

# Malnutrition in the ICU: Current recommendations for the assessment of nutritional status and a review of the use of albumin as an indicator of malnutrition

Kristen Fuhrmann Pharm D, Naree Panamonta MD, Shelley Roaten MS, RD, LD

## ABSTRACT

*Many critically ill patients have malnutrition at presentation or develop it during hospitalization, and this complication adversely affects outcomes, including length of stay, morbidity, and mortality. All ICU patients should be evaluated for malnutrition using simple screening tools, such as the Nutritional Risk Screening and Subjective Global Assessment. Laboratory tests, including serum albumin levels, are inaccurate indicators of malnutrition and do not provide a simple method for screening. In particular, albumin levels often fall rapidly because of transcapillary efflux and altered hepatic synthesis during acute illness. Current guidelines recommend that the nutritional status should be assessed by a review of recent energy intake, recent weight loss, and current body mass index and bedside assessment of muscle mass, fluid accumulation, and grip strength. An integrated analysis of nutritional status provides a better assessment and helps develop patient specific therapeutic interventions.*

## CASE

Patient X is a 55-year-old man, 72 inches tall, weighing 86 kg, status post motor vehicle accident with multisystem trauma. The patient is currently receiving sedation, pain control, and maintenance IV fluids; he received 3 liters of NS bolus, 2 units of FFP, and 2 units of PRBCs on admission. On admission, labs included CRP -2.1mg/dL, Alb -4.5 gm/dL and transthyretin (TTR, prealbumin) -17mg/dL. Labs on day three showed that the CRP was 37 mg/dL, Alb 1.2 gm/dL, and transthyretin 10 mg/dL. In an overall assessment of the patient, the clinician states that based on the most recent labs, enteral or parenteral nutrition needs to be started due to malnutrition as indicated by his low albumin.

**Corresponding author:** Kristen Fuhrmann, Pharm D  
**Contact Information:** Kristen.fuhrmann@umchealth-system.com  
**DOI:** 10.12746/swrccc2013.0104.038

## DISCUSSION

### MALNUTRITION IN THE ICU

Malnutrition is very common in critically ill patients, and its development is a function of the patients' preexisting nutritional status and severity of illness (degree of hypermetabolism). The characteristics of ICU patients have changed during the last decade; they now tend to be older and their medical disorders more complex with frequent comorbidity. These factors may contribute to malnutrition in the ICU. The combination of stress and undernutrition is associated with negative energy balances and the loss of lean body mass<sup>1</sup>. Critically ill patients often have a history of decreased food intake from anorexia, gastrointestinal symptoms, depression, anxiety, and other medical and surgical factors on presentation. Their food intake may have also been restricted for diagnostic or therapeutic procedures during hospital stay, and they may have nutrient loss from diarrhea, vomiting, polyuria, wounds, drainage tubes, and renal replacement therapy<sup>2,3</sup>. The major physiologic change

in critical illness is hypermetabolism. Decreased protein anabolic responses secondary to reduced physical activity and the use of neuromuscular blockade agents during mechanical ventilation and increased protein breakdown from infection, operative trauma, and commonly used drugs, such as corticosteroids, cause muscle wasting<sup>4</sup>. Large energy deficits can increase infectious complications, prolong mechanical ventilation, and increase ICU stay, morbidity, and mortality<sup>2, 5-7</sup>. Nutritional support can limit the loss of lean body mass during critical illness, and nutritional risk assessment is important to identify patients who may benefit from nutritional intervention in the ICU.

An appropriate nutritional history should review changes in weight or eating habits prior to hospitalization, comorbidities, functionality of the gastrointestinal tract, and ICU course. The physical examination should assess for temporal wasting, sarcopenia, signs of micronutrient deficiencies, fluid status, and the presence of non-healing wounds or drains as potential losses of nitrogen. Biochemical data, including the measurement of electrolytes and visceral proteins (e.g., albumin, prealbumin, transferrin, and retinol binding protein), are useful markers of inflammation and disease status but do not directly reflect the nutritional status of and provide little information about the nutritional status of critically ill patients<sup>8</sup>. Other measures to evaluate nutritional status, such as bioelectrical impedance, muscle function studies, creatinine-height index, anthropometric measures, and body composition studies, are cumbersome, impractical, and usually unavailable<sup>9</sup>. Scoring systems are helpful in determining overall disease severity and stratifying patient risk. The best screening tools are the Nutritional Risk Screening (NRS-2002) and the Subjective Global Assessment (SGA) (Table). The NRS includes four questions regarding body mass index, recent weight loss, dietary intake, and illness severity. The SGA is an alternative tool, incorporating the physical exam, comorbidities, weight, dietary history, and functional capacity, and has proven to be useful and reproducible in mechanically ventilated patients<sup>9</sup>.

**TABLE NUTRITION RISK SCREENING (NRS 2002)**

STEP 1: INITIAL SCREENING		YES	NO
1	Is BMI <20.5?		
2	Has the patient lost weight within the last 3 months?		
3	Has the patient had a reduced dietary intake in the last week.?		
4	Is the patient severely ill? (e.g., in intensive care)		

**Yes:** If the answer is “Yes” to any question, then additional screening is performed.

**No:** If the answer is “No” to all questions, the patient is rescreened at weekly intervals.

Adapted and abridged from Detsky. JPEN 1987; 11: 8-14

**BIOCHEMICAL MARKERS FOR MALNUTRITION**

There is a common misconception that serum albumin is an appropriate index of nutritional status and is often used as the sole marker for malnutrition by clinicians<sup>10, 11</sup>. Our introductory case highlights the need to shift from using albumin and other serum proteins as indicators of nutritional status to indicators of illness and to utilize more appropriate indices in assessing nutritional status in critically ill patients. However, the best method(s) to diagnose, quantify, and follow protein-energy malnutrition in both acute and chronically ill patients is uncertain<sup>12-14</sup>. In this section we will review the use of albumin as a nutritional marker and briefly discuss confounding factors in acutely ill patients which change albumin levels. In the last section of this review we will discuss recommendations for a more comprehensive nutritional assessment.

*Determinants of serum albumin:* Albumin is the body’s predominant serum binding protein and accounts for 75-80% of normal plasma colloid oncotic pressure and about half of the serum protein content<sup>10</sup>. Albumin is synthesized in the liver and has a variety of functions, including the maintenance of oncotic pressure and transport of molecules and drugs<sup>10, 15, 16</sup>.

Albumin has a half-life of ~18 days, and this makes it a poor choice for monitoring nutritional status day to day<sup>10</sup>. Serum albumin levels are influenced by illness, inflammation, exchange between intra- and extravascular compartments or transcapillary escape, hepatic synthesis and degradation, age, and multiple other factors<sup>10, 12, 14, 15</sup>. In inflammatory states, such as SIRS and sepsis, changes in the protective barrier of normal endothelial cells occurs<sup>17</sup>. Endothelial dysfunction increases vascular permeability resulting in the loss of intravascular fluid and its constituent proteins, such as albumin, into the interstitial space<sup>17, 18</sup>. This shift or “third spacing” of fluid and plasma proteins decreases measurable serum albumin<sup>18</sup>. Redistribution between the extravascular and intravascular space is affected by large amounts of intravenous fluids often needed in the critically ill patients who may have multiple infusions, require fluid boluses, or need blood products<sup>11</sup>. Thus, many factors acutely influence serum albumin levels; this protein has a large total body pool and long half-life, and consequently is nonspecific marker for assessment and monitoring of nutritional status<sup>12</sup>.

Multiple studies conducted in anorexic patients have reported a poor relationship between albumin levels and malnutrition by showing that even with very low BMIs and obvious malnutrition serum albumin levels remain normal<sup>10</sup>. Additionally, the Minnesota Starvation Experiment, perhaps one of most robust nutritional clinical studies performed, showed that in starved participants who all experienced a decrease in BMI and in lean body and fat mass, serum albumin only slightly decreased over the course of experiment and still remained within the normal range<sup>10</sup>.

*Inflammation and acute phase reactions:* The systemic response following inflammatory processes, including trauma, surgery, burns, autoimmune reactions, and cancer, is termed the acute phase response (APR)<sup>11</sup>. During the APR there is an increase in cytokine synthesis and release, followed by fluctuations in acute phase proteins (APP). Serum albumin, specifically, is referred to as a negative APP, as circulating serum levels decrease during inflammation and return to normal after the inflammation resolves<sup>11</sup>. This fluctuation

occurs due to reprioritization of protein synthesis towards immune mediators during the acute stage of critical illness and decreased need for other proteins not essential for immune function<sup>11, 14</sup>. The liver clears bacteria and bacterial products and produces and clears inflammatory mediators. Hepatocytes that have receptors for inflammatory mediators, such as interleukin-6 (IL-6) or tumor necrosis factor (TNF), will up-regulate the metabolic pathways for antiproteolytic enzymes, also called acute phase proteins (APPs)<sup>19</sup>. In sepsis, hepatocytes undergo a metabolic shift and reprioritize protein synthesis to aid in cellular repair and support the immune response. APPs enhance host defenses and other protective functions during immune responses; there is an increase or shift in the direction of positive APPs synthesis and conversely a decrease in the negative APPs synthesis<sup>19</sup>. Serum albumin, a negative APP, decreases as need for more immunomodulatory APPs increases.

#### CURRENT RECOMMENDATIONS FOR NUTRITIONAL ASSESSMENT

Nutrition assessment, as defined by the American Society for Parenteral and Enteral Nutrition (ASPEN), uses medical, nutritional and medication histories, anthropometric measurements, physical examination, and laboratory data to characterize the nutritional status of patients<sup>20</sup>. This global approach to nutrition assessment, including dietary history, clinical status, and social history, recognizes the fundamental relationship between nutritional status and severity of the illness<sup>20, 21</sup>.

Nutritional assessment helps identify patients who are at nutritional risk, meaning patients who either have actual malnutrition or who have the potential to become malnourished. The best approach involves the uses of several parameters for screening patients since no single parameter is a good indicator<sup>22</sup>. The overall consensus of the American Dietetic Association (ADA) and ASPEN recommends two or more of the following six characteristics for the diagnosis of adult patients having either severe or non-severe malnutrition: insufficient energy intake, weight loss, loss of muscle mass, loss of subcutaneous fat, localized fluid accumulation that can mask weight loss, and diminished functional status as measured

by hand grip strength <sup>22</sup>.

### 1. ENERGY INTAKE

Nutritional history includes recent changes in appetite or weight, ability to eat, bowel habits, activity level, nutrient intake, use of diets, feeding skills, types of feeding equipment used, food allergies or intolerances, and use of oral supplements <sup>23</sup>. The history is important to set a nutritional baseline in establishing the etiology of nutritional impairment, such as impediments to eating, absorbing, or both. Recent energy intake compared with estimated energy requirements is a primary standard in assessing malnutrition. When clinicians obtain the nutritional history and present illness from patients, they should estimate the energy requirements and compare them to the actual energy intake <sup>22, 24</sup>. Within the context of an acute illness or injury, severe malnutrition is defined as <50% of estimated energy requirements for > 5 days, and moderate malnutrition is defined as <75% of estimated energy requirements for >7 days <sup>22, 24</sup>.

### 2. INTERPRETATION OF WEIGHT LOSS/PHYSICAL FINDINGS

*Ideal Body Weight:* One standard parameter for evaluating changes in nutritional status involves review of the usual weight of an individual <sup>25</sup>. The ideal body weight (IBW), a comparison of the patient's current weight for height to the ideal body weight, can be used as a quantifying tool in the nutrition assessment process <sup>25</sup>. Interpretation of the IBW is as follows: 80-90% IBW is considered mild malnutrition, 70-79% is considered moderate malnutrition, and 0-69% is considered severe malnutrition <sup>25, 26</sup>

*Weight Loss:* Weight comparisons in adults can indicate severity of malnutrition and the percentage of weight lost from the usual baseline weight can be used as a parameter for malnutrition assessment. In adults with acute illness or injury, severe malnutrition is associated with an involuntary weight loss of >2% of usual body weight within 1 week, >5% weight loss within 1 month, and >7.5% weight loss in 3 months <sup>22</sup>. Moderate malnutrition is identified by a 1-2% weight loss within 1 week, a 5% weight loss within 1 month, and a 7.5% weight loss within 3 months. Careful evaluation of other factors affecting weight, such as hydration status, should be reviewed during this as-

essment <sup>22</sup>.

### 3. BODY FAT

*Body mass index (BMI):* BMI is a measure of weight for height and is an index used both as a measure of obesity and malnutrition. In adults, a BMI of less than 15 kg/m<sup>2</sup> is associated with a significant increase in morbidity, and a BMI of less than 18.5 kg/m<sup>2</sup> is considered underweight <sup>27-32</sup>. A BMI between 18.5 and 24.9 kg/m<sup>2</sup> is considered a healthy weight, a BMI between 25 and 29.9 kg/m<sup>2</sup> overweight, and a BMI of 30 kg/m<sup>2</sup> obese. Although the correlation between BMI and total body fat is relatively strong, variations in individuals do occur and can misclassify some patients as undernourished or obese using BMI alone.

*Body composition:* In adults, abdominal girth measurement is commonly used to indicate risk of coronary artery disease and other morbidity <sup>27, 29, 32</sup>. The waist circumference is measured by obtaining the distance around the smallest area right below the rib cage and above the umbilicus <sup>32</sup> and is used to determine excess abdominal fat. Waist girth circumference is not very useful for those with a BMI 35 kg/m<sup>2</sup> since at this level the incremental predictive power is lost <sup>27,29</sup>. Mid-arm circumference, mid-arm muscle circumference, and skinfold thickness estimate lean and fat mass <sup>27,29</sup>. Major limitations to these measurements in the intensive care setting include fluid shifts, changes in hydration status, and interobserver variability <sup>32</sup>. A moderate loss of subcutaneous fat (orbital, triceps, fat overlying the ribs) may represent severe malnutrition in acutely ill patients <sup>22, 29, 32</sup>. Therefore, physical examination, including palpation and inspection, should focus on possible fat and muscle wasting in temporal regions, thorax, deltoid muscle, and fine muscles of the hand <sup>22, 32</sup>.

### 4. MUSCLE MASS

A moderate loss of muscle mass will cause muscle wasting in the buttocks, temples, clavicles, scapula, and calf muscles in severely malnourished patients in critical care units <sup>22, 33</sup>. Nitrogen balance studies evaluate the adequacy of protein intake relative to need. Nitrogen metabolism is dependent on both energy and protein intake, and increasing en-

ergy intake often improves nitrogen balance <sup>33</sup>.

## 5. FLUID ACCUMULATION

In hospitalized patients, accurate interpretation of changes in weight can be complex, and clinicians must consider all factors that can contribute to weight changes during hospitalization. In the critically ill patients inflammation may cause fluid shifts which affect the body weight. For example, fluid shifts from the intravascular space to the extravascular space and from the intracellular space to the extracellular space with a concurrent decline in lean body mass can occur in malnutrition with little obvious change in weight. In addition, diuretic and resuscitation therapy, edema, ascites, and other fluid alterations can significantly alter body weight within short time periods and can conceal real changes in body weight <sup>34, 35</sup>.

## 6. REDUCED GRIP STRENGTH

The ability of an individual to function in his/her environment can be measured by the hand-grip dynamometry and forearm muscle dynamometry. These tests are inexpensive and easy to perform but may be difficult in the ICU <sup>32,36,37</sup>. Severely malnourished patients will have reduced grip strength <sup>22</sup>. Two studies have correlated handgrip and muscle dynamometry measurement with nutritional status, although the contribution of disease and injury to muscle strength was not measured within the studies <sup>36, 37</sup>. The assessment of range of motion of upper extremities can evaluate ability of patients to feed independently and may provide information regarding problems with energy intake in patients <sup>35-38</sup>.

## CONCLUSIONS

Patients in critical care units for more than 1-2 days need nutritional risk assessment, including evaluation of gastrointestinal function, and close monitoring <sup>39</sup>. They may need nutritional support to compensate for energy deficits. The enteral route is recommended in patients who have a functional gastrointestinal tract and who tolerate enteral feeding. Early initiation of tube feeds portends good patient outcomes and is recommended as soon as the he-

modynamic status is stabilized after any resuscitation period <sup>9</sup>. The ESPEN (European Society of Clinical Nutrition and Metabolism) guidelines recommend the addition of parenteral nutrition after 24-48 hours in the ICU patients receiving an inadequate amount of enteral feeding. However, The ASPEN/SCCM (Society of Critical Care Medicine) does not recommend the administration of parenteral nutrition during the first 7-10 days after admission <sup>40</sup>. Specific details about nutritional therapy are beyond the scope of this review.

The use of albumin as a nutritional marker is understandable given the clinical need for convenient, specific, and accurate nutritional indicators. Although serum albumin is not a good indicator of malnutrition, it has utility in identifying underlying diseases and the severity of acute illness and correlates with overall patient morbidity and mortality. Unfortunately, the perfect indicator does not exist, and multiple assessments need to be performed to identify malnutrition in the critically ill patients. Patients with a weight loss of more than 10%, poor dietary intake, loss of subcutaneous tissue, and muscle wasting represent a high risk group with poor outcomes <sup>41</sup>.

## KEY POINTS

1. Malnutrition is common in critically ill patients and influences outcomes.
2. Nutrition risk scores provide a good first step in patient evaluation.
3. Patients with malnutrition or at risk for the development of malnutrition need comprehensive evaluation using several indicators of nutritional status.
4. Serum albumin is not a good indicator of malnutrition and cannot inform therapeutic decisions.

**KEY WORDS-** malnutrition, albumin, critical care, assessment

---

**Author Affiliation:** Kristen Fuhrmann is a pharmacist at University Medical Center, Naree Panamonta is an Internal Medicine resident at TTUHSC, Shelley Roaten is a clinical dietician.

**Received:** 7/2/2013

**Accepted:** 8/30/2013

**Reviewers:** Kenneth Nugent MD, Scott O'Bannon Pharm D

**Published electronically:** 10/15/2013

**Conflict of Interest Disclosures:** None

---

## REFERENCES

1. Thibault R, Pichard C. Nutrition and clinical outcome in intensive care patients. *Curr Opin Clin Nutr Metab Care* 2010; 13(2):177-83.
2. ASPEN Board of Directors, Clinical Guideline Task Force for the use of parenteral and enteral nutrition in adult and pediatric patients. *JPEN J Parenter Enteral Nutr* 2002; 26(Suppl):1SA-138SA.
3. Singer P, Berger MM, Van den Berghe, et al. ESPEN guidelines on parenteral nutrition: intensive care. *Clin Nutr* 2009; 28:387-400.
4. Ziegler TR. Parenteral nutrition in the critically ill patient. *N Engl J Med* 2009; 361(11):1088-97.
5. Villet S, Chioloro RL, Bollmann MD, Revelly JP, Cayeux RNMC, Delarue J, Berger MM. Negative impact of hypocaloric feeding and energy balance on clinical outcome in ICU patients. *Clin Nutr* 2005; 24:502-9.
6. Robinson L, Diette GB, Song X, Brower RG, Krishnan JA. Low caloric intake is associated with nosocomial bloodstream infections in patients in the medical intensive care unit. *Crit Care Med* 2004; 32:350-7.
7. Dvir D, Cohen J, Singer P. Computerized energy balance and complications in critically ill patients: an observational study. *Clin Nutr* 2005; 25:37-44.
8. Schulman RC, Mechanick JI. Metabolic and nutrition support in the chronic critical illness syndrome. *Respir Care* 2012; 57(6):958-77.
9. Miller KR, Kilary LN, Lowen CC, Martindale RG, McClave SA. "CAN WE FEED?" A mnemonic to merge nutrition and intensive care assessment of the critically ill patient. *JPEN J Parenter Enteral Nutr* 2011; 35(5):643-59.
10. Friedman A, Fadem S. Reassessment of Albumin as a Nutritional Marker in Kidney Disease. *J Am Soc Nephrol* 2010; 21:223-230.
11. Banh L. Serum Proteins as Markers of Nutrition: What Are We Treating? *Nutrition Issues in Gastroenterology* 2006; 43:46-63.
12. Shenkin A. Serum Prealbumin: Is it a Marker of Nutritional Status or of Risk of Malnutrition? *Clinical Chemistry* 2006; 52(12):2177-8.
13. Fleck A, Raines G, Hawker F et al. Increased vascular permeability: a major cause of hypoalbuminemia in disease and injury. *Lancet* 1985; 1:781-5
14. Johnson M, Merline G, Sheldon J, et al. Clinical indications for plasma protein assays: transthyretin (prealbumin) in inflammation and malnutrition. *Clin Chem Lab Med* 2007; 45(3):419-426
15. Rothschild M, Oratz M, Schreiber S. Serum albumin. *Hepatology* 1988; 8:385-401
16. Rothschild M, Oratz M, Schreiber S. Regulation of albumin metabolism. *Annu Rev Med* 1975; 26:91-104.
17. Fisel RS, Are C, Barbul A. Vessel injury and capillary leak. *Crit Care Med* 2003; 31:S502-S511
18. Endemann D, Schiffrin E. Endothelial Dysfunction. *J Am Soc Nephrol* 2004; 15:1983-92.
19. Dhainaut JF, Marin N, Mignon A, et al. Hepatic response to sepsis: Interaction between coagulation and inflammatory processes. *Crit Care Med* 2001; 29(7):S42-45.
20. A.S.P.E.N. Board of Directors and Standards Committee. Definition of terms, style and conventions used in A.S.P.E.N. guidelines and standards. *Nutr Clin Pract* 2005; 20:281-285.
21. Barr J, Hecht M, Flavin K, et al. Outcomes in critically ill patients before and after the implementation of an evidence-based nutritional management protocol. *CHEST* 2004; 125:1446-1457.
22. White JV, Guenter P, Jensen G, et al. Consensus statement: Academy of nutrition and dietetics and American Society for Parenteral and Enteral Nutrition: Characteristics recommend for the identification and documentation of adult malnutrition (under nutrition). *JPEN J Parenter Enteral Nutr* 2012; 36:275.
23. Jeejeebhoy KN. Nutritional assessment. *Gastroenterol Clin North Am* 1998; 27(2):347-369.
24. Kondrup J. Can food intake in hospitals be improved? *Clin Nutr* 2001; 20:153-160.
25. Hopkins B. Assessment of nutritional status. In: Gottschlich MM, ed. *Nutrition Support Dietetics Core Curriculum*. 2nd ed. Silver Spring, MD: A.S.P.E.N.; 1993.
26. Blackburn GL, Bistrian BR. Nutritional and metabolic assessment of the hospitalized patient. *J Parenter Enteral Nutr* 1977; 1(1):11-22.
27. Charney DE, Meguid MM. Current concepts in nutritional assessment. *Arch Surg* 2002; 137(1):42-45.
28. A.S.P.E.N. Board of Directors. Definition of terms used in A.S.P.E.N. guidelines and standards. *J Parenter Enteral Nutr* 1995; 19:1-2.
29. ADA's definition for nutrition screening and assessment. *J Am Diet Assoc*. 1994; 838-839.
30. Centers for Disease Control and Prevention, National Center for Health Statistics. Prevalence of Overweight and

Obesity among Adults: United States, 1999–2000. Washington, DC: US Department of Health and Human Services; 2002.

31. Temblay A, Bandi V. Impact of body mass index on outcomes following critical care. *CHEST* 2003; 123: 1202-1207.

32. Hammond KA. Dietary and clinical assessment. In: Mahan LK, Escott-Stump S, eds. *Krause's Food, Nutrition, and Diet Therapy*. 11th ed. Philadelphia, PA: WB Saunders; 2004:407–435.

33. Homsy FN, Blackburn GL. Modern parenteral and enteral nutrition in critical care. *J Am Coll Nutr* 1983; 2:75-95.

34. Malone A. Anthropometric assessment. In: Charney P, Malone A, eds. *ADA pocket guide to nutrition assessment*, Chicago, IL: American Diabetes Association; 2004: 142-152.

35. Griffiths RD, Bongers T. Nutrition support for patients in the intensive care unit. *Postgrad Med J* 2005; 81:629-636.

36. Hunt DR, Rowlands BJ, Johnston D. Hand grip strength: a simple prognostic indicatory in surgical patients. *J Parenter Enteral Nutr* 1985; 9:701-704.

37. Kalfarentzos F, Spiliotis J, Velimezis G, et al. Prognostic nutritional index in gastrointestinal surgery. *Am J Surg* 1980; 139:160-167.

38. Petros S, Engelmann L. Enteral nutrition delivery and energy expenditure in medical intensive care patients. *Clin Nutr* 2006; 25:51-59.

39. Hiesmayr M. Nutrition risk assessment in the ICU. *Curr Opin Clin Nutr Metab Care* 2012; 15(2):174-80.

40. Singer P, Pichard C, Heidegger CP, Wernerman J. Considering energy deficit in the intensive care unit. *Curr Opin Clin Nutr Metab Care* 2010; 13(2):170-6.

41. Detsky AS, Smalley PS, Chang J. Is this patient malnourished? *JAMA* 1994; 271: 54-58.