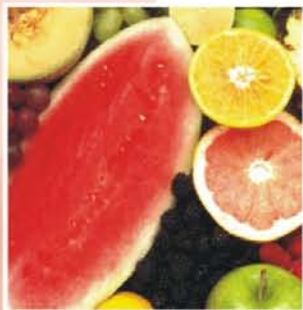


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ADVANCES IN DIET THERAPY

PRACTICAL MANUAL



NEW AGE INTERNATIONAL PUBLISHERS

V. Vimla

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IN DIET THERAPY**

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ADVANCES IN DIET THERAPY

PRACTICAL MANUAL

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**Dedicated to my beloved students
all over the country**

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Preface

As the incidence of diet related disorders is on the rise, the field of diet therapy is becoming increasingly important. Manipulation of diet in several disease conditions is considered to be essential for clinical management of diseases for reducing drug dosages or even to arrest the disease progress. Therapeutic modification of normal diet involves planning of therapeutic diet individually for each patient as the nutrient requirements vary from patient to patient. Hence a standard diet plan for a particular condition no more holds good. This has brought about a tremendous change in the role of dietitian in hospital diet care setting and made her an indispensable member of the medical team. Several professionals may be involved directly or indirectly with the nutritional care of people. However, registered dietitians are the only professionally educated group whose primary care and concern is the application of nutritional science to the health care of people. To make dietitians professionally competent and excel themselves in clinical setting, as students they need to be trained thoroughly in individual patient care. Numerous textbooks and journals are available in the field of nutrition and dietetics to attain theoretical knowledge. But students who are specializing in dietetics should be trained to handle practical situations in hospitals. To attain hands on experience, this type of practical manuals in dietetics are not available so far. As a teacher and expert diet counsellor, with an experience of 30 years, I have tried to evolve a suitable practical manual with basis as well as advances to gain hands on experience by dietetic student. I acknowledge various sources of textbooks, journals and hospitals which have contributed immensely for the development of this manual. I am also extremely indebted to Dr. M.V. Rao garu, the then Vice-chancellor of Acharya N G Ranga Agricultural University for encouraging to take up this work. I also thank our postgraduate students of nutrition [ANGRAU (Acharya N G Ranga Agricultural University)] for being a source of inspiration for this outcome. I hope this manual will be extremely useful for upcoming teachers, students of dietetics and dietitians.

V. Vimala

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Chapter 1

Therapeutic Modification of the Normal Diet—Their Uses in Dietary Treatment

The normal diet may be modified:

- to provide change in consistency as in fluid and soft diets.
- to increase or decrease the energy values—reducing diets.
- to include greater or lesser amounts of one or more nutrients—high protein and low fibre diets.
- to increase or decrease bulk—high and low fibre diets.
- to provide foods bland in flavour.
- to include or exclude specific foods as in allergic conditions.
- to modify the intervals of feeding.

1.1 MODIFICATION IN CONSISTENCY

These diets are used in the treatment of gastro intestinal tract. They can be from a very low residue diet to a very high fibre diet. Method of feeding is by mouth, unless otherwise indicated.

DIETS WITHOUT SOLIDS

Liquid Diets

Liquid diets consist of a variety of foods that are liquid or liquify at room temperature. These diets are used in:

- Febrile states
- Post operative conditions
- Wherever the patient is unable to tolerate solid food.

Liquid diets are of two types namely

- Clear fluid diet
- Full fluid diet

Clear Fluid Diet

This diet is indicated in:

- Acute illness
- Surgery
- Gastrointestinal disturbances.

2 *Advances in Diet Therapy*

A clear fluid diet is usually used for 1 or 2 days. After that a more liberal liquid diet is given. The amount per feeding is 30 – 60 ml/hour. As the patients tolerance improves, the amounts can be increased.

Foods Permitted

Tea with lemon and sugar
Coffee
Fat free broths.
Carbonated beverages
Cereal waters.

Full Fluid Diet

This diet is indicated when a patient is:

Acutely ill.

Unable to chew or swallow solid food.

This diet includes all foods which are liquid at room temperature.

It is free from cellulose and irritating condiments.

Iron is provided at inadequate levels.

Six or more feedings can be given daily.

The protein content of the diet can be increased by incorporating whole egg, egg white, nonfat dry milk in beverages and soups.

The calorie value of the diet can be increased by adding butter to cereal gruels and soups, glucose in beverages and using ice creams, dessert.

If decreased volume of fluid is desired, non fat dry milk can be substituted for the part of the fluid milk.

Foods Allowed

Beverages	—	Cocoa, coffee or tea.
Cereal	—	Fine or strained gruels.
Dessert	—	Soft custard, gelatin.
Eggs	—	Raw in broth with fruit juices or milk.
Fruit	—	All strained juices.
Meat	—	Strained in soups.
Vegetables	—	Puree, soups.
Miscellaneous	—	Butter, cocoa, sugar, salt.

Commercial Liquid Formulas

These are used to supplement other external diets.

These formulas vary in composition and source of nutrients.

Most of the formulas are milk based.

For persons who cannot tolerate milk, protein source is meat, soy or casein hydrolysate.

Fat and carbohydrate composition and proportions also vary to accommodate persons with different needs and restriction.

Criteria for Selection of Appropriate Formulae

- The protein sources
- The composition and proportion of fats and carbohydrates
- The osmolality
- The palatability
- The cost.

DIETS WITH SOLIDS

Soft and Low Fibre Diets

Soft diet is between liquid diet and normal diet.

Soft diet includes both liquid and solid foods which contain restricted amount of indigestible carbohydrates and no tough connective tissue. The diet can be made mechanically soft by cooking, mashing, pureeing the foods used in a normal diet. Further reduction in indigestible carbohydrate can be achieved by the use of refined breads, cereals immature vegetables and fruits.

The skin and seeds of fruits have to be removed.

Soft fruits like banana can be used as it is.

Tough connective tissue can be reduced, by selecting tender meat and cooking very soft.

Meat and meat broths have to be restricted because the nonprotein nitrogen products such as creatine, creatinine, purines and other products which are present in muscle tissue is extracted into the gravy which stimulates gastric juice.

Strong flavoured vegetables such as onions, radish, dried beans, cabbage, cauliflower have to be omitted if necessary.

With proper cooking (Short cooking time, vessel uncovered, serving immediately) it is desirable to eliminate those vegetables which the individual patient cannot tolerate.

It is not necessary to eliminate all spices, only gastric irritants like black pepper, chilli pepper, cloves etc., can be eliminated.

This diet is nutritionally adequate.

It is soft in texture and bland in flavour.

Low Residue Diets

The diet is made up of foods which can be completely absorbed, thereby leaving little or no residue for formation of faeces. This diet provides insufficient minerals and vitamins.

It must be supplemented.

Foods high in fibre should be omitted.

Food which contain residue but not fibre such as milk are also omitted or restricted.

Two cups of milk may be permitted per day. Strained fruits and vegetables without skins are usually permitted.

Meat should be tender or ground to reduce connective tissue.

The Diet is Usually Used in

- Severe diarrhoea to afford rest to the gastrointestinal tract.
- Acute phases of diverticulitis.
- Ulcerative colitis in initial stages.
- Operations.
- Partial intestinal obstruction.
- Whenever necessary to reduce bulk in the gastrointestinal tract.

High Fibre Diets

Dietary fibre plays a significant role in colonic function.

High fibre diet is mainly used for atonic constipation and diverticulosis.

This is a normal diet with fibre increased to 15–20 gms daily.

Fluid intake is also increased.

Concentrated foods should be replaced by those of greater bulk.

4 Advances in Diet Therapy

Foods which can be included in the diet are plenty of long fibered vegetables, salads, fruits and whole cereal grains.

Highly refined and concentrated foods, excessive amounts of rough brans and excessive seasoning should be avoided.

Intervals of feeding should be three meals daily.

Table 1.1 : Foods Allowed and Avoided in Low Residue Diet

<i>Foods allowed</i>	<i>Foods avoided</i>
Beverages Coffee, tea, fruit and vegetable juices, carbonated beverages, milk (2 cups/day) curds.	Milk or curds in excess of two cups.
Cereals Refined wheat bread, refined cereals and millets, dry cereals that are not whole grain, spaghetti, macaroni, noodles, rice.	Whole grain bread or cereals, brown or white rice.
Desserts Simple pudding made from milk allowance, plain ice cream, plain gelatin deserts, plain cakes, cookies and pies and sweets made from allowed foods.	All deserts containing coconut, nuts, seeds, fruits, jams, preserves, milk sweets.
Fats Cooking oils, butter, cream, mayonnaise.	Fried foods, high fat gravy, spicy salad dressings.
Fruits Fruit juices, peeled apricots, cherries, baked apple, ripe banana, orange and citrus fruits without membrane.	All others fruits with seeds and skins.
Meat/Meat Substitutes Tender chicken, fish, lamb, liver pork, eggs, cottage cheese.	Fried and highly seasoned, smoked or pickled meat, fish or poultry.
Soups Plain cream soup made from milk allowance, clear broth, noodles or rice.	Soups made with vegetables not allowed.

1.2 MODIFICATION IN NUTRIENTS

High Calorie Diets

These diets are prescribed for

- Weight loss
- Fever
- Hyperthyroidism
- Burns.

This is a normal diet with an increase in the calorie level to 3000 or more. If appetite is poor, small servings of highly reinforced foods are given.

The diet may be modified in consistency and flavour, according to specific needs.

Excessive amounts of bulky low calorie foods, fried foods or others which may interfere with appetite should be avoided.

Low Calorie Diet

These diets are prescribed for weight reduction in

- Diabetes Mellitus
- Cardiovascular diseases
- Hypertension
- Gout
- Gall bladder disease
- Preceding surgery.

This is a normal diet with energy values reduced to 1500, 1200 or 1000 calories.

Protein levels should be at 65 to 100 gms.

Supplements of Vitamin A and thiamine are usually required for diets below 1000 calories.

High Protein Diet

High protein diet of 100 – 125 g per day may be prescribed for a variety of conditions like

- Fever
- Hyper thyroidism
- Burns
- Nephrotic syndromes
- Haemorrhage
- After surgery
- Diarrhoea
- Ulcerative colitis
- Sprue
- Celiac disease
- Cystic fibrosis
- Infective hepatitis
- Elderly
- Alcoholics.

Low Protein Diet

Low protein diets are usually prescribed for conditions like

- Hepatic encephalopathy
- Acute and chronic glomerulonephritis
- Nephrosclerosis
- Acute and chronic renal failure
- In-born errors of metabolism.

In severe liver disorders, protein cannot be used for synthesis, the amino acids are catabolised and excess ammonia cannot be converted to urea for excretion and the patient develops hepatic coma.

In this situation protein levels must be decreased or completely restricted for a few days.

Patients with Kidney diseases such as acute and chronic glomerulonephritis, nephrosclerosis, acute and chronic renal failure require low protein diets since the kidney cannot excrete nitrogenous wastes.

Diets containing 18 to 22 gms of high biological value protein may be needed for the chronic uremic patients who is not being dialysed.

Low protein diets are also prescribed for patients with in-born errors of metabolism that result from lack of enzymes of the urea cycle.

In celiac disease, the protein gluten in wheat and rye brings out a metabolic defect in the intestinal mucosal cells.

The protein gluten is composed of two fractions glutenin and gliadin.

The latter protein causes the difficulty.

Treatment involves the removal of this protein from the diet. (Gliadin free diet).

The amino acid composition of the diet may need to be modified in cases of an inborn error of metabolism in which the amino acid content cannot be metabolized normally and as a result other metabolites are accumulated which are toxic.

In such cases, the amino acid from the diet is either decreased or completely eliminated.

Phenylalanine	—	Phenylketonuria
Methionine	—	Homocystinuria
Valine	—	Hypervalinemia

The treatment of maple syrup urine disease is difficult since the amount of three essential amino acids must be regulated namely valine, leucine and isoleucine.

Protein free diet

Is also recommended in cases like hepatic coma, acute aneuria.

Fat Controlled Diet

Since steatorrhoea occurs in gall bladder diseases and malabsorption syndrome the amount of fat is restricted.

Usually fat controlled diets are prescribed for—

- Gall bladder diseases
- Nontropical sprue
- Celiac disease
- Cystic fibrosis
- Atherosclerosis
- Myocardial infarction
- Hyperlipidemia etc.

Fat controlled diets regulate the amount and type of fat allowed.

The calories from fat should provide about 30% and 35% of the total calories with 10% from saturated fat and 12 – 14% from poly-unsaturated fats.

Even the intake of cholesterol also is reduced from the average daily intake of 600 to 300 mg.

Medium chain triglycerides are useful for individuals with those disorders that result in impairment of fat digestion or absorption.

Medium chain triglycerides do not require bile salts or micelle formation for absorption.

Low Sodium Diet

The mineral content of the diet may also be modified.

Four levels of sodium restriction are most often used — 250, 500, 1000 and 2400 – 4500 mgs.

The first diet is a severe restriction that excludes salty foods and salt in cooking and at the table.

This diet is used both to prevent and treat edema.

Therefore, it is prescribed for congestive heart failure, hypertension, toxemia of pregnancy, liver and renal diseases.

Some renal patients may not be able to regulate sodium excretion and become hyponatremic.

Then sodium must be added back by means of the diet.

In renal patients with chronic uremia it may be necessary to also restrict potassium and phosphorous.

Guidelines for reduction of phosphorous.

- ❑ Omit milk, yoghurt and ice cream. Use non dairy cream substitutes.
- ❑ Use meat poultry and fish only in amounts compatible, with high biological value protein intake.
- ❑ Exclude dried beans and peas.
- ❑ Omit cola beverages.
- ❑ Following above guidelines diets will contain about 15 mg phosphorous per 1 g protein. For example a 50 g protein diet will contain about 750 mg phosphorous.

Food High in Phosphorus

Cheese (Processed, Cheddar) liver, fish, egg, milk and milk products, whole grain cereals, lima beans, mushrooms, peas (cooked) etc.

Food Allergies and Food Intolerances

Specific foods may cause an immunologically mediated response in certain individuals.

Those foods which cause a known reaction are eliminated from the diet. Common offenders are milk, egg, wheat, chocolate and many other foods. Diagnoses of foods to which the individual has an allergic response can be accomplished through diaries, use of elimination diets, use of skin tests and use of laboratory tests.

Foods intolerance result in symptoms associated with the intake of that food but are not immunological responses.

A common example of food intolerance would be lactose intolerance. Food with milk are eliminated from the diets.

Lactose deficient subjects can tolerate lactose predigested milk products.

Modification in nutritional requirements in therapeutics do not occur in isolation. In some disorders, based on the patients condition many nutrient modifications need to be done. Dietitian has an important role in translating these diet prescription to meet the nutritional needs of the patients.

Table 1.2 : Foods Allowed and Avoided in Fat Restricted Diet

<i>Foods allowed</i>	<i>Foods avoided</i>
<p>Beverages Skim milk, coffee, tea, fruit juices.</p>	Whole milk, beverage with cream or ice cream.
<p>Cereals All cooked cereals without bran, macaroni, noodles, spaghetti.</p>	—
<p>Breads All kinds except those with added fat.</p>	Sweet rolls with fat, french toast.
<p>Desserts Fruit pudding, gelatin, cereal puddings using part of milk allowance.</p>	Sweets containing fats, chocolate cream, nuts, cookies, cakes, doughnuts, icecream, pastries, pies, rich puddings.
<p>Eggs - 3 per week</p>	Fried eggs.

Contd...

<i>Foods allowed</i>	<i>Foods avoided</i>
<p>Fat – Vegetable oil</p> <p>Meats Boiled, baked, roasted or stewed without fat, lean chicken, lamb, fish.</p> <p>Seasonings In moderation — Salt, pepper, spices, herbs, flavouring extracts.</p> <p>Soups — Clear</p> <p>Sweets Jam, jelly, marmalade sugars, sweets, with limited fat.</p> <p>Vegetables — All kinds</p> <p>Miscellaneous</p>	<p>Cooking fats, cream, salad dressing.</p> <p>Fatty meats, poultry or fish bacon, duck, goose, fish canned in oil, organ meats, smoked or spiced meats.</p> <p>Sometimes not tolerated — Pepper, meta sauced, excessive spices, vinegar.</p> <p>Cream soups.</p> <p>Sweets with excessive fat, nuts and chocolate.</p> <p>Strongly flavoured vegetables — Cabbage, cauliflower, cucumber, onion, radish, beans, dried cooked peas.</p> <p>Fried foods, gravies, nuts, butter, pickles, relishes, popcorn.</p>

Table 1.3 : Foods Avoided and Recommended on a Low Salt Diet

<i>Foods allowed</i>	<i>Foods avoided</i>
<p>Cereals Salt free bread, rice, wheat, millets, biscuits, muffins</p> <p>Meat/Meat substitutes Fresh or frozen lamb, liver, pork, chicken, fresh fish, salt free cheddar cheese, cottage cheese and peanut butter, egg.</p> <p>Milk Skim milk, milk.</p> <p>Beverages Tea, coffee, carbonated beverages, wine, beer.</p> <p>Fats Unsalted butter, oils.</p> <p>Vegetables Green beans, cabbage, cucumber, mushrooms, onions, peas, potato, pumpkin, radish, tomato</p> <p>Fruits Fresh fruits</p> <p>Deserts Plain gelatin desserts, cakes and cookies with low sodium baking powder.</p>	<p>Breads, crackers with salted tops, salted snack foods.</p> <p>Smoked, salted pickled meat, fish, poultry, beef etc.</p> <p>Butter milk, instant cocoa mixes.</p> <p>Softened water.</p> <p>Meat extractives, highly salted salad dressings.</p> <p>Brine cured vegetables, tomato pastes, sauces, purees, commercially canned vegetables, frozen peas, carrots frozen, lima beans.</p> <p>Fruits with sodium preservative, dried figs and raisins.</p>

PRACTICAL ASSIGNMENT

AIM: To discuss the characteristics of the following modified diets and also to highlight on which disease conditions they can be used.

Table 1.4 : Modified Diets and their Uses

<i>Sl. No.</i>	<i>Type of diet</i>	<i>Characteristics of the diet</i>	<i>Indications for use</i>
1.	Potassium restricted diet		
2.	Acid ash diet		
3.	Alkaline ash diet		
4.	High protein, high calorie diet		
5.	Low protein, low sodium diet		
6.	Low purine diet		

2. List 6 recipes suitable for the following modified diets.

I. Clear liquid diet

II. Semi solid diet

III. Bland diet

IV. Low residue diet

V. Low calorie diet

Chapter 2

Aspects of Nutritional Management (Problem Oriented Medical Record)

Major factors associated with the nutritional management of the client include:

- Assessment of her/his nutritional state, diagnosis of the nutritional problem(s).
- Proposed plan for care of the nutritional problem(s).
- Education of the client for self management of her/his nutritional problems.

Reassessment should be done after a definite time point.

2.1 SOAP FORMAT FOR NUTRITIONAL MANAGEMENT

SOAP format can be easily applied to all aspects of nutritional management *i.e.*,

- Assessment
- Care
- Education

SOAP means

- S : *Subjective evaluation*. This is the information collected from the patient or relatives.
- O : *Objective evaluation*. This includes actual measurements *i.e.*, assessment of anthropometric measurements, or analysis of blood/urine.
- A : *Assessment*. This is the reasoning process which results in the determination of nutritional status from which a nutritional care plan can be evolved.
- P : The plan for nutritional care or therapy should be written very specifically instead of vague generalities.

Basic Components of a Problem Oriented Medical Record Flow Chart using a Nutritional Problem as an Example

I. Data Base

Subjective: Dietary history
Family history
24-hour recall
Activity record
Physical signs
Food intolerances/allergies

Objective: Anthropometry
Height,
Weight,
Relative weight
Triceps fatfold
Body fat, %
Physical signs
Biochemical measurements
Diet order

II. Assessment

Example:

Dietary history and 24-hour recall indicate daily energy intake of 3000 Kcal.
Relative weight for height is 200% and triceps fatfold is 50% above normal.
Physical signs include several bulging fatfolds.

III. Plan

- (a) More information needed for diagnosis (Dx).
- (b) Specific treatment or nutritional care plan (Rx).

Example:

Implement physician prescribed diet of 1000 Kcal. Calculate current energy needs of client to predict expected weight loss for 1000 Kcal for 1 week. Weigh patient in one week to evaluate weight loss and diet adherence.

2.2 PRACTICAL ASSIGNMENT

AIM : To prepare a SOAP note for the case history of a patient given below.

Case History of Patient

Mr. Bheem Singh is 40 year old man. He had edematic feet from past two months. He was suffering from restlessness since two months. He was admitted in the hospital with a history of swelling of feet and distension of abdomen (ascites) from past two months.

Previous illness

Not a known case of diabetes mellitus
No Jaundice
No similar complaints in the past
He was alcoholic in the past, but stopped drinking now. He is a known smoker.

Physical examination

Height : 5'9"
Weight : 55 Kgs.
Temperature : 98.4°F
Pulse rate : 80/min
B.P. : 150/100

Laboratory data

Blood sugar : 90 mg/dl
Blood urea : 30 mg/dl
Serum cholesterol : 210 mg/dl
Serum creatinine : 1 mg/dl
Blood group : B+

Erythrocyte sedimentation rate (ESR)

1st hour 120mm
2nd hour 130mm
Urine colour : light yellow
Appearance : clear
Reaction : acidic
Albumin : 23 mg/24 hrs
Sugar : Nil
Bile salts : Nil
Bile pigments : Nil

Chapter 3

Cycle Menus for Special Diets

'General', 'house' and 'regular' are interchangeable terms used to identify the menu of diet served to patients who do not need a modified diet. 'Modified', 'special' or 'therapeutic' are usual terms used to refer the diets which have been altered in some way from the general diet and are prescribed for therapeutic purposes. In some cases, these diets are also prescribed for delay or prevention of symptoms associated with disease.

3.1 THE CYCLE MENU

Dietitians who are responsible for hospital finding or other group finding units develop a cycle menu. A great deal of time and effort goes into the development of cycle menu. Cycle menu should take into account nutritional quality of and adequacy of the day's diet, appropriate combinations of colours, flavours and textures, the budget facilities available. Furthermore, in the hospital the cycle menu should contain variety so that the menu can readily be modified for special diets.

Cycle menus are written to cover a definite period of time such as 1 to 3 months after which they are recycled. These menus are sufficiently flexible to incorporate seasonal changes.

Special diets are developed by serving the same foods as those served on regular cycle menu. Dependent upon the kind of modified diet required, the consistency seasoning or other aspect of the food may be altered. The recommended daily dietary allowance (RDA) for each nutrient should be met. Exceeding the RDA, in some cases can be as grave as error as too little. Examples of cycle menus from two hospitals are included.

**Table 3.1: Sample Cycle Menus
Hospital I**

	<i>Sunday</i>	<i>Monday</i>	<i>Tuesday</i>	<i>Wednesday</i>	<i>Thursday</i>	<i>Friday</i>	<i>Saturday</i>
Breakfast	Upma	Dosa with Chutney	Chapati with Dhal Fry	Idli with Chutney	Dosa with Chutney	Puri with Alu curry	Chapati Dhal
Lunch	Rice Avial Sambar Rasam Chapati Papad	Rice Cho-Chow, Peas Poriyal Mint chutney Dhal Rasam Puri	Ghee rice Mixed vegetable Korma Raita Butter milk Papad	Rice Chapati Kovai poriyal Tomato chutney Rasam Sambar Papad Butter milk	Vegetable pulao Curd rice Vegetable curry Papad Rasam Butter milk	Rice Chapati Carrot Poriyal Rasam Sambar Papad Butter milk	Rice Dhal palak Rasam Sambar Butter milk Veg. noodles
Special	Fish	Egg	Chicken	Fish	Beef	Chicken	–
Dinner	Rice Potato Poriyal Sambar Rasam Pickle Chapati Butter milk	Rice Green gram Poriyal Sambar Rasam Butter milk Papad Chapati	Rice Lady finger Poriyal Rasam Butter milk Pickle Chapati	Rice Beans pugath Sambar Rasam Pickle Papad Butter milk	Rice Chapati String bean Sambar Rasam Papad Butter milk	Rice Chapiti Cabbage poriyal Coconut chutney Rasam Pickle Butter milk	Tomato bath Coconut chutney Kesan bath Chapati Egg curry Butter milk

**Table 3.2 : Sample Cycle Menus
Hospital II**

<i>Day</i>	<i>Breakfast</i>	<i>Lunch</i>	<i>Dinner</i>
Monday	Idli-2 Dosa Chutney Sambar Grapes	Watermelon soup Tadka dal Chole/Baigan bartha Sprouted salad Vermicelli payasam	Palak soup Radish sambar Capsicum masala Mutterpaneer Tomato salad Orange
Tuesday	Idli-2 Uttappam-1 Roasted Bengal gram Chutney Sambar Papaya	Veg. thick soup Dalcha Rajmah cabbage Tossed salad Fruit custard Tomato	Lentil soup Tomato sambar Chikka kura Alu khurma Green salad Banana Carot
Wednesday	Idli-2 Veg. upma Tomato Chutney Sambar Pineapple	Soup Gongura dal Usal (dry)/Bhendimasala Cuamber Moong Dal payasam	Soup Drumstick sambar Onion pulusu Mixed veg. dry (DIB-Bendi) Cucumber salad
Thursday	Idli-2 Dosa Onion chutney Sambar Apple	Kidney bean soup Pancharatan dal Tinda Kairee pugath Tomato salad Channa dal sweet	Tomato thick soup Bhendi sambar Cucumber dry Avial Banana
Friday	Idli-2 Vermicelli upma Coriander chutney Sambar Watermelon	Veg. clear soup with chopped vegetables Mango dal Tomato curry (Minced nutrella dry) Shredded cabbage quarters Vermicelli payasam with Custard	Green peas soup Mixed veg. sambar Amchur karela Dal with quens Veg. Salad Orange

Contd...

<i>Day</i>	<i>Breakfast</i>	<i>Lunch</i>	<i>Dinner</i>
Saturday	Idli-2 Pesarattu Ginger chutney Sambar Orange	Cauliflower soup Moong dal fry Palak saag with paneer Tindli Cucumber salad Fruits salad	Tomato shorba Carrot sambar Padwal with channa dal Chikkudu kaya Sprouted methi salad Banana
Sunday	Plain bread Jam Butter Muskmelon	Hara dhanias soup Spring onion dal Drumstick kadi Lauki masala Rask ka meetha Cucumber salad	Potato thick soup Onion sambar Turai capsicum Methi mutter Orange

Rice and curds are common for Lunch and dinner

Table 3.3 : Exchange List

<i>Day</i>	<i>Breakfast</i>	<i>Lunch</i>	<i>Dinner</i>
Monday	Bread toast Jam Milk	Vanghibath Plain rice	Kichidi Plain rice
Tuesday	Cornflakes with milk	Tamarind rice Plain rice	Curd rice Plain rice
Wednesday	Idli Sambar Chutney	Zeera rice Plain rice	Normal
Thursday	Vermicilli Upma Chutney	Bisi-bella bath Plain rice	Lime rice Plain rice
Friday	Porridge	Palak rice Plain rice	Normal
Saturday	Veg. Sandwich Tomato sauce	Lime rice Plain rice	Curd rice Plain rice
Sunday	Tomato Kichidi	Til rice Plain rice	Normal

PRACTICAL ASSIGNMENT

1. Plan cycle menus for two special diets.

Chapter 4

Assessment of Nutritional Status

Nutritional assessment is used to

- Identify malnourished patients.
- Identify patients at risk or becoming malnourished.
- Provide data to serve as a basis for planning nutritional support to correct or prevent malnutrition.
- Provide information for effectiveness of nutritional support.

Who should be nutritionally assessed?

A. Conditions suggesting nutritional risk.

1. Inadequate nutrient intake.
2. Inadequate nutrient absorption.
3. Decreased nutrient utilisation.
4. Increased nutrient losses.
5. Increased nutrient requirements.

B. Those whose preliminary assessment show the following:

1. Serum albumin less than 3.2 g/dl.
2. Total lymphocytes less than 1500 mm³.
3. Nonvoluntary weight loss.
4. History of nutritional deficiency.
5. Statement from client indicating change in appetite.

4.1 METHODS OF NUTRITIONAL ASSESSMENT

Most of the information important to the nutritional assessment of the client will be obtained by careful evaluation of five major areas namely.

- Dietary/Nutrient data.
- Anthropometric measurements.
- Biochemical data—Laboratory values and results of organ function tests, X-rays, MRI scans, blood pressure etc.
- Clinical or physical signs.
- Economic, cultural, social and psychological factors.

The information on nutritional assessment is obtained by several members of the health care team. Dietitian is mainly responsible for collecting dietary/nutrient intake information. However, the dietitian should become

familiar with physical measurement values, as well as with the results of laboratory, organ function tests and the physical appearance of the client.

4.2 DIETARY/NUTRIENT DATA

Several factors affect food intake and nutrient utilization. Factors affecting food intake—Social, psychological, economic, cultural factors, immobility and excessive anorexia, cachexia, taste and odour, drugs, vomiting, mastication etc.

Factors affecting nutrient utilization— Swallowing, dysphagia, digestion, absorption, metabolism, immobility, organ dysfunction, radiation therapy, drugs, hormones etc.

In general, nutrients are obtained from foods, thus it is necessary to obtain information on food intake. Several methods can be used to obtain such information. These include:

- Dietary history.
- Dietary recall (24 hour recall).
- Food diary usually 3, 5 or 7 days.
- Food frequency.
- In a hospital, by checking for food remaining uneaten on tray.
- In the metabolic ward, each portion of food is weighed and uneaten portions are weighed back.
- For groups of people who routinely eat together such as children in a day care centre of hospital ward an assessment of food consumed by the group can be obtained by weighing the food served to the group and subtracting from it, the weight of the food remaining in the serving dishes at the end of the meal and the food discarded in the garbage.

Nutrient Intake

Once one has obtained the information on the foods consumed one of the several methods may be used to determine the nutrient content of foods. The method of choice would depend upon the accuracy desired. The nutrient content can be computed by following methods.

- Chemical analysis of foods.
- Use of Food Composition tables—Data banks, food composition tables, nutrient analysis of foods by industry.
- Through exchange lists.
- Nutritional labelling.
- Calculation of nutrients in food supplements.

For example, a label states that each pill has 80 mg. zinc sulfate. This means each pill has 32.4 mg. Zn.

Sample Calculation

ZnSO ₄	–	161.44 molecular weight
O	–	39.64%
S	–	19.86%
Zn	–	40.50%

<i>Element</i>	<i>Atomic Weight</i>	<i>Valence</i>
Zn	65.38	Zn ⁻²
S	32.06	–
O	16	SO ₄ ⁻²

Zn (65.38) + S (32.06) + O (16 × 4) = 161.44 Mol. Wt.

Zn (65.38/GMW (161.44)) = 0.404980 × 100 = 40.5% Zn

80 mg × 40.5% Zinc = 32.4 mg Zn

Recommended Daily Dietary Allowance

A standard against which nutrient intake can be evaluated is the RDA.

Estimation of Energy Requirements

Basal energy expenditure — (BEE) can be estimated from the Harris Benedict equation (1919).

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

$$\text{For women : BEE} = 655 + (9.6 \times W) + (1.7 \times H) - (4.7 \times A)$$

W = Actual weight in kilograms

H = Height in centimeters

A = Age in years.

Total energy expenditure is estimated by multiplying BEE by the appropriate activity/stress factor as listed below.

Maintenance :

Bed rest : $1.2 \times \text{BEE}$

Ambulatory : $1.3 \times \text{BEE}$

Anabolic : $1.5 \times \text{BEE}$

Stress and Starvation : $1.2 \times \text{BEE} \times \text{Percent change in metabolic activity.}$

4.3 ANTHROPOMETRIC MEASUREMENTS

Common anthropometric measurements currently in use include height, weight, wrist circumferences, mid arm, triceps, fat fold and upper mid arm circumference.

Evaluation of weight change : The importance of any weight change will depend on both the amount of change and the rate of change. The more rapid and larger the weight change, the more likely it is due to changes in water mass rather than to changes in lean body mass or fat. Therefore in evaluation of any weight change you should ascertain whether there was in the past or there is currently any edema or ascites, or any other evidence of fluid retention or loss. Once it has been established that the change is in one of the other body compartments, rapid and marked weight changes are usually associated with increased morbidity and mortality.

Table 4.1 : Evaluation of Weight Change

<i>Time</i>	<i>Significant wt. loss (% of change)</i>	<i>Severe weight loss (% of change)</i>
1 week	1–2	more than 2
1 month	5	more than 5
3 months	7.5	more than 7.5
6 months	10	more than 10

The other important anthropometric measurements after body height and weight are arm circumference (AC), triceps skin fold (TSF) thickness and arm muscle circumference derived from the other two measurements. Unlike height and weight they can be performed on bed ridden patients.

Table 4.2 : Standards for Upper Arm Anthropometry in Adults

<i>Parameter</i>	<i>Male</i>	<i>Female</i>
Triceps skin fold (mm)	12.5	16.5
Mid arm circumference (cm)	29.3	28.5
Mid arm muscle circumference (cm)	25.3	23.5

Measurement of Muscle Mass

Muscle mass can be measured not only by measuring arm muscle circumference but also creatinine height index.

Creatinine Height Index (CHI)

Creatinine is normally formed in an amount proportionate to muscle mass. Its urinary excretion is related to the amount of muscle mass. In patients of ideal body weight the creatinine coefficient is 23 mg/kg body weight for men and 18 mg/kg body weight for women.

Creatinine height is calculated with the following formula.

$$\text{CHI} = \frac{\text{Actual Urinary Creatinine}}{\text{Ideal Urinary Creatinine}} \times 100$$

Table 4.3 : Creatinine Height Index (CHI) Values in Muscle Mass Depletion (Black Burn *et. al.*, 1977)

<i>Muscle mass</i>	<i>CHI</i>
Normal	90 – 100%
Moderate depletion	40 – 90%
Severe depletion	Less than 40%

Factors which render the CHI invalid

- Improper sample collection.
- Decreased renal function.
- Inadequate fluid intake.
- Limb amputation.
- Deviation from ideal body weight.

Factors which increase creatinine excretion

- Severe exercise
- High meat diet
- Catabolic states *e.g.*, fever.

Factors which decrease CHI

- Renal disease.
- Medications.

Body Mass Index

BMI may be used as an estimate of body fatness.

When triceps fat fold measurements are not available, the following equation is used to calculate BMI.

$$\text{BMI} = \frac{\text{Weight}}{\text{Height}^2} \text{ or } \frac{W}{H^2}$$

Where W = Weight in kilograms

H² = Height in meters squared.

A women with a BMI over 27.3 or a man with BMI over 27.8 are at risk for health complications of obesity.

4.4 LABORATORY ASSESSMENT

Laboratory assessment is used primarily to detect subclinical deficiency states.

Subclinical deficiency states can be identified by two methods namely:

- Static biochemical tests
- Functional tests.

Bio-chemical tests

Subclinical deficiency states can be identified by measuring the levels of nutrient or its metabolite in a preselected biopsy material that reflects the total body content of the nutrient or size of the tissue store most sensitive to depletion. These measurements are termed as “Static bio-chemical tests”.

Bio-chemical tests are classified into two groups

- Measurement of a nutrient in biological fluid or tissue.
- Measurement of urinary excretion rate of nutrient.

Body fluid and tissues used

Whole blood, serum, plasma

Hair

Saliva

Amniotic fluid

Finger nails

Skin and buccal mucosa.

Plasma and serum levels tend to reflect recent dietary intake. Near normal plasma/serum nutrient concentration may be present even in the presence of severe depletion of body stores. In such cases alternative bio-chemical indices should be selected.

Erythrocytes

Reflects chronic nutrient status.

The analysis is difficult.

Erythrocytes contain only a small percent of the total body nutrient content.

They are unlikely to be a valid index of nutrient status.

Leukocytes

Leukocytes or specific cell types such as lymphocytes or neutrophils can be used to monitor short term change in nutritional status.

Relatively large blood sample is required; usually restricts the use of these indices to adults only.

Tissue stores

Liver, bone marrow, adipose tissue, are the storage sites for iron, vitamin E and calcium.

These materials are used as biopsy material in research and clinical settings.

4.5 MEASUREMENT OF URINARY EXCRETION RATE OF THE NUTRIENT OR ITS METABOLITE

- Urine specimens can be used for the bio-chemical assessment of some minerals, water soluble B complex vitamins, vit. C and protein provided that the renal function is normal. Urine cannot be used to assess vitamins A, D, E and K as metabolites are not excreted in the proportion to the amounts of these vitamins consumed, absorbed and metabolised.
- Urinary excretion assessment methods almost always reflect recent dietary intake rather than chronic nutritional status. The methods depend on the existence of a renal conservation mechanism.
- The urinary excretion of a nutrient or metabolite is reduced when the body stores of the nutrient is depleted with the exception of ascorbic acid and phosphorous.

In certain conditions like infections, consumption of antibiotics and in condition which produce negative balance, urinary excretion of a nutrient may occur despite depletion of body nutrient stores. 24 hour period urine sample is required for analysis. This approach assumes that daily urinary creatinine excretion is constant for a given individual, the amount being related to muscle mass.

Functional Tests

Some important examples of functional tests are:

- Measurement of abnormal metabolic products in blood or urine arising from suboptimal intakes of the nutrient (increased excretion of Xanthurenic acid in Vitamin B₆ deficiency).
- Measurement of changes in blood components of enzyme activities that are dependent on a given nutrient for example:
 - Erythrocyte glutathione peroxidase activity – Selenium activity
 - Erythrocyte glutathione reductase activity – Riboflavin
 - Erythrocyte glutathione transketolase activity – Thiamine
- In vitro tests of enzyme functions (d-uridine suppression test for Vitamin B₁₂ and folate).
- Induced responses and load tests in vivo (Tryptophan load test for vitamin B₆).
- Growth or developmental responses (e.g. Sexual maturation for zinc, cognitive performance for iron).

Evaluation of Laboratory Indices

Laboratory indices both static and functional are generally evaluated using two techniques namely.

- Comparing the observed values with references/normal values which are derived from a reference sample. Dependent upon the method used for bio-chemical analysis normal values may vary. However tables 7 and 8 contain laboratory values generally considered normal.
- Comparing the observed values with cut-off points based on data from subjects with clinical or functional manipulations of a nutrient deficiency.

The following are some of the methods used to evaluate nutrient status.

Evaluation of Protein Status

Protein status

Urinary creatinine excretion

Creatinine height index

3-Methyl histidine excretion

Visceral protein status

- Serum albumin
- Serum transferrin
- Serum thyroxine binding pre albumin (TBPA)
- Serum retinol binding protein (RBP)

Metabolic changes as indices of protein status

- Serum amino acid ratio
- Urinary 3-hydroxyprotein excretion
- Hydroxyproline creatinine ratio
- Hydroxyproline index
- Nitrogen balance studies

Evaluation of Vitamin Status

Some of the bio-chemical tests used for estimation of vitamin intake and stores are given in Table 4.4.

Evaluation of mineral status

Status with respect to the major minerals (Sodium, Potassium, Calcium and Magnesium) is commonly assessed using serum levels, (Table 4.5). The body normally maintains serum levels of these minerals within narrow limits because they have a very important role in electrolyte balance, nerve and muscle function.

Table 4.4 : Bio-Chemical Tests for Vitamin Intake and Stores

<i>Vitamin</i>	<i>Tests of intake levels</i>	<i>Tests of tissue stores</i>
A	Plasma or serum retinol	Liver retinol
D	-----	25 OHD, 1, 25 OH ₂ D
E	Plasma tocopherols	Erythrocyte fragility test
K	-----	Prothrombin time
C	Serum ascorbate	Leukocyte, Urinary ascorbate load test
Thiamine	Urinary thiamine excretion	Erythrocyte transketolase
Riboflavin	Urinary riboflavin excretion	Erythrocyte glutathione reductase, erythrocyte riboflavin, pyridoxamine oxidase.
Pyridoxine	Urinary Pyridoxine excretion	Tryptophan load test. Erythrocyte transaminase, Plasma pyridoxal phosphate.
B ₁₂	Serum B ₁₂	Serum B ₁₂ , methylmalonic acid excretion.
Folacin	Plasma folacin	Erythrocyte folacin, formino glutamate excretion test.
Niacin	-----	Urinary N-Methyl nicotamide 2-pyridone excretion.

Table 4.5: Assay for Major Mineral Status

<i>Mineral</i>	<i>Assays for functional status</i>	<i>Assay for stores</i>
Sodium	Serum sodium level, urine sodium excretion	In vivo neutron activation analysis
Potassium	Serum potassium level, urine potassium excretion	Serum potassium level
Calcium	Plasma ionized calcium, alkaline phosphate, Vit. D levels	Bone density measurement invivo, neutron activation analysis
Megnesium	Plasma magnesium, urine magnesium excretion	-----
Phosphorous	Plasma phosphate	In vivo neutron activation analysis

Assessment of iron status is closely linked with assessment of hematologic status. Hematology screening includes:

- White blood cell count (WBC)
- Red blood cell count (RBC)
- Hematocrit (HCT)
- Haemoglobin (HGH)
- Differential white cell count (DIFF)
- Mean cell/corpuscular volume (MCV)
- Mean cell/corpuscular haemoglobin (MCH)
- Mean cell/corpuscular haemoglobin concentration (MCHC)

The entire hematologic examinations should be considered in evaluating the significance of nutritional deficiency since dietary factors usually affect more than one aspect of the formed elements of the blood. But under field situation, tests to detect anaemia is restricted to only haemoglobin estimation. Several simplified methods of haemoglobin suitable for field situation have been developed recently ex Wong's method, cyanmethae myoglobin method and filter paper technique. The filter paper technique has been further modified by NIN.

The number of trace minerals considered essential for human nutrition has been growing. Some of the assays for trace minerals are given in table 4.6.

Table 4.6 : Assays for Trace Minerals

<i>Mineral</i>	<i>Intake</i>	<i>Functions</i>	<i>Stores</i>
Zinc	Hair, nail zinc	-----	Plasma zinc
Copper	-----	Presence of iron deficiency like anaemia	Ceruloplasmin, plasma copper, hair levels
Iodine	Thyroid hormone Thyroid stimulating hormone level	Thyroid hormone Thyroid stimulating hormone level	-----
Fluoride	Urinary fluoride level	-----	-----

Contd...

<i>Mineral</i>	<i>Intake</i>	<i>Functions</i>	<i>Stores</i>
Manganese	-----	Serum manganese	-----
Chromium	-----	Change in plasma levels following a meal	Hair levels
Selenium	Plasma selenium	Platelet glutathione peroxidase activity	Red cell selenium Platelet glutathione peroxidase activity.

4.5 CELL MEDIATED IMMUNE FUNCTION

1. Lymphocyte count
1500/mm³ is low normal value
2. Recall antigen skin testing for cell mediated immune response to various infected substances.

Procedure

- Inject antigens intradermally in the fore arm.
- Common antigens are mumps, candida and streptokinase, streptodornase (SK/SD).

Interpretation

- Measure in duration after 48 hours.
- Less than 5 mm for all 3 tests—negative reaction.
- Less than 10 mm—moderate
- More than 10 mm on any 1 test means there is immune competence.
- No induration means patient is allergic.

4.6 PROGNOSTIC NUTRITIONAL INDEX (PNI)

Estimates clinical risk

$$\text{PNI} = 158\% - (16.6 \times \text{ALB}) + (0.78 \times \text{TSF}) + (0.2 \times \text{TFN}) + (5.8 \times \text{DCH})$$

ALB = Serum albumin concentration (g/dl)

TSF = Triceps fat fold (mm)

TFN = Transferrin (g/dl)

DCH = Delayed cutaneous hypersensitivity.

Well nourished patient

$$\begin{aligned} \text{ALB} & 4.7 \text{ g/dl} \times 16.6 = 78.0 \\ \text{TFS} & 15 \text{ mm} \times 0.78 = 11.7 \\ \text{TFN} & 245 \text{ g/dl} \times 0.2 = 49.0 \\ \text{DCH} & 2 \times 5.8 = 110.6 \\ \text{Total} & = 150.3 \\ \text{PNI} & 158 - 150.3 = 7.7\% \end{aligned}$$

Malnourished patient

$$\begin{aligned} \text{ALB} & 2.8 \text{ g/dl} \times 16.6 = 46.5 \\ \text{TSF} & 10.2 \text{ mm} \times 0.78 = 8.0 \\ \text{TFN} & 160 \text{ g/dl} \times 0.2 = 32.0 \\ \text{DCH} & 1 \times 5.8 = 5.8 \\ \text{Total} & = 92.3 \\ \text{PNI} & 158 - 92.3 = 65.7\% \end{aligned}$$

The risk for complication for = $65.7 = 8.5$ times more than normal.

The malnourished patient

than well nourished patient. $\frac{7.7}{}$

Table 4.7 : Interpretation of Nutritional Assessment Values

<i>Observation</i>	<i>Deficit</i>			
	<i>None</i>	<i>Mild</i>	<i>Moderate</i>	<i>Severe</i>
TSF (% of standard)	90	90 – 51	50 – 30	30
AMC (% of standard)	90	90 – 81	80 – 70	70
CHI (% of standard)	90	90 – 81	80 – 71	70 – 60
Serum albumin (mg/dl)	3.5	3.5 – 3.2	3.2 – 2.8	2.8
Transferrin (mg/dl)	200	200 – 180	180 – 160	160
TIBC (Mcg/dl)	214	214 – 182	182 – 152	152
Total lymphocytes (per mm ³)	1501	1500 – 1201	1200 – 800	800

Table 4.8 : Blood and Serum Constituents

<i>Constituent</i>	<i>Normal value</i>
Amino Acids, Total Bicarbonate	35 – 65 mg/dl 24 – 30 m Eq/l
Bilirubin, total serum	0.2 – 1.4 mg/dl
Soluble, (Direct)	0.1 – 0.4 mg/dl
Unconjugated (indirect), serum	0.1 – 0.6 mg/dl
Carbohydrates	6 – 8 mg/dl
Fructose	
Glucose	
Nelson – Somogyi method	70 – 100 mg/dl
Folin – Wu	80 – 120 mg/dl
Glycogen	5 – 6 mg/dl
Carbon dioxide (CO ₂)	24 – 30 m Eq/l
Enzymes	1.4 – 4.1 Units/dl (Bondansky)
Alkaline phosphatase, serum	20 – 48 Im U/ml
Creatinine phosphokinase (CPK or CK), serum	5 – 50 Im U/ml
Lactic dehydrogenase (LDH), serum	40 – 60 Im U/ml
Serum glutamic oxalotransaminase (SGOT) (AST)	6 – 40 Units (Karmen)
Serum glutamic pyruvic transaminase (SGPT) (ALT)	6 – 36 Units/dl (Karmen)

Contd...

<i>Constituent</i>	<i>Normal value</i>
Hematology	
Erythrocytes (RBC)	42.5–5 million/mm
Leukocytes (WBC)	5 – 10 thousand/mm
Lymphocytes (WBC)	1 – 4 thousand/mm
Thrombocytes (platelets)	150 – 300 thousand/mm
Hematocrit (Volume % red cells)	
Men	40 – 54% RBC/vol
Women	37 - 47% RBC/vol
Haemoglobin	
Children (varies with age)	10 – 18 g/dl
Men	14 – 17 g/dl
Women	12 – 15 g/dl
Prothrombin time (Normal Vit. K)	10 – 15 sec.
Volume, blood	70 – 90 ml/kg B.W. or 8% to 9% of body wt.
Lipids	
Cholesterol, Serum	
Esters	
Free	100 – 180 mg/dl, 50 – 70% of total
HDL cholesterol	50 – 60 mg/dl
Children	
Men	55 mg/dl
Women	45 mg/dl
Total cholesterol	55 mg/dl
Fatty acids, serum	150 – 260 mg/dl
Free	
Total	8 – 30 mg/dl
17 – Hydrosycorticoids, plasma	350 – 450 mg/dl
Lipids, serum, total	5 – 25 mg/dl
Phospholipids, serum, total	450 – 850 mg/dl
Triglycerides	230 – 300 mg/dl
	140 – 300 mg/dl
Minerals	
Calcium, serum	9 – 11 mg/dl
Chlorides, serum	100 – 106 mEq/L
Iron, serum	70 – 140 mcg/dl
Magnesium, serum	75 – 175 mcg/dl
Phosphate	2–3 mg/dl or 1.6 – 2.4 mEq/L
Phosphorous, inorganic, serum	1.6 – 2.7 mEq/l
Potassium, serum	3 – 4.5 mg/dl
Sodium serum	16 – 20 mg/dl or 4 – 5 mEq/L

Contd...

<i>Constituent</i>	<i>Normal value</i>
Zinc	310 – 340 mg/dl or 136 – 145 mEq/L 90 – 110 mg/dl
Nitrogen	
Amino acid nitrogen, blood	
Ammonia, blood	4 – 8 mg/dl
Ammonia, plasma	75 – 196 mcg/dl
Ammonia, serum	56 – 122 mcg/dl
Creatine	0.15 – 0.30 mg/dl
Creatinine	0.2 – 0.9 mg/dl
Nonprotein nitrogen (NPN), blood	0.8 – 2 mg/dl
Phenylalanine	20 – 40 mg/dl
Taurine	0.7 – 1.0 mg/dl
Urea nitrogen (BUN), blood	0.4 – 0.8 mg/dl
Urea nitrogen (SUN), serum	8 – 20 mg/dl
Urea, blood	8 – 20 mg/dl
Uric acid	17 – 42 mg/dl 2.5 – 6 mg/dl
Organic acids	
Acetoacetic acid	0.8 – 2.8 mg/dl
Acetone, serum	0.3 – 2 mg/dl
Alpha-ketoglutarate	0.2 – 1.0 mg/dl
Beta-OH butyric acid	0.02 mM/L
Lactic acid	8 – 17 mg/dl
Pyruvic acid	0.4 – 2 mg/dl
Osmolality, serum	280 – 290 Mosm/kg plasma water
pH	7.35 – 7.45 pH
Protein	
Albumin, serum	3.5 – 5.5 g/dl
Albumin, globulin ratio	1.8 – 2.5
Globulin	1.3 – 2.7 g/dl
Fibrinogen, plasma	0.2 – 0.4 g/dl
Iron binding capacity, serum	250 – 450 mcg/dl
Transferrin	170 – 250 mg/dl
Transferrin saturation > 12 yr, male	≥ 20%
Transferrin saturation > 12 yr, female	≥ 15%
Vitamin B ₁₂	350 – 750 mg/dl
Vitamin E, tocopherol, serum	
Adults	1.0 – 1.2 mg/dl
Infants	0.23 – 0.43 mg/dl
Volume	8 to 9% of body Wt.

Table 4.9 : Normal Value of Urine and Stool Constituents

<i>Constituent</i>	<i>Normal value</i>
Urine	
Acetone (Ketones)	0.003 – 0.015 g/24 hr.
Ammonia	0.4 – 1.0 g/24 hr.
Bile	20 – 70 mEq/L.
Color	Straw
Creatine	0
Creatinine	1.0 – 1.5 g/24 hr.
Inorganic	
Calcium	0.1 – 0.3 g/24 hr.
Chloride (as NaCl)	10 – 15 g/24 hr.
Potassium	1.5 – 2.5 g/24 hr.
Sodium	2.0 – 5.0 g/24 hr.
Nitrogen, total	10 – 17 g/24 hr.
pH	5.5 – 8.0 pH
Protein	0 – 0.015 g/24 hr.
Specific gravity	1.003 – 1.030
Urea	20 – 35 g/24 hr.
Uric acid	0.5 – 0.8 g/24 hr.
Volume	1000 – 1500 ml/24 hr.
Stool	
Color	Brown, light brown on high milk diet, dark brown on high meat diet, black-blood present on high iron intake.
Bulk	
Drymatter	100 – 200 g/24 hr.
Protein	23 – 32 g/24 hr.
Fat, total	Minimal
Nitrogen	17.5 – 30% of dry matter 1.7 g/24 hr.
Moisture	Approximately 65%
Urobilinogen	40 – 280 mg/24 hr.

4.7 CLINICAL ASSESSMENT

Clinical assessment consists of a routine medical history and a physical examination to detect physical signs (*i.e.*, observation made by a qualified examiner) and symptom (*i.e.*, reported by the patient). Many of the critical physical signs are non-specific and must therefore be interpreted in conjunction with laboratory, anthropometric and dietary data before specific nutritional deficiencies can be identified.

Medical History

In clinical medicine, the medical history can be obtained by an interview with the patient and/or from the medical records. Two types of medical records are commonly used.

- Source oriented medical record (SOMR)
- Problem oriented medical record (POMR)

The SOMR consists of patient identification data, admission notes, physician orders, laboratory reports, medication records, progress notes and operating room records. The POMR consists of a defined data base, a complete problem list, the initial care plan and progress notes, as well as flow sheets and a discharge summary.

Physical Examination

The physical examination as defined by Jelliffe (1996) examiner, “Those changes, believed to be related to inadequate nutrition that can be seen or felt in superficial epithelial tissue, especially the skin, eyes, hair and buccal mucosa or in organs near the surface of the body”. A list of physical signs indicative or suggestive of malnutrition is presented in Table 4.10

Classification and Interpretation of Physical Signs

The World Health Organisations has classified, the most physical signs into three groups.

1. Signs indicating a probable deficiency of one or more nutrients.
2. Signs indicating probable long term malnutrition in combination with other factors.
3. Signs not related to nutritional status.

To assist in the interpretation, the physical signs are often combined into groups associated with a particular nutrient deficiency state or a system. The signs within group are often classified into three risk categories designated as high, moderate and low risk.

Q. What are the limitation of physical examination?

Table 4.10 : Physical Signs in Nutrient Deficiency and the Possible Mechanism of their Formation

<i>Deficiency</i>	<i>Physical signs</i>	<i>Mechanisms for signs</i>
Calories (Marasmus)	Sunken temples, prominence of bone structure, loss of muscle strength, sunken eyes.	Loss of muscle mass. Subcutaneous and retro-orbital (= behind the eye) fat.
Protein (kwashiorkar)	Hair dull and dry, easily pluckable dry, thin, scaly skin (flaky paint dermatosis); loss of muscle mass; poor wound healing.	Inadequate protein for new tissue growth
	Edema	Inadequate plasma protein levels; inappropriate growth hormone and corticosteroid secretion; increased capillary permeability.
	Parotid enlargement	Inappropriate growth hormone and corticosteroid secretion.
	Enlarged liver and spleen	Poor liver protein production, transport, and release.
	Apathy or irritability	Inadequate fuel supply to the brain; amino acid and electrolyte imbalance.
Essential fatty acids	Scaly skin lesions; rough, dry skin; poor wound healing	Inadequate essential fatty acid supply for membrane, prostaglandin and leukotriene formation leading to poor skin growth and repair.
Vitamin A	Dry skin; xerophthalmia (= dry eyes); Bitot's spots (= piling up of epithelium near cornea); keratomalacia (= softening of the cornea); follicular hyperkeratosis (= sand paper skin).	Abnormal growth of epithelial cells with conversion from columnar to undifferentiated squamous (= flat) cells, and failure to develop receptors for epidermal growth factor.
	Night-blindness	Failure to form rhodopsin.
Folate	Atrophy of tongue papillae (tongue appears to be shiny and red).	Decreased rate of tissue renewal due to poor formation of DNA.
Vitamin B ₁₂	Atrophy of tongue papillae.	Decreased rate of tissue renewal due to poor formation of DNA.
	Loss of sensation in hands and feet.	Decreased ability of nervous tissue to form neurotransmitters (e.g., epinephrine).
Thiamin	Beri-beri: Heart enlargement and failure; neurological changes (sensory loss, loss of reflexes, burning sensations in hands	Defective delivery of energy to tissues due to defective carbohydrate metabolism (failure to metabolize pyruvate).

Contd...

<i>Deficiency</i>	<i>Physical signs</i>	<i>Mechanisms for signs</i>
Biotin	and feet); muscle tenderness and loss of muscle strength; Wernick-Korsakoff syndrome. Scaly skin lesions; rough dry skin.	Poor metabolism of essential fatty acids: poor metabolism of phenylalanine.
Riboflavin	Abnormalities of the skin where the skin and mucous membrane meet: redness and fissuring of the corners of the eyelids, the mouth, the vulva and the anus, scalling around the nostrils and scrotum; deep red beefy tongue.	Abnormal oxidation — reduction function leading to poor tissue repair at sites of mechanical and chemical stress sites of constant new tissue generation.
Niacin	Pellagra (redness and scaling where the sun damages the skin); weakness, anorexia diarrhoea, mental changes (confusion and depression); scarlet raw tongue.	Inability to repair damaged tissue, especially DNA repair. Note that the metabolic abnormalities in niacin deficiency have been difficult to define.
Pyridoxine	Redness and fissuring around eyes; pellagralike picture. Depression	Impairment of intermediate metabolism; the function of other vitamins (niacin, is dependent on pyridoxine) affected. Failure to decarboxylate glutamic acid; failure to transaminate amino acids.
Vitamin C	Scurvy: Bleeding in skin (= petechiae) and gums; bone pain due to bleeding under the periostem; swelling at the ends of bones, giving (among other signs) a 'rosary' consisting of swellings at the points where the bony parts of the ribs join the cartilage.	Impaired collagen formation with difficulty in regenerating blood vessel walls (note that in daily life we are constantly having microtears in blood vessel walls that are constantly and rapidly repaired.
Vitamin D	Bone softening, swelling at the growing ends of long bones, rachitic rosary, Harrison's groove (where the diaphragm is attached to the ribs and draws, the softened ribs inwards), softening and bulging of the bones of the skull, and in an infant failure to close the fontanelles; knock knees and bowed legs.	Failure to absorb calcium and to form well structured new bone.
Vitamin E	Neurological changes consistent with a loss of spinal cord function; ceriod (wax-like) pigment deposition in smooth muscle destruction.	Failure of antioxidant function.
Vitamin K	Bleeding	Failure to form clotting factors.

Contd...

<i>Deficiency</i>	<i>Physical signs</i>	<i>Mechanisms for signs</i>
Copper	Pallor Skeletal deformities Hair with abnormal pigmentation.	Anaemia due to failure to form haemoglobin. Poor bone formation due to poor bone mineralization. Poor formation of extracellular tissue matrix.
Iron	Blue sclerae (= whites of the eyes), pallor especially of the conjunctivae. Koilonychiae (= spoon nails)	Failure to form haemoglobin.
Zinc	Infections of the skin and mucous membranes; diarrhoea Hypogonadism Growth failure: alopecia (= hair loss) Anorexia, hypogeusia (= loss of taste) Pallor	Failure to form proper keratin. Depressed cell mediated immunity. Lack of active enzyme for testosterone formation. Failure of active enzyme formation for new tissue growth. Inability to form active taste buds.
Selenium	Night-blindness Cardiomyopathy (= disease of the heart muscle).	Iron-deficiency type anaemia, due to lack of formation of porphyrins. Poor conversion of vitamin A to active forms.
Molybdenum Iodine	Tachycardia (= rapid heart rate), tachypnea (= rapid breathing rate), neurological disturbances. Lethargy; dry skin with abnormal doughy consistency; sparse hair with coarse texture; slowness of thinking. Enlargement of the thyroid gland.	Failure to maintain intracellular redox potential in tissue with high oxidative phosphorylation rates. Sulfur amino acid intolerance, due to lack of active sulfite oxidase. Lack of thyroid hormone. Excess accumulation of non-iodinated thyroid hormone precursor and excess stimulus for thyroid hormone to the pituitary, so excess thyroid stimulating hormone is released.

Disorders of nutrient excess or of abnormal metabolism may lead to characteristic physical signs. Table 4.11 list some of these signs.

Table 4.11 : Physical Signs of Nutrient Excess

<i>Nutrient in excess</i>	<i>Physical signs</i>	<i>Mechanisms for signs</i>
Vitamin A (acute)	Nausea, vomiting, confusion: on ophthalmological examination, the optic nerve appears pushed into the globe (= papilledema)	Increase intracranial pressure due to fluid and electrolyte imbalance.
Vitamin A (chronic)	Dry scaly rough skin, loss of body hair, brittle nails, mouth fissures Fatigue, lethargy, emotional inability, insomnia, severe throbbing headaches, exophthalmos (= bulging eyes), peripheral edema Bone and joint pain	Change in growth patterns of epithelial tissue. Difficulty in maintaining fluid and electrolyte balance leading to cerebral and peripheral edema. Abnormal calcification under the periosteum (= membrane that surrounds the bone).
Vitamin A (chronic)	Abdominal pain, constipation	Disordered gut motility due to abnormal fluid and electrolyte balance.
Vitamin D	Anorexia, nausea, vomiting, polyuria (= excess urination), polydipsia (= excess drinking), weakness	Hypercalcemia activation of brain receptors for vitamin D.
Hypercarotenemia	Yellow-orange coloring of the skin (not of eye)	Deposition in the skin excess carotene.
Fluorosis	Mottled teeth enamel	Poor formation of enamel due to excessive deposition of fluorids.

Table 4.12 : Physical Signs of Some Nutrition Related Disorders

<i>Disorder</i>	<i>Physical signs</i>	<i>Mechanisms</i>
Hyperlipidemia	Corneal arcus (= white deposits along the edges of the cornea); xanthomas (= flat to slightly raised deposits of lipid under the skin); xanthelasma (= raised yellow deposits of lipid around the eyes).	Deposition of excess lipid in inappropriate places.
Alcoholic cirrhosis	Ascites (= accumulation of fluid in the abdominal cavity); Red palms and soles; spider hemangiomas (= spider like arrays of tiny blood vessels); Jaundice or icterus (= yellowing of the skin mucous membranes and whites of eyes)	Fluid and electrolyte retention, decreased ability to maintain fluid in the vascular bed. Inability to metabolize estrogens. In ability to conjugate and excrete bile salts, with deposition in the skin and other membranes.

There are numerous conditions observable on physical examination which affect food consumption and assimilation. These include not only problems associated with the gastrointestinal tract, but also problems with neuromuscular function and with sensory perception. Table 4.13 is a partial compilation of physical findings which may be associated with impaired food consumption.

Table 4.13 : Signs and Symptoms Associated with Impaired Food Consumption

<i>Function</i>	<i>Sign/Symptom</i>
Vision Arms and hands	Blindness; restricted visual fields. Tremor; inability to recognize objects placed in hands; weakness; lesions on hands sufficient to prevent grasping; enlarged joints due to arthritis; stiffness for contractures.
Torso	Inability to sit up; swaying or instability on sitting up.
Respiration	Rapid or difficult breathing.
Head	Inability to maintain head erect; tremor.
Mouth	Cracked or fissured lips; poor dentition; dentures; lesions elsewhere in the mouth; tongue tremor and/or incoordination, paralysis of weakness.
Jaw	Pain or difficulty on moving jaw.
Swallowing	Pain or difficulty on swallowing.

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Chapter 5

Exchange Lists for Meal Planning

5.1 FOOD EXCHANGE LISTS

Exchange is a list of foods grouped together according to similarities in food values. In food exchange system the foods are divided into 6 main groups based on their nutrient composition. Each exchange group consist of foods in different amounts but provide equal amounts of nutrients so that any food item within a given exchange list can be substituted, thus making the diet planning simple, flexible and more practical.

The exchange system helps in planning a nutritionally balanced diet by choosing adequate number of exchanges from each group. The selection of foods from vegetable and fruit exchanges should be given importance in order to meet the vitamin A and C requirements. The six food exchange lists included in this system are (Table 5.1).

- Milk exchange list
- Meat exchange list
- Pulse exchange list
- Cereal exchange list
- Vegetable A exchange list
- Vegetable B exchange list
- Fruit exchange list
- Fat exchange list

Table 5.1 : Comprehensive Food Exchange List

<i>Exchange</i>	<i>Amount (g)</i>	<i>Measure of raw food</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Carbohydrate (g)</i>	<i>Fat (g)</i>
Milk	250	1 cup	170	8	12	10
(a) Meat	40	2 pieces/1 egg	70	7	Neg.*	5
(b) Pulse	30	3 tb.sp.	100	7	17	Neg.
(a) Vegetable A	100	1/2 cup	Neg.	Neg.	Neg.	Neg.
(b) Vegetable B	100-150	1/2 cup	40	2	7	Neg.
Fruit	80-100	1 portion	40	Neg.	10	Neg.
Cereal	20	3 tb.sp.	70	2	15	Neg.
Fat	5	1 tsp.	45	-	-	5

* Neg. – Negligible

MILK EXCHANGE LIST

The milk exchange list includes milk from common sources and a variety of milk products. Each exchange of milk provides 8 g protein, 12 g carbohydrate, 10 g fat and 170 K.cal.

Table 5.2 : Milk Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure</i>	<i>Energy (kcal)</i>	<i>Carbohydrate (g)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Calcium (mg)</i>
Milk buffalo	185	1 cup	210	9.3	8	12.0	388
Milk cows	250	1 1/4 cup	168	11.0	8	10.3	300
Curds	258	1 1/3 cup	155	7.7	8	10.3	384
Butter milk	1000	5 cup	150	50.0	8	17.0	300
Skimmed (low fat) milk	320	1 1/2 cup	120	12.0	8	5.0	384
(very low fat)	320	1 1/2 cup	93	14.7	8	–	384
Whole milk powder	31	2 tb.sp.	154	11.8	8	8.0	294
Skimmed milk powder	21	1 1/2 tb.sp.	75	10.7	8	–	287
Khoa	55	3 1/2 tb.sp.	231	11.3	8	17.2	231
Cheese	33	2 1/4 tb.sp.	115	2.1	8	8.3	260

MEAT EXCHANGE LIST

The meat exchange list includes meat, fish, poultry, egg and other commercial products rich in protein. Each meat exchange provides 7 g protein, 5 g fat, 70 K. cal and negligible carbohydrate.

Table 5.3 : Meat Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Energy (K. cal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>
Egg, hen	53	138	7	7.0
Egg yolk	39	81	7	11.3
Egg white	58	26	7	–
Fowl	27	29	7	0.2
Goat meat	32	38	7	1.2
Goat liver	35	38	7	1.1
Sheep liver	35	53	7	2.6
Mutton muscle	38	73	7	5.1
Pork	37	43	7	5.1
Prawn	37	32	7	0.4
Rohu	47	41	7	0.6
Katla	35	39	7	0.8
Beef	52	162	7	1.2
Crab	63	106	7	6.1

PULSE EXCHANGE LIST

The pulse exchange list includes pulses like bengal gram, green gram, black gram etc. Soyabean and its products are included in this exchange and each exchange provides approximately **7 g protein, negligible fat, 17 g carbohydrate and 100 K. cal.**

Table 5.4 : Pulse Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure (tb. sp.)</i>	<i>Energy (K. cal)</i>	<i>Carbohydrate (g)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Phosphorous (mg)</i>
Bengal gram whole	41	2 ³ / ₄	148	25	7	2.2	127.92
Green gram whole	29	2	197	16	7	1.8	94.59
Green gram dhal	28	2	101	17	7	0.3	113.40
Black gram dhal	29	2	121	17	7	0.4	111.65
Bengal gram dhal	34	2 ¹ / ₄	125	20	7	0.4	178.74
Red gram dhal	31	2	104	18	7	0.5	94.24
Soyabean	16	1	78	34	7	3.2	110.40
Rajmah	33	2 ¹ / ₄	115	20	7	0.4	135.30
Lentil	29	2	95	17	7	0.2	84.97
Peas (green)	100	6 ¹ / ₂	93	16	7	0.1	139.00
Peas (dry)	35.7	2 ¹ / ₂	112.5	20	7	0.3	107.00

CEREAL EXCHANGE LIST

Cereal exchange list includes cereals, bread products, some high carbohydrate vegetables like potato, colocasia and yam. One cereal exchange provides **15 g of carbohydrate, 70 K. cal, 2 g of protein and negligible fat.**

Table 5.5: Cereal Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure (tb. sp.)</i>	<i>Energy (K. cal)</i>	<i>Carbohydrate (g)</i>	<i>Protein (g)</i>	<i>Sodium (mg)</i>	<i>Potassium (mg)</i>
Rice	19	1 1/4	66	15	1.3	–	–
Rice flakes	20	4	65	15	1.3	2.18	30.8
Rice puffed	20	5	66	15	2.1	–	–
Wheat	21	2	69	15	2.3	3.5	56.8
Wheat flour	21	3	75	15	2.7	4.4	69.3
Wheat flour (refined)	21	3	73	15	2.3	1.95	27.3
Wheat semolina	20	1 1/2	69.6	15	2.0	4.2	16.6
Bread white	29	2	69	15	2.3	–	–
Bread brown	31	2	76	15	2.7	–	–
Bajra	22	2	79	15	2.6	2.39	67.5
Barley	22	1 1/2	72	15	2.5	2.53	–
Jowar	21	1 1/2	73	15	2.1	1.51	27.5
Maize (dry)	23	1 1/2	79	15	2.5	3.65	65.7
Ragi	21	1 1/2	69	15	1.5	2.31	85.6
Vermicelli	19	1 1/4	67	15	1.7	1.5	26.2
Oat meal	24	1 1/2	90	15	3.3	–	–
Biscuits (salt)	28	3	150	15	1.8	–	–
Biscuits (sweet)	21	2 1/2	96	15	1.3	–	–
Cake (plain)	31		88	15	1.0	–	–
Cake (sponge)	25		67	15	1.5	–	–
Colocasia	71	8 1/2 medium	69	15	2.1	6.39	390.5
Potato (sweet)	53	1 1/3 medium	64	15	0.6	4.7	208.29
Potato	66	1 1/4 big	64	15	1.0	0.6	163.02

VEGETABLE A EXCHANGE LIST

The vegetable A exchange list includes vegetables which contain 3% carbohydrate. These vegetables contribute some essential nutrients such as carotene, vitamin C and iron. Each exchange provides **negligible amounts of carbohydrate, protein and fat.**

Table 5.6 : Vegetable A Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure</i>	<i>Energy (K.cal)</i>	<i>Carbohydrate (g)</i>	<i>Iron (mg)</i>	<i>Fibre (g)</i>	<i>Carotene (ug)</i>	<i>Vitamin (mg)</i>
Bottle gourd	100	1 1/4 cup	12	2.5	0.91	0.6	0	0
Cucumber	100	1 medium	13	2.5	0.6	0.4	0	7
*Lettuce	100	1 small bundle	21	2.5	2.4	0.5	990	10
**Spinach	100	1 bundle	26	2.9	1.14	0.6	5580	28
Snake gourd	100	1 cup	28	3.3	1.51	0.8	96	0
Radish white	100	2 medium	17	3.4	0.4	0.8	3	15
Ridge gourd	100	1 cup	17	3.4	0.39	0.5	33	5
Ash gourd	100	1 1/4 cup	10	2.6	0.8	0.8	0	1
**Mustard leaves	100	–	34	3.2	16.3	0.8	2622	33
**Radish leaves	100	1 bundle	28	2.4	0.09	1.0	5295	81

* Rich in vitamin A.

** Rich in vitamin A and C.

VEGETABLE B EXCHANGE LIST

The vegetable B exchange list includes vegetables which contain 7% carbohydrate. Some vegetables with 20% carbohydrate such as potato, colocasia, etc., are included in the cereal exchange. Each exchange provides 7 g carbohydrate, 2 g protein, 40 K. cal and negligible fat.

Table 5.7 : Vegetable B Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure</i>	<i>Energy (K. cal)</i>	<i>Carbohydrate (g)</i>	<i>Protein (g)</i>	<i>Fibre (g)</i>	<i>Iron (mg)</i>	<i>Carotene (ug)</i>	<i>Vitamin C (mg)</i>
*Amaranth	115	3/4 bundle	52	7	4.6	1.26	26.0	*3983.6	**37.95
Beet root	80	1 1/4 medium	34	7	1.4	0.72	0.95	–	8.0
**Bitter gourd	167	2 cups	42	7	2.7	1.33	1.018	215.43	*160.32
Brinjal	175	3 1/2 medium	42	7	2.5	2.27	0.66	129.5	21.0
Broad beans	97	1 cup	47	7	4.4	1.94	1.35	8.73	11.64
*Cabbage	152	1 1/4 cup	41	7	2.7	2.5	1.216	182.4	*188.48
Capsicum	163	4 big	39	7	2.1	–	–	–	–
Carrot	66	2 medium	32	7	0.6	1.79	0.67	1247.4	1.98
Cauliflower	175	1 medium	53	7	4.5	2.1	2.1	52.5	98.0
*Colocasia leaves	103	1/2 bundle	58	7	4.0	5.07	17.5	*10586.34	12.36
**Drum sticks	192	1 big	50	7	4.8	9.2	0.34	211.2	*230.4
*Coriander leaves	111	8 bundles	49	7	3.7	1.33	1.55	*7678.98	149.85
Fenugreek leaves	117	4 1/2 bundles	57	7	5.1	1.28	2.25	2737.8	60.84
French beans	156	1 1/2 cup	41	7	2.6	2.8	–	205.92	37.44
*Knol-khol	184	3 medium	39	7	2.0	2.76	2.76	38.64	*156.4
Ladies finger	109	9 – 11 big	38	7	2.0	1.3	0.38	56.68	14.17
Mint leaves	121	2 1/4 bundles'	58	7	5.8	2.42	17.47	1960.2	32.67
Onion, small	56	2	33	7	1.0	3.36	6.72	8.4	1.12
Tomato	194	5 1/4 small	39	7	1.7	1.35	3.49	372.48	60.14
Turnip	113	4 medium	33	7	0.6	1.01	4.52	0	48.59

* Rich in vitamin A.

** Rich in vitamin C.

FRUIT EXCHANGE LIST

The fruit exchange list includes fruits which are locally available and commonly consumed. The amount per exchange has been calculated taking 10 g of carbohydrate as constant. Vitamin C content for each fruit exchange is provided as they are rich in the vitamin. Each exchange provides **10 g carbohydrate, 40 K. cal and negligible protein and fat.**

Table 5.8 : Fruit Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure</i>	<i>Energy (K. cal)</i>	<i>Carbohydrate (g)</i>	<i>Vitamin C (mg)</i>
Amla	74	16 medium	42	10	444.00
Apple	75	1 small	44	10	0.75
Banana	37	1/2 medium	42	10	2.59
Cherries	72		46	10	5.04
Dates	30	6 – 8	43	10	0.9
Grapes	61	5 – 8	41	10	0.61
Guava	80	1 1/4 big	45	10	169.6
Nimbu	92	1 medium	54	10	35.8
Mosambi	107	3/4 medium	46	10	53.5
Mango, ripe	59	1/2 small	44	10	9.44
Muskmelon	286	1/2 medium	49	10	74.36
Orange	92	1/2 medium	44	10	27.6
Papaya	139	1/4 medium	44	10	79.23
Pineapple	93	1 1/2 slice (round)	43	10	36.27
Pomegranate	69	1/2 medium	44	10	11.04
Raisins	13		41	10	0.13
Sapota	47	1/2 medium	46	10	2.82
Watermelon	303	2 big pieces	49	10	3.03

FAT EXCHANGE LIST

The fat exchange list includes all the oils and hydrogenated fats used for cooking purpose, commercial products such as margarine, mayonnaise and nuts and oil seeds. Each fat exchange which amounts to 5 g (1 t.sp.) provides **45 K. cal, 5 g of fat and negligible carbohydrate and protein.** Nuts contribute little protein.

Table 5.9 : Fat Exchange List

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure (t. sp.)</i>	<i>Energy (K. cal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>
Cooking oil	5	1	45	0	5
Hydrogenated oil	5	1	45	0	5
Ghee	5	1	45	0	5
Butter	6	1 1/4	45	0	5
Coconut, fresh	12	2 1/2	53	0.5	5

Contd..

<i>Foods</i>	<i>Amount (g)</i>	<i>Measure (t. sp.)</i>	<i>Energy (K. cal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>
Coconut, dry	8	1 1/4	53	0.3	5
Groundnuts, roasted	13	2 1/2	74	3.4	5
Gingelly seeds	12	2 1/2	58	2.2	5
Almonds	8	1 1/4	53	1.7	5
Walnut	8	1 1/4	55	1.3	5
Cashewnuts	11	2 1/4	66	2.3	5
Mayonnaise	6	1 1/4	45	Neg.	5
Bacon	10	2	60	3.5	5
Cream (light 20%)	25	5	45	Neg.	5
Cream 40%	15	3	45	Neg.	5

Table 5.10 : Approximate Nutrient Value of Common Cooked Foods

<i>Item</i>	<i>No. of servings</i>	<i>Weight of cooked preparation</i>	<i>Energy (K. cal)</i>	<i>Fat (g)</i>	<i>Protein (g)</i>
I. Cereal Preparations					
Rice	1 K	100	110	0.1	2
Idli	1	60	75	0.1	2
Plain dosa	1	40	125	3	3
Masala dosa	1	100	200	6	4
Phulka	1	35	80	0	3
Paratha	1	50	150	4	4
Upma	1 K	130	200	9	5
Sevian upma	1 K	80	130	4	3
Bread toasted	2 slices	50	170	1	4
Dhalia	1 K	140	165	7	5
Kichidi	1 K	100	210	7	4
Puri	1	25	80	3	2
II. Pulse Preparations					
Plain dhal	1 K	140	170	4	10
Sambar	1 K	160	81	2	4
Chole/Sundal	1 K	150	115	5	7
III. Vegetable Preparations					
With gravy	1 K	130	130	7	3
Dry	1 K	100	115	7	2
Bagara Baigan	1 K	170	230	20	3
Vegetable kofta	1 K	145	220	14	4

Contd...

<i>Item</i>	<i>No. of servings</i>	<i>Weight of cooked preparation</i>	<i>Energy (kcal)</i>	<i>Fat (g)</i>	<i>Protein (g)</i>
IV. Fried Snacks					
Bhaji	1	7	35	3	0.5
Samosa	1	65	210	12	2
Kachori	1	45	200	12	3
Potato bonda	1	40	100	5	3
Sago vada	1	30	100	6	1
Masala vada	1	20	56	2	2
Vada	1	20	65	3	3
Dahi vada	1	80	190	9	5
Vegetable cutlet	1	30	70	5	1
V. Chutneys					
Coconut/groundnut/ til/coriander	1 tb. sp.	25	64	5	3
Tomato	1 tb. sp.	20	10	0.3	0.3
VI. Non-Vegetarian Preparations					
Boiled egg	1	50	86	7.0	7.0
Omelette	1	65	155	14.0	7.0
Fried egg	1	50	155	14.0	7.0
Mutton curry	1 K	145	240	18	10.0
Chicken curry	1 K	125	260	15	26.0
Fish (fried)	2 pieces	85	220	12	18.0
VII. Bakery Products					
Biscuit	2	40	220	14	3
Cake	1	40	220	13	3
Vegetable puff	1	60	170	10	3
VIII. Sweets					
Laddu, burfi, etc.	1	60	250	15	8
Halwa (suji)	1 K	130	430	20	3
Double ka meetha	1 K	105	280	18	4
Custard/Puddings	1 K	110	180	6	5
Chikki	2	60	300	12	8
Jam/Jelly	1 tsp.	7	20	0.04	0.04

Ghafoorunissa and Kamala Krishnaswamy (1995) Diet and Heart disease, National Institute of Nutrition, Hyderabad.

FREE FOODS

A free food is any food or drink which contains less than 20 calories per serving. These foods can be taken in liberal amounts in two or three servings throughout the day, these foods include:

- Green leafy vegetables
- Tomato
- Radish
- Cucumber
- Cabbage
- Gourds (All)
- Lime
- Clear soups
- Butter milk
- Black tea and coffee
- Plain soda
- Coffee/Tea
- Carbonated water
- Mineral water
- Sugar substitutes

The food exchange lists mentioned above will help in planning appropriate menus for both normal and therapeutic use.

1. Explain the role of food exchange lists in planning of both normal and therapeutic diets.

Chapter 6

Management of Obesity

6.1 OBESITY

Obesity is a disease of multiple etiologies characterized by an excess accumulation of adipose tissue (more than 20% of the desirable weight) and due to enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyperplastic obesity) or a combination of both. The term overweight means a weight in excess of 10% of the average for a given sex, height and age.

Our body contains (body composition)

- The active mass (muscle, liver, heart etc.)
- The fatty mass (fat)
- The extra cellular fluid (blood, lymph etc.)
- The connective tissue (skin, bones connective tissue).

Structurally speaking, the state of obesity is characterized by an increase in the fatty mass at the expense of the other parts of the body. The water content of the body is never increased in case of obesity. Once obesity sets, in the most important thing is to identify the cause of obesity, for every cause of the problem holds the solution in itself.

Causes of Obesity

- Genetic factors
- Psychological factors
- Hormonal imbalance
- Sedentary lifestyle
- Excess consumption of calorie rich foods.

If a person is obese it is very essential to know the hazardous consequences of obesity.

Complications of Obesity

- Menstrual, uterine and ovarian abnormalities
- Cardiovascular hazards
- Gall bladder disease
- Arthritis
- Psychosocial disability
- Surgical and anesthetic risks
- Modest changes in B.P. and blood lipids

- Diabetes mellitus
- Gout
- Accidents
- Low life expectancy

The best recommendation for the control of major chronic and degenerative disorders is obesity control/weight reduction.

6.2 ASSESSMENT OF OBESITY

Although obesity can easily be identified at first sight, a precise assessment requires measurements and reference standards. The most widely used methods are two:

- Anthropometric assessment of growth and body composition.
- Laboratory assessment of body composition.

Anthropometric Assessment of Growth

Body weight: If a person is more than 10% standard weight we call him overweight, more than 20% is obese.

<i>% body weight excess of normal</i>	<i>Degree of obesity</i>
25	Mild
50	Moderate
75	Severe
100	Very severe

Weight/height ratio

$$\text{Body mass index (Quetelets Index)} = \frac{\text{Weight (Kg)}}{\text{Height}^2 \text{ (m)}}$$

Grading of obesity can be done based on BMI

- Grade III : > 40
- Grade II : 30 – 40
- Grade I : 25 – 29.9
- Note obese : < 25

- This index is disadvantageous as this does not distinguish between overweight due to obesity and muscular hypertrophy as it happens in athletes. It is also not a valid index for those under treatment or over 65 years, pregnant and lactating mothers.

Ponderal Index

$$PI = \frac{\text{Height (inches)}}{3\sqrt{\text{Weight (lbs)}}$$

An index less than 13 is associated with increased mortality.

Broca Index

$$\text{Height (cms)} - 100 = \text{Ideal weight (Kg)}$$

Corpulene Index

$$\frac{\text{Actual Weight}}{\text{Desirable Weight}}$$

This should not exceed 2

Waist to Hip Ratio

$$\frac{\text{Waist}}{\text{Hip}} = 0.7 \text{ (normal ratio)}$$

Upper body obesity in men : Quarter than 1.0

Upper body obesity in women : 0.85

Anthropometric Assessment of Body Composition**Assessment of body fat**

Skinfold measurements can be taken to measure body fat.

- Triceps
- Biceps
- Subscapular
- Superiliac

A combination of skinfolds (four skin folds) are particularly advisable for individuals who are undergoing pronounced weight gain.

Laboratory Assessment of Body Composition

Accurate methods for measuring specific components of body composition are available to assess the effects of nutrition intervention on body composition.

Selection of a method to measure body composition depends on its precision and accuracy, the objective of the study, cost, convenience to the subject, equipment and technical expertise required and the health of the subject.

The following methods are used for laboratory assessment of body composition.

- Underwater weighing
- Plethysmography
- Ultrasound technique
- Total body electrical conductivity method
- CT scan
- Estimation of total potassium using 40K
- Estimation of total body water using isotope dilution
- Neutron activation analysis

6.3 MANAGEMENT OF OBESITY

There are three basic components for the reduction of weight:

- Diet
- Exercise
- Behaviour modification

Diet

The main principle involved in planning *low calorie reducing diets* is to cut down the intake of cereals and fats which contribute energy to the body.

The diet should provide less calories than the requirement of the person.

By supplying less energy than what is required the body fat gets metabolized and supply energy.

When body fat is utilized for energy, body weight decreases.

The energy reduction per day depends on how much reduction is required by the person in a particular time.

Example:

Body weight of a person – 70 kg
Ideal weight required – 60 kg

Therefore, 10 kg weight should be reduced and one kilogram of fat gives 9000 K calories. If the energy requirement of the person is 2500 K. cal, cut down to 1500 K. cal so the remaining 1000 K. cal are met by metabolising the body fat. Hence, 1 kg reduction in body weight can be achieved in 9 days.

Calories

The daily calorie requirement could be divided into three categories based on

- 1000 K. cal
- 1200 K. cal
- 1500 K. cal

Protein

Normal recommended dietary allowances.

Usually the low kcal diet contains 45 – 70 gs of protein per day.

Protein will be around 20% kcal in a low kcal diet and 13–15% in diets with more kcal.

Fat

Around 30 and not more than 35% of kcal in the low kcal diet should come from fat.

Carbohydrate

Add together the kcal supplied by protein and fat.

Subtract this amount from the total kcal in the diet.

Divide the remaining kcal by 4, the number of kcal per gram of carbohydrate, to determine the grams of carbohydrate per day.

Sample calculations

1. Determination kcal supplied by protein.
 $50 \text{ gs of Pro} \times 4 \text{ cal/g of Pro} = 200 \text{ kcal provided by protein.}$
2. Determination of grams of fat per one day
 $1000 \text{ kcal} \times 30\% = 300 \text{ kcal from fat}$
 $300 \text{ kcal from fat} \div 9 \text{ kcal/g of fat} = 33 \text{ g of fat (around off to 35)}$
3. Determination of grams of carbohydrate per one day
 $200 \text{ kcal} + 300 \text{ kcal} = 500 \text{ kcal provided by protein and fat}$
 $1000 \text{ kcal} - 500 \text{ kcal} = 500 \text{ kcal provided by carbohydrate}$
 $500 \text{ kcal} \div 4 \text{ kcal/g of CHO} = 125 \text{ gs of carbohydrate.}$

The diet prescription is 125 g carbohydrate, 50 g protein, 35 g fat and 1000 kcal.

Tips for diet modification

- Reduce the total fat content in diet
- Reduce/avoid sugar containing dishes and sweets
- Relish large servings of salads
- Prefer whole cereal to refined cereals
- Consume a serving of millets for dietary fibre

- Avoid snacks in between meals
- Exclude pickle in oil
- Stop nibbling in between meals

6.4 EXERCISE

Exercise is not only required for obese people but it must be a daily habit for healthy living. In obese persons sudden beginning of exercise could be taxing. First physician should be consulted to clarify if the person is suffering with any chronic disorders. The exercising schedule should begin with light exercise like walking for about 20 minutes to a moderate level of brisk walking and jogging. Exercise should be done daily. Exercise should not replace a reducing diet. A low calorie diet will be much more effective if it is undertaken with exercise.

6.5 BEHAVIOUR MODIFICATION

It is very important to understand that reduction of weight is not the only criteria but its maintenance is the prime necessity.

Some Techniques Under Behaviour Modification

- Eat regularly in the same place
- Use smaller plates and containers
- Do not watch television or read while eating
- Chew the food thoroughly and take atleast 20 minutes to eat each meal.

PRACTICAL ASSIGNMENT

AIM: To plan a reducing diet for a obese person.

PROCEDURE: Analyse the case history given below critically and prepare SOAP NOTE and nutritional requirements (Energy, Fat, Protein, Carbohydrate) plan a day's menu and calculate the above nutrients.

CASE HISTORY-OBESITY

Mrs. Rita aged 30 is a rich housewife without much work. She has too many servants. She is fond of eating sweets, fried foods, ice creams etc. She is fond of giving parties and attending parties. She spends most of her time by watching T.V. She is a tall lady (5.5"). Her weight is 100 kgs. Her blood cholesterol level is 220 mgs/dl. Her B.P. is 90 mm/130 mm. Fat bulges are seen at the back and near the stomach. She is neither bothered about her condition nor familiar with the complications of her condition. She is relectant to go to a doctor. She thinks that putting on weight is a natural phenomena. She is not interested to modify her dietary habits.

SOAP NOTES ON THE CASE HISTORY

SUBJECTIVE EVALUATION

Name :

Age :

Dietary history

Physical signs

Food intolerances/Allergies

OBJECTIVE EVALUATION

Anthropometric assessment

Biochemical measurements

ASSESSMENT

Modified nutritional requirement

FOOD PLAN

Table 6.1 : Total Exchanges Per Day

<i>Food exchange</i>	<i>No exchange</i>	<i>Appropriation</i>	<i>Energy (K. cal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU

Table 6.2 : Distribution of Exchanges into Meals & Snacks

<i>Food exchange</i>	<i>No exchange</i>	<i>Menu</i>	<i>Energy (K. cal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>
	Breakfast					
	Mid-morning snack					
	Lunch					
	Snacks					
	Dinner					
	Bed time					
Total						

ANSWER THE FOLLOWING QUESTIONS

1. Briefly highlight the theories of obesity.

2. Suggest 10 recipes for a reducing diet giving reasons.

<i>Recipe</i>	<i>Reason</i>

<i>Recipe</i>	<i>Reason</i>

3. What are the various methods used for weight reduction apart from dietary modifications?

4. What are the ill effects of “starvation therapy” used for weight reduction?

Chapter 7

Diet in Diseases of Stomach and Duodenum

Disorders of the gastro-intestinal tract are classified as functional or organic in nature. Functional disturbances involve no alteration in structure. In organic diseases pathologic lesions are seen in tissue as in ulcers or carcinoma. Both disorders are characterized by changes in secretory activity and motility. Number of factors influence these changes.

7.1 FUNCTIONS OF STOMACH

Reservoir

The stomach can hold upto one litre of contents.

Trituration of food

The autrim grinds the food into small particles that are released into duodenum only when the particles size falls to 2 mm or less.

Controlled Release of Ingested Food

In the stomach the food is diluted to an isotonic state before it enters the duodenum.

Gastric emptying is controlled by complex interaction between pressure and resistance.

Osmoreceptors in the duodenum also control gastric emptying.

Gastric emptying also depends on the rate at which calories enter the duodenum.

Liquid meals empty at a rate of 2.3 K. cal/min.

Digestion of Protein

Acid and Pepsin break protein down into peptides. The gastric mucosal barrier prevents autodigestion of the gastric lining.

Intrinsic Factor and R. Protein

In the stomach at an acid pH, R. Protein binds to vitamin B₁₂ forms complex with R-Protein.

The released Vit. B₁₂ at the higher pH of the intestine binds to intrinsic factor.

The ideal mucosa then absorb Vit. B₁₂ from its complex with intrinsic factor.

Physiological Aspects of Stomach

Biochemically important specific secretory products of the gastric juice are.

- Hydrochloric acid secreted by the parietal cells.

- Enzyme–
 1. Pepsinogen secreted by the chief cells and activated to pepsin by the acid which also provides the acid medium required by this enzyme for its action on proteins.
 2. Renine
 3. A weak lipase.
- The haemopoietic factor, the absence of which is responsible for the development of pernicious anaemia.
- Mucus.
- Gastric juice also contains amino acids, histamine and other amines, urea, ammonia and neutral inorganic salts such as chloride.

Secretion of Gastric Juice

Gastric secretion has three phases:

- Psychic or cephalic phase
- Gastric phase
- Intestinal phase.

Psychic or cephalic phase—a nervous mechanism

Mediated through the vagus nerve.

Pleasurable sensations like thought, sight, smell and taste of palatable food evokes secretion.

Inhibition of secretion occurs as a result of worry, anxiety and sight or smell of disagreeable food.

Gastric phase—Mechanical and Chemical stimulation of the gastric mucosa in the pyloric antrum

Gastric mucosa releases gastrin.

Gastrin transmitted by blood stream stimulates secretion of gastric juice.

There is evidence to suggest that Vagus terminals in the stomach wall is involved in the production and liberation of this hormone.

Intestinal phase—Stimulated by certain products of gastric digestion when they enter the duodenum

The intestinal phase is inhibited by fat (before absorption).

The acidity, volume and especially peptic activity of the secretion is lowered.

The factor responsible for this effect has been termed enterogastrone.

Physical characteristics of gastric contents

Amount	– 20–100 ml (average 50 ml) An increase in volume may be due to hypertension or regurgitation from the duodenum.
Colour	– Whitish to colourless Dark brown, dark red or black colour indicates stale blood. Bright red colour would be due to freshly exuded blood. Blood in the residuum may be due to intubation, lesions such as carcinoma of the stomach, portal cirrhosis, peptic ulcer, acute gastritis and bleeding disorders.
Consistency	– It is fluid in nature with a small amount of ropy mucus which is derived from the nasopharynx. Increased amount of sediment indicates retention. Increased quantities of mucus point to inflammation of the stomach.
Organic acids	– Lactic, butyric and other fatty acids may be found in the residuum. Lactic acid may be secreted by the gastric mucosa in carcinoma of the stomach.

Organic acids can also result from stagnation of gastric contents with consequent bacterial action and fermentation.

Free and total acidity – It is expressed as the number of milliliters of tenth-normal sodium hydroxide needed to neutralize 100 ml of gastric contents (megs/litre).

Free acidity ranges from 0–30 with an average of 18.5 megs/litre.

Total acidity includes acidity due to free hydrochloric acid, hydrochloric acid combined to protein, acid salts (phosphates) and organic acids such as lactic acid. Total acidity ranges from 10–50 megs/litre. The average being 30.

Free HCl above 30 or total above 50 should be considered abnormal and indicative of the state of hyper acidity.

Common terms in relation to gastrointestinal disorders

Residum : The contents of fasting stomach.

Gastric atony : Due to lack of normal muscle tone of the stomach contractions are not of sufficient strength to move the food mass out of stomach at a normal rate.

Hyperperistalsis : Increased action of the musculature of the stomach and intestine.

Hypoacidity : Decreased amount of acid secretion. It is usually encountered in chronic gastritis, carcinoma of the stomach, gastric and other neuroses, secondary anaemias, tuberculosis, gastric ulcer, hyperthyroidism and in 20% of normals.

Hyper acidity : Hyper secretion of acid occurs in duodenal ulcers.

Hypo chlorhydria : Denotes a diminished amount of free acid.

Achlorhydria : No free acid is present although there is some peptic activity.

Achylia gastrica : Absence of both acid and enzyme activity.

7.2 GASTRITIS

This condition is an inflammation of the mucose of the stomach, occurring as an acute or chronic lesion with atrophy or hypertrophy.

Acute Gastritis

Acute gastritis is characterized by general inflammatory reaction of the mucosa with hyperemia, edema and exudation.

Chronic Gastritis

Chronic gastritis is characterized by altered resistance of the gastric mucosal barrier to hydrogen ions. Recurrent inflammation leads to glandular atrophy and changes in activities of the gastric mucosal cells.

Clinical Findings

Epigastric distress

Heart burn

Severe vomiting.

7.3 NUTRITIONAL MANAGEMENT

Acute Gastritis

Acute gastritis heals within 3 or 4 days.

Hence nutritional management is not the primary concern.

Remove the offending agent by gastric lavage or neutralize the offending agent with antibiotics.

Withhold food for 24–48 hours to allow the stomach to rest.

Replace water and electrolyte losses.
Give clear fluids after one or two days.
Gradual progression to bland, easily digestible food and then to normal diet.

Chronic Gastritis

Correct faulty dietary habits provide a relaxed atmosphere at bed time.
Provide 4 to 6 meals.
Nutritional requirements should be met.
Start with bland, soft diet and progress to normal diet.

7.4 PEPTIC ULCER

Peptic ulcer refers to a break in the mucosa that occurs in just a position to an acid secreting area of the stomach.

- Peptic ulcer usually occurs in non-acid secreting mucosa adjacent to acid secreting mucosa.
- The formation of an ulcer depends on two opposing factors operating at the site of ulceration.
- That is an imbalance between mucous secretion and surface glycoproteins protecting it.

Factors Influencing Gastric Secretion

Heredity
Blood group "O"
Diminished blood sugar levels (fasting).
Mental stress.
Increased parietal cells.
Raised serum calcium levels.
Smoking.
Strong tea, coffee, alcohol.
Meat soups and extractives.
Chillies and spices.
Protein rich foods.
Refined food intake.

Duodenal Ulcer

Patients have a higher acid output than normal and increased secretory drive during the non-stimulate state. Rapid gastric emptying is inhibited by acid and decreased duodenal pH.

Gastric Ulcer

Patients have normal or low acid output.
Factors altering mucosal defence are probably important.
These factors are,

- Duodeno gastric reflux of bile causing bile salt induced mucosal injury.
- Aspirin ingestion.

7.5 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 introduced into the stomach and the cotor inspects the food pipe and stomach and detect any breaks in the lining membrane. It takes 15–20 minutes. If there is cancer it can also be detected.

- Acid secretion of the stomach — gastric contents are aspirated through a tube. Basal acid output (BAO) measures the amount of acid secreted without any stimulus. In addition betazol hydrochloride may be injected to stimulate secretion for determination of peak output (PAO). Tests of acid content and output are used to detect hyper secretion and Zollinger-Ellison syndrome, rather than for diagnosis of ulcer.

Clinical Findings

Epigastric pain occurring as deep muscle contraction 1–3 hours after meals.
This pain can be dull, piercing burning or gnawing.
Hypermotility of the stomach gastric distention weight loss.

Laboratory Findings

Iron deficiency anaemia
Low plasma protein levels
Megaloblastic anaemia.

7.6 RATIONALE FOR TREATMENT

Treatment consist of

Drugs
Rest
Diet

Drugs

The following drugs are prescribed for ulcer healing.
Histamine Hydrogen receptor antagonists—inhibits basal and stimulated acid secretion. It should be taken with meals and bed time.
Antiacids—neutralizes excess acid. It should be taken between meals and at bed time, but should not be taken simultaneously with the above drug.
Anti cholinergic drugs—inhibits acid secretion.
Anti spasmodics—delays gastric emptying.

Rest

Both mental and physical rest is important.
Modification of living and work habits are important.

Diet

The development of potent drugs has largely replaced the role of diet in the treatment of peptic ulcer. The major goal of diet is to avoid extreme elevation of gastric acid secretion and direct irritation of gastric mucosa.
This necessitates only slight modifications in the patients normal diet.
The diet must be nutritionally adequate.
Regularity of meal timings should be maintained.
Individualization of diet is important.
The diet should be bland and soft in nature.
Spices causing dyspepsia are to be avoided.
Avoid Caffeine containing beverages and alcohol from the diet.
Citrus juices should be restricted.

Small and frequent feedings should not be advised as this leads to increased acid secretion.
3–4 hourly feeds are advisable.
Stomach distention with large quantities of food should be avoided.
Fried foods should be avoided.
Fats like cream, butter can be used with discretion.
Raw vegetables and coarsely cooked vegetables should be avoided.
Softly cooked, tender vegetables are suitable.
Frequent milk intake is not encouraged.
In some instances intakes of nutrients in excess of the recommended dietary allowances are desirable with emphasis on high quality protein, ascorbic acid and iron.

Foods Permitted

Wheat or millet chapathi, breakfast cereal, cooked rice, dhal, tender cooked vegetables, meat, fish, chicken, eggs, milk and milk products vegetable soups, potato, sweet potato, jam, fat for cooking, sugar jaggery jam desserts, fresh fruits, nuts light beverages.

Foods Avoided

Vegetable salads, vegetables with coarse fibre and overripe seeds, fried foods, condiments and spices, chutneys, pickles, alcohol.

Complications

Haemorrhage } are medico surgical
Perforation } emergencies.
Penetration } Affect the ability to
Obstruction } eat.

Clinical Effects of Gastric Surgery

- Dumping syndrome
- Diarrhoea
- Malabsorption
- Weight loss
- Iron, folate, B₁₂, Calcium and Vit. D deficiencies.

Nutritional Therapy

Bleeding ulcers

Early feeding after control of bleeding to replace protein and iron losses is recommended. Iron supplementation is essential TPN should be given immediately. There is more than 25% weight loss with hypoproteinemia.

Dumping, diarrhoea and early satiety problems

Dry solid meal, low in simple sugars but high in complex carbohydrate and protein should be given.
Avoid significant amount of milk and milk products due to functional lactose deficiency.
A drink of water, a sugar less tea after an hour of the meal is beneficial.
Adequate intake of red meat for haeme iron is recommended.
Oral supplements of iron and folic acid and Vitamin B₁₂ by injection, when indicated should be given to prevent anaemia.

7.7 PRACTICAL ASSIGNMENT

AIM : To plan modified diet for patient suffering with duodenal ulcer.

PROCEDURE : Collect case history of patient suffering with duodenal ulcer from near by hospital. Analyse the case history critically and formulate nutritional requirements. (Energy, protein, fat, iron and calcium) Plan a day's menu to suit the patients needs and calculate the nutrients mentioned *i.e.*, energy protein, iron and calcium.

CASE HISTORY — DUODENAL ULCER

History of present illness

General Information

Relevant past history (illness)

Dietetic history

General examination

Laboratory examination

Vitals and symptoms

Clinical impression

Treatment

Nutritional Therapy

Modified RDA

Energy
Protein
Fat
Iron
Calcium

FOOD PLAN**Table 7.1 : Total Exchanges Per Day**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Approximate amount</i>	<i>Energy K. cal</i>	<i>Protein (gs)</i>	<i>Iron (mgs)</i>	<i>Calcium (gs)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU**Table 7.2 : Distribution of Exchanges into Meals and Snacks**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Menu</i>	<i>Energy (K. cal)</i>	<i>Protein (gs)</i>	<i>Iron (Mg)</i>	<i>Calcium (gs)</i>
		Breakfast				
		Midmorning snacks				
		Lunch				
		Snacks				
		Dinner				
		Bed time				

ANSWER THE FOLLOWING QUESTIONS

1. Highlight "Milk therapy controversy" in the management of peptic ulcer.

2. What is the role of fibre in the management of peptic ulcer?

3. What are the causative factors for the development of post gastrectomy complications?

Chapter 8

Diet in Diseases of Liver

8.1 FUNCTIONS OF LIVER

Metabolism

Converts glucose into glycogen and triacylglycerols (absorptive period), converts galactose and fructose to glucose.

Converts amino acids into fatty acids which can be incorporated into triacylglycerols (absorptive period). Synthesizes triacylglycerols and secretes as very low density lipoproteins (absorptive period).

Produces glucose from glycogen and other sources (amino acids, lactic acid, pyruvic acid—gluconeogenesis) and releases the glucose into blood (post absorptive period).

Converts fatty acids to ketones (post absorptive period).

Produces urea through the urea cycle and releases it into the blood for excretion by the kidney. Amino acids are deaminized to produce ammonia, which in turn is used to produce urea.

Normal blood ammonia is 10–20 µg/dl.

Cholesterol Metabolism

Synthesizes cholesterol and releases into the blood.

Secretes cholesterol into the bile.

Converts cholesterol into bile acids.

Digestive Function (Through Bile Production and Secretion)

Synthesizes and secretes bile acids (necessary for emulsification of fat to facilitate digestion of fat).

Secretes into the bile a bicarbonate rich solution of inorganic ions, which helps to neutralize acid in GI tract.

Excretory Function

Secretes bilirubin (from breakdown of haemoglobin) and other bile pigments into the bile.

Excretes (Via bile) many endogenous and exogenous organic molecules as well as trace metals (Kupffer cells).

Biotransforms many endogenous and exogenous organic molecules into metabolites which can be excreted in bile or urine.

Removal of ammonia.

Synthesis of Plasma Proteins

Synthesizes and secretes plasma albumin, acute phase proteins, binding proteins (Steroid hormones, Vitamins such as Vitamin A and trace elements) lipo proteins.

Blood Clotting

Produces prothrombin and fibrinogen.

Facilitates absorption of Vitamin K (through bile secretion and emulsification of fats).

Endocrine Function

Secretes insulin like growth factor-I (IGF-I) in response to stimulation by growth hormone. IGF-I promotes growth by stimulating mitosis in various tissues.

Helps to activate Vitamin D.

Forms triiodothyroxine (T_3) from tyrosine (T_4).

Metabolizes hormones.

8.2 DIAGNOSIS OF LIVER DISEASE

Seventy five per cent of function can be lost before liver failure is clinically evident.

Diagnostic Tests

Metabolic functions

Blood ammonia

Serum protein concentration

Prothrombin time.

Formation and excretion of bile

Vanden Berg test

Urine bilirubin

Urine or stool urobilinogen

Serum bilirubin

Detoxification and excretion

BSP retention time.

Enzyme synthesis

Serum alkaline phosphatase (ALP)

Serum amino transaminases (SAT)

Serum glutamic-oxaloacetic transaminase (SGOT), or

Serum aspartic transaminase (AST)

Serum alanine transaminase (ALT) or

Serum glutamic-pyruvic transaminase (SGPT)

8.3 HEPATITIS

Hepatitis can be defined as inflammation of the liver cells or cells lining the biliary tract. It is characterized by both inflammation and necrosis of liver tissue and may be due to variety of reasons. This can be either acute or chronic. Some forms of hepatitis leads to rapid death, some progress to cirrhosis, while others continue as a low grade inflammation.

Viral Hepatitis

Hepatitis A is transmitted by fecal or oral route. Hepatitis B or Serum hepatitis is transmitted through blood transfusion. Hepatitis C is an acute hepatitis which is serologically different from A or B.

Chronic hepatitis is a chronic inflammation following hepatitis B or C. It can result from exposure to toxins also.

Clinical Manifestations

Clinical course of hepatitis has three phases of variable duration depending on the type of hepatitis and health of patient.

- The preicteric phase
- The icteric phase
- The convalescent phase

The Preicteric Phase

The preicteric phase is the phase that occurs before the development of Icterus (jaundice).

Symptoms of this phase include.

- Fatigue
- Headache
- Fever
- Anorexia
- Diarrhoea
- Nausea
- Vomiting
- Abdominal discomfort

The Icteric Phase

The icteric phase is characterized by inability to excrete bile.

- Jaundice
- Clay coloured stools
- Dark urine
- The symptoms of preicteric phase persist and may worsen.

The Convalescent Phase

Regression of jaundice occurs.

Appetite slowly returns.

To normal chronic fatigue may be the dominant symptom.

Laboratory Findings

- Fatty infiltration.
- Parenchymal cells of liver are damaged.
- Hepatomegaly.
- Total cholesterol remains normal, but the amount of esterified cholesterol decreases and free cholesterol raises.
- Serum and urine bilirubin levels are elevated.
- Levels of SGPT and SGOT are elevated.

8.4 PRINCIPLES OF TREATMENT

- Rest
- Abstinence from alcohol
- Diet

Rest

Rest in bed is essential during the acute stage of the disease.

The rest should be continued until:

- The appetite has returned to normal
- The liver is no longer tender
- The urine is free of bilirubin
- Normal colour has returned to the stools
- And serum bilirubin is less than 1.5 mg/10 ml.

This usually involves two to three weeks of bed rest.

Dietary Management

High calorie, high protein moderate fat, diet is prescribed.

Calories

3000 – 4000 K. cal/day.

A high carbohydrate diet is essential not only as a source of calories, but because of the protein value of carbohydrate in the prevention and treatment of injury to liver cells.

Protein

1.5 – 2 gs perday/kg body weight.

Ample intake of protein is essential for regeneration of liver cells.

Fat

Restrict fat if not tolerated.

When nausea and lack of appetite are present, fat and fatty foods are poorly tolerated.

The absorption of fat from the intestine is impaired because of lack of bile salt.

Fried foods are badly tolerated

The foods likely to cause dyspepsia should be avoided.

Foods Which Cause Dyspepsia

- Alcohol, strong tea, coffee, gravies and soups made from meat extracts.
- Raw vegetables, cucumber, onion, radish, tomatoes.
- Raw unripe fruit, dried fruits nuts, skins and peels of all fruits whether cooked or in puddings, cakes or jams.
- Pickles, spices and condiments.
- Tough, twice cooked or highly seasoned meats including sausages, bacon and pork.

Take precaution to prevent infecting others.

Sterilize dishes and trays.

ALCOHOLIC LIVER DISEASE (Fatty liver, alcoholic hepatitis, cirrhosis)

8.5 FATTY LIVER

Fat infiltration into the liver is known as fatty liver.

Laboratory Findings

- Increased BSP retention time.
- Elevated serum globulin and transaminase levels.
- Low serum albumin.

Therapy

- Bed rest
- No alcohol
- Good normal diet
- Moderate fat restriction

8.6 ALCOHOLIC HEPATITIS

Clinical Manifestation

- Fatigue
- Weakness
- Anorexia
- Fever
- Hepatomegaly.

Laboratory Findings

- Elevated transaminases
- Decreased prothrombin time
- Necrotic hepatocytes

Therapy

- Bed rest
- No alcohol
- Good normal diet
- Moderate fat restriction

8.7 CIRRHOSIS

Cirrhosis of the liver is a chronic disease characterized by irreversible distortion of normal hepatic architecture by connective tissue (fibrosis) in the forms of bands or walls (septa) which separate and surround nodules of regenerating liver cells. The septa and the growth of nodular cells is not organized into the normal liver architecture. These nodules do not maintain a normal relationship to the blood vessels and bile canaliculi of the liver lobule. As a result circulation and diffusion of nutrients, metabolic products and bile are poor.

Clinical Manifestations

- Weight loss
- Anorexia

Dietary Deficiencies

Thiamine
Folic acid
Pyridoxine
Fat soluble vitamins A, K and E.

8.8 DIETARY MANAGEMENT

Protein

1.0 – 1.5 gs protein/kg/day.
Food divided into 4–6 meals significantly improved nitrogen balance than food divided into three meals only.
Vegetable proteins and milk proteins are preferred but not meat proteins.
Vegetable proteins contain fewer AAA than meat proteins.
Milk has the added advantage of providing both calcium, vitamin A, D and riboflavin.

Manipulation of Amino acid ratio

Dietary enrichment with BCAA improves nutritional status as well as neurologic dysfunction.
Hypertonic dextrose solution with 14–23% BCAA can be used with step wise increase until 37–50% BCAA is reached.

To reduce Ammonia

Use Lactulose, a non-absorbable disaccharide, which acts by trapping N in the stool.
Administration of Neomycin. It reduces gut flora.
Use of BCAA.
Omit foods with preformed Ammonia like,

- Cheese
- Beef
- Bacon
- Gelatin
- Chicken
- Butter milk
- Potatoes
- Onions
- Peanut butter

Energy

High kilocalorie diet (50 K. cal/Kg body weight) is recommended.

Carbohydrate

Adequate carbohydrate *i.e.*, 300–400 gs should be provided to spare protein.
Excess carbohydrate may contribute to fatty liver.

Fat

Low to moderate fat (25% of K. cal) have to be provided if bile is inadequate
This should be in the form of 75% medium chain triglycerides which are better utilized in liver failure.

Sodium

If edema and ascites is present 500 mg sodium diet is prescribed.

Vitamins and minerals

Fat soluble vitamins and thiamine need to be supplemented.

Avoid iron supplements.

In liver disorders, copper excess is the problem.

Increase zinc intake.

Increased zinc intake will result in reduction in copper absorption and increases fecal copper excretion.

In Hepatic encephalopathy

Protein should be severely restricted (18–20g/day) sometimes even 0 per day. Maintenance of nitrogen balance is difficult.

Intravenous feeding may be used in some cases.

Hypertonic dextrose solution with 37–50% BCAA and low in AAA should be administered.

Alfa Keto analogs of BCAA can also be given.

All measures to reduce ammonia (previously mentioned) should be taken.

Fat solution containing 75% medium chain triglycerides and 25% long chain triglycerides may be better utilised.

Attention to electrolyte balance as well as acid base status is equally important.

Supplements of water and fat soluble vitamins should be given.

The patient should be shifted to enteral feeding as soon as possible.

Enteral nutrition maintains intestinal membrane integrity thus preventing bacterial translocation and subsequent multiple organ failure in critically ill patients.

8.9 PRACTICAL ASSIGNMENT

AIM: To plan modified diets for patients suffering with viral hepatitis and cirrhosis of liver.

Procedure: Collect case histories of patients suffering with viral hepatitis and cirrhosis from near by hospitals. Analyse the case histories critically, and formulate nutritional requirements (energy, protein, fat, carbohydrate) and plan a day's menu to suit the patients needs. Calculate the nutrients mentioned above as desired.

CASE HISTORY I — VIRAL HEPATITIS

History of present illness.

General information.

Relevant past history (illness).

Dietetic history.

General examination.

Laboratory examinations.

Vitals and systems.

Clinical impression.

Treatment.

Nutritional therapy.

Modified RDA

Energy

Protein

CHO

Fat

FOOD PLAN**Table 8.1 : Total Exchanges Per Day**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Approximate amount (g)</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU**Table 8.2 : Distribution of Exchanges into Meals and Snacks**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Menu</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>
		Breakfast				
		Midmorning Snacks				
		Lunch				
		Snack				
		Dinner				
		Bed time				

Case History III — Cirrhosis of Liver

History of Present Illness

General information

Relevant past history (illness)

Dietetic history

General examination

Laboratory examination

Vitals and systems

Clinical Therapy

Treatment

Nutritional therapy

Modified RDA

Energy

Protein

CHO

FAT

SODIUM

FOOD PLAN

Table 8.3 : Total Exchanges Per Day

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Approximate amount (g)</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						

MEAL PATTERN AND MENU

Table 8.4 : Distribution of Exchanges into Meals and Snacks

<i>Food exchange</i>	<i>Exchange No.</i>	<i>Menu</i>	<i>Energy (kcal)</i>	<i>CHO (g)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Sodium (mg)</i>
		Breakfast					
		Midmorning					
		Snack					
		Lunch					
		Snack					
		Dinner					
		Bed time					

ANSWER THE FOLLOWING QUESTIONS

1. What alteration in protein and nitrogen metabolism bring about dietary modifications in cirrhosis of liver?

2. Explain the basis for acites.

3. Why are the daily measurement of weight important?

4. What is the basis for the development of hepatic encephalopathy?

5. What dietary steps must to be taken immediately when hepatic encephalopathy is present?

6. What are the consequences of Alcoholism?

Chapter 9

Diet in Diseases of Gall Bladder

9.1 FUNCTIONS OF THE GALL BLADDER

- Concentrates bile
- Stores bile
- Bile is made in the liver.

Composition of Bile

- Water
- Bilirubin
- Bile acids (exist as bile salts combined with sodium and potassium).
 - Cholic acid combined with glycine-glycocholic and cholic acid combined with taurine-tauro cholic acid,
 - Chenodeoxycholic acid.
- Mucin
- Cholesterol
- Bile salts — (Circulate between the intestine and are absorbed by the portal vein and recirculated to the liver. Most bile salts are reabsorbed in the terminal ileum).

Process of Emptying of Bile into Duodevum

- Fat in duodenum influences the hormone Cholecystokinin (CCK) to be released into blood stream.
- Cholecystokinin influences contractions of the gall bladder and emptying bile into duct leading to duodenum.
- Sphincter of oddi relaxes simultaneously.

Functions of Bile

- Solubilization of fat in the intestinal lumen is the main function of bile.
- At the critical micellar concentration (0.15 M) bile salts combine with fatty acids and beta monoglycerides to form micelles of 3–10 mm in diameter with the hydrophilic polar group on the outside.
- Fat is solubilized in this aqueous environment.
- Enzymatic lipolysis becomes possible facilitating fat digestion.
- The bile salt and the fatty acids and/or a beta monoglycerides are dissociated in the jejunum.
- The fat is absorbed and the bile salt is reabsorbed into the ileum.

9.2 CHOLECYSTITIS

Cholecystitis is the inflammation and gall bladder wall.

Clinical Findings

Flatulence
Vomiting
Epigastric pain

9.3 GALL-STONES

Gall-stones are formed from bile salts and cholesterol. It may also contain bile pigments, bilirubin.

Clinical Findings

Pain (Gastric)
Excretion of bile salts
When bilirubin in blood is elevated, jaundice occurs.

Laboratory Findings

Cholesterol gall-stones — Enhanced activity of HMA — COA reductase the rate limiting enzyme in hepatic cholesterol synthesis.
Obese person — Increased hepatic cholesterol secretion.
Persons of Normal weight — Decreased bile acid pool size.

9.4 DIAGNOSTIC TESTS

Oral cholecystography
Intravenous cholangiography
Ultra sonography
CT Scan

9.5 TREATMENT

Dietary

A low fat diet is recommended to reduce symptoms (epigastric pain, bloating)

Fat tolerance is variable

The diets should be individualized.

There may be intolerance for specific foods like:

- Legumes
- Cabbage family
- Onion family
- Spicy foods.

Surgery

Treatment for gall-stones and cholecystitis is surgical.

A low fat diet for several weeks have to be given after surgery.

9.6 PRACTICAL ASSIGNMENT

AIM: To plan a day's diet for 45 year old obese women (Housewife) who is suffering with gall-stones and to calculate important nutrients in the diet.

Patients Profile

Age:

Sex:

Clinical diagnosis:

Diet Planning

Modified RDA

Calories

Proteins

Carbohydrates

Fat

Cholesterol

Consistency

FOOD PLAN

Table 9.1 : Total Exchanges Per Day

<i>Food Exchange</i>	<i>No. of exchanges</i>	<i>Approximate amount (g)</i>	<i>Nutrient</i>		
			<i>Calorie (kcal)</i>	<i>Fat (g)</i>	<i>Cholesterol (mgs)</i>
Milk					
Vegetable A					
Vegetable B					
Fruit					
Cereal					
Meat					
Pulse					
Fat					
Sugar					

MEAL PATTERN AND MENU

Table 9.2 : Distribution of Exchanges into Meals and Snacks

<i>Food Exchange</i>	<i>No. of exchanges</i>	<i>Menu</i>	<i>Nutrient</i>		
			<i>Calorie (kcal)</i>	<i>Fat (g)</i>	<i>Cholesterol (mgs)</i>
		Breakfast			
		Midmorning snacks			
		Lunch			
		Snack			
		Dinner			
		Bed time			

ANSWER THE FOLLOWING QUESTIONS

1. List the aspects of assistance and dietary counselling that should be provided for a patient who is suffering with gall-stones.

2. What is the role of Cholesterol in gall-stone formation?

Chapter 10

Diet in Diseases of Kidney

The management of patients in various stages of renal disease is the subject of ongoing controversy. The possibility of slowing the progression of chronic renal disease by diet is a central issue in the management of renal dysfunction. Another issue concerns the prevention of cardio vascular disease one of the most deadly complication of chronic renal disease.

10.1 ANATOMY OF THE KIDNEY AND URINARY SYSTEM

The kidneys are paired, somewhat flattened kidney bean shaped organs located at the back.

Each kidney consists of cortex, medula and pelvis.

Each kidney weighs about 60 gs and is the size of a fist.

The kidneys, ureter, urinary bladder and urethra make up the urinary system.

The kidneys are involved in urine formation and composition, whereas the other organs transport or store urine.

Functional unit of the kidney is the nephron.

Each kidney has more than 1,00,000 nephrons.

Each minute one fourth of the cardiac output passes through the glomeruli therefore in 4 – 5 minutes the entire blood supply would have passed through the kidney.

Each kidney filters 125 ml/min or 180 litres per day.

Normally only 1–2 litre of urine are excreted.

Functions of The Kidney

Maintains constant composition and volume of blood by removal of excess fluid and waste products.

The kidney maintains acid base balance of the body.

Toxic substances/metabolites/drugs are also removed from the body by the kidney.

Regulates blood pressure by production of the hormone renin and maintaince salt and water balance.

Stimulates red blood cell production by producing the hormone erythropoietin.

Maintains normal bone health by maintaining balance of calcium and phosphorous.

This is accomplished by (1) hydroxylation of in active vitamin D to 1, 25-dihydro cholocalciferol to promote absorption of calcium from G.I tract and (2) excretion of excess phosphorous.

10.2 NUTRITIONAL ASSESSMENT IN RENAL DISEASES

The following parameters are used to assess the nutritional status of patients suffering with kidney disorders.

- Anthropometric measurements
- Dietary information
- Renal functional tests
- Blood analysis

Anthropometric Measurements

The following anthropometric measurements are useful indices.

- Actual body weight
- Height
- Relative weight $\frac{\text{(Actual body weight)}}{\text{(Optimal body weight)}}$
- Body fat
- Muscle mass

Dietary Information

This can be obtained by various methods outlined in Chapter 2.

Clinical Measurement of Glomerular Filtration Rate (GFR)

GFR is estimated by measuring the plasma clearance of a substance like insulin or mannitol which appears in the glomerular filtrate in the same concentration as in plasma but which is neither reabsorbed nor secreted by the renal tubules.

Normal values for a healthy male adult

Blood flow to both kidneys	: 125 ml/min
Renal plasma flow	: 600 ml/min
Glomerular filtration rate	: 100–150 ml/min

Urea and creatinine clearances, although determined in part by tubular processes are convenient clinical indices of GFR.

$$\text{Clearance} = \frac{\text{Excretion rate}}{\text{Plasma concentration}}$$

Excretion rate = mg/ml urine × ml urine/min

Plasma concentration = mg/ml plasma

Normal clearance for men : 140–200 liter per day
97–140 ml/min

Women: 120–180 liter per day
85–125 ml/min

Creatinine clearance and serum creatinine levels are more reliable than blood urea levels as an index to serum changes in glomerular filtration rate when renal function is compromised.

Measurement of Tubular Function

The analysis of following parameters gives an idea about the extent of tubular damage.

- Urinary concentration
- Urinary sediment
- Proteinuria
- Urinary electrolytes.

Urinary Concentration

Normal urine concentration: 750–1400 milliosmoles per liter

Specific gravity 1.020–1.032

Reasons For Production of Concentrated Urine

Defective transport of sodium and urea with subsequent development of high concentration of solutes in the interstitial fluids of the renal medulla. Disease process which alter the function or structure of medulla.

- Tubular necrosis
- Hypercalcemic nephropathy

Starvation

Low protein diet

Overhydration

Reasons for Impairment of Urinary Dilution

Excessive antidiuretic hormone secretion-severe infection

Heart failure

Hepatic cirrhosis

Adrenal insufficiency

Renal insufficiency

Presence of Urinary Sediment (*MICROSCOPIC EXAMINATION*)

- RBC
- WBC
- Epithelial cells
- Casts-agglutination of protein cells or cellular debris
- Bacteria

Proteinuria

Protein excretion in excess of **150 mgs a day** or **10 mgs/100 ml urine** is abnormal.

Continuous proteinuria implies kidney disease.

Heavy proteinuria in excess of 4 g daily indicates a gross increase in glomerular permeability.

Proteinuria can also occur due to:

- Malignant hypertension
- Renal vein thrombus
- Renal venous congestion secondary to congestive heart failure.

Renal Excretion of Electrolytes

The excretion of sodium and potassium is used as a guide for dietary restriction in patients with renal disease.

For analysis 24, hour urine sample is collected.

The intake of sodium is adjusted to the output.

Appropriate ranges for urinary sodium on salt added diet are 50–80 meqs/day.

Blood Analysis

The following parameters are analysed:

- Serum protein
- Blood lipids
- Serum ferritin

BUN or SUN
Serum potassium
Serum sodium
Calcium
Phosphorous
Creatinine
Glucose.

Acceptable Values After 2 Days of Dialysis

BUN : 60 – 100 mg/dl
Alubumin : 3.4 – 5.0 g/dl
Potassium : 3.5 – 5.5 megs/dl
Calcium : 8.5 – 11.0 mgs/dl
Phosphorous : 3.2 – 4.0 mg/dl

10.3 ACUTE GLOMERULO NEPHRITIS

Nephritis or Bright's disease is an inflammation of kidney involving parenchymal and intestinal tissues. This disorder is confined to glomerulus only. It is most common in acute form in children of 3 to 10 years of age although it can also occur in adults past 50 years.

Clinical Findings

Edema

Tachycardia and hypertension

Characteristic urinary findings like:

- Proteinuria
- Hematuria
- Smoky appearance due to presence of red cells, haemoglobin and other haemoglobin decomposition products.

Nitrogen retention

Hypertension encephalopathy involving:

- Headache
- Vomiting
- Nausea
- Convulsions
- Coma

Finally oliguria and anuria which leads to development of acute renal failure.

10.4 OBJECTIVES OF DIETARY MANAGEMENT

Spare the diseased kidneys by minimising their work of excreting the end products of metabolism.

Provide optimal nutritional support.

Prevent uraemia caused by the accumulation of waste products of metabolism. This tendency should be minimised by reducing the amount of these products and by measures to promote their excretion by the damaged kidneys.

Adequate protein should be given unless there is oliguria or anuria.

Salt should be restricted if there is edema, hypertension or oliguria.

The fluid intake should be adjusted to output including losses in vomiting or diarrhoea.

MODIFICATION OF THE DIET

Acute phase of the illness

- Provide adequate fluid to maintain water balance.
- Provide non-protein calories to avoid excess tissue catabolism.
- Carbohydrate in the form of fruit juices can be provided.

Fluid

- During initial stages, the fluid should be decreased to allow for dispersal of oedema fluid. Daily weighing is needed to monitor overall fluid balance.
- In late stages the fluid intake is based on the volume of fluid excreted and an allowance of 500 ml/day is given for insensible water loss.
- The fluid is calculated taking into account the water consumed with the drugs, water present in milk, curds, buttermilk, tea, coffee, fruit spices etc. Daily fluid replacement should be 1000 ml plus daily amount excreted in the urine.
- 30 ml/kg body weight for infants
 - 20 ml/kg body weight for order children
 - 10 ml/kg body weight for adults.

Energy

- Requirements are calculated for the particular age and weight and 10% more for infection. Sufficient calories in the form of sugar, honey, glucose, sago, fats, oils and starchy foods are given without increasing the protein intake.
- By giving carbohydrate liberally protein catabolism and starvation ketosis are reduced. Above foods are not only rich in calories, but also poor in sodium and potassium.

Protein

- Daily protein intake should be restricted. A low protein diet is recommended to give rest to the kidneys. Complete and good quality proteins are better to ensure maximum utilisation. Out of the recommended protein 50% should be from animal protein. Fruits and vegetables which are usually low in protein can be prescribed provided they are also poor sources of sodium and potassium. 0.2 gs/kg body weight is recommended. During recovery phase protein may be increased to 0.5 gs/kg weight for adults and 0.75 gs/kg for the child. If Albuminuria is present the protein intake should be increased by the amount of protein lost in the urine.

Sodium

- When oedema and hypertension are present, sodium restriction of 500–1000 mg is generally indicated. In sodium restricted diets, the following foods are avoided.
- Salt during cooking or on table.
 - Baking powder and sodium bicarbonate which are added to cakes and pastries.
 - Sodium benzoate, potassium meta bisulphite which are added as preservatives in pickles, squashes and canned foods.
 - Papads, cheese, salted chips, nuts, popcorn and biscuits.

- ❑ Commercial soft drinks.
- ❑ Dried foods like fish, fruits and soup cubes.

Potassium

When the kidney don't work properly potassium builds up in the body and causes the heart to beat unevenly and stop suddenly.

When urine formation is reduced, potassium is also restricted.

Potassium content can be reduced in vegetables by cooking in excess water and discarding the water.

Spices and condiments can be used in small quantities as they are rich in potassium.

Fruits low in potassium are apple, guava, papaya, peas and pineapple.

Nuts, jaggery, instant coffee, chocolate and cocoa powder are rich in potassium.

Sugar, honey, arrow root, sago, unsalted butter, vegetable shortenings, vegetable oils and ghee are free foods.

10.5 CHRONIC GLOMERULU NEPHRITIS

Nephritis may be of short duration, subacute latent or chronic

CLINICAL FINDINGS

Latent stage

No symptoms

Nephritis may be detected only by laboratory tests.

Chronic stage

Proteinuria

Hematuria

Hypertension

Vascular change in the retina

The kidneys are unable to concentrate urine

There are both frequent urination and nocturia

The nephrotic syndrome characterized by massive edema and severe proteinuria may develop

Severe malnutrition

Depletion of plasma proteins

Anaemia

Finally symptoms of renal failure occur.

10.6 MODIFICATION OF DIET

Protein

Latent stage — Normal daily protein intake + The amount of protein lost with urine.

Nitrogen retention — Restriction of protein (40 gs).

Patients who are malnourished, edematous — 80–100 gs protein/daily and who lose much protein in the urine

Energy

Provide sufficient carbohydrate and fat to avoid breakdown of body protein 300 gs carbohydrate, 80 gs of fat will provide 1900 non-protein calories.

Sodium –

500 – 1000 mgs.

Potassium

Restrict potassium

Fluid

The inability of the kidney to concentrate urine makes it undesirable to restrict fluids.

Provide 1–2 litres of fluid.

Additional fluid may be necessary in the presence of fever, vomiting and nitrogen retention.

10.7 NEPHROSIS

The nephrotic syndrome is a syndrome consisting of albuminuria, hypoalbuminemia, hypogamma globulinemia and hyperlipidemia. As a result of hypo albuminemia, the patient becomes edematous and sodium is retained. The primary lesion in this condition lies in the glomerular basement membrane which becomes leaky to protein.

CLINICAL FINDINGS

Nephrosis is distinguished clinically from glomerulonephritis by the consistent absence of hypertension and haematuria and the usual absence of anaemia and nitrogen retention.

The following symptoms are seen:

Heavy proteinuria

Hypo albuminaemia

Peripheral oedema

Tissue wastage

Fatty liver

Malnutrition

Increased blood cholesterol level

Increased susceptibility to infection.

10.8 DIETARY TREATMENT

High protein, high calorie, high carbohydrate, salt restricted, moderate fat diet with restricted fluid are recommended.

Energy

To ensure protein use for tissue synthesis, sufficient kilo calories must be provided.

2000 K. cals is suggested.

Since appetite is usually, poor food must be appetising and much encouragement is needed.

Protein

Protein requirement depends on the phase of the disease.

Edematous phase

Protein allowance appropriate for the age and size of the patient + amount lost + modification for state of renal failure if any 90 – 120 g protein should be provided.

A high protein diet is required to meet the,

- Heavy losses of albumin

- Protein depletion of tissues
 - To build up resistance power to infections
- Pulse proteins should be mixed with cereals or milk to improve quality.
High quality proteins like egg, meat are preferred.
Energy and protein requirements for a nephrotic patient is given in table.

Table 10.1 : Requirement of Protein and Energy in Nephrosis

<i>Age (Years)</i>	<i>Protein g/kg body weight</i>	<i>Energy kcal/kg body weight</i>
0 – 1	6	100 – 130
1 – 3	3 – 4.5	90 – 100
3 – 6	3	80
7 – 12	2.5 – 3	70
Teenage	2.5	45 – 70
Adults	1.5 – 2.5	45

Source: Gnanasundaram S. and T. Ramamurthy (1988) “Annual Conference of Nutrition Society of India (Tamil Nadu Chapter).

Lipids

- Cholesterol intake should be reduced to below 300 mgs/day to prevent hyperlipidemia.
- High cholate containing foods should be avoided.
- Cholate is a cholesterol processor.
- Diet higher in dietary long chain saturate fatty acids is useful in reducing.
- Mevalonate production which is a cholesterol precursor.
- HMG-COA reductase** inhibitors are useful in reducing hyper cholesterolemia.

Sodium

- Sodium is restricted to reduce edema.
- The sodium can be restricted to 1g.

Other electrolytes

- Since calcium and potassium deficiency may accompany severe proteinuria, bone rarefaction and hypocalcemia are common in nephrosis potassium supplements are essential.
- Magnesium levels to be monitored closely and supplements should be provided.

Vitamins

- Vitamin supplements specially vitamin C are essential.

Fluid

- Fluid restriction is necessary if:
 - Oliguria is present.
 - If urine output is less than 25 ml/kg/24 hours.
 - When oedema is present.

10.9 ACUTE RENAL FAILURE

There is a sudden shutdown of renal function following metabolic insult or traumatic injury to normal kidneys. There is high mortality in which the nutritionist plays a supporting role.

CLINICAL AND LABORATORY FINDINGS

The urinary output as little as 20 to 200 ml/day (anuria or oliguria).

Accumulation of waste products of protein metabolism in blood.

Increased serum urea nitrogen and creatinine levels.

Diminished excretion of potassium.

Rise in serum potassium due to its release from tissue protein breakdown.

There is also increased phosphate and sulphate with decreased sodium, calcium and base bicarbonate.

The patient may be lethargic anoxic and suffer nausea and vomiting elevation of B.P.

Signs of ureamia.

Death is caused not because of rise in blood urea but potassium intoxication or water intoxication.

10.10 DIETARY CONSIDERATION IN CONSERVATIVE MANAGEMENT

Energy

A minimum of 600 – 1000 K. cal is necessary. A high calorie intake is desired mainly from carbohydrates and fats.

Protein

All foods containing protein is stopped if the patient is under conservative treatment and blood urea nitrogen is rising.

If the patient is on haemodialysis or peritoneal dialysis, 40 gs of protein is allowed to reduce endogenous protein breakdown.

Protein is restricted as per GFR or urea content of the blood.

Carbohydrate

A minimum of 100 g/day is essential to minimise tissue protein breakdown.

A minimum of 100 g/day is essential to minimise tissue protein breakdown.

If orally given 700 ml of glucose with lime juice can be given.

If the patient is not fed by mouth a mesogastric tube feeding of 700 ml of 15% glucose is administered.

Fluid

The total fluid permitted is 500 ml + losses through urine and gastrointestinal tract.

ELECTROLYTES

Sodium

Dilutional hyponatremia occurs due to water retention.

Water restriction than salt administration is indicated.

Sodium intake should be adjusted according to 24 hrs excretion.

Potassium

Potassium intoxication (hyperkalemia) occurs with a daily rise of 4 msserum potassium.

Potassium sources like tomato juice, coffee, tea, cocoa and potassium rich vegetables are avoided.

Haemodialysis or peritoneal dialysis is considered when blood urea level is over 200 mg/100 ml.

10.11 CHRONIC RENAL FAILURE

Chronic renal failure is characterized by the progressive irreversible loss of nephron function, regardless of the cause. As the number of functioning nephrons decrease, the remaining intact nephrons try to maintain homeostasis.

CLINICAL AND LABORATORY MANIFESTATION

Loss of appetite
Vomiting
Hiccups
Edema
Hypertension
Electrolyte imbalance usually hyperkalemia
Type IV hyperlipidemia with atherosclerosis
Uremia
Hyperphosphatemia
Hypo calcemia
Anaemia
Hyperuricemia
Peripheral neuropathy
Twitching
Convulsion
Coma.

10.12 OBJECTIVES OF MANAGEMENT

- To prevent protein catabolism and minimise uraemic toxicity.
- To avoid dehydration or overhydration
- To correct and maintain fluid and electrolyte balance.
- To maintain optimal nutritional status.
- To retard progression of renal failure thus postponing the ultimate necessity of dialysis.

DIETARY MANAGEMENT

The severity of renal failure is closely related to the Glomerular filtration rate. Solute differ in their modes of excretion. They will differ in the degree to which their excretion is affected by decrease in GFR. The management of solute problems associated with decreasing glomerular filtration rate can be conveniently divided into three groups corresponding to the three types of solute excretion models. Initially protein need to be restricted followed by potassium and then sodium.

Energy

Adequate kilo calories are mandatory.

CHO and fat must supply sufficient non-protein kilo calories to spare protein for tissue protein synthesis and to supply energy.

The end products of CHO and fat metabolism (carbon dioxide and water) do not impose a burden on the progressive renal failure.

If energy intake is inadequate, endogenous tissue catabolism takes place which further aggravates the existing uraemia.

35 – 50 K. cal/Kg/day is recommended.

Protein

There are two approaches to dietary protein management in the non-dialysed patient.

- Moderate protein restriction – to maintain BUN below 80 mg/dl.
- Severe protein restriction – to slow down the progression of renal disease.

Moderate protein restriction

Protein intake should be determined according to GFR. (Table 10.2)

Protein intake can also be determined by the serum urea nitrogen (SUN) to creatinine ratio (Table 10.3) in chronic renal failure patients not on dialysis. As the GFR decreases, the protein should be progressively of higher biological value.

A vegetarian diet with good balance of amino acids may be preferable.

Urinary and stool losses of protein should be replaced with caution.

Table 10.2 : Protein Intake Based on Glomerular Filtration Rate

<i>GFR (ml/min)</i>	<i>Protein (g/kg/IBW/d)</i>
25	No restriction
20 – 25	1.3
15 – 20	1
10 – 15	0.7
4 – 10	0.55 – 0.6

Table 10.3 : Sun to Creatinine Ratio and Recommended Intake of Dietary Protein for CRF Patients on Dialysis

<i>SUN to creatinine ratio (both in mg/dl)</i>	<i>g of protein</i>
3	20
4	25
5	40
6	45
7	50
8	60
9	70
10	75

Severe protein restriction

18 – 20 g protein (< 0.5 g/kg/day) is recommended.

Use Keto acids also.

Keto acids provide needed essential amino acids

They also promote transamination there by diminishing the urea load. Administer Amino acid formulas containing high amounts BCAA and reduced amounts of phenylalanine, isoleucine, lysine and methionine.

Sodium

Generally 1380 – 2070 mgs diet is recommended.

If dehydration or hypotension occurs, sodium intake should be raised.

Potassium

As renal disease becomes more severe there is risk of developing hyperkalemia.

Potassium restriction is indicated if levels reach above normal.

Avoid salt substitutes with potassium chloride.

Phosphorous

The less protein diet will reduce phosphorous intake.

Further reduction may be necessary if phosphorous levels in blood are high.

Guidelines for reduction of phosphorous

Omit milk, yogurt and icecream.

Use non-dairy cream substitutes.

Use meat, poultry and fish only in amounts compatible with high biological value protein intake.

Exclude brans and whole grain cereals and bread.

Exclude dried beans and peas.

Omit cola beverages.

If the above guidelines are followed the diets will contain about 15 mg phosphorous per 1 gm protein.

A 50 g protein diet will contain about 750 mg phosphorous.

Calcium

Calcium and active form of vitamin D supplementation should be given.

10.13 RENAL CALCULI

Renal calculi is found in the urinary tract namely kidneys, ureter, bladder or urethra. A urine concentrated with calcium phosphate and ammonium phosphate predisposes towards stone formation.

Types of calculi:

- Calcium phosphate calculi
- Calcium oxalate calculi
- Uric acid calculi
- Magnesium ammonium phosphate calculi

The most common type of calculi is calcium oxalate.

Treatment of urinary calculi

An acid or alkaline diet is not very effective in bringing about solution of stones already formed but may serve to prevent the recurrence of stones. Before planning a diet for renal calculi one should be familiar with acid producing, alkali producing and neutral foods (table 10.4) and also foods rich in calcium, phosphate, oxalates and purines (Table 10.5).

Table 10.4 : Acid, Alkali Producing and Neutral Foods

<i>Acid producing foods</i>	<i>Alkali producing foods</i>	<i>Neutral foods</i>
Bread	Milk	Butter
Whole wheat	Fruits	Coffee
Cereals	Vegetables	Fats
Cheese	Almonds	Sugar
Corn	Apricots (dried)	Tapioca
Eggs	Beans	Tea
Lentils	Beets grains	
Macaroni, spaghetti	Dates	
Noodles	Figs	
Meat, Fish	Peads (dried)	
Poultry	Raisins	
Peanuts	Spinach	
Rice	Foods prepared with	
Walnuts	baking powder or	
	Baking soda	

Table 10.5 : Foods Rich in Calcium, Phosphate, Oxalates and Purines

<i>Calcium</i>	<i>Phosphate</i>	<i>Oxalates</i>	<i>Purines</i>
Leafy vegetables	Whole cereals	Leafy vegetables	Meat
Milk and milk products	Bran	Grapes	Fish
Small fish with bones, prawns, crabs, ragi	Oatmeal	Tea	Animal tissues & Organs (kidney, liver, brain, heart)
	Nuts and oilseeds	Chocolate	
	Banana	Cocoa	
	Carrot	Coffee	
	Meat	Cola drinks	
	Fish	Beef	
	Egg	Cashewnuts	
	Milk and milk products	Beetroot and yam	
	Organ meat		
	Soft drinks		

10.14 DIETARY MANAGEMENT**Fluid**

A liberal fluid intake is important.

Fluids like water, coconut water, barley water, fruit juices and weak tea should be given in order to void over 2000 ml of urine per day.

The urine should be light in colour when voided.

Salt — Salt may be used in moderation

Foods

When the stone contains calcium, magnesium phosphate and carbonates, the urine is alkaline.

For such patients acid ash diet is prescribed.

In such diets, the fruits and vegetables so elected should not contribute more than 25 ml of base daily.

If stones consists of uric acid and cystine alkaline-ash diet should be given.

Use alkaline producing foods such as fruits, vegetables and milk.

Acid producing foods such as meat, eggs and cereals are restricted.

If the stones contain oxalates, oxalate rich foods such as beans, beets, greens, chocolate, cocoa, dried figs, plums, potatoes, tea and tomatoes should be omitted.

Fibre intake should be increased.

The consumption of refined carbohydrate should be decreased.

10.15 PRACTICAL ASSIGNMENT

AIM: 1. To plan a modified diet for a patient suffering with Nephritis/Nephrosis/Renal failure.

2. To learn about potassium content of food groups and to recognize foods high and low in potassium.

PROCEDURE: Collect a case history of a patient suffering with Nephritis/Nephrosis/Renal failure from nearby hospital. Analyse the case history critically and formulate modified nutritional requirements (protein, sodium, energy and potassium). Plan a day's menu to suit the patients needs and calculate the nutrients mentioned above.

Modified RDA

Energy

Protein

Sodium

Potassium

Fluid

FOOD PLAN**Table 10.6 : Total Exchanges Per Day**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Amount</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Sodium (mgs)</i>	<i>Potassium (mgs)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						

MEAL PATTERN AND MENU**Table 10.7 :**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>MENU</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Sodium (mgs)</i>	<i>Potassium (mgs)</i>
		Breakfast				
		Midmorning snacks				
		Lunch				
		Snacks				
		Dinner				
		Bed time				
		Total				

ANSWER THE FOLLOWING QUESTIONS

1. Highlight the dietary modifications for a Diabetic patient who is suffering with kidney disorder.

2. List out protein free foods.

3. Enlist two commercial preparations suitable for tube feeding and parenteral nutrition for renal disorders giving compositional details.

4. How do you manage renal osteodystrophy?

5. What are the management strategies for treating metabolic acidosis in renal patients?

Chapter 11

Diet in Diseases of Small Intestine and Colon

11.1 FUNCTIONS OF COLON

The functions of human colon are many. They include:

- Storage of intestinal contents prior to their elimination.
- Fermentation of the residues of digestive processes with the production of short chain fatty acids, ammonia, amines and other fermentation products together with the liberation of physically trapped or chemically bound materials (water and minerals).
- Absorption of water.
- The production of bacterial metabolic end products, bile acids.

Diet and Colonic Function

Foods which are consumed may alter colonic function by altering bacterial cell mass.

This may change both metabolic activity and physical excretes in the colon by increasing the faecal bulk.

Alteration in fat and protein content of the diet are not associated with alterations in faecal output.

However the effects of fibre may be marked.

Fibre refers to the skin, seeds and structural parts of plants and to the connective tissue fibres of meat. Plant fibres include cellulose, noncellulosic, polysaccharides, hemicelluloses, gums, mucilages, pectins and lignins.

They increase the bulk of stools and help it to hold water.

Different fibres appear to increase bacterial yields to different extents depending on the degree to which they themselves are degraded.

The type and form of fibre determines

- The amount entering the colon:
- Whether it contributes as a substrate for colonic bacterial metabolism.
- As a source of bulk for the colon with implication to water holding and colonic motility.

Faecal recovery also depends on the type of fibre consumed.

<i>Type of fibre</i>	<i>Recovery</i>
Cereal fibres	93%
Cellulose components	75 – 100%
Pectin	4 – 5%
Hemicellulosic materials from certain fruits and vegetables	Trace/Nil

Recent studies indicate that available carbohydrate may make an important contribution as a bacterial substrate than fibre.

Carbohydrate foods may be the major determinants of colonic metabolism and colonic retrieval of calories not absorbed in the small intestine.

Faecal protein losses may range from 4 – 8 gs.

Faecal fat losses are very minute ranging from 1 – 2 gs daily.

The entry of bile acids into the colon appear to be affected by nutrient intake.

Increased intake of fat of certain types of fibre greatly increase the losses of bile acids in the faeces.

Human colon also absorbs 300 – 350 mgs of bile acid daily.

In addition, free bile acids are deconjugated by colonic losses of water and electrolytes.

Daily colon absorbs 1,350 – 1700 ml water, 175 – 215 megs sodium and 115 – 155 meg chlorine and secretes 4 – 8 megs phosphorous and 60 megs HCO₃.

Fibre acting directly or through bile acid metabolism may influence these functions of the colon.

Residue

Residue refers to the volume of material still in the G.I tract after the digestion and absorption processes are completed.

Examples of residues include:

- Non digestible fibres
- Bacterial residue
- Desquamated cells of mucosa
- Digestive residues other than the residues listed above these residue includes food fibres, but food fibres do not include all residues.

Diagnostic Tests

Measurement of motility

X-ray examination is frequently used to determine emptying time of gastrointestinal tract.

Procedure

Patient fasts overnight.

Patient is given barium sulfate in one of several light fat products such as egnog, butter milk, malted milk, half milk + half cream etc.

Progress of the opaque meal (barium sulfate) along the gastrointestinal tract is followed by fluoroscopy.

X-rays may be taken prior to swallowing after swallowing and whenever desired.

11.2 BACTERIAL AND FUNCTIONAL DISORDERS OF GASTROINTESTINAL TRACT

DIARRHOEA

Diarrhoea, which is symptom and not a disease is the occurrence of frequent liquid stools. The passage of food through the intestines is abnormally rapid and impairs complete digestion, absorption and no chance for the fluid to be absorbed. Diarrhoea should be distinguished from dysentery. Diarrhoea refers to the character of the stools alone, while dysentery refers both to symptoms of intestinal dysfunction such as abdominal cramps and stool characteristics, chiefly the presence of blood and mucus.

Diarrhoea disorders may be acute or chronic. There are four types of diarrhoea.

- Osmotic diarrhoea
- Secretory diarrhoea
- Exudative diarrhoea
- Limited mucosal contact diarrhoea.

Osmotic Diarrhoea

It is caused by the presence of osmotically active solutes in intestinal tract that are poorly absorbed.
e.g. : dumping syndrome.

Secretory Diarrhoea

It occurs as a result of active secretion by the intestinal epithelium of electrolytes and water.

This diarrhoea is caused by:

- Bacterial exotoxins
- Laxatives such as castor oil
- Viruses
- Increased intestinal hormonal secretion.

Exudative Diarrhoea

This occurs as a result of mucosal damage.

There is an outpouring of mucus, blood and plasma proteins.

There is accumulation of electrolytes and water.

Examples are:

- Chronic ulcerative colitis
- Radiation enteritis.

These diarrhoea result due to inadequate mixing of chyme and inadequate exposure of chyme to intestinal epithelium.

Examples are:

- Crohn's disease
- Extensive bowel resection

11.3 NUTRITIONAL CARE

For all types of diarrhoea the nutritional care is similar.

Acute Diarrhoea

For severe acute diarrhoea, a fast of 24 – 48 hours is often prescribed to provide rest to the G.I tract.

The nature and severity of the disease determines the duration of the rest.

The nutritional care for adults include replacement of lost fluids and electrolytes by increasing the oral intake of liquids, sodium and potassium.

Liberal intake of fruit juice, soups etc., should be given.

If the patient is in critical condition saline solution with potassium should be administered.

If the parenteral feeding continues for longer than 72 hours, amino acids in a 3% solution may be added to prevent further protein catabolism.

Acute diarrhoea is most dangerous in infants.

In such cases, parenteral administrative of fluids and electrolytes is usually necessary.

Pectin has value in treatment of diarrhoea.

Scraped raw apple or liberal amounts of apple sauce may be given every two to four hours as tolerated. As the diarrhoea stops, the amounts given should be increased gradually.

The foods should be low in fibre and rich in protein and calories.

The return to normal diet is gradual.

High calorie and protein intake may be required for several months to correct protein deficiencies (calorie 3000, protein 100 – 150 gs, fat 100 – 120 gs).

Chronic Diarrhoea

Chronic diarrhoea may be associated with number of nutritional deficiencies.

There will be heavy loss of electrolytes, vitamins, minerals and protein which has to be replaced.

Potassium is the important electrolyte lost.

Loss of iron from gastro intestinal bleeding leads to anaemia.

Protein may be poorly digested and absorbed.

Because of antibiotic therapy, B vitamin deficiencies occur.

Even deficiencies of folic acid and vitamin B₁₂ can occur.

Liberal amounts of fluids should be given.

A diet low in fibre, high in protein and calories and moderate in fat content need to be advocated.

Adequate supplements of minerals and vitamins should be given.

11.4 CONSTIPATION

It is infrequent or difficult evacuation of the stool. Regular bowel evacuation varies with the individual from once to twice a day to once every two or three days.

Diet for Constipation

Increase dietary fibre to 15 – 20 gs per day.

Use whole grain cereals, fruits and vegetables.

Raw foods are beneficial.

Raw vegetable salads should be included.

Increase fluid intake.

Refined fibres as pectin may be beneficial.

Increase sugar or lactose in formula foods.

11.5 IRRITABLE BOWEL (COLON) SYNDROME

Increased tonicity of musculature of G.I tract will lead to spasmodic contractions of musculature stools are dry, hard and small. Other names are mucous colitis, spastic constipation and spastic colitis.

Symptoms

Pain – dull aching, cramping, or sharp and intermittent

Anorexia

Nausea

Vomiting

Headache

Palpitation

Heart burn

Constipation or diarrhoea

Weight loss

Nutritional Care

Remove food irritants.

These may vary from one person to another.

Thus individualized therapy is necessary.

Provide bulk in smooth form.

Fibre — extra soft fruits and vegetables should be provided.

Give 8 – 10 glasses of water a day.

If diarrhoea is present, diet restricted in fibre should be given.

Sometimes the use of anticholinergic or antidiarrhoeal agents may be necessary.

11.6 ORGANIC AND INFLAMMATORY DISEASES OF THE GASTROINTESTINAL TRACT

CROHN'S DISEASE

(Regional enteritis, regional ileitis)

Crohn's disease is a nonspecific inflammatory disease of the gastro intestinal tract as well as the colon. Usually it involves the terminal ileum. Edema (villi) fibrosis and inflammation of intestinal wall occur. One segment or multiple segments of the G.I tract can be involved. Complications include bowel obstruction, ulceration, fistulas (deep ulceration often leading to an internal organ as the bladder or vagina). All layers of the intestinal mucosa may be involved.

Clinical signs

Abdominal pain

Cramping

Diarrhoea, steatorhea

Weight loss

Fever

Weakness

Malnutrition

Fistula formation

Biochemical indices

Intestinal protein loss

Negative nitrogen balance

Anaemia

Nutritional care

It should be individualised.

Sufficient K. cal to maintain or even gain weight if under weight. 40 – 50 K. cal/kg of ideal body weight should be given.

1.0 – 1.5 g of protein/kg ideal body weight.

Nothing by mouth should be given if bowel needs to be rested.

Usually low fibre, low residue diet is given to reduce stool output and rest bowel.

Refined cereals and breads should be included.

Meats with no connective tissue.

Clear soups.

Fruit juices, cooked vegetables which are low in fibre.

Plain desserts.

Medium chain triglycerides may be useful to reduce steatorrhea if present.

Foods high in potassium should be given in case of prolonged diarrhoea.

For selected patients with severe malnutrition enteral or parenteral nutrition provides additional nutritional support.

Supplemental calcium, iron and magnesium should be given since these minerals are poorly absorbed.

Vitamin B₁₂ should be given if the ileum is affected.

Complication

Bowel resection (surgery) may be done.

11.7 DIVERTICULOSIS AND DIVERTICULITIS

This inflammation can be due to constipation, bacterial growth, rupture of mucosal cells.

Diverticulitis occurs when one of these sacs becomes inflamed and perforates.

Diverticulosis is fairly common and incidence increase with age.

Diverticula can occur all along the G.I tract but are more common in the colon than other areas.

This might occur due to structural weakness in wall of intestine (aging) and stagnation of fecal matter.

Clinical signs

Steady pain in lower abdomen

Abdominal distention

Constipation

Diarrhoea, steatorrhea

Chronic spasm, stasis

Fever

Megaloblastic anaemia

Nutritional Care

Adequate fluid

High fibre diet is recommended to promote motility of the G.I tract (diverticulosis)

In diverticulitis, a low fibre diet is used.

11.8 ULCERATIVE COLITIS

This is a diffuse inflammatory and ulcerative disease of unknown etiology involving the mucosa and sub-mucosa of large intestine/rectum.

Most individuals with ulcerative colitis are nervous, introspective, tense, apprehensive and emotionally tense.

Clinical signs

Mild abdominal discomfort

Urgent need to defecate frequently

Diarrhoea and rectal bleeding

Weight loss-loss of water, electrolytes, blood from the colon causes weight loss, dehydration and anaemia.

Nutritional care

Provide individual attention to the patient.

Cater to the patients food preferences and provide cheerful surroundings during meals.
Provide adequate K. cals *i.e.*, 40 – 50 K. cal/kg ideal body weight.
Provide high amounts of good quality protein *i.e.*, 1 – 1.5/kg of ideal body weight.
Some times patient is intolerant to milk.
Emphasis should be on tender meats, fish, poultry and eggs for those who cannot tolerate milk.
Use a low residue, low fibre diet to reduce the number of stools.
Small frequent meals may be tolerated better than a few large meals.
Supplementary vitamins and minerals are required specially iron and calcium.

11.9 PRACTICAL ASSIGNMENT

AIM: To plan high fibre and low residue diets for disorders of small intestine and colon.

Procedure: Plan a high fibre diet (heavy worker-female) and low residue diet (sedentary worker-male). Calculate energy, protein, fat and fibre content of the diet.

HIGH FIBRE DIET

Recommended dietary allowance

Energy

Protein

Fat

Fibre

FOOD PLAN**Table 11.1 : Total Exchanges Per Day**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Amount</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g) m</i>	<i>Fibre (g)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU**Table 11.2 : Distribution of Exchanges into Meals and Snacks**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>MENU</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Fibre (g)</i>
		Breakfast				
		Midmorning Snacks				
		Lunch				
		Snacks				
		Dinner				
		Bed time				
		Total				

Low Residue Diet

Recommended dietary allowance

Energy

Protein

Fibre

FOOD PLAN

Table 11.3 : Total Exchanges Per Day

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Amount</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Fibre (g)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU

Table 11.4 : Distribution of Exchanges into Meals and Snacks

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Menu</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Fibre (g)</i>
		Breakfast				
		Midmorning Snacks				
		Lunch				
		Snacks				
		Dinner				
		Bed time				
		Total				

ANSWER THE FOLLOWING QUESTIONS

1. Indicate the suitability of the above diets for diseases of small intestine and colon with full justification.

2. What are the points to be highlighted on nutritional management of celiac sprue?

3. Name few disorders and conditions associated with malabsorption.

4. What are the causative factors for excessive amount of exogenous fat in the stool?

Chapter 12

Diet in Diseases of Heart and Circulatory System

Heart diseases are mostly seen in middle aged people. But it can also affect people of different ages caused by atherosclerosis. Diseases of heart may affect.

- The pericardium (outer covering of the heart)
- The endocardium (membrane lining the heart)
- Myocardium (heart muscle)

12.1 DISEASES RELATED TO HEART FUNCTION AND CIRCULATION

If circulation through the tissues and kidneys is impaired, water and sodium are held in the tissue spaces. If compensation occurs, hypertension can result. Edema fluid collects in the extremities and with increasing heart failure. Fluid collects in the abdominal and chest cavities. This is called congestive heart failure.

- If failure to provide oxygen for brain occurs it will result in a stroke – cerebrovascular accident/ stroke.
- If there is a failure to supply blood (oxygen) to the heart, it can lead to coronary heart disease.

COMMON TERMS IN RELATION TO HEART DISORDERS

Arteriosclerosis

Thickening/hardening of the arterial wall.

Atherosclerosis

Soft amorphous lipid accumulation in the intima of the vessel (intima-inner most of the three coats of the artery).

Intimal plaques

This is formed from connective tissue proliferation with the deposition of lipids, cholesterol, blood platelets and calcium.

Thrombus

An enlarged plaque.

Occlusion

Thrombus closes off vessel.

Embolus

A thrombus which has broken off from the arterial wall and is circulating in the blood. If the embolus goes to the liver the embolus can be destroyed. While circulating, the embolus if large enough, is capable of occluding vessels.

Infarct

An area of coagulation necrosis in a tissue due to a local deprivation of oxygen and blood resulting from an obstruction of circulation to the area.

Myocardial infarction

The major cause is coronary thrombosis.

Aneurism

Weakening of vessel wall.

12.2 NUTRITIONAL ASSESSMENT OF CARDIAC PATIENTS

The following parameters are included the nutritional assessment of cardiac patients.

Nutrient Intake and Food Habits

- Diet history
- Food frequency
- Dietary recall

Anthropometric Assessment

- Body weight
- Body height
- Fat fold measurement

Laboratory Assessment

- Serum glucose, glucose tolerance test
- Serum uric acid
- Serum electrolytes
- Serum triglycerides (fasting)
- Serum cholesterol
- Serum lipoprotein profile
- Blood pressure
- Enzyme tests — SGOT, LDH, CPK

Physical Appearance

- Corneal arcus — white ring around the eye.
- Xanthelasma — small yellowish lumps around eyes.
- Xanthomas — fat deposits under skin around joints.

In this chapter, dietary management of hyperlipidemias, congestive heart failure and hypertension will be dealt.

12.3 HYPERLIPIDEMIA

Hyperlipidemia refers to an elevation of plasma or serum lipids such as cholesterol, phospholipids and triglycerides.

Classification of Hyper Lipo Proteinemias

Type	Elevated plasma/serum lipo proteins
I	Chylomicrons
IIA	Low density lipoprotein (LDL)
IIIB	LDL and very low density lipoprotein (VLDL)
III	Beta VLDL (cholesterol rich VLDL remnants)
IV	VLDL
V	Chylomicrons +, VLDL

DIAGNOSIS

Measurement of serum and plasma cholesterol and lipid profile. Risk analysis can be done by cholesterol levels.

Cholesterol Levels and Risk Analysis

Age	Moderate risk	High risk
2 – 19	> 170 mg/dl	> 185 mg/dl
20 – 29	>200 mg/dl	>220 mg/dl
30 – 39	>220 mg/dl	>240 mg/dl
40 +	> 240 mg/dl	>260 mg/dl

12.4 DIETARY MANAGEMENT

Goals

- To achieve and maintain optimal body weight
- To reduce plasma lipids to a normal range
- If hypertension is present, to reduce hypertension by reducing sodium intake
- To individualize drug and diet therapy.

Diet

With the exception of chylomicronemia, a single basic diet can be used to treat the hyper lipoproteinemias. Medium chain triglycerides should be substituted for other fats in the treatment of chylomicronemia.

Energy/Kilo Calories

Achieve and maintain desirable weight.

If the patient is maintaining his weight, his energy requirements can be determined from a food diary or recall.

For a patient who is obese, a reducing diet has to be prescribed.

Loss of body weight can reduce blood pressure.

Loss of body weight can reduce serum triglycerides, cholesterol and plasma glucose.

Fat

Total fat

Less than 30% of total kilo calories is recommended.

There is strong correlation between mean serum total cholesterol concentration and men percentage of kilo calories.

Saturated fat

Less than 10% of total kilocalories is recommended.

Saturated fatty acids increase serum cholesterol.

Palmitic and myristic acids are most effective in elevating serum cholesterol.

Lauric acid also raises serum cholesterol but not to the same extent as palmitic and myristic.

Stearic acid and SFA of 10 carbons or less have no effect on serum cholesterol.

Mono unsaturated fatty acids (MUFA)

10 – 15 per cent of total kilo calories are recommended.

MUFA do not have any effect on serum cholesterol if substituted for dietary carbohydrates.

Olive oil lowers serum cholesterol when substituted for saturated fats in the diet.

High MUFA did not lower HDL-C concentrations as did replacement of SFA with carbohydrates.

Omega-6-poly unsaturated fatty acids (PUFA)

Iso-caloric exchange of PUFA for SFA or MUFA lowered serum cholesterol.

If 10% of K. cal were exchanged for SFA, it lowered serum cholesterol by 13 mg/dl.

Major sources of PUFA are Linoleic and Linolenic acid (corn, cotton seed, safflower and soy oils)

Diets containing more than 7% of K. cal from Omega-6 PUFA are not recommended.

Omega-3 PUFA (Fish oils)

Fish oils reduce serum triglycerides concentration.

Substitution of Omega-3 PUFA for SFA lowers serum LDL.

Without changing SFA, fish oil has no effect on serum cholesterol.

Effect of Hydrogenation of Pufa

Hydrogenation produces trans fatty acids.

Hydrogenated fats generally are not as hyper-cholesterolemic as the solid animal fats.

However, hydrogenated fats are for less effective in lowering total and LDL cholesterol than the liquid vegetable oils from which they are made.

Carbohydrates

50 – 55% of total Kilo calories from carbohydrates is recommended.

Select complex carbohydrates.

Consumption of whole grain bread, cereal products fruits and vegetables are encouraged.

Small increases in soluble fibre intake (approximately 6 gs/day) moderately decreases serum cholesterol concentration regardless of the fibre source.

Insoluble fibre (wheat bran) does not lower serum triglycerides.

Cholesterol

< 300 mgs/day is recommended.

The dietary cholesterol has a definite independent plasma cholesterol raising effect when the diet is high in saturated fat as well.

Consumption of plant sterols are recommended.
Plant sterols interferes with the absorption of cholesterol.

Protein

10 – 20 per cent of total kilo calories is recommended.
If the patient is non-vegetarian, fish, poultry without skin and lean meats are recommended.
Use skim milk.
If the patient is vegetarian, select plant foods of high biological value.

Specific Dietary Guidelines

- Total fat intake should be less than 30% K. cal.
- Saturated fat intake should be less than 10% K. cal.
- Cholesterol intake should be less than 100 mgs/1000 K. cal not to exceed more than 300 mg per day.
- Poly unsaturated/saturated fat ratio should be increased to about 1.0 from the usual value of about 0.3.
- Protein intake should be approximately 10 – 20% K. cal.
- Foods rich in animal fat should be avoided.
- Non-vegetarians should increase fish consumption.
- Non-fat milk and vegetable oils are encouraged.
- Carbohydrate intake should constitute 50 – 55% of K. cal.
- Emphasis should be on complex carbohydrate.
- Sodium intake should be reduced to approximately 1g per 1000 K. cal and not exceed 3 gs/day.
- Avoid alcoholic beverages. If consumed not more than 50 ml of alcohol per day.
- Total K. cal should be sufficient to maintain the individuals best body weight.
- A wide variety of foods should be consumed.
- Supplements of vitamin A and vitamin E should be given.
- A banana a day keeps the doctor away. Banana contains magnesium and it is good for heart.

12.5 CONGESTIVE HEART FAILURE (CHF)

Compensation

If heart is slightly damaged and nearly normal circulation is maintained to all parts of the body it is considered as a period of compensation.

Decompensation

If heart is severely damaged the heart is no longer able to maintain the normal circulation to supply nutrients and oxygen to the tissues or to dispose of CO₂ and other waste. This condition is known as decompensation.

Symptoms

Dyspnea or exertion
Weakness
Chest pain
Hypertension
Edema
Gastrointestinal symptoms like loss of appetite, nausea, vomiting, digestive disorders.

Objectives of Treatment

- Maximum rest to the heart
- Prevention or elimination of edema
- Maintenance of good nutrition.

Dietary Modification

Energy

- Weight management is a critical factor and a first priority.
- Patients with normal weight are prescribed a maintenance level of calories *i.e.*, 1600 – 2000 K. cal.
- 1200 K. cal diet is suitable for an obese patient in bed.

Protein

- Normal protein allowance (1 g/kg body weight).

Fat

- A low fat diet with emphasis on increased amounts of poly unsaturated fatty acids.

Carbohydrates

- Complex carbohydrates should be given.

Vitamins and Minerals

- Normal allowance except sodium.

Sodium

- Degree of sodium restriction mainly depends on condition of individual patient.
- For patient on diuresis, prescribe 1.6 – 2.0 gs.
- Sodium restriction also depends on the capacity to reabsorb the sodium.

Fluid

- Fluid is restricted in advanced CHF.

Frequency of Feeding

- Five or six small meals should be given. This will reduce the burden on the heart.

Type of Food

- Soft non gaseous easily digested food should be given.

PROGRESSION OF DIET

Severe Decompensation

- No feedings on first and second day.
- Liquid foods should be given the next two to three days.
- Omit very hot or very cold beverages.
- Omit caffeine containing foods.
- Sodium should be severely restricted.

After Acute Phase

- Begin 5 – 6 small feedings of easily digested food.
- Two grams of sodium can be given.
- Limit kilo calories to 1000 to 1200 daily.
- Restrict saturated fats and cholesterol.

12.6 PRACTICAL ASSIGNMENT

- AIM:**
1. To plan a modified diet for a patient suffering with cardiovascular disease.
 2. To become familiar with fat, fatty acid and cholesterol contents of foods or groups of foods.

PROCEDURE: Collect case history of a patient suffering with hyperlipidemia from nearby hospital. Analyse the case history critically and formulate nutritional requirements (Energy, fat, cholesterol and sodium) based on the type of hyperlipidemia he has. Plan a day’s menu to suit the patients needs and calculate the nutrients mentioned above *i.e.*, energy, fat, cholesterol and sodium.

CASE HISTORY — HYPERLIPIDEMIA

History of present illness

General information

Relevant past history (illness)

Dietetic history

General examination

Laboratory examination

Vitals and symptoms

Clinical impression

Treatment

NUTRITIONAL THERAPY

Modified RDA

Energy

Fat

Cholesterol

Sodium

FOOD PLAN

Table 12.1 : Total Exchanges Per Day

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Amount (g)</i>	<i>Energy (K. cal)</i>	<i>Fat (g)</i>	<i>Cholesterol (mg)</i>	<i>Sodium (mg)</i>
Milk						
Vegetable A						
Vegetable B						
Fruit						
Cereal						
Meat						
Pulse						
Fat						
Sugar						
Total						

MEAL PATTERN AND MENU

Table 12.2 : Distribution of Exchanges into Meals and Snacks

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Menu</i>	<i>Energy (kcal)</i>	<i>Fat (g)</i>	<i>Cholesterol (mg)</i>	<i>Sodium (mg)</i>
		Breakfast				
		Midmorning snacks				
		Lunch				
		Snack				
		Dinner				
		Bed time				

ANSWER THE FOLLOWING QUESTIONS

1. What abnormal biochemical values your patient has suggests the type of hyper lipoproteinemia?

2. What dietary procedures may be effective in lowering serum cholesterol?

3. Highlight the role of fish oils in the management of cardiovascular diseases.

4. List some common sodium rich foods (> 75 mgs/100 g) which should be avoided in sodium restricted diet.

5. List some common low sodium foods (< 25 mg/100 g) which are suitable for a low sodium diet/sodium restricted diet.

6. Give practical suggestions in brief about ways to increase the palatability of sodium restricted diet.

12.7 TERMINOLOGY

Systolic pressure

This is the maximum pressure in an artery following contraction of the heart (Systole).

This is represented by the first number when blood pressure is recorded.

Diastolic pressure

The pressure in an artery when the heart is relaxed (diastole). This is represented by the second number when blood pressure is recorded.

Hypertension

Persistent diastolic blood pressure greater than 95 mm Hg. Hypertension is not a disease by itself, but is rather a sign of a disease process.

Borderline hypertension

Diastolic pressure between 90 – 95 mm Hg.

Systolic pressure between 140 – 160 mm Hg.

Pulse pressure

It is the difference between systolic and diastolic pressure.

12.8 PRIMARY HYPERTENSION

Primary hypertension is diastolic hypertension for which the cause is not apparent. It can be:

- Mild hypertension with diastolic values to 110 mm Hg.
- Moderate hypertension with diastolic values to 130 mm Hg, and
- Severe hypertension with diastolic values greater than 130 mm Hg.

Mild hypertension can be managed with diet alone.

Severe hypertension requires management with medication coupled with diet.

Management is life long.

Lapses in treatment will result in recurring of hypertension.

Clinical Findings

Primary hypertension itself does not cause symptoms.

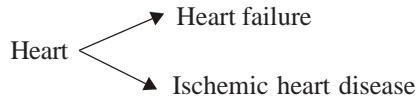
The symptoms are usually the result of the complication of the disease.

Morning occipital (back of the head) headache is seen.

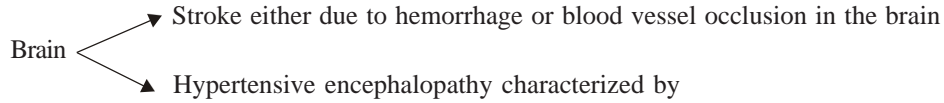
Complications of Hypertension

Hypertension damages

- Heart
- Aorta
- Brain
- Eye
- Kidney

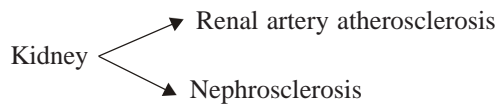


Aorta — Degeneration of medial elastic layer of the aorta, leads to aortic and aneurysm-frequently fatal.



- Severe headache
- Nausea
- Vomiting
- Confusion
- Coma
- Convulsion

Eye — Narrowing of arteries-hemorrhage-formation of exudates-loss of vision.



Necrotizing arteriolar fibrinoid changes leading to renal failure and ureamia.

12.9 MANAGEMENT OF ESSENTIAL HYPERTENSION

Management of hypertension include both

- Life style changes
- Drug therapy

Life style changes include

- A decrease in stress levels
- Dietary control
- Regular isotonic exercise
- Cessation of smoking
- Cessation or reduction of excess alcohol intake.

DIETARY MANAGEMENT

Restriction of Sodium

Sodium restriction is based on severity of hypertension.

In moderate cases, restriction is from 1500 – 2000 mgs per day.

In sodium sensitive individuals the more severe the sodium restriction the greater the decrease in blood pressure.

Sodium and salt content of some sodium restricted diets are given in the following table.

Table 12.3 : Sodium and Salt Content of Sodium Restricted Diet

<i>Modification</i>	<i>Sodium per day</i>		<i>Salt (g)</i>
	<i>mg</i>	<i>meq*</i>	
Severe	250	12	0.6
	500	22	1.3
Strict	1000	43	2.5
Moderate	1500	65	3.8
(No salted foods, and no added salt)	2070	90	5.0
Mild	2400	100	6.0
(no added salt)	2760	120	7.0

* To convert milligrams to milliequivalents divide milligrams by the atomic weight of the mineral.

e.g., $\frac{100}{23}$ mgs of Sodium = 4.35 meqs of Na.

Sources of Sodium

Naturally occurring sodium in foods

Foods high in sodium do not taste salty.

But these foods must be consumed in measured amounts.

Sodium added to foods

One tea spoon ful of salt is equivalent to 2000 mgs of sodium.

Cooking

Use reduced amount of salt in cooking for moderate restriction of sodium.

For severe restriction, do not use salt in cooking at all.

Select recipes using other combinations of seasonings as salt substitutes.

Do not keep salt on the table.

Food processing

Do not consume pickled, brined canned vegetables. Avoid baked foods.

Additives

Do not use baking powder, baking soda, sodium citrate, monosodium glutamate.

Sodium in drugs

Some drugs contain high sodium content.

Beware of such drugs.

Weight Control

Weight control is essential if the person is obese by prescribing weight reducing diets.

For each 10 kg excess body weight a 3 mm Hg increase in blood pressure is seen and vice versa.

Adults who develop obesity are more at risk than persons who have been obese since childhood.

Adult onset obesity is mainly upper body obesity.
Persons with upper body obesity are more likely to develop adult onset diabetes mellitus.
This leads to high levels of insulin secretion and insulin resistance.
High insulin levels leads to retention of sodium and counter regulatory hormone secretion.
Counter regulating hormones (both corticosteroids and growth hormones) are capable of causing hypertension.

Restriction of Fat Consumption

Restriction of saturated fat consumption is very essential.
This will also decrease the chances for the development of atherosclerosis.

Potassium and Calcium Intake

Increase potassium and calcium intake particularly in the form of fruits and dairy products.
Some diuretics like thiazides, thiazide derivatives, furosamide and ethacrynic acid decrease potassium levels. Hence, specific potassium supplements should be given.
Potassium supplements are contra indicated when spiranolactone or triampheterine are prescribed.
These diuretics have potassium sparing action.

MANAGEMENT OF SECONDARY HYPERTENSION

The management of secondary hypertension is undertaken based on the management of the underlying disease condition. When this management does not lead to correction of hypertension, treatment is undertaken as per primary hypertension.

12.10 PRACTICAL ASSIGNMENT

- AIM:**
1. To plan a modified diet for a patient suffering with hypertension.
 2. To learn about sodium content of food groups and recognize foods high and low in sodium.
 3. To know about processing procedures which increase the sodium content of foods.

PROCEDURE: A case history of a patient suffering with hypertension is given below. Analyse the case history critically and formulate nutritional requirements. (Energy, protein, fat, sodium and carbohydrates). Plan a day's menu to suit the patients needs and calculate the nutrients mentioned *i.e.*, energy, protein, fat, sodium and carbohydrates.

CASE HISTORY — HYPERTENSION

Mr. Anil Kumar had a blood pressure of 156/100 mm Hg. He went to a local physician on several occasions. His blood pressure varied from 145/95 to 160/110 mm Hg. His physician placed him on thiazide diuretic and sent him to you for dietary advice. In his chart you find the following data.

Anthropometric measurements

Weight : 210 lbs
Height : 5'11"

Laboratory analysis

Blood levels

Sodium : 138 meq/litre
Potassium : 3.9 meq/litre

Calcium	: 9 mg/litre
Magnesium	: 1.8 mg/dl
Renin levels (resting)	: 0.8 mg/ml/hr
Creatinine	: 1 mg/dl

On interview, you find that the patient is a factory worker, who carries his lunch to work. His wife does all the shopping, cooking and both he and she prefer South Indian food.

His favourite foods all contain salt. He enjoys baked foods, salty pickles and fried foods. He does not care for most fruits and vegetables, but likes beans and greens. He enjoys a drink before dinner. His work does not require much moving around but he does stand for about 7 hrs/day. He finds this tiring and does not feel that he has any energy left for exercise after work. He smokes 1^{1/2} packs of cigarettes per day.

ANSWER THE FOLLOWING QUESTIONS

1. What type of hypertension does Mr. Kumar have? and why?

2. What factors in his life style might have contributed to his hypertension?

3. What dietary measures would you advise him and his wife to take?

4. List out food products that contain sodium additives and figure out appropriate substitutes?

5. List out high potassium foods which are low in sodium.

Modified RDA for moderately active, obese and hypertensive patient.

Male :

Energy :

Carbohydrate :

Protein :

Fat:

Sodium :

FOOD PLAN

Table 12.4 : Total Exchanges Per Day

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>Amount (g)</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>CHO (g)</i>	<i>Sodium (mg)</i>
Milk							
Vegetable A							
Vegetable B							
Fruit							
Cereal							
Meat							
Pulse							
Fat							
Sugar							

MEAL PATTERN AND MENU**Table 12.5 : Distribution of Exchanges into Meals and Snacks**

<i>Food exchange</i>	<i>No. of exchanges</i>	<i>MENU</i>	<i>Energy (kcal)</i>	<i>Protein (g)</i>	<i>Fat (g)</i>	<i>Carbohydrate (g)</i>	<i>Sodium (mg)</i>
		Breakfast					
		Midmorning Snacks					
		Lunch					
		Snacks					
		Dinner					
		Bed time					

Chapter 13

Diabetes Mellitus and Management

Diabetes mellitus is a disease characterized by chronic hyperglycemia and glycosuria produced by an absolute or relative insufficiency of insulin. There will be striking changes in the metabolism of fat and protein and disorders in the structure and function of blood vessels. The early symptoms are usually related to the metabolic abnormalities and later developments involve complications with blood vessels such as retinopathy, nephrosclerosis and atherosclerosis.

13.1 PHYSIOLOGY OF NORMAL GLUCOSE HOMEOSTASIS

The chief hormones involved in glucose homeostasis

- Insulin
- Glucagon
- Epineprine
- Corticosteroids
- Growth hormone
- Somatostatin

Insulin is responsible for lowering blood glucose levels, while the other competent hormones act to raise them. Under normal circumstances, these hormones act to maintain blood glucose in 80 – 110 mg/dl range in the face of fluctuating intakes of carbohydrates.

13.2 INSULIN AND GLUCOSE METABOLISM

The process of insulin biosynthesis begins in the beta cells located in the islets of Langerhans of the pancreas. The insulin precursor, proinsulin is a high molecular weight protein synthesized in the rough endoplasmic reticulum. After transport to the plasma membrane, proinsulin is split, yielding insulin and c-protein which are secreted into the blood. C-protein has a long half life in the blood, so levels of this protein can serve as an indicator of the amount of insulin produced.

Insulin is required for entry of glucose into a large number of tissues, though tissues such as brain and kidney do not require insulin. Insulin sensitive tissues have receptors for the hormone on their cell surfaces. When insulin binds to these receptors glucose enters into the cell.

13.3 INSULIN SENSITIVE AND INSENSITIVE TISSUES

Insulin sensitive tissues : Muscle, fibroblasts, mammary gland, adipose tissue, anterior pituitary, lens of the eye, aorta, leukocytes.

Insulin insensitive tissues : Kidney tubules, intestinal mucosa, erythrocytes, liver, nerve tissues, brain (except part of hypothalamus).

13.4 RECEPTORS ON CELL SURFACES

Insulin appears to modulate its own surface and intracellular receptors. Low levels of insulin in the extra cellular fluid increase the number of circulating insulin receptors on cell surfaces. High levels of circulating insulin tend to decrease the number of receptors, thereby diminishing the ability of the cells to transport glucose.

This decrease is called down regulation or down modulation. Other factors which cause down regulation of insulin receptors include antireceptors, antibodies and elevated levels of free fatty acids.

Insulin promoters

- Tissue glycolysis
- Fatty acid synthesis
- Intracellular deposition of glucose through stimulation of glycogen synthesis.
- Insulin accelerates hepatic glycolysis by increasing levels of the enzymes glucokinase.
- Phospho fructokinase and suppressing levels of enzymes glyconeogenic pathways in particular phospho end pyruvate carboxykinase.

13.5 INSULIN AND TRANSPORT OF OTHER NUTRIENTS

Insulin causes active transport of branched chain amino acids (valine, leucine and isoleucine) and the aromatic amino acids phenylalanine and tyrosine into cells.

Insulin encourages protein anabolism. This hormone also increases the transcription of DNA and translation of RNA, both required for protein synthesis.

Of the several pathways of glucose metabolism which are insulin insensitive too are of major pathological importance in the diabetic.

One is the formation of glycosylated hemoglobin.

This forms the basis of a common test for “control” in diabetes.

R.B.C. stay in circulation for 120 days.

During that time, the hemoglobin they contain becomes progressively more glycosyated.

This glycosylation reflects the mean levels of glucose to which RBC has been exposed

The second pathway is the formation of sorbitol.

In the presence of hyperglycemia toxic accumulation of sorbitol may occur in insensitive tissues such as Schwann cells.

Nerve myoinositol uptake is inhibited. The accumulation of polyols leads to nerve dysfunction.

13.6 CLASSIFICATION OF DIABETES MELLITUS AND GLUCOSE TOLERANCE

- Insulin dependent diabetes mellitus (IDDM – Type IA and Type IB)
- Non Insulin dependent diabetes mellitus (NIDDM – Type II)
- Impaired glucose tolerance
- Gestational diabetes mellitus
- Secondary diabetes mellitus.

Clinical signs and symptoms

Insulin dependent Diabetes Mellitus

Type IA (young) – Polyphagia, polydipsia, polyurea, Hyperglycemia, ketonemia, glycosuria ketoacidosis, ketonuria – may develop rapidly and the patient needs immediate medication.

Type IB (older) – Intermediate between Type IA and Type II
Rarely develops ketoacidosis

Nonspecific symptoms
Hyperglycemia, Glycosuria.

Non Insulin dependent Diabetes Mellitus (Type II)
poly-dipsia, poly urea, polyphagia, fatigue, weakness, blurred vision, frequent infection (with the fungus candida albicans)
Impotence, ketoacidosis rarely occurs

Table 13.1 : Comparison of IDDM and NIDDM

<i>Parameters</i>	<i>IDDM (Type I)</i>		<i>NIDDM</i>
	<i>Type IA</i>	<i>Type I B</i>	<i>(Type II)</i>
Primary Pathology	Pancreatic beta cell insufficiency		uncoordinated insulin secretion and organ unresponsiveness to Insulin. Insulin secretion inadequate for needs.
Plasma insulin levels	Nil	Nil	Low or normal
Insulin receptors	Normal	Normal	Low or normal
Usual age at on set	< 20 years	< 40 years	> 40 years
Prevalence among all diabetics	10%	10%	80%
Genetic basis	Yes	Yes	Yes
HLA (tissue type) association	Yes	Variable	No
Islet cell antibodies	50–80%	Positive at diagnosis	Less than 5% positive at diagnosis
Environmental factors	Infection	Probably infection	Obesity (truncal)
Obesity at on set	Rare	Variable	Common
Appearance of initial symptoms	Rapid	Variable	Slow
Ketoacidosis	Frequent	Rare	Rare
Nonketotic hyperosmolar coma	Uncommon	Uncommon	Common

13.7 DIAGNOSIS AND MONITORING OF DIABETES MELLITUS**THE GLUCOSE TOLERANCE TEST (GTT)**

The glucose tolerance test is designed to determine the blood glucose response at given intervals after consumption of parenteral injections of a set amount of glucose. This test is mainly used to

- Detect diabetes mellitus
- To follow diabetes mellitus

There are two types of GTTS namely the oral GTT and Iv GTT

The oral GTT – Standard amount of glucose is administered by mouth.

Iv GTT – Glucose load is administered intravenously.

Blood is drawn prior to the administration of glucose (fasting) and thereafter 1 hour, 2nd and 3rd hour and glucose is estimated

Table 13.2 : Glucose values above which the OGTT may be considered abnormal (mg/dl)

<i>Duration</i>	<i>Whole blood</i>	<i>Serum or Plasma</i>
Fasting	110	130
1 hour	160	185
2 hour	120	140
3 hour	110	0

The validity of glucose tolerance test depends on number of factors.

- Diet prior to testing. A high fat diet preceding the GTT leads to relatively poor glucose tolerance particularly in elderly.
- Emotional state : Anxiety associated with the test leads to the release of epinephrine which in turn leads to glucose release from the liver.

13.8 MEASUREMENT OF GLYCOLYLATED HAEMOGLOBIN LEVELS

Persistent hyperglycemia results in glycosylation of many proteins, a process in which glucose becomes covalently bound to lysine residues of the proteins, the best studied is haemoglobin where glycosylation results in haemoglobin A_{1c}. Because the glycosylation is rapid and largely irreversible, Hgb_{1c} in erythrocyte accumulates over the 120 days of cell life span. HgbA_{1c} levels therefore reflect the average blood glucose levels present four to six weeks earlier. Clinically HgbA_{1c} levels are measured indirectly by measuring the total levels of HgbA₁ of which HgbA_{1c} is a fraction. HgbA₁ levels are useful for long term assessment of control or maintenance of normal blood glucose levels.

Table 13.3 : Haemoglobin A_{1c} and Haemoglobin A₁ Values in normal Adults and Diabetics

	<i>HgbA_{1c}</i>	<i>Hg bA₁</i>	<i>Mean plasma glucose</i>
Normal	3.8 – 6.3%	5 – 9%	90 – 110mg/dl
Diabetic tight control	< 7.5%	< 9%	< 160 mg/dl
moderate control	5 – 9.5%	9 – 11%	160 – 230 mg/dl
Fair/poor control	9.5 – 12%	11 – 14%	230 – 310 mg/dl

13.9 MONITORING URINE GLUCOSE AND KETONE LEVELS

Glycosuria is usually measured by

- Clinitest (Modification of Benedict's test)
- Clinistix, Testape, Diastix (Paper strip method)

Both methods are dependent upon a normal glomerular filtration rate (120ml/min) and renal threshold for glucose reabsorption by the tubules (180 mg/dl)

Ketonuria

Ketonuria is quantitatively detected by reacting the acetoacetate in the urine sample with nitro pruside and 10mm acetone (ketostix, acetest, labstix). Beta hydroxy butyrate which lacks a ketone group does not react with nitropruside. In mild cases of ketonemia the ratio of beta hydroxybutyrate to acetoacetate is about 1. In severe cases of keto acidosis the ratio may go as high as 6.

Patients with impaired kidney function and glomerular filtration rates may have ketonemia without ketonuria.

Self Blood Glucose Monitoring

Blood, rather than urine glucose levels are the levels of physiologic significance in diabetes.

Urine glucose levels lag behind blood levels and are dependent on renal function. Via self (or home) assess blood glucose measurement, the patient can blood glucose profile over 24 hours and detect both hyperglycemia and hypoglycemia.

To obtain the blood, the patient pricks the tip of a finger.

The blood is then applied to a test strip containing the glucose oxidase reagents and a chromogen, the colour of which changes with the concentration of glucose in the blood.

The colour obtained can either be read by eye or with a meter.

There is an upper limit to the concentration of glucose measurable using the various test strips (250 mgs/dl Dextrostix)

13.10 A NEW NON INVASIVE TECHNIQUE FOR BLOOD GLUCOSE MONITORING

It involves a small laser light and a detector. The absorbance of the blood at a certain wave length depends on the concentration of glucose in the blood.

This absorbance can be measured by sending light of the appropriate wave length through the tip of the finger and measuring the amount of the light that has been absorbed.

13.11 MANAGEMENT OF DIABETES MELLITUS

Although it is not possible to cure diabetes completely, diabetics can lead almost a normal life if they follow certain do's and don'ts scrupulously. Cooperation of the patient is very important in the management of diabetes.

The main modes of treatment of diabetes are

- Diet
- Exercise
- Drugs
- Education

Dietary management

A well designed meal plan is an important cornerstone in the management of diabetes mellitus.

13.12 GOALS OF NUTRITIONAL THERAPY

- Achieve physiologic blood glucose levels
- Maintain desirable plasma lipid levels
- Reduce complications of diabetes mellitus
- Retard development of atherosclerosis
- Provide optimal selection of nutrients
- Attain and maintain desirable body weight
- Meet energy needs in a timely manner.
- Individualize to preferences and food available.
- Address special requirements (such as pregnancy)
- Tailor for therapeutic needs (such as renal failure)

The nutrition plan

Calories

An excessive calorie intake results in weight gain and obesity

Obesity is an important factor in terms of target cell resistance to insulin action.
 Attainment of ideal weight results in reduced glucose levels and an increase in target cell response to insulin.
 With moderate obesity, there is a four fold increase in diabetes mellitus.
 With severe obesity, a tenfold increase was observed.
 Calorie requirement depends on the weight of a person.

Table 13.4 Calorie Requirement Based on Weight/age.

<i>Category</i>	<i>Calorie requirement</i>
Over weight	20 Kcals/Kg/day
Ideal weight	30 Kcals/Kg/day
Under weight	40 Kcals/Kg/day
Elderly person above 50 years	10% less calories for each additional decade
Children - 1st year	1000 calories
For girls 1 – 12 years	1000 + 100 calories per year of age upto 12 years.
For boys 1 – 12 years	1000 + 125 calories per year of age after 12 years

Thus the calorie content of the diet for all diabetics should be set at a level which will permit them to maintain their desired body weight and in children and adolescents allow for a normal rate of growth and development.

Carbohydrates

Recommendation with regard to optimum carbohydrate content of the diet for diabetes is controversial. The present recommendation is to provide **generous amounts of complex carbohydrates and fibre and restricted fat.**

Carbohydrates should provide 50 – 60 per cent of energy

Complex carbohydrates should account for approximately 2/3 of total carbohydrate. Among this,

60 – 70% should be complex carbohydrate

30 – 40% should be simple carbohydrate

Advantages

Stimulates glucose use (glycolysis and glycogenesis) in many tissues.

Attenuates hepatic glucose production.

Increases tissue insulin sensitivity.

Increases insulin receptor number.

Lowers post prandial serum triglyceride

Disadvantages

May increase post prandial plasma glucose

Temporarily may worsen glycemic control

Tend to increase fasting serum triglycerides.

Type of carbohydrate

It is the type of carbohydrate that determines the glycemic response than the actual quantity.

Simple carbohydrates from commonly used food tend to raise blood glucose more than complex carbohydrates from starchy foods.

The glycemic response to 50g of glucose is much greater than the response to a variety of foods providing 50 g starch.

Glucose, maltose, and sucrose produce large increase in blood glucose but fructose does not. Fructose can be used as a sweetener for diabetic patients.

Glycemic Index

Glycemic index of a food is determined by measuring the area under blood glucose curve obtained by consumption of the test food expressed as a percentage of the area obtained by giving the same quantity of carbohydrates in the form of glucose (50 gs).

Different complex carbohydrates evoke different glycemic responses.

Bread or potatoes raise blood glucose more than beans.

Low glycemic index of Beans may be due to

- High soluble fibre content
- Food form
- Naturally occurring starch blockers.

Not only different complex carbohydrates but in different form also evoke different glycemic response.

Factors affecting glycemic response to food

- Rate of insulin
- Prestomach hydrolysis
- Stomach hydrolysis
- Gastric emptying rate
- Intestinal hydrolysis and absorption
- Physical form of food
 - Consistency of food – whether liquid, puree or solid
 - Raw or Cooked,
 - Whole or chopped.
- Nature of starch
 - Amylose to Amylopectin ratio
 - Amylopectin is shown to be digested more quickly than amylose.
 - Glucose chains of amylose are more bound to each other by hydrogen bonds making them less available
 - Legumes contain 30 – 40% of amylose and 60 – 70% of amylopectin
- Cooking method
 - Ground rice produces greater glucose response than unground rice amylopectin. Boiling and pressure cooking of legumes resulted in faster rates of digestion than roasting
- Antinutrients
 - Phylates, lectins and enzyme inhibitor may lower the glycemic response of food.
 - Acarbose an enzyme inhibitor present only in wheat reduces blood glucose response.
- Fibre content
- Food ingredients
- Pancreatic hormones
- Gut hormones

Fibre

The therapeutic value of fibre for diabetes became evident over the last decade. High fibre intake improves glycemic control and reduces insulin requirements. It is the soluble fibres that are more effective in producing favourable effect in carbohydrate and lipid metabolism when compared to insoluble fibres.

13.13 MODE OF ACTION OF FIBRE

Soluble fibre prolongs the rate of and gastric emptying and intestinal transit time. It forms a gel with water and thickens the unstirred layer

Carbohydrates are thus packed and insulated from the action of the digestive enzymes in the intestines, thus reducing the rate of absorption.

Insoluble fibre reduce gastric emptying and intestinal transit time. High fibre diet enhances the response of gastric inhibitory polypeptide (GIP) which is a stimulus for insulin secretion.

There is an enhanced plasma somatostatin release after a fibre rich diet. Somatostatin delays the absorption of carbohydrates from the small intestine.

Advantages

- Slows nutrient digestion and absorption
- Decreases post prandial plasma glucose
- Increases tissue insulin sensitivity
- Increases insulin receptor number
- stimulates glucose use
- Attenuates hepatic glucose output
- Decreases counter regulatory hormone release (such as glucagon)
- Lowers serum cholesterol
- Lowers fasting and post prandial serum triglycerides
- May attenuate hepatic cholesterol synthesis

Disadvantages

- Increases intestinal gas
- May cause abdominal discomfort
- May alter availability of minerals

People at risk of deficiencies i.e. post menopausal women and elderly should be careful with the intake of fibre.

Optimum level of fibre would be 25/g per 1000 calories High fibre intakes are not recommended for individuals with inflammatory bowel disease.

Protein

Protein should provide 12 – 20% of energy intake

An additional 30 g may be necessary during pregnancy and lactation

Protein requirement is increased in malnutrition, surgery or wound healing.

In insulin dependent diabetics adequate dietary protein (1 – 1.5g/kg body weight) is necessary for growth and development.

In diabetic nephropathy, protein restriction may vary between 0.4 – 0.6 g per kilogram

The source of protein is as important as amount.

Protein from vegetable source is preferable to that from animal sources.

Fat

The amount and type of fat plays an important role in the diet of a diabetics. Diabetes of all types have a greater incidence of hyperlipidemia and atherosclerosis than do the nondiabetics fat intake should be monitored carefully.

High fat diets offer short term benefits for glycomic control and have no adverse effects on insulin requirements over two or three weeks.

But long term use of high fat diets has many metabolic disadvantages like

- Increased risk of heart attacks
- Cause insulin resistance
- Impair intracellular glucose metabolism
- Decreases the number of insulin receptors
- Rate of glycolysis is lower.
- Glycogen synthesis, glycogen accumulation and glucose oxidation are lower
- High serum levels of free fatty acids are seen

The present recommendation of total fat is 20 – 30% of total calories.

Saturated, monosaturated and polyunsaturated fatty acids are given in the ratio of 1 : 1 : 1

- Saturated fats – less than 10%
- polyunsaturated fats – 6 – 8%
- Monounsaturated fats-remaining fat

Dietary cholesterol should be less than 300 mgs/day.

In severe hypercholesterolemia, it may be necessary to restrict dietary cholesterol to 100 – 150 mg/day. These recommendations should be further modified to accommodate the nutritional management of accompanying disorders such as obesity and hypertension.

Sweeteners : The use of modest amount of sorbitol, mannitol or xylose probably poses no risk to diabetic or non diabetic individuals.

Fructose offers advantages over sucrose,

- It tastes sweeter
- Is metabolised without insulin
- Produces less hyperglycemia

Long term safety of fructose for person with diabetes is not established. Non caloric or non nutritive sweeteners enjoy wide use in beverages and other products. Pregnant women and children should avoid saccharin.

PRACTICAL ASSIGNMENT

Aim : To plan modified diets for the following diabetic patients

- A school going insulin dependent diabetic patient
- A 25 year old pregnant mother suffering with gestational diabetes mellitus
- A fifty five year old non insulin dependent diabetic suffering with kidney failure
- Middle aged obese diabetic women.

Procedure : Collect case histories of above patients from diabetic clinics. Analyse the case histories critically and formulate nutritional requirements and cycle menus to meet the patients needs. Prepare consolidated report for each patient with all the above particulars giving necessary dietary counselling tips.

ANSWER THE FOLLOWING QUESTIONS

1. What are the complications and risks of uncontrolled diabetes mellitus ?

2. Name five foods which have low glycemic index ?

3. Mention five foods which can be consumed in unlimited amounts by a diabetic ?

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