The assessment of body composition in clinical conditions

By A. J. RICH, *Department of Surgery, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP*

The clinician is frequently urged to take account of the state of nutrition of his patients. This being the case, the questions he or she will ask are 'Is the patient undernourished?', 'Is the undernutrition a hazard?', 'How much nutritional support is required?', 'Is the support effective?'. Answers to these questions will usually be provided on the basis of a nutritional assessment and so play an important part in clinical decisions regarding treatment and prognosis. Most of the methods for nutritional assessment have been used extensively and successfully as public health indices of nutrition for a number of years. The newer methods of direct measurement of body composition are limited at present to experimental studies. This review will examine the practicality of such methods in aiding the clinician to answer the questions posed above.

Normal nutrition assumes adequacy of the body's complement of a multitude of nutrients and micronutrients. However, protein and fat are generally regarded as being the most important because they represent sources of endogenous energy during starvation (Cahill, 1970). The absolute amounts of fat and protein are assumed to give some indication of an individual's capacity to withstand a period of inadequate nutrient intake and hence of his over-all nutritional status. In order to assess this capacity it is usual to regard the body as consisting of several major compartments; usually fat, protein and water (Moore *et al.* 1963). However, the body does not lend itself easily to measurement of such compartments. Although much of the body fat is found subcutaneously, the splanchnic organs contain appreciable quantities (Forbes *et al.* 1953) which are not easily measured or may not be utilized during starvation (Garrow *et al.* 1965). Protein too is distributed widely throughout the different tissues of the body (Table 1). Furthermore, although the relationship between the different components within each compartment may be clearly defined during health there is good evidence that such relationships may become altered during depletion and disease (Moore & Brennan,

Table 1. *The protein content of body tissues*

(calculated from Forbes *et al.* 1953)

	g/kg
Muscle	22
Skeleton	20
Viscera and skin	18
Extracellular	17
Fat	6

1975). For example, Picou *et al.* (1966) demonstrated that during severe protein
—calorie malnutrition in children, much of the remaining body protein lay within
the extracellular compartment which had been relatively spared during the process
of general protein attrition.

Nevertheless, most of the commonly used techniques of nutritional assessment
are based, directly or indirectly, on a static assessment of body composition. The
methods and their relation to body composition are shown in Fig. 1.

Whole-body protein can be estimated from total body potassium or nitrogen
measurements (Goode & Hawkins, 1978; Hill *et al.* 1978). The skeletal muscle
component may be assessed from the arm muscle circumference (AMC) (Jelliffe,
1966) or from the urinary excretion of creatinine or 3–methylhistidine (3MH)
(Young *et al.* 1973; Bistrian, Blackburn, Sherman *et al.* 1975). Visceral protein,
that component of body protein contained within the soft organs of the body, is
indicated by plasma levels of albumin and transferrin (Blackburn *et al.* 1977).
Measurement of the cutaneous response to injected antigen and the peripheral
blood lymphocyte count are also used to indicate this component (Bistrian,
Blackburn, Scrimshaw *et al.* 1975). The size of the body fat compartment may be
derived from measurements of single or multiple skinfold thickness (Durnin &
Womersley, 1974) or obtained by subtraction of body protein mass estimates from
the body-weight. Body-weight itself is a crude index of body composition but
cannot be regarded as typical of one particular compartment.

Can these measurements be used to provide answers to the clinician's questions
in terms of an individual patient?

Is the patient undernourished?

The implication of the term undernutrition is of a subnormal state incompatible
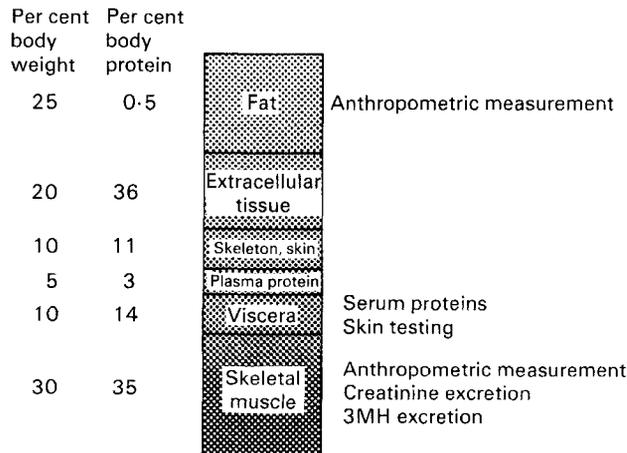with health. Measurements of indices of nutrition (such as body-weight) for a

Fig. 1. The major subdivisions of body composition and some of the methods used for their
measurement. The size of each division is proportional to its contribution to body-weight. The
relative proportion of total body protein contained is also indicated.

given healthy population can usually be represented by a frequency curve of normal or gaussian distribution. Somewhere at the two extremes lie values that could be regarded clinically as almost certainly indicative or over- or undernutrition. In accordance with a normal distribution, about 95% of the healthy population would lie within ±2 standard deviations (SD) above and below the mean and 99·7% would lie within the range ±3 SD. At some point at the appropriate end of the curve the probability that an individual with that particular value is in fact a normally nourished and healthy individual with a low measurement must become highly unlikely. Beyond this point it would seem reasonable to class any observation as being indicative of undernutrition. However, defining this point for any particular measurement of body composition may be difficult. One problem lies in obtaining a sufficiently large sample of the normally nourished population to draw up the curve. It is not sufficient to utilize information derived from alternative populations since these almost certainly differ from the individual under consideration, and may show an abnormal or skewed distribution.

Kelmsley *et al.* (1962) considered that the quartile (the portion of the normal curve containing the lower 25% of the population) below mean standard weight for height was a fair lower-limit for normal weight. For their population this represented 93–94% of the mean standard weight and they suggested that this was also a reasonable definition of underweight in the clinical sense.

Blackburn *et al.* (1977) suggested that norms for body-weight/height, arm muscle circumference and triceps skinfold thickness (TSF) could be defined as a fixed percentage of Jelliffe's standards (Jelliffe, 1966). A measurement between 60 and 90% of standard was said to indicate moderate depletion and below 60%, severe depletion. Other workers have applied this method of analysis to local standards.

Gray & Gray (1979) have criticized both the use of Jelliffe's standards and this type of analysis. These authors applied the criteria above to data from the Ten-State Nutrition Survey (Frisancho, 1974) and the Health and Nutrition Examination Survey (National Centre for Health Statistics, 1977) of healthy American adults and found that a considerable number of this population would be classified as depleted. This anomaly arose partly because Jelliffe's standards were substantially different from those of the US population and were not stratified for age, but also because the application of a fixed percentage of standard did not allow for differing coefficients of variation inherent in measurements of different aspects of nutrition.

Gray & Gray (1979) and Burgert & Anderson (1979) suggest that observations should be compared to percentiles of the appropriate standard rather than to percentages. This approach would allow for differing coefficients of variation between measurements but would still require adequate local standards. Moreover, it is by no means clear which percentile would be most appropriate as a division between normal and abnormal nutrition. The quartile below normal as suggested by Kelmsley *et al.* (1962) seems likely to exclude many individuals who would be regarded clinically as 'scrawney' but of normal nutrition and Bistrian (1980) has

suggested that suitable percentiles should be clearly associated with dysfunction.

An additional factor complicating the selection of a suitable percentile is the observation that loss of fat is functionally less important than loss of protein (Bistrian, 1980). This would suggest that different percentiles might be required to indicate undernutrition of different body compartments. Such differences are suggested from an examination of the results cited by Gray & Gray (1979) derived from a healthy American population. The 5th percentile for weight/height lay between 62 and 72% of standard; for AMC it lay between 82 and 84% of standard; and for TSF between 33 and 53% of standard. The considerable variations in the 5th percentile values in this survey and in the data reported by Frisancho (1974) suggest that wide variations in subcutaneous fat thicknesses occur in the normal population without apparent dysfunction but that considerably less variation occurs in the other measurements.

There is some, largely circumstantial, evidence to corroborate the use of the 5th percentile. Keys *et al.* (1950) found that a loss of active cell mass of about 20% in previously healthy subjects impaired physical fitness. Spurr *et al.* (1979) found evidence of reduced cardiac response to work in chronically undernourished subjects averaging a weight deficit of 28% compared to nutritionally normal controls. These figures are not too far removed from the 5th percentile figures for weight/height and AMC noted above.

It is less clear at what level TSF measurements indicate dysfunction. Indeed, Bistrian (1980) has indicated that this index is of limited clinical value in the diagnosis of undernutrition. However, as Frisancho (1974) has pointed out, the quantity of subcutaneous fat does indicate the size of the energy reserve. If reduced, this may imply a reduced ability to withstand a period of inadequate nutritional intake. For this reason, and to simplify the diagnosis of undernutrition by anthropometry, it may also be appropriate to use the arbitrary 5th percentile for TSF.

The laboratory markers of nutrition are usually considered to indicate depletion if they lie below the lower limit of normal for the laboratory concerned. Most laboratory 'normal' ranges are 2 SD above and below the laboratory mean. As discussed above, the use of a standard based upon the range of observations in a 'normal' population is a satisfactory method of delineating nutrition. However, most laboratories deal with samples from a population that is selected in that a majority of its members have been admitted to hospital for the diagnosis and treatment of illness. This may not reflect a healthy population and there are some results which suggest that nutritional measurements from hospital patients may be slightly lower than a control population of hospital employees (Bollet & Owens, 1973).

However, it is unlikely that these small differences would significantly alter biochemical identification of an undernourished individual. Far more important are the effects of factors other than nutrition on the laboratory measurements.

Plasma protein concentrations depend on the balance of synthesis on the one hand and utilization, catabolism, excretion, hydration and transvascular transfer

on the other. Anything that influences any of these, irrespective of its influence on nutrition, is bound to reflect in the plasma protein levels. Thus stress (Kinney, 1976), sepsis (Meakins, 1976) and cancer (Craig & Waterhouse, 1957) are known to influence plasma protein levels. One or more of these factors are found in most hospital patients and it seems unlikely that their complex interrelations can be defined or differentiated from each other in the individual patient.

Subnormal plasma protein levels are well documented in severe protein–calorie deficiency in the developing countries and correlate well with other measurements of nutrition (Young & Hill, 1978). They are useful in defining nutrition in epidemiological or experimental studies of groups. However, in experimental starvation mean plasma albumin levels do not drop for several weeks (James *et al.* 1976; Shetty *et al.* 1979) and may even rise in some individuals (Shenkin & Steele, 1978). Because of this slow response and the complicating factors indicated above, plasma albumin and transferrin lack specificity and are of little value in assessing an individual's visceral protein compartment.

The peripheral blood lymphocyte count and the response to an injected antigen (delayed cutaneous hypersensitivity, DCH) are also used to assess the visceral protein compartment and there is some evidence to support the view that the size of the lean body mass (LBM) is related to immunocompetence (Spanier *et al.* 1976). A lymphocyte count below $3000/mm^3$ is said to demonstrate an immune deficiency (Blackburn *et al.* 1977) as is a negative response to an injected antigen (Bistrian, Blackburn, Scrimshaw *et al.* 1975). Both these measurements can be materially influenced by factors other than nutrition (Millar, 1978) and the DCH response is additionally subject to observer error (Sokal, 1975). Without the ability to separate the effects of undernutrition and those of stress, sepsis and age it is difficult to justify the application of such tests as an index of immunocompetence or visceral protein in the individual.

The 24 h urinary excretion of creatinine or 3MH has been used to evaluate the muscle protein component of the LBM. Creatine is found almost entirely within muscle as creatine phosphate. It spontaneously and irreversibly dehydrates at a relatively constant rate to form creatinine which is excreted unchanged in the urine. Measurement of urinary creatinine in normal individuals is indicative of total muscle mass and is also closely related to LBM (Forbes & Bruining, 1976). Bistrian, Blackburn, Sherman *et al.* (1975) described the use of a creatinine–height index (CHI) to relate actual creatinine excretion to the expected excretion for an adult of the same sex and height but of standard weight. Excretion values were expressed as a percentage of standard and values between 60 and 80% were said to represent moderate depletion of somatic protein, below 60%, severe (Blackburn *et al.* 1977).

The difficulties inherent in using a fixed percentage of standard to describe undernutrition have been discussed above. In addition, the standards given are derived from a small sample of young individuals (Butterworth & Blackburn, 1975) and will certainly give rise to inaccuracies when applied to more elderly subjects. However, the most serious criticism of the method lies in the difficulty of ensuring

accurate urine collections which may result in fluctuations of individual creatinine excretion of up to 36% (Greenblatt *et al.* 1976). This inconsistent and unquantifiable error makes it unlikely that urinary creatinine excretion could be used to identify undernutrition even if the objections to standards could be overcome.

3MH is an amino acid found mainly in myofibrillar protein. When the protein is broken down 3MH is released but not recycled and is excreted in the urine. Urinary excretion of 3MH should thus be representative of myofibrillar protein and has been used as an index of muscle protein turnover (Williamson *et al.* 1977) and suggested as an index of the nutritional state (James, 1978). However, the same criticisms regarding adequacy of collection of urine may be made and there appear to be no adequate normal standards available for comparison. This further limits the usefulness of this test as an index of undernutrition.

Whole body potassium estimations of lean body mass are based on measurements of the gamma-ray emissions from the naturally occurring isotope of potassium, ^{40}K. This forms a constant proportion of total potassium which is contained mainly within body cells. However, even within the LBM the potassium content varies from tissue to tissue (Table 2) but for purposes of calculation an average value is assumed and the LBM expressed in kg. However, since differential losses of protein occur during the process of undernutrition (Moore *et al.* 1963) it can no longer be assumed that the same average value remains valid for the undernourished patient.

Hill and co-workers have measured whole body nitrogen as an index of total body protein (Hill *et al.* 1978). The subject is irradiated with a small dose of neutrons and this high energy bombardment creates an isotope of nitrogen (and other elements) which emits gamma-rays for a short time. This emission can be measured and gives an indication of the whole body content of nitrogen. Since this is a primary component of all protein the whole body content can be calculated by multiplying total body nitrogen by 6.25 (Hill *et al.* 1978). As up to one-third of body protein is extracellular (Moore *et al.* 1963) this method will tend to over-estimate the LBM which is generally regarded as containing the majority of the labile protein important in states of depletion.

Both of these methods seem capable of direct measurements of various aspects of body protein composition and in conjunction with other measurements (Forse &

Table 2. *The potassium content of body tissues*

(Calculated from Forbes & Lewis, 1956)

	mmol/g
Muscle	80
Viscera and skin	56
Extracellular	45
Skeleton	27
Fat	19

Shizgal, 1980) can be used to indicate fat or water content. However, they do not readily lend themselves to the indification of undernutrition in the individual because of a lack of suitable normal standards. Most groups using these methods have validated their technique on the local population but have a limited number of observations at the upper and lower extremes of normal nutrition (Goode & Hawkins, 1978). Once this objection can be overcome it should be easy to identify the undernourished individual with confidence.

From the foregoing it can be seen that it is difficult to give an adequate answer to the question, 'Is the patient undernourished?'. The chief reason for this is a lack of adequate standards and the biochemical methods appear to lack specificity. For clinical purposes the most relevant reply might be, 'Why do you want to know?'. Clinicians usually want to know so that they can weigh the risks of therapy against the hazards of being undernourished. This question is now discussed.

Is the undernutrition hazardous?

There is no doubt that the gross, long-standing malnutrition as seen in the developing countries results in an increased morbidity and mortality for the populations concerned. It is far less clear whether the relatively minor undernutrition described in hospital inpatients (Bistrian *et al.* 1974; Hill *et al.* 1977) has a significant effect on outcome (Hill, 1979). Indeed, within the hospital it is particularly difficult to separate the effects of poor nutrition from those of age, disease, sepsis and stress that often accompany it. Nevertheless, it is usually assumed that a combination of disease and nutritional depletion in hospitalized patients is likely to result in higher risks of morbidity and mortality. Is it possible to use the standard methods of assessment of body composition to predict the risk of undernutrition for an individual?

On the simplest level, body-weights for height of 10% below standard do not appear to be associated with other evidence of undernutrition (Bistrian *et al.* 1976) or with increased length of stay after surgery (Faintuch *et al.* 1979). Low body-weight was not associated with post-operative morbidity (Higgens *et al.* 1981) or mortality (Ryan & Taft, 1980). Abel *et al.* (1976) found slightly increased rates of morbidity and mortality in a small group of patients undergoing cardiac surgery who were 15% or more below ideal weight.

In terms of loss of weight, a loss of 10% or more in the previous 6 months does not seem to be associated with an increased risk of post-surgical complications (Klidjian *et al.* 1980). Weight loss as a single measurement lacks two important determinants; absolute weight (which would admit obesity) and time (which would admit rapid loss of weight). Mullen, Gertner *et al.* (1979) have used weight loss/unit time as an indicator of risk and report that significant loss occurs at rates exceeding 0·2% of the usual weight/d for a 7 d period.

Anthropometric methods indicating a single compartment might be expected to indicate individual risk with more precision. Klidjian *et al.* (1980) found that an AMC below 85% of standard was indicative of a high risk of post-operative

complications in their study of patients undergoing major surgery. However, the test was not particularly selective, only 39% of patients with this low value actually developing complications. Mullen, Gertner *et al.* (1979) found that AMC did not predict post-operative complications.

TSF is not usually regarded as an appropriate predictive index because of the lesser role of fat and the wide individual variations in the measurement (Michel *et al.* 1981). Forse & Shizgal (1980) found only a poor correlation between TSF and indices of protein nutrition. Mullen and co-workers reported TSF as being a significant variable (Mullen, Buzby *et al.* 1979), although in an earlier study they had failed to demonstrate its predictive value (Mullen, Gertner *et al.* 1979).

Plasma albumin levels below 3·0 g/l were found to be associated with a complication rate two-and-a-half times greater than that of patients with levels above this (Mullen, Gertner *et al.* 1979). However, 45% of patients with plasma albumins below this level did not develop complications. Klidjian *et al.* (1980) also found a strong association between low albumin levels and post-operative complications, but again with rather poor selectivity, 48% of their high-risk group recovering without complications. Ryan & Taft (1980) found that the plasma albumin level was unassociated with the complication rate following major abdominal surgery.

Mullen, Gertner *et al.* (1979) and Buzby *et al.* (1980) have also reported plasma transferrin as a sensitive index of the risk in undernourished patients facing surgery. Review of their results suggests that transferrin levels are also rather unselective, over half the patients with subnormal levels (220 mg/100 ml) avoiding complications. Ryan & Taft (1980) found that transferrin levels failed to predict morbidity and mortality in 389 patients undergoing abdominal surgery, over one-third of their study group having transferrin levels below this standard.

Lymphocyte counts below 1200/mm^3 are said to indicate relative anergy resulting from depletion of the visceral protein compartment (Blackburn *et al.* 1977). A retrospective survey of 105 patients found that those with a lymphocyte count below 1000/mm^3 developed significantly more post-operative sepsis than did those with counts above this level (Lewis & Klein, 1979). However, Mullen, Gertner *et al.* (1979) and Ryan & Taft (1980) found that this measurement had no prognostic significance.

DCH testing is difficult to evaluate because of the large individual variations in response. Mullen, Gertner *et al.* (1979) and Meakins *et al.* (1977) reported an increased risk of complications in patients with a poor or absent response to testing with one or more antigens. However, other large-scale studies report the contrary (Ryan & Taft, 1980; Brown *et al.* 1982). These opposing findings can probably be explained on the basis of population variables and on the various combinations of disease and therapy within each group. This diversity, however, suggests such tests have little prognostic value for the individual.

A large proportion of abnormal CHI measurements are reported in nutritional surveys (Blackburn *et al.* 1977). Mullen, Gertner *et al.* (1979) reported an abnormal CHI in 65% of their patients but demonstrated no predictive value

towards morbidity and mortality. No reports are available on the predictive value of urinary 3MH excretion.

For the direct measurements of body protein using whole body potassium or nitrogen no absolute figures indicating risk are available. Blackburn & Kaminski (1980) suggest that every protein molecule is vital and this would imply that any departure from normal was detrimental. Keys *et al.* (1950) reported that a loss of body protein of about 20% impaired physical fitness. Passmore (1965) suggested that 8·5 kg protein was the minimum required for life. Garrow (1982) has assessed a gradual loss of approximately one-third of body protein as survivable. Unfortunately there is no experimental or clinical data to support these theoretical figures, or to relate them to absolute quantities of LBM.

It is clear from the foregoing that although there is some evidence that abnormal body composition as assessed by these measurements may indicate an increased risk, it is still impossible to calculate what this risk is on an individual basis. A number of observers have attempted to overcome this problem using predictive indices based on multiple regression equations of a number of anthropometric and laboratory tests. Typical is that of Buzby *et al.* (1980) who devised a prognostic nutritional index (PNI) based on measurements of plasma albumin and transferrin, TSF and DCH. Values for an individual were inserted into the formula to give a figure for the percentage risk of that individual developing post-operative complications. The effectiveness of the PNI was tested by using it in a prospective study of 100 patients undergoing gastrointestinal surgery. The results are somewhat difficult to interpret because some of the patients were obese and many were given post-operative parenteral nutrition. Patients with pre-operative PNI values of over 50% were considered to be a high-risk group and 89% of all complications occurred in this group. However, less than half of the group actually developed complications so although the index displayed a reasonable degree of sensitivity it was not particularly selective. Such a test would, therefore, be of limited use in assessing the hazard of undernutrition in individual circumstances.

It is apparent that static measurements of body composition used singly or in combination provide little help to the clinician in deciding if an individual patient is at risk from his undernutrition. This is perhaps not unexpected if the risks themselves are considered. Morbidity and mortality in surgical patients usually derives from the failure of a vital body system rather than from exhaustion of energy or protein resources. Cardiopulmonary failure, for example, is a prime cause of post-operative morbidity and mortality (Anscombe, 1957) and renal and hepatic dysfunction may occur during the post-operative period. For the survival of an individual the function of these body systems is probably more important than the absolute amount of protein or energy they contain and the quality of the remaining protein is more important than the quantity. Clearcut functional deterioration in muscle and respiratory function in experimental undernutrition has been reported (Keys *et al.* 1950) so it would seem logical to attempt to relate such changes to morbidity and mortality in undernourished individuals. Klidjian *et al.* (1980) have reported a correlation between hand-grip strength and post-operative

complications, and Moran *et al.* (1980) have suggested that similar results may be obtained from tests of pulmonary musculature. It may be appropriate to develop similar functional tests for other systems documented as suffering deterioration during undernutrition and to attempt to validate them for prediction of risk in the individual.

However, it is perhaps indicative of the rather poor performance of static measurements of body composition in predicting a risk that a number of experienced surgical nutritionists place little reliance on such tests. Several workers (Hill, 1979; Grant *et al.* 1981; Jeejeebhoy, 1981; MacBurney & Wilmore, 1981) have suggested that for clinical purposes a perfectly adequate assessment of nutrition and its risks can be obtained from a good history and physical examination together with a knowledge of the diagnosis and proposed treatment.

How much nutritional support is required?

Energy and protein requirements are largely a function of the size of the LBM and its metabolic rate (Moore *et al.* 1963). It follows that estimates of the size of this compartment give an indication of basal requirements. Additional requirements for hypermetabolism depend on a number of factors including its cause and pre-existing nutritional depletion, and cannot be derived directly from compositional studies (Elwyn, 1980). Calculation of the actual amount of nutritional support required obviously depends on current nutrient intake which will require assessment by conventional methods.

The Harris–Benedict equations use height, weight, age and sex to calculate resting metabolic expenditure (RME) (Harris & Benedict, 1919). These variables are closely related to the size of the LBM (Moore *et al.* 1963) and the contemporary techniques of continuous expired-air analysis have confirmed that these equations provide realistic estimates (Long *et al.* 1979). Since these measurements are simple to make there seems little justification in using more elaborate methods to estimate LBM as an index of RME. Methods to calculate the RME from the AMC or from the fat-free mass using skinfold thickness do not appear to have been applied clinically, although urinary creatinine excretion has been considered in the experimental situation (Moore, 1980). As expected, the body cell mass as measured by whole body potassium is an accurate index of energy expenditure (Kinney *et al.* 1963) but obviously lacks the clinical flexibility of height and weight measurements. Although whole body nitrogen measurements have not been used to estimate energy requirements their relationship to protein rather than to the LBM might make them less suitable, especially in the depleted individual.

Clinical usage and responses suggest that simple measurement of height and weight and the use of the Harris–Benedict equations produce estimates of energy requirements that are perfectly satisfactory (Grant, 1980; Rich & Wright, 1980). More sophisticated methods are not required for most patients.

Is the nutritional support effective?

The aim of nutritional support is to replete the protein and energy compartments or prevent them from becoming further depleted. From a practical point of view this implies that measurements of the protein or fat compartment should show the appropriate change or at least remain static to indicate successful support.

It is rarely possible to fully replete patients in hospital. Restoration of the LBM without excess fat deposition can take several months, extending well into the period of convalescence (Moore *et al.* 1963). It is unlikely to occur at the low levels of physical activity normally associated with inpatient care and still less likely in ill, stressed and septic surgical patients. Thus the compartmental changes that occur in an individual as the result of nutritional support are likely to be relatively small over periods of 1 or 2 weeks.

As an example, an undernourished man weighing 55 kg might be expected to have an LBM of about 25 kg and a fat compartment of 12·5 kg. If it is assumed that he has achieved a generously large, positive daily balance of 10 g nitrogen and 4270 kJ (1000 kcals) and these excesses are wholly transported to their respective compartments, then the over-all increases in 1 week would amount to approximately 438 g protein and 779 g fat.

Are such changes detectable in an individual using the conventional methods of nutritional assessment? It is doubtful if anthropometric methods are sufficiently sensitive. Skinfold thicknesses are subject to observer error of $\pm 1·5$ mm. In terms of total body fat this represents as much as 1·9 kg (Durnin & Womersley, 1974), well outside the magnitude of change calculated above. Even if observer accuracy is assumed and three or four skinfold thicknesses are used the error is still large. Durnin & Womersley (1974) described a standard error for their method of $\pm 5\%$. This approximates to $\pm 2·7$ kg fat in individual terms, again well outside the changes that are being assessed.

There are no formulae to translate AMC directly into an index of somatic protein mass but it is usually assumed that a percentage change in AMC is equivalent to a change in total muscle protein. If it is assumed that the calculations in the example are reasonable, that new protein went equally to muscle mass and non-muscle lean mass (219 g each) and that arm muscle is 10% of the total muscle mass, then the expected increase in circumference can be calculated as approximately 10 mm. This is inside the probable error of AMC measurement. Even assuming that arm circumference can be measured with complete accuracy the error in measuring TSF detailed above can be calculated to give rise to an error in the calculation of the AMC of ± 290 mm.

Plasma albumin levels, indicating the visceral component of the LBM, are stated to increase in parallel with improvement in nutrition (Valerio *et al.* 1978). However, in studies where this finding is reported, additional treatment has been provided for the disease process and it is difficult to avoid the conclusion that the changes in plasma albumin levels are merely the response to successful treatment for the primary condition. In a study of fit young volunteers given only half their

daily requirements for a 6-month period, plasma albumin levels fell by only 10% when active tissue (equivalent to LBM) fell by 27% (Keys *et al.* 1950). These authors observed that plasma volume changes accounted entirely for the drop in plasma albumin levels and hence there was no significant change in the amount of circulating albumin. During the 12-week recovery period of this same group, plasma albumin levels increased by 8% while active tissue increased by 10%.

Plasma transferrin levels respond only slowly to severe protein restrictions in normal subjects (Shetty *et al.* 1979) and the response to refeeding is unpredictable (Ingenbleek *et al.* 1975). These experimental findings and the variable effects of stress, sepsis and hydration make it difficult to interpret changes in plasma levels on an individual basis.

Reservations have been expressed above about the validity of the CHI as a day-to-day indicator of muscle mass. Forse & Shizgal (1980) found that changes in body composition were not reflected in the CHI. Urinary excretion of 3MH appears to be a sensitive index of the changes in muscle protein breakdown that occur in the individual during starvation (Young *et al.* 1973) and sepsis (Long *et al.* 1977). Successful nutritional support should result in a reduction in muscle protein breakdown, apparent as a reduction in urinary 3MH excretion. However, this measurement does not appear to have been used for this purpose in the clinical setting, possibly because of collection difficulties, outlined above.

Depression of immune competence, and presumably depletion of the visceral protein component, is said to be reversed by nutritional repletion. The work of Meakins *et al.* (1977) is usually quoted in support of this statement. These authors used DCH as an index of immune function in 354 patients undergoing major surgery but their paper gives no details of patients' nutrition nor the feeding regimens that were used in a few subjects. It would appear that the changes in DCH response were equally likely to have been due to treatment of the primary disease process. Law *et al.* (1973) also reported improved responses in DCH following intravenous feeding but since their results appear to be derived from only six patients, these findings should be interpreted with caution. Changes in lymphocyte count following nutritional support do not appear to have been reported in man. Dionigi *et al.* (1977) found no lymphocyte response to refeeding in animals.

Direct measurements of body protein from whole body potassium or nitrogen appear to be attractive methods of assessing the response of the LBM to nutritional support because they avoid many of the assumptions of the other methods. However, errors in individual measurement can arise as a result of a number of factors including the habitus of the patient and the geometry of the whole body counter (Hawkins & Goode, 1976). Goode & Hawkins (1978) report an experimental error of 3.6% in the measurement of total body potassium, equivalent to about 190 g protein in a 55 kg man. Whole body nitrogen measurements have an error of $\pm 1.6\%$ of body-weight (Oxby *et al.* 1978). This amounts to approximately 187 g protein for the same subject. In both cases the probable range of measurement error is of the same order of magnitude as the

likely change in body protein calculated above so that it would seem probable that these methods are suitable for short-term assessment of nutritional support. Unfortunately, the techniques are not universally available because of the high capital costs involved but Hill has shown that they can be used even for sick patients (Hill *et al.* 1978).

It has been difficult to demonstrate that the standard methods of nutritional assessment based on body composition are sensitive enough to measure the small changes that take place over the period of 1 week. On the other hand, the observant clinician is likely to notice subtle changes in his patient over such a period. Positive signs of tissue anabolism are filling-out of facial contours, increased voluntary activity and the 'hair' sign; lassitude, weakness and muscle wasting indicate continuing catabolism. Such clinical observations usually precede significant changes in nutritional measurements by several weeks.

Conclusions

From this review it is apparent that the routine nutritional tests used to indicate body composition are of limited aid to the clinician wanting to make a decision about an individual patient he suspects of being undernourished.

If suitable local standards are available it should be possible to state with a reasonable degree of certainty that the patient is undernourished on the basis of anthropometric methods. Individual risk is more difficult to define. Static measurements of body composition are excellent for the study of population groups and research purposes but bear little relation to function. Since it is functional failure that is ultimately responsible for patient morbidity and mortality it is less important for the clinician to know how much is left rather than how well it works. It is suggested that functional tests of systems known to be adversely affected by moderate undernutrition would be a useful aid to the clinical assessment of individual nutritional risk.

Basal requirements for nutritional support can be easily calculated from measurement of height and weight and are reasonably specific. Calculation of additional requirements for stress cannot be made solely on the basis of body composition.

The efficacy of the nutritional support can best be assessed on clinical grounds. Changes in body composition can be detected with certainty only some time after they become clinically apparent.

In general, clinical judgement is a better aid to decision making in the undernourished patient than specialized measurements of body composition.

REFERENCES

Abel, R. M., Fischer, J. E., Buckley, M. J., Barnett, G. O. & Austen, W. G. (1976). *Ann. Surg.* **111**, 45.
Anscombe, A. R. (1957). *Pulmonary Complications of Abdominal Surgery*. London: Lloyd-Luke.
Bistrian, B. R. (1980). *Am. J. clin. Nutr.* **33**, 2211.
Bistrian, B. R., Blackburn, G. L., Hallowell, E. & Heddle, R. (1974). *J. Am. med. Ass.* **230**, 858.
Bistrian, B. R., Blackburn, G. L., Scrimshaw, N. S. & Platt, J. P. (1975). *Am. J. clin. Nutr.* **28**, 1148.

Bistrian, B. R., Blackburn, G. L., Sherman, M. & Scrimshaw, N. S. (1975). *Surg. Gynaec. Obstet.* **141,** 512.

Bistrian, B. R., Blackburn, G. L., Vitale, J., Cochran, D. & Naylor, J. (1976). *J. Am. med. Ass.* **235,** 1567.

Blackburn, G. L., Bistrian, B. R., Maini, B. S., Schlamm, H. T. & Smith, M. F. (1977). *J. parent. ent. Nutr.* **1,** 11.

Blackburn, G. L., & Kaminski, M. V. (1980). In *Practical Nutritional Support*, p. 166 [S. J. Karran and K. G. M. M. Alberti, editors]. Tunbridge Wells: Pitman Medical.

Bollet, A. J. & Owens, S. (1973). *Am. J. clin. Nutr.* **26,** 931.

Brown, R., Bancewicz, J., Hamid, J., Patel, N. J., Ward, C. A., Farrand, R. J., Pumphrey, R. S. H. & Irving, M. (1982). *Br. med. J.* **284,** 851.

Burgert, S. L. & Anderson, C. F. (1979). *Am. J. clin. Nutr.* **32,** 2136.

Butterworth, C. E. & Blackburn, G. L. (1975). *Nutrition Today.* **10,** 8.

Buzby, G. P., Mullen, J. L., Matthews, D. C., Hobbs, C. L. & Rosato, E. F. (1980). *Am. J. Surg.* **139,** 160.

Cahill, G. F. (1970). *New Engl. J. med.* **282,** 668.

Craig, A. B. & Waterhouse, C. (1957). *Cancer* **10,** 1106.

Dionigi, R., Zonta, A., Dominioni, L., Gens, F. & Ballabio, A. (1977). *Ann. Surg.* **185,** 467.

Durnin, J. V. G. A. & Womersley, J. (1974). *Br. J. Nutr.* **32,** 77.

Elwyn, D. H. (1980). *Crit. Care Med.* **8,** 9.

Faintuch, J., Faintuch, J. J., Machado, C. C. & Raia, A. A. (1979). *J. parent. ent. Nutr.* **3,** 369.

Forbes, G. B. & Bruining, G. J. (1976). *Am. J. clin. Nutr.* **29,** 1359.

Forbes, G. B., Cooper, A. R. & Mitchell, H. H. (1953). *J. Hum. Biol.* **203,** 359.

Forbes, G. B. & Lewis, A. M. (1956). *J. clin. Invest.* **35,** 596.

Forse, R. A. & Shizgal, H. M. (1980). *Surgery* **88,** 17.

Frisancho, A. R. (1974). *Am. J. clin. Nutr.* **27,** 1052.

Garrow, J. S. (1982). *Proc. Nutr. Soc.* **41,** 175.

Garrow, J. S., Fletcher, K. & Halliday, D. (1965). *J. clin. Invest.* **44,** 417.

Goode, A. W. & Hawkins, T. (1978). In *Advances in Parenteral Nutrition*, p. 557 [I. D. A. Johnston, editor]. Lancaster: MTP Press.

Grant, J. P. (1980). *Handbook of Total Parenteral Nutrition.* Philadelphia: W. B. Saunders.

Grant, J. P., Custer, P. B. & Thurlow, J. (1981). *Surg. clin. N. Am.* **61,** 437.

Gray, G. E. & Gray, L. K. (1979). *J. parent. ent. Nutr.* **3,** 366.

Greenblatt, D. J., Ransil, B. J., Harmatz, J. S., Smith, T. W., Duhme, D. W. & Koch-Weser, J. (1976). *J. clin. pharmac.* **16,** 321.

Harris, J. A. & Benedict, F. G. (1919). Publication no. 279. Washington DC: Carnegie Institute of Washington.

Hawkins, T. & Goode, A. W. (1976). *Phys. Med. Biol.* **21,** 293.

Higgens, C. S., Keighley, M. R. B. & Allen, R. N. (1981). *J. Roy. Soc. Med.* **74,** 571.

Hill, G. L. (1979). *Med. J. Aust.* **2,** 465.

Hill, G. L., McCarthy, I. D., Collins, J. P. & Smith, A. H. (1978). *Br. J. Surg.* **65,** 732.

Hill, G. L., Pickford, I., Young, G. A., Schorah, C. J., Blackett, R. L., Burkinshaw, L., Warren, J. V. & Morgan, D. B. (1977). *Lancet* i, 689.

Ingenbleek, Y., Van Den Shrieck, H. G., De Nayer, P. & De Visscher, M. (1975). *Clin. Chim. Acta* **63,** 61.

James, W. P. T. (1978). In *Advances in Parenteral Nutrition*, p. 521 [I. D. A. Johnston, editor]. Lancaster: MTP Press.

James, W. P. T., Sender, P. M. & Waterlow, J. C. (1976). In *Plasma Protein Turnover*, p. 251 [R. Bianchi, G. Bariani and A. S. McFarlane, editors]. London: Macmillan.

Jeejeebhoy, K. N. (1981). In *Nutrition and the Surgical Patient*, p. 118 [G. L. Hill, editor]. Edinburgh: Churchill Livingstone.

Jelliffe, D. B. (1966). *The Assessment of the Nutritional Status of the Community*, WHO monograph 53. Geneva: WHO.

Kelmsley, W. F. F., Billewicz, W. Z. & Thomson, A. M. (1962). *Br. J. prev. soc. Med.* **16,** 189.

Keys, A., Brozek, J., Henschel, A., Mickelsen, O. & Taylor, H. L. (1950). *Biology of Human Starvation* Minneapolis: University of Minnesota Press.

Kinney, J. M. (1976). In *Metabolism and the Response to Injury*, p. 237 [A. W. Wilkinson, editor]. Tunbridge Wells: Pitman Medical.

Kinney, J. M., Lister, J. & Moore, F. D. (1963). *Ann. N.Y. Acad. Sci.* **110**, 711.

Klidjian, A. M., Foster, K. J., Kammerling, R. M., Cooper, A. & Karran, S. J. (1980). *Br. Med. J.* **281**, 899.

Law, D. K., Dudrick, S. J. & Abdou, N. I. (1973). *Ann. Int. Med.* **79**, 545.

Lewis, R. T. & Klein, H. (1979). *J. surg. Res.* **26**, 365.

Long, C. L., Schaffel, N., Greiger, J. W., Schiller, W. R. & Blakemore, W. S. (1979). *J. parent. ent. Nutr.* **3**, 452.

Long, C. L., Schiller, W. R., Blakemore, W. S., Greiger, J. W., O'Dell, M. & Henderson, K. (1977). *Am. J. clin. Nutr.* **30**, 1349.

MacBurney, M. & Wilmore, D. W. (1981). *Surg. clin. N. Am.* **61**, 571.

Meakins, J. L. (1976). *Surg. clin. N. Am.* **56**, 847.

Meakins, J. L., Pietsch, J. B., Bubenick, O., Kelly, R., Rode, H., Gordon, J. & MacLean, L. D. (1977). *Ann. Surg.* **186**, 241.

Michel, L., Serrano, A. & Malt, R. A. (1981). *New Engl. J. Med.* **304**, 1147.

Miller, C. L. (1978). *J. parent. ent. Nutr.* **2**, 554.

Moore, F. D. (1980). *J. parent. ent. Nutr.* **4**, 228.

Moore, F. D. & Brennan, M. F. (1975). In *Manual of Surgical Nutrition*, p. 169 [W. F. Ballinger, J. A. Collins, W. R. Drucker, S. J. Dudrick and R. Zeppa, editors]. Philadelphia: W. B. Saunders.

Moore, F. D., Olesen, K. H., McMurray, J. D., Parker, H. V., Ball, M. R. & Boyden, C. M. (1963). *The Body Cell Mass and its Supporting Environment: Body Composition in Health and Disease*. Philadelphia: W. B. Saunders.

Moran, L. R., Thurlow, J., Custer, P. & Grant, J. P. (1980). *J. parent. ent. Nutr.* **4**, 595.

Mullen, J. L., Buzby, G. P. & Waldman, M. T. (1979). *Surg. Forum* **30**, 80.

Mullen, J. L., Gertner, M. H., Buzby, G. P., Goodhart, G. L. & Rosato, E. F. (1979). *Arch. Surg.* **114**, 121.

National Centre for Health Statistics. (1977). *Advance data* **14**, 1.

Oxby, C. B., McCarthy, I., Burkinshaw, I., Ellis, R. E. & Hill, G. L. (1978). *Int. J. appl. Radiat.* **29**, 205.

Passmore, R. (1965). In *Human Body Composition; Approaches and Applications*, p. 121 [J. Brozek, editor]. Oxford: Pergamon Press.

Picou, D., Halliday, D. & Garrow, J. S. (1966). *Clin. Sci.* **30**, 345.

Rich, A. J. & Wright, P. D. (1980). *Br. J. Surg.* **67**, 313.

Ryan, J. A. & Taft, D. A. (1980). *Surg. Forum* **31**, 96.

Shenkin, A. & Steele, L. W. (1978). *Proc. Nutr. Soc.* **37**, 95.

Shetty, P. S., Watrasiewicz, K. E., Jung, R. I. & James, W. P. T. (1979). *Lancet* ii, 230.

Sokal, J. E. (1975). *New Engl. J. Med.* **293**, 501.

Spanier, A. H., Meakins, J. L., MacLean, L. D. & Shizgal, H. M. (1976). *Surg. Forum* **27**, 332.

Spurr, G. B., Barac-Nieto, M. & Maksud, M. D. (1979). *Am. J. clin. Nutr.* **32**, 767.

Valerio, D., Overett, L., Malcolm, A. & Blackburn, G. L. (1978). *Surg. Forum* **29**, 145.

Williamson, D. H., Farrell, R., Kerr, A. & Smith, R. (1977). *Clin. Sci. mol. Med.* **52**, 527.

Young, G. A. & Hill, G. L. (1978). *Am. J. clin. Nutr.* **31**, 429.

Young, V. R., Havenberg, L. N., Bilmazes, C. & Munroe, H. N. (1973). *Metabolism* **22**, 1429.

Printed in Great Britain