SURGICAL NUTRITION

Edited by

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Preface

Although the importance of nutritional replenishment of the surgical patient has been recognized for over 50 years, there has been an enormous accumulation of scientific data regarding the pathophysiologic response to nutritional states during the past two decades. Furthermore, the development of safe and efficacious means for providing nutritional support—via both the enteral and parenteral routes—has allowed the physician to more aggressively treat malnutrition prior to the appearance of morbid complications resulting from inadvertent starvation during the pre- and postoperative periods.

This text is organized to summarize the current state of the art in a manner that allows the surgical practitioner to concisely review the pathophysiologic mechanisms of surgical malnutrition as well as the therapeutic approaches for providing replenishment. The reader is provided a comprehensive review of techniques for nutritional assessment, methods of nutrient delivery, and composition of currently utilized enteral and parenteral solutions. Detailed diagnostic and therapeutic management of those surgical disease states with special nutritional requirements is outlined, including gastrointestinal disease, morbid obesity, trauma and burns, cancer, neonatal and pediatric abnormalities, sepsis, and postoperative respiratory failure. In addition, areas of controversy are presented, allowing the reader a glimpse of current research activity, which may clarify present uncertainty with regard to the expected consequences of inappropriate nutritional therapy.

The editors are appreciative of the thoughtful and comprehensive contributions of the individual authors. These combined efforts have provided the reader with an extraordinary opportunity to obtain an updated base of knowledge with which to derive practical solutions to frequently encountered clinical problems. The experienced clinician is aware that few, if any, "patent" regimens are available to deal with the myriad of pre- and postoperative malnutrition syndromes. Instead, a successful outcome is dependent on expert clinical

and laboratory evaluation, knowledge of expected biologic response to surgical disease and nutritional deprivation, and prompt therapeutic intervention.

With these thoughts in mind, our objective has been to create a text that would provide the practitioner with a concisely outlined review of surgical nutrition.

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Contents

1 Nutritional Assessment in Surgical Patients

David N. Herndon

Nutritional support may be required in the surgical patient who is malnourished or who is catabolic as a result of his illness. Disease or injury may prevent normal intake by central depression of the appetite center or by interruption of gastrointestinal (GI) function. Many patients present with evidence of malnutrition. Increased metabolic demands, characteristic of many surgical diseases, exacerbate any preexisting deficit. Nutritional assessment requires a knowledge of the general metabolic requirements of the individual patient in addition to the ability to estimate the degree of any preexisting deficits in body fuel stores.

Hypermetabolism, increased glucose flow, and severe protein and fat wasting are characteristic of the human response to major trauma, infections, and surgical complications. In 1930 Sir David Cuthbertson first demonstrated persistent negative nitrogen, potassium, zinc, sulfur, and creatinine balance consequent to femur fractures in a small animal model.[1] Patients subjected to moderate operative trauma such as cholecystectomy become only slightly hypermetabolic, while patients with peritonitis have increases in metabolic rate of 5 to 25 percent above normal; multiply-traumatized, respirator-dependent patients can have metabolic requirements 30 to 75 percent above normal. The metabolic rate of patients with burns greater than 40 percent total body surface

area is 100 percent greater than normal.[2,3] In starving, 20 to 35 percent total body surface area burned patients, tissue breakdown liberates as much as 20–25 g/m²·day of nitrogen.[4] This approaches lethality from the exhaustion of essential protein stores within 3 to 4 weeks postinjury. Adult patients subjected to elective surgery lose 5 to 10 percent of their admission weight; patients experiencing multiple trauma and sepsis experience approximately 20 to 30 percent weight loss.[2] Patients with burns covering more than 40 percent total body surface area and treated with the most vigorous possible oral alimentation have been shown to lose 25 percent of their preadmission weight by 3 weeks postinjury.[5] In burned patients, the precise energy requirements needed to maintain weight and nitrogen equilibrium have been precisely calculated by Curreri et al.[6] From linear regression analysis of weight change versus predicted dietary intakes, energy requirements have been found to be 25 kcal/kg plus 40 kcal per percent body surface area burn per 24 hours. For large burns, this requirement can reach more than 5000–6000 kcal/day.[6] In other surgical patients, actual metabolic demands can be predicted by assuming basic requirements of 25 kcal/kg·24 hr and adding an additional amount equal to the percentage increase in metabolic rate caused by the injury. Metabolic rates may be determined by direct or indirect calorimetry if available, this being by far the most accurate method of determining maintenance demands in any given setting. Indirect calorimetry is simple and clinically most useful; it involves measurement of oxygen intake and carbon dioxide output by volumetric techniques using transparent head canopies.[3]

Precise determination of nitrogen balance is also a good method for assessing the degree of catabolism and for evaluating the adequacy of support, once instituted. For routine clinical use, nitrogen balance can be estimated in any hospital by using a formula based on urine urea nitrogen excreted over a 24-hour period.[7,48,50] A 24-hour urine collection can be processed for nitrogen determination (expressed in g/l); multiplying this by the total volume of urine excreted in a 24-hour period, total urine urea nitrogen loss per 24 hours is determined. Urea nitrogen is the predominant route of nitrogen loss, but an approximate correction factor for nonurea urine nitrogen loss must be added (approximately 3 g/day). Fecal losses of 1 g nitrogen per stool should also be considered. Nitrogen intake can be estimated by dividing total grams of protein consumed by the constant 6.25. Nitrogen balance can then be calculated by the following equation:

$$\text{Nitrogen balance/24 hr} = \frac{\text{Grams protein consumed/24 hr}}{6.25} - [\text{Grams urea urine nitrogen formed/24 hr} + 3\text{ g (nonurea nitrogen loss)} + 1\text{ g} \times \text{Number of stools/24 hr}]$$

This formula is simple to use and fairly accurate when patients are off all oral intake or on controlled steady state diets. Accuracy diminishes with large losses from diarrhea, gastrointestinal bleeding, skin losses or losses from pro-

tein-wasting nephropathies. Elective postoperative patients, who are essentially fasting and have less than 10 percent increases in metabolic rate, will excrete approximately 8 g urea nitrogen per 24 hours. Patients with major injury, such as multiple long bone fractures as in Cuthbertson's original model, or infections whose metabolic rate is 20 to 30 percent above normal will excrete 9 to 11 g of urea nitrogen per 24 hours; the septic, respirator-dependent or multiply traumatized patient with a metabolic rate of 75 percent above normal excretes 11 to 14 g urea nitrogen per day; major burns with metabolic rates 100 percent above normal have urea nitrogen losses as high as 20 g/day.[8] The adequacy of nutritional support is determined by following serial body weights as well as by following calculated nitrogen balance. Maintenance of admission body mass can be expected only if a zero balance is achieved. Repletion of preexisting deficits cannot be expected unless a positive nitrogen balance of approximately 5 g/24 hr is achieved.

The failure to meet the large caloric and protein requirements necessary for weight maintenance, nitrogen balance, and energy equilibrium results in delayed and abnormal wound healing. Defects in fibroblast and white blood cell metabolic function have been demonstrated. Isolated leukocytes from patients with large burn injuries have been shown to consume only one-half to one-third the amount of oxygen of normal leukocytes.[9] Demonstrable deficiencies in immune competence[10,11] and cellular membrane active transport occur and can be reversed by proper nutritional support.[12] Feeding an optimal diet to starved mice has been shown to abolish susceptibility to viral infection,[13] and improved antibody production in depleted individuals supplemented with protein has been demonstrated.[14] Leukocyte derangements subsequent to starvation may also be improved by caloric repletion.[15] Law et al. showed enhanced delayed skin reactivity and in vitro lymphocyte proliferation in response to mitogens after intravenous hyperalimentation in man.[11,16] Gross and Newberne in a detailed review of the role of nutrition in immunologic function[17] conclude that protein calorie malnutrition most profoundly effects cell mediated immunity via defective phagocytic function; impaired chemotaxis; and defective bactericidal, candidacidal, and metabolic function. Isolated vitamin deficiencies can result in marked reduction of these same individual immunologic functions.

Identifying the patient in need of nutritional support requires, in addition to recognition of the hypermetabolic state, an estimation of the degree of nutritional deficit already present. Protein calorie malnutrition has been demonstrated in 25 to 50 percent of surgical and medical patients hospitalized for more than 2 weeks.[18,19] Surgical patients of all social classes may be depleted from increased metabolic demands; from decreased intake as a result of the anorexia of malignancy, infection, or chronic disease; or from malabsorptive disorders.

The primary tool for assessing nutritional status is the patient's history with the degree of weight loss being the cornerstone. Studly, in a 1936[20] study of 46 patients operated upon for chronic ulcer disease, demonstrated the percentage of preoperative weight loss to be the principal factor correlating with

operative mortality. Mortality in patients who had lost less than 20 percent of their body weight was 3.5 percent, whereas patients with weight losses of more than 20 percent from their pre-morbid body weight had operative mortalities of 33 percent. In starvation states, a gradual total body weight loss of 40 to 50 percent may be tolerated before death ensues; but any underlying organic disease, intercurrent infection, or stress, including surgery, will hasten the starving patient's demise.[21] Kinney has shown that only 1584 kcal are liberated per kg of weight lost in starving patients following minor operative trauma.[22] Therefore, major losses of lean body mass must be incurred to satisfy only meager increases in energy demands, with a resultant devastating effect on the total body nitrogen economy. The most important historical information is percent body weight loss from the pre-illness optimal weight. Of significance but less import is the comparison of the patient's body weight to ideal body weight for his height, age, and sex. (Body weight, age and height standards are available in most hospital nutrition departments. An excellent compilation is the Canadian Weight Height Survey.[23])

Specific historical analysis for vitamin deficiencies is mandatory. Levy et al, in a study of a randomly selected United States hospital population, found only 12 percent of 120 patients studied to have normal serum levels of vitamins tested. Forty-five percent of patients were deficient in folic acid, and approximately 30 percent were deficient in thiamine, nicotinic acid, and pyridoxine. Ten to 15 percent of the patients were deficient in vitamin B_{12}, vitamin C, riboflavin, vitamin E, vitamin A, and pantothenic acid. Thirty-eight percent of the patients had two deficiencies. Clinical signs of hypovitaminosis were present in 46 patients. None, however, had eye signs or bone changes classic for rickets and scurvy. Symptoms included neuropathy, with either diminished or absent reflexes, decreased sensation, or paresthesia. The most common symptom was glossitis with papulary atrophy of the tongue, which was seen in 33 patients. Ninety percent of the patients with hypovitaminemia also had decreased serum albumin levels.[24] Night blindness and skin changes are other common symptoms.

Historical quantitation of the kind and amount of food eaten by a patient is important, and the hospital dietician may be very helpful in obtaining this information. A full gastrointestinal history is also important to identify alimentary dysfunction, e.g., gastric outlet obstruction or malabsorption syndromes.

A combination of more objective techniques for assessing protein calorie malnutrition have been popularized by Blackburn, Bistrian, Kaminsky, Jeejeebhoy, and others.[18,25–29] Anthropometric measurements compared to standards include weight in relation to height, triceps skin-fold thickness, and arm muscle circumference. Other parameters include measurements of 24-hour urinary creatinine excretion compared to height; blood counts with focus on total lymphocyte count and hemoglobin; serum albumin; serum transferrin or total iron binding capacity; and results of delayed hypersensitivity skin tests.

Midarm circumference is measured with a tape measure at the halfway point between the acromial tip and the olecranon process of the left arm; triceps

skin fold is measured with caliper at the same level. Arm muscle circumference is equal to arm circumference in centimeters minus .314 times the triceps skin-fold in millimeters. Most of these authors use the standard values obtained by Geliffe in 1966.[30] Burgett has pointed out multiple inaccuracies in these standards as applied to the United States population[31] and suggests using contemporary standards,[32,33] which detail percentile versus age for both sexes. In a well-controlled study, he found Geliffe's standards high for the triceps skin-fold thickness of men and arm muscle circumference of women. The standards were significantly low for skin-fold thickness in women and arm circumference and arm muscle circumference in men.[31] The values presented as norms are seen in Table 1-1. Measured results can be compared with the standards and degrees of protein and fat loss estimated, since arm muscle circumference is an index of protein content and triceps skin fold an index of fat content. Values equal to 60 to 80 percent of the standards indicate moderate nutritional depletion, and values less than 60 percent of the standards represent severe malnutrition.

The creatinine-height index as applied to adults was described by Bistrian et al.,[34] and is an adaptation of a technique suggested by Viteri and Alvarado for children.[35] To derive the creatinine-height index,[27] the creatinine excreted per 24 hours on a creatinine and creatine-free diet is divided by the expected 24-hour urinary excretion of creatinine by a person of the same sex with an ideal weight for the patient's height (as determined by ideal height for weight charts), multiplied by 100

$$\text{Creatinine-height index} = \frac{\text{Actual urinary creatinine/24 hr (mg)}}{\text{Ideal urinary creatinine/24 hr (mg) for same height and sex}} \times 100 .$$

The standard values have been determined to be 23 mg creatinine per kilogram ideal body weight for men, and 18 mg creatinine per kilogram ideal body weight for women. The creatinine-height index was found to be a more sensitive parameter of nutritional status than weight for height, nitrogen balance, or serum albumin levels.[34]

Laboratory methods for measuring the visceral protein compartment are

Table 1-1. Anthropometric measurements standards from the literature

		Standards	
		Men	Women
Triceps skin-fold thickness (mm)	Burgett[31]	10.1	19.9
	Geliffe[30]	12.5	16.5
	Frisancho[33]	12.0	22
Arm circumference (cm)	Burgett	30.2	26.4
	Geliffe	29.3	28.5
	Frisancho	31.2	28.6
Arm muscle circumference (cm)	Burgett	26.9	20.1
	Geliffe	25.3	23.2
	Frisancho	27	21.6

primarily measurements of albumin and transferrin. The normal value for albumin is 4.5 g/dl; mild impairments are between 3.0 and 3.5 g/dl; moderate impairments are between 3.0 and 2.5 g/dl; and severe impairments are less than 2.5 g/dl. The half-life of albumin is 16 to 18 days; a more sensitive indicator in terms of time is serum transferrin, which has a half-life of .6 to 8 days. Transferrin can be determined directly at some hospitals, but more frequently, its concentration is derived from the total iron binding capacity. Serum transferrin is equal to the total iron binding capacity times 0.8 minus 43. Above 200 mg/dl is normal. A result of 200 to 180 mg/dl is a slight impairment; 180 to 160 mg/dl is a moderate impairment; and less than 160 ml/dl is a severe impairment.

Immune competence is assessed by the total lymphocyte count and reactivity to standard skin tests. Total lymphocyte count equals the percent of lymphocytes in a peripheral blood smear times the total white blood cell count. Normal is greater than 1700. A total lymphocyte count of 1700 is a mild impairment; 1000 a moderate impairment; and less than 800, a severe impairment.

Delayed hypersensitivity skin testing is done by the Sokal method.[36] Tests are positive when the diameter of induration is greater than 5 mm. The most commonly used recall antigens are mumps, candida, streptokinase/streptodornase, tricophyton mix, and purified protein derivative (PPD). A normal patient reacts to at least two of these. Relative anergy is defined as reaction to one antigen, and total anergy is reaction to none. The use of other visceral protein markers, such as prealbumin, retinol binding protein, and C_3, have been proposed but are not as widely accepted as the above listed parameters.[37] They do have shorter half-lives and may show responses to therapy more quickly than the standard visceral markers.

From tabulated results of the above tests, kwashiorkor (protein malnutrition in excess of calorie depletion) is diagnosed by observing decreased visceral protein stores with maintenance of somatic mass, as determined by anthropometric measurements. Low levels of total iron binding capacity, albumin, total lymphocyte count, and varying degrees of anergy are characteristic. In marasmus (significant caloric and protein malnutrition), anthropometric indices are reduced before visceral protein indices, but both are usually reduced.

Kaminsky et al.[38] correlated admission serum transferrin levels with hospital mortality and found that patients with levels less than 170 mg/dl had 2.5 times the mortality of those with higher levels. Meakins et al., in skin testing 354 surgical patients with PPD, mumps, candidin, trichophytin, and streptokinase/streptodornase (Varidase), found that mortality in patients abnormal at admission and who stayed abnormal through their hospital course was 74 percent, whereas those who were normal or improved their skin reactivity in serial testings had mortalities of only 2.1 percent.[39] Daly et al.,[40] in a study of 140 cancer patients, showed a 49 percent response to chemotherapy in patients with positive skin reactivity, compared to only 27 percent of anergic patients who responded to chemotherapy.

There is some evidence that nutritional support can reverse white cell dysfunction[41] and restore skin test reactivity.[41,42] Copeland et al.[43] found that

17 of 24 cancer patients receiving chemotherapy had negative skin tests initially, but 13 of these 17 patients had positive skin tests after an average of 11.4 ± 5.5 days of hyperalimentation. Harvey et al.,[44,45] in a study of 161 cancer patients, showed that 27 of 32 who were initially anergic became immunocompetent with nutritional therapy and had a mortality rate of 11 percent as compared to 100 percent in the 5 patients who remained anergic throughout their hospital stay.[45] Serum albumin and transferrin levels were used as parameters for nutritional assessment in both of these two studies and seem to correlate well with clinical courses.

Anthropometric parameters for individual patient analysis and prediction have been seriously questioned.[46,47] Shizgal et al.[46] compared weight to height, serum albumin, triceps skin fold, and creatinine to height index with more precise measurements of body composition determined by radioisotope dilution techniques in 216 patients. The best correlation was between weight to height and body cell mass ($r = 0.82$), and the poorest correlation was between creatinine height index and body cell mass. By means of a multiple linear regression analysis, the biochemical and anthropometric parameters were combined and correlated with the nutritional status as determined by body composition measurements. The resultant regression predicted malnutrition in 22 of the 69 normal subjects (false positive rate of 32 percent) and failed to predict the presence of malnutrition in 9 of the 79 malnourished patients (false negative rate of 11 percent).[47] Therefore, utilization of anthropomorphic parameters as predictors of morbidity and mortality for individual patients has questionable reliability.

More recently, a mathematical compilation of nutritional parameters into a prognostic nutritional index has been advocated.[48,49] Mullen et al. performed nutritional and immunologic assessments of 64 consecutive surgical patients, using the following parameters: weight loss, triceps skin fold, midarm circumference, creatinine-height index, serum albumin, serum transferrin, total lymphocyte count, serum complement, serum electrophoresis, lymphocyte and T-cell rosette formation, neutrophil migration, and delayed hypersensitivity. Ninety-seven percent of their patients had an abnormality in at least one of these measurements; 35 percent had abnormalities of at least three. Forty-six percent gave a history of greater than 0.2 percent weight loss per day from their usual weight. Approximately one-third of the patients had deficient triceps skin folds and midarm muscle circumferences. Creatinine-height index and delayed hypersensitivity were abnormal in 65 and 62 percent of the patients, respectively. They correlated these parameters to patient outcome in terms of operative mortality and morbidity; and it was determined that only the serum albumin, serum transferrin, and delayed hypersensitivity tests had significant correlation. Fifty-five percent of the patients with serum albumin levels less than 3 g/dl had postoperative complications 2.5 times the rate of those with albumin levels greater than 3 g/dl. Patients with serum transferrin levels below 220 mg/dl had five times the complication rate of patients with higher transferrin levels, with 41 percent of the patients with levels below 220 mg/dl having

complications. Thirty-three percent of patients anergic to all but one skin recall antigen had postoperative complications, compared to only 14 percent of those with normal skin tests.[47]

Buzby et al.[48] and Mullen et al.[50] developed their prognostic nutritional index by evaluating the strongest of these parameters in 161 surgical patients. The only parameters with predictive value were found to be serum albumin, serum transferrin, delayed hypersensitivity skin testing, and the single anthropometric measurement of triceps skin fold. (Of all the anthropometric data analyzed by Shizgal, triceps skin fold had the highest correlation with body composition, r = .79). When these variables were correlated with clinical course, an indicator of operative morbidity and mortality on the basis of nutritional status was developed. This prognostic nutritional indicator is as follows:

$$\begin{aligned}\text{Index} = {} & 158 - 16.6 \times \text{Serum albumin (mg/dl)} \\ & - 0.78 \times \text{Serum transferrin (mg/dl)} - 0.20 \times \text{Tricep skin fold (mm)} \\ & - 0.58 \times \text{Delayed hypersensitivity to mumps,} \\ & \quad \text{streptokinase/streptodornase, and candida} \\ & \quad \text{(graded 0 for nonreactors, 1 for <5 mm reactivity,} \\ & \quad \text{2 for } \geq 5 \text{ mm reactivity)}\end{aligned}$$

The greater the index, the greater the risk of complications. Patients with an index greater than 40 were judged to be at high risk.

The validity of this model was assessed prospectively in 100 patients undergoing major nonemergency gastrointestinal and thoracic surgery. A highly significant increase in the actual incidence of death ($p < 0.0007$), complications ($p < 0.0005$), sepsis ($p < 0.015$), and major sepsis ($p < 0.005$) was noted as predicted risk increased. Patients identified prospectively as high risk had a sixfold increase in mortality in relation to those identified as low risk.[48] Blackburn,[27] in his discussion of Buzby et al., has advocated the use of a less complex formula:

$$\begin{aligned}50\%\ \text{survival} = {} & [\text{Albumin (g/dl)} - \text{Delayed hypersensitivity} \\ & (1, \text{positive}; 2, \text{negative}) - 1.7] \times 100\end{aligned}$$

A patient with an albumin level less than 2.6 g/dl has less than a 5-percent chance of being immuncompetent, not becoming infected, and surviving; indeed, the 50/50 level is 3.2 g/dl.[48]

SUMMARY

Nutritional assessment in the surgical patient requires a determination of the degree of hypermetabolism caused by his disease state. This may be es-

timated by indirect calorimetry or by nitrogen balance determinations. Caloric needs for maintaining this requirement may then be computed. The degree of the patient's nutritional depletion at the time of initial presentation must also be determined so that candidates for preoperative hyperalimentation may be identified. In hypermetabolic patients an estimation may be made of the amount of additional substrate which must be provided above required maintenance levels to replete preexisting deficits. A variety of clinical and laboratory measurements to estimate nutritional status have been presented. The most useful of these are patient history and physical examination, delayed hypersensitivity skin tests, serum albumin, serum transferrin, and, perhaps, triceps skin-fold measurements. Routine analysis of these simple measurements in patients at apparent high-risk historically should help to define the patient group in which intensive nutritional support may decrease morbidity and mortality.

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