UNIT 17 NUTRITIONAL MANAGEMENT OF NEUROLOGICAL DISORDERS

Structure

17.1 INTRODUCTION

Your friend's aged father, over a period of time has become very confused, forgetful and is unable to do many things he could do earlier. He has also developed eating problems. Laxmi's baby suffered from anoxia (lack of oxygen) at birth which has led to developmental impairment and difficulty in chewing and swallowing. Ramesh suffered spinal injury in a road accident. This has resulted in a partial paralysis with associated impaired feeding ability. As a nutrition expert, how do you manage these situations? First of all, you must understand that all of these occur as a consequence to neurological problems. In this unit, we will provide you with the salient guidelines for the better management of these disorders.

Objectives

After studying this unit, you will be able to:

- identify some common neurological disorders, their etiology and clinical features,
- explain the consequences of these disorders on feeding and nutrition, and
- **suggest** feeding and dietary recommendations to meet the needs of these disorders,

Let us begin our discussion by first understanding what are the different types of neurological disorders.

17.2 COMMON NEUROLOGICAL DISORDERS

Neurological disorders may be of two types from the nutritional view point. What are these? Let us read and find out.

i) *Neurological disorders arising due to imbalanced nutritional intake (deficiency or excess)* – Common examples are the neurological manifestations of beriberi, pellagra, pernicious anaemia, Wernicke Korsakoff syndrome due to nutrient deficits and stroke, hypertension and diabetes due to nutrient excesses, unbalanced diet leading to malnutrition. Alcoholism and malabsorption could also be other causative factors.

ii) *Neurological disorders of non-nutritional etiology* – Some of the common disorders are Alzheimer's disease, Parkinson's disease, epilepsy, spinal and neuro trauma. Dysphagia (difficulty in swallowing), though not a disease, is a symptom which may occur in several neurological disorders.

In this unit, we will focus on the common neurological disorders of non-nutritional etiology. Before we move on to their discussion, let us quickly review a few relevant aspects about Central Nervous System (CNS).

17.3 THE CENTRAL NERVOUS SYSTEM (CNS) – SOME RELEVANT PHYSIOLOGICAL ASPECTS

A brief overview of some of the consequences of CNS damage will help us to understand neurological disorders better. Refer to Figure 17.1. This figure will help you recall the major components and parts of the CNS i.e the brain and the spinal cord, about which you have already studied in the **Applied** Physiology Course (MFN-001) in Unit 9.

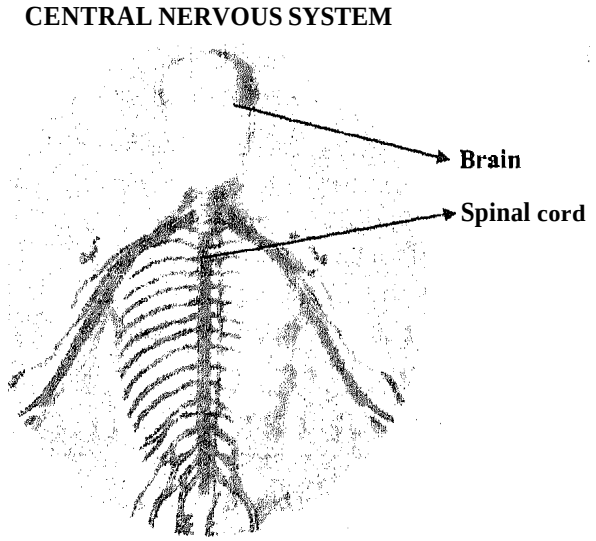

Figure 17.1: Structure and components of CNS

Lesions of different parts of the CNS can result in different dysfunctions with different nutritional significance. Signs of weakness are the most quantifiable clinical symptom of any neurological disease. Any damage to any part of the CNS results in the inability of the body to meet its nutritional and metabolic needs, Some consequences of CNS damage of nutritional significance are given in the Table 17.1. Figure 17.2 illustrates the lobes of the cerebral cortex which may get damaged.

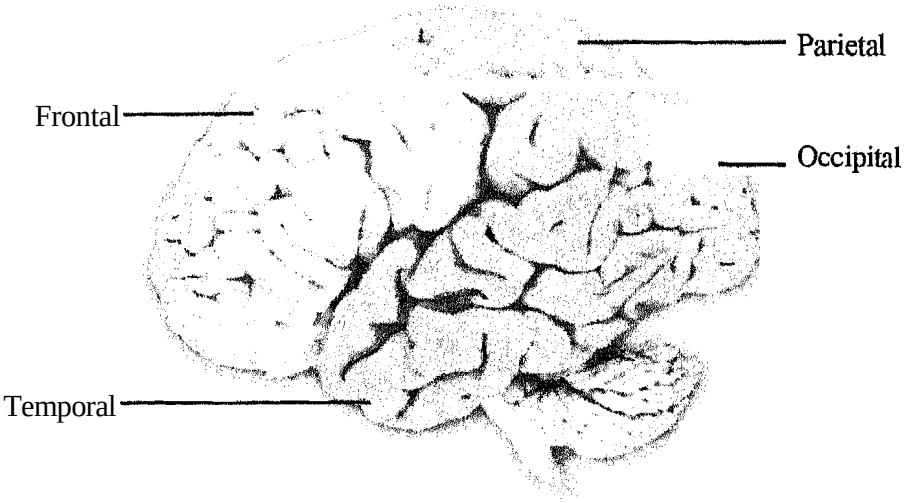

Figure 17.2: Lobes of Cerebral Cortex

Table 17.1: Some consequences of CNS damage of nutritional significance

Area of Damage	Consequences
Frontal lobe-base	Loss of sense of smell, visual changes
Frontal lobe-central	Motor apraxia or inability to carry out a complex activity, inspite of understanding it
Frontal lobe-posterior	Seizures (convulsions)
Temporal lobe	Memory and speech impairment
Occipital lobe	Vision deficits
Brain stem	Damage of cranial nerves which innervate face and head, including eyes, ears, jaws, tongue, pharynx and facial muscles. Dysphagia and aspiration risk can occur.
Hypothalamus	Damage of center for hunger and satiety, leading to overeating or anorexia problems.
Spinal Cord	Motor impairment/ paralysis based on location of injury with resultant feeding difficulties.
Peripheral nerves and neuromotor junction	Impaired nutritional and metabolic balance.

What are the nutritional issues related to neurological disorders? What should be the goals of nutritional care? How to feed such patients where dysphagia is the commonest problem? Let us read and find out about these and many other issues next.

17.4 NEUROLOGICAL DISEASES: FEEDING AND NUTRITIONAL ISSUES - GENERAL GOALS OF NUTRITIONAL CARE

Nutritional management of the patients with neurological disease is complex, as mechanisms and abilities needed for adequate nourishment get impaired. Reduced functional capacity would impair the ability to procure and prepare food. Self feeding impairment may arise due to limb weakness, poor positioning of the body due to hemi paresis or partial paralysis. Hemianopsia or half sided blindness, apraxia (inability to perform), mental confusion, fatigue and early satiety can affect feeding. Weakness of the tongue, facial and masticator muscles can lead to prolonged feeding time and coughing or choking while eating. Chewing and swallowing difficulties could also arise. Emotional and metabolic stress and trauma can compound the eating and nutritional problems as these can have an effect on the nutritional requirements.

In view of the long term disabling consequences, most neurological disorders require special rehabilitative care. Personalized nutrition care plays a vital role in this rehabilitation, for healing and recovery, along with physical, mental and social support and involvement of the patient and the family.

Let us then move on to the nutritional care. The important goals of nutritional care are to:

- prevent further disability,
- restore or achieve optimal potential of the patients physical, mental and social abilities, and
- improve the quality of life of the patient.

In order to meet these goals, the following are necessary:

- Assessing the nutritional, physical and other parameters of the individual and monitoring the same at intervals to assess the improvement.
- Regular evaluation to improve patient outcome in term of quality and quantity of food consumed, weight changes, clinical assessment.
- Eating and feeding assessment to identify need for special foods or eating devices as illustrated in Figure 17.3.
- Dysphagia assessment for textural and other food or feeding modifications.
- Nutritional therapy recommendations based on individual needs. For example, nutritional deficiencies like anemia may have to be identified as iron is required by the neurotransmitters serotonin and dopamine.
- Nutrition counseling of the patient, family and caregiver.

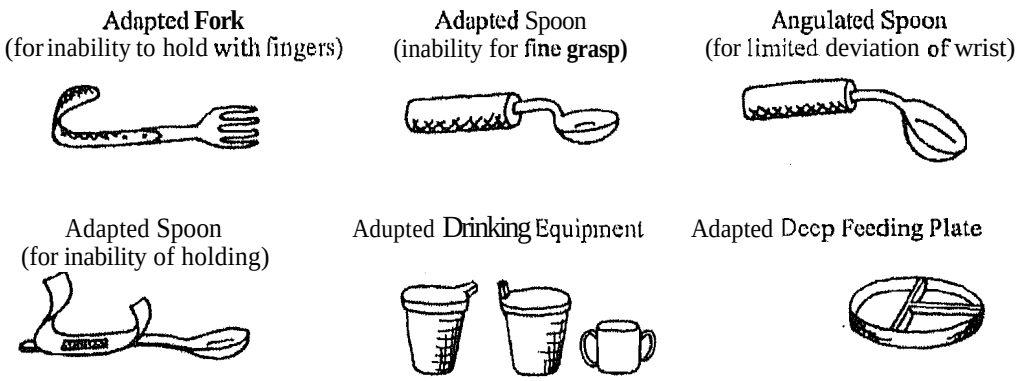

Figure 173: Examples of modified eating equipments

A few additional points related to the patient care that need to be kept in mind include paying attention to the patients living situation, facilities, help, economic status, psychosocial aspects and emotional adjustments. Neglect or overprotection should be avoided to help develop a positive sense of self and the will to fight against a difficult situation. A combined effort of the rehabilitation team including the occupational and speech therapists is desirable, besides the nutrition professional. Next, let us move on to the most common neurological disorder, that is dysphagia. But before that, let us quickly review what we have learnt so far.

Check Your Progress Exercise 1

1. Enlist three common neurological disorders of

   a) nutritional origin .....

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   b) non-nutritional origin: .....

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2. State five consequences of neurological damage which can affect feeding or nutrition.

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3. Give two important goals of nutritional care of persons with neurological disorders.

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4. Match the following:

	A		B
i)	Occipital lobe	a)	Loss of sense of smell and visual changes
ii)	Frontal lobe-base	b)	Feeding difficulties
iii)	Temporal lobe	c)	Vision deficits
iv)	Spinal Cord	d)	Impaired nutritional and metabolic balance
v)	Peripheral nerves	e)	Memory and speech impairment

17.5 DYSPHAGIA

Dysphagia is the inability to swallow or *difficulty* in swallowing. It is a common problem in those with neurological disorders and can occur in any of the three phases of swallowing: oral, *pharyngeal* and oesophageal. Many patients may not be able to consume adequate intake of food and hence special feeding methods such as enteral feeding may have to be adopted.

What is the cause of dysphagia? The etiology and the clinical sign/symptoms are described next.

17.5.1 Etiology and Clinical Features

The cause of this neurological disorder can be mechanical or paralytic. The mechanical cause is primarily due to surgical resection or to alteration of one or more organs of swallowing owing to trauma, obstruction, cancer or other disease. The *paralytic* type results from **a** lesion in the cerebral cortex or cranial nerves of the brain stem in particular the medulla oblongata. The most common cause of the paralytic type is accident, head injury, brain diseases which may affect the nerves causing dysphagia.

If we understand the swallowing process, we can understand the problems that arise in neurological damage better. So let us get to know more about the swallowing process. Swallowing has three phases:

3 Oral Phase: In this, food is placed in the mouth, mixed with saliva, chewed if required and formed into a bolus by the tongue. The tongue pushes the food to the rear of the oral cavity against the hard and soft palate.

ii) *Pharyngeal Phase*: This is initiated when the bolus is propelled into the oesophagus. The soft palate gets raised at this stage to close off the nasopharynx to prevent nasopharyngeal regurgitation.

iii) *Oesophageal Phase*: This is the final phase in which the bolus continues through the oesophagus into the stomach. This phase is involuntary.

In neurological disorders, damage to cranial nerves can lead to weak and poorly co-ordinated tongue movements, which results in problems in completing the oral phase of swallowing. Weakened lip muscles cause an incomplete mouth closure with sucking difficulties. There may be a difficulty in forming a cohesive bolus and moving it through the oral cavity. Loss of sensation and facial weakness can result in food being pocketed in the buccal recesses of the mouth, without the patient being aware of it. Poorly co-ordinated pharyngeal phase can cause gagging, choking and nasopharyngeal regurgitation. Aspiration of food into the lungs is a complication of dysphagia, unless care is taken.

Thus, some symptoms commonly associated with dysphagia are drooling, coughing, and choking while eating, oral food retention, gurgling voice quality and feeling of lump in the throat. There is an increased risk of aspiration and pneumonia.

Having studied the etiology and symptoms, let us next learn about the feeding and nutritional management of dysphagia,

17.5.2 Feeding and 'Nutritional Management

A multidisciplinary approach is essential for successful management of dysphagia. Patient, nurse, physician, dietitian and swallowing therapist need lo coordinate in this endeavour. For feeding, patient should be upright at 90 degree angle with hips flexed, feet flat on the floor and head slightly forward. Food of liquid consistency often causes the greatest problem. Hence, attention has to be paid to the consistency and texture of food as enumerated herewith.

Consistency and Texture: Aspiration of liquids, including water, into the lungs can result in complications. To avoid this problem and yet fulfilling the fluid needs, liquids could be thickened with starch or milk powder. Intake of caffeine beverages is best limited as these have a diuretic effect and may lead to mild dehydration and fatigue. Foods that form a cohesive bolus within the mouth must be selected. Those that break apart such as plain rice, chopped meat should not be given. Sticky foods that adhere to the mouth should be avoided as they cause oral manipulation problems and fatigue. Dual texture must not be given such as canned fruit with juice, soup with noodles, and dry cereal with milk. One could moisten food with gravy to facilitate a bolus.

A variety of food items should be served in an appetizing manner with as many characteristics to a normal food. Patients should bc gradual progressive transitions in the texture and consistency of food viz., pureed to ground to soft textured foods and eventually to all textured foods.

Hence, paying attention to improved and appropriate taste, texture and temperature of food facilitates swallowing. Cool temperature, sauces and gravies which lubricate food and prevent its fragmentation in the oral cavity, makes swallowing easier. Moist and soft foods are usually better tolerated. Table 17.2 and 17.3 summarize some feeding tips for dysphagia.

In acute cases of dysphagia, if oral intake is not possible or inadequate, or if there are increased metabolic demands for nutrients, then nutritional support may be required. Enteral nutrition needs to be given,

Table 17.2: Some dietary tips for dysphagia

Dysfunction	Dietary Tips
• Slow / weak / uncoordinated swallowing	– Semisolid consisterícy to form a cohesive bolus – Textured foods as diced cooked vegetables – Small frequent meals
• Poor oro-motor Control	– Well seasoned, flavoured, aromatic, sweet foods. – Cold temperatures Avoid: sticky, bulky and thin liquids – Semisolid foods that form a cohesive bolus – Small frequent meals Avoid: slippery and sticky foods and purees and thin liquids
• Reduced oral Sensation	– Food placed in most sensitive area of mouth – Different textures, not mixed, to maximize sensation – Highly seasoned foods – Cold temperatures Avoid: I-Iot foods and mixing of different textures

Source: Krause's Food, Nutrition and Diet Therapy 2000. Mahan L.K and Escott Stump S. Medical Nutritional Therapy for Neurologic Disorders. W.B.Saunders Co. London, Toronto 10th Ed.

Table 17.3 gives us a list of desirable and undesirable foods based on the consistency and texture.

Table 17.3: Food consistency and textures

Desirable Foods	Undesirable Foods
o *Foods **forming** a cohesive **bolus:*** Egg dishes, soft cheese and paneer, pasta dishes, rice with gravy, ground meats with gravy, gelatin based desserts, hot cereals and vegetables in a gravy or sauce.	• *Foods that fall apart*: Dry crumbly bread, dry cracker biscuits, thin pureed foods, plain chopped raw vegetables and fruit, plain ground meat, thin cereals and plain dry rice.
• ***Medium** thick* liquids: Vegetable juice, blenderized cream soups, blenderized fruit juices, milk shakes and soft custard.	• Sticky *and* Bulky ***Foods:*** Fresh white bread, plain mashed potato, banana, bran enriched cereals, large pieces of meat, raw vegetables and fruit.
• *Semi soft/thick* liquids: Curd/ yoghurt, pureed fruit, ice cream, soft smooth desserts, frozen shakes and frozen juices.	• Thin *Liquids:* Apple, orange and other citrus juices, milk, tea, coffee, water and soda.

Source: Krause's Food, Nutrition and Diet Therapy 2000. Mahan L.K and Escott-Stump S. Medical Nutritional Therapy for Neurologic Disorders. W.B.Saunders Co. London, Toronto 10th Ed

With this we end our study on dysphagia. We move on to Alzheimer's disease next.

17.6 ALZHEIMER'S DISEASE

Named after the German neurologist who first described it, Alzheimer's disease is the most common cause of progressive dementia, due to the degeneration of nerve cells in the brain and shrinkage of brain matter. Extra cellular deposits of amyloid forming protein or amyloid plaques are reported in the cerebral vessels. Let us now find out what factors lead to this disorder and what its clinical features are.

17.6.1 Etiology and Clinical Features

The probable risk factors include a genetic basis, head injury, low education level, down syndrome and mother's age at birth. However, no single factor has been proven to be responsible for this disease.

The clinical manifestations of Alzheimer's disease along with the nutrition related changes may be divided into three stages. Impairment of a wide range of neurological functions is involved, being a disease of the cortical neurons. The three stages are:

Stage I – There is an increased forgetfulness, anxiety and depression. Associated nutrition related changes include difficulty in food preparation, forgetting to eat, taste and smell changes, altered food choices and impaired appetite regulation.

- *Stage II* – There is a memory loss, especially for the recent events. There is disorientation and personality changes occur. Dietary manifestations include an increase in energy requirements as a result of agitation, holding food in the mouth, forgetting to eat and swallow, forgetting the use of eating equipment except perhaps a spoon and eating with hand.

- *Stage III*– This is characterized by severe mental confusion, psychosis, memory loss, personal neglect and distinct feeding problems. There may be no recognition of food with refusal to open the mouth for eating,

Persons with Alzheimer's disease thus have impaired ability to recognize hunger, thirst, or satiety. They are prone to dehydration. As the disease progresses, their attention span reduce, they are easily distracted and forgetful and may stop eating or not eat enough or try and eat inedible items. Eventually nutritional support may be required to sustain them.

The nutritional management of such patients is discussed next.

17.6.2 Feeding and Nutritional Management

Keeping the clinical manifestations of Alzheimer's disease in mind, treatment involves personalized care, keeping the patient well nourished, reducing anxiety and stress and improving the quality of life. The main objectives of nutritional management, hence, are to:

- provide adequate nutrition,
- prevent malnutrition, and
- devise methods to tackle feeding problems.

Several strategies may have to he used to achieve these objectives, keeping the functional impairments in mind. Some of these, based on the stage of the disease and individual needs are:

- supervising meal times with minimal distractions,
- assessing chewing and swallowing ability and providing foods of appropriate consistency,
- initiating the activity of eating, by making the person touch or taste the food,
- giving one food at a time in small bowls so as to avoid stress of food choices,
- supervising to avoid eating of spill food or inedible items,
- giving only a spoon or finger foods, in case of inability to use other eating equipment,
- encouraging individual appropriate feeding techniques,
- permitting adequate feeding time to increase intake,
- use of nutrient dense foods, frequent snacks and nutritional supplements to avoid malnutrition,

- , avoiding finger foods and using only a small spoon in case of tendency to take a large holus, and
- guarding against aspiration, in case of dysphagia.

To ensure adequate food and nutrient intake, continuous assessment of nutritional status is desirable, supported by behaviour modification, if required. Patient guidance and supervision including continuous verbal instructions during each step of feeding may also be needed, like to eat, chew and stop chewing.

Let us move on to the discussion of the next disorder i.e. Parkinson's disease.

17.7 PARKINSON'S DISEASE

Parkinson's disease is a degenerative central nervous system (CNS) condition characterized by progressive loss of cells within substantia nigra. Substantia nigra is a portion of the midbrain, as illustrated in Figure 17.4, which is thought to be involved in certain aspects of movement and attention. It consists of two subdivisions, the pars *compacta* and the *pars reticulata*. The cells within the substantia nigra release the neurotransmitter dopamine and it is the loss of dopamine that is primarily responsible for the motor defects.

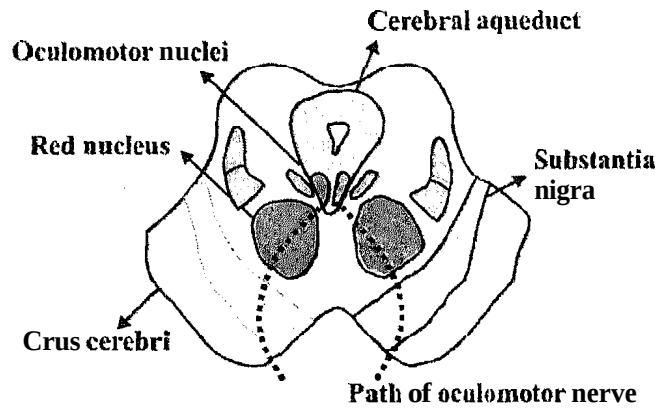

Figure 17.4: The substantia nigra

The disease is much more common in senior citizens and is slightly more prevalent in men than women, What are the causative factors and the clinical features of this disease? Read the next section and find out.

17.7.1 Etiology and Clinical Features

The *cause* of Parkinson's is unknown. Genetic predisposition (in most cases the reason for the death of these dopamine neurons is unknown), and exposure to neurotoxins and industrial toxins are said to be important risk factors. Viral infection such as encephalitis can also produce the disease condition.

The common clinical features of the disease include:

- slowness of movement
- inability to initiate movements
- muscular rigidity
- resting tremor
- postural instability
- weight loss.

Parkinsonism describes the common symptoms of Parkinson's disease such as **tremor, rigidity, akinesia (inability to initiate movements)** or bradykinesia (slow movement) **and postural instability.**

Considering the pathology of Parkinson's disease, the feeding and nutritional care is a crucial aspect in the management of the patients suffering from this disease. The next section focuses on the nutritional management of Parkinson's disease.

17.7.2 Management: Drug, Feeding and Nutritional Care

There is no cure yet for Parkinson's disease, but its symptoms can be minimized with drug therapy. Levodopa or a precursor of dopamine is used mostly. Once levodopa enters the brain it can be decarboxylated to dopamine thus replenishing the depleted neurolransmitter dopamine. Levodopa may produce gastric symptoms and nausea, which can interfere with food intake. For many patients these symptoms are mild and tolerance to nausea does develop. The large amino acids generated from metabolic breakdown of proteins can inhibit the absorption of levodopa and hence is best to have it 1 hour before meals. Very large protein meals can reduce the effect of levodopa, hence managing/manipulating the proteins (intake) well can give a better performance in patients. For example patient wishing to remain in an optimum state of activity could benefit from redistributing the protein. Day time restriction of dietary protein-10 g or less upto 5 pm has been shown to improve the efficacy of levodapa. After 5 pm the remaining day's protein requirement can be consumed. This way the patient can have adequate performance of day time activities.

Hence from our discussion above, it is evident that nutrient-drug interaction is an important aspect that we need to consider in the nutritional management of Parkinson's disease. We have already touched on this aspect earlier in Unit 7.

Weight loss is also an occasional problem with patients. This could be due to increased calorie needs resulting from involuntary movements, difficulty in fecding, nausea, medicine related factors, dementia, depression and dysphagia could be causative factors.

Constipation is also a problem in Parkinson's disease patients due to low grade autonomic function or medication that may contribute to constipation. Difficulty in swallowing too can reduce fluid and fibre intake leading to constipation.

As the disease progresses some food related difficulties appear. These are:

- difficulty in food preparation and eating due to tremors,
- gradual development of chewing and swallowing difficulties and risk of aspiration,

  tendency for constipation, and
- prolonged meal times, up to 1 hour, due to muscle rigidity leading to an impaired head and neck control and hence feeding difficulties.

These problems have to be taken into consideration while planning meals. For example:

- Foods rich in fibre and which can be cut into pieces and made into cohesive bites could be given.
- Very liquidy foods may be difficult to handle, but care should be taken to ensure fluid intake is adequate to prevent constipation and hypertension (low blood pressure).
- Small frequent meals with more carbohydrates and less fat may be better tolerated, in view of the gastric side effects and delayed gastric emptying.
- Diets given should be balanced and nutritionally adequate.
- Frequent intake of high-protein snacks has deleterious effects upon Parkinson disease control. Hence this should be avoided.
- Supplemention of vitamin B_6 (pyridoxine) should be avoided as this vitamin can facilitate the premature conversion of levodopa to dopamine thus reducing the potency of the drug.

Thus, the dietary goals in Parkinson disease can be highlighted as under.

Dietary Coals in Parkinson's Disease

The main goals include:

- maintain desirable weight
- promote absortion of anti-parkinson drug levodopa
- lessen swallowing difficulties as a result of disease of medication,
- alter food consistency or texture,
- drink sufficient fluids or have good source of fibre for effective bowel function, and prevent constipation, and
- redistribute the protein

So in our discussion above we have learnt about the drug and diet suited for Parkinson's disease patients. Remember besides the dietary goals:

INDIVIDUALIZE TREATMENT ACCORDING TO EACH PATIENTS SYMPTOMS

Let us now take a break and answer the questions given in the check your progress exercise 2.

Check Your Progress Exercise 2

1. What do you unclerstand by the term 'dysphagia'? Why are the thicker fluids preferable in dysphagic patients?

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2. Name three foods that form a cohesive bolus and three foods that fall apart.

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3. What are the main objectives of nutritional management of Alzheimer's disease?

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4. Why is knowledge of drug nutrient interaction important in the nutritional management of Parkinson's disease?

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Now we move on to the study of epilepsy and its nutritional management.

17.8 EPILEPSY

Epilepsy is *a neuromuscular disorder in which transient seizures recur, due to an abnormal brain activity.* The brain, through an orderly electric excitation of its nerve cells, controls all activities of the body. When however, due to some reason, the discharge is unregulated and chaotic, an epileptic seizure can occur. Seizures may occur spontaneously or may be triggered by a stimulus. What factors lead to these and what are its clinical features? Let us read and find out in our next section.

17.8.1 Etiology and Clinical Features

This disorder usually starts in childhood, with the peak incidence between birth and two years. Etiological factors include birth trauma, head injury, brain infection, and metabolic imbalance in the body, neurotoxins or a genetic basis. It may also be idiopathic or of unknown origin. About 1/3rd persons outgrow this condition and do not require medication. In 1/3rd, drugs can control the seizures. In the remaining, the condition usually gets worse.

Any of the etiological factors in epilepsy, can result in intermittent derangemenl of the nervous system due to a sudden excessive disorderly discharge of cerebral neurons. Different types of seizures may occur:

1) *Generalized or tonic-clonic seizures,* where the entire brain cortex is involved and post seizure disorientation may last for a few minutes to few hours,

2) *Petit nzal or absence seizures* which involve no post seizure fatigue or disorientation, and

3) *Partial seizures,* where there is an epileptogenic focus in the brain tissue, but electrical activity may spread across the entire brain.

The type of seizure determines the drug therapy and nutrient requirement. Anticonvulsant drug reactions are of relevance in nutritional management.

The general symptoms of epilepsy include weakness, fainting, uncoordinated muscle movement. Based on the drug, common side effects include nausea, vomiting, anorexia or increased appetite, diarrhoea or constipation, decreased taste sensation, increased vitamin D and K catabolism, low levels of serum calcium, vitamins B, and B_{12} and folate. Long-term usage may lead to rickets in children and cause liver damage.

The management of epileptic patients is described next.

17.8.2 Management: Drug, Feeding and Nutritional Care

The primary treatment of epilepsy is anticonvulsant drugs. The focus of nutritional management is a diet, which is appropriate for growth during childhood and maintenance in adults. Nutrient drug interactions also must be considered and remedial steps must be taken. Whereas anticonvulsant drugs may cause side effects of nutritional significance, folic acid supplements can interfere with the action of one of the drugs– Phenytoin. Food intake can delay the absorption of phenobarbital. Low serum albumin due to a state of malnutrition can result in a higher drug concentration in the blood and thus toxicity.

In mild/moderate epilepsy, *'ketogenic diets'* are sometimes recommended. A ketogenic diet is a *high fat diet, with a ratio of 4:1 or 3:1 of fat to carbohydrate and protein calories.* Usually about *75%* of the recommended energy intake for weight and height is given. Protein given is about 1g/ kg for growth. This leaves a minimal amount of carbohydrate to make up the calories. This diet may be used for upto three years and has been reported effective in young children. A ketogenic diet is designed to produce ketone bodies as a result of incomplete oxidation of fat although the exact mechanism is not known. The ketone body produced by incomplete oxidation

of fat (acetone, acetoacetic acid and β hydroxybutric acid) are thought to have an anticonvulsant action and hence are beneficial.

The ketogenic diet is initiated after an initial period of Pasting for 24-72 hours, till ketosis is established. There are two types of ketogenic diets:

a) traditional diet, using long chain triglycerides, and

b) the medium chain triglyceride diet (containing coconut and palm kernel oil) which results in ketosis easily.

Next, let us look at the dietary recommendations.

Dietary Recommendations

A 3:1 ketogenic diet is recommended. The time taken for reversing the usual ratio of **1:3** to **3:1** is about 4 days. How will we know if our ketogenic diet is working? Very easy, just test the urine for ketone bodies. There are a few foods to be avoided in a ketogenic diet. These are listed in Table 17.4 along with those foods which may be given as desired.

Table 17.4: Foods to be included and avoided in a ketogenic diet

Foods to Avoid for Ketogenic Diet	*Foods to be Given as Desired*
The following foods contain substantial amounts of carbohydrates and should be avoided. – all breads and cereals – cakes or cookies, pastries – carbonated beverages, sherbet and sweet juices – puddings and pies – candy and chewing gum – jams, jellies, marmalade and honey – syrups, sugar and condensed milk	The following foods contain negligible amounts of protein, fat and carbohydrate and may be used more frequently. – broth or consommé – coffee (normal and decaffienatcd) and tea – unsweetened cocoa powder and gelatin – mustard dry, salt and pepper, parsley and other herbs, and – vinegar.

For preparing meals you could choose one of the following food/food items:

- Meat/cheese, chicken (30 g), egg (1), fish (50 g), cottage cheese (50 g), processed cheese (30 g), pulse (30 g)
- Cereal: bread (25 g), wheat (20 g), crackers (20 g), rice (50 g cooked). Noodles (50 g cooked)
- Vegetables (100 g): beans, broccoli, cabbage, cauliflower, cucumber, eggplant mushrooms, mustard greens, radish, spinach, tomatoes, turnip.
- Fats: Butter (5 g), cooking fat (5 g), nuts–almonds, walnuts (5 g), whipped cream (60 g)
- Fruits: Apple (40 g), apricots (60 g), banana (30 g), gooseberries (50 g), cherries (40 g), grapes (40 g), orange (100 g), mango (35 g), melon (100 g), papaya (60 g), peach (60 g), pineapple (40 g), plums (40 g)
- Milk: Butter milk (120 g), skimmed milk (120 g).

With these dietary recommendations we end our study of epilepsy. Finally, let us review the neuro trauma and the spinal trauma and the nutritional management of patients suffering from these traumas.

17.9 NEURO TRAUMA

Neuro or head trauma includes brain injury, skull fractures, extraparenchymal or internal brain haemorrhage. Brain injury can be divided into three types. These include:

- Concussion means brief loss of conciousness (< 6 hours),
- Contrusion is similar to a bruise on the skin, and
- Comminution means splintering of bone in many fractures.

Like other cases of major injury and trauma, as you now know from Unit 5, brain injury or trauma also results in a systemic hypermetabolic, hypercatabolic response. This affects the entire body as body reserves get mobilized. If this resultant hypermetabolic state remains unchecked, a sequence of organ failure can result.

Neuro trauma results in production of cytokines, these effect the metabolism.. Some of the effects of this are fever, neutrophilia (type of while blood cells which provide important defence mechanism are increased in number), muscle breakdown, altered amino acid metabolism, increased organ demise. How to prevent these conditions? Let us read and find it out in our following sub-section on nutritional management.

Feeding and Nutritional Management

The main objective of nutritional management is to counteract the hypermetabolism associated with inflammation. The basal energy expenditure (BEE) in neuro trauma patients can be 170-160% of the normal, along with a negative nitrogen balance and weight loss.

The nutritional treatment has mostly two phases. Let us get to know what these are.

a) *Initial Phase* – In this, the life threatening conditions need to bc controlled first. Soon the nutritional support must start because hypermetabolism contributes lo excess energy expenditure and hypercatabolism to increased protein demand. This increased energy (1.75 × 2 x BEE) and protein demand (1.5-2.5 g/ kg IBW) must be met initially via nutritional support methods or enteral / parenteral nutrition. If nutritional replacement is not provided, 10% decrease in lean body mass can occur within a week, with up to 30% loss in two to three weeks. This is associated with increased mortality.

b) *Rehabilitation Phase* – Once the patient stabilizes and starts recovering, bcsidcs nutritional replacement, there is a need to assess functional disabilities related to eating. Dysphagia, difficulty in chewing, physical handicaps in eating can arise as an outcome of the injury. Accordingly, food consistency and texture may need to be adjusted to ensure adequate intake.

In the end, let us review the spinal trauma and its management.

17.10 SPINAL TRAUMA

Spinal trauma or spinal cord injury, commonly due to accidents, falls, sports injury can result in serious disabling consequences. The spinal cord damage disrupts the transmission of nerve impulses from the brain to the peripheral nerves and muscles, resulting in a loss of muscle function, depending on the site of injury. Immobilization commonly occurs due to quadriplegia (paralysis from neck down) or paraplegia (paralysis of the lower part of the body). Metabolic consequences of the trauma include negative nitrogen balance, low serum albumin, loss of calcium, loss of bone and skin collagen and weight loss. Malnutrition is a frequent outcome. The spinal cord responds to insult in a similar manner as the brain. Bleeding and confusion may appear first and then fibrosis. In general, frequency of complications which affect the nutrition may vary and these are constipation, pressure, ulcers, obesity and pain.

So then let us get to know how to manage this condition.

Feeding and Nutritional Management

The main objectives of nutritional management are to meet the nutritional needs of the initial acute phase and then the rehabilitation phase. During the latter phase, special feeding requirements have to be considered along with the requirements of associated complications. What are these phases? Let us read and find out.

- ***Acute*** Phase—Nutritional support should start within 3-5 days or as early as possible to prevent the onset of malnutrition and secondary illness. Frequent assessment of the metabolic rate is desirable, as it tends to vary. This can avoid the complication of overfeeding. Reported recommendations for adults are about 23 Kcal/kg for quadriplegics and 28 Kcal/kg for paraplegics, keeping in mind that increase in metabolic rate is less compared to other trauma conditions.
- *Rehabilitation* ***Phase*** – Once the patients condition stabilizes, individualized diets need to be recommended, based on the energy and nutrient requirements and feeding capacity.

It is usually desirable to adjust energy to maintain the weight 10-15 lbs below the IBW to prevent excess weight gain, which can add to the medical problems and physical handicap. Protein intake should be based on requirements and must be adequate to maintain muscle mass and tissue integrity. A negative nitrogen balance should be guarded against.

Minerals and vitamins must be adequate based on requirements. Supplements may be needed if the dietary intake is poor.

Common complications with spinal trauma patients include pressure sores or decubitus ulcers, hypercalciuria and renal stones, constipation due to neurogenic bowel and depression. Nutrition has a relevant role in all conditions. Pressure sores develop due to immobilization, loss of pressure sensation, poor circulation and skin breakdown. Anaemia occurs resulting in less oxygen supply to the sore area, low serum albumin, excess weight loss also contribute to the complications. The nutritional management lies in making up the protein deficit. About 1.52 g protein/kg is recommended with supplements of vitamin C and zinc. Hypercalciuria and thus a tendency for renal stones, arises due to prolonged immobility. A balanced intake of calcium and phosphorus is beneficial. While protein requirements may be increased, excess should be avoided, as it can cause calcium withdrawal from bones.

Constipation arising due to neurogenic bowel requires a regular bowel schedule with high fibre and fluid intake.

Depression often follows recovery as the person comes to term with disability. An adequate balanced diet resulting in slight increased muscle mass, with concerned nutritional care can help improve quality of life.

Check Your Progress Exercise 3

1. List five foods which have substantial amount of carbohydrates and need to be avoided in epilepsy?

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2. Why are ketogenic diets beneficial in epilepsy?

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3. Why are energy requirements mostly greater in neuro trauma as compared to spinal trauma?

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17.11 LET US SUM UP

In this unit, we learnt about nervous system and the related disorders, which are termed as 'neurological disorders'. Neurological disorders may be of nutritional or non-nutritional etiology, but both require nutritional intervention. Common non-nutritional neurological disorders, some being progressive in nature, in which feeding and nutrition are important, are Alzheimer's disease, Parkinson's disease, epilepsy, neuro and spinal trauma.

Here, we realized that dysphagia is a problem common to many of the disorders. Personalized nutrition care plays an important role, its objectives being to maintain adequate nutrition, prevent further disability and restore potential function. Social, psychological and emotion support improves patient outcome, best achieved by the combined efforts of a rehabilitation team including the family, occupational and speech therapists and nutritionist.

Finally, we learnt that individual nutritional needs have to be assessed and adequate energy, protein, energy sources and minerals and vitamins provided to promote functional, tissue and organ integrity. Eating skills and eating desire also requires assessment and persons may require help to develop new ways of eating using special foods or eating equipment and utensils. Nutritional support may be needed in some neurological diseases. Drug nutrient interactions may also require attention. Social, psychological and emotion support improve patient outcome, which can be best achieved by the combined efforts of a rehabilitation team including the family, physicians, nurse, dietitians, occupational and speech therapist, caregivers, family.

17.12 GLOSSARY

Amyloid : a starch-like glycoprotein.

Alzheimer's disease : a neurological disorder arising due to degeneration of nerve cells in the brain and shrinkage of brain matter, with formation of amyloid plaques in the cerebral vessels. It is the most common cause of progressive dementia.

Apraxia : neurological impairment charactcrizcd by inability to perform activities inspite of ability to understand and carry out the activity.

Bradykinesia : an abnormal slowness of movements; may be clue to neurological damage.

Decubitus ulcers : a bedsore which is caused by pressure over bony areas. The most common sites for it to occur are the hips, elbows and heels.

Dementia : progressive mental deterioration due to organic brain disease.

Dysphagia : an inability to swallow or difficulty in swallowing.

Down syndrome : a combination of birth defects caused by the presence of an extra copy of the 21st chromosome.

Enteral nutrition : a way to provide food through a tube placed in the nose, the stomach or the small intestine. A tube in the nose is called a nasogastric or nasoenteral tube while through the skin into the stomach is called a gastrostomy or percutaneous endoscope.

Epilepsy : a neuromuscular disorder in which there is recurrence of transient seizures due to unregulated and chaotic or abnormal electrical excitation of the brain nerve cells.

Hemanopsia : defective vision or blindness in half the visual field.

Hemiparesis : paralysis affecting one side of the body.

Ketogenic diet : a high fat diet with restricted carbohydrate that produces ketosis and production of ketone bodies in the human system.

Medium Chain Triglycride : are medium chain fatty acid esters of glycerol. These are fatty acids containing 6 to 12 carbon atoms. They are constituents of coconut and palm kernel oils.

Paraplegia : paralysis of half of the body, usually referring to the lower half or lower extremities.

Parenteral nutrition : also known as hyperalimentation, is an administration of a nutritionally adequate solution through a catheter into the vena cava; used in cases of long-term coma or severe burns or severe gastrointestinal syndromes.

Parkinson's disease : a chronic progressive nerve disease characterized by muscle tremors, weakness, rigid movements, halting gait, drooping posture and expressionless facial appearance.

Quadriplegia : neck down paralysis of all four extremities.

Wernicke Korsakoff Syndrome : a brain disorder involving loss of specific brain functions, due to thiamine deficiency.

Tremor : is the rhythmic, oscillating shaking movement of the whole body or just a certain part of it, caused by problems of the neurons responsible from muscle action.

17.13 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1. a) Nutritional origin neurological disorders: Neurological manifestations of nutritional disorders: beri beri, pellagra, pernicious anaemia, Wernicke-Korsakoff syndrome and stroke.

   b) Non-nutritional neurological disorders; Dysphagia, Alzheimer's disease, Parkinson's disease, Epilepsy, Spinal and Neuro Trauma.

2. Five consequences which can effect feeding include: i) Weakness of tongue, facial and masticator muscles, ii) Hemisperesis and paralysis, iii) Apraxia, iv) Hemianopsia, and v) Metabolic stress and trauma.

3. Important goals of nutritional care of persons with neurological disorde s are:

   i) Prevent further disability, and

   ii) Restore or achieve optimal potential of the patient and improve quality of life.

4. i) – c)

   ii) – a)

   iii) – e)

   iv) – b)

   v) – d)

Check Your Progress Exercise 2

1. The thicker fluid is preferred in dysphagic patients due to the inability to swallow or difficulty in swallowing. Thicker fluids are easier to swallow and lower the risk for aspiration.

2. Three foods that form a cohesive bolus are egg dishes, soft cheese, vegetables or ground meats in gravy. Three foods that fall apart are dry crumbly bread, plain chopped raw vegetables and fruit, plain dry rice.

3. The main objectives are to:

   - provide adequate nutrition,
   - prevent malnutrition, and
   - device methods to tackle feeding problems.

4. Nutrient drug interaction is important as tlie large amino acid generated from metabolic breakdown of proteins in this condition can inhibit the absorption of main therapeutic drug levodopa and hence it is best to give this drug one hour after meal, Further levodopa produces gastric symptoms and nausea and thus interferes with satisfactory food intake.

Check Your Progress Exercise 3

1. Foods which have substantial amount of carbohydrates include all breads and cereals, cakes, cookies and pastries, puddings and pies, candy and chewing gum, jam, jellies, sugar and condensed milk.

2. Ketogenic diets produce ketone bodies which are believed lo behave as inhibitory neurotransmitters, producing an anticonvulsant effect.

3. Energy requirements tend to be greater in neuro trauma as compared lo spinal trauma as the increase in metabolic rate is higher in neuro trauma as compared to spinal injury trauma. In the latter, the extent of increase of metabolic rate is less due to lower metabolic activity of the damaged denervated muscles.