# Chapter 13 Clinical Nutrition

A number of nutritional problems arise during illness. The obvious one is with regard to diseases of the gastro-intestinal system causing difficulties with digestion and absorption of foods. But there are also a number of problems to be addressed during infective illness and in diseases of organs like the heart, kidney and the liver. It is proposed to outline current practice in provision of nutritional support during the more common illnesses.

## Acute diarrhoea

Acute diarrhoea is responsible for deaths of 1.7 million children under the age of five years according to WHO estimates. Diarrhoeal disease is usually associated with strains of food-borne bacteria like *E.coli*, *Salmonella*, and *Campylobacter* and some viruses (e.g. rotavirus), but from time to time it may be due to pathogens like *Shigella* and *Vibrio cholera* with significant risks of mortality. According to WHO approximately 80 per cent of all episodes of diarrhoea are acute watery diarrhoea; 10 per cent are acute dysentery; 10 per cent are persistent.

There has been a major change in the scientific understanding of the pathophysiology of diarrhoea. In the past the practice was to prescribe antibiotics to deal with the infection, and intravenous fluids to counter the dehydration. Research in the past several decades has revealed that toxins produced by the invading microbes alter normal function of the enterocyte. Under normal conditions water and electrolyte absorption and secretion occur throughout the intestine. In the small intestine water and electrolytes are mainly absorbed by the villous cells and secreted by the crypt cells simultaneously resulting in a bidirectional movement of water and electrolytes between the intestinal lumen and blood. In the normal state there is a net absorption of over 90 per cent of the fluid in the small intestine, so that about one litre reaches the large intestine. Enetrotoxins like the cholera toxin and the heat labile and heat stable toxin of E.coli disrupt the normal traffic of water and electrolytes. The toxins activate adenylate cyclase inside the cell which in turn leads to an increase in intracellular cyclic AMP. The increased AMP blocks sodium absorption from the lumen and stimulates chloride secretion by the crypt cells into the lumen. Together these processes cause excessive loss of electrolytes (sodium, chloride, potassium, and bicarbonate) into the gut lumen drawing water with it. (See Fig.13.1).

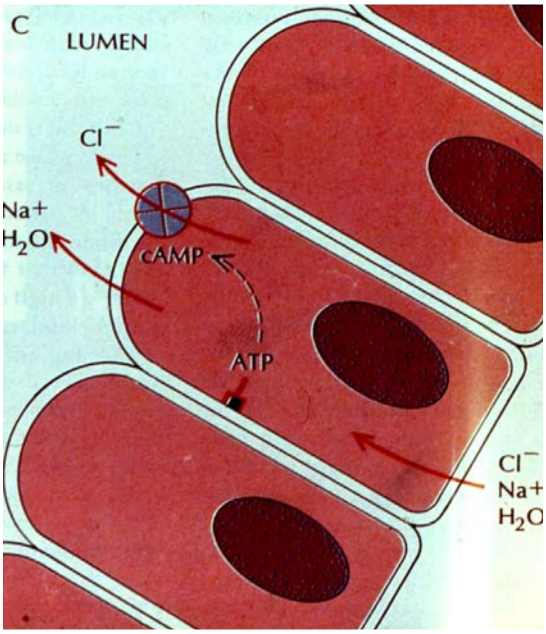


**Figure 13.1 Secretory diarrhoea**

In a way diarrhoea may be looked upon as "water-electrolyte" malnutrition. Loss of water causes signs of dehydration; loss of potassium from the tissues causes cardiac and neuromuscular problems; and loss of bicarbonate upsets the pH balance of the body causing acidosis with disruption of metabolic processes. Certain viruses produce diarrhoea by disrupting the normal absorptive function with the result that there is incomplete absorption of various dietary components and secondary "osmotic diarrhoea" results.

Normally sodium absorption is linked to glucose or amino acid transport mechanisms across the brush border of the enterocytes, and water gets transported passively with the other molecules. These mechanisms remain intact during diarrhoea. Even in severe diarrhoea glucose and other carrier-mediated sodium absorption remains intact Thus provided sodium is supplied in the presence of glucose or amino acids water absorption occurs both thorough and between the mucosal cells. It is, however, necessary to supply the salts, sugar and water in the correct proportion. Typically, in a healthy individual 25 g of sodium is secreted into the lumen each day as a constituent of the numerous gut secretions and a further 5 g enters through the food. Sodium ions are then reabsorbed by an active transport process which is facilitated by the presence of glucose in the gut lumen. This is the principle on which oral rehydration therapy is based. (See Fig. 13.2)

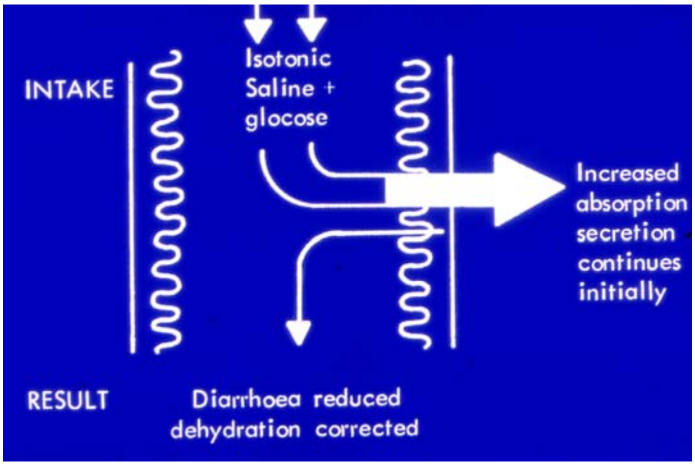


**Figure 13.2 Basis of oral rehydration therapy**

The original proportions of sugar and salt were determined in adult patients suffering from cholera which causes a massive loss of fluid and electrolytes. But it worked in children with acute diarrhoea and has been widely promoted. More recently the formula has been revised to provide a reduced osmolality formula. (See Table 13.1)

**Table 13.1 Composition of oral rehydration solution**

| Substance | Amount (g/litre of water) | |
|---|---|---|
| | Original formula | Reduced osmolality formula |
| Sodium chloride | 3.5 | 2.6 |
| Potassium chloride | 1.5 | 1.5 |
| Trisodium citrate | 2.9 | 2.9 |
| Glucose | 20.0 | 13.5 |

Glucose is not so widely available and is expensive. But since digestion remains unaffected it was found that sucrose, a household substance, can be used instead in twice the amount as glucose. Starch digestion is also unaffected and rice water has also been used with benefit.

If there is severe dehydration the first priority is to give intravenous fluids to prevent shock and get kidneys functioning. The preferred fluid is Ringer's lactate 100ml/kg. Of this 30ml/kg is given within the first hour for infants and children <12 months old, and within the first 30 minutes for older children. The remainder 70ml/kg is given more slowly over 5 hours for infants and young children and 2 ½ hours for older children. If facilities for intravenous therapy are not available the ORS fluid can be given by nasogastric tube or orally 20ml/kg/hour for 6 hours to achieve a total intake of 120ml/kg. After the initial period of replenishing the losses oral rehydration fluid is given at the rate of 50 – 100 ml (and 100 – 200 ml for older children) after each loose stool.

**Nutritional support in acute diarrhoea**. Acute diarrhoea is the commonest antecedent of childhood malnutrition. Recurrent episodes of diarrhoea with little time in between for catch-up create a vicious circle in which progressive nutritional deterioration predisposes to further attacks of diarrhoea which tend to be prolonged. (See Fig. 13.3). Children with more severe diarrhoea, dehydration and acidosis have a greater reduction in dietary intake.

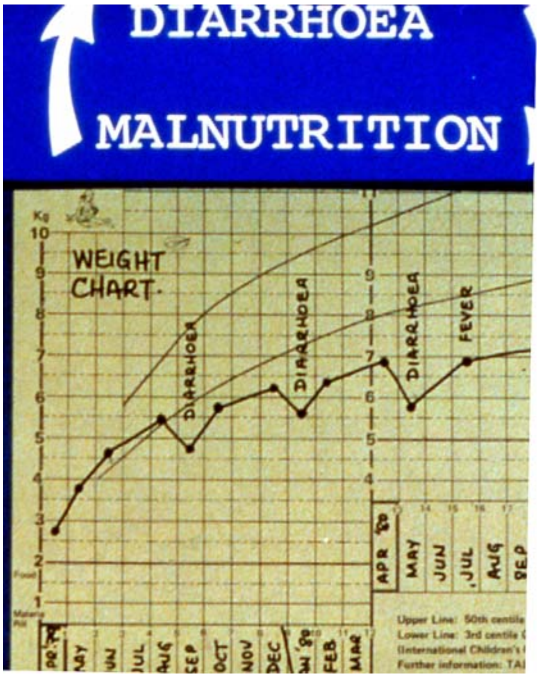


**Figure 13.3 Recurrent attacks of diarrhoea resulting in malnutrition.**

Abnormalities of digestion and/or mucosal transport of carbohydrate, protein and fat occur frequently in diarrhoea. Nutritional status may continue to deteriorate even after the more obvious symptoms have subsided. Several studies have shown that continued feeding during illness and supplementation with certain nutrients such as zinc may reduce the frequency and duration of abnormal mucosal function following diarrhoea. Breast fed infants are particularly protected from the more severe nutritional effects of diarrhoea.

A number of clinical trials conducted in different regions of the world have demonstrated the beneficial effects of nutritional support. Those subjects who were fed early in the course of treatment tended to gain more weight than those who were not. Even if patients experienced anorexia initially, their appetite returned within a day or so as recovery from acidosis and dehydration occurred. In those patients in whom breast feeding was continued illness tended to be of short duration. With non-human milk feeding treatment failure was twice as common as with breast milk. As a result two general treatment strategies have evolved:

1. Continued feeding of different amounts of food during illness to avoid nutritional deficiencies, and in breast fed infants to persist with breast feeding.
2. Increased feeding after the episode of diarrhoea for catch-up growth. Mixed diets prepared from locally available foods (In Peru noodles cooked in evaporated milk; In Mexico mixture of rice, chicken and other common foods) is superior to soy-protein based formula.
3. Supplementation with zinc has a positive impact.

If malabsorption is suspected it is most commonly due to maldigestion of carbohydrate, which can be detected by stool pH of <6.5 and reducing substance in stool. Intake of the suspect carbohydrate is reduced or substituted by another macronutrient like fat.

**Persistent diarrhoea**. Diarrhoea which begins as an acute episode but continues beyond 2 weeks is referred to as "persistent diarrhoea". In developing countries an estimated 3 to 10 per cent children with acute diarrhoea develop persistent diarrhoea. Nearly all pathogens known to cause diarrhoea have been associated with it. The most common pathogens reported are Shigella, Enterotoxigenic E.coli, Enteropathogenic E.coli, Enteroadherent E.coli, Cryptosporidium and Giardia. Histological examination of intestinal biopsies shows non-specific villous atrophy and inflammation. (See Fig. 13.4).

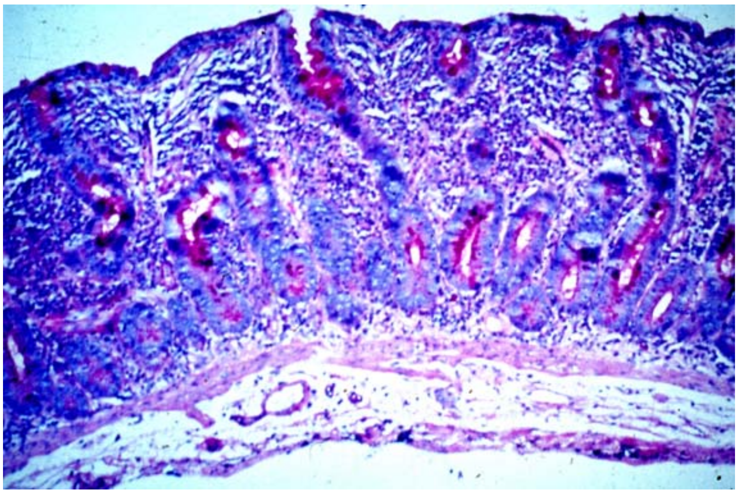

**Figure 13.4 Villous atrophy in a child with persistent diarrhoea**

**Nutritional support**. During the course of the illness patients develop cow's milk intolerance, carbohydrate maldigestion and bacterial overgrowth resulting in marked deterioration of the nutrition and general condition. To make matters worse digestive disturbances make oral intake most difficult. (See Fig. 13.5)

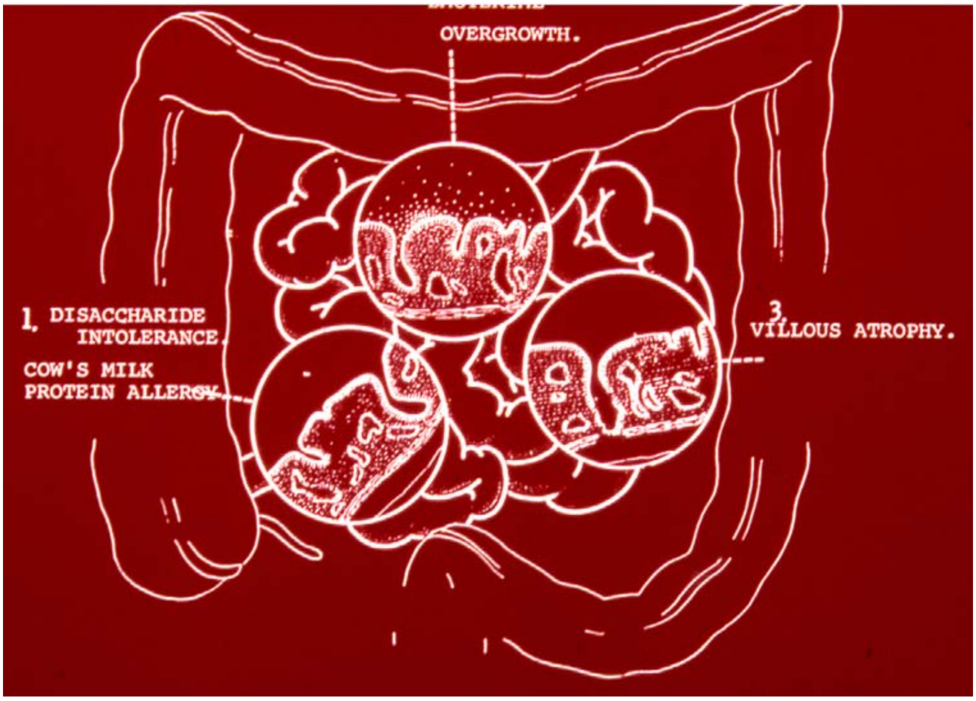


**Figure 13.5 Villous atrophy, sugar and protein intolerance and bacterial overgrowth make oral feeding difficult.**

Oral rehydration therapy is effective and prevents dehydration. The difficulty lies in getting nutrients into the child because of the damage to the gut mucosa. There is rapid deterioration of the nutritional status. (See Fig. 13.6)

**Figure 13.6 Persistent diarrhoea with marked nutritional deterioration.**

Often the only approach feasible for feeding is by total parenteral nutrition (TPN) or elemental diet. Both have their specific problems. TPN carries risks and requires expert supervision in a tertiary care centre. It is also expensive and the required ingredients may not be available in resource poor countries. Elemental diets are also expensive and not easily available in developing countries. Workers in Manchester (UK) have used banked human milk fed orally in the management of persistent diarrhoea. In a series of 13 children eleven were successfully treated. All the patients were severely malnourished, and had deteriorated while fed on a range of modified formulas. Seven infants responded promptly with cessation of weight loss and diarrhoea; in four others human milk helped re-establish oral nutrition after a period of TPN.

Several centres in developing countries use locally available foods like rice/lentil mix with yogurt and have obtained satisfactory results. Based on such experiences WHO has recommended two diets – one starch based and containing low lactose and the other a lactose free with reduced starch as follows:

**Table 13.2 Starch based low lactose diet for use in Persistent Diarrhoea**

| Food | Amount |
|---|---|
| Full fat dried milk | 11g |
| (or whole liquid milk) | (85 ml) |
| Rice | 15g |
| Vegetable oil | 3.5 g |
| Sugar | 3 g |
| Water to make | 200 ml |

**Table 13.3 Lactose free diet with reduced sugar**

| Food | Amount |
|---|---|
| Whole egg | 64 g |
| Rice | 3 g |
| Vegetable oil | 4 g |
| Glucose | 3 g |
| Water to make | 200 ml |

The main objective is to get food into the intestinal lumen based on what is tolerated. Lactose and disaccharides may precipitate purging depending upon the damage to the brush border and availability of enzymes. Presence of food in the intestine has a trophic effect because of secretion of gut hormones, principally enteroglucagon, and this may help to usher in healing.

**HIV infection.**

In HIV infection the relationship between immune deficiency, enteric and other opportunistic infections, malabsorption and malnutrition induced immune defects create a vicious cycle resulting in progressive nutritional deterioration. The goal of nutritional management is to maintain normal growth, maintain lean body mass, provide sufficient energy intake, and control malabsorption if encountered. Children may appear to be well nourished because of increased fat mass, but may have decreased lean body mass and not growing at the expected rate for age. Fever and recurrent infections increase metabolic demands and should be met.
An important cause of malnutrition in HIV infection is decreased oral intake because oral ulcers, oesophagitis, anorexia, depression, increased nutrient loss through diarrhoea caused by opportunistic infections, malabsorption and increased metabolic requirement.

# Nutritional support in cardiac, renal and hepatic disease

Growth failure is common in **congenital heart disease**. Cyanotic lesions are more commonly associated with delayed weight gain and growth. The severity of growth failure and delay in skeletal maturation are directly related to the degree of hypoxemia. Acyanotic lesions affect weight more severely than height.

Congestive heart failure results in decreased cardiac output and reduced blood flow to the kidneys and the gut. This may result in impaired absorption, altered intestinal motility, and delayed gastric emptying. Additionally, drugs used for treatment may further affect appetite. The current approach is i) to increase energy intake from maintenance requirement of 75-120 kcal/kg/day by 20 to 100% depending on appetite if there is growth failure, or surgery or stress; ii) provide high calorie food by decreasing water content and raising protein (8-10%), carbohydrate (35-65%), and fat (35-50%); iii). restrict sodium; iv) maintain urine osmolality below 400 mosm/l.

Children with **chronic renal failure** experience many nutritional and metabolic abnormalities like acidosis, huperkalaemia, hypermagnesaemia, anaemia, oedema, and growth failure.

Fluid restriction requires calorie dense formula feeding for infants. Increasing the concentration of formula results in increased renal solute load and hence carbohydrate and fat provide an effective way of raising calorie density. The minimal amount of protein required for growth must be provided and this should be adjusted as the child gains weight. It is usual to recommend protein intake of 1.8/g/kg/day for infants under 1 year of age; 1.0 – 1.5 g/kg/day for infants aged 1 to 2 years, and 1g/kg/day for children aged 2 to 16 years. Majority of the protein should be of high biological value with an optimal amino acid profile. In infants this is achieved through breast milk. In bottle fed infants a low solute whey based formula diluted to achieve required to achieve required protein concentration is used. The energy content may have to be raised by means of glucose polymer and or fat emulsion. Weaning foods can be introduced at 4 to 6 months in the form of rice and pureed fruit.

Infants and children with chronic renal insufficiency are assumed to have normal energy requirements and at all ages the estimated average requirements for energy should be met. Growth should be assessed on an individual basis and appropriate adjustments to energy intake made.

Children on regular peritoneal dialysis are exposed to the risk of protein loss in the dialysis effluent and may require additional protein supplement. Nutritional support with various nutrients is illustrated in table 13.4.

**Table 13.4 Fluid and Nutrient recommendations for Children on Peritoneal Dialysis**

| Age | 0-1 yr | 1-3 yr | 4-10 yr | 11-18 yr |
|---|---|---|---|---|
| Energy (kcal/kg) | <6 mos. 108<br>6mos -1yr 98 | 102 | 4-6 yr 90<br>7-10 yr 70 | Girls 11-14 yr 47<br>Girls 15-18 yr 40<br>Boys 11-14 yr 55<br>Boys 15-18 yr 45 |
| Protein (g/kg) | 2.5-4.0 | 2.0-2.5 | 2.0-2.5 | 1.5 |
| Sodium | 1-3 mEq/kg if needed | Same as for predialysis | Same as for predialysis | Same as for predialysis |
| Calcium | Same as for predialysis | Same as for predialysis | Same as for predialysis | Same as for predialysis |
| Potassium (mEq/kg) | 1-3 if needed | 1-3 if needed | 1-3 if needed | 1-3 if needed |
| Vitamins | Multivitamin 1 mg folic acid, vitamin D | Multivitamin 1 mg folic acid Vitamin D as needed | B complex vitamins containing 1 mg folic acid 10 mg pyridoxine, 60 mg ascorbic acid, 5 mg pantothenic acid 1 mg thiamine, 1.2 mg riboflavin 3µg $B_{12}$, 300µg biotin, 15 mg niacin, vitamin D if needed | B complex vitamin containing 1 mg folic acid, 10 mg pyridoxine, 60 mg ascorbic acid, 10 mg pantothenic acid, 1.5 mg thiamine, 1.7 mg riboflavin, 6 µg B12, 300µg biotin, 20 mg niacin, vitamin D if needed |
| Trace minerals | Supplement zinc or copper if needed; iron usually needed | Supplement zinc and copper if needed; iron usually needed | Supplement zinc and copper if needed; iron usually needed | Supplement zinc and copper if needed; iron usually needed |
| Fluid | Provide insensible loss + urinary output + ultra filtration capacity | Usually unrestricted | Usually unrestricted | Usually unrestricted |

Diseases that impair liver function ultimately affect absorption and utilization of nutrients. Children with **chronic liver disease** require between 100 to 150% of the RDA to maintain growth. Protein restriction is not necessary except in situations where hepatic encephalopathy threatens. If there is fat malabsorption, as in the case of biliary obstruction, foods containing medium chain triglycerides may be used to provide energy. Anorexia and fat maldigestion are major problems especially in a child with cholestasis. Because of abnormal gluconeogenesis, peripheral oxidation of protein occurs which may account for the muscle wasting seen in children with chronic liver disease.

As a rule of thumb urgent and aggressive nutritional support is indicated if the mid-arm circumference is <10 cm after one month of age and <13 cm from four months of age. The following strategy may be adopted for improving the nutritional status:

1. Increase energy intake to 140-200% of the recommended daily allowance. In babies this can be achieved by supplementing feeds with extra fat and carbohydrate. It is best to do so in stages in order to establish intestinal tolerance since the supplemented feeds have high osmolality.
2. Medium chain triglycerides (MCT) should be substituted for half to two-thirds of the long chain triglycerides. MCT is more soluble, does not need lipolysis and is absorbed even when intraluminal bile concentration is low.
3. Attention needs to be paid to intake of essential fatty acids – linoleic and linolenic acids- aiming to achieve an intake ratio of 5:1 for linoleic to linolenic acid.
4. Extra intake of fat soluble vitamins is needed. (5 000 to 20 000 units of vitamin A; 100-800 mg vitamin E; vitamin D 800 – 1 000 units; and 5 – 10 mg vitamin K daily).
5. If fluid intake is to be restricted a complex carbohydrate like maltodextrin is useful for restricting osmolality of the feeds.
6. Up to 4g/kg/day of protein is often tolerated without precipitating encephalopathy. A whole protein source is preferred because of its trophic effects on the gut.

Once complications like ascites or encephalopathy develop a modular system wherein individual components of the feed are varied is helpful.

## Malabsorption Syndromes

Malabsorption manifests itself by frequency of stools and failure to thrive. If malabsorption of fat is predominant the stools tend to be bulky, greasy and float in water.

**Coeliac disease**. The cause is gastro-intestinal intolerance of gluten and only occurs after infants have been weaned onto gluten containing solids. In some instances the onset is late in life. There is a strong family history and the incidence in first degree relatives is between 5 to 19 per cent, indicating a genetic basis to the condition. Gluten is present in wheat, oats and rye. Gluten is a large complex molecule consisting of gliadin, glutenin and globulins. Gliadin is composed of about 40 different components of which α-gliadin fraction is the most toxic.

Children with coeliac disease present with symptoms of malabsorption usually a few months to a year after weaning. Late onset is not uncommon coinciding with times of increased nutritional demand like infancy, adolescence, pregnancy and lactation. Often there are minimal symptoms and the child is short with a refractory anaemia. The only evidence suggestive of malabsorption is iron deficiency anaemia, low serum folate, low albumin and low serum calcium. The diagnosis is established by jejunal biopsy which shows villous atrophy. Diagnosis of coeliac disease is confirmed by demonstration of normal histology after a period on gluten free diet, followed by histological relapse after gluten challenge.

Once the diagnosis is established the subject must remain on gluten free diet for life. In the acutely ill child it may be necessary to stop all gluten in the diet and supplement with vitamins and oral iron.

**Nutritional management**. A strategy of management of coeliac disease is as follows:

- Initiate a strict gluten free diet
- Add supplements of deficient nutrients like iron, folic acid and calcium
- Ensure regular supervision and review of progress
- Repeat intestinal biopsy if clinical progress is suboptimal

Table 13.5 gives a list of foods that do not contain gluten and are safe to eat, as well as a list of foods that must be avoided.

**Table 13.5 Gluten content of foods**

| Gluten-free foods | Gluten containing foods to avoid |
|---|---|
| Cereals | Cereals |
| Corn, corn meal, popcorn<br>Rice<br>Buckwheat, millet, sorghum, teff<br>Cassava, tapioca, sago | Wheat, rye, barley, oats<br>Bulgur wheat, drum wheat<br>Semolina, couscous, spaghetti<br>Wheat and oat bran |
| Flour | Flour |
| Corn flour, polenta<br>Rice flour and ground rice<br>Soy flour<br>Potato flour, potato starch<br>Bean flour, chickpea flour, split-pea flour | Wheat, rye, barley, oat<br>Bread and crispbread |
| Nuts, beans, pulses and lentils<br>Meat, poultry, eggs, milk butter, margarine, fats and oils<br>Fresh fruit and vegetables | |
| Breakfast cereals | Breakfast cereals |
| Cornflakes, rice crispies | All other breakfast cereals |

A decrease in the incidence of coeliac disease has been noticed in recent years in several countries. It is believed that this largely due to improvements in the rates of exclusive breast feeding, and withholding of introduction of cereals until after the age of six months.

**Cystic fibrosis** is an autosomal recessive condition that results in abnormal exocrine gland secretion that results in a variety of symptoms and signs. Usually the symptoms are of both gastro-intestinal and respiratory tract involvement. The diagnosis is confirmed by sweat electrolyte sodium of more than 60 mmol/l and chloride of more than 60 mmol/l.

The management of the condition centres on treatment of the respiratory condition and management of malnutrition resulting from pancreatic enzyme deficiency. Deficiency of pancreatic lipase and trypsin leads to diminished absorption of protein and fat. Starch and carbohydrates are absorbed well.

**Nutritional support**. The mainstay of the treatment of pancreatic malabsorption is the administration of pancreatic supplements with food. A number of formulations are commercially available in the form of powder, granules, capsules and tablets with varying costs. They all contain the enzymes lipase and protease in different proportions. Taken just before a meal the dose is adjusted to reduce stool frequency and to improve the consistency until no further improvement is achieved.

Steatorrhoea is seldom controlled by pancreatic supplements alone and reduction of fat in the diet is always necessary. This is achieved by removal of visible fat from meat, avoidance of foods with high fat content like all fried foods, cream, cheese and use of partly skimmed (for infants) or skimmed milk (for older children). Reduction of fat requires addition of extra protein and carbohydrate to make up the calories.

These children require high energy intakes to allow for inadequate absorption even in the presence of pancreatic supplements. Protein content can be normal provided the protein supplied is of high quality so that possible deficiencies of essential amino acids are minimised. In order to obtain high energy and protein intakes it is usually necessary to provide children with three good quality main meals and supplementary carbohydrate snacks in between meals. Vitamins, particularly fat soluble vitamins should be provided in water soluble form. Extra vitamin E (50 mg. daily children; 200 mg daily adults) is advisable since neurological degeneration has been reported in some instances. Cystic fibrosis causes progressive fibrosis and destruction of the pancreas so that eventually diabetes results.

The aim of dietary management is to provide nutrition in forms likely to be absorbed with least difficulty. Fats are not absorbed well, so the diet should initially be low in fat which is then gradually increased. Medium chain triglycerides are water soluble and do not require bile salts. They can be digested by brush border lipases. Hence they are an attractive choice if available. Their drawback is that they do not contain long chain fatty acids. These should be introduced gradually as soon as possible at a rate of 1 ml/kg body weight.

Human milk fat is well tolerated, and so breast feeding should be continued.

Protein is less efficiently absorbed than peptides or amino acids. Preparations of various protein hydrolysates are commercially marketed. If used source of energy should be added together with vitamins and minerals. Protein requirement is 120 – 130 % of recommended daily allowance per kg body weight.

Glucose is usually well absorbed but in large amounts osmolality causes diarrhoea. To overcome this problem a variety synthetic glucose polymers are marketed. They are useful and so are maltodextrins since they all have less osmolality relative to their energy content. Energy intake should be 130 – 150% of recommended daily allowance per kg body weight.

Diet is gradually built up using low concentration of protein, carbohydrates and fat and adjusting to tolerance together with mineral and vitamin supplements.

**Inflammatory bowel disease.**

Crohn's disease. The aetiology is not known but there are suggestions that it may be an infection caused by a mycobacterium – mycobacterium avium subspecies paratuberculosis. Failure to thrive, weight loss, short stature, vitamin deficiency mineral deficiency can pre-empt other symptoms and signs by many years. Dietary management has an important role to play in order to maintain the nutritional status and immune functions.

Dietary management follows one or more of the following four patterns:

1. **Elemental diet**. This is provided in the form of amino acids, glucose, fatty acids, vitamins and minerals to meet daily requirements and the needs for catch-up growth. Elemental diet with the exclusion of all other foods given for six weeks has proven as effective as steroids in bringing about remission. The drawback is the unpleasant taste and compliance is poor. Hence often has to be given by nasogastric tube. The second drawback is the cost. Many authors have commented on its efficacy being as good as steroids. Remission is induced within a few days of introduction of the diet and it has been shown that linear growth six months after treatment with elemental diet is much better than in those patients treated with steroids. The mechanism of action of elemental diets is not known, but the observation that remission occurs twice as commonly with an amino acids based elemental diet compared to with polymeric feeds (75% vs. 36%) suggests that exclusion of intact protein from the gut may be the operative factor.

**Table 13.6 Elemental diets and their constituents**

Elemental diets are designed to bypass the normal digestive processes so as to allow the gut to recover. They are a safe alternative to parenteral nutrition in situations where bowel sounds are audible but gut function is poor. Since elemental diets do not contain intact protein they are hypo-allergenic and can also be used when there is intolerance to milk or other protein, or as the basis for an elimination diet.
Protein consists of short chain peptides or free amino acids which require little or no hydrolysis by intestinal peptidases.

Carbohydrate is generally in the form of hydrolysed corn starch or short chain glucose polymers. Hence such diets are free of lactose and other disaccharides.
Elemental diets are usually low in fat, though a small addition of linoleic acid is needed to prevent deficiency of essential fatty acids. Where triglyceride is present it is in the form of medium chain triglycerides (MCT).

Examples of formulations specifically designed for use in children are:

*Neocate*
Free of lactose, sucrose, gluten, milk, and all intact proteins.
Protein: synthetic L-amino acids in a pattern based on human milk.
Contains fat, including a small amount of MCTs.
Carbohydrate: maltodextrin.
Osmolality per 100 ml feed 320 mosmol/kg
Energy per 100 ml feed 71 kcal

*Peptide 0-2*
Free of lactose, sucrose, gluten, milk, and intact protein.
Protein: low molecular weight peptides from hydrolysed non-milk protein, and L-amino acids.
Fat: long chain fat
Carbohydrate: maltodextrin.

Osmolality per 100 ml feed 195 mosmol/kg
Energy per 100 ml feed 70 kcal

*Elemental 028*
Free of lactose, sucrose, gluten, milk, and all intact protein.
Protein: synthetic L-amino acids with pattern based on human milk.
Fat: long chain fat.
Carbohydrate: maltodextrin.
Osmolality per 100 ml feed 520mosmol/l
Energy per 100 ml feed 53 kcal

*Flexical*
Free of lactose, sucrose, gluten, and intact milk protein.
Protein: casein hydrolysate, 70% free amino acids, 30% small peptides.
Fat: long chain fat and MCTs.
Carbohydrate: corn syrup solids and tapioca starch.
Osmolality per 100 ml feed 550/kg
Energy per 100 ml feed 100 kcal

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2. **Elimination diet**. This is based on the belief that some of the symptoms and signs are due to food intolerance. The treatment is commenced with elemental diet until remission, and then foods are gradually introduced, in stages of one new food per day, until there is evidence of relapse. The most recently introduced food is withdrawn as being the most likely cause. The initial diet is of chicken, pear and rice. Then other food items are introduced from a list of foods least likely to cause intolerance. This approach is best suited when there are definite symptoms, and most difficult when the only symptom is poor growth.

3. **High energy diet**. The principle behind this diet is to encourage growth and weight gain by increasing the total energy content. The diet is rich in good quality protein and high in carbohydrate together with vitamin supplements, iron, folic acid and vitamin $B_{12}$. If energy intake is not increased using carbohydrate then recourse is taken to medium chain triglycerides. This regimen is best suited when gastro intestinal symptoms are minimal.

4. **Total parenteral nutrition (TPN)**. In a crisis when the general condition of the patient has deteriorated to the extent that nothing can be tolerated by mouth recourse is taken to TPN. The objective is to rest the gut while nutrition is provided by the parenteral route. The decision to use TPN should never be taken lightly because of the risk of infection, the cost, and the need to carefully monitor intake and output of nutrients.

**Ulcerative colitis** usually affects older children. There is anorexia, weight loss secondary to blood and protein loss in the bowel lead to nutritional deterioration. Fat malabsorption occurs in approximately 40 per cent of the patients. Low fat regimen with incorporation of medium chain triglycerides helps. In particular reducing stool fat helps to prevent calcium loss.

Progress of the disease is not affected by dietary treatment. During an acute flare up total parenteral nutrition is the only way of providing nutritional support.  In less severe cases low residue diet is commonly used. (See table 13.7)

**Table 13.7 Low and minimal residual foods**

| Foods free of fibre | Low fibre foods (g fibre/100 g food) |
|---|---|
| Egg | Corn flour (0.3) |
| Fish | White rice (0.8) |
| Meat | Tapioca (trace) |
| Milk | Spaghetti, white (0.2) |
| Cheese | |
| Butter and margarine | |
| Cooking fats and oils | |
| Plain yogurt | |
| | |
| Sugar | Asparagus (1.0) |
| Honey | Cucumber (0.4) |
| Jams | Pumpkin (0.5) |
| | tomato pulp no skin no pips (0.90 |
| Clear soups and broths | Grapes no skin and pips (0.9) |
| Fizzy drinks | Melon (0.9) |
| Tea and coffee | Fresh fruit juice (trace) |

Many centres use milk free diet. (See table 13.8) It is not clear whether the benefits observed are due to removal of milk protein or lactose.

**Table 13.8 Lactose content of foods**

| Foods with high lactose content (> 1g/100 g) | Foods with low lactose content (< 1g/100 g) | Foods free of lactose |
|---|---|---|
| Milks of all species; milk based desserts; ice cream; yogurt | Hard cheese; cream; butter; margarine; | Soy milk; margarine made from vegetable oil; vegetable products; oils; cooking fat |
| Cream soups; milk chocolate | Fizzy drinks; filled chocolate products | Glucose; fructose; sucrose; most fruit drinks |
| Processed milk or cheese based fruit; most infant breakfast cereals and rusks | Some adult breakfast cereals; some breads; biscuits and baked goods; some flavoured crisps | Vegetables; potatoes; peas; beans; lentils; nuts |
| | | Fruit; |
| | | Rice; wheat; barley; oats; maize; sago; semolina; tapioca; most breads; most adult type breakfast cereals; pasta |

## Intestinal resection

A number of conditions like volvulus in the newborn; intestinal vascular accidents affecting mesenteric artery; necrotising enterocolitis; obstruction due to meconium; Crohn’s disease and trauma may require bowel resection. A number of nutritional problems arise because loss of absorptive area. In a term newborn the length of the small intestine is 250 – 300 cm. When residual length after resection is less than 75 cm there is difficulty with digestion and absorption. A rapid transit time with sugar intolerance, presence of unabsorbed bile salts and bacterial over growth give rise to persistent diarrhoea of short bowel syndrome. Resection of short segment of the gut causes mild malabsorption. In massive resection there is profound depletion of bile salts and steatorrhoea predominates. Unabsorbed dietary fat is converted to hydroxyl fatty acids by the gut bacteria and these exacerbate the diarrhoea.

A prolonged period of parenteral nutrition is necessary during the period of profuse diarrhoea and loss of nutrients. If nutrition is maintained considerable intestinal adaptation takes place over time. Adaptation is response to nutrients in the bowel lumen, hence it is necessary to offer small feeds as soon as possible. Small feeds of breast milk facilitate adaptation not only because of the trophic effect of nutrients but also because of gut hormone response to feeding. Human milk has factors that stimulate mucosal hyperplasia and this increases brush border enzymes. Several of the growth factors of human milk, in particular epidermal growth factor (EGF), are being marketed as the result of recombinant technology and awaiting future trials for assessing their usefulness.

Main intestinal adaptation takes place in the first two months after resection, and oral nutrition is usually possible after that. Adaptation continues much longer and tolerance of a normal diet is usual by 6 to 12 months. Long term survivors develop almost normal absorptive capacity over time.

Ileal resection usually presents more problems than removal of jejunum alone. The ileum is able to compensate for loss of jejunum, but certain specific functions of the ileum cannot be met by the jejunum. Terminal ileum is the site of absorption of vitamin B12. The ileum is also involved in absorption of bile salts; unabsorbed bile salts are deconjugated by gut bacteria and together with unabsorbed fatty acids cause troublesome diarrhoea. If the ileo-caecal valve is removed in the resection, colonic bacteria invade and colonise the small bowel and interfere with absorption of nutrients.

One specific problem is that of gastric hyper secretion which can cause peptic ulceration as well as diarrhoea because of acid bowel content and ineffective pancreatic enzyme function in acid medium. Over time gastric secretion returns to normal, but at its height may the acidity may need to be controlled with cimetidine and antacids.

**Nutritional support**. Oral nutrition should be commenced as soon as possible for the reasons discussed above. While breast milk feeding is being gradually introduced, nutrition can be maintained in a number of ways. Elemental diets have been used by several workers. They do not require intestinal enzymes for digestion. Their problem is high osmolality because many were originally designed for use in adults. The formulae listed in table 13.4 are for paediatric use. For improving energy content of feeds several water soluble dextrin preparations are commercially available. An example is *caloreen* which provide 390 kcal/100g and is gluten, lactose and fructose free. For suspected malabsorption of fat MCT may be substituted. They are water soluble and do not require bile salts. The problem with MCT is that because of small particle size they have high osmolality. Long chain triglycerides should be introduced when feasible because they are potent stimulators of enterogastrone and provide essential fatty acids.

Several of the products sited above may not be available in resource poor countries.