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# Nutritional Assessment: Current Concepts and Guidelines for the Busy Physician

David S. Seres

**Malnutrition is associated with a great deal of morbidity. The prevention of malnourishment with early intervention is much more effective in improving outcome than is reacting once a patient has become ill and has obvious nutritional deficits. It is not difficult to become proficient at screening patients for nutritional risk as most of the information that is used in the nutritional assessment is already being gathered in the clinical setting. It is important to distinguish between the effects of improper nourishment and the effects of catabolic disease when assessing nutritional status. The basics of nutrition assessment for the practicing clinician and the pathophysiology of the different states of malnutrition are reviewed.**

## INTRODUCTION

The importance of appropriate nutritional assessment cannot be understated. It has even been said that altered nutritional markers can account for 50% of the variance in response to any given therapy. Despite the impact that the underlying nutritional state has on prognosis and outcome, however, we as physicians pay far too little attention and are poorly trained in assessing our patients and screening for *nutritional risk.* Compounding our inadequate education is a lack of agreement between different disciplines as to how to refer to nutritional markers and states of malnutrition, and the mistaken identification of markers of dysmetabolism as reflective of nutritional intake. It is the goal of this article to reintroduce some of the nutritional assessment terminology, concepts, and techniques, and to review the current clinical understanding of these conditions.

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David S. Seres, M.D., CNSP, Chair, Physician Certification, National Board of Nutrition Support Certification, Clinical Instructor, Albert Einstein College of Medicine, Assistant Attending Physician, Beth Israel Medical Center, New York, NY.

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Nutritional assessment serves several goals. The most important of these is to identify patients at nutritional risk early, particularly those with systemic disease, to prevent the development of a state of nutritional depletion or excess, both of which will adversely affect prognosis. The nutritional screening and evaluation process should identify those patients who may benefit from nutritional interventions, including those at higher risk for responding poorly to, or developing complications from, medical or surgical interventions. For example, a patient with cancer who is to undergo a major surgical procedure is far more likely to develop wound healing problems and/or infections if they have lost a significant amount of weight prior to surgery (1).

Nutritional screening and assessment are best performed by a multidisciplinary team. Each member of the medical team, the dietitian, the nurse, the pharmacist, the ancillary personnel, and the physician, participates in the processes and comes to the patient with a unique perspective and knowledge base. Physicians are often absent from the nutrition assessment process, for a number of reasons and in response to a multitude of seemingly more urgent pressures. But, as long as physicians are the final decision makers where patient care is concerned, we must be at least cognizant of the patient's nutritional state, as it will affect the entire course of the patient's illness and response to our therapeutic interventions. Further, the physician is the continuous link between the patient and the medical system and will be the one tracking the patient's course.

Whether or not the patient has access to all members of the "nutrition team" will depend on the setting in which the patient is being treated. The dietitian is most often the focal person in assessing patients in the hospital, but is too frequently excluded from the evaluation of patients with chronic disease in the outpatient or home environment. The nurse is often the main source of information on the patient's intake and social history in the hospital, long-term care, or home environment, but in the office setting is often busy with other tasks that preclude this kind of information gathering. The patient's relationship with the outpatient pharmacist is severely limited by the pressures of the high volume of business required in the current managed care environment. It falls, then, on the shoulders of the physician, or physician extender, to find time in the ongoing clinical assessment of patients to make at least subjective assessments of the nutritional status of the patient. The good news is that this assessment can be accomplished with very little time and by using skills we already possess.

## REVISITING NUTRITIONAL TERMINOLOGY AND CONCEPTS

### Malnutrition and Body Weight

*Malnutrition* is typically associated with under-nourishment, yet it also encompasses excess nourishment and is best defined as an imbalance between *energy* intake and utilization. Nutritional states are defined based on their effect on the health of the organism. Therefore, the term "nutritional risk" better serves to describe patients' states of malnutrition. For instance, medical obesity is defined not based on the cosmetic effect of excess weight, but on the effect of the excess weight on predicted longevity or the risk for developing co-morbidities. There are conventions by which these conditions are defined, based either on a one-time assessment, or based on changes in weight over time (2).

A person may be deemed malnourished based on a stable weight below normal, due to a loss of an arbitrary amount of weight, or due to a loss of a significant percentage of baseline weight. Commonly, the appropriateness of a given weight for an individual is determined relative to their height. Ideal weight is determined by insurance companies based on longevity and is available on published tables. The Body Mass Index (BMI) is determined as weight/height$^2$ (3) See Table 1; also go to: http://nhlbisupport.com/bmi/bmicalc.htm (4) for easy calculation.

A BMI of 20 to 25 is deemed normal. Most guidelines identify patients at nutritional risk if they are:

- <80% of ideal weight,
- Have a body mass index less than 20,
- Have lost 5% of baseline or 5 pounds in one month, or
- Have lost 10 pounds or 10% of usual body weight in 6 months.

When reporting under- or over-nourishment as a risk factor, it should be made clear in the assessment whether the determination was made based on a single

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**Table 1**
**Body Mass Index (BMI) (3)**

$$BMI = \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

OR

$$\text{weight (in pounds)} / \text{height (in inches}^2) \times 703$$

| *Classification* | *BMI* |
|---|---|
| Severe or morbid obesity | > 40 |
| Moderate obesity | 30–40 |
| Mild obesity | 27.5–30 |
| Obesity | >27.5 |
| Appropriate weight (19–34 yr) | 19–25 |
| Appropriate weight (>35 yr) | 21–27 |
| Mild malnutrition | 17–18.5 |
| Moderate malnutrition | 16–17 |
| Severe malnutrition | <16 |

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**Table 2**
**Evaluation of Recent Weight Change (5)**

$$\text{Recent weight change} = \frac{\text{UBW} - \text{current weight} \times 100}{\text{UBW}}$$

| *Time Period* | *Significant Loss* | *Severe Loss* |
|---|---|---|
| 1 week | 1-2% | >2% |
| 1 month | 5% | >5% |
| 3 months | 7.5% | > 7.5% |
| 6 months | 10% | > 10% |

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measurement or based on changes over time. The degree to which a given loss of weight has impact on nutritional risk is summarized in Table 2.

If weight loss is the determinant of malnourishment, we are usually concerned with *unintentional* loss (except in the case of patients with eating disorders). What is often confusing about these defining characteristics of malnutrition is that a patient who remains obese after losing a significant amount of weight may carry the same sort of risk profile as a chronically undernourished patient.

## Serum Proteins

In contrast to those with pure malnutrition, patients with systemic illness have an alteration in the metabolism of energy substrates (6). The term *dysmetabolism* may be more appropriate to describe the "nutritional" alterations seen in these patients. Hypoalbuminemia and reduced prealbumin and transferrin levels in the blood are due to the effect of circulating inflammatory modulators, often occur in the face of normal or at least adequate nourishment, and do not reflect the adequacy of intake. This concept is quite foreign to most healthcare practitioners, as we have all been taught to think that a reduction in serum albumin reflects a protein "deficiency." Furthermore, there are a number of illnesses, metabolic derangements, and therapies that may affect serum albumin (Table 3) (7).

The term *kwashiorkor* is often used to describe the state of dysmetabolism seen in our ill patients. The term actually reflects a specific syndrome that develops almost exclusively in children who suddenly develop hypoalbuminemia and ascites, are irritable, and have characteristic skin and hair changes. The occurrence of this condition is frequently associated with periods of endemic starvation, but the patients suffering from kwashiorkor do not have to be undernourished to develop the syndrome. Although the diet was characteristically low in protein and high in carbohydrates in the original descriptions of the syndrome, patients with kwashiorkor are not necessarily deficient in protein in their diet. In fact, kwashiorkor has been described in populations of breast-fed infants of adequately nourished mothers.

The clinical syndrome of kwashiorkor is best described as a state of dysmetabolism due to the misuse of protein by the body as an energy substrate instead of as a building block. The breakdown of albumin and other serum proteins to make acute-phase reactants, and the suppression of hepatic protein synthesis may also play roles. The mechanism for the development of

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**Table 3**
**Factors Affecting Serum Albumin Levels (7)**

*Increased*
- Dehydration
- Exogenous albumin (transient)

*Decreased*
- Increased intravascular volume
  - Overhydration
  - Eclampsia
- Inflammatory states
  - Infection
  - Catabolic stress
  - Trauma/post-operative states
  - Burns
  - Collagen vascular diseases
  - Cancer
- Hepatic failure
- Protein losing states
  - Nephrotic syndrome
  - Enteropathies
- Kwashiorkor
- Corticosteroid use

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hypoalbuminemia is likely the same for most patients with systemic illness as it is in patients with kwashiorkor, that is, due to inflammation and not due to inadequate protein intake. Because the syndrome of kwashiorkor is distinct, the term should not be applied broadly to hypoalbuminemic patients. "Hypoalbuminemia of catabolism" would better describe the hypoalbuminemic patients with systemic illness (8).

Whether we are discussing kwashiorkor or the hypoalbuminemia of catabolism, it has become quite clear that, while both are states of nutritional risk, neither of these states of malnutrition is due solely to malnourishment. Many studies and years of clinical experience have shown us that undernourished patients do not become hypoalbuminemic until, and unless, they become ill. Additionally, ill patients may become hypoalbuminemic without being malnourished. Take for instance the patient that we often see in the intensive care unit. They may be obese and otherwise without catabolic illness until the day of admission for an acute sepsis. The following day the albumin level will, as often as not, be significantly decreased. Even immediate nutrition support will not stop this drop in albumin, which will continue to worsen as long as this patient remains septic (9).

Other proteins have been proposed as markers of nourishment. These include prealbumin and transferrin. Both of these proteins are reverse acute-phase reactants. This means that serum concentrations of these proteins drop during states of inflammation, as does albumin. Prealbumin, in particular, has become a popular "marker" for assessing the nutritional state of our patients. Unlike albumin, which may take six weeks to normalize, prealbumin has a half-life of 48-72 hours and may normalize within a week once the inflammation has resolved. While normalization indicates an improvement in risk, it may occur whether intake is adequate or inadequate and does not, therefore, reflect nutritional adequacy. Levels of both prealbumin and transferrin are affected by numerous factors in addition to inflammation. Both are increased in advanced renal disease and by use of oral contraceptives. Transferrin is increased in iron deficiency and decreased in non-iron deficient anemias. Prealbumin levels are increased and transferrin levels decreased when corticosteroids are administered (10).

Catabolism, and not inadequate intake, is the real challenge to our patients and to our ability to provide appropriate nourishment. As suggested above, the nutritional result of catabolism is an alteration in energy metabolism. In the normal human, carbohydrate and fat are the preferred energy sources. In the stressed individual, inflammatory cascades are activated that wholly alter the use of substrates. Fat enters futile cycling, insulin resistance and other mechanisms shunt carbohydrate away from normal energy forming mechanisms, and protein becomes a preferred energy fuel. While we can assuredly provide patients with excess nourishment, either by enteral or parenteral routes, the substrates will not be put to their normal uses (6).

By identifying a patient as being at nutritional risk, we hope to intervene, or to refer patients to a nutrition professional, *before* their nutritional risk translates into systemic disease with the concurrent development of

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**Table 4**
**Nutritional Review of Systems (12,13)**

- Physical or functional Changes?
  - Clothing size/fit changes/belt notch change
  - Change in appearance/distribution of fat
  - Changes in activities/deterioration in activities of daily living
- Gastroenterological symptoms
  - Nausea, early satiety
  - Diarrhea
  - Constipation
  - Dry mouth
  - Lack of appetite
  - Difficulty swallowing
- Gustatory symptoms
  - Food aversions
  - Altered or lost taste/smell
- Dietary intake
  - Changes in preferences
  - Changes in portion size/usual amount consumed

**Table 5**
**Other Components of Patients' History that May Affect Nutritional Status (12–14)**

- Diagnoses/co-morbidities
- Altered mental state
- Delirium
- Dementia
- Catabolic illness
  - Cancer
  - HIV
  - Renal failure
  - Systemic infection
  - Inflammatory bowel disease, etc.
- Alcoholism
- Degenerative Neurological Disease
- Diabetes
- Socioeconomic factors
  - Independent vs. institutionalized or dependant on home aid
  - Income
  - Accessibility of a variety of foods (e.g. how close and of what quality are the foods at the grocery store?)

catabolism. All patients are at nutritional risk once catabolism and hypoproteinemia have developed, and close monitoring of intake and involvement of nutrition specialists should be considered (1).

The nutritional effects of catabolic illness include not only a decrement in serum proteins, but also a reduction in the lean mass. Catabolic patients may actually gain adipose weight while losing muscle. No amounts of protein, calories, or special nutrients have ever been shown to completely overcome these metabolic derangements. This is why nourishing an ill patient is so much more difficult and why preventing catabolic illness is so important from a nutritional perspective, particularly when one considers that lean mass is one of the most important determinants of survival in any catabolic illness (11).

## ASSESSING NUTRITIONAL RISK

The simplest and most effective method for monitoring nutritional status is following a patient's weight at every visit, or having the patients weigh themselves at home. While most doctors' offices weigh their patients regularly, weight fluctuations are often overlooked. Since weight may fluctuate by several pounds each day, using a graphic record so that trends can be easily identified make the weight a far more sensitive indicator.

Since a patient's weight is influenced by so many factors such as edema or the contents of the pockets of an anorectic, it is easy to dismiss weights as inaccurately reflective of the patient's nutritional status. Therefore, a second line of investigation is needed to improve sensitivity in the physician's office. Patients often report their food intake inaccurately, whether intentionally or not. A simple set of questions can be designed to approach the problem from a number of angles and may improve the sensitivity of the review of systems (Table 4) (12,13).

Enlisting the patient in the process will also make the physician's job easier. A quick explanation of the relationship between proper nourishment and recovery from disease will often motivate the patient to perform simple tasks such as observing themselves in a mirror for habitus changes or keeping simple diet records. During the physical examination, the clinician should

take notice of an increase in the redundancy of skin, consistent with weight loss, and the presence of rashes such as angular stomatitis, non-specific eczematous rashes, and more severe signs of deficiency. Zinc deficiency can occur in patients with chronic diarrhea and causes a characteristic rash, classically reported on the shins. Lanugo hair is usually a late sign of severe malnourishment. By the time the presence of muscle wasting is evident, the loss of muscle mass is so severe as to put the patient at very high nutritional risk. Therefore, a high degree of suspicion and the use of a few simple tools should help the physician to earlier identify patients at risk. See Table 5 for additional factors that may affect nutritional status.

More sophisticated testing is available for the interested practitioner. These involve attempting to assess and monitor the relative composition of the patients' body, either by direct measurement or by inference. A properly trained practitioner, especially when monitoring patients over time, may make reliable assessments, however, techniques of assessing anthropometrics do not yield, for the most part, useful quantitative information. Tracking the information obtained over time may reveal alterations in habitus sooner than they would be detected with simple observation. This is especially valuable in uncovering loss of lean mass when there has been no change in overall weight. These tests are easy to master and include measurements such as waist to hip ratio, mid-arm circumference, and the measurement of several different skin thicknesses using calipers. Then, by applying the values obtained to nomograms, estimates may be derived for lean and fat mass.

## CONCLUSION

Sensitivity to nutritional risk on the part of the physician or physician extender may lead to the identification of patients at risk for developing nutrition-related morbidity and mortality. Using tools already at the physician's disposal, patients can be identified who would benefit from nutritional intervention, or might need referral to a nutrition practitioner. Included in the nutritional assessment must be the understanding of the difference between malnourishment and dysmetabolism and their different effects on the patient. ■

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