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2

NUTRITION AND THE SURGICAL PATIENT

EDITED BY
GRAHAM L. HILL

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Nutrition and the Surgical Patient

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Preface

While appreciating the importance of surgical nutrition many surgeons are finding the subject confusing and somewhat full of contradictions. The problem has been compounded recently by a myriad of journal articles and a host of short monographs (most of them reports from conferences) which frequently tend to eulogise nutritional therapy at the expense of solid science. It is not surprising therefore that the practising surgeon is finding it difficult to distinguish nutritional fact from nutritional fancy.

When the Editorial Board of Clinical Surgery International asked me to compile this volume on surgical nutrition I saw the opportunity to make amends and to provide a monograph that not only would be scientifically sound but would be of practical value to the busy surgeon in clinical practice. To this end I invited a team of international authorities in surgical nutrition to contribute and give a balanced appraisal of their subject in a way which would be understandable and of value to the journeyman surgeon. Nearly all these experts are busy clinicians themselves and understand the practical implication of what they write. The contributions have come from the United States, Canada, England, Scotland, Denmark, Sweden and New Zealand. We believe that the opinion expressed in each case reflects accurately the present state of the art.

I would like to thank each of the contributors, the publishers and the many other people who have helped make this book possible. In particular I would like to thank my wife for her help and Mrs Karen Hunter who coped with the considerable typing burden with unfailing good humour.

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DONALD S. McLAREN

Introduction

This chapter is designed to introduce the reader to the variety of clinical manifestations of nutritional disorders that may occur in the surgical patient together with an account of the underlying disease processes that give rise to them and the influence they may have on prognosis.

It is only in recent years that the susceptibility of many patients requiring surgery to become malnourished and the importance of maintaining normal nutritional status in all such patients have become generally appreciated. The implications for the practice of surgery of the pioneering studies of Cuthbertson (1936) and Moore & Ball (1952) on the metabolic response to injury were ill-appreciated. It took most surgeons a long time to realise that the forms of malnutrition endemic among young children in most developing countries occur frequently in diseases necessitating surgery and that they might be induced by certain surgical procedures. The methodology of assessment of nutritional status (see Ch. 3) was largely developed during the study of childhood malnutrition (McLaren 1976). A powerful impetus to the investigation of the nutrition of surgical patients has been given by developments in the techniques of parenteral nutrition and elemental diets, considered in detail in subsequent chapters.

Malnutrition syndromes

Protein-energy malnutrition (PEM)

The most fundamental nutritional requirement of the body is for energy. This is usually raised well above the normal range in surgical patients and for a variety of reasons energy intake and availability frequently fall short of requirement. In this energy-deficit state glycogen stores are exhausted within a day or two, fat stores last several weeks but protein is increasingly catabolised for energy, most coming from muscle breakdown but almost all tissues are affected. In prolonged and severe deficiency synthesis of enzymes, peptide hormones, transport proteins and components of the immune system is diminished.

The resulting malnutrition is thus a combination of protein and energy deficiency, now usually termed protein-energy malnutrition (PEM). In surgical patients, as in malnourished children in whom it was first characterised (McLaren 1966), the clinical state of severe deficiency varies, depending on the degree to which protein or energy deficit predominates. It is not generally realised by surgeons that the term PEM does not denote a precise clinical syndrome but covers a whole spectrum of disease, the severe forms of which go by the names of kwashiorkor (predominantly protein deficiency) and marasmus (predominantly energy deficiency) and an intermediate form (marasmic kwashiorkor). Apart from growth failure and impairment of intellectual development the syndromes of kwashiorkor and marasmus seen in adult surgical patients are essentially those described in malnourished children. It should, however, also be recognized that these syndromes are not due to protein/energy deficiency alone but are accompanied to a varying degree by biochemical and sometimes clinical evidence of vitamin or element deficiencies, the most important of which are discussed below.

Marasmus (*general inanition, cachexia, semistarvation*)
This results from an overall deficit in intake and/or utilisation of food. Emaciation due to wasting of depot fat and skeletal muscle is generalized but is most evident where these tissues normally give the figure its rounded configuration. The skin becomes thinned, dry, loose and inelastic, pale and cold with a tendency to cyanosis in the cold. It is often pigmented in patches, of a brownish colour anywhere on the body, most commonly on the face, but not sufficiently marked to be confused with pellagra. Sometimes heaping up of keratin around hair follicles, perifollicular hyperkeratosis, occurs mainly on the extensor aspects of the upper arms and legs and chest. The hair is dry, brittle and falls out easily.

Achlorhydria and diarrhoea are frequent, the latter often being terminal. There is bradycardia, lowered systolic and diastolic blood pressure, low venous pressure, reduced cardiac ouput and heart size. Vital capacity, respiratory rate, minute volume and efficiency are all reduced. The endocrine system is not uniformly affected. Growth hormone is decreased but thyrotrophic and adrenotrophic hormones are unaffected, as are corticosteroids. Gonadal atrophy leads to loss of libido, and amenorrhoea in the female. Hypothermia is frequent and contributes to death. The intellect remains clear but there is personality change, with inability to concentrate, irritability and apathy. Anaemia is usually mild, normocytic normochromic in type. Work capacity is reduced, due to muscle wasting, anaemia and cardio-respiratory decompensation. Weight loss may be as much as 50 per cent, with skeletal muscle, adipose tissue, skin, liver and intestine accounting for much of this. Total body water and extracellular water are increased in relation to total body weight, the loss of adipose tissue, very low in water content, accounting for much of this. Dependent oedema may occur and this is unrelated to hypoalbuminaemia as plasma proteins remain virtually normal. Intracellular water is decreased and the extracellular/intracellular water ratio may be useful in assessment (Elwyn et al 1975).

Kwashiorkor (acute visceral attrition)

In the fully developed state this presents as a dramatic syndrome in contrast to the generalized wasting of marasmus. In surgical patients many of the florid features of the childhood disease may be lacking and intermediate forms of mild degree are not infrequent.

Subcutaneous fat is generally well preserved but muscle wasting and oedema are usually present, the latter being associated with low plasma albumin and other transport proteins (e.g. transferrin and pre-albumin). Apathy, irritability and anorexia are usually marked. The skin lesions consist of desquamation over pressure points and between the legs. There is depigmentation in dark-skinned races and a fine branny desquamation and hyperpigmentation, often over the forehead, in the fair-skinned. Hair changes are similar to those in marasmus and often more marked. The liver is usually enlarged and infiltrated with fat. Anaemia is commonly of the iron deficiency type. Hypogonadism, gynaecomastia, pancreatic and intestinal dysfunction are sometimes present.

Protein deficiency in experimental animals (Daly et al 1972) and in patients (Irwin & Hunt 1974) has been shown to impair colonic wound healing, and postoperative parenteral nutrition improves anastomotic strength in depleted rats (Steiger et al 1973). There is considerable evidence of impaired cell-mediated immunity in undernourished surgical patients, although humoral immunity is unaffected (Law et al 1973 & 1974, Bistrian et al 1975). T system impairment has been demonstrated by delayed skin reactivity to primary or secondary antigens and reduced in-vitro lymphocyte responses to PHA. B system impairment resulted in reduced IgM antibody response to keyhole limpet haemocyanin. In both dogs (Dionigi et al 1977) and man (Law et al 1973) most tests of cell-mediated immunity returned to normal after total parenteral nutrition (see Ch. 12).

Surgical conditions associated with PEM

These are, in general, states in which (1) protein and energy metabolism are markedly increased, (2) excessive losses of body fluids occur, or (3) an underlying disease predisposes to malnutrition. Frequently a combination of factors is present.

Severe trauma, extensive surgery, generalized sepsis, hyperthyroidism, advanced cancer, pregnancy and infancy are all associated with hypermetabolism. Excessive losses occur in burns, fistulas (Himal et al 1974, Kaminsky & Deitel 1975), abscesses and acute pancreatitis (Blackburn et al 1976). Patients with various forms of malabsorption, including the blind loop syndrome, and massive resections not infrequently develop PEM, as do a subset of patients undergoing open-heart surgery, primarily those with rheumatic valvular heart disease (Abel et al 1976).

Fat malabsorption (steatorrhoea)

Although this is a clinical sign and not a syndrome attributable to deficiency of a nutrient, it constitutes an entity common to many surgical conditions and leads to a variety of nutritional deficiencies discussed here. Steatorrhoea after surgery occurs most commonly and severely in resection of ileum, particularly

terminal ileum and to a milder degree after gastro-enterostomy or vagotomy. Losowsky et al (1974) discuss the operative conditions and possible mechanisms involved.

Gut resection has been accompanied by megaloblastic anaemia and tetany (Pullan 1959), magnesium deficiency (Fletcher et al 1960), PEM of the kwashiorkor type (Harrison & Booth 1960) and hypochloraemic alkalosis (LeVeen et al 1967). Gastric surgery has been reported to lead to osteomalacia (Anon 1966), magnesium deficiency (Booth et al 1963) and kwashiorkor (Neale et al 1967).

Essential fatty acids (EFA)

About 10 g of essential polyunsaturated fat, mainly in the form of linoleic acid, is required daily and most diets readily supply this. Essential fatty acids have three known functions: they may be used as an energy substrate like any other fatty acid, they are an important component of phospholipid bilayers of cell membranes, and they are precursors of prostaglandins, thromboxane, which causes platelet aggregation and vasoconstriction, and prostacyclin, which inhibits these actions.

The main EFA, linoleic acid (18:2ω6), gives rise to a series of ω6 fatty acids and in deficiency not only are these fatty acids reduced in plasma but the normal inhibition of oleic acid (18:1ω9) by linoleic acid is interfered with and an abnormal fatty acid 20:3ω9 makes its appearance. Linoleic acid normally gives rise to arachidonic acid (20:4ω6), which is also one of the EFA and in deficiency the hallmark is often taken as a plasma ratio of 20:3ω9/20:4ω6 of 0.4 or over.

Manifestations of EFA deficiency in man are similar to those reported in experimental animals and include dermatitis, decreased growth, diarrhoea, fatty liver and abnormal platelet function (Press 1980). Biochemical changes have been reported in patients with carcinoma of the oesophagus (Wapnick et al 1974) and abnormal plasma fatty acids and skin lesions in patients after gut resection with no more than 90 cm of small bowel remaining (Press et al 1974). The time course of development of changes was found to be as follows by Goodgame et al (1978) during fat-free parenteral nutrition: 1 week, eicosatrienoic and linoleic acid plasma levels abnormal; 2 weeks, arachidonic acid abnormal; 4 weeks, triene: tetraene ratio 0.4; and skin lesions after 2 months. Biochemical and clinical changes respond to i.v. administration of 100–300 g linoleate in the form of intralipid.

Vitamin A (retinol)

Deficiency is characterised by night blindness, occasionally xerosis of the skin and perifollicular hyperkeratosis and in advanced cases xerophthalmia and keratomalacia. Absorption of this fat-soluble vitamin is impaired in all causes of steatorrhoea (see above) and vitamin A-responsive impairment of dark adaptation has been reported after total gastrectomy (Adams et al 1960). In a recent study (Cohen et al 1979) large oral or i.m. doses of vitamin A (300 000–450 000 iu daily for 7 days) were shown to prevent postoperative

depression of the total lymphocyte count and response in a mixed lymphocyte reaction to a pool of stimulating cells which occurred in patients not receiving vitamin A. This vitamin is known to act as an adjuvant and steroid antagonist in the immune response (Cohen & Cohen 1973).

Vitamin B_{12} (cobalamin)
Deficiency as indicated by lowered plasma concentration (normal 150–750 pg/ml) results in megaloblastic anaemia, subacute combined degeneration of the spinal cord, and sometimes retrobulbar neuropathy. Symptoms take several years to appear because of large body stores and at this time after gastrectomy, due to absence of intrinsic factor, 10–20 per cent of patients have low plasma vitamin B_{12} and 4 per cent have mild megaloblastic anaemia (Hoffbrand 1967). Deficiency is common in the blind loop syndrome, partly as a result of bacterial colonisation of the loop (Goldstein 1971) and is a less frequent cause of megaloblastic anaemia in Crohn's disease than folic acid deficiency (Dawson 1972).

Folic acid
Plasma folate (normal 3–21 ng/ml) reflects recent dietary intake and deficiency is best detected by red cell folate (normal 160–640 ng/ml whole blood, corrected to packed cell volume of 45 per cent). As a long-term effect of gastrectomy folic acid absorption may be impaired (Elsborg 1974) but megaloblastic anaemia is more commonly due to deficiency of vitamin B_{12} than of folic acid (Hoffbrand 1967). It has been reported complicating Crohn's disease (Dawson 1972).

Vitamin C (ascorbic acid)
In deficiency fibrous collagen is not formed, being replaced by a non-fibrous precursor leading to a weakness of connective tissue in skin, bone and capillaries. Wound healing is markedly delayed. Vitamin C absorption is reported to be impaired in patients undergoing vagotomy for peptic ulcer, probably as a result of reduced gastric acid secretion (Macdonald & Cohen 1972). Ascorbic acid facilitates the absorption of iron and this occurs in post-gastrectomy patients (Baird et al 1974).

Vitamin K
Most of the daily requirement for this vitamin, required by the liver for the synthesis of prothrombin (factor II) and four other coagulation factors, is obtained from bacterial synthesis in the gut and not from the diet. Reduction of quantitative prothrombin below 80 per cent of normal indicates deficiency and reduction below 20 per cent is associated with active bleeding. Bleeding and coagulation times are usually not altered significantly until the prothrombin level has fallen below 20 per cent. Bile acids are necessary for vitamin K absorption and surgical intervention in obstructive jaundice must be preceded by phytonadione (vitamin K_1) 10 mg s.c. or i.m. The newborn is especially susceptible to hypoprothrombinaemia due to inactivity of gut flora,

bile secretion or liver synthesis and surgery must be accompanied by phytonadione 1 or 2 mg i.m. In the presence of severe liver damage hypoprothrombinaemia unresponsive to vitamin K occurs.

Water and electrolytes

Dehydration and electrolyte imbalance are potential problems in all major surgical procedures but are readily controlled in modern practice. However, the introduction of long-term nutritional maintenance by elemental diets or parenteral feeding has encouraged surgeons to perform extensive bowel resection that was not previously possible. Absorptive defects may result depending on the degree of resection, the area resected, the presence or absence of the ileocaecal valve, and the state of the remaining valve (Zamchek & Broikman 1973) and also in reversible bypass procedures for the treatment or morbid obesity. Immediately postoperatively fluid and electrolyte losses due to vomiting and diarrhoea, fistula or intra-abdominal sepsis can threaten survival.

Potassium depletion (serum K < 3.8 mEq/1) is common in the early phase of the short bowel syndrome, severe burns and trauma. When serum K falls below 3.0 mEq/1 muscular weakness may result in hypoventilation, paralytic ileus, hypotension, tetany, kaliopoenic nephropathy and characteristic ECG changes.

Calcium, magnesium and phosphate deficiencies tend to develop several weeks later (see below).

Iron

Iron deficiency anaemia may occur in any debilitated surgical patient as a result of poor appetite, chronic blood loss or underlying disease. It is one of the commonest long-term complications of partial gastrectomy, with a higher incidence in women, and is the most frequent cause of anaemia in these patients. There is probably impairment of the mechanism normally responsible for increased iron absorption when iron status is lowered but reduced iron intake also appears to play a part (Baird 1967). Response to iron is good and vitamin C facilitates absorption (Baird et al 1974). Tovey & Clarke (1980) stress the need for a prophylactic approach to anaemia after partial gastrectomy.

Calcium, phosphorus and vitamin D

These are best considered together as vitamin D is concerned with the metabolism of both calcium and phosphorus.

Hypocalcaemia (serum calcium <2.5 mmol/1) is usually associated with prolonged immobilisation due to increased urinary excretion, malabsorption states with steatorrhoea and intestinal drainage. The characteristic signs of tetany, prolonged Q-T interval of the ECG and a mild encephalopathy usually appear when the serum calcium falls below 1.75 mmol/1.

Phosphate depletion has been neglected until recent years when, as a result of the long-term management of patients with haemodialysis and parenteral nutrition, it has become evident that it is responsible for disorders of many