III YEAR –V SEMESTER
COURSE CODE: 7BHF5C1

CORE COURSE - IX – DIET THERAPY

Objectives:

1. Know the principles of diet therapy
2. Understand the modifications of normal diet for therapeutic purposes

Unit - I

Basic concepts of diet therapy

Therapeutic adaptations of normal diet, principles and classification of therapeutic diets.

Routine Hospital Diets: Regular, light, soft fluid, parenteral and enteral feeding. Nutritional care for overweight and obese, Underweight.

Unit - II

Febrile conditions – Typhoid, Tuberculosis and Malarial infections and surgical conditions, GI tract diseases, intestinal diseases and Anemia.

Malabsorption syndrome, celiac sprue, tropical sprue. Intestinal brush border deficiencies, protein losing enteropathy.

Unit - III

Diseases of the Liver – Jaundice, Cirrhosis of liver, Viral Hepatitis, Hepatic Encephalopathy, Wilson's disease. Diseases of Gall Bladder and Pancreas – Cholelithiasis, Cholecystitis, cholecystectomy, Pancreatitis .Diet in disease of the endocrine pancreas – diabetes mellitus.

Unit - IV

Diseases of the cardiovascular system – Atherosclerosis and hyper tension, Diseases of Musculoskeletal system, renal diseases – glomerular nephritis – acute and chronic, End stage renal disease and dialysis.

Unit - V

Management of cancer, Surgery, trauma and burns.Inborn errors of metabolism – biochemical basis and nutritional Management of PKU and Maple Syrup Urine Disease, Allergies: Food allergy, types of allergens, reactions – diagnosis and treatment.

Books for Reference:

1. Mahaj L K Arlin M T (1992) **Kruse's Food, Nutrition and Diet Therapy,** 8th Ed W B Saunders Company, London
2. Mahaj L K Arlin M T (1992) **Kruse's Food, Nutrition and Diet Therapy,** 8th Ed W B Saunders Company, London
3. Joshi S A (1992) **Nutrition and Dietetics**, Tata McGraw Hill Publications, New Delhi
4. Ruth .A.Roth IX (2007) **Nutrition and Diet Therapy** Thomson Delmar Learning, Australia
5. Townsend,C.E(2000) **Nutrition and Diet therapy**, VII edition, Delmar Publisher Albany
6. Williams, M.H (2002) **Nutrition for health and fitness,** Mc Graw Hill, Boston
7. Gibney, M.J et al (2005) **Clinical Nutrition** I edition Blackwell Science

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UNIT I

INTRODUCTION

Diet therapy is concerned with the modification of the normal diet to meet the requirements of the sick individual. The main purposes are

- To maintain good nutritional status;
- To correct deficiencies which may be present;
- To provide rest to the whole body;
- To improve the body's ability to metabolise the nutrients; and
- To bring about changes in body weight whenever necessary.

Diet therapy in most instances is not a remedy by itself but a measure which supplements or makes the medical or surgical treatment more effective.

Therapeutic nutrition begins with the normal diet. Advantages of using normal diet as a basis for therapeutic diets are

- It emphasizes the similarity of psychological and social needs of those who are ill and those who are well, even though there is quantitative and qualitative difference in requirements.
- Food preparation is simplified when the modified diet is based upon the family meal pattern and the number of items required for special preparation is reduced to minimum.
- The calculated values for the basic plan are useful in finding out the effects of addition or omission of certain foods, for example, if vegetables are restricted vitamin A and C deficiency can occur.

Factors to be consider in planning therapeutic diets

The alteration of the normal diet requires an appreciation of

- The underlying disease conditions which require a change in the diet;
- The possible duration of the disease;
- The factors in the diet which must be altered to overcome these conditions; and
- The patient's tolerance for food by mouth. In planning meals for a patient his economic status, his food preferences, his occupation and time of meals should also be considered.

The normal diet may be modified

- To provide change in consistency as in fluid and soft diets;
- To increase or decrease the energy value;
- To include greater or lesser amounts of one or more nutrients, for example, high protein, low sodium, etc;
- To increase or decrease bulk-high and low fibre diets; and
- To provide foods bland in flavour.

The planning of a therapeutic diet implies the ability to adopt the principles of normal nutrition to the various regimens for adequacy, correctness, economy and palatability. It requires recognition of the need for dietary supplements such as vitamin and mineral concentrates when the nature of the diet itself imposes severe

restrictions, the patient's appetite is poor, absorption and utilization are impaired so and the diet cannot meet the needs of optimum nutrition.

Dietary history should help in planning each diet. The dietary history reveals the patient's past habits of eating with respect to dietary adequacy, likes and dislikes, meal hours, where meals are eaten, budgetary problems, ability to obtain and prepare foods. The likes and dislikes of patients are respected because food habits are deep-seated and it is not possible to change them overnight. It requires considerable encouragement and understanding on the part of the doctor-nurse-dietician team to bring about important changes in the diet. Intelligent planning of therapeutic diets necessitates consideration of food costs, the avoidance of waste, and retention of nutrients so that the diet is economically practicable.

TYPES OF DIET

Clear-fluid diet

- Whenever an acute illness or surgery produces a marked intolerance for food as may be evident by nausea, vomiting, anorexia, distension and diarrhoea, it is advisable to restrict the intake of food.
- In acute infections before diagnosis, in acute inflammatory conditions of the intestinal tract, following surgery of the colon or rectum when it is desirable to prevent evacuation from the bowel, etc. clear fluid diet is suggested.
- This diet is also given to relieve thirst, to supply the tissues with water, to aid in the removal of gas.

> The diet is made up of clear liquids that leave no residue; it is non-gas forming, non-irritating and non-stimulating to peristaltic action.

- This diet is entirely inadequate from nutritional standpoint since it is deficient in protein, minerals, vitamins, and calories.
- It should not be continued for more than 24 to 48 hours.
- The amount of fluid is usually restricted to 30 to 60 ml per hour at first, gradually increasing the amount, as per improvement in the patient's tolerance. This diet must provide 300k cal and no protein.
- This diet can meet the requirement of fluids and some minerals and can be given with 1 to 2 hour intervals.

FULL FLUID DIET

- This diet bridges the gap between the clear fluid and soft diet.
- It is used following surgery, acute gastritis, acute infections and during diarrhoeal episodes.
- This diet is also suggested when milk is permitted and for patients not requiring special diet but too ill to eat solid or semisolid foods.
- In this diet foods which are liquid or which readily become liquid on reaching the stomach are given.
- This diet may be made entirely adequate and may be used over an extended time without fear of developing deficiencies, provided it is carefully planned.
- This diet is given at intervals of 2-4 hours intervals. This diet gives 1200kcal and 35g of protein.

SOFT DIET

- This is one of the most frequently used routine diets; many hospital patients are placed on this until a diagnosis is made.
- It bridges the gap between acute illness and convalescence. It may be used in acute infections, following surgery, and for patients who are unable to chew.
- The soft diet is made up of simple, easily digested food and contains no harsh fibre, low in fat and with mild or no seasoning.
- It is nutritionally adequate when planned on the basis of a normal diet. Patients with dental problems are given mechanically soft diet.
- It is often modified further for certain pathologic conditions as bland and low residue diets. In this diet, three meals with intermediate feedings should be given.
- This diet should provide 1500 kcal and 35-40 g of protein. Light diet should be given before regular diet.

REGULAR AND NORMAL DIET

- It is the most frequently used diet in all hospitals.
- It is used for ambulatory and bed ridden patients whose condition does not necessitate a special diet of one of the routine diets.
- Many special diets progress ultimately to a regular diet.

Table gives contents allowed for soft diet, full fluid and clear fluid diet.

Table: Contents of soft, full-fluid and clear-fluid diets

Foods allowed			
Types of food	**Soft diet**	**Full-fluid**	**Clear-fluid**
Cereals	Refined, finely ground whole grain	Gruels, porridges kanji, ragi malt	Barley water
Pulses	All dals	Dal soups, dal payasam	Dal water
Vegetables and Fruits	Juices, pureed, cooked and mashed or baked, ripe banana	Strained juices, cooked and pureed fruits and vegetables.	Clear strained fruit juice
Milk	Milk and milk products, cheese, fine cream	Milk and milk beverages, milk shake, lassi	Whey water

Fats and oils	Butter, oil, cream, margarine	Butter, oil and cream	--
Meat and fish	All except pork, minced fish, poultry	--	--
Eggs	All except fried	Only in beverages	--
Sugar and jaggery	All	Sugar, jaggery and glucose	Sugar or glucose
Nuts and oil seeds	None	None	None
Beverages	All	Tea, coffee, egg, non-carbonated beverages	Tea, coffee (without milk) carbonated beverages, coconut water
Soups	All	Strained	Fat free broth
Desserts	Custard, kheer, pudding	Custard, ice cream plain gelatin	Plain gelatin

- The regular hospital diet is simple in character and preparation, with ease of digestion, and calculated to afford maximum nourishment with minimum effort to the body.
- The diet is well balanced, adequate in nutritional value and attractively served to stimulate a possible poor appetite. This diet gives 1800-2000 kcal and 42-45g of protein.

SPECIAL FEEDING METHOD

Enteral nutrition can be provided either orally or by tube feeding. By definition enteral means "within or by the way of the gastrointestinal tract". In practice enteral nutrition is generally considered as tube feeding

Oral feeding is the best for the nourishment of a patient. But in the following conditions it is not possible to give the feeding orally and tube feeding or parenteral feeding is resorted.

- Those who cannot swallow due to paralysis of the muscles of swallowing (diphtheria, poliomyelitis) or cancer of the oral cavity or larynx.

- Those who cannot be persuaded to eat.
- Those with persistent anorexia requiring forced feeding.
- Semiconscious or unconscious patients.
- Severe malabsorption requiring administration of unpalatable formula.
- Short bowel syndrome.
- Those who are undernourished or at risk of becoming so.
- Those who cannot digest and absorb.
- After surgery.
- Patients with neurological and renal disorders or have chronic fevers or diabetes.
- Babies of very low birth weight.

Tube Feeding

This is done by passing a tube into the stomach or duodenum through the nose which is called nasogastric feeding or directly by surgical operation into stomach known as gastrostomic and into jejunum is called jejunostomic feeding.

A satisfactory tube feeding must be

- Nutritionally adequate
- Well tolerated by patient so that vomiting is not induced
- Easily digested with no unfavourable reactions such as distension, diarrhoea or constipation.
- Easily prepared and
- Inexpensive.

Diet	**Indications**	**Foods to be included and avoided**
High-fibre diet	Atonic constipation Atherosclerosis Obesity Diabetes	Foods included: All fibre richvegetables such as greens like amaranth, cabbage, lettuce, celery spinach, etc. special stress on raw vegetable salads. All fruits, with skins when tender whole grain cereals and breads with bran in fine division. Milk, meat, fish, fowl, eggs as desired for normal nutrition. Foods to avoid: Highly refined and concentrated foods, fried foods, excessive amounts of coarse bran, excessive seasonings. Intervals of feeding: three meals daily, water and fruit juices between meals before breakfast and

		before retiring.
Soft moderately high fibre diet.	When there is abnormal irritation of the intestines, bulk must be provided in smooth and finely divided form Spastic constipation Mucous colitis Peptic ulcer.	Food included: modify soft diet to include only pureed fruits and vegetables in increased amounts. Foods to avoid: see soft diet Intervals of feeding: three to six small meals.
Very low residue diet	In severe diarrhoea the gastrointestinal tract needs to be given rest. Ulcerative colitis during initial stages of treatment. Preceding and following surgery on the colon or rectum when no movement is desired for several days. Partial intestinal obstruction.	Foods include: Tender meat, fish, or fowl, clear fat free soups, fruit juices gelatin desserts, eggs, arrowroot cookies, refined cereals and breads, carbonated beverages, coffee, tea, butter and sugar. Foods to avoid: Coarse breads and cereals; cheese, milk rich desserts, excessive sweets. Intervals of feeding: Usually three meals daily.
Bland diet	To prevent stimulation of peristalisis and flow of gastric juice by mechanical or chemical irritation, and to reduce inflammation gastric and duodenal ulcers peptic ulcer.	

OBESITY

INTRODUCTION

One of the most common problems related to lifestyle today is overweight. Severe overweight or obesity is a key risk factor in the development of many chronic diseases such as heart and respiratory diseases, non-insulin-dependent diabetes mellitus or Type 2 diabetes mellitus, hypertension and some cancers, as well as early death.

Obesity and overweight are serious problems that pose a huge and growing financial burden on national resources. However, the conditions are largely preventable through sensible lifestyle changes.

> **Obesity:** is a condition characterized by excess body fat, usually defined as weighing 20% above desirable weight. **Over weight** is a condition where the body weight is 10-20 per cent greater than the mean standard weight for age, height and sex.

CAUSES

Obesity is a multifactorial disease influenced by both genetic and environmental factors like social, behavioural, psychological, metabolic, cellular and molecular factors

1. **Genetic factors**
   - Genetic base regulates species differences in body fat and sexual differences within the species.
   - Genetic inheritance influences 50-70%
   - If both parents are obese, chances of inheritance is 80% and with one parent obese the chance is 50%.
   - ? receptor in adipose tissue is causative factor
2. **Age and sex**
   - It can occur at any age, in either sex as long as the person is under positive energy balance.
   - As per NFI studies females are more prone to become obese than males
3. **Eating habits**
   - Nibbling between meals.
   - Fast eating with less chewing.
   - Responding to external cues rather than internal hunger.
   - Fond of cooking varieties of food.
   - Frequent business lunches.
   - Forcible consumption to prevent leftovers.
   - Eating food outside more frequently, consumption of highly processed and junk foods.
   - Cultural practices influencing consumption of more sweets and fats.
   - Non inclusion of fruits and vegetables or eating more non vegetarian foods.
   - Depression leading to overeating.

- Aggression and sophisticated marketing practices.

4. **Physical activity:** Sedentary life, not participating in games and sports and using vehicles for commuting.
5. **Stress:** Excess release of endorphins, leads to self gratification, self punishment, depression anxiety
6. **Endocrine factors:** Hypothyroidism, hypogonodism and also stages like puberty, pregnancy and menopause
7. **Trauma:** Damage to hypothalamus leads to lack of regulation of appetite or satiety
8. Prosperity and civilization

Theories on obesity

1. **Fat cell theory:** The number of fat cells is determined during early in life to provide space to store fat. Juvenile obesity - increase in the number of fat cells, increase adult obesity due to increase in size of fat cells.
2. **Set point theory:** Once the body reaches ideal body weight or set point, body gets signals for food intake.
3. **Leptin:** Obesity gene called ob codes for protein, leptin, acts as a hormone of hypothalamus to promote negative energy balance.

ASSESSMENT

Body weight: >10% of normal body weight is classified as over weight >20% of normal body weight is classified as obesity

% Body weight >normal	**Degree of obesity**
25	Mild
50	Moderate
75	Severe
100	Very severe/ morbidity

Body mass index (Quetlet index) weight (kg)/height 2(m)

<18.5	**Under weight**
18.5-24.9	Normal
25-29.9	Grade I obesity
30-40	Grade II obesity
>40	Grade III obesity

Body Mass Index: Weight (in kilograms) divided by height squared (in meters); a value of 30 or more indicates obesity.

Waist circumference

1. Men: >40 inches (>102cm)
2. Women: >35 inches (88cm)

Measurement of body fat

3. Harpender calipers: fat at triceps (85-90mm)
4. Lange calipers: abdomen and sub scapular regions
5. USA MRNL calipers: sub coastal sites

Waist to hip ratio: Normal 0.7

Broka's Index= Height in cm-100 = ideal weight (kg)

Metabolic changes

Glucose tolerance	
Sensitivity to insulin	
Plasma insulin	
Response to starvation	
Production of ketone bodies	
Plasma free fatty acids	
Plasma triglycerides	
Plasma Cholesterol	
Plasma uric acid	
Sensitivity to growth hormone	
Urinary 17-hydroxycorticoids	

Regional distribution of adipose tissue

Weight gain in the area of and above the waist (apple type) is more dangerous than weight gain around the hips and flank area (pear type).

Fat cells in the upper body have different qualities than those found in hips and thighs.

TREATMENT

Strategies for weight loss and weight maintenance

1. **Diet therapy**
2. **Physical exercise**
3. **Stress management**
4. **Pharmacotherapy**
5. **Weight loss surgery**

DIET THERAPY

Diet to promote weight loss are generally divided into four categories: low-fat, low-carbohydrate, low-calorie, and very low calorie.

Very low-calorie diets

- Very low calorie diets provide 200-800 k cal/day while maintaining normal protein intake and limiting calories from both fat and carbohydrates.
- They subject the body to starvation and produce an average weekly weight loss of 1.5-2.5 kilograms.
- These diets are not recommended for general use as they are associated with adverse side effects such as loss of lean muscle mass, increased risks of gout, and electrolyte imbalances.
- People attempting these diets must be monitored closely by a physician to prevent complications.

Low-calorie diets (Reducing diet)

Low-calorie diets usually produce an energy deficit of 500-1000 calories per day, which can result in a 0.5 kilogram weight loss per week.

Low-carbohydrate diets (weight maintenance diet)

- Provides 1500-1800 k cal.
- Introduced when body weights are reduced to optimal level due to reducing diets. Low carbohydrate diets such as Atkin's and protein power are relatively high in fat and protein.
- They are very popular in the press but are not recommended by the American Heart Association.
- A review of 94 trials found that weight loss was associated with increased satiety and thus decreased calorie consumption.
- No adverse affect from low carbohydrate diets were detected.

Low-fat diets

Low-fat diets involve the reduction of the percentage of fat in one's diet. Calorie consumption is reduced but not purposely.

Principles of dietetic management

Low calorie, normal protein, vitamin and mineral (except sodium), restricted carbohydrate, fat liberal fluid, and high fibre

- **Energy:** 29k cal/kg ideal body weight for moderate worker
  25 k cal/kg ideal body weight for moderate worker
  - Consumption of low Glycemic index foods
    1. By promoting satiety
    2. By promoting fat oxidation at the expense of carbohydrate oxidation
- **Protein:** 0.8-1 g/kg body weight
- **Carbohydrate:** rich foods like potatoes, rice, sugar and fruits like banana should be avoided.
- **Fat:** Low or no fat.
- **Vitamins:** as fat is restricted, fat soluble vitamins also become restricted and so they should be supplemented orally
- **Minerals:** restricted sodium level and more calcium to maintain normal weight. Depressing certain hormones are also helpful in destruction of fat cells
- **Fluids:** liberal amount of water

High fibre diets

- Low in calorie density
- Green leafy vegetables provide vitamins and minerals
- Satiety
- Regulate bowel movements
- Reduce blood cholesterol
- Promote chewing and decreases rate of digestion

PHYSICAL EXCERCISE

- Low calorie diet with exercise is effective in weight loss.
- Aerobic exercises directly increases energy expenditure, preserves lean body mass and prevents decrease in basal energy expenditure.
- Regulates appetite and set point

Stress management

- Diaphragmatic breathing
- Deep muscle relaxation
- Meditation, yoga and physical activity

Pharmacotherapy: (BMI>30)

Drug therapy may be used as part of comprehensive weight loss programme along with diet therapy, physical activity

- Not preferred without changing the life style

Behaviour therapy:

- This involves changing diets (by eating smaller meals), habits (by cutting down certain types of food)
- Physical activities (by making a conscious effort to exercise for a longer period) to new behaviour that encourage weight loss

WEIGHT LOSS SURGERY

- Bariatric surgery ("weight loss surgery") is the use of surgical interventions in the treatment of obesity.
- As every operation may have complications, surgery is only recommended for severely obese/ morbid obese people (BMI>40) who have failed to lose weight by dietary modification and pharmacological treatment.
- Weight loss surgery relies on various principles; the most common approaches are reducing the volume of the stomach, producing an earlier sense of satiation (e.g. by adjustable gastric banding and vertical banded gastroplasty) and reduce the length of bowel that food will be in contact with, directly reducing absorption (gastric bypass surgery). Complications from weight loss surgery are frequent.

COMPLICATIONS OF OBESITY

Obesity has been reported to cause or exacerbate a large number of health problems, which has known impact on both life expectancy and quality of life. Obesity leads to direct physical disability since the feet have to carry extra load, complications like flat feet, osteoarthritis of knee, hips and lumbar spine are common. The abdominal muscles that support the viscera and leg muscles are infiltered with fat hence their normal mechanical action is impaired with abdominal hernias and varicose veins. Adipose tissue around chest and diaphragm interferes with respiration and predisposes bronchitis.

1. **Metabolic disorders**
   - There's a close association between obesity and diabetes. In simple obesity, there is insulin resistance especially in muscle and there is hyper-insulinemia because of impaired insulin uptake by receptors in target tissues.
   - Plasma cholesterol level is generally high therefore prone to develop gall stones.
   - Excess cholesterol in plasma leads to deposition in intima of arteries producing atherosclerosis.
   - Gout is common.
2. **Cardiovascular disorders** Apart from atherosclerosis, obese people develop high blood pressure and increased incidence of varicose veins.
3. **Sleep apnea** A transitory cessation of breathing is increasingly identified in obese children and adolescents.
4. **Prone to accidents** obese people are likely to meet with accidents by falling down on slippery floors and crossing streets.
5. **Gall Stones** Obese people have higher output of cholesterol in bile, with a lower concentration. So their bile is constantly in danger of forming gall stones.

6. **Osteoarthritis** Degenerative disease of weight-bearing joints is a very common complication of obesity, in the knees of middle-aged women and causes significant disability.
7. **Obstetrical risks** obese women when pregnant have greater obstetrical risks because of hypertension, diabetes and postpartum infection. Restriction of weight gain in these individuals may cause complication during pregnancy. There is an increased risk of neural tube defects in newborn of obese mothers. There is reduced fertility in obese subjects.
8. **Psychological disturbances** obese adolescents have been found to have personality characteristics of self blame, withdrawal and feeling of inferiority.
9. **Low life expectancy** The statistics of Metropolitan Life Insurance USA shows that for a man aged 45 with an increase of 12 kg body weight above standard weight reduces his life expectancy by 25 per cent. The relative risk associated with obesity decreases with age over 75.
10. **Cancer** Certain cancers (colon, rectum and prostate in men; uterus, biliary tract, breast and ovary in women) digestive tract diseases (gallstones, reflux, oesophagitis) and skin disorders are more prevalent in the obese.

Advantages of weight loss

- Reduction in blood pressure
- Reduction in total cholesterol and LDL cholesterol
- Increase in physical activity improving HDL levels
- Lowering of elevated blood glucose level

Weight loss reduces the risk factors for diabetes mellitus and coronary heart disease and cancer.

COMPLICATIONS

Medical field	Condition	Medical field	Condition
Cardiovascular	• Ischemic heart disease: angina and myocardial infarction • Congestive heart failure: 12% attributable to obesity • High blood pressure: present in 85% of those with BMI>25 • High cholesterol	Gastrointestinal	• Gastroesophageal reflux disease • Fatty liver disease • Cholelithiasis (gallstones) • Hernia

	• Thrombosis and pulmonary embolism		
Endocrine and reproductive	• Diabetes mellitus • Polycystic ovarian syndrome • Menstrual disorders • Infertility • Complications fr om pregnancy • Birth defects	Respiratory	• Obstructive sleep apnea • Obesity hypoventila tion syndrome • Asthma • Complications from general anaesthesia
Musculoskele tal	• Gout • Immobility • Osteoarthritis • Low back pain	Psychological	• Depression in women • Low self esteem • Body dysmorphic disorder • Social stigmatization
Neurologic	• Stroke • Meralgia paresthetica • Headache • Carpal tunnel syndrome • Dementia • Idiopathic intracranial hypertension	Skin	• Stretch marks • Acanthosis nigricans • Lymphedema • Cellulitis • Carbuncles • Intertrigo
Cancer	• Breast • Esophageal • Colorectal • Liver • Gallbladder • Pancreatic • Stomach, prostate • Endomaterial, cervical • Ovarian • Kidney • Non-Hodgkin's lymphoma • Multiple	Genitourinar y	• Erectile dysfunction • Urinary incontinence • Chronic renal failure • Hypogonadism • Stillbirth

	myeloma		

UNDERWEIGHT

INTRODUCTION

- Half the Indian rural population is underweight with up to three-quarters in deprived communities.
- A very large proportion of rural Indian households have inadequate food supplies. Chronic energy deficiency is due to chronic food deficiency.
- People whose BMI is less than 18.5 are considered as underweight.

LIMITATION OF UNDERWEIGHT

- Adults with BMI < 18.5 have reduced VO2 max, reduced capacity for sustained heavy work and a lower productivity.
- Pregnant women show a proportional increase in the risk of an underweight baby born in relation to post-partum BMI and the BMI ranges from 25 to 16 or less.
- Progressively greater proportions of time are spent off work or in bed ill when BMI levels are below 17 in men and women.
- There is a progressive curvilinear increase in mortality in group of men with BMI ranging from 18.5 to below 16.0.
- Immunological defects are seen particularly in men with low BMI concomitant micronutrient deficiencies are present.
- Undernourished children have lower height and weight.
- The age of menarche is delayed in undernourished girls by 1-2 years.

AETIOLOGY

Some of the causes of underweight are:

- Starvation occurs either due to famine conditions or an inadequate diet inadequate in proteins or an attempt at reducing weight. During starvation fatty tissue is lost and the skeletal, heart muscles and small intestine are atrophied losing its absorptive function. This results in low blood pressure, marked emaciation, loss of hair and inelastic skin. Since the feeding programme should be gradual initially glucose water, fruit juice and skimmed milk powder may be given..
- Underweight also results from debilitating diseases like tuberculosis, diabetes, malabsorption syndrome or cancer. Infections are common among them. In these cases tonics are not useful as they only help to improve the appetite but they do not increase weight. It is wise and necessary to spend money on nourishing high calorie foods.

DIETARY MODIFICATION

A high calorie, high protein, high fat diet with liberal vitamin intake is recommended. But before going into the diet the first step is to determine and eliminate the cause for under eating. Then a balanced diet should be planned based on the requirements.

- **Energy** The calorie requirements vary depending upon the activities. For increasing weight the total calorie intake should be in excess of the energy requirement. An additional 500 k cal per day is recommended. The increase should be gradual over one or two weeks otherwise digestive disturbances may occur.
- **Proteins** Instead of 1 g of protein, over 1.2g per kg is recommended for tissue building. Good quality protein is completely utilized by the body so as far as possible best protein sources must be liberally included in the initial stage.
- **Fats** Even though fat content is increased easily digestible fats are to be included. Fried and fatty foods are not recommended as they may cause diarrhoea. Fatty food should not be taken at the beginning of a meal as they reduce appetite. High calorie fatty foods such as cream, butter, margarine and oils help to increase the weight.
- **Carbohydrates** High carbohydrate sources must form the basis of the diet. Leafy vegetables should be restricted and preference to be given to potato and yam. Dried fruits, sweets, nuts, desserts, jam, jelly, cereals, cereal products and non vegetarian foods are rich source of energy and can be liberally included in the diet. The number of meals should be increased. Two feeds incorporating soups, juices or sweets in between major meals improve the nutritive value of the diet. Easily digestible foods should be given. Porridge, cutlets, desserts, potato chips, high protein drinks like milk, malted milk, and badam kheer can be included. Thick soups are easily digestible and highly nutritious.
- **Vitamins** and minerals With a liberal diet there is no need for extra vitamin and mineral supplement.
- **Fluids** Fluids should not be taken before or with a meal but only after a meal so that food intake is not reduced. Enough fluids must be taken so as to avoid constipation.

UNIT II

FEVER

DEFINITION

- Fever is an elevation in body temperature above normal which may occur due to exogenous and endogenous factors.

CAUSES

Endogenous factors: antigen-antibody, malignancy and graft rejections.
Exogenous factors: bacteria or fungi.
Development of fever due to exogenous agents

TYPES

Short duration fever: colds, tonsillitis, influenza and typhoid
Chronic fever: tuberculosis
Intermittent: malaria
Drugs like aspirin are effective in reducing fever because they inhibit prostaglandin synthesis.

METABOLIC CHANGES IN FEVER

- An increase in the metabolic rate @ 13 per cent for 10C rise in body temperature (7 per cent for 10F); increase is also due to restlessness and hence caloric need increases.
- Decreased glycogen stores and decreased stores of adipose tissue.
- Increased catabolism of protein places an additional burden upon the kidneys.
- Accelerated loss of body water owing to increased perspiration and the excretion of body wastes.
- Increased excretion of sodium and potassium.

GENERAL DIETARY CONSIDERATION

ENERGYIncreased by 50%, if the temperature is high and tissue damage is high should be able to ingest 600-1200 k cal daily.

Protein: During prolonged illness 100g of protein with liberal calorie intake for efficient utilization. High protein beverages can be supplemented.

Carbohydrates: Glycogen stores are replenished by readily absorbable glucose.

Fats: Judiciously increased. Fried foods to be avoided.

Minerals: Sufficient intake of NaCl through soups, fruit juices and milk for calcium.

Vitamins: Vitamin A, C and B-complex requirements increase in proportion to calories. Oral therapy of antibiotics in short courses interferes the absorption of B-complex vitamins, hence necessitates supplementation.

Fluid: Must be liberal to compensate the losses from sweat and to permit adequate volume of urine for excreting the waste. 2500-5000ml/day.

Ease of digestion: Bland, readily digested, soft or of regular consistency food should be used to facilitate digestion and rapid absorption.

Intervals of feeding:

- Small quantities of food at intervals of 2-3 hrs to provide nutrition without overtaxing the digestive system.
- Upon improvement 3 meals can be given along with bedtime feeding
- The duration of fevers is shortened by antibiotic and drug therapy therefore the nutritional needs can be met without difficulty
- In acute fevers as the patient's appetite is often very poor, small feedings of soft or liquid food at frequent intervals is desired
- Sufficient intake of fluids and salt is essential

If the illness persists for more than few days high calorie and high protein is emphasized.

1. **Tuberculosis**
2. **Typhoid**
3. **Influenza**
4. **Malaria**

TUBERCULOSIS

Tuberculosis is an infectious disease caused by the bacillus Mycobacterium tuberculosis. It affects the lymph nodes, intestine, meninges, bones and joints, skin etc.

Symptoms

Pulmonary tuberculosis is accompanied by

- wasting of tissues
- exhaustion
- cough
- expectoration (cough with respiratory secretions) and
- fever

The acute phase resembles pneumonia, with high fever and increased circulation and respiration.

The chronic phase is accompanied by low-grade fever. Though the metabolic rate is high, it is lower than in acute fever.

Diet:

Energy High calorie diet with 2500 to 3000 Kcal. It is not desirable to gain more than 10 per cent above the ideal body weight.

Protein Serum albumin levels are decreased, therefore, 80 to 120 g of protein per day is desirable.

Minerals

- The drug isoniazid interferes with vitamin D metabolism.
- Decreased absorption of calcium and phosphorus. Therefore liberal amount of calcium for healing tuberculosis lesions.
- Iron as per the level of haemorrhages.
- Calcium, iron and phosphorus help in regeneration of cells, blood and fluids.

Vitamins

Vitamin A and C requirement increases.

Isoniazid is antimetabolite of pyridoxine and prevents the formation of pyridoxal phosphate. Hence 50 to 100 g of pyridoxine is necessary.

Diet:

High calorie, high protein, high vitamin, high minerals, high fluid and soft diet is recommended. Along with high calorie, high protein, high vitamin and minerals. Include one litre of milk and 3-4 eggs/day.

TYPHOID

Typhoid is caused by Salmonella typhi and Paratyphoid B by S.scottmulleri is an infectious disease with an acute fever of short duration.

Source of infection: Feaces and urine of the patient or carriers. Drinking water, milk, food infected with intestinal contents of the patients.

Signs and symptoms:

- Continued high inflammation of the intestine
- Formation of intestinal ulcers, haemorrhage
- Enlargement of spleen
- Peyer's patches or flat patches of lymphatic tissue at small intestine and ileum
- Diarrhoea or constipation and severe stomach ache
- Anorexia and headache due to poor nutrient absorption

Diet:

High calorie, high protein, high carbohydrate, low fat, high fluid, low fibre and bland diet.

At first clear fluid diet followed by full fluid and soft diet is suggested.

Febrile condition alters water and electrolyte balance, so more liquids must be included.

INFLUENZA

Influenza is an acute infection of short duration. It spreads from person to person by contact and inhalation of virus. A sneeze or cough from an infected individual produces many droplets containing virus and these may be inhaled by those nearby.

Symptoms

Headache, lassitude, myalgia, shivering, sore throat, pyrexia and fever.

Diet:

General principles of dietary treatment are followed for influenza patient.

MALARIA

Four species of the genus plasmodium are responsible for human malaria. P. vivax, P. malaria, P.ovale and P. falciparum.

Signs and symptoms:

Shaking chills (the cold stage), fever (the hot stage) to 41°C or higher and the sweating stage.

Associated symptoms include fatigue, headache, dizziness, gastrointestinal symptoms (anorexia, nausea, mild diarrhoea, and vomiting, abdominal cramps) myalgia, arthralgia, backache and dry cough.

Diet:

Dietary management is same as that of fever.

GASTRO INTESTINAL DISEASE

INTRODUCTION

The digestive tract is a group of 6 separate and interrelated organ systems designed to digest food, absorb nutrients and eliminate metabolized products.

These organ systems are:

- The oesophagus
- The stomach
- The pancreas
- The hepatobiliary tract (liver and bile ducts, gall bladder)
- The small intestine
- The colon

These organ systems have individual and interrelated abnormalities requiring different methods to assess and evaluate.

The gastro intestinal tract converts foods to the form usable by the body for carrying out various functions. Any diseases of the gastro intestinal tract therefore will affect the availability of nutrients to the body and the nutritional status of the individual.

Gastro intestinal diseases

Indigestion	Abdominal discomfort
Peptic ulcer	Erosion of mucus membrane, Helicobacter pylori
Carcinoma	Any part of the GI tract
Dumping syndrome	Low regulation on emptying
Steatorrhoea	Fat in stools
Lactose intolerance	Low -? glucosidase lactase
Coeliac disease	Gluten sensitive enteropathy
Tropical sprue	Reaction to gliadin
Irritable bowel syndrome	Change in life cycle
Inflammatory bowel disease	Inflammation of mucus
Ulcerative colitis	Bloody diarrhoea
Intestinal gas and flatulence	High fibre

Diverticular disease	Sac like herniations
Diarrhoea	
Constipation	

PEPTIC ULCER

A **peptic ulcer** is an erosion in the lining of the stomach, duodenum, or oesophagus. An ulcer is a sore or erosion that forms when the lining of the digestive system is corroded by the acidic digestive juices, if neglected, leads to formation of a hole. It is estimated that between 5% to 10% of adults globally are affected by peptic ulcer at least once in their lifetime.

When peptic ulcer affects the stomach it is called gastric ulcer. If it affects duodenum it is called a duodenal ulcer, while an esophageal ulcer is an ulcer in the oesophagus. When the lining of these organs is corroded by acidic digestive juices secreted by stomach cells peptic ulcers can form.

The first symptom of peptic ulcer is usually stomach pain - caused by the ulcer and intensified by stomach acid coming in contact with it. However, it is not uncommon for people to have a peptic ulcer and no symptoms at all. Even though stomach ache is the first symptom, most stomach aches are not serious and do not mean the individual is ill.

1. **Aetiology**
2. **Symptoms and Clinical Findings**
3. **Diagnosis**
4. **Treatment**
5. **Drugs**
6. **Dietary management**
7. **Dietary guidelines**

AETIOLOGY

- **Bacterial infection:** Helicobacter pyroli is the chief cause of ulcer. It is spiral shaped, unipolar flagellum and is associated with astral gastritis and duodenitis in the presence of gastric metaphasia. If Helicobacter pylori is cleared by antibiotic treatment especially with colloidal bismuth and amoxicillin, the associated gastritis improves ulcer healing and recurrence rates may be lower.
- **Genetic factors:** It is common in persons with blood group 'O' than in those of other groups and possibly in those with HLA-B5 antigens. People who are first degree relatives of patients with duodenal ulcer have an increased risk of developing duodenal ulcer.
- **Sex:** Men are affected two to three times more frequently than women.
- **Age:** The incidence is high between 20 and 40 years though the average age of incidence has increased. During these years career and personal strivings are at a peak.
- **Stress:** People who are highly nervous and emotional and who worry, fear and feel anxiety are particularly susceptible. These emotional and nervous factors in turn may lead to hyper¬secretion and hypermotility of the stomach. The nervous control of the vascular system in the gastric or duodenal walls may be so disturbed that there is diminution in the blood supply to the mucosa of the stomach and duodenum making it susceptible to acid secretion.
- **Potentially irritant substances:** Caffeine, ethanol, aspirin and nicotine may delay healing but there is little evidence to show that these substances induce ulcer. Chillies, pepper, ginger, garam masala, meat soups, strong tea or coffee and protein rich foods increase the secretion of hydrochloric acid and aggravate the condition.
- **High fibre diet:** In India the incidence of peptic ulcer is low where the staple diet is millet or wheat compared to rice eating areas. This theory is yet to be confirmed.
- **Emergency injuries:** Stress ulcers occur in conjunction with emergency injuries such as burns or long-term rehabilitation processes.

SYMPTOMS

- Epigastric pain, heart burn etc., due to reflux of acid into oesophagus occurring as deep hunger contraction 1 to 3 hours after meals is often the chief complaint. The pain may be described as dull, piercing, burning or gnawing and is usually relieved by taking food or alkalies.
- Discomfort and flatulence in upper part of abdomen. The basis for the pain may be the action of un-neutralised hydrochloric acid on exposed nerve fibres at the site of the ulcer.
- Pain is also associated with hypermotility of the stomach or gastric distension following ingestion of large amounts of food or liquids.
- Low plasma protein levels are often present and delay rapid and complete healing of the ulcer.
- Weight loss and iron deficiency anaemia are common.
- The intake of iron, ascorbic acid, and B-complex vitamins, particularly thiamine may be less than desirable because of self imposed limitation of green leafy vegetables and other good sources of these nutrients.

- In some instances, haemorrhage is the first indication of an ulcer and requires surgical intervention. Other complications such as obstruction, perforation and carcinoma are treated surgically.
- Bleeding ulcers can result in vomiting known as haematemesis (dark brown in color).
- There are spasms of pyloric canal and this may give rise to a feeling of sickness, distension and prevent taking food.

DIAGNOSIS

- **Radiographic examination with barium meal** The technique, though simple, carries a fairly substantial risk of missing the problem.
- **Endoscopy** A flexible tube made of fibre optic bundles is introduced into the stomach and the endoscopist inspects the food pipe and stomach and detects any breaks in the lining membrane. It takes 15-20 minutes. If there is cancer, it can also be detected by an endoscope.
- **Biopsy** of lining tissue.
- **Acid secretion of the stomach** In this the acid output after stimulation by pentagastrin is measured. It is useful for further investigation if surgery is contemplated. Acid output is higher than normal in duodenal ulcer and low or absent in patients with carcinoma of the stomach.

TREATMENT

Therapeutic goal:

- To relieve the symptoms
- To allow healing of ulcer
- To prevent complications like surgery.

DRUGS

It is important to neutralize the excess acid produced.

- Antacids were the earliest and most logical method of providing relief. They continue to be effective but have fallen out of routine use because they are cumbersome to prescribe, large quantities have to be taken several times a day.
- A group of chemicals are available that block certain vital steps in acid production by the stomach called H2 blockers. They are effective, devoid of major side effects and are given once or at the most twice daily.
- A third group of drugs, instead of attempting to lower or neutralize acid, they block the resistance of the lining of the stomach and prevent breakdown.

DIETARY MANAGEMENT

It was customary to suggest bland diet for ulcer patients. Bland diet is a diet which is mechanically, chemically and thermally non-irritating.

Mechanically irritating foods include those with indigestible carbohydrate, such as whole grains and most raw fruits and vegetables. Foods believed to be chemically irritating because of their stimulatory effect on gastric secretion include meat extractives, caffeine, alcohol, and some spicy foods. The capsaicin present in chillies causes shedding of surface stomach cells and may cause peptic distress. Foods believed to be thermally irritating such as very hot or iced liquids may cause pain. This diet prevents irritation to the mucosa, avoids increase in acidity and aids in control of pain. Sippy's diet, Lenhartz's diet and Meulangracht diets were given in the past.

The so-called bland diets used in the past for treatment of peptic ulcer have proved to be unwarranted and ineffective. A more liberal individual approach prevails today in modern clinical practice.

- **Sound total nutrition:** There must be optimal overall nutritional intake to support recovery and maintain healthy tissue, based on individual needs and food tolerance.
- **Protein foods:** Milk and protein foods do have some buffering effect but they also evoke gastric secretions more than carbohydrates and fats. Milk should be included as a source of nutrient factors for healing purposes. Protein provides the necessary amino acids for synthesis of tissue protein which helps in healing ulcer.
- **Fat:** Moderate amount of fat helps to suppress gastric secretion and motility through the enterogastrone mechanism. Fats such as cream, butter and. olive oil can be particularly helpful in a thin patient. Fried foods are not advised as they are difficult to digest and often aggravate the symptoms.
- **Ascorbic acid:** It helps in wound healing hence citrus fruit juice and tomato juice can be given. The pH of food before ingestion has little significance. No food is sufficiently acidic i.e., by itself to effect a significant change in pH or cause direct irritation on an ulcer. For majority of patients hospitalized for an active peptic ulcer some type of bland diet is commonly used.

- **Gas formers:** In addition certain foods traditionally forbidden include strongly flavoured vegetables such as cabbage, cauliflower, onions turnips and fried foods. Restriction of these foods is based on subjective evidence from patients who experience distress following ingestion of these items.
- **Fibre:** A regular diet, including good food sources of dietary fibre, has proved to be beneficial.

DIETARY GUIDELINES

- A patient on a bland diet or regular diet, should be aware of foods needed for a nutritionally adequate diet and the importance of including these daily.
- He should select food from a wide variety of foods, omitting foods known to distress to the patient.
- Moderate use of seasonings are permitted.

- Regularity of mealtimes is essential. The patient gets benefited by small and frequent meals.
- In between meals, protein rich snacks should be taken.
- Moderate amounts of food should be eaten. Heavy meals should be avoided. Volume of any foods sufficient to exert astral pressure against the stomach wall stimulates gastric secretion through the gastric mechanism.
- The diet should be planned in consultation with patient, taking into consideration his preferences, cultural pattern and economic status.
- Meals eaten outside the house will not cause any problem if good judgement is used in food selection.
- Meals should be eaten in a relaxed atmosphere and should forget personal or family problems while eating.
- A short rest before and after meals may be conducive for greater enjoyment of meals.
- Food should be eaten slowly and chewed well. How one eats is more important than what one eats because fast eating provokes gastric feeding reflex

DIARRHOEA

Diarrhoea is the passage of 3 or more loose or liquid stools per day, or more frequently than normal for the individual. It is usually a symptom of gastrointestinal infection, which can be caused by a variety of bacterial, viral and parasitic organisms. Infection is spread through contaminated food or drinking-water, or from person to person as a result of poor hygiene

TYPES OF DIARRHOEA

Three clinical syndromes of diarrhoea reflecting a different pathogenesis and treatment approaches

1. **Acute watery diarrhoea:** with a sudden change in bowel habits resulting in loose and frequent stools without blood, more voluminous in nature, sometimes associated with fever
   Causes:
   - Infections of bacteria (50%)
     Ex: E. Coli, Salmonella, Shigella, Clostridium difficile (C. difficile), Campylobacter
   - Parasites: giardia, entamoeba,
   - Viral infections (30-40%)
     Rota, Corono, Astro, Norwalk and Adeno virus
   - Food poisoning due to bacteria infecting food and stored improperly.
   - Numerous medications may also cause diarrhoea
     Ex: antacids, antibiotics, antihypertensive medications, laxatives, magnesium supplements, potassium supplements and various cardiac medications
2. **Dysentery:** Diarrhoea with visible blood, fever and tenesmus
3. **Chronic diarrhoea:** Long lasting diarrhoea due to non infectious causes.
   Causes:

- Sensitivity to gluten or inherited metabolic disorders
- Vagotomy or gastrectomy, cholecystectomy
- Endocrine diseases as hyperthyroidism, diabetes mellitus, hypoparathyroidism

Osmotic diarrhoea: Passage of large, frothy acidic stools with severe dehydration and hypernatremia

- Due to high osmolar solutions

Physiological Changes in the body

- Water constitutes about 75% at birth and 60% in later age of body weight.
- Diarrhoea losses comes from ECF and replacement should be with sodium rich and low potassium solutions
- Loss of the water from the body causes a reduction or shrinkage in the volume of extracellular compartment

1. **Excessive loss of sodium in diarrhoeal stools**
   - Causes decline in serum and ECF sodium level (hyponatremia)
   - Osmolality of ECF fall causing movement of water from extra cellular to intracellular compartment
   - Decrease in blood volume due to depletion of extracellular compartment
   - Results in weak thready pulse and fall in blood pressure
   - Extremities appear cold due to low hydrostatic pressure in the renal glomeruli
   - Filtration of urine reduced
   - The quantity and frequency of urination falls down
2. **Excessive potassium lost through stools**
   - Abdominal distension and hypotonicity of muscles
3. **Excessive bicarbonates loss**
   - Breathing will be deep and rapid if base level of bicarbonates fall to 12m mol/l

ACUTE DIARRHOEA

Acute diarrhoea in weaning:

- Indigestion
- Introducing weaning foods too early
- Low secretion of digestive enzymes
- Poor food hygiene
- Improper handling of food
- Food intolerance and food allergy

Diet in weaning diarrhoea:

- Fluid management
- Encouragement for breast feeding
- Better food hygiene
- Improvement of nutritional status of children
- Environmental sanitation

FLUID MANAGEMENT
To maintain renal function with electrolyte and pH balance

- Early replacement of fluid losses at the first sign of liquid stool

- Giving plenty of fluids during illness to prevent dehydration
- Small sips to prevent hyperactive gastro-colic reflex

Initial management with any fluid:

- Offering ample fluids as the child can take orally without vomiting
- Coconut water, butter milk, rice kanji with salt,
- lemon-sugar-salt beverage, weak tea

Oral rehydration therapy with homemade solutions:

ORAL REHYDRATION

for prolonged diarrhoea and dehydration

Composition of ORS (WHO)

Components	Amount g/lit
Glucose	20 (90 mEqNa)
Sodium chloride	3.5 (20mEqK)
citrate	2.9 (80 mEq CI)
Sodium carbonate	2.5 (30 mEq C03)
Potassium chloride	5

For 1 yr child 1000 ml/24 hrs

1. Continue breast feeding during attack of diarrhoea as it
   - Aids in recovery of nutrients and rehydration capacity
   - Prevents further infection
2. The bowel should not be rested
3. Avoid milk and other lactose containing products for a day or two
4. Milk should be diluted with equal volume of boiled and cooled water and feed along with ORS till diarrhoea stops
5. Fermented milk with S. haemophilus and L. bulgaricus reduces growth of bacteria
6. As the child develops PEM, give easily digestible, nutritionally balanced diet
7. Rice based solutions potato, millet, maize and other cereal flours
8. For older infants well cooked milled cereal with lentils preferred
9. Mashed bananas
10. Iso osmolar diets
11. Little oil and fats
12. Precooked and amylase rich food

In chronic diarrhoea

- Low milk- milk free- starch free diet in succession
- Usage of ORT
- Avoiding inappropriate antibiotics
- Supplementation of vitamin A, zinc and folic acid

DIARRHOEA IN ADULTS

1. **Acute**
   - Replacement of lost fluids and electrolytes
   - Pectin from cooked apples or other soluble fibre
   - Avoid caffeine as it enhances CAM level
   - Avoid sugar, alcohol, lactose, fructose and sucrose rich foods
   - Introduce solids as diarrhoea stops.
2. **Chronic**
   - Replacement of nutrients parenterally and enterally
   - Loss of K alters bowel motility, anorexia and bowel distress
   - Loss of iron in gastro intestinal bleeding causes anaemia
   - Nutrient deficiencies cause mucosal changes and enzyme secretion.

MALABSORBTION SYNDROME

STEATORRHOEA

Steatorrhoea is the presence of excess fat in feces. Stools may also float due to excess lipid, have an oily appearance and foul-smelling. An oily anal leakage or some level of fecal incontinence may occur. There is increased fat excretion, which can be measured by determining the fecal fat level.

Signs and symptoms

- Foul-smelling feces
- Bulky stool
- Loose stool
- Pale stool
- Greasy stool

LACTOSE INTOLERANCE

Lactose intolerance is the inability or insufficient ability to digest lactose, a sugar found in milk and milk products. Lactose intolerance is caused by a deficiency of the enzyme lactase, which is produced by the cells lining the small intestine. Lactase breaks down lactose into two simpler forms of sugar called glucose and galactose, which are then absorbed into the bloodstream as glucose.

Lactose intolerance is commonly seen among infants. Studies show that 50% of adult Asian population also show intolerance to lactose. In infants it is often due to a congenital error of metabolism due to the deficiency of the enzyme ?-galactosidase. Lack of lactase also leads to it.

Growth retardation is seen in children. Clinical features seen in others include –

- Flatulence
- Distention and bloating
- Frothy diarrhoea
- Stools with lactose and lactic acid

Retention of undigested disaccharides in the intestines increases the osmotic load of the intra-luminal contents and cause an excess quantity of fluid to be drawn into the intestine in order to dilute the sugar concentration. This leads to an increase in motility of the intestines and causes cramps, bloating and diarrhoea. The undigested sugars are fermented by the bacteria in the colon leading to the formation of lactic acid. The lactic acid is excreted in the stools and this irritates the bowels leading to diarrhoea. The fermentation results in excessive carbon dioxide production leading to bloating of the stomach.

CELIAC DISEASE

Coeliac disease, often called gluten-sensitive enteropathy or non-tropical sprue is caused by a reaction to gliadin in wheat protein fraction. The resulting damage to the villi of the intestinal mucosa results in potential or actual malabsorption of virtually all nutrients.

> Sprue or Coeliac disease is inflammatory disease of the small intestine caused by ingestion of wheat in individuals predisposed.

The most common symptoms in children between 6 months to 3 years of age are:

- Diarrhoea
- Growth failure
- Vomiting
- Bloated abdomen and
- Stools that are abnormal in appearance, odour and quantity.

Adults may experience weight loss despite increased appetite, weakness and fatigue. They may also suffer from anaemia and osteopenic bone disease.

In gliadin-free diet, wheat, oat, rye and barley are excluded. Products made from corn, potato, rice, soybean, tapioca, arrowroot, amaranth seeds and millets can be included in the diet. Medium chain triglycerides may help provide calories and a vehicle for fat soluble nutrients.

TROPICAL SPRUE

It is a rare digestive disease where the small intestine cannot absorb nutrients properly. It may be the sequela of an acute infectious diarrhoea, with subsequent contamination of the bowel by bacteria. The intestinal villi are shortened. The gastric mucosa may be atrophied and inflamed with diminished secretion of hydrochloric acid and intrinsic factor.

Symptoms include diarrhoea, anorexia and abdominal distention as well as symptoms of nutritional deficiency.

Treatment involves restoration of fluids, electrolytes and nutrients. Tropical sprue often responds promptly to antibiotics and folate therapy. Along with other nutrients as needed, folate is given orally, 5 mg/ day, along with intramuscular vitamin B12 (1000 μg/month).

INFLAMATORY BOWL DISEASE

The term inflammatory bowel disease is applied to three conditions which have similar symptoms but are clinically different.

- Ulcerative colitis
- Crhon's disease
- Short bowel syndrome.

In the first two conditions there are mucosal tissue lesions, in the third there may be repeated removal of the affected sections of the intestine - all three leading to typical, similar symptoms.

ETIOLOGY

Ulcerative colitis is a disease that causes inflammation and sores, called ulcers, in the lining of the rectum and colon. Ulcers form wherever inflammation has killed the cells that usually line the colon, then bleed and produce pus. Inflammation in the colon also causes the colon to empty frequently, causing diarrhoea.

Crhon's disease, also known as regional enteritis, is an inflammatory disease of the intestines that may affect any part of the gastrointestinal tract from mouth to anus, causing a wide variety of symptoms. It primarily causes abdominal pain, diarrhoea (which may be bloody if inflammation is at its worst), vomiting, or weight loss, but may also cause complications outside the gastrointestinal tract such as skin rashes, arthritis, inflammation of the eye, tiredness, and lack of concentration.

Both ulcerative colitis and Crhon's disease have severe and devastating nutritional consequences. They may be distinguished by two differences –
Anatomic distribution – Crhon's disease can occur in any part of the gastro-intestinal tract – colon, small intestine, colorectal area.

Ulcerative colitis is confined to the colon and rectum.

Nature of tissue changes – In crhon's disease the inflammation is chronic and can involve any part of the intestinal wall. This often leads to strictures and fistula formation.

In ulcerative colitis, the inflammatory changes are acute and last for brief periods. The changes are also confined to the mucosal and submucosal tissue layers of the intestine.

Clinical Symptoms

The common clinical symptom is a chronic, bloody diarrhoea. Ulceration of the mucous membrane of the intestines leads to problems such as anorexia, edema, anemia, negative nitrogen balance, electrolyte disturbances etc. Weight loss is common.

MANAGEMENT

In the management of these two diseases, the emphasis is on restoring optimal nutrition. Antibacterial, anti-inflammatory medications and corticosteroids are administered along with supportive care. Nutrient deficiencies should be avoided by giving nutritional supplements. Local trauma to inflamed areas should be prevented. Medium chain triglycerides are well tolerated and may be given along with a protein, energy, mineral and vitamin rich diet, low in residue.

Intestinal gas and flatulence

Flatulence is the state of having excessive stomach or intestinal gas. This can result in uncomfortable feeling of bloating, as well as increased belching (burping) or passing of gas from the rectum. It is found to be common in all people.

Most people produce about 1-3 pints of gas per day.

Ulcerative colitis starts in the rectum and progress in a retrograde fashion.

Source: Mahan Kathleen L. and Sylvia Escott-stump, 2000, Krause's Food Nutrition and diet therapy, WB Saunders Company Philadelphia, USA.

Consumption of large amounts of dietary fibre (especially soluble fibre), resistant starch, and lactose in persons who are lactase deficient or modest amounts of alcohol sugars such as sorbitol may result in increased gas production in the colon and increased flatulence. Consumption of unusual amounts of fructose or even sucrose may also result in increased amounts of faecal substrate. Legumes produce flatus or gas due to the presence of stachyose and raffinose.

Diverticulitis

Small out pouching in the gastro-intestinal tract is called diverticula. Diverticula usually occur at weakened areas of the tissue resulting from such factors as tissue irritation or secretory or muscular malfunctioning. Diverticulosis, the condition where the diverticula are formed, is seen mostly in elderly persons with weakened muscular in the bowel wall. Diverticula can occur in any part of gastro intestinal tract but are generally seen in the colon and sometimes in the esophagus.

Constipation

INTRODUCTION

Constipation is defined as having a bowel movement fewer than three times per week.

- Stools in constipation are usually hard, dry, small in size, and difficult to eliminate.
- Painful bowel movement and often experiencing strain during defecation, bloating, and sensation of a full bowel
- Coated tongue, foul breath and lack of appetite
- Constipation is a condition resulting from insufficient frequency of defecation, deficient quantity of stool or production of abnormally hard and dry stools.

Certain factors which could lead to constipation are

- Low intake of water: As the food passes through the colon water is reabsorbed from it. If water intake is already low, this causes the stools to become hard and dry and difficult to evacuate.
- Excessive use of laxatives and certain other drugs can eventually lead to constipation.
- Loss of muscle tone of intestinal muscles, as in the elderly, can also lead to constipation.
- Lack of sufficient physical exercise could lead to constipation
- A diet low in fibre leads to constipation emphasizing the need for a high fibre diet. Fibre in the diet helps to absorb and hold water which makes the stools soft and helps in their easy passage. Fibre also decreases the transit time through the intestines and helps in easy defecation.

SYMPTOMS associated with constipation

- Dull headache
- Lassitude
- Anorexia
- Lower back pain
- Coated tongue
- Foul breath

CAUSES

Constipation occurs when the colon absorbs too much water from the digested residue or if the colon's muscle contractions are slow and sluggish, causing the stool to move through the colon too slowly. As a result, stools can become hard and dry.

1. Low fiber in the diet
2. Lack of physical activity (especially in the elderly)
3. Medications
    - Pain relieving medications (especially narcotics)
    - Antacids that contain aluminum and calcium

- Blood pressure medications (calcium channel blockers)
- Antiparkinson drugs
- Antispasmodics
- Antidepressants
- Iron supplements
- Diuretics
- Anticonvulsants

4. Milk
5. Irritable bowel syndrome
6. Changes in life style or routine such as pregnancy, aging, and travel
7. Abuse of laxatives
8. Ignoring the urge to have a bowel movement
9. Dehydration
10. Specific diseases or conditions, such as stroke (most common)
    - **Neurological disorders**
        - Multiple sclerosis
        - Parkinson's disease
        - Chronic idiopathic intestinal pseudo obstruction
        - Stroke
        - Spinal cord injuries
    - **Metabolic and endocrine conditions**
        - Diabetes
        - Uremia
        - Hypocalcaemia
        - Poor glycemic control
        - Hypothyroidism

Problems with the colon and rectum

Intestinal obstruction, scar tissue-also called as adhesions diverticulosis, tumors, colorectal stricture, or cancer can compress, squeeze, or narrow the intestine and rectum and cause constipation

CLASSIFICATION

Constipation refers to difficult or painful defecation with less than 3 motions per week.

The frequency of bowel movements varies among individuals and is related to their personal habits. Both daily elimination or evacuation once in two or three days may be normal. Constipation may lead to malaise, headache, coated tongue, foul breath and lack of appetite.

Spastic constipation

A spasm in the intestines causes food to move in an irregular manner. It is also very painful. The spasm may be caused by irritation of the intestinal mucosa through excessive use of –

- Alcohol
- Tea/coffee
- Spices.
- Bran
- Laxatives

High strung and nervous people are affected by this type of constipation. Dietary fibre plays an important role in the prevention of constipation.

Fibre has water absorption capacity which contributes to the bulk forming laxative effect and influences the transit time of the food mass through the digestive tract. Water held by fibre increase the volume of feces and softens the stools, causing the colon muscles to contract and propel the food residue quickly.

Dietary fibre plays an important role in the prevention and treatment of diverticular disease also.

Atonic constipation

Muscle tone of the intestinal wall is reduced so that peristaltic movements are impaired. As a result, food mass cannot move at a normal rate down the tract. Bacteria acts on the stagnated food, causing some of the symptoms of constipation. Moreover the longer the food stays in the intestines, the more the water absorbed. This make the stools dry and hard.

Atonic constipation occurs due to

- A diet poor in fibre
- Insufficient intake of fluids
- Lack of physical exercise
- Illness
- Pregnancy etc

COMPLICATIONS OF CONSTIPATION

Sometimes constipation can lead to complications.
These complications include hemorrhoids, caused by straining for bowel movement, or anal fissures, tears in the skin around the anus caused when hard stool stretches the sphincter muscle.

TREATMENT OF COMPLICATION

Although treatment depends on the cause, severity, and duration of the constipation, in most cases dietary and lifestyle changes will help to relieve symptoms and also help prevent them from recurring.

1. **Diet**
   - Eating a well-balanced, high-fiber diet that includes beans, bran, whole grains, fresh fruits, and vegetables
   - A diet with enough fiber (20 to 35 gms/day) helps the body form soft, bulky stool.
2. **Lifestyle changes**
   - Drinking sufficient water and other liquids
   - Engaging in daily exercise
   - Having enough time for bowel movement.
   - Not ignoring the bowel movement.
3. **Laxative**
   - Laxatives taken by mouth are available in liquid, tablet, gum powder, and granule forms. They work in various ways

ANEAMIA

INTRODUCTION

Iron deficiency anemia is the most prevalent micro nutrient deficiency in the world. It affects more than 2000 million people in both developing and developed countries. Young children and pregnant women are the most affected group with a global prevalence of 40% and 50% respectively. Indian studies show over 65% prevalence among preschool children (Seshadri, 1994). Anemia is a public health problem in India with up to 70% of pregnant women being anemic. Nutritional anemia is most commonly caused by a deficiency of iron.

CAUSES

Anemia is caused by several factors that often occur together. The principal causes are -

- Negative iron balance due to inadequate dietary intake.
- Low dietary bioavailability.
- Iron loss from the body

During periods of rapid growth as in infancy, early childhood, adolescence and pregnancy, the blood volume expands and extra iron is required to maintain the concentration of blood components like hemoglobin. When the requirements are not met, anemia could occur.

Foods which supply

Heme iron	Non heme iron
Liver	• **Green leafy vegetables**
Mutton	• **Rice flakes**
Fish	• **Gingelly seeds**
Crab	• **Soybean**
Prawns	• **Bengal gram roasted** • **Dates (dried)** • **Bajra**

The quality of iron supplied to the body – heme and non heme iron, the presence of iron absorption inhibitors and promoters – also affect the iron made available to the body.

Iron loss mainly occurs during menstruation, iron transfer to the fetus during pregnancy, blood loss during child birth, helminth infestation and malaria. It can also occur in accidental hemorrhages, and in bleeding ulcers and hemorrhoids.

Inhibitors and Enhancers of iron absorption

Inhibitors	Enhancers
• **Low gastric acidity**	**Increased acidity**
• **Phytates and oxalates**	**Ascorbic acid**
• **Polyphenols** • **Minerals**	**Presence of meatBody's need**
• **Infection**	

SCREENING OF IRON DEFICIENCY

Iron deficiency is the point at which iron stores are completely exhausted and any deficiency of functional iron is associated with liabilities.

*Serum Ferritin	Hemoglobin	Diagnosis
Normal	Normal	No iron deficiency
Low	Normal	Storage depletion
Low	Low	Iron deficiency anemia
Normal	Low	Other causes of anemia

*__Ferritin__ is the storage form of iron in the body.

Consequences of iron deficiency anemia

- **Pregnancy outcome** Anemia during pregnancy is associated with an increased risk to the fetus and the mother. The incidence of low birth weight, premature births and perinatal mortality is higher when the mother is even slightly anemic. Anemia is also a cause of maternal deaths. Changes in neurotransmitter levels of fetal brain are seen if mother is anemic.
- **Mental and motor development**
  Anemia, even mild anemia, affects the psychomotor and cognitive function of infants. Iron deficiency anemia at a critical period of brain growth may produce irreversible abnormalities. Lack of sufficient iron can affect attention, concentration and memory among school aged children.
- **Effect on growth**
  Anemic children have a poor growth status as indicated by their weight and height. During adolescence iron deficiency slows down the tempo of growth and the 'catch up' growth may not be optimum. Low iron stores in girls may contribute to a delayed age at menarche.

CLINICAL FEATURES

- The structure and function of epithelial cells is affected by iron deficiency anemia.
- The tongue, mouth, stomach and nails are affected. The skin becomes pale and the inside of lower eyelid is also pale Finger nails became thin and flat and koilonychia develops (spoon shaped nails). Achlorhydria is also seen.
- There is atrophy of the lingual papillae and the tongue look smooth and waxy. Angular stomatitis and dysphagia may be present.
- Lassitude, fatigue, breathlessness on exertion, palpitations, dizziness, headache, insomnia are some common features.

- Steady, severe and gnawing pain in upper abdomen.
- Nausea, vomiting, fever, tachycardia, abdominal distention

- Decreased levels of serum calcium, sodium and potassium.
- Hyper metabolic state.
- Serum amylase levels are used as a diagnostic test for pancreatitis. They are elevated within 24-28 hours after the onset of the disease.
- At a later stage diabetes, steatorrhoea and weight loss are observed.
- Hypotension
- Defect in the absorption of vitamin B12.

Hemoglobin cut off points used to define anemia

Group	Hemoglobin (g/dl)	Hematocrit
Children 6 months to 5 yrs	11.0	33
5 – 11 yrs	11.5	34
12-13 yrs	12.0	36
Non pregnant women	12.0	36
Pregnant women	11.0	33
Men	13	39
Source: WHO/UNICEF, 1998		

UNIT III

LIVER

INTRODUCTION

The liver is a vital organ, which, through its network of biochemical reactions controls the internal environment of the body. **The liver is located in the upper right hand side of the abdomen, mostly behind the rib cage and is the largest gland of the body and rightly known as the body's chemical workshop**. When the liver is diseased and does not function normally, various metabolic difficulties and clinical symptoms are observed. It is important to have a clear understanding of the normal metabolic functions of the liver before suggesting nutritional modifications in liver disease.

Different organs and diseases

To access and the gastro intestinal tract and diagnose the problems and diseases, various methods and techniques are used.

Three pathological changes may follow damage to the liver namely:

1. **Fatty infiltration:** It is the deposition of droplets of fat in the cells. This process is completely reversible, but if the damage is severe or long lasting it may be followed by necrosis or fibrosis.
2. **Necrosis** or death of the cells may be slight and only involve part of the lobule, e.g. ishchaemia due to heart failure causes central necrosis around the hepatic vein, or it may be massive and cause widespread destruction of the organs.
3. **Cirrhosis:** Fibrosis, the end result of any liver damage which leads to necrosis and also occurs in the absence of obvious necrosis gives rise to the condition cirrhosis.

Major Agents responsible for liver damage

1. Dietary deficiencies: Fatty changes seen in the liver in kwashiorkor may be attributed to a low protein intake and reduced capacity to secreate B-lipoproteins
2. Infective agents –Virus can cause infections and damage the liver
3. Toxic agents – Alcohol is known to have direct action on the lipid metabolism in the liver by enhancing fatty acid synthesis, decreasing fatty acid oxidation and producing specific stimulation to triglyceride formation.
4. Certain drugs and chemicals

CIRRHOSIS OF LIVER

Cirrhosis of the liver is a condition in which the liver slowly deteriorates and malfunctions characterised by destruction of liver cells, distortion of the normal lobular architecture with over growth of fibrous tissue and nodular regeneration of cells.This is advanced liver damage, characterized by dense scarring where the lobes are covered with fibrous tissue. It can occur in the following conditions:

- **Chronic alcohol abuse**
- **Unresolved viral hepatitis**

TYPES OF CIRRHOSIS

Three basic types of cirrhosis have been described.

1. Diffuse hepatic fibrosis (portal or Laennec's cirrhosis)
2. Post necrotic scarring which occurs in livers which have been the seat of massive necrosis
3. Biliary cirrhosis, which occurs as a result of biliary obstruction and superimposed infection

Cirrhosis is the end point of many types of liver damages.

- Necrosis
- Fatty infiltration
- Fibrosis and nodular regeneration

Cirrhosis takes few months to many years to develop

CAUSES OF CIRRHOSIS

Excessive alcohol consumption

- Deficiency of proteins, carbohydrates and vitamins
- Affects lipid metabolism by
- Fatty acid synthesis
- Fatty acid oxidation
- Stimulation to produce triglycerides
- Release of lipoproteins
- Uptake of circulatory lipids
- Risk of hepatitis B and C
- Malnutrition: Deficiency of Vitamin A, β-carotene, Vitamin E and C
- Iron overload (Heamochromatosis), Wilson's disease (disturbance in Cu metabolism)

- Chronic viral hepatitis B,C,D and G
- Chronic bile duct obstruction
- Aflatoxins

Presence of drugs and toxins like aflatoxin.

- Malnutrition
- Intestinal by pass
- Vascular disorders
- Metabolic disorders

Hemo chromatosis – where in excess of iron is deposited.

Wilsons disease – where in copper is present in free form due to the deficiency of ceruloplasmin.

CLINICAL SYMPTOMS

- Gastro intestinal disturbances like nausea, vomiting anorexia, distention and epigastric pain.
- Jaundice
- Edema, ascites, esophageal varices gastro intestinal bleeding, anemia – especially macrocytic anemia.
- Steatorrhoea
- Circulatory changes - reddening of the palms (palmar erythema)
- Endocrine changes - loss of libido and hair loss
- The liver may be shrunken or enlarged.
- Portal hypertension

Hepato cellular damage, necrosis of hepatocytes and fibrosis are characteristic of cirrhosis and it may lead to hepato cellular carcinoma.

COMPLICATIONS OF CIRRHOSIS

1. Build up of pressure in the veins that go to the liver (portal hypertension)
2. Collection of fluid in the abdominal cavity (ascites)
3. Alteration of brain function caused by toxins which are not removed by the liver (hepatic encephalopathy)
4. Bleeding from dilated veins in the oesophagus (oesophageal varices)
5. Infection in the ascitic fluid (spontaneous bacterial peritonitis)
6. Malnutrition and liver cancer.

Viral Hepatitis

Inflammation of the liver which occurs due to viral infection is known as viral hepatitis. It is a multisystem disease involving the lymph nodes, spleen, gastro intestinal tract, bone marrow and pancreas in addition to the liver.

The virus may be hepatitis A, B, C, D, E or G virus. Hepatitis may also be caused by excessive alcohol consumption, drugs or toxins. In viral hepatitis, the virus may be transmitted through contaminated food or water or through infected blood used for transfusions or contaminated needles and syringes.

- **Hepatitis A:** It is caused by a picornavirus and mainly transmitted by the fecal-oral route, often associated with ingestion of contaminated food. They do not produce chronic liver disease and get resolved within a few weeks.Hepatitis E also occurs through contaminated food and water. Antibodies are made by the immune system in the body that confer immunity against future infection. Adequate rest, remaining hydrated and abstinence to alcohol helps in speedy recovery.
- **Hepatitis B:** It is caused by a hepadnavirus. Hepatitis B infection occurs through contact with blood and blood products or other body fluids, unsterilized needles/syringes etc. contact with a person who has been tested positive for hepatitis B Surface Antigen (HBSAG) can also cause infection. This can progress to chronic hepatitis, cirrhosis and liver cancer.
- **Hepatitis C:** Type C hepatitis was previously referred to as "non-A, non-B hepatitis," because the causative virus had not been identified, but it was known to be neither hepatitis A nor hepatitis B. The hepatitis C virus (HCV) usually is spread by shared needles among drug abusers, blood transfusion, hemodialysis, and needle sticks and can also cross the placenta. Hepatitis C may lead to a chronic form of hepatitis culminating in cirrhosis.

The symptoms of hepatitis A, B and C are the same. If symptoms occur, they might include:

- Tiredness
- Loss of appetite
- Nausea
- Abdominal discomfort
- Dark urine
- Clay-colored bowel movements
- General symptoms include lassitude, abdominal pain, flatulence, anorexia, nausea, vomiting, diarrhea, headache, fever etc. Optimal nutrition and rest are essential for the injured liver cells to recover.

Common symptoms

- Jaundice: It is the yellowing of the skin and eyes (jaundice).Jaundice is a common symptom of liver disease where in the sclera, skin and mucous membranes become yellow. The blood bilirubin concentration increases to 40μmol/L.

- Jaundice can be classified as obstructive jaundice, haemolytic jaundice and Toxic Jaundice

DIAGNOSIS

Liver function test: For biochemical and clinical evaluation of the status of liver function, a special set of laboratory tests are conducted on blood and urine of the patient and are called liver function tests. These include the following.

In blood

- Total and conjugated serum bilirubin test (conjugated bilirubin glucuronide)
- Serum gluatamic transaminase test (SGOT)
- Serum glutamic pyruvic transaminase test(SGPT)
- Serum alkaline phosphatase test
- Serum total protein test
- Serum albumin test
- Serum globulin test
- Serum albumin-globulin ratio
- Serum cholesterol
- **Flocculation tests-** These tests are performed by adding serum to reagents such as thymol, zinc sulphate and cephalin –cholesterol. Albumin will retard the precipitation or turgidity when these reagents are added whereas globulin will tend to cause precipitation.
- **Carbohydrate tolerance test:** Glucose, galactose and glucagon tolerance tests are abnormal. In liver dysfunction, glycogen storage is impaired and the administration of glucagon leads to hypoglycaemia. Galactose tolerance tests are abnormal in liver disease since the ability to convert galactose to glucose is impaired.
- **BSP test (Bromosulphalein test):** The intravenous injection of sulfobromophthaline is given and then BSP concentration is measured in plasma. BSP is conjugated with glutathione and is excreted in a manner similar to that of bilirubin. An increase in the retention of BSP is indicative of liver dysfunction.
- **Impaired prothrombin time:** This test measures the clotting time. Increases prothrombin time may indicate liver damage.
- **Elevated enzymes:** Lactic dehydrogenase, Transaminase(glutamic-oxalacetic and glutamic –pyruvic), alkaline phosphatise, leucine amino peptidase, 5' nucleotidase, phopho-hexose isomerase.
- **In urine:** Bile salts, Bile pigment, urobilinogen

HEPATIC ENCEPHALOPATHY

A liver which is failing cannot carry out its functions of inactivating and detoxifying certain substances, nor can it metabolize other substances. The hepatic encephalopathy that results brings about changes in consciousness behavior and neurological status. Ammonia is formed predominantly in the gastro intestinal tract as a result of protein break down and gastro intestinal bleeding and preoduction by gut bacteria. The ammonia crosses the blood brain barrier to bring about changes.

Hepatic encephalopathy is a worsening of brain function that occurs when the liver is no longer able to remove toxic substances in the blood. Hepatic encephalopathy is caused by disorders that affect the liver. These include disorders that reduce liver function (such as cirrhosis or hepatitis) and conditions in which blood circulation does not enter the liver.

CLINICAL SYMPTOMS

- Disorders of consciousness
- Alterations in motor function.
- Apathy, confusion, drowsiness and coma.

HEPATIC COMA

Hepatic coma is a condition of severe end-stage liver dysfunction that is accompanied by unresponsiveness (coma). It is often a feature of hepatic encephalopathy. Hepatic coma occurs as a complication of viral hepatitis and cirrhosis.

Causes

Certain products which are absorbed and not metabolized or detoxified by the liver due to its malfunctioning, reach the brain and cause changes in the brain resulting in hepatic coma.

This occurs due to acute hepato cellular failure after viral hepatitis or alcoholic hepatitis. Other causes are –

- Fatty livers
- Drugs such as paracetamol and anti T.B drugs
- Poisoning
- Lepto spiral infection.

Clinical features and complications –

- Increase in circulating blood ammonia which crosses the blood – brain barrier and enters the brain.
- Mental changes
- Flapping tremors
- Odour of breath (fetid breath)
- Abnormal Electro encephalogram (EEG)

Hemorrhages occur due to the decreased synthesis of coagulation factors by the liver. Hypoglycemia occurs due to depleted glycogen stores and inability of the liver to produce glucose from amino acids.

DISEASE OF GALL BLADDER

Cholecystitis

Cholecystitis results from a low grade chronic infection leading to inflammation of the gall bladder. Changes in the gall bladder mucosa which affect its absorptive powers are brought about by the infection. Under normal circumstances, the cholesterol in bile, which is insoluble in water, is made soluble by the action of other substances like bile acids. During infection and the resulting mucosal changes, the solubility ratios of the bile ingredients are changed.

Cholecystitis is inflammation of the gallbladder, usually resulting from a gallstone blocking the cystic duct.
Cholelithias is the presence of gallstones in the gallbladder.

Types of Cholecystitis

Cholecystitis is classified as acute or chronic.

- **Acute Cholecystitis:** Acute cholecystitis begins suddenly, resulting in severe, steady pain in the upper abdomen. At least 95% of people with acute cholecystitis have gallstones. The inflammation almost always begins without infection, although infection may follow later. Inflammation may cause the gallbladder to fill with fluid and its walls to thicken.
- **Chronic Cholecystitis:** Chronic cholecystitis is gallbladder inflammation that has lasted a long time. It almost always results from gallstones. It is characterized by repeated attacks of pain (biliary colic). In chronic cholecystitis, the gallbladder is damaged by repeated attacks of acute inflammation, usually due to gallstones, and may become thick-walled, scarred, and small. The gallbladder usually contains sludge (microscopic particles of materials similar to those in gallstones), or gallstones that either block its opening into the cystic duct or reside in the cystic duct itself.

Cholelithiasis

Very frequently cholesterol precipitates causing gall stones of almost pure cholesterol. This condition is known as cholelithiasis. A high dietary fat intake over a long period also predisposes towards gall stone formation as more cholesterol is produced.

Cholelithiasis occurs when bile (a digestive fluid) has too much cholesterol or bilirubin, or when the bile is very concentrated due to underlying disease. Obesity as well as rapid weight loss are risk factors for cholelithiasis.

Pancreatitis is inflammation of the pancreas. The pancreas is a large gland behind the stomach and close to the duodenum—the first part of the small intestine. The pancreas secretes digestive juices, or enzymes, into the duodenum through a tube called the pancreatic duct. Pancreatic enzymes join with bile—a liquid produced in the liver and stored in the gallbladder—to digest food. The pancreas also releases the hormones insulin and glucagon into the bloodstream. These hormones help the body regulate the glucose it takes from food for energy.

DIABETES MELLITUS

INTRODUCTION

Diabetes Mellitus is a metabolic disorder which has become very common in recent years. Because of its high prevalence, India is being described as the ‘Diabetic Capital’. Its occurrence is closely linked to lifestyle and it is not as benign as people imagine it to be.

Under normal circumstances, food is consumed and digested to give glucose, amino acids and fatty acids which enter the blood stream. The pancreas to the hormone insulin and release it into the bloodstream. Glucose is carried to the target cells and enters the cell with the help of insulin which is then utilized by the cell to give it energy thus the levels of glucose in blood decreases. Glucose is carried to the target cells and enters the cell with the help of insulin. The glucose is then utilized by the cell to give it energy thus the levels of glucose in blood decrease.

Diabetes mellitus is a disorder in which blood sugar (glucose) levels are abnormally high because the body does not produce enough insulin to meet its needs.

MECHANISM

Increased anterior pituitary, adrenal cortex, thyroid and ? cells of islets of langerhans. Diabetes mellitus is associated with excessive “glycosuria”, diabetes insipidus in which the excessive urine without sugar, is caused by either kidney (nephrogenic DI) or pituitary gland (central DI) damage.

CLASSIFICATION

Type I diabetes mellitus (IDDM)

- Type I diabetes affects children or adults, was traditionally termed “Juvenile diabetes”

AETIOLOGY and comparative features of IDDM and NIDDM

Factor	IDDM	NIDDM
Age of onset	Juvenile	Maturity
Sex	M:F= 1:1	M>F (India)
Incidence		
Genetic locus	Chromosome 6 (HLA class II genes)	Chromosome 11 (mutation of glucokinase gene)
Environmental factors		
	Infections:Triggering of autoimmune	Life style: obesity

	reaction of islets due to coxsackie	
	Acute stress: Physical injury, surgery and emotional distress	Abdominal fat: high waist/hip ratio
	Diet: Wheat and milk proteins	Pregnancy: increased placental hormones
	Immunological factors: auto immune disease	Insulin resistance: due to abnormal insulin molecule

DIAGNOSIS

Several tests are used in the diagnosis of diabetes.

Glycosuria

- By glucose dip-sticks method(Diastix)
- Benedict's test

Table : Interpretation of Benedict's test

Colour	Approximate Sugar in		
	Report	Urine g%	Blood mg%
Green discolouration	0 to trace	-	< 200
Green precipitate	+	0.25	200 – 250
Greenish-yellow	++	0.5	250 – 300
Precipitate			
Yellowish-orange	+++	1.0	300 – 350
Precipitate			
Brick red	++++	>2.0	>350

precipitate			

Ketonuria: High amounts traced by nitro prusside reaction or by ace test tab or ketostix paper sticks.

Random blood sugar: If the fasting plasma glucose is greater than 140 mg/ dl or the random plasma glucose is greater than 200 mg/dl indicates diabetes.

Glucose tolerance test

- 75 g of glucose dissolved in 250-300 ml of water is given.
- After 2 hours of administration of glucose, blood and urine specimens are collected every 30 minutes.

National Diabetes Data Group (NDDG) in United States and the WHO expert committee on diabetes mellitus set down the criteria given in Table

Table : Blood glucose levels

	Fasting		**2 hrs after 75g. glucose (oral)**	
	Plasma mg%	**Whole blood mg%**	**Plasma mg%**	**Whole blood mg%**
Normal	<100	<80	<140	<120
Impaired glucose tolerance	100-140	80-120	140-200	120-180
Diabetes mellitus	>140	>120	>200	>180

WHO Technical Report Series No.727, 1985.

Impaired Glucose Tolerance

Renal threshold of diabetic patient is more than 180mg of glucose/dl.

TREATMENT

Clinical criteria

- Relief from symptoms
- Reduction in obesity
- Prevention of acute and chronic complications
- Presence of adequate energy for normal work performance.
- In childhood diabetes to promote normal development
- During pregnancy, to deliver the normal baby without complications

Bio-chemical criteria

- Urine and blood estimates for glucose levels
- Glycosylated Hb give the trend of glucose levels of the past 2-3 months
- Maintaining normal serum lipid profile

DIETARY MANAGEMENT

- Food exchange lists help in preventing both hypo and hyper glycemia.
- Diet is based on type of insulin intake

Table:Types of insulin and their action

Type	Onset hours	Peak Action hours	Duration hours
• Rapid action-short duration- Regular soluble crystalline semi lente	½ ½	2-4 2-4	6-8 10-12
• Intermediate action and duration lente NPH (neutral Protamine Hagedom)	2 2	6-12 6-12	18-24 18-24
• Delayed action- Prolonged duration- Protamine Zinc Insulin (PZI) Ultralente	4-8 4-8	14-20 16-18	24-36 18-24

- The action of insulin and the peak absorption of carbohydrate should be at the same time.

Insulin and meal distribution of calories

Type of insulin	Breakfast	Noon	Mid AN	Evening	Bed time
None	1/3	1/3		1/3	
Short acting	2/5	1/5		2/5	
Intermediate	1/7	2/7	1/7	2/7	1/7
Acting NPH Long acting	1/5	2/5		2/5	80-160 kcal

Long acting with regular insulin at BF	1/3	1/3		1/3	

- **Carbohydrate:** High carbohydrate and high fibre diet improves monocyte insulin receptor binding capacity, 60-65% total calories.
- **Proteins:** A diet high in protein (20% of k cal). Requirement for adults is 1gm/kg body weight and for children 1-1.5g/kg body weight
- **Fat:** 15-25% of total calories from PUFA

Vitamins and minerals:

- Needed to overcome oxidative stress and deficiency
- Vitamin C, vitamin E, magnesium and zinc are needed
- Normal requirement of calcium
- Vitamin D deficiency contributes to Impaired Glucose Tolerance
- Chromium supplementation reduces insulin dose

Dietary fibre: About 25-50g of dietary fibre and complex carbohydrate for type I and II diabetes.

- Decreases insulin requirements
- Increases peripheral tissue insulin sensitivity
- Decreases serum cholesterol and triglycerides
- Aids in weight control
- Decreases blood pressure.

Artificial Sweeteners: Sugar substitutes, non caloric and high intense sweetners. Ideal sweetener:

- Pleasant taste with no after taste
- Sweet as sucrose
- Colourless, odourless
- Readily soluble, stable
- Nontoxic, does not promote dental cavities
- Functional and economically feasible
- Without metabolic abnormalities

Low calorie sweeteners

- **Polyols:** 2.4 k cal/ g, synthesized from carbohydrate like starch, sucrose, glucose, invert sugar, xylose, sugar alcohols xylitol, sorbitol, mannitol, maltitol, lactitol etc 40-50g/day for adults and 30 g/day for children.

Non-calorie sweeteners

- **Cyclamate:** 30 times sweeter than sucrose, heat stable.
- **Acelsulfame-K:** Synthetic derivative of acetoacetic acid, non digestive, after taste, high concentration has metallic flavour, 200 times sweeter than 3% sucrose heat stable.
- **Alitame:** Di peptide based amide, 2000 times sweeter than sucrose.
- **Aspartame:** Made of aspartic acid and phenylalanine 180- 200 times sweeter than sucrose. Not heat stable.
- **Saccharin:** Sodium ortho benzene sulphonamide, stable, 300 times sweeter than sucrose.

- **Sucralose:** 600 times sweeter than sucrose easily soluble in water, stable
- **Oral hypoglycaemic drugs:** Sulphonylureas, biguanides

GLYCEMIC INDEX

Ranking of the foods as per post prandial blood glucose response compared to reference diet.

Factors which influence the glycemic index

- The presence of nonabsorbable oligosaccharides and viscous dietary fibre such as pectins, ?-glucans and gum fruits, vegetables and cereals reduce the efficiency of enzyme hydrolysis and slows down the rate at which glucose enters the blood stream.
- Starch is encased in its seed coat or coarsely ground is not efficiently hydrolysed to glucose because digestive enzymes are prevented from reaching the starch.
- Starch granules subjected to moist heat and subsequent cooling become dense and less available to enzyme action. Retrograded starch has low glycemic index.
- Rice bran, which is rich in fibre and oil has low glycemic index.
- High amylose rice varieties which are slowly digested are potentially useful in low glycemic diets.
- Chapathis, which need chewing have lower glycemic index compared to wheat 'kanjee'.
- Raw food has lower glycemic index than cooked food.
- Foods rich in fat and protein have low glycemic index.
- Phytic acid present in whole grains decrease glycemic index.
- Natural food has low glycemic index compared to processed foods.
- The glycemic index for fructose is low (23) compared to lactose (46), sucrose (65) or glucose (97). Fructose produces lower risk of blood sugar than glucose. In view of its effect on serum lipid, it can be taken only in moderation.

COMPLICATION

- **ACUTE COMPLICATION**
- **CHRONIC COMPLICATION**

ACUTE COMPLICATION

Diabetic ketoacidosis:

- Elevated levels of ketone bodies in the blood decrease the blood's pH, leading to diabetic keto acidosis.
- Ketoacidosis can easily become severe enough to cause hypertension, shock, and death
- More common in type I diabetes than type II.

Hyperglycemia hyperosmolar state

- This results in loss of water and an increase in blood osmolarity.
- If fluid is not replaced (by mouth or intravenous), the osmotic effect of high glucose levels, combined with the loss of water, will eventually lead to dehydration.

- Lethargy may ultimately progress to coma, though this is more common in type II diabetes than type I.

Hypoglycemia

- Hypoglycemia, is abnormally low blood glucose
- Severe hypoglycaemia may lead to loss of consciousness leading to coma, seizures, or even brain damage and death

CHRONIC COMPLICATION

Chronic elevation of blood glucose level leads to damage of blood vessels (angiopathy).
The damage to small blood vessels leads to a microangiopathy, which can cause one or more of the following:

- Diabetic retinopathy - growth of friable and poor-quality new blood vessels in the retina lead to severe vision loss.
- Diabetic neuropathy - abnormal and decreased sensation, starting with the feet but potentially in other nerves, later often fingers and hands. When combined with damaged blood vessels this can lead to diabetic foot.
- Diabetic nephropathy - damage to the kidney which can lead to chronic renal failure, eventually requiring dialysis. Diabetes mellitus is the most common cause of adult kidney failure worldwide in the developed world.
- Diabetic cardiomyopathy - damage to the heart, leading to diastolic dysfunction and eventually heart failure.

Macrovascular disease leads to cardiovascular disease, to which accelerated atherosclerosis is a contributor:

- Coronary artery disease, leading to angina or myocardial infarction ("heart attack")
- Stroke(mainly the ischemic type)

DIETARY GUIDELINE

- Energy intake is based on age, sex, actual weight in relation to desirable weight, activity and occupation
- For children
  Boys: basal requirements 1000kcal+125k cal x in no of years
  Boys: basal requirements 1000kcal+100k cal x no of years
- should maintain 10% of ideal body weight in
  Broka's index = Height in cm - 100 = Ideal body weight in kg
- Energy needs 20 k cal/kg- overweight,
  30 k cal/kg- Normal
  40 k cal/kg- underweight
- Distribution of calories for each meal if no insulin is given:

Breakfast & Midmorning	33%
Lunch &	33%

Tea	
Dinner & Before going to bed	33%

- Avoid simple sugars and junk foods, insulin based on need, calorie based on insulin
- Include low GI food
- Mixture of oils
- High protein and water soluble fibre-hypoglycemic
- Timely intake in between meals to avoid hypoglycemic stress
- Use food exchange lists
- Avoid fasting and feasting
- Include hypoglycemic foods like fenugreek
- Sodium greater than 6g/day and 3 g/ hypertensive diabetics
- Include whole wheat instead of rice- acarbose
- Diet should meet the needs of antioxidants, micronutrients and phytochemicals
- Supplement vitamins and minerals if needed.

- Starch encased in its seed coat or coarsely ground is not efficiently hydrolysed to glucose because digestive enzymes are prevented from reaching the starch
- Starch granules subjected to moist heat and subsequent cooling become dense and less available to enzyme action. Retrograded starch has low glycemic index.
- Rice bran, which is rich in fibre and oil has low glycemic index.
- High amylase rice varieties which are slowly digested are potentially useful in low glycaemic diets.
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- Natural food has low glycemic index compared to processed foods.
- The GI for fructose is low (23) compared to lactose (46), sucrose (65) or glucose (97). Fructose produces lower risk of blood sugar than glucose. In view of its effect on serum lipid, it can be taken only in moderation.

In general high-fibre foods with low glycemic indices (e.g., beans, vegetables, whole fruit and whole grains) are the preferred forms of carbohydrate in a defensive nutrition plan. Meal combination is also an important factor in managing blood glucose levels. Combining protein, fat and carbohydrate at meals and snacks i.e., small serving of nuts and a piece of fruit, can lead to better control of blood glucose levels and less insulin release than meals or snacks that consist mainly of carbohydrate like bread and jam.

Soluble dietary fibre combined with lower glycemic index food is good for diabetics. While planning diets glycemic load that is the amount of carbohydrate derived from the food is also considered.

Glycemic load is also important. Carrots have high glycemic index. The amount of carbohydrate is so low in a carrot that eating a carrot will have little effect on blood

glucose or insulin concentrations which has low glycemic load. Glycemic index is more important when the total carbohydrate content of the diet is high.

Foods to be avoided	Eaten in Moderation	Foods permitted
Simple sugars (glucose, honey, syrup, dried fruits, cake, candy, fried foods, alcohol, nuts, jaggery, sweetened juices).	Fats, cereals, pulses, meat, egg, nuts, roots, fruits, artificial sweetener.	Green leafy vegetables, fruits except banana, lemon, clear soups, onion, mint, spices, salads, plain coffee or tea, skimmed and butter milk, spices.

Myths and Facts

Myths	**Facts**
Diabetics can eat wheat but not rice Any amount of wheat can be consumed. Fasting can be compensated by the next meal. Feasting can be done by fasting the next meal. Diabetic diet is a special diet. Fruits can be eaten in unlimited quantities. Vegetables can be eaten in unlimited quantities. Liquids are easily digestible.	Both have similar glycemic index and raise the blood sugar to a similar extent. Many diabetics feel it convenient to count and limit the number of chapathis and chew count is more and satiety is better. Wheat is more nutritious as it is richer in protein, fibre, and B-vitamins. Large quantities of wheat increases blood sugar. Fasting can lead to hypoglycemia. It is dangerous particularly who are on oral medicines or insulin. This results in hyperglycemia. This is a normal diet eaten regularly in moderation avoiding certain foods. Citrus fruits and apples can be taken. Banana and mango are high

	in fructose when eaten in unlimited quantities cholesterol levels may increase. If eaten raw or with little fat or no coconut. Preparations like kanjee are not preferred as they have high GI. Chapathis/rotis are preferred.
Fruit juices/cola drinks can be taken. Bitter foods like fenugreek and bitter gourd are good. Sugar is to be totally avoided. Avoid carbohydrates and fats	It is better to avoid empty calories like cola drinks. It is better to eat the fruit as such as glycemic index and nutritive value better. Fenugreek seed (not leaves) to be taken 25g/day to have an impact. This may not apply to all other bitter foods. Sweets where concentrated sugar and fat are used like mysorepak are avoided totally. Permitted levels of 10g of sucrose can be taken. Artificial sweeteners can be added in coffee and tea like aspartame. Best is to appreciate bitterness in tea or coffee. Take good carbohydrates and good fats saturated fats trans-fatty acids unsaturated fatty acids complex carbohydrates

Constipation

Constipation is a condition resulting from insufficient frequency of defecation, deficient quantity of stool or production of abnormally hard and dry stools.

The frequency of passage of stools varies from individual to individual and from race to race. It is important to develop regular habits of defecation right from childhood.

However excessive parental concern over toilet training of the child is not good as children often try to seek attention by not passing stools!

Certain factors which could lead to constipation are

- Low intake of water: As the food passes through the colon water is reabsorbed from it. If water intake is already low, this causes the stools to become hard and dry and difficult to evacuate.
- Excessive use of laxatives and certain other drugs can eventually lead to constipation.
- Loss of muscle tone of intestinal muscles, as in the elderly, can also lead to constipation.
- Lack of sufficient physical exercise could lead to constipation
- A diet low in fibre leads to constipation emphasizing the need for a high fibre diet. Fibre in the diet helps to absorb and hold water which makes the stools soft and helps in their easy passage. Fibre also decreases the transit time through the intestines and helps in easy defecation.

SYMPTOMS associated with constipation

- Dull headache
- Lassitude
- Anorexia
- Lower back pain
- Coated tongue
- Foul breath

CLASSIFICATION

Constipation refers to difficult or painful defecation with less than 3 motions per week.

The frequency of bowel movements varies among individuals and is related to their personal habits. Both daily elimination or evacuation once in two or three days may be normal. Constipation may lead to malaise, headache, coated tongue, foul breath and lack of appetite.

Spastic constipation

A spasm in the intestines causes food to move in an irregular manner. It is also very painful. The spasm may be caused by irritation of the intestinal mucosa through excessive use of –

- Alcohol
- Tea/coffee
- Spices.
- Bran
- Laxatives

High strung and nervous people are affected by this type of constipation. Dietary fibre plays an important role in the prevention of constipation.

Fibre has water absorption capacity which contributes to the bulk forming laxative effect and influences the transit time of the food mass through the digestive tract. Water held by fibre increase the volume of feces and softens the stools, causing the colon muscles to contract and propel the food residue quickly.

Dietary fibre plays an important role in the prevention and treatment of diverticular disease also.

Atonic constipation

Muscle tone of the intestinal wall is reduced so that peristaltic movements are impaired. As a result, food mass cannot move at a normal rate down the tract. Bacteria acts on the stagnated food, causing some of the symptoms of constipation. Moreover the longer the food stays in the intestines, the more the water absorbed. This make the stools dry and hard.

Atonic constipation occurs due to

- A diet poor in fibre
- Insufficient intake of fluids
- Lack of physical exercise
- Illness
- Pregnancy etc

UNIT IV

CARDIOVASCULAR DISEASE

INTRODUCTION

Heart disease or cardiovascular diseases is the class of diseases that involve the heart or blood vessels (arteries and veins). While the term technically refers to any disease that affects the cardiovascular system, it is usually used to refer to those related to atherosclerosis (arterial disease). These conditions have similar causes, mechanisms, and treatments.

Cardiovascular disease (CVD) is a major cause of morbidity and mortality in the western world. There are a number of well-established risk factors for CVD including smoking, hypertension and family history. In terms of nutrition, a diet high in fat, particularly saturated fat, has been shown to be associated with CVD incidence. The observation that Greenland Eskimos have a low incidence of CVD despite a high saturated fat intake has led to much scientific and public interest in the role of n-3 fatty acids found in fish and fish oils in the prevention and treatment of disease, and particularly CVD. Heart disease affects people of all ages, but is most frequent in middle age and is most often caused by atherosclerosis.

No single factor is an absolute cause either of atherosclerosis or of coronary disease. Many factors are interrelated and to the extent that they are present they increase the risk of the disease. Major risk factors are elevated serum cholesterol, hypertension, cigarette smoking, Diabetes mellitus, and marked obesity. Other risk factors include a family history of early heart disease other lipid abnormalities, lack of physical activity and stress.

To treat coronary artery disease (CAD) it is important to first recognize it. The prediction, diagnosis and treatment of CAD is based on the risk factors which help in identifying the individuals at risk. These risk factors are either modifiable or non modifiable.

Table: Modifiable and non modifiable risk factors

Life style	**Biochemical/physiological characteristic (modifiable)**	**Personal characteristics (non modifiable)**
Diet high in saturated fat, cholesterol & energy	Elevated Blood pressure	Age
Tobacco	Elevated plasma total	Sex

smoking	cholesterol, LDL cholesterol	
Excess consumption of alcohol		Family history of CHD at an early age (men <55years, women<65 years)
Physical activity	Low plasma, HDL cholesterol Elevated plasma triglycerides. Hyperglycemia/ diabetes Obesity Thrombogenic factors	Personal history of CHD or other atherosclerotic vascular disease.

Diseases of heart can be caused to

- Pericardium
- Myocardium
- Endocardium
- Blood vessels within the heart, those leaving the heart or the heart valves

CAUSES

- Coronary artery disease
- Angina pectoris: pain in the chest provoked by exercise
- Hypertension
- Peripheral artery disease
- Rheumatic heart disease
- Congenital heart disease
  - Chronic inability of the heart to maintain an adequate output of the blood from the ventricles leads to congestion or over distension of veins
- Atherosclerosis
  - Thickening of walls of small arteries due to ageing or hypertension

RISK FACTORS

Risk for cardiovascular disease

- Cigarette smoking
- Hypertension
- High serum total cholesterol & LDL
- Low HDL
- Diabetes mellitus
- Obesity
- Physical inactivity
- Family history of coronary heart disease
- Hyper triglyceridemia.
- Small, dense LDL particles

- Increased lipoprotein(a)
- Increased serum homocysteine
- Abnormalities in several clotting factors
- High levels of serum reactive protein

It has been shown that there is an association between atherosclerosis and various nutrients to varying degrees. There is a definite association between types of dietary fat and elevated blood lipid levels. Controlling blood cholesterol level is a major means of controlling heart disease. While elevated cholesterol levels are major contributor to heart disease, dietary saturated fat contributes to the cholesterol level.

LIPOPROTEINS

Since lipoproteins are the major transport forms of lipids in the blood, an increase in one or more of these plasma lipoproteins creates a condition called hyper lipoproteinemia which is a risk factor for heart disease. Lipoproteins are formed in the intestinal wall after the ingestion of a meal. They have a high triglyceride content and are cleared from the blood by lipoprotein lipase. The liver also synthesizes lipoproteins from endogenous fat sources.

The various lipo proteins are:

- **Chylomicrons –** produced from exogenous fat and consisting of a large percentage of triglycerides.
- **Very low density lipoproteins (VLDL),** formed from endogenous fats in the liver also contain more of lipids and less of proteins.
- **Intermediate density lipoproteins (IDL)** deliver endogenous triglycerides to the cells for their use.
- **Low Density Lipoprotein (LDL)** carry two thirds of the plasma cholesterol. Since LDL carries more cholesterol to the cell, it is the main agent of concern.
- **High Density Lipoprotein (HDL)** carries less cholesterol and more protein and is also formed in the liver from endogenous fat sources. It carries cholesterol from the tissues to the liver for breaking down and excretion and therefore higher levels of HDL are considered cardio protective.

Apo proteins

Apo proteins refer to the protein part of a combined protein. Apo proteins are mostly synthesized by the liver and few by intestinal mucosa. Defects in the synthesis of these Apo proteins can lead to a disturbance in the serum lipoprotein level. Most of these changes are familial and rare. For example in a deficiency of apoprotein A, HDL cannot be formed and cholesterol accumulates in the tissues. Similarly Apo lipoprotein E deficiency is characterized by low LDL concentration and non formation of chylomicrons. The cholesterol rich remnants of chylomicrons accumulate in the blood and cause lipid deposits in the skin called xanthomas.

ROLE OF FAT IN DEVELOPMENT OF ATHEROSCLEROSIS

- Main forms of fat are cholesterol and triglycerides.
- Fat is transported to the parts of body with the help of lipoproteins
- Liver places cholesterol along with triglycerides in the lipoprotein packages

There are five stages to atherosclerosis

- **Phase I:** Asymptomatic phase which consists of fatty streaks which are non-obstructive lipid filled cells
- **Phase II:** Consists of plaque with high lipid content and prone to rupture with main lipid being LDL
- **Phase III:** Acute complicated phase with rupture of non-occlusive thrombus
- **Phase IV:** Acute complicated lesions with occlusive thrombus which are associated with angina/myocardial infarction.
- **Phase V:** Fibrotic or occlusive lesions.

Angina pectoris: Chest discomfort if often reported by most patients especially those which are chronic cases of dyslipidemia and/or hypertension

DEVELOPMENT OF ATHEROMA

The sequence of development of atheroma is given in the schematic diagram. Hyperlipidaemia and excess of modified lipoprotein fractions/peroxides/free radicals

All the three layers – intima, media and adventia of the arteries are damaged in atherosclerosis.

Atherosclerosis causative fatty acids		
	High intake of saturated fatty acid can lead to	• Increased plasma --- Increased cholesterol atheroma formation. • Increased LDL receptors --- Increased LDL cholesterol. • Arrhythmia --- Thrombosis. • Increased Lipid oxidation --- - Injury to coronary arteries. • Increased factor VII level --- - Thrombosis. • Decreased antiaggregatory prostacyclin. • Decreased HDL cholesterol. • Plasma triglycerides.
	High cholesterol intake can lead to	• Plasma cholesterol accumulation • LDL synthesis • LDL catabolism by cells.

Low ? – 3 fatty acids intake	• Arrhythmia ----- Thrombosis • Increased Fibrinogen ---- Thrombosis ---- Fibrous plaque • Increased platelet aggregation --- Thrombosis. • Increased lipoprotein (a) - Fibrous plaque • Increased inflammation – injury to coronary arteries. • B.P --- Injury to coronary arteries.

Table: Desirable cholesterol levels

Desirable cholesterol levels	mg/dl
Total cholesterol	< 200
HDL cholesterol	> 40
LDL cholesterol	< 150
VLDL cholesterol	< 40

CLINICAL EFFECTS

Manifested in medium sized muscular arteries like

- Coronary artery
- Carotid artery
- Cerebral arteries
- Iliac and superficial femoral arteries

Atherosclerotic lesions due to partial or total occlusions of the lumen
Formation of thrombus because of cracks and fissures
Angina pectoris
Myocardial infarction (death of heart muscle tissue)

CONGESTIVE HEART FAILURE

1. Severe dilation of heart leads to impairement of circulatory system leading to failure of kidney
2. Sodium and water is held in tissues causing odema in extremities, abdominal and chest cavities Ischemic death (Deficient blood supply)

RISK FACTORS

Category I

Risk factors for which intervention have been **proven** to lower CVD risk are

- Cigarette smoking
- LDL cholesterol
- High fat/ cholesterol diet
- Hypertension
- Left ventricular hypertrophy
- Thrombogenic factor

Category II

Risk factors for which interventions are **likely** to lower CVD risk

- Diabetes mellitus
- Physical inactivity
- HDL cholesterol
- Triglycerides, small dense LDL
- Obesity
- Postmenopausal status

Category III

Risk factors associated with increased CVD risk may reduce if the following are modified

- Physiological factors
- Lipoprotein A
- Homoecysteine
- Oxidative stress
- No alcohol consumption

Category IV

Risk factors associated with increased CVD risk which **cannot** be modified

- Age
- Male gender
- Family history of early onset of CVD

ROLE OF NUTRIENTS PREVENTING AND PROMOTING

Nutrients apart from fat also play an important role in preventing or promoting atherosclerosis by changing blood levels of cholesterol, HDL and triglycerides. These include:

- Calcium in large amounts lower the cholesterol and triglyceride levels.
- Carbohydrates, especially simple sugars in large quantities tend to cause excessive production of triglycerides and increase the risk in susceptible individuals of forming atherosclerotic plaque.
- Chromium may have a protective role against plaque formation. When chromium is supplemented HDL levels increase and total cholesterol levels drop.

- Copper deficiency has been shown to increase serum cholesterol levels in rats. Although the evidence is in-conclusive, copper may protect against plaque formation.
- Soluble dietary fibre has been documented to reduce cholesterol levels.
- Severe deficiency of iron increase blood lipid levels but moderate anemia lowers blood lipid levels.
- Experiments showed that zinc in large quantities reduce HDL levels. Evidence suggests that large amounts of zinc increase the risk of the disease.
- Omega 3 fatty acids, Eicosapentanoic acid (EPA) and docosahexaenoic acid (DHA) have been found to reduce platelet aggregation and thus reduce thrombus formation. They also decrease the formation of LDL and have anti inflammatory effects.

Thus it is seen that many nutrients and not just fat affect the formation of atherosclerotic plaque and the diet as a whole should be considered to control or prevent atherosclerosis.

Myocardial infarction or heart attack occurs when the blood supply to the heart is cut off. The portion of the heart muscle serviced by the blocked artery is deprived of oxygen and nutrients and slowly dies. The area of necrosis in the heart muscle is called a myocardial infarct. The functioning of the heart muscle is reduced to this extent. Thus if a large portion of the heart becomes dysfunctional, death results. Acute damage to the heart muscle due to myocardial infarction leads to an increase in levels of certain enzymes and an enzyme assay helps in diagnosing myocardial infarction. The enzymes from the damaged heart muscle escape into the intestitium, some of them are denatured but others reach the circulation.

DIETARY MANAGEMENT

Low calorie, low fat (High PUFA with n-6 to n-3 ratio of 4-10:1), low carbohydrate, high fibre, normal protein, vitamins and minerals.

1. **Total energy**
   - Total energy is restricted to maintain the normal weight
   - Mild degree of weight loss for the cardiac patient of normal weight
   - For the obese: 1000-1200 Kcal/day
2. **Fat**
   - 20% of total calories
   - The proportion of SFA:MUFA:PUFA - 5:6:4
   - High intake of PUFA helps in
     - Increasing esterification of cholesterol
     - Decrease in thrombosis
     - Decreased accumulation of cholesterol
     - Decreased synthesis of VLDL cholesterol
     - Decreased production of LDL and triglycerides
   - High intake of MUFA is
     - Conducive to thrombolysis
     - Anti-inflammatory
3. **Cholesterol:** Cholesterol of < 300 mg in diet (cholesterol level in blood> 260 mg/dl is dangerous to health)
4. **Carbohydrates:** Intake is reduced as total calories are reduced
5. **Proteins, vitamins and minerals:** Normal
6. **Sodium:** restriction if hypertension is present and 1600-2300mg for CVD
   Low glycemic foods should be used
   Inclusion of functional foods i.e. vitamin-E and C, carotenoids, selenium and soya proteins
   Allyl sulphur compounds of garlic is antithrombolic in nature

HYPERTENSION

DEFINITION

Hypertension or **high blood pressure** is a condition in which the blood pressure in the arteries is chronically elevated. With every heart beat, the heart pumps blood through the arteries to the rest of the body. Blood pressure is the force of blood that is pushing up against the walls of the blood vessels. If the pressure is too high, the heart has to work harder to pump, and this could lead to organ damage and several illnesses such as heart attack, stroke, heart failure, aneurysm or renal failure.

WHO defines hypertension is a condition in which systolic pressure exceeds 160 mm Hg and diastolic pressure exceeds 95 mm Hg. With diastolic pressures of 100 or more therapy should be initiated with drugs as well as diet.

The systolic blood pressure, corresponds to the pressure in the arteries as the heart contracts and pumps blood forward into the arteries. The diastolic pressure represents the pressure in the arteries as the heart relaxes after the contraction. The diastolic pressure reflects the lowest pressure to which the arteries are exposed.

Hypertension is elevated blood pressure indicating the symptom of underlying progress of disease

Systolic pressure > 160mm Hg
Diastolic pressure> 95mm Hg

Hypertension impairs pumping action of heart leading to damage to heart, brain and kidneys.
Hypertension is increased cardiac output and increased total peripheral resistance

High Blood Pressure Overview

The heart pumps blood into the arteries with enough force to push blood to the far reaches of each organ from the top of the head to the bottom of the feet. Blood pressure can be defined as the pressure of blood on the walls of the arteries as it circulates through the body. Blood pressure is highest as its leaves the heart through the aorta and gradually decreases as it enters smaller and smaller blood vessels (arteries, arterioles, and capillaries). Blood returns in the veins leading to the heart, aided by gravity and muscle contraction

CAUSES

- Diseases of CVD, pituitary gland and ovaries
- Renal-glomerulonephritis, poly nephritis, polycystic renal disease
- Tumors of brain or adrenal gland
- Hyperthyroidism
- Predisposing factors such as heredity, stress, obesity, smoking and high viscocity of blood

TYPES

When the cause of hypertension is unknown, it is called essential hypertension.

1. **Mild hypertension** Diastolic pressure is 90 to 104 mm Hg. Treatment is based on weight loss, sodium restriction and behavioural techniques.
2. **Moderate hypertension** Diastolic pressure is 105 to 119 mm Hg in moderate hypertension. Nutritional therapy is supported by drugs such as blockers.
3. **Severe hypertension** Diastolic pressure is 120 to 130 mm Hg and above. Apart from giving treatment for moderate hypertension peripheral vasodilators are given. Diet therapy revolves around potassium replacement in the use of drugs and nutritional support for weight management and sodium modification.

SYMPTOMS

Headache, dizziness, impaired vision, failing memory, shortness of breath, pain over heart, GI disturbance

DIETARY MANAGEMENT

Earlier Kempner's rice fruit diet was suggested for hypertension. This diet is very restrictive, deficient in many nutrients.

1. **Energy** An obese patient must be reduced to normal body weight with low calorie diet. About 20 k cal/kg of ideal body weight are prescribed for a sedentary worker and 25 k cal/kg of body weight for moderately active worker. Alcohol consumption should be reduced.
2. **Protein** A diet of 60g protein is necessary to maintain proper nutrition. In severe hypertension, protein restriction to 20g as advocated by Kempner may be necessary as a temporary measure since protein foods are rich in sodium.
3. **Fats** As they are prone to atherosclerosis, it is advisable to avoid a high intake of animal or hydrogenated fat. About 20g vegetable oil is permitted.
4. **Carbohydrate** Easily available carbohydrate is of great help in the management of high blood pressure.
5. **Sodium** Increased intake of sodium in diet leads to increased intravascular volume and thus increases cardiac output, elevating blood pressure. And at the cellular level, increased intracellular sodium is exchanged for increased intracellular calcium with its potent effects of augmented vascular tone and vascular hypertrophy with resulting persistent hypertension. Restricted sodium and a decrease in the sodium/potassium ratio in the diet is preferred. Moderate sodium restriction 2-3 g/day reduces diastolic pressure 6-10 mm of Hg and enhances the blood pressure lowering effect of diuretic therapy. American Heart Association prescribed 2g for mild and 1g sodium for moderate levels. Recent studies have shown that sodium restriction accompanied by weight reduction can effectively control mild or moderate arterial blood pressure. Pregnant women with toxemia need a moderate rather than low salt intake. Restrictions for a moderate low sodium diet of 1000 mg.
6. **Potassium's** role in hypertension is actually the result of a complex interplay with sodium, calcium and magnesium found in all living cells and in blood. For example, low levels of potassium cause the body to retain sodium and water and this can elevate blood pressure. Research suggests that the risk of stroke, a common consequence of high blood pressure, relates inversely with the amount of potassium in the diet and the lowest risk is among the high potassium low sodium group. Daily requirement of potassium is 3500 mg/day. Fruits and vegetables should be taken liberally to meet potassium requirement.

RENAL DISEASE

INTRODUCTION

The kidneys are glands that are located in the abdominal region just above the pelvis on either side of the body. When functioning normally, the kidneys separate and filter excess water and waste from the blood stream. The kidneys are responsible for producing urine, which is used to flush away the toxins. The kidneys also maintain a healthy balance of fluids and electrolytes, or salt compounds, in the body.

The basic filtering unit of the kidney is called the nephron. Every kidney has about a million **nephrons.** In the nephron, tiny blood vessels called capillaries intertwine with tiny urine-carrying tubules. By filtration, the small ions and molecules from the blood are removed. The valuable components among these are re absorbed. The excess is sent out with the urine. Each day, the kidneys pump about 200 litres of blood through 140 miles of tubes and millions of filters.

FUNCTIONS OF KIDNEY

Kidney act like a 24-hour cleaning crew for the blood.

- They filter out waste products. They get rid of excess water. They balance electrolytes in blood such as potassium and sodium. They remove excess acid.
- They regulate blood pressure.
- They also produce a hormone erythropoietin to help the bone marrow make red blood cells.
- They maintain Calcium levels in the body.
- The kidneys also function as a part of the endocrine system producing erythropoietin and calcitriol. Erythropoietin is involved in the production of red blood cells and calcitriol plays a role in bone formation

NEPHRITIS

Nephritis is inflammation of the kidneys. It can be either acute or chronic and is a serious condition. It may be caused by a bacterial infection of the kidneys (pyelonephritis) or exposure to a toxin. However, it more commonly develops from an abnormal immune reaction, which can occur in two ways:

1. An antibody can attack either the kidney itself or a substance that stimulates an immune reaction (antigen) attached to kidney cells, or
2. An antigen and antibody can combine somewhere else in the body, forming an immune complex, and then attach to cells in the kidney.

Some types of nephritis involve infiltration of kidney tissues by white blood cells and deposits of antibodies. In other types of nephritis, inflammation may consist of tissue swelling or scarring without white blood cells or antibodies. Nephritis can occur anywhere in the kidneys. It can cause death if not treated in initial stages. It often strikes during childhood or adolescence.

In rare cases nephritis can be genetically inherited, though it may not present in childhood.

Nephritis is a serious medical condition which is the ninth highest cause of human death. As the kidneys inflame, they begin to excrete needed protein from the body into the urine stream. This condition is called proteinuria. Loss of necessary protein due to nephritis can result in several life-threatening symptoms. Most dangerous in cases of nephritis is the loss of protein that keeps blood from clotting. This can result in blood clots causing sudden stroke.

SYMPTOMS

- Pain in the kidney that extends till the urethra.
- Highly colored scanty urine.
- Dull pain in the back and fever.

What are the symptoms of Nephritis

Less commonly, nephritis involves the tubules and the tissues that surround them (tubulointerstitial tissues). Such inflammation is called tubulointerstitial nephritis. A kidney tubule is a microscopic tube that carries fluid and substances filtered from the blood in the glomerulus to the duct that drains urine into the pelvis of the kidney. Tubulointerstitial tissues surround each of the tubules and separate one tubule from another.

When inflammation damages the tubules and the tubulointerstitial tissues, the kidneys may become unable to concentrate urine, eliminate (excrete) metabolic waste products from the body, or balance the excretion of sodium and other electrolytes, such as potassium. When the tubules and tubulointerstitial tissues are damaged, kidney failure often develops.

CAUSES

- Infection of the throat.
- **Rheumatic fever or scarlet fever** is usually followed by nephritis.
- Wrong dietary habits, excessive drinking, and habitual use of chemical agents. Frequent use of painkillers and suppressive medical treatment are the main causes.
- **Nutritional deficiencies** also contribute.

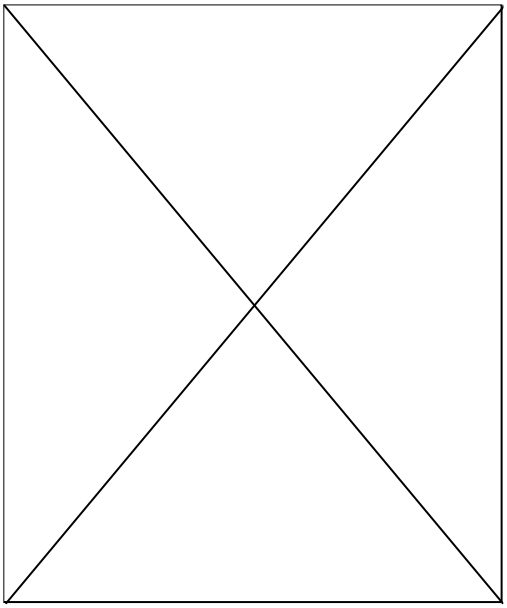
URINARY CALCULI

Urinary calculi are solid particles in the urinary system. They may cause pain, nausea, vomiting, hematuria, and, possibly, chills and fever from secondary infection.

Nephrolithiasis: refers to the condition of having kidney stones.
Urolithiasis: refers to the condition of having calculi in the urinary tract
Ureterolithiasis is the condition of having a calculus in the ureter
Bladder stones usually applies to urolithiasis of the bladder in non-human animals such as dogs and cats.

Kidney stones do not have single, well-defined cause, but are the result of a combination of factors. A stone is created when the urine does not have the correct balance of fluid and a combination of minerals and acids. When the urine contains more crystal-forming substances than the fluid can dilute, crystals can form. Normally the urine contains components that prevent these crystals from attaching to each other. However, when these substances fall below their normal proportions, stones can form out of an accumulation of crystals.

Table Composition of Urinary Calculi.



Composition of Urinary Calculi		
Composition	**Percentage of All Calculi**	**Common Causes**
Calcium oxalate	70	Hypercalciuria Hyperparathyroidism Hypocitruria Renal tubular acidosis
Calcium phosphate	15	Hypercalciuria Hyperparathyroidism Hypocitruria Renal tubular acidosis

Cystine **Magnesium ammonium phosphate (struvite)**	2 3	Cystinuria UTI caused by urea-splitting bacteria
Uric acid	10	Hyperuricosuria Increased urine acidity

RENAL FAILURE

INTRODUCTION

Renal failure is described as a decrease in the glomerular filtration rate. Biochemically, renal failure is typically detected by an elevated serum creatinine level. Problems frequently encountered in kidney malfunction include abnormal fluid levels in the body, deranged acid levels, abnormal levels of potassium, calcium, phosphate, and (in the longer term) anemia. Depending on the cause, hematuria (blood loss in the urine) and proteinuria (protein loss in the urine) may occur. Long-term kidney problems have significant repercussions on other diseases, such as cardiovascular disease.

In renal failure the kidneys undergo cellular death and are unable to filter wastes, produce urine and maintain fluid balances. This dysfunction causes a buildup of toxins in the body which can affect the blood, brain and heart, as well as other complications. Renal failure is very serious and even deadly if left untreated.

TYPES

There are two types of renal failure: acute and chronic. Acute renal failure occurs suddenly and is usually initiated by underlying causes, for example dehydration, infection, serious injury to the kidney . Acute renal failure is often reversible with no lasting damage.

Acute renal failure can occur for any number of different reasons, but the three most common explanations are blood flow problems, toxic damage caused by medicines, infections and poisons and kidney blockage. Blood circulation problems can manifest due to blood loss or dehydration. A sepsis infection, or an accumulation of toxins or bacteria, can negatively impact the kidneys as well. Some antibiotics, pain medications, blood pressure medicines and X-ray dyes have also been shown to harm the kidneys, though this is more common in people who already suffer from

related, common health problems. In addition, kidney stones, tumors, enlarged prostate glands and injury can damage the kidneys and result in acute renal failure.

Chronic renal failure is more serious than acute renal failure because symptoms may not appear until the kidneys are extremely damaged. Chronic renal failure can be caused by other long term diseases, such as diabetes and high blood pressure. Chronic renal failure can worsen over time, especially when the problem has gone undiagnosed and treatment is delayed.

DIALYSIS

Dialysis is a type of renal replacement therapy which is used to provide an artificial replacement for lost kidney function due to renal failure. It is a life support treatment and does not treat any kidney diseases. Dialysis may be used for very sick patients who have suddenly lost their kidney function (acute renal failure) or for quite stable patients who have permanently lost their kidney function (end stage renal failure).

Dialysis is an imperfect treatment to replace kidney function because it does not correct the endocrine functions of the kidney. Dialysis treatments replace some of these functions through diffusion (waste removal) and ultra filtration (fluid removal).

- **Principle**

  Types of dialysis

  Renal dialysis falls into two main divisions

- **Haemodialysis**
- **Peritoneal dialysis**

PRINCIPLES OF DIALYSIS

Dialysis works on the principles of the diffusion of solutes and ultra filtration of fluid across a semi-permeable membrane. Diffusion describes a property of substances in water. Substances in water tend to move from an area of high concentration to an area of low concentration. Blood flows by one side of a semi-permeable membrane, and a dialysate, or special dialysis fluid, flows by the opposite side. A semi permeable membrane is a thin layer of material that contains various sized holes, or pores. Smaller solutes and fluid pass through the membrane, but the membrane blocks the passage of larger substances (for example, red blood cells, large proteins).

HEAMODIALYSIS

Haemodialysis is a replacement therapy; it is a method for removing waste products from blood when the patient's kidneys suffer from irrevocable renal failure. This procedure is accomplished with a mechanical device sometimes called an artificial

kidney, which is connected to the patient with an extracorporeal blood circulation device of lines and filter.

A diffusion process allows the exchange of particles between two compartments, one for blood and the other for dialytic fluid. These are separated by a semi permeable membrane, which is assembled within the filter. In this way, contaminants go from blood flux to dialytic fluid and are removed.

PERITONEAL DIALYSIS

Peritoneal dialysis makes use of peritoneal membrane, a lining of the abdomen, to store dialysis fluid temporarily, so that dialysis takes place within the body. This means that patients manage their own care to a greater extent than haemodialysis patients, who have to attend hospitals or clinics very frequently.

Dialysis takes about 1-2 hours each day, rather than about 4 hours several times per week. In this case however the patient has an operation in which a catheter is inserted into the abdomen and dialysis fluid is then administered through this.

The peritoneal membrane takes the place of the artificial semi-permeable membrane in kidney machines, allowing excess water and waste products to pass into the dialysis fluid, which is then trained away.

Both forms of dialysis have their benefits and drawbacks and in either case regular monitoring is essential.

CANCER

INTRODUCTION

A tumor occurs when cells within a tissue grow in an uncontrolled and progressive fashion. If the tumor grows rapidly and invades adjacent normal tissue and the cells are irregular in appearance, it is malignant and is called a cancer. Cancer requires the development of cells with functionally altered DNA. The source of these alterations may be damage by radiation, chemicals, alteration in gene expression etc. Diet can influence cancer occurrence and growth. Experimental models of chemically induced carcinogenesis have shown the macronutrients, various vitamins such as folic acid, vitamin B12, riboflavin, retinol, β- carotene and alpha tocopherol and some minerals including selenium, zinc, magnesium and calcium to modulate cancer risk. Other nutrients such as lycopene and resveratrol also influence carcinogenesis pathways. Present research shows that foods and nutrients have multiple role in cell signaling, growth and apoptosis and influence tumor growth and cancer survival.

Cancer: Cellular tumour whose natural course is fatal cancer growths are invasive and spread easily.
Neoplasm: Any new or abnormal growth
Sarcoma: Tumor, usally malignant, arising from connective tissues
Carcinoma: Tumor usually malignant, arising from epithelial tissues.
Apoptosis: Programmed cell death.

CAUSES OF CELL DEVELOPMENT

Basic cause of cancer development is a fundamental loss of control over normal cell reproduction. This may be due to

- Mutations – as a result of loss of one or more regulatory genes of the cell nucleus. This may be inherited or due to exposure to some environmental agent.
- Chemical carcinogens which interfere with the structure/ function of the genes. These include cigarette smoking, exposure to pesticide residues, pollution, food additives, and contaminants.
- Radiation – damages DNA, causes breakages and incorrect joining of chromosomes, X-Rays, atomic exhausts, sunlight are all examples of harmful radiation.
- Oncogenic viruses – these interfere with the function of the regulatory genes.

Impact of tumor on Host Metabolism

Malnutrition is a consequence of cancer and results due to the cancer itself or its therapy. The impact of the tumor on the host metabolism leads to cachexia and impaired immune function.

- **Protein metabolism:** Cancer causes increased turnover of whole body protein as well as increased rate of protein synthesis in the liver. However skeletal muscle is not only degraded more but also synthesized less. The tumor has the capacity to retain nitrogen and increase its weight while the rest of the body is wasting.
- **Fat metabolism:** More fat is metabolized and the rate of lipolysis is higher for patients with progressive disease. The plasma concentration of free fatty acids also increases. Fat is oxidized and total body fat decreases in the patient.
- **Glucose metabolism:** Glucose metabolism is altered in the disease condition. Glucose intolerance, insulin sensitivity and decreased responsiveness to insulin are commonly seen.

SYSTEMATOC EFFECTS OF CANCER

Cancer and various antitumor treatments can have adverse effects on the nutritional status of the affected patient. It is important have an understanding of these effects in order to provide nutritional support.

As a tumor grows it produces both local and distant effects. The distant effects are known as paraneoplastic syndromes. These include –

1. Anorexia or loss of appetite occurs due to the presence of an malignant tumour Anorexia is intensified by fear, depression, sepsis and treatment. When severe it can lead to protein calorie malnutrition and wasting of body tissues – commonly known as cachexia. The anorexia and weight loss produced in cancer depends on the type of tumour. The maximum loss is seen in gastric cancers and pancreatic cancers (> 80%) while colon, prostrate and lung cancers also lead to considerable weight loss (48-61%). Breast cancers cause a weight loss of 30 – 40%. In general there is an average loss of 63% weight in general cancer populations.
2. Some tumours produce insulin like substances and use up glucose leading to hypoglycemia.
3. Serious weight loss and nutrient deficiencies have effect on metabolic and immune function with resulting morbidity and mortality.
4. Tube feeding helps in maintaining host weight but it is important to follow it with anti-tumour therapy. Otherwise it may lead to undesirable metabolic reactions.
5. Increased rate of Cori cycling is seen in cancer patients. In this cycle glucose is metabolized to lactate which is resynthesized to glucose. This cycle uses up 6 ATP molecules. However only two ATPs are produced by glycolysis through which the glucose molecules pass through. Thus this is a 'futile cycle' in which more energy is used up than produced and is important for development of weight loss.
6. Tumors also mobilize fat stores for their own use. This not only depletes fat stores by also increases plasma lipid levels which can be immunosuppressive.
7. Excess body fat depletion, increased lepolysis, increase in free fatty acid level, decreased lipogenesis and decreased in activity of serum lipoprotein lipase are all attributed to stress response to illness leading to an increase in catecholemines.
8. Increased whole body protein turnover is seen. Persistent muscle proteins break down and a decrease in plasma branched chain amino acids is seen. Albumin synthesis is depressed due to malnutrition and there is an increased degradation by tumour liposmes both leading to hypo albuminemia.
9. Taste and appetite changes. Unpleasant and unacceptable alterations in the taste of foods occur. The taste threshold for sweetness is elevated while that for bitterness is lowered.

10. Hypercalcemia is a common metabolic complication as the tumour releases substances which cause bone resorption. This leads to nausea, muscle weakness, excess urine, increase in blood pressure and anorexia.

CANCER

INTRODUCTION

A tumor occurs when cells within a tissue grow in an uncontrolled and progressive fashion. If the tumor grows rapidly and invades adjacent normal tissue and the cells are irregular in appearance, it is malignant and is called a cancer. Cancer requires the development of cells with functionally altered DNA. The source of these alterations may be damage by radiation, chemicals, alteration in gene expression etc. Diet can influence cancer occurrence and growth. Experimental models of chemically induced carcinogenesis have shown the macronutrients, various vitamins such as folic acid, vitamin B12, riboflavin, retinol, β- carotene and alpha tocopherol and some minerals including selenium, zinc, magnesium and calcium to modulate cancer risk. Other nutrients such as lycopene and resveratrol also influence carcinogenesis pathways. Present research shows that foods and nutrients have multiple role in cell signaling, growth and apoptosis and influence tumor growth and cancer survival.

Cancer: Cellular tumour whose natural course is fatal cancer growths are invasive and spread easily.
Neoplasm: Any new or abnormal growth
Sarcoma: Tumor, usally malignant, arising from connective tissues
Carcinoma: Tumor usually malignant, arising from epithelial tissues.
Apoptosis: Programmed cell death.

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TYPES OF CANCER

Classified by Body System.
Cancer has the potential to affect every organ in the body. The cells within malignant tumors have the ability to invade neighboring tissues and organs, thus spreading the disease. It is also possible for cancerous cells to break free from the tumor and enter the bloodstream, in turn spreading the disease to other organs. This process of spreading is called metastasis.

When cancer has metastasized and has affected other areas of the body, the disease is still referred to the organ of origination. For instance, if cervical cancer spreads to the lungs, it is still called cervical cancer, not lung cancer.

Blood Cancer: The cells in the bone marrow that give rise to red blood cells, white blood cells, and platelets can sometimes become cancerous. These cancers are leukemia or lymphoma.

Bone Cancer: Bone cancer is a relatively rare type of cancer that can affect both children and adults, but primarily affects children and teens.

Brain Cancer: Brain tumors can be malignant (cancerous) or benign (non-cancerous). They affect both children and adults. Malignant brain tumors don't often spread beyond the brain. However, other types of cancer have the ability to spread to the brain.

Breast Cancer: Breast cancer is a common type of cancer that affects women and much less commonly, men. More than 200,000 women are diagnosed with breast cancer in the United States each year.

Digestive/Gastrointestinal Cancers This is a broad category of cancer that affects everything from the esophagus to the anus. Each type is specific and has its own symptoms, causes, and treatments.

Endocrine Cancers: The endocrine system is an instrumental part of the body that is responsible for glandular and hormonal activity. Thyroid cancer is the most common of the endocrine cancer types and generally, the least fatal.

Eye Cancer: Like other organs in the human body, the eyes are vulnerable to cancer as well. Eye cancer can affect both children and adults.

Genitourinary Cancers: These types of cancer affect the male genitalia and urinary tract.

Gynecologic Cancers: This group of cancer types affect the organs of the female reproductive system. Specialized oncologists called gynecologic oncologists are recommended for treating gynecologic cancer.

Head and Neck Cancer: Most head and neck cancers affect moist mucosal surfaces of the head and neck, like the mouth, throat, and nose. Causes of head and neck cancer vary, but cigarette smoking plays a role. Current research suggests a strong HPV link in the development of some head and neck cancer.

Respiratory Cancers: Cigarette smoking is the primary cause for cancer affecting the respiratory system. Exposure to asbestos is also a factor.

Skin Cancers: Non-melanoma skin cancer is the most common type of cancer among men and women. Exposure to the UV rays of the sun is the primary cause for non-melanoma skin cancer and also melanoma.

OTHER CLASSIFICATION

Cancer is a complex set of diseases. Each cancer is unique in the way it grows and develops, its chances of spreading, the way it affects one`s body and the symptoms one may experience. Several factors, including location and how the cancerous cells appear under the microscope, determine how cancer is diagnosed. All cancers, however, fall into one of four broad categories:

1. **Carcinoma**
2. **Sarcoma**
3. **Leukemias**
4. **Lymphomas**

CARCINOMA

Carcinoma is a malignant neoplasm of epithelial origin. It is a tumor that arises in the tissues that line the body`s organs like the nose, the colon, the penis, breasts, prostrate, urinary bladder, and the ureter. About 80% of all cancer cases are carcinomas.

Basal cell carcinoma

This flesh-colored, wart-like, pearly, smooth, non-scaly papule (bump) is a typical basal cell carcinoma. In this case, it is on a common, sun-exposed area of the face -- the forehead.

This basal cell carcinoma is 5 to 6 centimeters across, red, has well-defined (demarcated) borders and sprinkled brown pigment along the margins. This cancer is located on the person's back.

SARCOMA

Sarcomas are tumors that originate in bone, muscle, cartilage, fibrous tissue or fat. Ewing sarcoma (Family of tumors) and Kaposi`s sarcoma are the common types of sarcomas.

LEUKEMIA

Leukemia are cancers of the blood or blood-forming organs. When leukemia develops, the body produces a large number of abnormal blood cells. In most types of leukemia, the abnormal cells are white blood cells. The leukemia cells usually look different from normal blood cells, and they do not function properly.

LYMPHOMAS

lymphomas affect the lymphatic system, a network of vessels and nodes that acts as the body`s filter. The lymphatic system distributes nutrients to blood and tissue, and prevents bacteria and other foreign "invaders" from entering the bloodstream. There are over 20 types of lymphoma. Hodgkin`s disease is one type of lymphoma. All other lymphomas are grouped together and are called non-Hodgkin`s lymphoma. Non-Hodgkin`s lymphoma may occur in a single lymph node, a group of lymph nodes, or in another organ.

DIETARY

Cancer patient need to take enough nutrients to meet the following goals:

1. Prevent or reverse nutritional deficiencies
2. Decrease side effects of cancer and its treatment
3. Maximize quality of life

The most common ways are

The most common ways are

By mouth

To meet the nutritional needs by taking in nutritious foods and drinks. Able to get enough nutrients by eating high-calorie, high-protein meals supplemented with snacks, canned liquid nutrition products, and homemade drinks and shakes. If that proves to be too hard, or if calorie and nutrient needs have greatly increased, need to use a feeding tube.

By feeding tube

Tube feedings are used most often to maintain good nutrition or boost weight in people who have a poor appetite. Tube feedings can be given at home, if needed, with the help of family, friends, or caregivers. Once tube feedings begin, usually start to feel better because nutritional needs are being met.

By vein (total parenteral nutrition)

Tube feedings may not always provide all the needed fluids and nutrients in people with serious digestive problems. In these cases, nutrient solutions can be given through a vein.
This type of therapy is called intravenous hyperalimentation or total parenteral nutrition (TPN). TPN is most often used when someone has:

- Surgery on the digestive system
- Complete blockage of the bowel
- Severe vomiting or diarrhea
- Complications from cancer or treatment that prevent eating or using a feeding tube
- Like tube feedings, TPN can be given at home.

TREATMENT

Different cancer treatments can cause different kinds of problems that may make it hard to eat or drink. Here mentioned some tips on how to manage nutrition problems depending on the type of treatment.

1. **Surgery**
2. **Radiation Therapy**
3. **Chemotherapy**

SURGERY

Surgery is done to remove cancer cells and nearby tissue. It is often used with radiation therapy and chemotherapy. After surgery, the body needs extra calories and protein for wound healing and

recovery.
This is the time when many people have some pain and fatigue. They also may be unable to eat a normal diet because of surgery-related side effects. The body's ability to use nutrients may also be changed by surgery that takes out any part of the mouth, esophagus, stomach, small intestine, colon, or rectum.

SURGERY

The stress response to surgery is characterized by increased secretion of pituitary hormones and activation of the sympathetic nervous system. The changes in pituitary secretion have secondary effects on hormone secretion from target organs (Table). For example, release of corticotrophin from the pituitary stimulates cortisol secretion from the adrenal cortex. Arginine vasopressin is secreted from the posterior pituitary and has effects on the kidney. In the pancreas, glucagon is released and insulin secretion may be diminished. The overall metabolic effect of the hormonal changes is increased catabolism which mobilizes substrates to provide energy sources, and a mechanism to retain salt and water and maintain fluid volume and cardiovascular homeostasis.

CLASSIFICATION OF SURGERY

Day Surgery
A growing number of conditions needing operation can now be treated by day surgery, usually at the day surgery unit of a hospital. The patient arrives, has the operation and goes home on the same day. The hospital will be able to answer any questions patients may have about specific arrangements for day surgery. Day surgery units have developed considerable experience in ensuring that this type of surgery goes smoothly for the patient and relatives.

Elective surgery
This term is used for operations that are planned in advance, to distinguish them from operations performed as an emergency.

Emergency surgery
Operations that require immediate admission to hospital, usually through the accident and emergency department. They are usually performed within 24 hours, and may be done immediately or during the night for serious or life-threatening conditions. Examples include acute appendicitis; haemorrhage, perforation or obstruction of the intestines; major trauma, including many fractures; and a ruptured spleen or aneurysm.

Keyhole surgery
Advances in surgical treatments have enabled many conditions to be treated by "keyhole surgery", which involves very small incisions and less pain and trauma for the

patient than in conventional surgery. The surgeon can see the area to be operated on by looking through a fine tube with a light on the end (known as a fibre optic light source) and carries out the operation by using special instruments inserted through the tube. Removal of gall bladder or gallstones and some operations on the prostate gland or on joints may be suitable for keyhole surgery. The operations are carried out under anaesthetic.

Laparoscopic surgery
This is similar to keyhole surgery but refers especially to operations performed inside the abdomen and in the peritoneum (the lining of the abdomen).

Microsurgery
Surgeons use a powerful magnifying device to enable them to operate on tiny structures such as small arteries, nerves, the bones of the middle ear or inside the eye. Delicate and extremely small instruments are used.

Organ Transplant Surgery
Where a patient has a failing organ, for example a diseased kidney, it is sometimes possible to replace it with a healthy one donated by another human being. In the case of a kidney the donor may be a relative or someone who has recently died and had completed an organ donor card requesting that their body be used to help others. Transplant surgery is sometimes offered to patients with liver or serious heart failure. Surgeons who carry out this type of surgery require special training in immunology to help prevent the body from rejecting the organ. Unfortunately many patients are denied the opportunity of a transplant because of the great shortage of donors.

CLASSIFICATION OF SURGICAL DISEASE

1. Trauma
    1. Mechanical
    2. Thermal
    3. Chemical
    4. Electrical
2. Infection
3. Neoplasia
    1. Benign
    2. Malignant
4. Anatomical abnormalities
    1. Congenital
    2. Acquired
5. Metabolic & Hormonal disorder
6. Infarction & Ischamia
7. Others

1. Obstruction
2. Parasite diseases
3. Vein varicose

SURGERY AND NUTRITINAL CARE

Nutrient Reserves

- Nutrient reserves can be built up prior to elective surgery to fortify a patients
- Protein deficiencies are common.
- Sufficient kilocalories are required
- Extra carbohydrates maintain glycogen stores
- Vitamin / mineral deficiencies should be corrected
- Water balance should be assessed

Immediate Preoperative Period

- Patients aren't typically directed not to take anything orally for at least eight hours prior to surgery.
- Prior to gastrointestinal surgery, a nonresidue diet may be prescribed.
- Nonresidue elemental formulas provide complete diet in liquid form.

Notes:

Why is it recommended to avoid eating eight hours before surgery? (to prevent aspiration of food during anesthesia and to prevent complications due to food in stomach) What can aspiration cause? How does anesthesia contribute to risk of aspiration? What complications could occur with food in the stomach during surgery? What are the implications for emergency surgery?

Non residue diet:

1. Diet includes those foods that are free of fiber, seeds and skins.
2. Prohibited foods include fruits, vegetables, cheese, milk, potatoes, unrefined rice, fats, and pepper.
3. Vitamin / mineral supplements are required for prolonged non residue diet.

Post surgical Nonresidue Diet

1. Non residue diet plus:
2. Processed cheese, mild cream cheeses
3. Potatoes
4. Bread without bran.
5. All desserts except those containing fruit and nuts
6. Condiments as desired.

POST OPERATIVE NUTRITIONAL CARE

1. Post operative nutrient losses are great, but food intake is diminished.
2. Protein losses occur during surgery from tissue breakdown and blood loss.

3. Catabolism usually occurs after surgery (tissue breakdown and loss exceed tissue buildup).
4. Negative nitrogen balance may occur.

Need for Increased Ptotein

- Building tissue for wound healing
- Controlling shock
- Controlling edema
- Healing bone
- Resisting infection
- Transporting lipids

Problems Resulting From Protein Deficiency

1. Poor healing of wounds and fractures
2. Rapture of suture lines (dehiscence)
3. Depressed heart and lung function
4. Anemia, liver damage
5. Failure of gastrointestinal stomas to function
6. Reduced resistance to infection
7. Extensive weight loss
8. Increased mortality risk.

Other Post operative Concerns

1. Ensure sufficient fluids to prevent dehydration.
2. Provide sufficient non protein calories for energy in order to spare protein for tissue building
3. Ensure adequate vitamins
4. Ensure adequate potassium, phosphorus, iron, zinc.
5. Avoid electrolyte imbalances.

Initial Intravenous Fluid and Electrolytes

- Oral feeding is encouraged soon after surgery.
- Routine postoperative intravenous fluids supply hydration and electrolytes, not k calories and nutrients.

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7. Others

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    2. Parasite diseases
    3. Vein varicose

METHODS OF FEEDING

1. Enteral: nourishment through regular gastrointestinal route, either by regular oral feedings or by the tube feedings.
2. Parenteral: nourishment through small peripheral veirs or large central vein

ORAL FEEDING

1. Allows more needed nutrients to be added
2. Stimulates normal action of the gastrointestinal tract.
3. Can usually resume once regular bowel sounds return.
4. Progresses from clear to full liquids, then to a soft or regular diet.

TRAUMA

Trauma (or any injury) results in tissue damage. Immediately after traumatic injury, a pattern of local reactions and systemic changes is launched. This reparative process involves almost all organ systems.

The local response to trauma serves three goals: stop blood loss, clear tissue debris, and restore normal biological function in the affected area, with the use of scar tissue.

Limitation of blood loss. This begins with a brief constriction of the blood vessels to reduce blood flow to the affected area. Meanwhile, platelets are activated to form a clot or mesh of fibrin to block the bleeding blood vessels. The platelets then release substances such as histamine, serotonin, and cytokines, which activate the next stage of healing, inflammation.

Clearing of tissue debris. Once the bleeding is under control, the body begins to remove damaged and dysfunctional tissue through the inflammatory response. Only after the debris is completely reabsorbed can the body lay down a new tissue framework. Inflammation requires the activation of certain enzyme systems and pro-inflammatory cells that dissolve the damaged tissue.

During this inflammatory period, blood flow to the wound is increased. This vasodilation, which follows the intense vasoconstriction seen immediately after the wound is created, is mediated by chemicals such as histamine, prostaglandins, and those found in the complement cascade, which is part of the immune system response to injury. Under their influence the blood vessel walls in the area of the wound become leaky, allowing repair cells and protein-rich plasma to gather in the damaged tissues. This process results in swelling.

The plasma spilling into the wound serves multiple functions. It dilutes any irritants in the injured area and brings protein molecules called fibrinogen, which link with each other, forming a fibrin mesh big enough to occupy the entire wound. This clot helps trap foreign particles, enhances immune cell effectiveness, and forms the scaffolding over which new tissues are laid down.

Neutrophils are among the first white blood cells to arrive at the site of injury. They remove the dead and dying cells, blood clots, and the fibrin mesh to clean up the wound. Other immune cells, including monocytes, lymphocytes, eosinophils, and basophils, join the neutrophils later. Together these immune cells engulf and digest any bacteria.

Scar Tissue: Long-Term Healing

Once the bleeding has stopped and the body's immediate inflammatory response has been activated, long-term healing and tissue regeneration can begin. The growth of new tissue consists of three different processes:

Angiogenesis, or creation of new blood vessels

Formation of granulation tissue

Remodeling of the scar to suit changing functional requirements

Angiogenesis. The cells lining the damaged capillaries start multiplying to form fresh blood vessels, a process known as angiogenesis. The new capillaries not only help clear the dead tissue but also support the growing cells by supplying oxygen and nutrients.

BURNS

INTRODUCTION

A patient with extensive burns requires rigorous nutritional care. Burns cause a tremendous loss of tissue and additional tissue destruction and nitrogen loss continue. The greatest increase in metabolic rate occurs in patients with extensive burns. The hyper metabolism caused by injury induces a catabolic state characterized by erosion of lean body mass, negative nitrogen balance, altered glucose metabolism etc in proportion to the extent and severity of the injury.

PHYSICAL DESTRUCTION OF SKIN

The extent of a burn wound is defined as the percentage of total body surface damaged and may be determined by the Rule of Nines. This divides the body into areas of 9% or multiples of nine (figure I) and is modified for estimating the extent of burn injury in children

CLASSIFICATION OF BURNS

The depth of a burn injury refers to the amount of skin, and on some occasions other tissue, damaged or destroyed. Skin has two layers, the superficial epidermis and the deeper dermis that overlies subcutaneous tissue. Burns are classified as **Superficial (1st Degree), Partial Thickness (2nd Degree) and Full Thickness (3rd Degree)** depending on how deep and severe they penetrate the skin's surface.

FIRST DEGREE BURNS

First-degree burns affect only the epidermis, or outer layer of skin. The burn site is red, painful, dry, and with no blisters. Mild sunburn is an example. Long-term tissue damage is rare and usually consists of an increase or decrease in the skin color.

Causes
In most cases, first-degree burns are caused by the following:

- mild sunburn
- flash burn - a sudden, brief burst of heat

Symptoms
The following are the most common signs and symptoms of a first-degree burn.

- redness
- dry skin
- skin that is painful to touch
- pain usually lasts 48 to 72 hours and then subsides
- peeling skin

The symptoms of a first-degree burn may resemble other conditions or medical problems.

SECOND DEGREE BURNS

Second-degree burns involve the epidermis and part of the dermis layer of skin. The burn site appears red, blistered, and may be swollen and painful.

Causes
In most cases, second-degree burns are caused by the following:

- scald injuries
- flames
- skin that briefly comes in contact with a hot object

Symptoms
The following are the most common signs and symptoms of a second-degree burn.

However, each child/person may experience symptoms differently. Symptoms may include:

- blisters
- deep redness
- burned area may appear wet and shiny
- skin that is painful to the touch
- burn may be white or discolored in an irregular pattern

The symptoms of a second-degree burn may resemble other conditions or medical problems.

THIRD DEGREE BURNS

Third-degree burns destroy the epidermis and dermis. Third-degree burns may also damage the underlying bones, muscles, and tendons. The burn site appears white or charred. There is no sensation in the area since the nerve endings are destroyed.
Second and third-degree burns require the immediate attention of a physician or other healthcare provider. Listed below you will find additional information relating to first-, second-, or third-degree burns.

Causes

In most cases, third-degree burns are caused by the following:

- a scalding liquid
- skin that comes in contact with a hot object for an extended period of time
- flames from a fire
- an electrical source
- a chemical source

Symptoms

The following are the most common symptoms of a third-degree burn. However, each child/person may experience symptoms differently. Symptoms may include:

- dry and leathery skin
- black, white, brown, or yellow skin
- swelling
- lack of pain because nerve endings have been destroyed

Large third-degree burns heal slowly and poorly without medical attention. Because the epidermis and hair follicles are destroyed, new skin will not grow.

NUTRITIONAL REQUIREMENTS

Nutritional support represents one of the most important cornerstones in the management of patients with a moderate to severe burn injury.

Patients suffering burn over 20 percent or more of their body surfaces require special diets during the healing process. Such severe burning significantly boosts the rate at which bodies metabolize nutrients, creating a risk of malnutrition. Among the key nutritional considerations for severe burn victims are increased protein and high-calorie diets. Burn victims lose protein through their wounds as body systems attempt to regenerate skin and muscle tissues. And extra carbohydrates and fat in burn victims' diets keep energy levels high -- a key ingredient to recovery. Burn Injury Online notes severe burn victims often require up to two extra servings of protein per day through lean meat, fish or dairy. And consuming extra carbohydrates keeps enough body fuel on board, channeling proteins to the process of skin and tissue repair. Fat also provides needed calories for fueling the bodies of burn victims. Doctors often recommend that burn patients receive up to 30 percent of their calories from fat.

Energy

The energy expenditure in burns exceeds that of any other injury. Oxygen consumption increases and peaks by day 10 post burn to 2.5 times that of the normal. The caloric needs are estimated roughly on basis of following guidelines – 25 kcals/kg body weight + 40 kcals x % body burns.

Calorie requirements peak between 6th and 10th post burn day and decline to normal levels with complete skin coverage.
Underfeeding has a negative impact on wound healing, immunocompetence and mortality. Over feeding causes hyperglycemia, fatty livers and elevated co2 production. Patient should be kept warm and pain relief provided.

Protein

Protein is the most important nutrient compromised by a burn injury - protein needs can be approximated by using the formula – 1g/kg body wt + 3g x % body burns.
The protein source should be of high biological value. Arginine enriched diet improves cell mediated immunity and healing.
Nitrogen balance is a useful parameter for estimating adequacy of protein intake in burns patient. This can be calculated by the formula –
Nitrogen intake – (24 hour urine urea nitrogen + fecal nitrogen loss g/24 hours + wound nitrogen loss g/24 hours)
Wound nitrogen loss is given as –
<10% open wound=0.02 g nitrogen/kg/day.
11% to 30% open wound=0.05 g nitrogen/kg/day.
>30% open wound=0.12 g nitrogen/kg/day.
Nitrogen excretion should be decreased as the wounds heals or are grafted.
Efficient use of nitrogen requires simultaneous administration of potassium which is excreted heavily after a burn. The potassium-to-nitrogen ratio should be 6:1 until the late convalescent phase.

Carbohydrates
The burn wound metabolizes large quantities of glucose to lactic acid; this pathway is favoured by healing wounds. Hyperglycemia is common during the acute phase following thermal injury and this should be monitored and controlled using exogenous insulin carbohydrates are beneficial as nutritional substrates and help in utilization of protein. However excessive carbohydrate intake causes side effects.

Fats
Conservative ingestion of fats is beneficial. Giving 15-20% of non protein calories as fats is optimal supplementation with omega – 3 fatty acids improve immuno competence. The administration both enterally & parenterally, of low fat formula results in less pneumonia, improved respiratory function, faster recovery of nutrition status and a shorter length of care.

Micronutrients
Daily multivitamin supplementation is important. Enrichment with vitamins A and C and zinc is also necessary.

- 5000 IU of vitamin A per 1000 kcalories helps in epithelialization and maintenance of immunologic response.
- Vitamin C is important in collagen synthesis and immune function and required in increased amount of wound healing, its usual doses 500 mg twice daily . Ingestion of 1gram of vitamin C is also necessary.
- Supplementation with 220 mg of zinc sulphate daily is recommended.
- Administration of calcium to treat hypocalcemia, and supplemental magnesium & phosphorus are given to prevent gastrointestinal irritation.

GUIDELINES FOR FEEDING

Patients with burns ≤ 20% of body surface area can be given oral feeding. Enteral feeding should be given if the burn area exceeds 20%. Enteral feeding should be started as early as possible. A lactose free formula with a high protein content can be selected.

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For a burns patient, body weight is the most important parameter that guides nutritional support. The adequacy of nutritional support can be seen in the increasing body weight.

- satisfied
- Improved tube feeding tolerance
- Decreased incidence of bacterial translocation
- Decreased number of infectious episodes

- Decreased antibiotic therapy
- Improved nitrogen balance
- Reduced urinary catecholamines
- Diminished serum glucagon
- Suppressed hypermetabolic response
- Enhanced visceral protein status

The administration both enterally & parenterally, of low fat formula results in less pneumonia, improved respiratory function, faster recovery of nutrition status and a shorter length of care.

FOOD ALLERGY

INTRODUCTION

Food allergy is also a leading cause of anaphylaxis (a severe, potentially fatal allergic reaction; please see Anaphylaxis article in this supplement for more information) presenting to emergency departments [2]. Annually, there are approximately 200 deaths in the United States attributed to food allergy [5]. At present, there are no accurate data regarding food allergy-related deaths in Canada.

Accurate diagnosis and appropriate management of food allergy are critical since accidental exposure to even minute quantities of the food causing the allergic reaction may result in anaphylaxis [6]. This article provides an overview of current literature related to the epidemiology, pathophysiology, diagnosis, and appropriate management of food allergy. This review focuses primarily on immunoglobulin E (IgE)-mediated food-allergic reactions.

DEFINITION

The term food allergy is used to describe an adverse immunologic response to a food protein. It is important to distinguish food allergy from other non-immune-mediated adverse reactions to foods, particularly since more than 20% of adults and children alter their diets due to perceived food allergy [4]. Adverse reactions that are not classified as food allergy include food intolerances secondary to metabolic disorders (e.g., lactose intolerance), reactions to toxic contaminants (e.g. histamine produced by scombroid fish contaminated by Salmonella organisms) or pharmacologically active food components (e.g. caffeine in coffee causing jitteriness, tyramine in aged cheeses triggering migraine). Other conditions which are associated with symptoms similar to food allergy include auriculotemporal syndrome (a disorder characterized by facial flushing and salivation that may follow trauma to the parotid gland), and gustatory rhinitis [2–4].

EPIDEMIOLOGY

Many food allergies, particularly allergies to milk, egg, soy, and wheat, are usually outgrown within the first ten years of life [9]. In contrast, allergies to peanut, tree nuts, fish, and shellfish are often lifelong, although 20% of individuals may outgrow peanut allergy [10]. Peanut and tree nuts are responsible for the most serious allergic reactions and food-allergy related fatalities [11]. Canadian prevalence estimates for five of the major food allergens (i.e., peanut, tree nuts, fish, shellfish, and sesame)

Children with atopic disorders tend to have a higher prevalence of food allergy; approximately 35% of children with moderate-to-severe atopic dermatitis have IgE-mediated food allergy [12]. Studies have also shown that children with atopic dermatitis and food allergy have a higher prevalence of allergic rhinitis and asthma. Approximately 75% of children with atopic dermatitis develop allergic rhinitis and 80% develop asthma [13].

CLINICAL SYMPTOMS

Children with atopic disorders tend to have a higher prevalence of food allergy; approximately 35% of children with moderate-to-severe atopic dermatitis have IgE-mediated food allergy [12]. Studies have also shown that children with atopic dermatitis and food allergy have a higher prevalence of allergic rhinitis and asthma. Approximately 75% of children with atopic dermatitis develop allergic rhinitis and 80% develop asthma [13].

Skin reactions are the most common clinical manifestations of allergic reactions to food and include acute urticaria (hives), angioedema (swelling) and erythema (redness of the skin). Typical respiratory tract symptoms include laryngeal edema, rhinorrhea, and bronchospasm. GI-related signs and symptoms of food allergy include nausea, vomiting, abdominal pain, and diarrhea.

The mildest IgE-mediated reaction is the oral allergy syndrome, which causes tingling and itching of the mouth and pharynx. This is typically triggered after consumption of fresh fruits and vegetables in pollen-allergic individuals. It is caused by cross reactivity of IgE antibodies to certain pollens with proteins in some fresh fruits and vegetables (see Table 3) [6]. For example, individuals with ragweed allergy may experience oropharyngeal symptoms following the ingestion of bananas or melons, and patients with birch pollen allergy may experience these symptoms following the ingestion of raw carrots, celery or apple. Fortunately, these proteins are heat labile, enabling allergic individuals to eat these foods when cooked. Allergy skin tests are usually negative to

commercial food extracts in individuals with oral allergy syndrome, but are positive to the fresh food. Also, progression to systemic symptoms is rare, but may occur in a small proportion of patients with the condition

DIAGNOSIS

The diagnosis of a food allergy requires a detailed history and physical examination, and diagnostic tests, such as skin prick tests (SPT) and/or serum-specific IgE testing to foods (ImmunoCAP®). In some cases, oral food challenges may also be required [2–4].

Referral to an allergist is important to confirm the diagnosis of a suspected food allergy. Patients should avoid the food in question until assessment, and an epinephrine auto-injector should be prescribed, even if the diagnosis is uncertain

TREATMENT

There is currently no treatment for food allergy, beyond avoidance of the responsible food(s). Once a food allergy is diagnosed, strict elimination of the offending food allergen from the diet is necessary. A properly managed, well-balanced elimination diet can lead to resolution of symptoms while maintaining nutritional status. When the elimination diet is used as treatment, the identified food allergens are removed from the diet indefinitely, unless evidence exists that the food allergy has resolved

PREVENTION

Strategies for the prevention of food allergy have been extensively studied. Prior recommendations suggesting avoidance of highly allergenic foods in infant diets and the diets of pregnant/breastfeeding mothers have not been shown to decrease the prevalence of food allergy or atopic disease [6]. Recent guidelines from the American Academy of Pediatrics state that “no current convincing evidence exists to recommend specific avoidance of certain foods beyond 4-6 months of age for the prevention of allergy”

CONCLUSION

Food allergy is an important clinical problem of increasing prevalence. Assessment by an allergist is very important for appropriate diagnosis and treatment. Diagnosis currently relies on a careful history and diagnostic tests,

such as SPT, serum-specific IgE testing (where appropriate) and, if indicated, oral food challenges. The mainstay of treatment is avoidance of the responsible food(s) and appropriate, prompt response to allergic reactions with epinephrine. Further insights into the pathophysiology of food allergy will lead to the development of improved methods for prevention, diagnosis, and management of the disorder.