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copper plays a role in iron metabolism, melanin synthesis, energy production, neurotransmitter synthesis, and CNS function; the syn thesis and cross-linking of elastin and collagen; and the scavenging of superoxide radicals. Dietary sources of copper include shellfish, liver, nuts, legumes, bran, and organ meats. Deficiency Dietary copper deficiency is relatively rare, although it has been described in premature infants who are fed milk diets and in infants with malabsorption (Table 344-2). Copper-deficiency anemia (refractory to therapeutic iron) has been reported in patients with malabsorptive diseases and nephrotic syndrome and in patients treated for Wilson's disease with chronic high doses of oral zinc, which can interfere with copper absorption. Menkes kinky hair syndrome is an X-linked metabolic disturbance of copper metabolism character ized by intellectual disability, hypocupremia, and decreased circulating ceruloplasmin (Chap. 425). This syndrome is caused by mutations in the copper-transporting ATP7A gene. Children with this disease often die within 5 years because of dissecting aneurysms or cardiac rupture. Aceruloplasminemia is a rare autosomal recessive disease character ized by tissue iron overload, mental deterioration, microcytic anemia, and low serum iron and copper concentrations. The diagnosis of copper deficiency is usually based on low serum levels of copper (<65 µg/dL) and low ceruloplasmin levels (<20 mg/ dL). Serum levels of copper may be elevated in pregnancy or stress con ditions since ceruloplasmin is an acute-phase reactant and 90% of cir culating copper is bound to ceruloplasmin. It has been suggested that mild or subclinical copper deficiency is more common than expected; at-risk individuals include patients with cholestasis or chronic diar rheal diseases, dialysis patients, and people on long-term zinc supple ments. The role of copper in cardiovascular disease, immune function, bone health, or neurodegenerative diseases is still unclear. Toxicity Copper toxicity is usually accidental (Table 344-2). In severe cases, kidney failure, liver failure, and coma may ensue. In Wil son's disease, mutations in the copper-transporting ATP7B gene lead to accumulation of copper in the liver and brain, with low blood levels due to decreased ceruloplasmin (Chap. 427). A potential negative role of copper in the pathogenesis of Alzheimer's disease has been reported. ■ ■SELENIUM Selenium, in the form of selenocysteine, is a component of the enzyme glutathione peroxidase, which serves to protect proteins, cell mem branes, lipids, and nucleic acids from oxidant molecules. As such, selenium is being actively studied as a chemopreventive agent against certain cancers, such as prostate cancer. However, it remains unclear whether selenium is effective as a chemopreventive agent or whether it increases cancer risk (e.g., prostate cancer). Convincing

evidence for a protective effect of selenium on cognitive decline or cardiovascular disease risk is presently lacking. Selenocysteine is also found in the deiodinase enzymes, which mediate the deiodination of thyroxine to triiodothyronine (Chap. 394). Rich dietary sources of selenium include seafood, muscle meat, and cereals, although the selenium content of cereal is determined by the soil concentration. Countries with low soil concentrations include parts of Scandinavia, China, and New Zealand. Keshan disease is an endemic cardiomyopathy found in children and young women residing in regions of China where dietary intake of selenium is low (<20 µg/d). Concomitant deficiencies of iodine and selenium may worsen the clinical manifestations of cretinism. Chronic ingestion of large amounts of selenium leads to selenosis, character ized by hair and nail brittleness and loss, garlic breath odor, skin rash, myopathy, irritability, and other abnormalities of the nervous system. ■ ■CHROMIUM Chromium potentiates the action of insulin in patients with impaired glucose tolerance, presumably by increasing insulin receptor–medi ated signaling, although its usefulness in treating type 2 diabetes is uncertain. In addition, improvement in blood lipid profiles has been reported in some patients. The usefulness of chromium supplements in muscle building has not been substantiated. Rich food sources of chromium include yeast, meat, and grain products. Chromium in the

trivalent state is found in supplements and is largely nontoxic; however, chromium-6 is a product of stainless steel welding and is a known pul monary carcinogen as well as a cause of liver, kidney, and CNS damage.

■ ■MAGNESIUM See Chap. 421. ■ ■FLUORIDE, MANGANESE, AND ULTRATRACE ELEMENTS An essential function for fluoride in humans has not been described, although it is useful for the maintenance of structure in teeth and bones. Adult fluorosis results in mottled and pitted defects in tooth enamel as well as brittle bone (skeletal fluorosis). Manganese and molybdenum deficiencies have been reported in patients with rare genetic abnormalities and in a few patients receiv ing prolonged total parenteral nutrition. Several manganese-specific enzymes have been identified (e.g., manganese superoxide dismutase). Deficiencies of manganese have been reported to result in bone demin eralization, poor growth, ataxia, disturbances in carbohydrate and lipid metabolism, and convulsions. Ultratrace elements are defined as those needed in amounts <1 mg/d. Essentiality has not been established for most ultratrace elements, although selenium, chromium, and iodine are clearly essential (Chap. 394). Molybdenum is necessary for the activity of sulfite and xanthine oxidase, and molybdenum deficiency may result in skeletal and brain lesions. ■ ■FURTHER READING Combs GF Jr, Mcclung JP: The Vitamins: Fundamental Aspects in CHAPTER 345 Nutrition and Health, 6th ed. London, Academic Press, 2022. DeAngelo SL et al: Selenoproteins and tRNA-Sec: Regulators of can cer redox homeostasis. Trends Cancer 9:1006, 2023. Fan D et al: Cell signaling pathways based on vitamin C and their appli cation in cancer therapy. Biomed Pharmacother 162:114695, 2023. Imdad A et al: Vitamin A supplementation for preventing morbid Malnutrition and Nutritional Assessment ity and mortality in children from six months to five years of age. Cochrane Database Syst Rev 3:CD008524, 2022. Lassi ZS et al: Zinc supplementation for the promotion of growth and prevention of infections in infants less than six months of age. Cochrane Database Syst Rev 4:CD010205, 2020. Mechanick JI et al: Clinical practice guidelines for the perioperative nutrition, metabolic, and nonsurgical support of patients undergoing bariatric procedures–2019 update. Surg Obes Relat Dis 16:175, 2020. Ota Y et al: Comprehensive review of Wernicke encephalopathy: Pathophysiology, clinical symptoms and imaging findings. Jpn J Radiol 38:809, 2020. Staub E et al: Enteral zinc supplementation for prevention of morbid ity and

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Malnutrition and

Nutritional Assessment Malnutrition occurs in 30–50% of hospitalized patients depending on the setting, the patient’s diagnosis, and the criteria that are used to diagnose malnutrition. Notable adverse outcomes associated with mal nutrition include poor wound healing, compromised immune status, and impaired organ function. These can lead to increased length of hospital stay, readmissions, higher mortality, and increased health care costs. It is now widely appreciated that acute or chronic inflammation

At risk for malnutrition

Use validated screening tools Risk screening Diagnostic Assessment Assessment criteria

Phenotypic

Non-volitional weight loss

Low body mass index

Reduced muscle mass

Etiologic

Reduced food intake or assimilationa

Disease burden/inflammatory conditionb Diagnosis Meets criteria for malnutrition diagnosis

Requires at least 1 Phenotypic criterion and

1 Etiologic criterion Severity Grading Determine severity of malnutrition

Severity determined based on Phenotypic

criterion FIGURE 345-1 Global Leadership Initiative on Malnutrition (GLIM) diagnostic scheme for screening, assessment, diagnosis, and grading of malnutrition. a≤50% of energy requirement >1 week, or any reduction for >2 weeks, or any chronic gastrointestinal condition that adversely impacts food assimilation or absorption. bAcute disease/injury (e.g., major infection, burns, trauma or closed head injury) or chronic disease (e.g., malignant disease, chronic obstructive pulmonary disease, congestive heart failure, chronic renal disease or any disease with chronic or recurrent inflammation) or C-reactive protein may be used as a supportive laboratory measure (mild inflammation: 3.0-9.9 mg/L, moderate inflammation: 10-50 mg/L, severe inflammation: >50 mg/L). (Reproduced with permission from Jensen GL et al: GLIM criteria for the diagnosis of malnutrition: A consensus report from the global clinical nutrition community. JPEN J Parenter Enteral Nutr 43:32, 2019, Figure 1.) PART 10 Disorders of the Gastrointestinal System contributes to the

pathophysiology of malnutrition. The presence of inflammation can also render historic nutrition parameters, like albumin and prealbumin, unreliable. In patients with high levels of inflammation, nutrition care is supportive. At moderate or low levels of inflammation, nutrition care may be therapeutic in reducing the nutritional deficits and improving clinical outcomes. ■ ■MALNUTRITION DIAGNOSIS IN CLINICAL SETTINGS Several tools have been used to diagnose malnutrition, such as the Sub jective Global Assessment, Mini Nutrition Assessment, and the Acad emy of Nutrition and Dietetics and American Society for Parenteral and Enteral Nutrition Indicators of Malnutrition. These tools use com mon parameters such as dietary intake and weight history, muscle and fat loss, body mass index (BMI), and/or physical function to diagnose malnutrition. However, nuances between them lead to incomparable malnutrition prevalence rates in the literature. TABLE 345-1 Thresholds for Severity Grading of Malnutrition Into Stage 1 (Moderate) and Stage 2 (Severe) Malnutrition WEIGHT LOSS (%) LOW BODY MASS INDEX (kg/m2)b REDUCED MUSCLE MASSc Stage 1/moderate malnutrition (requires 1 phenotypic criterion that meets this grade) 5%–10% within the past 6 months, or 10%–20% beyond 6 months Stage 2/severe malnutrition (requires 1 phenotypic criterion that meets this grade)

> 10% within the past 6 months, or 20% beyond 6 months aSeverity grading is based on the noted phenotypic criteria, whereas the etiologic criteria described in the text and Figure 345-1 are used to provide the context to guide intervention and anticipated outcomes. bFurther research is needed to secure consensus reference body mass index for Asian populations in clinical settings. cFor example, appendicular lean mass index (kg/m2) by dual-energy absorptiometry or corresponding standards using other body composition methods such as bioelectrical impedance analysis, computed tomography, or magnetic resonance imaging. When not available or by regional preference, physical examination or standard anthropometric measures such as mid-arm muscle or calf circumferences may be used. Functional assessments such as hand-grip strength may be used as a supportive measure. Source: Reproduced with permission from Jensen GL et al: GLIM criteria for the diagnosis of malnutrition: A consensus report from the global clinical nutrition community JPEN J Parenter Enteral Nutr 43:32, 2019, Table 4.

In 2019, four international nutrition societies introduced the Global Leadership Initiative on Malnutrition (GLIM) consensus criteria for diagnosing malnutrition in adults (Fig. 345-1). GLIM incorporates broadly available nutrition parameters that are commonly used in other diagnostic tools to enable data from these earlier assessment methods to be described in the GLIM framework. To diagnose malnutrition and its severity, GLIM requires one etiologic criterion (inflammation/ disease burden or reduced food intake/assimilation) and one phenotypic criterion (weight loss, low BMI, or reduced muscle mass) (Table 345-1). To facilitate the diagnosis of malnutrition in settings with variable availability and expertise of personnel and equipment across clinical settings, several options are suggested for the phenotypic criterion. The phenotypic criterion is used to define the severity of malnutrition. Recent publications highlight GLIM’s widespread adoption in hospitals and clinical settings, showing predictive value for adverse outcomes. ■ ■NUTRITION ASSESSMENT FOR PROTEINCALORIE MALNUTRITION The Nutrition Care

Process in the hospital setting involves screening for malnutrition risk, comprehensive nutrition assessment of patients at malnutrition risk, development of a nutrition care plan, and moni toring for needed adjustments to the plan. Screening for the risk of malnutrition is typically undertaken by the bedside nurse or by a clini cal dietitian using validated tools such as the Malnutrition Screening Tool, Malnutrition Universal Screening Tool, Nutritional Risk Screen ing 2002, or other in-house tools. A comprehensive nutrition assess ment includes a review of medical, surgical, and social history; dietary intake and weight history; medication profile; available laboratory, diagnostic tests, body composition, and anthropometric measures; and nutrition-focused physical examination. The clinical dietitian designs an individualized intervention and monitoring plan for the patient. The nutrition assessment findings and care plan are shared with the physician who may make a malnutrition diagnosis and prescribe the recommended nutrition care plan. The response to nutrition therapy is monitored with adjustments to the care plan as needed. Micronutrient deficiencies of clinical relevance may be detected in association with malnutrition, but a detailed discussion of their assessment is beyond the scope of this chapter (see Chap. 344). In ambulatory clinic settings, physicians may have less access to dietitian support than in inpatient settings. However, the GLIM criteria are designed to be feasible even in an outpatient setting. Nurses can obtain measures of height and weight (for BMI) and calf or arm cir cumference to assess muscle mass. A diagnosis of malnutrition can be made in the clinic with referral of the patient to a dietitian for a detailed nutrition assessment and development of a care plan. ■ ■COMPONENTS OF A DETAILED NUTRITIONAL ASSESSMENT Medical/Surgical History and Clinical Diagnosis The risk of malnutrition varies widely across medical/surgical diagnoses and clinical settings. During the early stages of diseases, malnutrition may be less likely than with disease progression. Knowledge of a patient’s PHENOTYPIC CRITERIAa <20 if <70 years, <22 if ≥70 years Mild-to-moderate deficit (per validated assessment methods; see below) <18.5 if <70 years, <20 if ≥70 years Severe deficit (per validated assessment methods; see below)

medical/surgical history and associated clinical diagnoses is especially helpful in discerning the likelihood of malnutrition and inflamma tion. Several conditions or diseases are characterized by severe acute inflammatory response (e.g., critical illness), whereas others are more typically associated with a chronic inflammatory response that is mild to moderate in severity (e.g., chronic cardiometabolic, oncologic, or gastrointestinal disease, or organ failure) and may be relapsing and remitting. It is common for acute inflammatory events to be superim posed on patients with chronic conditions; for example, a patient with chronic renal disease may be admitted to the hospital with sepsis, such that the acute increase in inflammation should be time-limited while the chronic inflammation will continue. The inflammatory milieu, especially when severe, may modify nutrient requirements by elevating resting energy expenditure and promoting anabolic resistance and catabolism in muscle. Inflamma tion also promotes anorexia, decreases food intake, and further com promises nutritional status. A deteriorating course may result because severe inflammation may reduce the benefit of nutritional interven tions to being supportive rather than therapeutic, and the associated malnutrition may diminish the effectiveness of medical therapies. It is also imperative to recognize medical/surgical conditions that place patients at increased risk of becoming malnourished because they have increased nutritional requirements or compromised intake or assimilation. Chronic gastrointestinal disease, even if relapsing and remitting, may limit nutrient assimilation. Therefore, patients with seemingly adequate oral intake may still be at risk of malnutrition due to compromised nutrient assimilation. Clinical Signs and Physical Examination Nonspecific clinical indicators of inflammation include fever, hypothermia, and tachy cardia. The

nutrition-focused physical examination should identify edema as well as physical signs of weight gain/loss. Physical findings of weight loss associated with decreased muscle and subcutaneous fat mass should be noted. If edema is present, an estimation of dry weight may be needed to identify actual weight loss. Anthropometric Data Nonvolitional weight loss is a well-val idated nutrition parameter associated with underlying disease or inflammation. The degree and duration of nonvolitional weight loss determine its clinical significance. A 5–10% loss of body weight over 6 months is clinically relevant, whereas >10% loss over the same dura tion is severe. The trend in body weight may be obtained from patients, their family or caregivers, and the medical record. Obtaining body weight at each clinic, emergency department, and hospital visit will support efforts to detect changes in this important parameter. Training staff members to weigh patients consistently without shoes and heavy outer garments and maintaining scales and stadiometers in good calibration facilitate acquisition of trustworthy weight data. Clinical sites may maintain chair or bed scales for patients who cannot stand. Height may be estimated by doubling the arm span measurement from the sternal notch to the end of the longest finger or by measurement of knee height using a caliper. BMI, defined as weight (kg)/height (m2), is used primarily to screen for overweight or obesity. The National Institutes of Health/World Health Organization BMI categories for adults are as follows: BMI <18.5 kg/m2 = underweight, BMI 18.5–24.9 kg/m2 = desirable, BMI 25.0–29.9 kg/m2 = overweight, and BMI ≥30 kg/m2 = obese. Note that patients of any BMI status can be malnourished. Overweight or obese patients can be malnourished due to weight loss or loss of muscle mass (e.g., sarcopenic obesity). Similarly, being underweight does not equate to malnutrition. BMI <18.5 kg/m2 is infrequent in economically developed countries. Classical anthropometric measurements such as arm and calf cir cumference can be helpful. However, measuring the calf circumfer ence may be more accessible as it requires less specialized training in comparison to measuring the arm circumference. Additionally, calf circumference has established cutoff values validated for various BMI ranges and ethnicities. Clinically available body composition mea sures include bioelectrical impedance analysis (BIA), dual-energy x-ray

absorptiometry (DXA), and air displacement plethysmography (Table 345-2). BIA has the advantage of being portable and is used for patients in diverse clinical settings. Abdominal computed tomography (CT) and magnetic resonance imaging (MRI) taken for disease diagnostic purposes can be interpreted to measure muscle mass, though they are not routinely used to aid in the diagnosis of malnutrition.

Laboratory Indicators There is, unfortunately, no biomarker descriptive of comprehensive nutritional status. Laboratory findings (Table 345-2) should be evaluated in combination with other assess ment parameters to appropriately diagnose malnutrition. Albumin and prealbumin are often available in patients with suspected malnutrition; however, these measures have poor sensitivity and specificity as indi cators of malnutrition. Albumin and prealbumin are negative acutephase reactants with levels that are reduced by the systemic response to injury, disease, or inflammation. C-reactive protein, a positive acutephase reactant, may be useful to identify the presence of inflammation, particularly if the clinical context is unclear about the extent of inflam mation. Trends in repeated levels of C-reactive protein over time may help to clarify the extent and trajectory of the inflammatory challenge. Research suggests that interleukin 6, and perhaps other cytokines, may offer promise as indicators of inflammatory status. Nonspecific labora tory indicators that are often associated with inflammatory response include leukocytosis and hyperglycemia. Additional tests that may be obtained to help confirm the presence of inflammatory

response include 24-h urine urea nitrogen and indirect calorimetry to measure energy expenditure. In the setting of a severe acute systemic inflamma tory response, negative nitrogen balance and elevated resting energy expenditure are anticipated. CHAPTER 345 Diet History Brief diet surveys are available to target specific aspects of the diet, such as food patterns, to prevent disease or track intake of nutrients of key importance such as calcium, vitamin D, and phosphorus for bone disease management. However, more specific information about the adequacy of intake of key nutrients or food patterns relative to estimated requirements is most often obtained by consultation with a clinical dietitian. Since patients often present to health care practitioners with acute medical events superimposed upon chronic health conditions, it is common for patients to have had decreased food intake and progressive malnutrition for extended peri ods prior to assessment. Therefore, compromised dietary intake must not be overlooked so that appropriate intervention may be undertaken. Malnutrition and Nutritional Assessment Assessment of the adequacy of nutritional intake must include oral nutrition supplements, enteral tube feedings, and parenteral nutrition both during hospitalization and as a component of home nutrition support. Ongoing reassessment of actual intake received by the patient is undertaken in hospital settings, most commonly by the clinical dietitian, the bedside nurse, and the nutrition support team if available. Changes in the adequacy of oral intake, body weight, or tolerance to enteral or parenteral feedings would all require adjustment of the feed ings to optimize patient care. A comprehensive assessment should also include a review of medi cations, vitamin/mineral supplements, or herbal remedies with atten tion to undesirable nutrition-sensitive side effects such as anorexia, dysgeusia, oral mucositis, nausea, vomiting, diarrhea, and constipation. Potential drug–nutrient interactions should also be identified to enable alternative therapeutic options. Functional Outcomes Advanced malnutrition is accompanied by declines in muscle mass and function that can be detected by various functional test outlined in Table 345-2. Among them, hand grip strength may have better clinical utility since it is easily assessed and helps to identify which patients benefit most from individualized nutritional support. In summary, malnutrition puts patients at risk of adverse outcomes. Therefore, timely diagnosis and treatment of malnutrition should be provided to improve patient outcome. Robust evidence exists dem onstrating that a multipronged approach to malnutrition intervention reduces mortality and holds promise to improve the quality of life of patients.

PART 10 Disorders of the Gastrointestinal System TABLE 345-2 Common Body Composition Studies, Laboratories, and Other Studies Used in Nutrition Assessment TEST NOTES Body Composition Studies (Recommended) Air plethysmography May be used to assess body composition. It comprises a dual-chamber, sealed compartment containing an oscillating diaphragm, which allows it to measure body volume using Poisson’s law, and compute fat mass and fat-free mass. However, its validity in a racially diverse population needs further studies, and it is not readily available in most hospitals. Bioelectrical impedance analysis (BIA) A promising method to assess body composition as the equipment is easily portable. It uses the opposition of an electrical current through body tissues (i.e., impedance) and population-specific equations to estimate total body water and body composition. BIA equations are highly device and population specific. Fever, some medications, and fluid/electrolyte disturbances can influence its precision and accuracy. Calf circumference A practical and valid clinical estimate of skeletal muscle mass, in which low calf circumference indicates muscle loss. Ethnic specific reference ranges are available, and measurements should be adjusted in persons with body mass index different from the normal range (underweight, overweight, or obese). The technique requires less training than mid-upper arm circumference. Dual energy x-ray absorptiometry (DXA) May be used to assess regional and

total fat mass in a diverse group of adult clinical patients. However, its validity in assessing muscle mass is unclear. Imaging with computed tomography (CT) or magnetic resonance imaging (MRI) May be used to quantify fat and fat-free mass when scans are taken for other diagnostic purposes. Both are costly, and CT entails x-ray exposure. Mid-upper arm circumference Mid-upper arm circumference is the circumference of the upper arm measured at the midpoint between the olecranon process and the acromion process. It estimates both muscle and subcutaneous fat stores, but references are population specific. Take note that this measurement technique requires reliability training. Ultrasound A promising method to assess body composition as the equipment is easily portable. It uses high-frequency sound waves to capture live images of muscle tissues. Since most studies examined a single muscle, it is unclear if results can be extrapolated to reflect overall nutritional status. More research is needed to establish standardization of measurement protocol. Laboratory Tests and Other Studies (Recommended) Complete blood count with differential May be used to screen for nutritional anemias (iron, B12, and folate deficiencies) and thrombocytopenia (vitamin C and folate deficiencies). C-reactive protein (CRP) May be used to confirm systemic inflammation. While inflammation may be associated with malnutrition, CRP lacks specificity as a biomarker for diagnosing malnutrition. Nevertheless, when combined with other nutrition assessment methods, CRP can complement the diagnostic process for malnutrition. In addition, elevated levels may be associated with reduced food intake and a lack of response to nutritional interventions. Indirect calorimetry May be used to determine resting energy expenditure (REE) to aid in determining caloric intake goal. Predictive equations that were based on REE measures in specific populations are also used to estimate REE, especially when indirect calorimetry is not available. Nitrogen balance (NB) May be used to reflect the degree of catabolism and adequacy of protein replacement delivery in patients with normal renal and liver function. Method requires collection of 24-h urine. Nitrogen balance = (protein delivery [g]/6.25) − (urinary urea nitrogen + 4 insensible losses). Specific micronutrients May be used to validate clinical symptoms of micronutrient deficiencies. Exercise caution with interpretation since levels can be influenced by acute illness. Laboratory Tests and Other Studies (Not Recommended) Blood urea nitrogen (BUN), serum creatinine Not recommended as malnutrition indicators. Although low BUN and serum creatinine may reflect reduced muscle mass, their levels are influenced by factors unrelated to malnutrition (e.g., renal and hepatic insufficiency). Cholesterol Not recommended as a malnutrition indicator. While becoming hypocholesterolemic (<120 mg/dL) during hospitalization may be linked to poorer clinical outcomes, insufficient evidence supports its use for diagnosing malnutrition and evaluating nutritional interventions. Creatinine height index (CHI) Not recommended as a malnutrition indicator. CHI = (24-h urinary creatinine excretion/ideal urinary creatinine for gender and height) × 100. Although urinary creatinine excretion reflects muscle mass, it can be influenced by renal insufficiency, meat consumption, physical activity, and trauma. Requires accurate 24-h urine collection. Cytokines Not recommended as malnutrition indicators. Their cutoff values and role as an indicator of inflammatory status are still being studied. Electrocardiogram Not recommended as a malnutrition indicator. Although prolonged QT interval may be present in severely malnourished patients, the former can be influenced by factors unrelated to malnutrition. Fecal fat test May be used to validate clinical symptoms of fat malabsorption. It reflects the percentage of dietary fat that the body does not absorb. Requires stool samples collected over 72 h during ingestion of high-fat diet. Serum proteins (albumin, prealbumin, transferrin, and retinol-binding protein) Not recommended as a malnutrition indicator. Their levels are influenced by factors other than malnutrition (e.g., systemic inflammation, hepatic and renal insufficiency, protein-losing enteropathies, corticosteroids, hydration, and iron status), reducing their specificity in diagnosing malnutrition and evaluating nutritional interventions. However, they remain valuable

for predicting clinical outcomes. Skin testing—recall antigens Not recommended as a malnutrition indicator. Although delayed hypersensitivity is associated with malnutrition, the former can be influenced by factors unrelated to malnutrition. Total lymphocyte count Not recommended as malnutrition indicator. Low levels are influenced by factors unrelated to malnutrition. Urine 3-methylhistidine Not recommended as a malnutrition indicator. Although it reflects muscle mass, its level is influenced by meat intake and poorly reflects changes in body protein stores. Requires accurate 24-h urine collection (Continued)

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