years and has been reported effective in young children. A ketogenic diet is designed

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to produce ketone bodies as a result of incomplete oxidation of fat although the exact mechanism is not known. The ketone body produced by incomplete oxidation of fat (acetone, acetoacetic acid and  $\beta$  hydroxybutyric acid) are thought to have an anticonvulsant action and hence are beneficial.

The ketogenic diet is initiated after an initial period of fasting for 24-72 hours, till ketosis is established. There are two types of ketogenic diets:

- a) traditional diet, using long chain triglycerides, and
- b) the medium chain triglyceride diet (containing coconut and palm kernel oil) which results in ketosis easily.

Next, let us look at the dietary recommendations.

***Dietary Recommendations***

A ketogenic diet is recommended. The time taken for reversing the usual ratio of 1:3 to 3:1 is about 4 days. How will we know if our ketogenic diet is working? Very easy, just test the urine for ketone bodies. There are a few foods to be avoided in a ketogenic diet. These are listed in Table 17.4 along with those foods which may be given as desired.

**Table 17.4: Foods to be included and avoided in a ketogenic diet**

| <i>Foods to Avoid for Ketogenic Diet</i>                                                                                                                                                                                                                                                                                                                                                                                                                             | <i>Foods to be Given as Desired</i>                                                                                                                                                                                                                                                                                                                                                                               |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <p>The following foods contain substantial amounts of carbohydrates and <b>should be avoided.</b></p> <ul style="list-style-type: none"> <li>- all breads and cereals</li> <li>- cakes or cookies, <b>pastries</b></li> <li>- carbonated beverages, <b>sherbet</b> and <b>sweet juices</b></li> <li>- puddings and pies</li> <li>- candy and chewing gum</li> <li>- jams, jellies, marmalade and honey</li> <li>- syrups, sugar and condensed <b>milk</b></li> </ul> | <p>The following foods contain negligible amounts of protein, <b>fat</b> and carbohydrate and may be used more frequently.</p> <ul style="list-style-type: none"> <li>- broth or <b>consommé</b></li> <li>- coffee (normal and decaffeinated) and tea</li> <li>- unsweetened cocoa powder and gelatin</li> <li>- mustard dry, salt and pepper, parsley and other herbs, and</li> <li>- <b>vinegar.</b></li> </ul> |

For preparing meals you could choose one of the following food/food items:

- Meat/cheese, chicken (30 g), egg (1), fish (50 g), cottage cheese (50 g), processed cheese (30 g), pulse (30 g)
- Cereal: bread (25 g), wheat (20 g), crackers (20 g), rice (50 g cooked). Noodles (50 g cooked)

- Vegetables (100 g): beans, broccoli, cabbage, cauliflower, cucumber, eggplant mushrooms, mustard greens, radish, spinach, tomatoes, turnip.
- Fats: Butter (5 g), cooking fat (5 g), nuts-almonds, walnuts (5 g), whipped cream , (60 g)
- Fruits: Apple (40 g), apricots (60 g), banana (30 g), gooseberries (50 g), cherries (40 g), grapes (40 g), orange (100 g), mango (35 g), melon (100 g), papaya (60 g), peach (60 g), pineapple (40 g), plums (40 g)
- Milk: Butter milk (120 g), skimmed milk (120 g).

With these dietary recommendations we end our study of epilepsy. Finally, let us review the neuro trauma and the spinal trauma and the nutritional management of patients suffering from these traumas.

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## 17.10 NEURO TRAUMA

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Neuro or head trauma includes brain injury, skull fractures, extraparenchymal or internal brain haemorrhage, Brain injury can be divided into three types. These include:

Concussion means brief loss of consciousness (< 6 hours),

Contrusion is similar to a bruise on the skin, and

Comminution means splintering of bone in many fractures.

Like other cases of major injury and trauma, as you now know from Unit 5, brain injury or trauma also results in a systemic hypemetabolic, hypercatabolic response. This affects the entire body as body reserves get mobilized. If this resultant hypermetabolic state remains unchecked, a sequence of organ failure can result.

Neuro trauma results in production of cytokines, these effect the metabolism.. Some of the effects of this are fever, neutrophilia (type of white blood cells which provide important defence mechanism are increased in number), muscle breakdown, altered amino acid metabolism, increased organ demise. How to prevent these conditions? Let us read and find it out in our following sub-section on nutritional management.

### ***Feeding and Nutritional Management***

The main objective of nutritional management is to counteract the hypermetabolism associated with inflammation. The basal energy expenditure (BEE) in neuro trauma patients can be 170-160% of the normal, along with a negative nitrogen balance and weight loss.

The nutritional treatment has mostly two phases. Let us get to know what these are.

- Initial Phase — In this, the life threatening conditions need to be controlled first. Soon the nutritional support must start because hypermetabolism

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contributes 10 excess energy expenditure and hypercatabolism to increased protein demand. This increased energy ( $1.75 \times 2 \times \text{BEE}$ ) and protein demand ( $1.5\text{-}2.5 \text{ g/kg 1B W}$ ) must be met initially via nutritional support methods or enteral / parenteral , nutrition. If nutritional replacement is not provided, 10% decrease in lean body mass can occur within a week, with up to 30% loss in two to three weeks. This is associated with increased mortality.

- Rehabilitation Phase—Once the patient stabilizes and starts recovering, besides nutritional replacement, there is a need to assess functional disabilities related to eating. Dysphagia, difficulty in chewing, physical handicaps in eating can arise as an outcome of the injury. Accordingly, food consistency and texture may need to be adjusted to ensure adequate intake.

In the end, let us review the spinal trauma and its management.

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## 17.11 SPINAL TRAUMA

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Spinal trauma or spinal cord injury, commonly due to accidents, falls, sports injury can result in serious disabling consequences. The spinal cord damage disrupts the transmission of nerve impulses from the brain to the peripheral nerves and muscles, resulting in a loss of muscle function, depending on the site of injury. Immobilization commonly occurs due to quadriplegia (paralysis from neck down) or paraplegia (paralysis of the lower part of the body). Metabolic consequences of the trauma include negative nitrogen balance, low serum albumin, loss of calcium, loss of bone and skin collagen and weight loss. Malnutrition is a frequent outcome. The spinal cord responds to insult in a similar manner as the brain. Bleeding and confusion may appear first and then fibrosis. In general, frequency of complications which affect the nutrition may vary and these are constipation, pressure, ulcers, obesity and pain.

So then let us get to know how to manage this condition.

### *Feeding and Nutritional Management*

The main objectives of nutritional management are to meet the nutritional needs of the initial acute phase and then the rehabilitation phase. During the latter phase, special feeding requirements have to be considered along with the requirements of associated complications. What are these phases? Let us read and find out.

- Acute Phase—Nutritional support should start within 3-5 days or as early as possible to prevent the onset of malnutrition and secondary illness. Frequent assessment of the metabolic rate is desirable, as it tends to vary. This can avoid the complication of overfeeding. Reported recommendations for adults are about 23 Kcal/kg for quadriplegics and 28 Kcal/kg for paraplegics, keeping in mind that increase in metabolic rate is less compared to other trauma conditions.
- Rehabilitation Phase—Once the patient's condition stabilizes, individualized diets need to be recommended, based on the energy and nutrient requirements and



feeding capacity.

It is usually desirable to adjust energy to maintain the weight 10-15 lbs below the IBW to prevent excess weight gain, which can add to the medical problems and physical handicap. Protein intake should be based on requirements and must be adequate to maintain muscle mass and tissue integrity. A negative nitrogen balance should be guarded against.

Minerals and vitamins must be adequate based on requirements. Supplements may be needed if the dietary intake is poor.

Common complications with spinal trauma patients include pressure sores or decubitus ulcers, hypercalciuria and renal stones, constipation due to neurogenic bowel and depression. Nutrition has a relevant role in all conditions. Pressure sores develop due to immobilization, loss of pressure sensation, poor circulation and skin breakdown. Anaemia occurs resulting in less oxygen supply to the sore area, low serum albumin, excess weight loss also contribute to the complications.

The nutritional management lies in making up the protein deficit. About 1.52 g protein/kg is recommended with supplements of vitamin C and zinc. Hypercalciuria and thus a tendency for renal stones, arises due to prolonged immobility. A balanced intake of calcium and phosphorus is beneficial. While protein requirements may be increased, excess should be avoided, as it can cause calcium withdrawal from bones.

Constipation arising due to neurogenic bowel requires a regular bowel schedule with high fibre and fluid intake.

Depression often follows recovery as the person comes to term with disability. An adequate balanced diet resulting in slight increased muscle mass, with concerned nutritional care can help improve quality of life.

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## **17.12 LET US SUM UP**

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In this unit, we learnt about nervous system and the related disorders, which are termed as 'neurological disorders'. Neurological disorders may be of nutritional or non-nutritional etiology, but both require nutritional intervention. Common non-nutritional neurological disorders, some being progressive in nature, in which feeding and nutrition are important, are Alzheimer's disease, Parkinson's disease, epilepsy, neuro and spinal trauma.

Here, we realized that dysphagia is a problem common to many of the disorders. Personalized nutrition care plays an important role, its objectives being to maintain adequate nutrition, prevent further disability and restore potential function. Social, psychological and emotion support improves patient outcome, best achieved by the combined efforts of a rehabilitation team including the family, occupational and speech therapists and nutritionist.

Finally, we learnt that individual nutritional needs have to be assessed and adequate energy, protein, energy sources and minerals and vitamins provided to

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promote functional, tissue and organ integrity. Eating skills and eating desire also requires assessment and persons may require help to develop new ways of eating using special foods or eating equipment and utensils. Nutritional support may be needed in some neurological diseases. Drug nutrient interactions may also require attention. Social, psychological and emotion support improve patient outcome, which can be best achieved by the combined efforts of a rehabilitation team including the family, physicians, nurse, dietitians, occupational and speech therapist, caregivers, family.

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### 17.13 GLOSSARY

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|                            |                                                                                                                                                                                                                                  |
|----------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Amyloid</b>             | : a starch-like glycoprotein.                                                                                                                                                                                                    |
| <b>Alzheimer's disease</b> | : a neurological disorder arising due to degeneration of nerve cells in the brain and shrinkage of brain matter, with formation of amyloid plaques in the cerebral vessels. It is the most common cause of progressive dementia. |
| <b>Apraxia</b>             | : neurological impairment characterized by inability to perform activities in spite of ability to understand and carry out the activity.                                                                                         |
| <b>Bradykinesia</b>        | : an abnormal slowness of movements; may be due to neurological damage.                                                                                                                                                          |

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### 17.14 CHECK YOUR PROGRESS

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- 1). Enlist three common neurological disorders of
  - a) nutritional origin
  - b) non-nutritional origin:
- 2). State five consequences of neurological damage which can affect feeding or nutrition.
- 3). Give two important goals of nutritional care of persons with neurological disorders.
- 4). What do you understand by the term 'dysphagia'? Why are the thicker fluids preferable in dysphagic patients?
- 5). What are the main objectives of nutritional management of Alzheimer's disease

# 18

## PAEDIATRIC AND GERIATRIC NUTRITION

### NOTES

#### STRUCTURE

- 18.1 Learning Objective
- 18.2 Introduction
- 18.3 Paediatric Problems and Nutritional Management
- 18.4 Geriatric Nutrition
- 18.5 Let Us Sum Up
- 18.6 Glossary
- 18.7 Check Your Progress

### 18.1 LEARNING OBJECTIVE

After studying this unit you will be able to:

- Discuss a few common paediatric problems and their nutritional management,
- Enumerate the nutritional assessment tools for the elderly, and
- Elaborate on the nutrition support for the elderly.

### 18.2 INTRODUCTION

Every stage has its unique requirements due to different changing needs. Adequate and optimum nutrition support is very important during the early critical periods of life to achieve normal growth and development. Besides, certain groups of children with different types of medical conditions may have feeding difficulties. In this last unit, we will focus on paediatric geriatric problems and their nutritional management. Many of these problems you may have already studied earlier in this course. Here in the first part of this unit we shall review them, particularly in the context of paediatric nutrition.

Due to the changes in physiological function with aging, effect on absorption,

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retention and utilization of nutrients commonly occurs. With age the requirements for macro- and micronutrients also changes. Further, malnutrition and other problems amongst elderly persons have been observed — be it hospitalized patients, nursing home residents or outpatients. With the help of nutritional assessment the elderly at risk can be given preventive and treatment care as required. The second part of this unit deals with nutritional assessment tools and nutrition support for the elderly.

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### 18.3 PAEDIATRIC PROBLEMS AND NUTRITIONAL MANGEMENT

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The process of accepting and digesting food in adequate amounts to meet nutrition needs is termed as feeding. Certain groups of children with different types of medical conditions may have feeding difficulties for example infants/ with cardiopulmonary, genetic or metabolic disorder may have poor intake and may lead to slower weight gain. Other conditions like various neurological conditions such as cerebral palsy, structural abnormality or brain injury may affect the motor or swallowing reflex.

The first step in treatment is to assess the child's feeding disorder. This requires a multidisciplinary team who need to diagnose assess and develop appropriate treatment plan. In the following sub-section, we shall learn about a few common paediatric problems and their nutritional management. These problems include congenital heart disease, low birth weight/preterm, lactose intolerance and celiac disease. We begin with congenital heart disease.

#### 18.3.1 Congenital Heart Disease (CHD)

The relationship between CHD, malnutrition and growth is well documented. Different types of cardiac defects are associated with different patterns of growth retardation. Here we are not going into the details related to these cardiac defects. What we must, however, know, is that, patients with acyanotic heart disease show greater growth deficit in weight. Cyanotic heart disease patients have greater growth deficit in stature — demonstrated by both decreased height and weight.

Those infants born with CHD also show a greater incidence (6-14%) of intra uterine growth retardation (IUGR), viral illness and chromosomal abnormality.

The various factors related to causing malnutrition among CHD patients are hypoxia (reduction in oxygen supply) and haemodynamic factors, nutritional intake, metabolic requirements and impaired nutrient absorption.

Nutritional and metabolic factors that contribute to the malnutrition of CHD infant are elevated BMR, unable to coordinate to suck, swallow and laborious breathing/ sucking, delayed gastric emptying and impaired gastrointestinal motility, decreased gastric capacity — leading to early satiety, thus compromised

energy and protein intake. Hence, the intake is below the requirement and is different from normal.

### Specialized Nutritional Requirements

The nutritional requirement for catch up growth for infants with CHD is listed in Table 18.1. By catch up growth we mean to catch up or make up for the earlier deficit in growth caused by CHD. Calorie and protein requirements (given as per kg body weight) are increased due to catch up growth and hyper metabolism. Sodium intake is to be closely monitored due to existing congestive heart failure (CHF). Other important nutrients are potassium, vitamins, and iron. Fluid requirements are individualized based on degree of cardiac compromise, diuretic therapy, fluid intolerance, congestive heart failure (CHF), pulmonary hypertension,

### NOTES

**Table 18.1: Nutritional requirements for catch up growth for infants with CHD**

| Age (yr)           | 0-0.5 | 0.5-1 | 1-3  |
|--------------------|-------|-------|------|
| Energy(Kcal/kg)    | 108   | 98    | 102  |
| Protein(g/kg)      | 2.2   | 1.5   | 1.23 |
| Sodium(mg/day)     | 230   | 500   | 650  |
| Potassium (mg/day) | 650   | 850   | 1100 |

The following calculation/formula may also be useful to calculate the calorie and protein content:

$$\text{Catch-up growth (Kcal/kg)} = \frac{\text{RDA Kcal/kg for weight} \times \text{ideal weight}}{\text{Actual weight (kg)}}$$

$$\text{Protein (g/kg)} = \frac{8\% \text{ of catch-up growth calories}}{\text{Catch-up growth (Kcal/kg)} \times 0.08}$$

Source: Food and Nutrition Board, Recommended Dietary Allowance, 10<sup>th</sup> ed. National Academy of Science, 1989; 284.

Achieving calorie and protein requirements with restricted fluid intake, you would realize, is a formidable challenge. High calorie intake (0.8-1 Kcal/ml), we learnt above, is recommended as it helps ill weight gain. Increasing formula calorie density can be achieved by either formula concentration or formula supplementation.

### Formula Concentration/ Supplementation

Formula concentration can be done by decreasing the amount of water added in the mixing of the formula. Standard formula has calorie density 0.67 Kcal/ml. The advancement in density should be gradual with careful monitoring, ensuring

## NOTES

gastrointestinal and metabolic tolerance. As for proteins, diet should contain between 8-10 % . Attention needs be paid to the sodium content of the feed on increasing the formula by concentration method. In infants with severe CHD and failure to thrive, formula density should not exceed 0.8 Kcal/ml. Severely malnourished CHD infants may develop lactase deficiency and thus use of soya based infant formulas is suggested.

Increased calorie density of a formula may be achieved through supplementation with carbohydrate or fat module. Addition of glucose and lipid maintains a lower renal solute load. On adding more than one module component, maintenance of calorie distribution is gradual and stepwise. Care is to be taken that with supplementation the percentage of calories from proteins does not fall to less than 6-7 (energy percent or percentage of energy). Breast feeding/expressed breast milk is the best food for the infant. Breast milk contains 0.66 Kcal/ml; although provides nutritional and immunologic advantages it is not able to meet adequate calorie and protein intake. Thus breast-feeding may be alternated with high calorie density formula to ensure total intake of 120-140 Kcal/kg /day.

### ***Post-surgical Nutrition Support***

Nutrition support should be started as soon as possible. If oral not possible, enteral nutrition support should be provided. Nutritional support can be enhanced with continuous intermittent nasogastric feeding either by 24 hours/12 hrs continuous feeding, Paediatric and Geriatric 12 hours nocturnal continuous feeding, or by Bolus feeds. In case enteral support cannot be provided for more than one week, initiate TPN.

Solid food feeding/weaning to be initiated at the same time as normal term infant

To sum up, CHD contribute significantly to malnutrition, morbidity and mortality. It is important to recognize patients who are undernourished or at risk, so that they can receive appropriate treatment. The surgery outcome is also observed to be better with improved nutritional status. Thus it is important for proper diet counseling to provide ideal nutrition therapy to improve nutritional status of (CHI) patients.

Next, let us learn about the nutritional support for the preterm/low birth weight infant.

### **18.3.2 Preterm/Low Birth Weight**

The foetal and neonatal health is mainly dependent on the birth weight and it has been well recognized that perinatal (from birth upto one year) morbidity and mortality is closely related to low birth weight.

You may have come across the terms — premature, small for gestational age, intrauterine growth retardation etc. What do these terms mean? Let us get to know the definition of these terms.

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'Low Birth Weight' is weight of infant less than 2500g at birth.

'Preznturity' is defined when delivery is at less than 37 completed weeks.

Small for Gestational Age (SGA) is defined as infants affected by intrauterine growth restriction (IUGR).

Intrauterine Growth Retardation (IUGR) is a condition where the growth of the foetus is abnormal, as a result of reduced blood flow through the placenta (which is the source of the baby's nutrition).

The main possible causes and risk factors for LBW/premature births/SGA are said to be poor nutrition, cigarette smoking, alcohol and drug intake, young age of mother, poor stature, and some complication during pregnancy. Some other factors like faetal infections, congenital malfunctions, chromosomal abnormalities etc. are also seen to be present along with LBW or premature deliveries.

What are the health consequences of these conditions?

There is an increased neonatal mortality and growth deficit: and neurological development due to LBW or preterm delivery or due to IUGR. It is also postulated that developing coronay vascular disease (C V D), high blood pressure, diabetes, hyperlipidemia and obstructive lung disease is associated with low birth weight.

Prevention of IUGR, or preterm bilth has been seen in case of mothers who stopped smoking and are on a balanced protein energy diet and with control of urinary tract md vagina infection.

We shall elaborate on the nutritional management of preterm infants next.

Nutrition Management and Feeding the Premature Infant

There are numerous nutritional risk factors in premature infants. These include:

- Elevated metabolic rate, thus increasing the protein, fat, energy requirements.

- Excessive urinary and evaporative losses.

- Immature gastrointestinal tract (poor gastric emptying and improper peristalsis).

- Respiratory distress and hypoxia,

There are number of feeding problems faced by the premature infant due to:

- Poor sucking reflex,

- Difficulty in swallowing and breathing,

- Small gastric capacity,

- Reduced intestinal molility, and

- Getting tired easily after being fed or handled.

As for the nutritional requirements, there is an increased nutrient need due to catchup growth with approximately 110-130 Kcal/kg body weight/day. The nutrient requirements are elaborated further herewith.

## NOTES

Nutrient requirements.

**Energy** : For preterm infants 120 Kcal/kg/day,  
For normal infants 108 Kcal/kg/day.

**Proteins** : Care to be taken to give adequate protein (for proper growth), if excess proteins /insufficient intake, metabolic acidosis [azotemia may occur.

For preterm an intake of 3.5-4.0 gm/kg/day and for normal infant 2.2 gm/ kg/day.

Vitamin and mineral: Care to be taken for calcium and phosphorus intake in preterm infant. Due to inefficient sodium conservation mechanism in preterm infants, the sodium requirement is increased to 3.0-3.5 meq/kg/day. From 2 weeks to 2 months, both preterm and term infants require iron supplementation (2-3 mg/kg/day of supplemental iron.

**Fluid needs** - In case of premature infants:

weighting less than 1000 g fluid needs is 150 ml/kg/day.

weighing more than 1000 g fluid needs is 100-150 ml/kg/day.

for term infant's fluid needs is 100 ml/kg/day.

So then considering the feeding problems and the enhanced nutrient requirement what are the feeding options for premature or LBW infants. Read and find out.

Feeding Options for Premature/LBW Babies

Different workers have tried different methods and since all methods are successful, it is dependent on the individual infant's needs and problems without imposing stress on the infant's metabolic and excretory system.

Some studies have shown that tube feeding can be done for infants born less than 34 weeks of gestation as these infants have sucking, fatigue and swallowing reflex problems. In another study, infants having very low birth weight (less than 1500 g) were initially fed by tube feeding (half strength formula) for 3-4 days until stable and then gradually increased the strength of the formula depending on the tolerance. In case of absence of complications like reflux, abdominal distension, diarrhoea etc, oral intake can be started.

Some researches have observed that SGA infants who weighed between 1800-2500 mg, given on-demand bottle-feeding responded well. Thus, emphasis is more on bottle-feeding and breast-feeding to avoid the various complications of tube feeding. It has been observed that with full term, as well as, premature normal



**NOTES**

infants, on-demand feeding schedule is better than scheduled feedings. The reason is its ability to:

- a controls calorie intake.
- avoid over or under feeding.
- consume adequate fluid and nutrient intake in less time.
- less health care costs. Paediatric and Geriatric Nutrition — Special
- improves fat absorption. Considerations
- reduces risk of NEC (Necrotizing enterocolitis).
- enhances mother-baby bonding.

For preterm infants, it has observed that except for a few nutrients (e.g. vitamin C, D, sodium, folic acid), the breast milk of mother (of the preterm infants) has been found to be higher in nutrients and is unique for low birth weight babies. The weaning age for the preterm infants should be delayed and be based on the corrected chronological age. However, in cases of nervous system disorders, hyperbilirubinemia, heart problems etc, a modified demand feeding is better.

However, if infants cannot be breast fed or bottle-fed, under such circumstances enteral or parenteral nutrition may be provided. Table 1 8.2 gives the calorie and protein intake of preterm infants on enteral and parenteral support.

**Table 18.2: Estimated calorie and protein intake of preterm infants**

| Nutrients      | Body Weight (g) |           |            |
|----------------|-----------------|-----------|------------|
|                | 900-1200        | 1200-1500 | 1500-1800  |
| Proteins (TPN) | 3.5             | 3.4       | 3.2        |
| Proteins (EN)  | 4.0             | 3.9       | <b>3.6</b> |
| Energy (TPN)   | 101             | 108       | 109        |
| Energy (EN)    | 119             | 127       | 128        |

TPN: Total parenteral nutrition EN : Enteral Nutrition

For enteral feeding following need to be considered:

- In preterm calorie density to be 0.8 Kcal/ml,
- LBW formulas used: protein — a ratio of 60:40 whey to casein; fat — having MCT (medium chain triglyceride) for better absorption, LCT (long chain triglyceride) for essential fatty acids.
- In full term infants — formulas providing adequate proteins, carbohydrate,

fats and other nutrients.

### ***Parenteral Nutrition Support***

#### **NOTES**

Parenteral nutrition support is the provision of dextrose, amino acids, electrolytes, vitamins, minerals and trace elements with or without fat. The initial considerations are:

Calorie and protein goals —based on nutritional assessment and keeping in mind

the maintenance and growth needs.

The duration of TPN and thus choosing the different access routes (central/peripheral).

Total fluid allowance.

Parenteral nutrition is more used for preterm infants who are less than 1500 g and less than 30 weeks of gestation age, as their GI tract is immature. As both risks and benefits are found to be associated with TPN, thus, various nutrition routes are used as per required individually. 451

**Glucose:** Initiated at the rate of 6 mg/kg/min and increased upto 12-14 mg/kg/min, but care to be taken to prevent hyperglycemia.

**Proteins :** 1.5-2.0 g/ kg/day and increased to 3.5-4.0 g/kg/day, cysteine is considered to be an essential nutrient for preterm infant.

**Lipids:** The recommendations vary from 0.5-1.0 g/kg/day to 3.00 g/kg/day.  
**Electrolytes:** Sodium 3.0-4.0 mmol/kg/day and potassium 2.0-3.0 mmol/kg/day.

**Vitamins:**The suggested parenteral intake of vitamin is:

|                         |                   |
|-------------------------|-------------------|
| <b>Vitamin A:</b>       | 280-500 µg/kg/day |
| Vitamin E:              | 2.8 µg/kg/day     |
| Vitamin K               | 100 µg/kg/day     |
| Vitamin D:              | 4 µg/kg/day       |
| Ascorbic Acid:          | 25 µg/kg/day      |
| Thiamine:               | 350 µg/kg/day     |
| Riboflavin:             | 150 µg/kg/day     |
| Pyridoxine:             | 180 µg/kg/day     |
| <b>Niacin:</b>          | 6.8 µg/kg/day     |
| Pantothenate:           | 2.0 µg/kg/day     |
| <b>Biotin:</b>          | 6.0 µg/kg/day     |
| Folate:                 | 56 µg/kg/day      |
| Vitamin B <sub>12</sub> | 0.3 µg/kg/day     |

**NOTES**

From our discussion above, it is evident that feeding the IBW infant is challenging. Monitoring of the nutritional status of these infants is essential to observe the growth. The various parameters used are daily body weight record, weekly length and head circumference and periodic biochemical parameter assessment.

We may end our discussion by summing up that specialized nutrition needs for preterm and/or low birth weight infants should be carefully monitored for prevention of morbidity and promoting optimal growth and development.

**18.3.3 Lactose Intolerance**

Lactose is the name of the sugar found in milk. Lactose intolerance, you must be aware by now, occurs when the body is unable to breakdown the lactose that is in consumed foods. In children, lactose intolerance is presented with persistent diarrhoea lasting more than 14 days. Other symptoms may include stomach pain, cramping and gas. Shedding of lactase producing villi in the intestine cause the lactose malabsorption. It is mostly a transitory phenomena and patients recover when the mucosa returns to normal. During the malabsorption period, a lactose free restricted diet is given to control the diarrhoea.

Confirmatory test for lactose intolerance is the test of stools for its pH and reducing substance. It is confirmed for lactose intolerance if the pH is below 5.5, and reducing substance are more than 10% in a stool.

While most infants can tolerate milk-based and dairy products, many children develop Paediatric and Geriatric an intolerance as they mature.

**Diet intervention**

Lactose is present in dairy products such as milk, cheese, yoghurt, ice cream etc. Hidden sources of lactose may include bread, candy, cookies, biscuits, sauces, gravies, soups etc. Hence, depending upon the amount of lactose an individual can handle, major or minor dietary restrictions may be imposed.

Most lactose-intolerant children can digest yoghurt and buttermilk. On settling of the diarrhoea, they should begin with yoghurt which is better tolerated as during its fermentation it becomes richer in bacteria which produces  $\beta$  galactoside - this hydrolyzes lactose. Later milk (50 ml/kg/day) may be tried if tolerated.

Because dairy products are restricted or avoided, which are a major source of calcium, an important mineral for children to develop strong bones, it is essential that other foods rich in calcium be given to make up for the loss. Tofu, broccoli, pulses (bengal gram whole, horse gram, rajmah), nuts and oilseeds, green leafy vegetables (particularly amaranth, fenugreek), fish and sea foods are excellent sources of this mineral besides dairy products.

Further, use of lactose free formulas can be advised, like soya feeds, Amylase rich foods are advised and rice based (ORS) advised.

## NOTES

### 18.3.4 Celiac Disease

Celiac disease (also called gluten enteropathy), as you may recall studying in Unit 14, sub-section 14.2.8.1, is an intestinal disorder that results from an abnormal immunological reaction to gluten, a protein found in wheat, barley, rye, and, to a lesser extent, oats. It is characterized by enteropathy of small intestines. It is said to be due to genetic, as well as, environmental factors. It is seen all over the world, as well as, in India. The clinical manifestation of celiac disease in children below 3-4 yrs of age is chronic diarrhoea, abdominal distension, extreme irritability and failure to thrive. The diagnosis of celiac disease is established with the help of three biopsies. Long term implication of untreated celiac disease could be growth stunting, osteoporosis and dental enamel defects.

Treating the patient with celiac disease emphasizes the importance of maintaining dietary compliance. The nutritional management of celiac disease is therefore highlighted next.

#### *Nutritional Management Celiac Disease*

A gluten-free diet is the major therapy for celiac disease patients, particularly children, and may be needed for lifelong adherence,

Gluten-free diet, as we learnt earlier, is a diet including food and drink, which does not contain gluten. In other words, a diet in which food having gliadin or related prolamin fraction has to be excluded e.g. wheat, rye and barley. Usage of oats is still under consideration. Various common natural gluten-free flours and cereals to be used in celiac disease are rice, maize, chick pea flour (Channa atta), black gram flour (besan), corn flour, potato flour, bajra, singhara ka atta, kuttu ka atta, soyabean flour. Other food sources that are safe in celiac disease include meats (poultry, fish, meat), milk, fruits and vegetables, butter, oil etc. Very few commercial gluten-free products are available in our country. It is indeed very important to monitor the child on gluten-free diet

A well-balanced diet with emphasis on intake of the following nutrients is recommended.

**Energy:** initially the children are seen to be malnourished, thus 100-120 Kcal/kg body weight/day with gradual increase to 150 Kcal/kg bodyweight/day to be given younger children can be given frequent feeding — upto 6-8 feeds in a day.

**Proteins:** should be provided upto 15% (percent of energy). The intake can be increased from 0.5 to 1g/kg body weight/day.

**Fat:** intake of fat can be from 25 to 50 % (i.e. 25-50 percentage of energy from fat of the total calories).

**Micronutrients:** supplementation of various micronutrients is advised for

celiac disease. These include vitamin A, folic acid, copper, calcium potassium and zinc.

## NOTES

Counseling and compliance: If symptoms do not improve, the child's diet should be reviewed and evaluated. A decrease in diarrhoea, increase in height and weight are some of the signs for improvement of the celiac disease condition. Proper diet counseling and follow up is very important to ensure compliance to the gluten - free diet.

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## 18.4 GERIATRIC NUTRITION

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Aging has been defined as "a series of time related processes that ultimately bring life to a close. " Persons of 60 years of age and older are defined as elderly by WHO. Successful aging is said to be multidimensional and has been defined as "encompassing the avoidance of disease and disability, maintenance of cognitive and physical function and sustained social and productive activity".

By 2022, around 13% of global population (1 billion people) would be above 60 years of age. It is expected that 1/3rd of this total population will be in developing countries. Considerations The increment being approximately 3% per year in developing countries and 1 % per year in developed countries. It is important that the elderly live a healthy and functional life than live with chronic disabilities. Since elderly are more susceptible to chronic and degenerative problems it would be interesting to know more about the physical and physiological changes linked with aging.

### 18.4.1 Physical and Physiological Changes

Every stage has its unique requirements due to different changing needs. With respect to nutrition and health, four different basic areas are of importance — physical, psychosocial, socioeconomic and nutritional changes. Let us review these changes.

**A. Cellular senescence** — Aging brings about an irreversible state of growth arrest at the cellular level. Though the cells are viable and metabolically active but their genetic function are different from the normal cells. Various clinical biomarkers have shown that oxidative stress at cell/mitochondria level increases with aging that is characterized by lower concentration of carotene, vitamin E and C levels.

**B. Physiological changes — The various physiological changes include:**

- 1) **Integumentary Tissues:** The skin, hair and nails covering and protecting the body are the integumentary tissues. Depigmentation of hair and wrinkling of skin (due to alteration in connective tissue composition) is

## NOTES

observed with aging.

**Wound healing becomes slower with age:** The dermis of skin where 7-hydroxy cholesterol is converted to cholecalciferol (Vitamin. D) also decreases with age, thus the older persons require longer exposure to sun to produce the given quantity of the vitamin.

- 2) **Body Composition:** After 30 years of age 1-2% decline in lean body mass annually is observed. The total body water, bone mass and lean body mass decreases. The body fat mass increases, weight gain is commonly observed in men (central abdominal area) and in women (post menopause). With aging the cardiac, renal and pulmonary functions decrease.
- 3) **Pulmonary and Respiratory System:** With aging the chest wall becomes stiffer and less compliant and the muscular force of the diaphragm is reduced causing less compliance and less recoil of lungs. Thus, there is decrease in the maximum amount of air movement in and out of the lungs. With the decreasing function, the hygiene of respiratory airways -ability to clear microbial pathogens in compromised.
- 4) **Cardiovascular and Circulatory System:** Aging affects the cardiac muscle causing a diminished resting cardiac output. The capacity of myocardium for cellular repair is also reduced with aging. With aging, there is stiffening of the blood vessels, causing rise in systolic blood pressure. The integrity of the blood vessels is also altered.
- 5) **Renal and Urogenital System:** Various studies have shown a decrease in the creatinine and insulin clearance. Due to the changes in glomerular structure of the kidney and circulatory senescence (aging) filtration is affected and thus the renal clearance. However, this per se does not affect the net nitrogen excretion. With aging, the male urogenital system undergoes hypertrophy of prostate gland and this affects the urine flow from the bladder.
- 6) **Oral cavity and alimentary tract:** Various functional changes and decline in secretory function occur in the digestive tract with aging. These include:

**Oral cavity** — The dentition are affected, (loosening of teeth, tooth decay and gum deterioration with age), saliva secretion decreases leading to dry mouth (xerostomia).

**Gastric** — Reduced gastric secretion and gastric emptying is common in elderly. Some authors consider it to be due to aging whereas other investigators consider it to be due to helicobacter pylori. This affects the iron availability from the diet. Vitamin B<sub>2</sub> deficiency also observed due to decreased gastric intrinsic factor.

Due to the reduction in intestinal/ colon motility and poor muscle tone of abdomen along with decrease in dietary fiber intake and low physical activity associated with aging, constipation is a common problem observed.

## NOTES

- 7) **Musculoskeletal System:** Osteopenia (decrease in bone mineral content) is observed with aging. There is an average of 30% decline in the bone mineral density from 30-40 yrs of age to 90 years. Other factors affecting low bone mass are smoking, alcohol consumption, reduced physical activity, lower calcium intake and absorption and use of steroids. In women, immediately following menopause, the rate of bone loss is higher due to decrease in estrogen production. Musculoskeletal atrophy and decrease in functional loss of muscle strength is also observed due to decrease in anabolic hormones affecting the physical function and the metabolic rate.
- 8) **Gonads and Reproductive System:** With aging, there is decrease in pituitary hormone secretion causing decrease in gonadal hormone production, which causes number of changes. In women with the decrease and cessation of oestrogen hormone, menopause occurs.
- 9) **Endocrine Systems and Metabolism:** As mentioned earlier, the main endocrine gland—pituitary gland secretion decreases. Another important feature in elderly is decline in insulin production and thus declines in glucose tolerance. The diet- induced thermogenesis, basal and resting metabolism is reduced with aging.
- 10) **Hematopoietic and Immune System:** Though the circulating red blood cells or the white cell count or platelet number does not normally change with age, but some other changes occur like vitamin B malabsorption affecting the red blood cell production. The white cells, with aging, due to disrupted signaling causes changes like intrinsic function deficits— hyporesponsiveness to proliferation etc.
- 11) **Central and Peripheral Nervous System:** Age related changes occur in various senses related to the cranial nerves—vision, hearing, taste and smell, These are discussed next.

Vision — Presbyopia (loss of accommodation function and loss of muscular function) and opacity of the lens (cataract) occur with aging. Some studies suggest that there could be delay in cataract formation if diets rich in antioxidant vitamins are consumed.

Age related hearing loss is another feature observed with aging.

With aging taste and smell activity also decreases (hypogeusia and hyposmia respectively), thus affecting the appetite of the persons. Decrease in some micronutrient (Zn, Cu, some vitamins) are also said to be associated with decrease flavour perception.

Cognitive functions like intellectual, reasoning memory functions tend to decrease with age either as a consequence of neurodegenerative change. Important role of vitamin B and antioxidants have been mentioned by some authors.

## NOTES

Peripheral nervous system The vibratory and pain perception declines/dulled with age and this may have adverse implication in early organic disease detection. With Considerations respect to central nervous system, myelination of axons of nerve needs to be maintained and may be vulnerable to stress and free radicals.

12) **Drug metabolism (Polypharmacy)** — As older persons are sometimes prescribed number of medications, care is to be taken, as there is reduction in renal and hepatic clearance. Medications that may have possible interaction with appetite and gastrointestinal motion and electrolyte imbalance include anticonvulsants, antidepressants, non-steroidal anti-inflammatory agents, anti-arrhythmias, diuretics etc.

**C. Psychosocial changes**— is dependent on the integrity vs. despair of the individual and is also seen to affect the individual's health and nutrition.

**D. Socioeconomic status (SES)** — The nutrition has also been found to be related to the of the individual, which may change with retirement, and other health issues of the elderly.

The various changes described above influence the nutrient need and nutritional management of elderly. We shall review the nutrient requirement and changes required for the elderly next.

### 18.4.2 Nutritional Changes and Requirement

Nutrition is affected in two way —due to the changes in physiological function with aging having effect on absorption, retention and utilization of nutrients and on the other hand the extent to which a particular nutrient or diet pattern affects the senescence (aging. To a certain extent, optimal nutrient intake, food and selection can help in a better life of an aged person in addition to his/her genetic constitution. By 1970, it was suggested that with age the requirements for macro-and micronutrients change. The RDA have been given for 51+ age group by National Research Council (1989,US). These requirements include:

**Energy and Protein:** Decreased physical activity and changes in body composition and decreased basal metabolic rate affects the macronutrient energy, protein requirements. It has been established that 0.8 gm protein [kg body weight/day in universally recommended providing about 15-20 en% (percent of energy). The protein intake may be increased or decreased depending on illness/convalescence.

The estimated energy requirements decrease by 0.5-1.0 % per year and are based on physical activity level, weight, height and age of the individual - 25-30



**NOTES**

Kcal/kg/day (increased in hypermetabolic state). With aging loss of muscle mass and strength (sarcopenia) is observed. It has been seen that decrease in physical activity causes these body composition changes. These processes lead to a lower energy requirement. Due to the decrease in lean tissue, the BMR declines by about 5 % per decade. Thus for maintenance of lean body mass, physical activity is most important. It is also important to emphasize the elderly to maintain adequate energy intake to prevent either underweight or overweight. Underweight may be observed in persons who have 'anorexia of aging' and depression. They may have gait instability, falls, fractures, delayed wound healing etc.

**Carbohydrate:** It is usually recommended that approximately 55% (percent of energy) be provided from carbohydrate foods with emphasis on complex carbohydrates. Aging has been associated with decreased glucose tolerance and it can be corrected to an extent with increased dietary fiber and exercise.

**Fats:** In elderly fat intake is recommended to be limited to 20% (percent of energy) with emphasis on quality of fats used. Emphasis is on right proportion of PUFA, MUFA and prevention of coronary artery disease.

**Vitamin and Minerals:** Decreased gastric secretion and intestinal motility affect the absorption and intake of nutrients especially calcium, iron, vitamin B12 and fiber in the diet. The dietary fiber intake is also reduced with age.

**Calcium, Phosphorus and Vitamin D :** As mentioned earlier, due to the change in skeletal system, vitamin D and calcium needs increase with age. Bone mineral loss as part of aging needs special emphasis on calcium and phosphorus intake. The upper intake level of phosphorus is lower than the requirement for younger adult. Keeping in view the greater prevalence of impaired renal function with aging FAO/WHO recommends a lower magnesium intake than the younger adult.

**B-Complex Vitamins:** Various B complex vitamins especially B<sub>12</sub>, folic acid, B<sub>6</sub> and riboflavin are found to affect favourably the cognitive function and n-3 fatty acids help in preserving the cerebral at the cellular level.

**Vitamin A, Iron and Zinc:** The recommendation for iron is said to decrease in elderly women. Intake of vitamin E and zinc is said to help in the immune and defense system.

**Water and electrolyte:** Intake for individuals are said to be consistent throughout adulthood. For adults 30 ml/kg body weight /day is recommended.

There may be increased risk of dehydration among elderly when on diuretics or laxatives as they have a lower thirst and have decreased urine concentrating ability. Thus, we may conclude that for elderly a healthy diet with emphasis on green vegetable, fruit, whole grain product, low fat milk products, fish and legumes along with life style modification in physical activity and smoking is important. Unnecessary and overdose of supplements should be avoided, but on

the other hand adequate supplementation as Required would help in improving the nutritional status of the elderly.

## NOTES

### 18.4.3 Nutritional Assessment

Nutritional factor is defined as "characteristic occurrence that increases the likelihood that an individual has or will have problems with nutritional status". Various factors contributing to this are age, poverty, disease, dietary intake, and physical condition

Nutritional assessment methods commonly used are enumerated herewith.

1. **Body Weight** : Measurements remain to be the most practical and cost effective parameter. Rapid weight loss such as 2% in one week or up to 10% in 6 months is considered significant and BMI calculation is useful for comparison since skin fold measurements are not effective due to skin turgor changes.
2. **Physical Examination**: Based on the physical examination such as presence of oedema and vitamin deficiency symptoms, diagnosis can be made.
3. **Biochemistry** : Hypoalbuminemia is not considered to be an index of under nutrition, but it is of importance to identify the high risk subset of older persons requiring nutritional intervention. Levels less than 3 g/dl needs caution and intervention.
4. **Haematology** : A lower total lymphocyte count (TLC) is also helpful in stratification of severity of under nutrition. If levels are less than <1500, it is associated with decrease immune function and malnutrition.

Nutritional status assessed traditionally with the use of anthropometric, biochemical, and dietary analysis has drawbacks. These include:

Data for elders is compared with normal values for younger persons.

Physical condition of elders makes some measurements difficult.

RDA's for those over 50 years of age not available.

Nutrient deficiency signs and symptoms are inappropriately attributed to normal aging.

Nutritional screening programmes, therefore, have been designed to assess for presence of malnutrition and some of the causes. Next, we shall look at the nutritional assessment tools, which may be used for the assessment of at risk elderly individuals.

### Nutritional Assessment Tools

Malnutrition/protein energy malnutrition amongst elderly persons has been

observed in various studies — be it hospitalized patients, nursing home residents or outpatients. With the help of the assessment tools the elderly at risk can be given preventive and treatment care as required.

The assessment can be done by observation of following tools.

1. **Nutrition Screening Initiative (NSI) :** (NSI) tries to identify basic risk factors - inappropriate food intake, poverty, social isolation, disability, acute / chronic disease, chronic medication use, and the age of subject. It is a valuable epidemiological tool and increases awareness of undernutrition among patients and caregivers.

**Drawback** — A large number of personnel required at different screening levels, there is lack of professional supervision and patient compliances in the beginning.

2. **Mini Nutritional Assessment (MNA) Tool :** It is a comprehensive and simple tool, which is able to categorize the subjects into three different categories like well nourished, at risk and undernourished. In most of the cases this tool eliminates the need for more invasive test such as blood sampling.

The MNA was developed and validated jointly by the Center for Internal Medicine and Clinical Gerontology of Toulouse (France), the Clinical Nutrition Programme at the University of New Mexico (United States), and the Nestle Research Center in Lausanne (Switzerland). The objective of this tool was to screen and assess the nutrition status as part of the standard evaluation of elderly patients in clinics, nursing homes, hospitals, or among those who are otherwise frail. The MNA is easy to administer, patient friendly, inexpensive, very sensitive (96%), highly specific (98%), and reproducible.

The MNA comprises 18 items grouped in four sections:(1) anthropometric assessment (weight, height, arm and calf circumferences, and weight loss); (2) general assessment (six questions related to lifestyle, medication, and mobility); (3) dietary assessment (eight questions related to number of meals, food and fluid intake, and autonomy of feeding); and (4) subjective assessment (self-perception of health and nutrition

The response to each item in the MNA had a numerical score. The total MNA score is calculated as the sum of the points assigned to the responses of the 18 items. The maximum value of the final score is 30. According to the obtained score using the questionnaire the MNA stratifies patients in: well nourished ( $24 \leq \text{MNA} < 30$ ), at risk of undernutrition ( $17 \leq \text{MNA} < 23$ ), and undernourished ( $\text{MNA} < 17$ ).

The MNA is specifically designed to guide nutritional intervention by identifying the risk factors requiring correction. In fact, it is both a screening and assessment tool for the identification of malnutrition in the elderly. The management and

## NOTES

**NOTES**

nutritional intervention guidelines using MNA are as follows:

***A. For those categorized as well-nourished***

They need only be reminded of the importance of continuing good dietary habits. The MNA assessment should be repeated to detect any changes in status.

***B. Persons at risk of undernutrition***

An MNA score between 17 and 24 indicates that the patient is at risk for malnutrition with a good prognosis, given early intervention. Thus, factors leading to undernutrition must be identified and strategies should be devised to treat those conditions. The MNA examination should be readministered 3 to 6 months later to evaluate the success of intervention and to determine if the patient remains at risk of malnutrition.

***C. Undernourished persons***

An MNA score below 17 indicates that the patient is suffering from undernutrition. Thus in this situation the diagnosis must be confirmed by laboratory tests, besides improving the nutrient intake and frequent monitoring will be necessary.

In community-dwelling elderly persons, the MNA detects risk of malnutrition and life-style characteristics associated with nutritional risk while albumin levels and the BMI are still in the normal range. Also, IVNA is associated with diminished cognitive function, diminished ability to care for one's self, and with reduced functional capacities. In outpatients and in hospitalized patients, the NINA is predictive outcome and cost of care. Nutritional status assessed on admission by the MNA reflects the patient's nutritional condition, degree of autonomy, and current treatment. In home care patients and nursing home residents, the MNA is related to living conditions, meal patterns, and chronic medical conditions and allows targeted intervention.

The MNA was designed to provide the health care professionals with a quick, macroscopic view of a number of important areas that affect the nutritional well-being of an elderly patient. As such, it is not useful in assessing whether a patient has a specific vitamin or mineral deficiency, especially in the case of Vitamin D, folate, vitamin B<sub>12</sub>, iron and zinc. Another weakness of the MNA is that a number of questions target independent-living elders but not elders in long-term care or elders receiving nutrition support.

A sample of the mini nutritional assessment tool is given in Table 18.3 for your reference.

3. Scales (Screening Tool) : The basic biochemical and anthropometric parameters are used in this screening.
4. Recognizing the different causes of under nutrition by simple mnemonics - WEIGHT LOSS. Let us see what it stands for.

**NOTES**

W — Wandering and forgetting to eat / weight loss due to 'anorexia of aging'.

E — Emotional /psychological problems like depression etc. and their medication have effect on food intake.

I — Insufficient funds.

G — Gastrointestinal tract problems.

H — Hypo(hyperthyroid or other endocrine problems should be looked into as they cause weight loss, depression, gastrointestinal tract problem.

T — Tremors or other neurological problems interfering feeding.

L — Low salt or unappetizing diet due to diet restriction because of variety of medical illness.

O — Oral problems like tooth loss or use of dentures also effects intake of food.

S — Swallowing — Dysphagia related various neurological problems.

S — Shopping and food preparation.

Having learnt about the nutritional assessment tools let us look at some problems linked with elderly.

**18.4.4 Health and Feeding Problems among Elderly**

Malnutrition, both obesity and undernutrition, are common problems linked with elderly. Some of the common disorders such as Alzheimer's disease, Parkinson's disease etc. are also age related about which you may recall studying in the last unit. Dysphagia (difficulty in swallowing), though not a disease, is a disorder which may accompany several neurological disorders. We shall not dwell on the nutritional management of these neurological disorders here in this unit since we have already covered them earlier.

Here, let us look at obesity as a problem among elderly. Obesity, we know, is correlated to number of diseases like coronary artery disease, diabetes, sleep apnea, osteoarthritis, peripheral vascular disease. Hence, its treatment and management is crucial. The mainstay of treatment of obesity in elderly should be combination of exercise, healthy eating and behaviour modification, with emphasis on micronutrient supplementation. Undernutrition, among elderly, can be diet induced or may be due to other reasons. We will next elaborate on nutrition support for the elderly during undernutrition next

**18.4.5 Nutrition Support— Parenteral/Enteral/Oral**

Undernutrition either due to dietary deficit or due to a medical complication may

arise in the elderly. The nutritional support, therefore, needs to be individually modified based on changes in metabolism. us review each of the nutrition support i.e. parenteral, enteral and oral and their indications

## NOTES

Parenteral feeding is used in cases when neither oral nor enteral feeding is possible. For example, in case of non-functional GI tract. Close monitoring is required in this feeding. Use of low osmolality nutritional preparation is also emphasized.

During prolonged use, central line is preferred and care needs to be taken to prevent catheters related infections.

Use of pharmacological methods is the second line of treatment for undernutrition, if the initial nutritional support fails.

Enteral feeding: Enteral, as you may already be aware, is defined as provision of nutrition support through the GI tract or by accessing the gut. The pre-requisites/special features include

To be resorted to if individual is unable to feed orally.

If GI tract intact, resort to enteral feeding.

For short term support nasogastric or nasoenteric; for prolonged use of enteral route — gastrostomy/jejunostomy to be used.

Choice from the different polymeric formulas, preferably of low viscosity so that easier route in case of small bore tubes.

Specific formulation can be given as and when required by patient e.g. diabetic or renal feeds

Continuous feeding to begin with half strength feed (at the rate of 30 ml/hr; followed by full strength at 25 ml/hr) preferred over bolus feeding due to risk of aspiration

Cyclical feeding is another example of continuous feeding when feeds are given for a particular time of day e.g. 12-18 hours. Interrupted feeding is also found to be most practical method. It is continuous feed for 4-6 hours intermitted with few hours of no feeding.

While feeding, positioning the patient at an angle of 30° decreases risk of aspiration.

PEG (percutaneous endoscopic gastrostomy) is another good way of feeding.

Usually caloric concentration of feeds is around 1/Kcal/ml

Finally, let us get to know about the oral nutrition repletion.

Oral Nutritional Repletion: This is the most ideal feeding method. The nutrient requirements are highlighted herewith.

### ***Energy Requirements***

Based on the Benedict — Harris equation for men and women, the daily energy requirements can be calculated

For Men :  $66 + 13.7 W - 6.8 A$

For Women:  $66.5 + 9.6 W + 1.8 H - 4.7 A$ , where W = weight (kg), H = Height(cm),

A = Age (Yrs)

OR

For practical purposes 35 Kcal/kg/day in case of hyper metabolic condition and baseline requirement of 25-30 Kcal/kgBW/day.

In case the elderly is having pulmonary insufficiency; the fat en% (percent of energy) should be increased to 50%.

Proteins : Normally 1g/kg BW /day is the protein requirement which may be increased to 1.5 g/kgBW/day in stress conditions. However, in case of renal and hepatic insufficiency protein intake is reduced as per condition.

Fluid and Electrolytes: Care to be taken that adequate fluid be given to the elderly. If patient is unable to take the required daily needs, then formulated nutritional supplements could be given if oral intake is poor.

If taking orally (supplemented feed), should take care regarding the following:

- Correct flavour of feed
- To be given in between meals; approximately 1 hour before meals
- In case of malabsorption syndromes, hydrolyzed preparations given

Vitamins and trace elements: Decreased gastric secretion, absorption interaction with medication may increase certain vitamin and trace elements requirement. For e.g. calcium, vitamin. A, C, D, E, B6 and zinc requirements.

Requirements in special situations: In disease conditions like renal, cardiac, hepatic insufficiency etc., the requirements are as per the disease state, about which we have already studied earlier in this course

To sum up, having gone through the discussion above, you may have realized that it is a challenge to meet the nutritional requirements of elderly due to the various factors.

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## 18.5 LET US SUM UP

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This unit focused on the paediatric and geriatric problems and their nutritional management.

Initially, the common paediatric problems such as congenital heart disease, low birth weight, lactose intolerance, celiac disease were highlighted. Their symptoms etiology were described, with particular focus on their nutritional management.

## NOTES

The second part of the unit dealt with the physiological changes linked with ageing and the nutritional changes and requirement related to this phase. Nutritional screening programmes to assess the presence of malnutrition and some of the causes among elderly was covered, followed by health and feeding problems and the nutritional support for the elderly.

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### 18.6 GLOSSARY

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|                                |                                                                                                                                       |
|--------------------------------|---------------------------------------------------------------------------------------------------------------------------------------|
| <b>Acyanotic Heart Disease</b> | : broad term of congenital heart defect involving the walls of the chamber or obstruction in the valves.                              |
| <b>en</b>                      | : percentage of energy of total calories.                                                                                             |
| <b>Gastrostomy</b>             | : refers to a surgical opening into the stomach. Creation of an artificial external opening into the stomach for nutritional support. |
| <b>Geriatrics</b>              | : the branch of medicine specializing in medical problems associated with old age.                                                    |
| <b>Hypoalbuminemia</b>         | : refers to an abnormally low level of albumin in blood.                                                                              |
| <b>Hypoxia</b>                 | : reduction of oxygen supply to tissues below physiological levels. refers to surgical creation of an opening between the             |
| <b>jejunum</b>                 | : and üle anterior abdominal wall; to allow enteral feeding.                                                                          |
| <b>Senescence</b>              | : the process of growing old; a consequence of advancing age or of a premature aging process from disease.                            |

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### 18.7 CHECK YOUR PROGRESS

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1. List a few paediatric problems, which have special nutritional requirements.
2. Why does the calorie and protein requirement increase in CHD?
3. List the energy, protein and fluid requirements for a preterm infant.
4. Which food/food component is not tolerated during celiac disease and why?



5. Give the basic nutritional requirements of elderly.

6. Give the situation when parenteral and enteral feeding is indicated in the elderly.

**NOTES**