

A joint meeting of the Scottish Section and the Clinical Nutrition and Metabolism Group of the Nutrition Society and the East of Scotland British Dietetic Association was held at the Institute of Electrical Engineers, Glasgow on 27–28 March 2003

Symposium on ‘Nutrition in the clinical management of disease’

Changing perspectives in the nutritional management of disease

Angela M. Madden

Department of Health and Human Sciences, London Metropolitan University, Holloway Road, London N7 8DB, UK

There have been substantial changes in the nutritional management of many diseases in the last 20 years, which have been accompanied by a growing recognition of its importance. Many of the changes in clinical nutrition have been associated with the introduction of standards, clinical audit and the implementation of evidence-based practice, which has led to a re-evaluation of some established dietary interventions using a hierarchy-of-evidence approach. Although there are few randomised controlled trials on which to base such work, the examination of other, often less-robust, evidence has led to some traditional dietary interventions being modified. Examples in gastroenterology include the use of low-fat diets in gall bladder disease and the restriction of protein in hepatic encephalopathy, where the current evidence suggests that neither should be used routinely in clinical practice. Where therapeutic dietary restrictions are required, as with low-Na diets in ascites, there is very little information on how these restrictions influence total nutrient intake and, if intake is impaired, how the detrimental effects of an inadequate intake should be balanced with the therapeutic effects of restriction. Studies are required to ensure that nutritional interventions are not only effective but also free from undesirable side effects. The mode and timing of the delivery of nutritional support has also been re-evaluated and the benefits of early enteral feeding have been recognised. The delivery of dietary advice is a new area that is being considered, with practitioners in clinical nutrition using behaviour-change skills to facilitate optimum nutrition rather than simply providing patients with advice. For such developments to continue in clinical nutrition it is essential that all practice should be systematically evaluated and, where necessary, modified in the light of sound current research findings, and that gaps in our present knowledge base are identified and addressed.

Nutritional management of disease: Dietary interventions: Re-evaluation of evidence

The nutritional management of many diseases has changed substantially in the last 20 years. It is difficult to quantify this change directly, but an indication is given by the increase in the number of practitioners working in this field (membership of the Clinical Metabolism and Nutrition Group of the Nutrition Society has increased from 30 to > 1200 in the last 14 years despite overall membership of the Society remaining stable; membership of the British Dietetic Association has doubled over the same period and, since its formation in 1995, so has that of the Parenteral and Enteral Nutrition Group of the British Association for Parenteral and Enteral Nutrition) and by the greater number of relevant peer-review publications and scientific meetings

(the number of clinical nutrition papers published per year has quadrupled in the same time frame). The inclusion of clinical nutrition in the *Core Curriculum for Nutrition in the Education of Health Professionals* (Nutrition Task Force, 1994) indicates that this increase is not simply numerical but demonstrates an increasing acceptance of the importance of work in this field.

Causes of change

Whilst few developments are brought about by single factors, the advances that have occurred in clinical nutrition in the last two decades can probably be linked to those

Abbreviation: RCT, randomised controlled trials.

Corresponding author: Dr Angela M. Madden, email a.madden@londonmet.ac.uk

occurring across the health care spectrum following the introduction of the routine use of clinical standards and their audit in the 1980s (Dixon, 1996). These developments rapidly led on to, or at least were paralleled by, the application of evidence-based practice or, in the absence of evidence, an aspiration towards this approach. The concept of evidence-based practice is, however, not new; the philosophy originated in the mid 19th century (Sackett *et al.* 1996). What is new is its general acceptance as a fundamental principle of clinical practice. This approach has been facilitated by the simultaneous revolution in information technology, which has enabled most practitioners to examine the evidence available through shared research findings (e.g. on research databases) and to search peer-reviewed publications via the Internet (Latchford, 2002). The distillation and evaluation of evidence by experts through the Cochrane Collaboration has further supported this work (Cochrane Library, 2003). The questioning of established practice in the light of the evidence available has revealed that some practice has been based on perceived wisdom that is no longer pertinent to nutritional management in the 21st century. This finding does not imply that past generations working in clinical nutrition were cavalier or negligent, but that they did not have access to the subsequent developments in other clinical areas (including medicine, surgery, pharmacology and medical physics) that can now facilitate further investigation and gathering of more reliable evidence on which to base current nutritional management. It does, however, suggest that the evaluation of practice is an ongoing process of development and that in due course current innovations may be shown not to be a permanent solution.

Examining the evidence

When examining the available research findings on which to base practice, health care professionals often use a hierarchy of evidence to rank the importance of study results and to inform practice (Guyatt *et al.* 1995; Greenhalgh, 2001; Evans, 2003). Traditionally, such hierarchies have been headed by randomised controlled trials (RCT; Guyatt *et al.* 1995), and more recently by systematic reviews and meta-analyses (Shekelle *et al.* 1999; Scottish Intercollegiate Guideline Network, 2000; Evans, 2003). Although this approach may provide a useful tool in many disciplines, more consideration is required to its application in clinical nutrition where an absence of RCT, systematic reviews and meta-analyses may suggest, perhaps erroneously, that no good evidence is available. The areas of clinical nutrition in which the evidence has been systematically examined are often determined by local opportunities, available funding or personal interest and, to date, a limited but increasing number of systematic reviews have been completed in the area of nutritional management of disease (Cochrane Library, 2003).

The findings from well-conducted RCT are best used to evaluate the effectiveness of an intervention. They may not, however, address other aspects of care that are relevant in clinical nutrition, including the appropriateness and feasibility of a particular treatment, and these issues may require research studies of different design (Evans, 2003). In

Table 1. Hierarchy of evidence showing the relative order of importance of research findings to be considered when making decisions about interventions in clinical nutrition (adapted from Elia *et al.* 2000; Scottish Intercollegiate Guideline Network, 2000; Greenhalgh, 2001)

Systematic reviews and meta-analysis	Strongest evidence
Randomised controlled trials with definitive results*	↓
Randomised controlled trials with non-definitive results	↓
Cohort and case-control studies	↓
Cross-sectional studies	↓
Case reports	↓
Opinions and/or clinical experience of respected authorities†	Weakest evidence

*Definitive results are those with confidence intervals that do not overlap the threshold clinically-significant effect.

†Published in peer-reviewed journals or by recognised specialist societies or committees of experts.

addition, the capacity to examine some nutrition interventions by RCT may be limited, e.g. by ethical considerations associated with not feeding patients, the lack of suitable food-based placebos or the size of study required to show a statistical difference between treatments. As a consequence, a broader view of the evidence is important in clinical nutrition and requires a hierarchy that addresses all aspects of care and includes evidence that is practical and relevant to the situation being examined. A number of alternative approaches have proposed rating schemes that include other non-RCT research evidence including the opinions and/or clinical experience of respected authorities (Shekelle *et al.* 1999; Scottish Intercollegiate Guideline Network, 2000; Greenhalgh, 2001; Mulrow & Lohr, 2001; Evans, 2003; Table 1). A good example of the consideration of a fuller range of evidence in clinical nutrition is the evaluation of malnutrition screening tools by Elia *et al.* (2000) where no RCT and a limited number of other studies have been carried out. Where this type of evaluation is required, it is essential that it is undertaken carefully; in the absence of accepted criteria to evaluate less-robust types of evidence, the Scottish Intercollegiate Guidelines Network (2000) advises that non-analytical studies and expert opinion should be considered only if published either in a peer-reviewed journal or in a report by a recognised specialist society or committee of experts.

Gastroenterology

Most of the systematic reviews and meta-analyses of the nutritional management of adults with gastrointestinal disorders published to date have been concentrated in a number of specific areas, including inflammatory bowel disease, irritable bowel syndrome, pancreatitis, post-surgical nutrition support and hepatic encephalopathy (Table 2). Each review and analysis undertaken has focused on one or two clearly-defined research questions in order to provide a definite answer on which future management can be based. In some cases they have concluded that there are insufficient data available to enable the question to be answered

Table 2. Systematic reviews and meta-analyses of studies undertaken to examine the nutritional management of selected gastrointestinal disorders in adults

Clinical area	Reference	Main conclusion
Inflammatory bowel disease	Fernandez-Bañares <i>et al.</i> (1995)	Peptide-based enteral feeds are less effective than steroids in inducing remission in active Crohn's disease. The data on elemental and whole-protein-based diets are less conclusive (sixteen studies)
	Griffiths <i>et al.</i> (1995)	Enteral nutrition is less effective than corticosteroids in the treatment of active Crohn's disease (eight studies). There is no difference in the efficacy of elemental v. non-elemental diets (five studies)
	Middleton <i>et al.</i> (1995)	Long-chain triacylglycerols in enteral feeds are negatively associated with remission rates in patients with active Crohn's disease (twenty studies)
	Messori <i>et al.</i> (1996)	Defined formula diets are less effective than steroids in inducing remission in active Crohn's disease (seven studies)
	Zachos <i>et al.</i> (2003)	Enteral nutrition is less effective than corticosteroid therapy in inducing remission in active Crohn's disease (four studies). There is no difference in the remission-inducing effects of elemental and non-elemental diets (nine studies)
Irritable bowel syndrome	Niec <i>et al.</i> (1998)	It is unclear if adverse food reactions are a key factor in exacerbating symptoms (seven studies)
Pancreatitis	Al-Omran <i>et al.</i> (2003)	There are insufficient data to conclude whether enteral or total parenteral nutrition is more effective in acute pancreatitis, although there was a trend towards a reduction in adverse outcomes with enteral nutrition (two studies)
Hepatic encephalopathy	Eriksson & Cohn (1989)	BCAA do not influence outcome in patients with acute hepatic encephalopathy (seven studies). BCAA induce positive N balance to the same extent as dietary protein without exacerbating encephalopathy as frequently in patients with chronic hepatic encephalopathy (four studies)
	Naylor <i>et al.</i> (1989)	Parenteral BCAA are associated with a marked improvement in mental recovery. Uncertainty about effects on mortality (five studies)
	Fabbri <i>et al.</i> (1996)	Oral BCAA may be useful in the prevention and treatment of patients with advanced cirrhosis who are intolerant to dietary protein (two studies)

BCAA, Branched-chain amino acids.

irrefutably (Niec *et al.* 1998; Al-Omran *et al.* 2003), and in many cases the authors conclude that further RCT are required. Thus, in addition to the areas that have not been examined strenuously, there are areas in which only specific questions have been addressed and others in which no conclusions have been made. This lack of systematic evidence on which to base the nutritional management of patients requires that evidence ranked lower in the hierarchy be evaluated. Two areas of gastroenterology in which this type of assessment has been undertaken and has led to a re-evaluation of dietary treatment are gall bladder disease and hepatic encephalopathy.

A survey of eighty-seven hospital-based dietitians in the UK showed that 92 % of them advised patients with gall stones to follow a low-fat diet in order to avoid pain, although 59 % considered that this dietary treatment required clarification (Madden, 1992). An examination of published studies shows that there is little evidence to support the use of low-fat diets in gall bladder disease and that what is available could be described as unconvincing.

A number of studies have shown that there is no association between the symptoms described by individuals and whether or not they have gall stones or an abnormal gall bladder (Bainton *et al.* 1976; Rome Group for the Epidemiology and Prevention of Cholelithiasis, 1984; Glambek *et al.* 1989; Diehl *et al.* 1990). Similarly, studies comparing reported dietary fat intolerance in patients with and without gall bladder disease have found no significant difference between the two groups (Price, 1963; Koch & Donaldson, 1964; Sampliner *et al.* 1970; Jorgensen, 1989),

and patients who describe fat intolerance have been shown to be unable to detect dietary fat given in a blind challenge (Taggart & Billington, 1966). Whilst an intake of dietary fat is likely to provoke contraction of the gall bladder, by stimulating the secretion of cholecystokinin in the small intestine, it has been shown that an equivalent secretion of cholecystokinin can be provoked by dietary protein (Hopman *et al.* 1985). Gall bladder contractions may also occur independently of cholecystokinin secretion in response to cephalic stimulation and it has been shown in healthy volunteers that the extent of gall bladder contraction provoked by an appetising fatty meal was not significantly different from that provoked by sham feeding (Hopman *et al.* 1987). These studies suggest that there is no specific mechanism by which fat alone is particularly likely to provoke greater gall bladder contractions and thus cause greater pain than other aspects of dietary intake. Even if there were such a mechanism, it could be argued that regular contraction of the gall bladder is potentially useful, at least in the prevention of gall stone formation as stagnant bile is more lithogenic. These findings suggest, therefore, that potential benefit from severely restricting dietary fat is unlikely (Madden, 1992). Ideally, such conclusions should be supported by an RCT, preferably double blinded, but the trial would be difficult to conduct in the face of entrenched views about the values of fat restriction held by both patients and professional staff. In terms of research and clinical priorities it is unlikely that such a trial will be undertaken because an unnecessary low-fat diet is unlikely to have a grossly detrimental effect on most patients, unless it is taken

to extremes, and for some patients it will represent a healthier alternative to their current intake.

The same cannot be said about the use of low-protein diets in the treatment of hepatic encephalopathy. The restriction of dietary protein became an established treatment for hepatic encephalopathy in the 1950s following the publication of observations that nitrogenous substances were associated with hepatic coma (Phillips *et al.* 1952). Although the observations supporting this restriction were uncontrolled and made in a small number of patients, dietary protein restriction became a central pillar of treatment in patients with hepatic encephalopathy (Sherlock *et al.* 1954, 1956) and remained relatively unchallenged for several decades. The results from N-balance and re-feeding studies undertaken in the 1990s showed that patients with cirrhosis require a higher intake of dietary protein, providing a minimum of 0.8 g/kg per d, in order to maintain N balance (Nielsen *et al.* 1993, 1995; Kondrup *et al.* 1997). It is likely, therefore, that the inadequate provision of dietary protein is a major contributor to the high prevalence of malnutrition observed in this patient population (Italian Multicentre Cooperative Project on Nutrition in Liver Cirrhosis, 1994; Caregaro *et al.* 1996; Loguerico *et al.* 1996). The dilemma of the apparently detrimental effects of dietary protein in patients with increased protein requirements has not been reconciled by an RCT (Soulsby & Morgan, 1999). However, the examination of published data suggests that the benefits associated with restricting dietary protein are tenuous whilst those associated with increasing protein intake to meet requirements are more convincing. These findings have been taken into account in European guidelines for feeding patients with cirrhosis, stating that those with well-compensated disease should receive 1.0–1.3 g protein/kg body weight per d, whilst in malnourished patients and those with decompensated disease this level should be increased to 1.5 g/kg per d (Plauth *et al.* 1997). These levels are higher than the 0.6 g/kg per d recommended for groups of healthy adults (Department of Health, 1991) and substantially more than the 20–30 g protein/d previously recommended for cirrhotic patients recovering from acute encephalopathy (Uribe & Conn, 1994).

Intake recommendations in disease

One of the difficulties of making specific recommendations about nutrient intake in particular patient groups is the need for these recommendations to be interpreted meaningfully at the clinical level. For example, the recommended protein intakes described earlier are expressed per kg body weight per d, as are energy intakes in the same guidelines, but these recommendations do not define how body weight should be determined (Plauth *et al.* 1997). In patients with chronic liver disease, body weight may be increased by the presence of gross fluid retention or be reduced as a consequence of accompanying malnutrition, and it is thus necessary to indicate whether actual weight, estimated dry weight or ideal weight should be used to calculate the recommended nutrient intake. This differentiation is necessary if optimum intake is to be achieved, as fluid-related variations in body weight may account for ± 25 kg (Arroyo *et al.* 1991). However, it is extremely difficult to measure dry weight

accurately and, to date, no studies have undertaken this investigation with the purpose of informing recommended nutrient intakes. Accurate measurement of body composition requires the application of a four-component model for which densitometry (using either underwater weighing or plethysmography), dual-energy x-ray absorptiometry and total body water measurements are used to determine fat-free mass and total body protein (Fuller *et al.* 1992). This measurement has been undertaken in a small number of patients with cirrhosis (Madden *et al.* 1998), and although this method is limited by the practicality of undertaking three techniques simultaneously, it could be used for further studies to refine the recommendations for energy and nutrient intakes in this patient population and others with similar fluid-retention problems.

Until this refinement is undertaken, estimated energy requirements will probably continue to be calculated from BMR, determined using prediction formulae, with adjustments made for activity level and disease process (Schofield, 1985; Elia, 1990). Whilst these calculations provide a useful and easily available goal for nutritional support, there is increasing awareness of the limitation of using formulae to predict BMR in individuals, especially those with specific clinical conditions (Daly *et al.* 1985; Hébuterne *et al.* 1996; Frankenfield *et al.* 1998; Aliprandi *et al.* 2001; Batterham *et al.* 2003). For example, in 100 patients with cirrhosis a comparison of values for resting energy expenditure calculated using seven prediction formulae (Harris & Benedict, 1919; Cunningham, 1980; Schofield, 1985; Owen *et al.* 1986, 1987; Mifflin *et al.* 1990; Müller *et al.* 1993) with those determined using indirect calorimetry showed substantial variation, whilst no significant differences were observed between predicted and measured values in a comparable group of healthy volunteers (Madden & Morgan, 1999). This finding is not surprising, because all but one of the prediction formulae examined were developed for use in a healthy population. However, the main concern is that the 95 % limits of agreement for individual patients showed that values predicted using the Schofield equations (1985) varied between overestimating measured values by 1576 kJ (377 kcal) and underestimating them by 2286 kJ (547 kcal), with the other formulae giving similar results. An attempt to devise a specific prediction equation for this patient population failed when an examination of other patient variables that were significantly associated with measured values, including fat-free mass and a clinical score (Pugh *et al.* 1973), showed that only 61 % of the variation in resting energy expenditure could be explained. This finding suggests that the measurement of energy expenditure in this patient population should be considered routine at least until the means to estimate energy requirements more accurately is available. The development of easy-to-use metabolic carts and, more recently a portable hand-held version, make these measurements a realistic proposition (Med Gem; Viasys Healthcare Ltd, Warwick, Warks., UK).

Nutrient restriction v. nutrition support

Whilst the absence of absolute intake guidelines in many disease situations is recognised, there has been an increased

awareness that dietary restrictions that are often based on good scientific observations may, in addition to their therapeutic role, contribute to an impaired nutrient intake. Although few studies have been undertaken, this effect has been demonstrated in patients with liver disease in relation to advising dietary Na restriction in the treatment of fluid retention (Soulsby *et al.* 1997). Soulsby *et al.* (1997) carried out an investigation on six cirrhotic patients with mild to moderate ascites who were randomised to either a diet containing 40 mmol Na or a no-added-salt regimen for a 4-week period and then treatments were crossed over. Intakes of energy and protein, assessed by 7 d weighed dietary records, were lower on the more restrictive diet and were associated with a reduction in mid-arm muscle circumference. Whilst the number of patients was small, these findings demonstrate a relationship between the extent of therapeutic restriction and nutrient intake that merits further investigation. By contrast, Wicks *et al.* (1995) observed no difference between the mean adequacy of energy intake in five patients with primary biliary cirrhosis following a low-Na diet compared with that of patients whose diet was not restricted. However, again the numbers were very small, and the patients were not controlled for their demographic or clinical profiles. The results from a larger study of thirty-nine patients with hypertension (Korhonen *et al.* 2000) showed no substantial change in nutrient intake on a Na-restricted regimen; however, the diet was more liberal than that of the patients in the study of Soulsby *et al.* (1997) and the patients were free-living and relatively well. Undoubtedly, the compromising effects of specific therapeutic dietary restrictions on total nutrient intake can be ameliorated, at least to a certain extent, by intensive dietetic input and the provision of appropriate supplementation in well-motivated and adequately-supported patients. Indeed, this approach is fundamental to the work of most clinical dietitians. However, more evidence is required to quantify the negative effects of dietary restriction of Na and other nutrients in different patient groups so that an optimum balance between dietary restriction and nutrition support can be achieved. The continual development of new drugs and other medical treatments that can influence this balance suggest that the attainment of optimum balance should be an ongoing task.

Evaluation of nutritional status

The routine assessment of patients' nutritional status is essential in order to monitor any potential negative effects of dietary restriction. The past decade has seen the publication of a plethora of screening and assessment tools that can be used for this purpose (Green & McLaren, 1998; Arrowsmith, 1999; Elia *et al.* 2000). A recent systematic evaluation of published papers describing forty-four tools designed to assess a patient's nutritional status or to identify those at risk from malnutrition found that few papers included sufficient details about the tools' development or provided adequate statistical evidence that they had been appropriately validated (Jones, 2002). At present there are no conclusive published data to indicate whether any of these tools are useful in clinical practice.

While still not meeting the stringent statistical criteria recommended by Jones (2002), a recently developed global assessment tool for use in patients with liver disease has been shown to be repeatable, to have internal and external validity and to be predictive of clinical outcome (Madden *et al.* 1997; Madden, 1998). The tool categorises patients into one of three groups (adequately nourished, moderately malnourished or severely malnourished) on the basis of an anthropometric evaluation and a dietary and clinical history. From these data, approximate BMI derived from estimated dry body weight (Mendenhall, 1992; Wicks & Madden, 1994), mid-arm muscle circumference (Bishop *et al.* 1981) and the adequacy of energy intake relative to estimated requirements are then entered into a simple algorithm (Madden, 1998). The presence of other potentially-important factors, e.g. recent weight loss or severe symptoms like steatorrhoea, that are likely to impair nutritional status can be taken into account by making a subjective amendment to the final nutritional category. In an evaluation of 271 patients who had undergone liver transplants the category of nutritional status attributed by the assessment tool before surgery was found to be significantly associated with survival on the 90th post-operative day ($P < 0.005$). However, independently none of the individual variables contributing to the tool showed a significant association with survival, suggesting that their combination in the tool provides a useful instrument in the management of these patients (Madden, 1998). At present, few studies have been carried out to fully evaluate most nutritional assessment tools and this evaluation must be undertaken rigorously, especially in disease-specific tools, if they are to play a useful role in clinical nutrition.

Mode and timing of nutrition support

In many clinical situations the nutritional status of patients is unambiguous and no compromise is required between dietary restriction and nutrition support; the patient clearly needs feeding and the key question is then how. The mode of delivery of nutritional support has changed considerably over the last 20 years, with a reduction in the use of parenteral nutrition and the use of more enteric nutritional support provided through different routes (Waitzberg *et al.* 2000; Elia *et al.* 2001).

In addition to the introduction of clear guidelines on the route of feeding, the issue of when to feed has also been more clearly addressed (Lewis *et al.* 2001). Two decades ago the introduction of early post-operative enteral feeding before the restoration of gastrointestinal motility might have been considered inappropriate and potentially dangerous. Indeed, the restoration of bowel sounds had been used as a determinant of when enteral feeding should re-commence (Payne-James *et al.* 1987). The publication of a study by Kudsk *et al.* (1992) showed clearly that early post-operative enteral feeding could be undertaken safely and that it had clinical advantages over parenteral nutrition. Furthermore, these findings were supported by the conclusion of a meta-analysis of eight other studies (Moore *et al.* 1992). Interestingly, the benefits of enteral feeding of patients immediately after gastric surgery had been first reported approximately

70 years earlier in the same journal (Andresen, 1918). The reason why it took so long for these benefits to be examined more closely before being adopted as good practice can only be left to speculation. The answer may relate, at least initially, to the focus of nutrition at that time being directed towards newly discovered vitamins (Rosenfeld, 1997) and to the absence, until relatively recently, of the necessary technology, e.g. fine-bore double-lumen tubes and endoscopic procedures (Baskin, 1992). With technological advances came the introduction of parenteral feeding, which then dominated the attention of many experts on nutrition support, and a large proportion of their research efforts and funding, at the expense of enteral nutrition support (Dudrick *et al.* 1968). In the last decade this shortcoming has been redressed and early enteral feeding is now regarded as not only a means to provide necessary energy and nutrients but also a tool to increase immune function and increase resistance to complications (Bengmark, 2002).

Facilitating dietary advice

The provision of nutritional support to acutely-ill patients may require their consent or that of their representative, but in many clinical situations it also requires their full cooperation. This provision may be difficult if nutrition support with or without dietary modification is required over a long period of time. In public health nutrition the difficulties of helping individuals to change their eating habits has been recognised and it has been shown that behavioural approaches can be effective in promoting fruit and vegetable intake (Resnicow *et al.* 2001) and reducing dietary fat (Wen *et al.* 2002). However, until recently little attention has been given to this area in the clinical management of disease. Rapoport (1998) has described how dietitians can act as facilitators of change, using behaviour-change skills rather than simply providing patients with advice. No RCT have yet been undertaken to examine the potential benefits of this approach in clinical nutrition, although it has been used effectively in other areas of health care including depression (Fava *et al.* 2002), insomnia (Edinger *et al.* 2001) and chronic fatigue syndrome (Deale *et al.* 1997).

The future

The changes that have occurred in nutritional management over the last 20 years can be summarised as challenging established practice and using a scientific evidence-based approach to consider all aspects of nutritional care. This process requires an ongoing and continual re-evaluation of practice in the light of developments both within the field of nutrition and in other areas of medicine, both clinical and research. This approach provides a huge challenge to all practitioners and nutrition scientists and relies on the integration of the two areas so that good clinical studies can be based on sound nutritional science. Rather than passively watching perspectives change, the challenge is to change perspectives by engaging actively in this ongoing process of evaluation.

Acknowledgements

Thank you to the following who helped provide the data on membership of UK organisations with an interest in clinical nutrition and on scientific publications indexed as 'clinical nutrition' and 'clinical*assessment': Judith Beeston, Pauline Cotterill, Correen Finney, Nick Kariagiannis, George Knapp, Dr Jackie Landman and Dr David McCarthy.

References

- Aliprandi G, Bissolotti L, Turla D, Vallet M, Scarazzato M & Fredi M (2001) The use of REE determination in a clinical setting applied to respiratory disease. *Acta Diabetologica* **38**, 27–30.
- Al-Omran M, Groof A & Wilke D (2003) Enteral versus parenteral nutrition for acute pancreatitis (Cochrane Review). In *The Cochrane Library Issue 1*. Oxford: Update Software.
- Andresen AFR (1918) Immediate jejunal feeding after gastroenterostomy. *Annals of Surgery* **67**, 565–566.
- Arrowsmith H (1999) A critical evaluation of the use of nutrition screening tools by nurses. *British Journal of Nursing* **8**, 1483–1490.
- Arroyo V, Ginès P, Jiménez W & Rodés J (1991) Ascites, renal failure, and electrolyte disorders in cirrhosis. Pathogenesis, diagnosis and treatment. In *Oxford Textbook of Clinical Hepatology*, vol. 1, pp. 429–470 [N McIntyre, JP Benhamou, J Bircher, M Rizzetto and J Rodés, editors]. Oxford: Oxford University Press.
- Bainton D, Davies GT, Evans KT & Gravelle IH (1976) Gallbladder disease. Prevalence in a South Wales industrial town. *New England Journal of Medicine* **295**, 1147–1149.
- Baskin WN (1992) Advances in enteral nutrition techniques. *American Journal of Gastroenterology* **87**, 1547–1553.
- Batterham MJ, Morgan-Jones J, Greenop P, Garsia R, Gold J & Caterson I (2003) Calculating energy requirements for men with HIV/AIDS in the era of highly active antiretroviral therapy. *European Journal of Clinical Nutrition* **57**, 209–217.
- Bengmark S (2002) Enteral nutrition in HPB surgery: Past and future. *Journal of Hepatobiliary and Pancreatic Surgery* **9**, 448–458.
- Bishop CW, Bowen PE & Ritchey SJ (1981) Norms for nutritional assessment of American adults by upper arm anthropometry. *American Journal of Clinical Nutrition* **34**, 2530–2539.
- Caregaro L, Alberino F, Amodio P, Merkel C, Bolognesi M, Angeli P & Gatta A (1996) Malnutrition in alcoholic and virus-related cirrhosis. *American Journal of Clinical Nutrition* **63**, 602–609.
- Cochrane Library (2003) <http://www.update-software.com/Cochrane/>
- Cunningham JJ (1980) A reanalysis of the factors influencing basal metabolic rate in normal adults. *American Journal of Clinical Nutrition* **33**, 2372–2374.
- Daly JM, Heymsfield SB, Head CA, Harvey LP, Nixon DW, Katzef H & Grossman GD (1985) Human energy-requirements: Overestimation by widely used prediction equation. *American Journal of Clinical Nutrition* **42**, 1170–1174.
- Deale A, Chalder T, Marks I & Wessely S (1997) Cognitive behavior therapy for chronic fatigue syndrome: A randomized controlled trial. *American Journal of Psychiatry* **154**, 408–414.
- Department of Health (1991) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom. Report on Health and Social Subjects* no. 41. London: H.M. Stationery Office.
- Diehl AK, Sugarek NJ & Todd KH (1990) Clinical evaluation of gallstone disease: Usefulness of symptoms and signs in diagnosis. *American Journal of Medicine* **89**, 29–33.

- Dixon N (1996) *Good Practice in Clinical Audit. A Summary of Selected Literature to Support Criteria for Clinical Audit*. London: National Centre for Clinical Audit.
- Dudrick SJ, Wilmore DW, Vars HM & Rhoads JE (1968) Long-term total parenteral nutrition with growth, development and positive nitrogen balance. *Surgery* **64**, 134–142.
- Edinger JD, Wohlgemuth WK, Radtke RA, Marsh GR & Quillian RE (2001) Cognitive behavioural therapy for treatment of chronic primary insomnia: A randomized controlled trial. *Journal of the American Medical Association* **285**, 1856–1864.
- Elia M (1990) Artificial nutrition support. *Medicine International* **82**, 3392–3396.
- Elia M, Baxter JP, Jackson A, Mason P, Rollins H, Sandars J, Thomas A & Ward J (2000) *Guidelines for Detection and Management of Malnutrition. A Report by the Malnutrition Advisory Group*. Maidenhead, Berks.: BAPEN.
- Elia M, Stratton RJ, Holden C, Meadows N, Micklewright A, Russell C, Scott D, Thomas A, Shaffer J, Wheatley C & Woods S (2001) Home artificial nutritional support: The value of the British Artificial Nutrition Survey. *Clinical Nutrition* **20**, Suppl. 1, 61–66.
- Eriksson LS & Conn HO (1989) Branched-chain amino acids in the management of hepatic encephalopathy: An analysis of variants. *Hepatology* **10**, 228–246.
- Evans D (2003) Hierarchy of evidence: A framework for ranking evidence evaluating healthcare interventions. *Journal of Clinical Nursing* **12**, 77–84.
- Fabbri A, Magrini N, Bianchi G, Zoli M & Marchesini G (1996) Overview of randomised clinical trials of oral branched-chain amino acid treatment in chronic hepatic encephalopathy. *Journal of Parenteral and Enteral Nutrition* **20**, 159–164.
- Fava GA, Ruini C, Rafanelli C & Grandi S (2002) Cognitive behavior approach to loss of clinical effect during long-term antidepressant treatment: A pilot study. *American Journal of Psychiatry* **159**, 2094–2095.
- Fernandez-Bañares F, Cabre E, Esteve-Comas M & Gassull MA (1995) How effective is enteral nutrition in inducing clinical remission in active Crohn's disease? A meta-analysis of the randomized clinical trials. *Journal of Parenteral and Enteral Nutrition* **19**, 356–364.
- Frankenfield DC, Muth ER & Rowe WA (1998) The Harris-Benedict studies of basal metabolism: History and limitations. *Journal of the Dietetic Association* **98**, 439–445.
- Fuller NJ, Jebb SA, Laskey MA, Coward WA & Elia M (1992) Four-component model for the assessment of body composition in humans: Comparison with alternative methods and evaluation of the density and hydration of fat-free mass. *Clinical Science* **82**, 687–693.
- Glambek I, Arnesjo B & Soreide O (1989) Correlation between gallstones and abdominal symptoms in a random population. *Scandinavian Journal of Gastroenterology* **23**, 277–281.
- Green SM & McLaren SG (1998) Nutritional assessment and screening: Instrument selection. *British Journal of Community Nursing* **3**, 233–242.
- Greenhalgh T (2001) *How to Read a Paper*, 2nd ed. London: BMJ Books.
- Griffiths AM, Ohlsson A, Sherman PM & Sutherland LR (1995) Meta-analysis of enteral nutrition as a primary treatment of active Crohn's disease. *Gastroenterology* **108**, 1056–1067.
- Guyatt GH, Sackett DL, Sinclair JC, Hayward R, Cook DJ & Cook RJ (1995) Users' guide to the medical literature IX. A method for grading health care recommendations. *Journal of the American Medical Association* **274**, 1800–1804.
- Harris JA & Benedict TG (1919) *Biometric Studies of Basal Metabolism in Man*. Carnegie Institute of Washington Publication no. 279. Washington, DC: Carnegie Institute of Washington.
- Hébuterne X, Hastier P, Peroux JL, Zeboudj N, Delmont JP & Rampal P (1996) Resting energy expenditure in patients with alcoholic chronic pancreatitis. *Digestive Diseases and Sciences* **41**, 533–539.
- Hopman WPM, Jansen JBMJ & Lamers CBHW (1985) Comparative study of the effects of equal amounts of fat, protein and starch on plasma cholecystokinin in man. *Scandinavian Journal of Gastroenterology* **20**, 843–847.
- Hopman WPM, Jansen JBMJ, Rosenbausch G & Lamers CBHW (1987) Cephalic stimulation of gallbladder contraction in humans: Role of cholecystokinin and the cholinergic system. *Digestion* **38**, 197–203.
- Italian Multicentre Cooperative Project on Nutrition in Liver Cirrhosis (1994) Nutritional status in cirrhosis. *Journal of Hepatology* **21**, 317–325.
- Jones JM (2002) The methodology of nutritional screening and assessment tools. *Journal of Human Nutrition and Dietetics* **15**, 59–71.
- Jorgensen T (1989) Abdominal symptoms and gallstone disease: An epidemiological investigation. *Hepatology* **9**, 856–860.
- Koch JP & Donaldson RM (1964) A survey of food intolerances in hospitalised patients. *New England Journal of Medicine* **271**, 657–660.
- Kondrup J, Nielsen K & Juul A (1997) Effect of long-term refeeding on protein metabolism in patients with cirrhosis of the liver. *British Journal of Nutrition* **77**, 197–212.
- Korhonen MH, Jarvinen RMK, Sarkkinen ES & Uusitupa MIJ (2000) Effects of a salt-restricted diet on the intake of other nutrients. *American Journal of Clinical Nutrition* **72**, 414–420.
- Kudsk KA, Croce MA, Fabian TC, Minard G, Tolley EA, Poret HA, Kuhl MR & Brown RO (1992) Enteral versus parenteral feeding: Effects on septic morbidity after blunt and penetrating abdominal trauma. *Annals of Surgery* **215**, 503–515.
- Latchford G (2002) *Computers at Work Survey*. Birmingham: NHS Information Authority.
- Lewis SJ, Egger M, Sylvester PA & Thomas S (2001) Early enteral feeding versus 'nil by mouth' after gastrointestinal surgery: Systematic review and meta-analysis of controlled trials. *British Medical Journal* **323**, 773–776.
- Loguerico C, Sava E, Siculo P, Castellano L & Narciso O (1996) Nutritional status and survival of patients with liver cirrhosis: Anthropometric evaluation. *Minerva Gastroenterologica e Dietologica* **42**, 57–60.
- Madden A (1992) The role of low fat diets in the management of gall-bladder disease. *Journal of Human Nutrition and Dietetics* **5**, 267–273.
- Madden AM (1998) Nutritional status and body composition in patients with chronic liver disease. PhD Thesis, University of London.
- Madden AM, Fuller NJ, Jennings G, Elia M & Morgan MY (1998) Assessment of fat-free mass in patients with cirrhosis. *Hepatology* **28**, Suppl., 610A.
- Madden AM & Morgan MY (1999) Resting energy expenditure should be measured in patients with cirrhosis, not predicted. *Hepatology* **30**, 655–664.
- Madden AM, Soulsby CT & Morgan MY (1997) Assessment of nutrition in patients with cirrhosis. *Journal of Hepatology* **26**, Suppl. 1, 125.
- Mendenhall CL (1992) Protein-calorie malnutrition in alcoholic liver disease. In *Nutrition and Alcohol*, pp. 363–384 [RR Watson and B Watzl, editors]. Boca Raton, FL: CRC Press.
- Messori A, Trallori G, D'Albasio G, Milla M, Vannozzi G & Pacini F (1996) Defined-formula diets versus steroids in the treatment of active Crohn's disease: A meta-analysis. *Scandinavian Journal of Gastroenterology* **31**, 267–272.

- Middleton SJ, Rucker JT, Kirby GA, Riordan AM & Hunter JO (1995) Long-chain triglycerides reduce the efficacy of enteral feed in patients with active Crohns-disease. *Clinical Nutrition* **14**, 229–236.
- Mifflin MD, StJeor ST, Hill LA, Scott BJ, Daugherty SA & Koh YO (1990) A new predictive equation for resting energy expenditure in healthy individuals. *American Journal of Clinical Nutrition* **51**, 241–247.
- Moore FA, Feliciano DF, Andrassy JR, McArdle AH, McBooth FV, Morgenstein-Wagner TB, Kellum JM, Welling RE & Moore EE (1992) Early enteral feeding, compared with parenteral, reduces postoperative septic complications: the results of a meta-analysis. *Annals of Surgery* **216**, 172–183.
- Müller MJ, Böttcher J & Selberg O (1993) Energy expenditure and substrate metabolism in liver cirrhosis. *International Journal of Obesity* **17**, Suppl. 1, S102–S106.
- Mulrow CD & Lohr KN (2001) Proof and policy from medical research evidence. *Journal of Health Politics, Policy and Law* **26**, 249–266.
- Naylor CD, O'Rourke K, Detsky AS & Baker JP (1989) Parenteral nutrition with branched-chain amino acids in hepatic encephalopathy. A meta-analysis. *Gastroenterology* **97**, 1033–1042.
- Niec AM, Frankum B & Talley NJ (1998) Are adverse food reactions linked to irritable bowel syndrome? *American Journal of Gastroenterology* **93**, 2184–2190.
- Nielsen K, Kondrup J, Martinsen L, Dossing H, Larsson B, Stilling B & Jensen MG (1995) Long-term oral refeeding of patients with cirrhosis. *British Journal of Nutrition* **74**, 557–567.
- Nielsen K, Kondrup J, Martinsen L, Stilling B & Wikman B (1993) Nutritional assessment and adequacy of dietary intake in hospitalised patients with cirrhosis. *British Journal of Nutrition* **69**, 665–679.
- Nutrition Task Force (1994) *Core Curriculum for Nutrition in the Education of Health Professionals*. London: Department of Health.
- Owen OE, Holup JL, D'Alessio DA, Craig ES, Polansky M, Smalley KJ, Kavle EC, Bushman MC, Owen LR, Mozzoli MA, Kendrick ZV & Boden GH (1987) A reappraisal of the caloric requirements of men. *American Journal of Clinical Nutrition* **46**, 875–885.
- Owen OE, Kavle E, Owen RS, Polansky M, Caprio S, Mozzoli MA, Kendrick ZV, Bushman MC & Boden G (1986) A reappraisal of caloric requirements in healthy women. *American Journal of Clinical Nutrition* **44**, 1–19.
- Payne-James JJ, Rees RG & Silk DBA (1987) Bowel sounds. *Anaesthesia* **42**, 893–894.
- Phillips GB, Schwarz R, Gabuzda GJ Jr & Davidson CS (1952) The syndrome of impending hepatic coma in patients with cirrhosis of the liver given certain nitrogenous substances. *New England Journal of Medicine* **247**, 239–246.
- Plauth M, Merli M, Kondrup J, Weimann A, Ferenci P & Müller MJ (1997) ESPEN guidelines for nutrition in liver disease and transplantation. *Clinical Nutrition* **16**, 43–55.
- Price WH (1963) Gallbladder dyspepsia. *British Medical Journal* **2**, 138–141.
- Pugh RNH, Murray-Lyon IM, Dawson JL, Pietroni MC & Williams R (1973) Transection of the oesophagus for bleeding oesophageal varices. *British Journal of Surgery* **60**, 646–649.
- Rapoport L (1998) Integrating cognitive behaviour therapy into dietetic practice: A challenge for dietitians. *Journal of Human Nutrition and Dietetics* **11**, 227–237.
- Resnicow K, Jackson A, Wang T, De AK, McCarty F, Dudley WN & Baranowski T (2001) A motivational interviewing intervention to increase fruit and vegetable intake through Black churches: Results of the Eat for Life trial. *American Journal of Public Health* **91**, 1686–1693.
- Rome Group for the Epidemiology and Prevention of Cholelithiasis (1984) Prevalence of gallstone disease in an Italian adult female population. *American Journal of Epidemiology* **119**, 796–805.
- Rosenfeld L (1997) Vitamine-vitamin. The early years of discovery. *Clinical Chemistry* **43**, 680–685.
- Sackett DL, Rosenberg WMC, Gray JAM, Haynes RB & Richardson WS (1996) Evidence based medicine: What it is and what it isn't. *British Medical Journal* **312**, 71–72.
- Sampliner RE, Bennett PH, Comess LJ, Rose FA & Burch TA (1970) Gallbladder disease in Pima Indians. *New England Journal of Medicine* **283**, 1358–1364.
- Schofield WN (1985) Predicting basal metabolic rate, new standards and review of previous work. *Human Nutrition: Clinical Nutrition* **39C**, Suppl. 1, 5–41.
- Scottish Intercollegiate Guideline Network (2000) *Grading System for Recommendations in Evidence-Based Clinical Guidelines*. Edinburgh: Scottish Intercollegiate Guideline Network.
- Shekelle PG, Woolf SH, Eccles M & Grimshaw J (1999) Clinical guidelines: Developing guidelines. *British Medical Journal* **318**, 593–596.
- Sherlock S, Summerskill WHJ & Dawson AM (1956) Treatment and prognosis of hepatic coma. *Lancet* **ii**, 689–694.
- Sherlock S, Summerskill WHJ, White LP & Phear EA (1954) Portal-systemic encephalopathy. Neurological complications of liver disease. *Lancet* **ii**, 455–457.
- Soulsby CT, Madden AM & Morgan MY (1997) The effect of dietary sodium restriction on energy and protein intake in patients with cirrhosis. *Hepatology* **26**, 383A.
- Soulsby CT & Morgan MY (1999) Dietary management of hepatic encephalopathy in cirrhotic patients: Survey of current practice in United Kingdom. *British Medical Journal* **318**, 1391.
- Taggart D & Billington BP (1966) Fatty foods and dyspepsia. *Lancet* **ii**, 464–466.
- Uribe M & Conn HO (1994) Dietary management of portal-systemic encephalopathy. In *Hepatic Encephalopathy: Syndromes and Therapies*, pp. 331–349 [HO Conn and J J Bircher, editors]. Bloomington, CA: Medi-Ed Press.
- Waitzberg DL, Plopper C & Terra RM (2000) Access routes for nutritional therapy. *World Journal of Surgery* **24**, 1468–1476.
- Wen DB, Ehret C, Pedersen M, Snetselaar L, Johnson M, Tinker L, Hollinger D, Ilona L, Bland K, Sivertsen D, Ocke, Staats L & Beedoe JW (2002) Results of an adjunct dietary intervention program in the Women's Health Initiative. *Journal of the American Dietetic Association* **102**, 1631–1637.
- Wicks C, Bray GP & Williams R (1995) Nutritional assessment in primary biliary cirrhosis: The effect of disease severity. *Clinical Nutrition* **14**, 29–34.
- Wicks C & Madden A (1994) *A Practical Guide to Nutrition in Liver Disease*, 2nd ed., p.5. Birmingham: Liver Interest Group of the British Dietetic Association.
- Zachos M, Tondeur M & Griffiths AM (2003) Enteral nutritional therapy for induction of remission in Crohn's disease (Cochrane Review). In *The Cochrane Library Issue 1*. Oxford: Update Software.