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Undernutrition in hospital patients

By Susan Holmes

Aims and intended learning outcomes

This article describes undernutrition, its potential causes and consequences, and its importance in the care of hospital patients.

After reading this article you should be able to:

- Recognise that undernutrition causes significant morbidity due to the loss of lean body mass (LBM).
- Understand the body’s response to physiological stress.
- Be aware of potential interactions between nutrition and drugs.
- Identify the factors that contribute to undernutrition in hospital patients.
- Recognise the role of nutrition in good clinical practice and effective clinical governance.

Around 40 per cent of hospital patients are malnourished regardless of age or illness (McWhirter and Pennington 1994). Moreover, the longer the hospital stay, the more likely patients are to experience malnutrition. Indeed, 70 per cent or more are malnourished on discharge (McWhirter and Pennington 1994, RCP 2002).

Although undernutrition affects individuals in different ways depending on their age, illness and initial body weight, those who are undernourished have significantly longer hospital stays and experience greater morbidity and mortality (Forman 1996, Warnold and Lundholm 1984). Acutely ill patients are also more likely to die if undernourished (RCP 2002). Yet, while undernutrition may be attributable to illness, it can often be prevented and clinical outcome can be improved with appropriate care. The fact that this does not occur indicates the low priority afforded to nutrition. Evidence reveals that neither managers nor clinicians view nutrition as important (Maryon-Davis and Bristow 1999). Furthermore, the focus on illness means that nutrition tends to be overlooked, and because patients are cared for by many healthcare professionals in different disciplines, no one group accepts responsibility for nutrition. This is not surprising as nurses and doctors are not trained to anticipate nutritional problems and as a result nutrition is often neglected through lack of awareness of its clinical benefits (Lennard-Jones 1992, RCP 2002).

Defining malnutrition

The term ‘malnutrition’ literally means bad nutrition and applies to any condition in which deficiency, excess or imbalance of energy, protein or other nutrients adversely affects body function and/or clinical outcome (RCP 2002). It may, therefore, reflect both under- and overnutrition, deficiencies in specific nutrients or dietary imbalance because of disproportionate intake of particular foods/food components (Keller 1993). Definitions of the terms used in clinical nutrition are outlined in Box 1.

Although this article focuses on undernutrition, the problems of overnutrition (overweight/obesity) must not be overlooked as they are the most prevalent nutritional disorders in the western world (Drummond 2002, WHO 1998). In England and Wales, more than 50 per cent of women and 60 per cent of men are overweight, and 17 per cent and 21 per cent respectively are obese (Joint Health Surveys Unit 1999). Thus, obesity is a significant public health problem.
issue. It is also important in terms of undernutrition as it may mask significant nutritional deficits.

The terms ‘malnutrition’ and ‘undernutrition’ as used in this article, refer to the wasting condition resulting from deficiency of protein and energy accompanied by varying degrees of trace nutrient deficiency. This is the most common form of undernutrition in hospital (Pinchcofsky-Devin and Kaminski 1986, RCP 2002, Torun and Cherv 1994).

**Inadequate energy intake** There are two types of energy depletion: chronic protein-energy deficiency, and acute undernutrition demonstrated by recent, unintentional weight loss. Chronic protein-energy deficiency, as its name suggests, is a chronic disorder representing a prolonged period of inadequate energy intake reflecting, for example, starvation or conditions such as cardiac disease, cancer, chronic respiratory or gastrointestinal (GI) disorders. It is manifested by a significant reduction in skinfold thickness and arm muscle circumference, due to loss of subcutaneous fat, and associated muscle wasting, due to protein loss from skeletal muscles. Such wasting also affects internal organs, such as the heart, kidneys and liver.

In contrast, acute undernutrition is primarily associated with acute and life-threatening conditions, which result in hypermetabolism and increase the demand for protein and energy at a time when intake is usually reduced. In such circumstances, undernutrition can develop rapidly. There is increasing evidence that acute food deprivation can have adverse functional effects impairing convalescence (RCP 2002).

**Trace nutrient deficiency** Vitamin and mineral deficiencies may affect any patient who has increased requirements, for example, an increased demand for vitamins during periods of sepsis. Such deficiencies particularly affect nutrients that are stored in the body in limited amounts, such as the water-soluble vitamins (the vitamin B complex and vitamin C).

**TIME OUT 1**

Reflect on your current patient caseload and consider how many patients may be malnourished. Is their condition acute or chronic? Can you identify reasons why they have become undernourished? Make a list of the possible contributing factors and add to this list as you read through the article.

**Consequences of undernutrition**

Although the effects of undernutrition on recovery are well-recognised, clinical surveys continue to reveal an unacceptably high incidence of undernutrition in hospitals (Ela and Stratton 2000, McWhirter and Pennington 1994). This increases re-admission rates (Sullivan 1992, Tierney et al 1994) and the costs of patient care (Larsson et al 1990, McCamish 1993, Robinson et al 1987).

Undernutrition affects many body functions, influences responses to disease/infection and reduces quality of life (Ela 2000). More obvious effects include weight loss, depletion of subcutaneous fat and progressive muscle wasting (Brozek 1990). Indeed, because weight is easily monitored it is a commonly used indicator of nutritional status (Box 2). The body mass index (BMI), which is strongly related to the percentage fat mass adjusted for height in middle-aged adults, is a better marker (RCP 2002). However, like weight, it cannot distinguish
between fat and LBM and so may be misleading. Both measurements are readily distorted by, for example, oedema or muscle loss due to neurological disorders.

Undernutrition causes apathy, malaise and lethargy, even when not associated with disease. During illness, those who are undernourished often have low morale, finding it difficult to care for themselves. Many exhibit a reduced will to recover (Silk 1994). Undernutrition also significantly decreases the desire and the ability to eat, further reducing nutritional status, and creating a cycle of apathy and disinterest in food, from which it can be difficult to escape.

More specifically, the decline in body mass, with physical weakness, inhibits mobility, increasing liability to deep vein thrombosis and pressure sores (Close 1993). Respiratory muscle weakness causes difficulty in expectorating, increasing susceptibility to chest infection (Arora and Rochester 1982). The risk of cardiac failure is also enhanced (Heymsfield et al 1978). Immunocompetence declines, increasing the risk of infection, which in turn reduces nutritional status (Ward 2002).

Undernourished surgical patients develop increased complications (Warnold and Lundholm 1984), for example, wound healing is prevented and dehiscence is common (Trujillo 1993).

**TIME OUT 2**

Consider the patients that you identified as undernourished in Time Out 1, how has their condition been affected by their poor nutritional status? What steps can you take to improve their condition?

**Causes of undernutrition**

There are two main ways in which undernutrition develops. The first, protein-energy malnutrition (PEM), arises during acute injury or illness when increased nutrient requirements and loss of body protein are common (Bessey et al 1989, Wernerman et al 1985). Additional stressors such as pain and anxiety can exacerbate PEM.

The second cause is inadequate nutrient intake during a time of increased nutritional demand or over a prolonged period of reduced dietary consumption, perhaps exacerbated by impaired absorption or metabolism. This is particularly common in older people and those with disabilities and chronic or mental illness. While some degree of adaptation occurs during the early stages of weight loss, resulting in preservation of LBM, PEM eventually develops. However, regardless of the cause (Box 3), PEM may be exacerbated by hospitalisation, treatment and/or a range of psychosocial factors.

**Adaptive response to starvation**

The ‘normal’ response to starvation must be understood before the response to physiological stress is considered. In healthy adults, available stored energy comprises approximately 200g glycogen, 6,000g protein and, although subject to individual variation, 15,000g fat. During starvation, glycogen stores are rapidly depleted (15-20 hours) (glycolysis), supplying about 800kcal. Following this, protein is mobilised from skeletal muscles, converted to glucose in the liver (gluconeogenesis) and released into the circulation. As much as 75g protein may be used for this purpose daily and is reflected by a negative nitrogen balance. The body adapts over three to four days, using fat as an energy source, thus reducing protein catabolism and decreasing nitrogen excretion.

Fatty acids released from fat may be used directly for energy or converted to glucose or ketone bodies (ketogenesis). Body tissues adapt to metabolising ketone bodies for energy and 70 per cent of the brain’s energy needs can be supplied in this way. This conversion process is promoted by a progressive decline in circulating levels of insulin and differs from the usual situation when severe ketonaemia stimulates insulin secretion. Since insulin strongly inhibits ketogenesis, feedback mechanisms prevent ketosis from reaching pathological levels. In the final stage of starvation, fat stores are depleted and total energy needs are obtained from plasma proteins and proteins in visceral organs, and this ultimately results in death.

These responses are primarily protective, designed to arrest the progress of PEM (Hoffer 2001). Fat loss is slowed by reduced energy expenditure associated with a decline in basal metabolic rate (BMR) and in LBM (Shetty 1999). Muscle protein initially bears the brunt of the loss, while organ tissues are relatively spared so that, as long as the intake of energy and protein is not too low, adaptation reduces energy and protein requirements, restoring homeostasis and maintaining physiological functions (Hoffer 2001). Healthy adults can tolerate a 35-40 per cent loss of body weight (1,200-1,800g body protein) before death occurs. However, a smaller weight loss is fatal in those who are nutritionally depleted or underweight before starvation occurs. Patients who are acutely ill do not adapt as readily to starvation.

**Physiological stress response to illness, trauma or infection**

The body’s response to severe injury, trauma, infection, wounds, or surgery, is characterised by a significant hormonal reaction. The initial response – the
‘ebb phase’ – is primarily a response to shock. It is characterised by a decrease in blood pressure and reduced cardiac output, body temperature and oxygen consumption, which results in hypovolaemia, hypoperfusion and lactic acidosis. This is followed by the ‘flow phase’, where adaptation occurs and body resources are mobilised to counteract these negative effects. In contrast to the ebb phase, which is brief, the flow phase may last for several weeks or longer, resulting in a neuroendocrine response, which stimulates hypermetabolism (Figure 1).

The hormonal response, mediated by the sympathetic nervous system, causes marked increase in the release of catecholamines – adrenaline (epinephrine) and noradrenaline (norepinephrine) – and other stress hormones, such as cortisol. There is also a concurrent decrease in anabolic hormone secretion, for example, human growth hormone and testosterone.

A sustained increase in body temperature and marked elevation in the demand for glucose also occurs. The demand for glucose is met by catecholamine-stimulated glycogenolysis (breakdown of stored glycose), gluconeogenesis (synthesis of glucose) and fat mobilisation. Cortisol mobilises amino acids from skeletal muscle leading to rapid muscle breakdown. These combined effects cause significant elevation of BMR and, hence, energy demand is increased. Fifty per cent of this is drawn from body fat, 30 per cent from carbohydrate and 20 per cent or more from protein, which rapidly depletes body tissues and LBM (Bessey et al 1989, Kester et al 1987). This may be masked by sodium and water retention due to release of aldosterone and antidiuretic hormone (ADH), which helps to support circulating blood volume. Weight loss might not be apparent until diuresis occurs.

The severity of the catabolic response depends largely on the degree of injury or infection (Table 1), and thus a previously protective mechanism becomes self-destructive. Controlling this response while also supporting the metabolic demand is vital to avoid further deterioration. If sufficient nutrients, particularly carbohydrate or fat, are not available, then body protein is catabolised to provide energy. Blood loss, exudates and discharges exacerbate protein loss. PEM can develop rapidly and, if uncontrolled, will progress to multiple organ failure (Cerra 1987, Wernerman et al 1985). It is the magnitude of LBM loss that produces the morbidity and mortality associated with PEM.

Anabolism does not usually occur until wounds are healed or infection is resolved. Once this is achieved, corticosteroid release gradually declines followed by spontaneous diuresis and reduced nitrogen excretion. This transition lasts one or two days and is followed by anabolism, during which LBM and muscular strength increase. The total nitrogen loss is eventually regained, though recovery is slower than the rate at which it was lost (Souba and Wilmore 1988).

Drug-nutrient interactions

Although drug-nutrient interactions can produce unexpected therapeutic effects or additional health problems, they are poorly understood by healthcare professionals. Drugs may interfere with nutrient absorption, digestion, metabolism, use or excretion, potentiating undernutrition. Conversely, nutritional status and diet can affect the metabolism and function of drugs. Drugs may act centrally or peripherally, causing
anorexia or decreasing appetite due to side effects. Centrally-acting drugs include dopaminergics (for example, levodopa), serotonergics (for example, clomipramine) and endorphin modulators (for example, naloxone), while peripheral-acting agents include those inhibiting gastric emptying and bulking agents. Moreover, many drugs alter GI function, causing nausea, vomiting, diarrhoea or constipation, thus decreasing appetite.

Dietary factors may decrease, delay or enhance drug absorption, altering their availability, solubility or the length of time they remain in the intestinal tract. For example, since absorption of many antibiotics (for example penicillins, ketoconazole and rifampicin) is decreased by food, they should be given one hour before or two hours after meals (Kirk 1995). Other examples include erythromycin, which is susceptible to inactivation by gastric acid and decreased absorption when given after food or nutritional supplements (Segal and Kaminski 1996), and tetracyclines, which form complexes with calcium, thus significantly decreasing therapeautic efficacy.

Hot food, high viscosity solutions and, to a lesser extent, high protein and carbohydrate foods, all delay gastric emptying and may reduce the half-life of some drugs, including theophylline (Dickerson 1988). The pH of the GI tract also affects drug disposition and prolonged retention in gastric acid may accelerate dissolution of basic drugs, such as penicillin, while delaying that of acidic drugs, for example, tetracyclines.

The rate of metabolism in the liver and elsewhere (for example, lungs, kidneys) is an important determinant of drug effects. Drug metabolism occurs in two stages. Although there are exceptions, phase I usually involves oxidation, which alters the chemical composition and either activates or deactivates the drug. Phase II generally conjugates oxidised drug activity of drug-metabolising enzymes so that drug concentrations decline more slowly, increasing drug efficacy.

Inadequate nutrient intake

Inadequate nutrient intake is common during illness and potentiates undernutrition. Risk factors include:

- Increased nutritional demand.
- Increased nutrient losses.
- Impaired digestion and absorption.
- Inability or unwillingness to eat.

There are many reasons why patients do not eat adequately (Box 4). Many can be attributed to the disruption of sociocultural, psychological and physiological factors that occurs as a result of illness and/or hospitalisation. For example, admission to hospital may cause significant stress markedly reducing interest in food. Similarly, although it is known that eating improves when some control is exerted over the diet, patients can rarely influence either their food or times of eating. Personal idiosyncrasies about eating, combined with the expectation of poor quality food (Audit Commission 2001), may mean that meals are not eaten, even when the food is adequate. While acknowledging the difficulty in addressing individual likes and dislikes, a remarkable inflexibility remains in relation to the choices offered to patients. Grossman (2002) reports that hospital catering does not reflect the habits of people in the ‘real world’ and as a result patients have to change their eating behaviour. Addressing such matters can enhance food consumption.

**REFERENCES**


Prolonged starvation often contributes to under-nutrition. For example, fasting for diagnostic tests mitigates against optimal nutrient intake and may be exacerbated by pre-operative starvation. Many patients are starved for significantly longer than is necessary (Greenfield et al. 1997, Maclean and Renwick 1993). As the fasting stomach secretes up to 50mL/hr of gastric juice, and clear fluids rapidly leave the stomach (half the volume disappears within 10-20 minutes (Sutherland et al. 1987)), this is illogical. Although those with delayed gastric emptying should not drink before anaesthesia, there is overwhelming evidence to suggest that clear fluids should be permitted up to two hours before it is given (Greenfield et al 1997).

Similarly, hospitalised patients regularly miss 11-27 per cent of meals (Eastwood 1997). Although clinical investigations account for 7.6 per cent of these, more than 92 per cent result from illness and/or the quality of food provided: this is exacerbated by mealtimes visits by medical personnel (Deutekom et al. 1991). Thus, it is not surprising that many patients are vulnerable to nutritional deple-

TIME OUT 5

Reflecting on a patient in your care, list the factors that may contribute to an inadequate nutrient intake. Design a care plan for that patient taking these into account and monitor its impact in terms of food consumption, nutritional status and general wellbeing. Discuss your care plan with a dietitian to evaluate its potential effectiveness.

Increased nutrient demands The patient’s illness and the treatment received may affect nutrient demands. The process of wound healing will also

Box 4. Factors contributing to inadequate nutrient intake

- Ageing
- Chronic or mental illness
- Alcoholism or drug addiction
- Food idiosyncrasies and/or avoidance of particular foods/food groups
- Inadequate dentition
- Social isolation
- Poverty
- Anorexia/appetite loss
- Taste changes
- Malabsorption
- Ignorance or lack of information regarding appropriate food choices
- Hospitalisation and treatment received

Box 5. Examples of physical symptoms inhibiting food consumption

- Anorexia
- Dysphagia
- Nausea and vomiting
- Taste changes
- Tooth decay and/or periodontal disease
- Oral mucositis (stomatitis)
- Diarrhoea/constipation
- Dyspnoea and respiratory distress
- Pain

The patient’s illness and its treatment may affect nutrient demands. The process of wound healing will also


increase nutrient requirements (Whitney and Heitkemper 1999).

**Increased nutrient losses** Nutrient losses are increased by blood loss, exudates and discharges, as well as by any condition affecting intestinal function, such as inflammatory bowel disease, drug-induced intestinal disorders and chronic diarrhoea.

**Nutritional needs**

The relationship between good nutrition and clinical outcome is well-established and hospitals have a duty to ensure that patients’ nutritional needs are met (NHS Estates 2001). Guidelines for hospital catering stipulate that the diet should deliver an energy intake of 1,800-2,200kcal and a protein intake of 45-55g per patient per day (DoH 1996).

Allison (1999) suggests that these recommendations are low for some patients and that 30kcal and 1.0-1.5g protein per day are required for each kilogram of body weight, which he claims should meet the needs of the majority of patients.

However, consumption of apparently adequate amounts of food does not necessarily mean that patients are appropriately nourished. Dietary imbalance or vitamin losses resulting during food preparation and storage are common, and hospital diets are known to be, at best, adequate only for maintenance rather than replenishment (Maryon-Davis and Bristow 1999). For example, Simon (1991) showed that the food served may be deficient in one or more essential nutrients, including not only energy but also nutrients such as folic acid, iron and vitamin D. It cannot, therefore, be assumed that a nutritionally adequate diet is always available to patients. The NHS Plan (DoH 2000) and the Better Hospital Food plan (NHS Estates 2001) have established targets for improvements in hospital food in England. These include:

- A new menu framework describing the meals to be provided and the minimum choice to be offered, plus a menu brochure is to be offered to each patient.
- Twenty-four-hour access to food by providing better food in ward kitchens, snack boxes and ‘lite bites’ (Box 6).
- Two of the dishes developed by a panel of leading chefs must be offered daily.
- Provision of snacks with mid-afternoon and evening beverages.
- The nutritional requirements of a variety of patients must be met and checked by a state-registered dietitian.

This four-year project, supported by £40 million of investment funding, has yet to be evaluated. However, as long as the majority of studies regarding the quality of hospital food address matters of personal taste/satisfaction rather than nutritional adequacy, and nutrition continues to be viewed as a ‘hotel’ service rather than as an integral part of clinical care (Allison 1999), its nutritional benefits may be limited. Undernutrition may persist unless healthcare practitioners become proactive in its prevention.

At the same time, attempts must be made to reduce the high levels of food wastage (Allison 1999, Barton et al 2000), which range between 16 and 67 per cent (Edwards and Nash 1997, Stephen et al 1998). Although Grossman (2002) claims that levels of food waste are decreasing, many factors, which have clinical and financial implications, contribute to this problem. These range from palatability to portion size and from individual appetite to inadequate help with eating.

In clinical terms, wastage contributes to nutritional depletion. For example, Stephen et al (1998) showed that patients were consuming only 70-75 per cent of the required energy and 70 per cent of the necessary protein. Even lower consumption was demonstrated by Wilson (1998), who suggests that the average energy deficit is approximately 58 per cent of daily requirements per patient. Therefore, it is not surprising that undernutrition affects so many patients. In financial terms, the total cost of wastage is at least £45 million per year (Allison 1999). It is little wonder that significant attention is now being directed towards this aspect of hospital food provision.

**Nutrition and clinical governance**

Undernutrition is also important in terms of clinical governance, which aims to guarantee high quality health care and to ensure that clinical, managerial and educational practice is based on the best available clinical evidence (McSherry and Haddock 1999).

It is for this reason that nutrition is one of the central features of the Essence of Care benchmarking project (DoH 2001) designed to improve the quality of ‘fundamental and essential aspects of care’, building on the NHS Plan (DoH 2000). It is sobering to realise that health professionals often fail to ‘get the basics right’ (NHS Estates 2001). Indeed, Kester P et al (1987) Metabolic response to trauma. Contemporary Orthopaedics. 14, 53-59.


Nutrition

Crossman (2002) suggests that: ‘If you cannot get the food right you will probably never get everything else right’. Nutrition should be fully integrated into clinical care. Relatively simple actions can promote eating and enhance patient nutrition (Davis 2002, Ellis 2002). Examples include:

- Choosing food shortly before consumption rather than the day before.
- Ensuring that cutlery and crockery are clean.
- Ensuring that ice cream has not melted before it is delivered to patients.
- Offering foods appropriate to the patient.

The RCP (2002) highlights reasons why nutrition ‘fits’ neatly into a clinical governance approach:

- Nutrition is clearly linked to health, wellbeing and clinical outcome.
- Patients and carers are concerned about the quality of food and nutrition.
- Simple measuring tools are available for assessing nutritional status and monitoring consumption.
- Improving patient nutrition can contribute to considerable saving of resources in the longer term.

Conclusion

Nutrition is fundamental to good clinical care and provides a good indicator of the quality of care delivered. Many hospitals have now developed clinical guidelines and standards designed to overcome undernutrition and promote eating, building on the requirements outlined in the NHS Plan (DoH 2000), to ensure that the quality and availability of food are improved. Standards have been established for nutritional screening and assessment (Elia 2001), which should now be in place in all clinical environments (DoH 2000). Using a structured approach to nutrition (DoH 2001), nurses can ensure that all patients are screened on admission and that appropriate action is taken to meet individual nutritional needs.

Undernutrition affects significant numbers of hospital patients in whom it delays recovery and increases the cost of hospitalisation. It also increases the risk of mortality. The prevention of undernutrition in hospital patients requires collaboration and should be a primary aim of all healthcare professionals. However, no group of healthcare professionals currently accepts responsibility for ensuring adequate nutrition of general hospital patients. Although nurses cannot accept sole responsibility, they can and should play a central role in patient feeding. Similarly, recognition of the risk of undernutrition must, for most patients, rest with nurses. It is, therefore, incumbent on nurses to ensure that their knowledge of nutrition is adequate to permit them to fulfil this role.


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