UNIT 14 NUTRITIONAL MANAGEMENT OF GASTROINTESTINAL DISEASES AND DISORDERS

Structure

14.1 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 cmplying, diarrhoea or very severe ones such as the dumping syndrome, malabsorption syndromes arid 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.

Objectives

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 GASTROINTESTINAL DISEASES AND DISORDERS

Before discussing the many gastrointestinal problems, it is useful to understand the basic physiology of the gastrointestinal tract. Figure 14.1 illustrates the different parts of the gastrointestinal tract. You would recall from your Applied Physiology Course (MFN-001), Unit 6 that the gastrointestinalsystem 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.

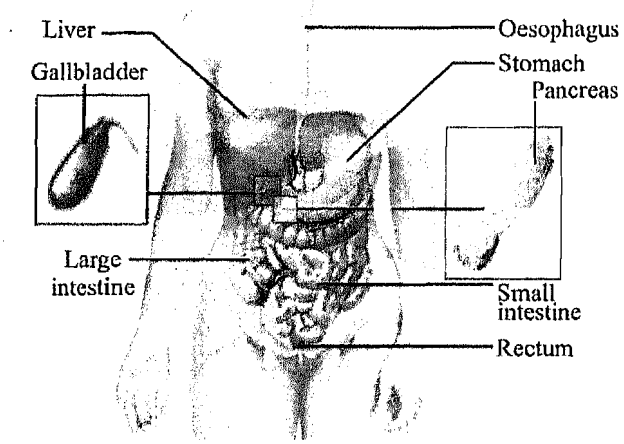

Figure 14.1: Various parts of the GI tract

Looking at the functions, you canunderstand that any disorder or diseases of a particular part can effect 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.

14.2.1 Diarrhoea

What is diarrhoea? Diarrhoea is characterized by the ***frequent evacuation*** of ***liquid stools, usually exceeding 300 ml, accompanied by an excessive loss of 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 ans 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. Table 14.1 highlights the causes for acute and chronic diarrhoea.

Table **14.1:** Causes of diarrhoea

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; *Shigella*)	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

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 associaled with lactose intolerance (sugar lactose is not digcsted due to lack of enzyme lactase in the intestine), dumping syndrome (multiple symptoms rclatcd 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 cxotoxins 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 proccdurc. This type of diarrhoea is usually complicated by steatorrhoea (increased amount of fat in feces).

Now, let us have a look at the consequences of diarrhoea with the help of flow chart given in Figure 14.2.

Consequences of Diarrhoea

All of us must have suffered from diarrhoea atleast once in our lifetime. I-low 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 inteslines. 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.

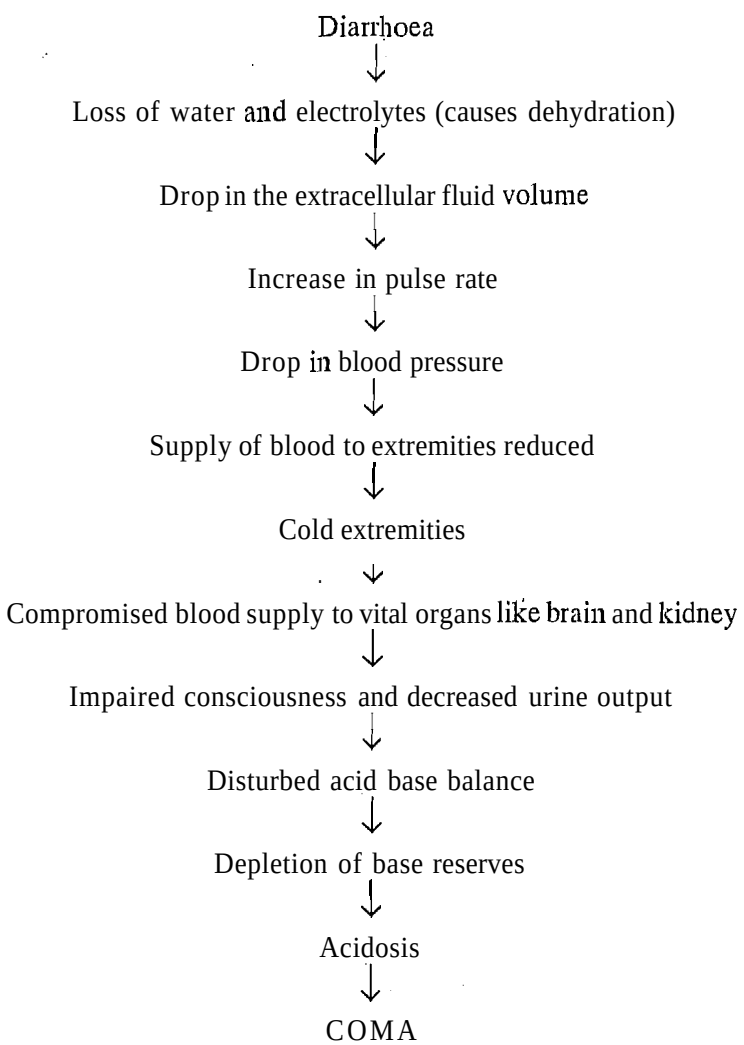

Figure 14.2: Consequences of diarrhoea

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.

We will now continue our discussion to learn about the treatment and management of diarrhoea.

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 morlalities. 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, gaslric leavage 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, inoderate 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 rehydartion)
3. Potassium (mEq/L) = 20 (prevents acidosis)
4. Chloride (mEq/L) = 80 (favours rehydration)
5. Bicarbonale (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 diarrhoea1 episode and a quicker recovery. Calories can be provided through easily digestible carbohydrates. 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, emulisified fats or those, which are rich in medium chain triglycerides, may be added in restricted amounts. Fats like butler, 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 carbohydratesi.e. principally starches should be preferred. Glucose, sugar, honey, jaggery, potato, yam, colocasia, rice, sago, semolina, refined flour, pastas can be incorporated lo 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. Table 14.2 and 14.3 give the low fibre and low residue foods. 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.

Information provided in Box 14.1 relates to the concept of residue read it carefully.

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.

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 rnucosal damage in chronic diarrhoea, which in turn impair absorption and synlhesis of various essential substances in the body. The vitamins of importance are B complex vitamins especially folic acid, vitamin B_{12} 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 wc 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 parentslcarelakers 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 with 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 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 bc 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 CEILDREN; MAKE NECESSARY MODIFICATIONS IN THE NORMAL DAILY DIET. GIVE FREQUENT LIQUIDS OR/AND LOW RESIDUE SOFT DIGESTIVE FEEDINGS. GIVE BLAND AND LOW FIBRE DIETS.

We shall next proceed over to discuss about another common disorder of 'the gastro-intestinal tract viz., constipation.

14.2.2 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, rouhage and potassium
   b) vitamin B Complex deficiency
   c) irregular defeaction habit and poor personel hygiene.
   d) excessive purgation or use of enema.
   e) 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 worry are also some common causes. The causes can be classified under two heads – systemic and gastrointestinal – as highlighted in Table 14.4.

Table 14.4: General causes of constipation

Systemic	Gastrointestinal
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

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, as depicted in the Figure 14.3. --

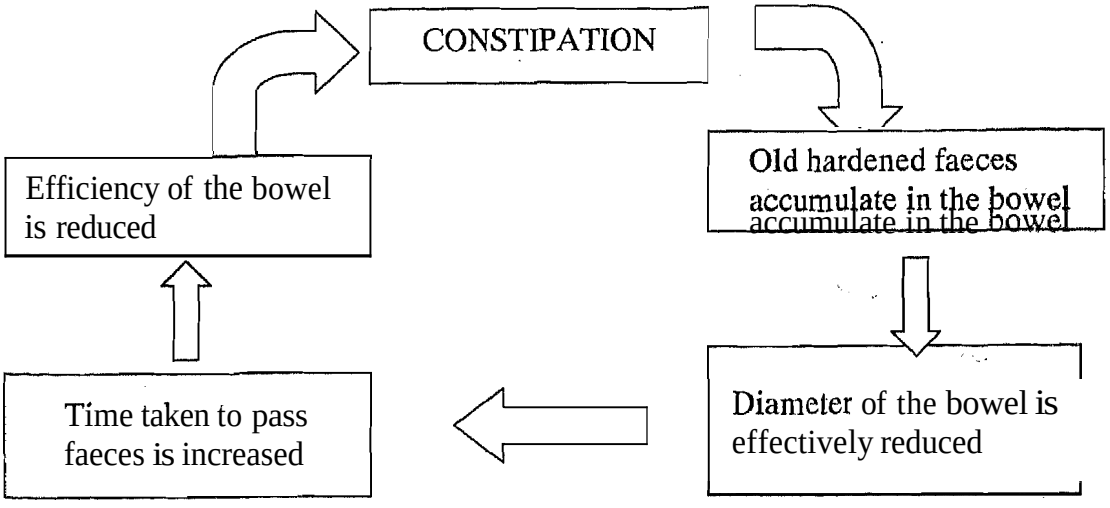

Figure 14.3: Complications in constipation

Apart from this, the list of other complications for constipation have been mentioned in Table 14.5.

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)

What can be done to prevent constipation? Can dietary management help relieve the problem? Let us find out.

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 structural1 functional change in the gastrointestinal tract. In our subsequent discussions, we will deal with the dietary management of conslipation. 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 flalulence 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 toile.

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.

Table **14.6:** Foods to be given freely in constipation

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 **14.7:** Foods to be **avoided**/ restricted during 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.

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.

Now let us gear ourselves to some check your progress exercise, before we move any further.

Check Your Progress Exercise 1

1. Make a list of 10 foods low in dietary fibre and 5 foods low in residue.

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2. Suggest ways how you can improve the dietary fibre intake of an elderly man 65 years of age. Justify your selection.

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3. 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?

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4. Suggest a feeding schedule for a child recovering from acute diarrhoea and justify your selection.

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In the section discussed above we learnt about the etiology and management of two distinct disorders of the bowel viz., diarrhoea and constipation. We shall continue our discussion on certain other common disorders of the gastrointestinal tract such as oesophagitis and gastroesophaus reflux disease.

How many of us get the symptoms of heartburn, acidity and bloating often? Do we know as to why it happens? It is important to learn about these symptoms in view of their wide prevalence among the general masses, as well as, among patients. Let us examine and find out the details of the associated condition.

14.2.3 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 oesopltagus 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 an acute/chronic inflammation of the oesophageal wall. It is associaled with the common syinptom of heartburn (burning epigastric substernal pain). Other symptoms are regurgitation and dysphagia (difficulty in swallowing). Difficulty in swallowing occurs due to pain associaled with inflammation of the tissues of the oesophagus. Regurgitation of the acidic gastric contents can be 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 **Oesophngitis*** – 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 pancreaticenzymes. 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 hydrocliloric acid (HCl) and pepsin, with symptoms of substernal burning, cramping, pressure sensation or severe pain. 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 pnenmonitis. 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 arc 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 (cemetidine).
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).

14.2.4 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 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 hiatus 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 latter 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 complicatioils 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 complicatioils 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, symptoins 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 by impairing gastric emptying. Also, it is inappropriate to advise to reduce the fat content of their meals, as 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 LESP 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.

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 of 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 relevarice 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) and 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.

Next, we shall discuss the symptoms of hiatus hernia.

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 antacids to relieve burning sensation
- large or sliding hernia may require surgical treatment

In this section, we learnt about oesophagitis and gastroesophgeal 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.

Check **Your Progress Exercise *2***

1. List a few common symptoms of oesophagitis. Write two lines about nutritional management of chronic oesophagitis.

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2. Give the causes that lead to the development of GERD. Also highlight the points that you would keep in mind while counseling such patients.

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3. Name the most common complications of GERD. List any five foods that must be avoided during this disease.

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In this section we shall learn about some more diseases of the gastrointestinal tract. Some of them would be as common as dyspepsia and gastritis which we all must have suffered from at some point of time in our lives. Another disease which we shall discuss thereafter is diverticulosis which is relatively less common as compared to dyspepsia and gastritis.

14.2.5 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, discoinfort 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.

Etiology

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 tlie 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.

Let us move on to some complications of chronic dyspepsia.

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 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 tlie etiology, symptoms and complications of dyspepsia it must be clear that treatment and management of this disorder does not require any major 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 tlie 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 consuming 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 yoy 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 a acid neutralizer to as severe to result in the development of ulcers. Let us Iearn about this disorder in detail.

14.2.6 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 inflammatioii 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, bums, 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 liypercmia, 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.

Let us now explore the causalive factors of acute gastritis.

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_{12} (Pernicious anaemia). The chronic gastritis is clinically more important than the acute gastritis.

Gastroscopic observation shows 3 types of chronic gastritis:

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_{12}, 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 irritants and 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 cooked form. 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.

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_{12}: Supplementation with vitamin B_{12} 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.

Abrief list of foods to be avoided is given below in Table 14.8.

Table **14.8: Foods/substances** to be **avoided**

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

So far we have discussed about dyspepsia and gastritis in this section. Next, we shall discuss about yet another disorder of the gastrointestinal tract viz., diverticulosis.

14.2.7 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 of 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 *diverticuli.* 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,
- the movement of waste through the colon,
- 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 now highlight some important aspects of the dietary management.

Dietary Management

Most of the diseases which we have discussed so far do not require any major changes in the nutrient intake. The patients generally benefit from a high fibre diet. Hence, a greater amount of bulk or fiber in the diet will promote soft, bulky stools that pass more swiftly and are defecated more easily. Also, the intra luminal pressure generated in the lower colon would be less and the fewer diverticuli will be formed. An increased intake of fluid must be emphasized. A decreased intake of fat in the diet 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 intravenous antibiotics. Eating a high-fiber diet and taking psyllium supplement are beneficial. Maintain overall nutrition. Some important aspects, which need to be taken care of, include:

1. High-fiber *Diet:* Population 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 all 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.

We now need to attempt the check your progress exercise **3** given herewith' to recapitulate what we have learnt so far.

Check Your Progress Exercise 3

1. Differentiate between the following:

   a) Acute and chronic gastritis

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   b) Diverticulosis and Diverticulitis

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2. Discuss the dietary principles involved in the management of dyspepsia.

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3. Discuss the diet therapy for the treatment of diverticulosis.

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So far we have learnt about a wide spectrum of diseases of the gastrointestinal tract which if .managed efficiently do not result in the development of any serious consequences. In this section we shall discuss about another common disorder of the GI tract viz, peptic ulcer. Let us understand it in a little more detail.

14.2.8 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.

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 rnucosal integrity and permit proteolylic 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.

Table 14.9: Factors affecting mucosa

Destructive Factors	Defensive Factors
Hydrochloric acid	Epithelial cells barrier
Pepsin (Proleolytic enzyme)	Mucous
Psychological factors (stress and anxiety)	Gastric blood flow
Gastric irritants (alcohol, caffeine, excess tea, meat extracts and spices)	Regulation of acid secretion
Nicotine and Tobacco	Ability of the body to regenerate the cells
Anti inflammatory drugs/ Analgesics	

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 rnucosal 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 of 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.

Table 14.10: Pathogenic factors

Gastric Ulcer	Duodenal Ulcer
Seen at the back of the stomach	Appears within 3 cms of the pyloris
Abnormal pyloric function	Increased acid secretory capacity
Duodeiiogastric reflux	Increased basal acid secretion
Defective gastric mucosal defenses	Increased parietal cell mass and sensitivity
Decreased mucosal blood flow, Prostaglandin production, Bicarbonate production, Gel layer	Prolonged meal secretory response
H. pylori infection	Abnormal gastric emptying
	Abnormal duodenal mucosal defenses
	Decreased bicarbonate secretion

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 pectic 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 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 tlie management of pectic ulcers. Recent development of new drugs like Cimetidine and Ranitidine (Hz 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 tlie 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 the 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 tlie patient in achieving and maintaining an ideal body weight. An adequate energy intake is a must to prevent subsequent weight Ioss 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 and bleed. Adequate protein intake ensures synthesis of new tissues essential for healing. Normal milk protein is valued for their buffering action but it is supposed to provide temporary relief because the products of protein digestion (amino acid and polypeptides) reach tlie antrum and they stimulate the secretion of gastrin and gastric acid, Milk 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. Restricte'd intake of milk gives relief for a short period as it contains the prostaglandin PGE2-a protective agent against stress induced ulcers.

Proteins from cottage cheese, egg, chicken and fish in adequate amounts is beneficial for regeneration of cells. Protein supplemenls 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 tlie small intestine stimulate entrogastrone, 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. *Pylroi.* 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 ntucosa: 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 nonnally 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 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 biocarbonate synthesis. 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 repulation of being gas formers has also been questioned. Clinical observations have shown that tolerance for 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 is 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 ark:

- 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 liberalized according 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 tarry 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 g/hour (especially in stages of acute pain) followed by small feedings of easily digestible foods like soft cooked eggs, custards, refined flour products, cottage chkese, low fibre vegetables like gourds, clear soup with no seasoning and herbs, soft over ripe fruit 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 -

- a marked recovery from pain.
- a 6 meal pattern followed
- light, bland, low fibre diet
- mechanical/thermal, chemical irritation of gastric mucosa to be avoided
- 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
- a 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.

This section dealt with one of the most serious forms of gastrointestinal disorder wherein diet plays an important role in the causation, treatment, as well as, management of the disease. We hope that you will benefit from the discussion above. Before we proceed further try to attempt the check your progress exercise 4.

Check Your Progress Exercise 4

1. What is the difference between gastric and duodenal ulcer?

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2. What is the recent highlight in the etiology of peptic ulcer?

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3. Why is the current management approach of peptic ulcer called liberalized?

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We have learnt about a host of diseases in the previous sections. In our last section on GI disorders we will brief ourselves on certain common malabsorption syndromes viz., celiac disease, steatorrhoea, lactose intolerance and inflammatory bowel disease. Some of these disorders are diseases in itself while others are symptoms of another underlying disease. We shall begin our section with an overview on rnalabsorption syndromes.

14.2.9 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 '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. These are tabulated in Table 14.11.

Table 14.11: Principal causes of malabsorption

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	: Cholestyrainine, Antibiotics, excess laxatives
Infections	: Giardia and parasitic infestation, tuberculosis and bacterial overgrowth.

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 us 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 us begin with celiac disease,

14.2.9.1 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, losses 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 neurologicdisorders, 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 B_{12} 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 complicationsmainly due to nutrient absorption problems leading to malnutrition. These complicationsare highlighted in Table 14.12:

Table 14.12: Complications due to Celiac's disease

Congenital defects — in babies born to celiac mothers	Miscarriage
Osteoporosis (weak and brittle bones)	
Lymphoma (can develop in the intestine)	Stunted growth in children
Seizures or convulsions	Anaemia

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, symptomsand complications the major objectives of dietary management include the following:

- Providing a nutritionally adequate diet
- Strict restriction of gluten foods
- Vitamin 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.

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 1.4.13gives an idea of the gluten sources to be avoided by the subject, the food that can be taken are from the non-gluten sources.

Table **14.13:** Gluten **and** Non-gluten sources

Gluten Sources	Non-gluten Sources
Beverages: cereal beverages, ovaltine, beer and ale	Coffee, tea, chocolate drinks (pure cocoa) wine
Milk beverages that contain malt	Whole, toned, skim milk and buttermilk
Meat and meat products: Breaded meats commercially available	Pure meats, fish, poultry, eggs, cottage cheese, peanut butter
Fats and oils: Commercial gravies, white sauce and cream sauces	Butter fats and oils
Cereal and *cereal products:* Bread, wheat, oals, rye, malt, pastry flour (maida), bran, barley, wafers, pasta.	Rice, potato flour and soya flour, pure corn, popcorn.
Vegetables: Breaded vegetables with any of the sauces, white sauces etc.	All fresh vegetables, canned and pured.
Fruits: Any fillings e.g. pies etc.	All fresh fruits
Snacks: Pastries, patties, pizzas, samosas, mathris etc.	Milk base sweets (pure) without addition of any cereal products.

Let us have a look at few of the tips whicli 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 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, fruit, and most dairy products are free of gluten.

Another common malabsorption syndrome listed in the section 14.8 seatorrhoea which is discussed below.

14.2.9.2 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 faces*. 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
- a 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.

14.2.9.3 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 vili are structurally damaged.
- Surgical causes in which large part of jejunum is removed.

As discussed above, the patients usually experience the symptoms as highlighted next:

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.

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 individuals tolerance level) can be given several times a day.
- Small amounts of milk in moderate lactose 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.
- Lactose enzymes are available these can be added in the milk.
- Deficiency of lactose 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.

14.2.9.4 Inflammatory Bowel Disease (IBD)

Inflammatory bowel disease is a general term used to refer to chronic inflammatory condition of the intestine. It is applied to three conditions having similar symptoms but different underlying clinical problem. It includes:

1. Ulcerative colitis
2. Crohn's disease
3. Short bowel syndrome

When the inflammation is in rectum with extension into the colon without affecting the right colon or small intestine, the disease is called *ulcerutive 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 intestines the colon and even the colorectal region. However, in ulcerative colitis, the inflamination is confined to 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.

I-Iaving 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.
- 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 are observed in patients.

So then, what can be done to manage these conditions? The next section focuses on the nutritional management of inflammatory bowel diseases.

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/support growth of children and adolescents. Supplementation with multivitamin preparations (1-5 times above RDA) in necessary as this condition precipitates deficiency of many nutrients, decreases absorption and increases requirements.

Overemphasis on fibre may be avoided in patients with strictures as it may lead to blockage.

Streatorrhoea 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.

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.

Next, we will understand the etiology of ulcerative colitis.

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_2 as compared to the normal excretion of 2 g. In severe ulcerative colitis, 20 g N_2 (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 / IBW 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 alieady 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 / 2.2 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 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 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.

We will now discuss about another inflammatory bowel disease i.e short bowel syndrome.

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 bum 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 wit11 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 B_{12} and zinc.

Let us read and find out the details of dietary management.

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.

Check Your Progress Exercise 5

I. Desribe the term lactose intolerance.

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2. Write five lines about celiac disease? Give five foods containing gluten.

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3. Differentiate between Crohn's disease and Ulcerative colitis.

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4. **A** patient suffering from inflammatory bowel disease is at high risk to malnutrition. Why?

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14.3 LET US SUM UP

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, 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.

1144 GLOSSARY

Acidosis : Increase in concentration of acidic substances in the blood.

Crohn's disease : **a** chronic inflammatory disease of unknown etiology involving small and large intestines that results in diarrhoea, strictures, fistula and malabsorption.

Fistula : an abnormal passage between two internal organs or from an internal organ to the surface of the body.

Welicobacter pylori : a type of bacteria that can chronically infect the stomach, thought lo be the primary contributor to the development of gastritis, peptic ulcers and even gastric cancers.

Hypokalemia : a condition with low serum potassium levels characterized by poor muscle tone, impaired gut motility and electrocardiographic changes.

Hyponatremia : a decrease in serum sodium level in the blood commonly found in dehydration.

Melena : black tarry stools indicative of gastrointestinal bleeding.

Prebiotics : non-digestible food products that stirnulute the growth of symbiotic bacterial species already present in the colon.

Probiotics : microbial foods or supplements that can he used to change or reestablish the intestinal flora and improve the health of the host.

Prostaglandins : one of the several potent hormone-like compounds made from PUFA which have diverse effects, both harmful and useful.

Regurgitation : is the controlled flow of stomach contents back into the oesophagus and mouth.

Residue : the fecal contents, including bacteria, GI secretions and foods not digested and absorbed.

Short Bowel **Syndrome** : a malabsorption syndrome resulting from major resections **of** the small bowel characterized by diarrhoea, steatorrhoca and malnutrition.

14.5 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1. Dietary fibre rich foods - whole grain cereals, legumes, whole pulses, lcnfy vegetables, pear, guava, oilseeds, methi seeds, peas, beans, ladies finger, lotus stem. Low residue foods. Refers to volume of inaterials remaining after the digestive processes. Food high in dietary fibre are considered to bc high residue foods. However, low fibre foods need not neccessrily be low residue food e.g. milk is low fibre but a high residue food.

2. Use of methods like sprouting, fermentation etc.; salads and raw fruit may be given in grated form to be able to chew easily, whole pulses and legumes may be sbaked overnight to improve their digestibility, soya flour or whole Bengal gram flour may be added to wheal flour, bran and powdered supplements of fibre may also be given to individuals who do not eat sufficient amounts of fibrous foods.

3. Coconut water, dal water, kanji, barley water, vegetable stock/soup can help promote rehydration. The basic concept behind combining salt, sugar and water

is to prepare a solution of specific osmolarity that would favour water absorption, thus leading to rehydration.

4. Feed the child with WHO-ORS standard preparation/home based rehydration solutions, continue breast feeding young infants, whilc providing energy rich mixturc of weaning foods for older children. This would help in preventing dehydration and prevent further malnourishment of the child.

Check Your Progress Exercise 2

1. Heartburn or burning epigastric substernal pain, regurgitation and dysphagia are the common symptoms. The objectives of nutritional care are to: prevent irritation of the inflammed oesophageal mucosa in the acute phase; prevent oesophageal reflux; and decrease the irritating capacity or acidity of the gastric juice.

2. Decreased muscle tone or abnormal relaxation of the LES; reduced stomach motility or hiatus hernia due to advancing age, pregnancy, obesity or side effects of certain drugs can result in the development of GERD. Patients should be counseled regarding: restricting smoking/alcohol intake, avoiding lying down bending after eating meals, keeping the head and neck raised slightly while lying down, and avoiding astringent or highly flavoured foods.

3. Hiatus Hernia is the most common cause of GERD. Avoid any five of the following: carbonated beverages, citrus fruits and juices, tomato products, spicy foods, coffee, pepper, alcohol, garlic, onion, chocolate, cream sauces, gravies, margarine, butter and oil.

Check Your Progress Exercise 3

1. a) Acute gastritis results due to over eating, overuse of alcohol, tobacco, excess dose of aspirin, anti-inflammatory drugs, increased bile acids, trauma, surgery, shock, jaundice, renal failure, burns, infection while chronic gastritis precedes development of an organic gastric lesion, a cancer or a ulcer. It may be caused by bacterial infections and impaired mucosal defense mechanisms,

   b) Diverticulosis is the herniation of the mucosa through the muscular layers of colonic walls while diverticulitis is an inflammation of the diverticuli.

2. The *lifestyle* ***modifications*** in terms of work schedules, eating behaviour like consuming meals when extremely tired, preventing episodes of GERD, avoiding abdominal discomfort and drug use, quitting alcohol and smoking would help to alleviate the symptoms of dyspepsia.

3. The diet therapy for a patient suffering from diverticulitis may be limited Lo clear liquids progressing to full liquids and then to low residue and fibre soft to normal diet. With improvement, in inflammation, an increase in fibre intake is recommended.

Check Your Progress Exercise 4

1. Gastric and duodenal ulcers are two forms of peptic ulcers. Duodenal ulcers generally occur in the duodenal cap i.e., just above the point of entry of alkaline pancreatic juice. Gastric ulcer on the other hand refer to localized erosion of the lesser curvature of the antrum of stomach.

2. Etiological factors in peptic ulcer involves the following:

   - Destructive factors (HCL, pepsin, psychological, factors, gastric irritants, nicotin and tobacco and anti-inflammatory drugs.
   - Defensive factors (epithelial cells barrier mucous, gastric blood flow, regulation of acid secretion, ability of the body to regenerate the cells.

3. The conventional approach involved restrictions of almost all foods except milk and cream (Sippy's diet). The liberal approach involves giving a variety of foods other than those which may increase damage to the ulcerated area. Since it is the individual patient who is the focus of treatment, it becomes important to treat the patient completely according to his tolerances and desires.

Check **Your** Progress **Exercise 5**

1. Lactose intolerance refers to inability of the body to digest and assimilate lactose. It occurs as a consequence of lactase deficiency, the degree of which may vary from hypolactasia to lactase persistence.

2. Celiac disease is an autoimmune inflammatory disease of the small intestine and is precipitated by the ingestion of gluten, a component of wheat protein while tropical sprue is a malabsorption symdromc of unknown causes that is prevalent in the tropics and sub-tropics.

Five foods containing gluten include: *wheat,* refined flour (maida) rye, barley, ***arid*** *possibly* oats and many processed foods.

3. **a)** Crohn's disease can occur in any part of the GI tract whereas in Ulcerative Colitis, the inflammation is confined to colon and rectum.

b) In Crohn's disease, the inflammatory changes become chronic and can involve any part of GI tract. Inflammatory process follows transmural pattern i.e., affecting the entire thickness of the wall of the small intestine. This leads to strictures resulting in a partial or complete obstruction or formation of fistulas. Ulcerative Colitis relates to acute inflammation of colon lasting for a few weeks and is limited to mucosal and sub mucosal tissues layers of the intestine.

4. The patient is at risk of malnutrition due to:

Decreased food intake due to associated abdominal discomfort.

- Malabsorption due to decreased absorptive surface, bile salt deficiency, bacterial overgrowth and use of drugs.
- Increased secretion and nutrient losses due to GI blood losses, electrolyte, trace mineral losses.
- Increased requirements due to inflammation, fever, increased intestinal cell turnover, haemolysis.
- Drugs interference related to corticosteroids, sulfasalazine, cholestyramine

Deficiency of folate, vitamin A, vitamin C, vitamin D, low serum zinc, copper and iron are observed.