



# Surgical Nutrition

Edited by

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# Preface

This volume is an attempt to characterize the relatively new field of nutritional support in its current developmental phase. Since the demonstration by Dudrick, Rhoads, and coworkers of the feasibility of total parenteral nutrition using primarily glucose-protein hydrolysate mixtures administered into a central vein, the field of nutritional support has grown rapidly. The phase when the technique was popularized, roughly between 1968 and 1976 or 1977, manifested almost unabashed and probably, in retrospect, appropriate enthusiasm for what was justifiably seen as a new, important, and therapeutically life-saving technique. Acclaim was uncritical, and excesses, as with any new form of therapy, were common. Popularization substituted for careful scientific investigation and application of this powerful technique.

In a previous volume, *Total Parenteral Nutrition*, my coauthors and I attempted to summarize the knowledge as it existed until 1975. The emphasis in that volume was on the characterization of the field and its uses, and a rough appreciation of what the indications and complications were, but in general the book was a practical guide to a new and important therapeutic tool. Since that time, a necessary period of retrenchment, evaluation, and contemplation has taken place in the field of nutritional support. In the best sense, this period has consisted of an attempt to place the field of nutritional support on a more firm scientific basis by the somewhat overdue application of new research techniques and technology to the clinical and laboratory area. Thus, unabashed enthusiasm has been replaced by a more careful and balanced appreciation for such things as energy requirements, the question of caloric source, trace metals, and, above all, a concern for the requirement for the demonstration of efficacy.

To a certain extent, this current volume reflects the alteration in the field of nutritional support. It is entitled *Surgical Nutrition* because I am, in fact, a practicing surgeon and because, although the contributions of others to this field have been notable, it has been and largely remains an area of interest to surgeons since their patients are, by and large, the ones in most critical need of nutritional support. This is not to say that physicians would not benefit from a more critical and aggressive appraisal of their patients' nutritional needs, nor does it deny the need for the more



widespread teaching of nutritional requirements in medical school curricula, which still remains woefully lacking. Rather, it acknowledges that other disciplines have been somewhat less prone to adopt nutritional support as an essential part of their armamentarium.

This volume also reflects changing interests in the field of nutritional support. Growing appreciation for the importance of the preclinical sciences toward progress in clinical areas is reflected in the first several sections, including chapters on protein, carbohydrate, fat, vitamin, and trace metal metabolism, as well as other areas that are important, such as body composition, hormone and energy interchange, fluid and electrolytes, and methods of evaluation of efficacy. The growing appreciation that the enteral route may offer significant advantages, not the least of which may be in the immunologic area, is reflected in the inclusion of several chapters on enteral techniques emphasizing both the theoretical and practical applications of the enteral approach in normal and disease states. The preponderance of discussion of the importance of caloric source, be it glucose or fat, and the multiple references in this regard represent not careless editing, as the casual reader might suppose, but rather the growing importance of this debate within the area of nutritional support. To my count, there are no less than five individual statements of the proper ratios of calorie distribution between glucose and fat. All five differ in perspective and indicate both the depths of division and the breadth of interest in this particular topic. Other topics that may overlap are also included because of my feeling that open discussion and widespread debate of controversial points is not only necessary but desirable, and because in a field that is so immature, there must be free dis-

cussion rather than dogmatically held points of view.

Once again, I have been privileged to work with the members of the staff at Little, Brown. Many of them are friends in addition to collaborators, including Fred Belliveau, Vice President and General Manager of the Medical Division, and Lin Richter Paterson, Editor-in-Chief. Cynthia Baron has been invaluable in her position as Associate Book Editor for the volume. I would also like to acknowledge the friendship, collaboration, and inspiration of the many colleagues with whom I have had the privilege of working over the past decade or so, including Ronald Abel, John Ryan, Herbert Freund, Alfonso Aguirre, Bengt Jeppsson, Robert Bower, Kenneth Kern, Howard James, and Bill Chance, to name but a few. Rita Colley, Jean Wilson, and Vickie Duty are excellent representatives of our nursing staff, and Laura Matarese, our Dietitian, is represented in this volume. It has been my good fortune, over my professional career, to have been blessed with an excellent office staff, including Catherine Sullivan, Pat Walk, Kay Powell, Tricia Robertson, Shirley Davis, Marty Ridder, and Barbara Daria, all of whom have contributed to this volume.

Finally, and most importantly, one must not forget one's family, whose forbearance, support, and love make possible the tasks that are accomplished. *Surgical Nutrition* in every way belongs not only to my wife, Karen, but to my children, Erich and Alexandra, as well, who shoulder the burden of the inevitable intrusion upon our family life, yet manage to keep our relationship intact.

J. E. F.

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# **General Considerations**

**I**



# Body Composition

Harry M. Shizgal

## LEAN BODY MASS AND BODY FAT

Total body mass, or body weight (BWt), is composed of two major components: body fat and the lean body mass (LBM). The lean body mass was originally defined by densitometric measurements based on underwater weighing procedures. This methodology was based on the concept that the lean body mass was that component of body composition with a density of 1.100, whereas the neutral body fat had a density of 0.900 [1]. Thus, Behnke et al. [2], employing the diving tank of Bethesda, determined the LBM by measuring total body water (TBW) and specific gravity ( $d$ ).

$$\%LBM = 100 - \frac{213.66}{d} - 77.49 \frac{(TBW)}{BWt} - 137.4$$

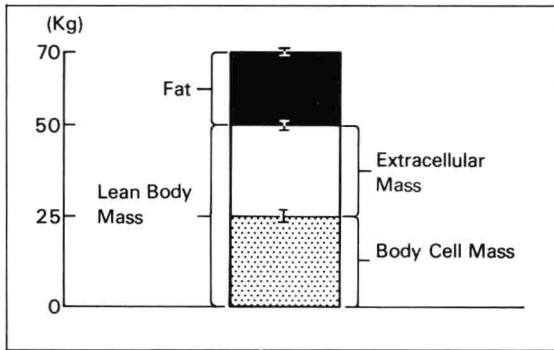
The lean body mass, as defined by densitometry, was originally regarded as containing between 2 and 10 percent essential lipids. Thus, conceptually there was a small difference between the lean body mass, as defined by densitometry, and the "fat-free body." The measurement of specific gravity by underwater weighing is difficult to perform, especially in the clinical setting. Currently, lean body mass is commonly determined by measuring total body water (TBW) and using the relationship

$$LBM = \frac{TBW}{0.73}$$

This relationship is based on the observations of Pace and Rathburn [8] that the water content of the fat-free body is 73 percent. The lean body mass, as defined by this relationship, is equivalent to the fat-free mass. Thus, body fat (BF) can be determined as follows:

$$BF = BWt - LBM$$

The calculations of body fat and lean body mass from the measurement of TBW are based on the assumption that the water content of the fat-free mass is constant. The hydration of the fat-free mass is fairly constant in a healthy homogeneous population, but varies considerably with illness. The water content of the fat-free body has been estimated to vary from a maximum of 85 percent with severe anasarca to a minimum of



**Figure 1-1.** The mean ( $\pm$  SEM) body composition of 25 normal volunteers. Body weight is the sum of body fat and the lean body mass, which in turn is subdivided into two compartments: the extracellular mass and the body cell mass.

67 percent with marked dehydration. The Pace-Rathburn formula thus results in a first approximation of body fat and lean body mass.

Moore et al. [7] developed a nomogram to calculate the hydration of the fat-free body and thus improve the precision of the body fat and lean body mass determination. The ratio of intracellular water volume to total body water (ICW/TBW) and the ratio of observed to expected extracellular water (ECW) are the indices that are used to determine the hydration of the fat-free body. Although this nomogram was described by Moore et al., they have seldom used it to calculate either body fat or LBM, but have instead employed the Pace-Rathburn formula.

In a healthy and homogeneous population, a relationship exists between the lean body mass and metabolic indexes such as oxygen consumption, carbon dioxide production, caloric requirement, and work performance. However, such a relationship does not exist with heterogeneous populations or in the presence of pathologic states. This is because the lean body mass is anatomically and metabolically heterogeneous, being composed of both the body cell mass and the extracellular mass (Figure 1-1). The latter represents the extracellular supporting component of body composition and is composed of such composition elements as the skeleton, carti-

lage, fascia, tendons, plasma volume, extracellular fluids, and pathologic water accumulations. These components of the body are not metabolically active, do not consume oxygen, and do not perform work. Rather, they are largely concerned with transport and support.

## BODY CELL MASS

The body cell mass is the metabolically active, energy-exchanging mass of the body. The body cell mass was defined by Moore et al. [7] "as that component of body composition containing the oxygen-exchanging, potassium-rich, glucose-oxidizing, work-performing tissue." It is therefore that component of body composition that can serve as a reference for the metabolic activity of the body as measured by oxygen consumption, carbon dioxide production, caloric requirement, and work performance. The body cell mass is the sum of all the cellular elements of the body and thus includes the cellular components of both skeletal and smooth muscle, the viscera (liver, kidneys, lungs, gastrointestinal tract), and the central nervous system. Also included are the cells of tissues with a sparse cellular population such as bone, cartilage, tendon, and adipose tissue. In the normal healthy individual, the skeletal muscle cell mass has been estimated to comprise 60 percent of the total cell mass, whereas the visceral cell mass accounts for 20 percent. The remaining 20 percent is made up by the red cell mass and the cells in the peripheral connective tissues such as bone, cartilage, tendon, adipose, and so forth [6].

A direct measure of the body cell mass is currently unavailable. However, the size of the body cell mass can be estimated by a variety of indirect measurements. The approach most commonly employed is the measurement of total body potassium. Potassium is the most abundant intracellular cation, with over 98 percent of total body potassium located within the intracellular compartment. Furthermore, the intracellular potassium concentration varies within a narrow range. As a result, total body potassium

is linearly related to the size of the body cell mass [7]. In addition, total body potassium is linearly related to both the intracellular water volume [7] and to total body nitrogen [3]. The ratio of potassium to nitrogen is fairly constant in all tissues in both normal and pathologic states [13, 14]. Kinney et al. have also demonstrated a relationship between body potassium and resting metabolic expenditure [4]. Considerable data therefore exist supporting the use of body potassium to estimate the size of the body cell mass.

Total exchangeable potassium ( $K_e$ ), as measured by isotope dilution with an isotope of potassium, is equivalent to total body potassium. Several studies have reported differences between  $K_e$  and total body potassium. However, in each instance these differences were extremely small, statistically insignificant, and less than the measurement errors of the techniques employed. The most precise and accurate measurement of  $K_e$  is obtained by isotope dilution using a radioactive isotope of potassium. However, the radioactive isotopes of potassium that are currently available are relatively unstable and decay rapidly. The half-life of  $^{42}\text{K}$  is 12.5 hours. As a result, 86.6 percent of the available  $^{42}\text{K}$  decays in the initial 36 hours. By three days only 1.7 percent remains. This short half-life is not a problem if the  $K_e$  measurement can be planned to coincide with the delivery of the isotope. However, with clinical studies, this is often difficult because the availability of patients is usually unpredictable.

To overcome the difficulties and expense associated with the use of the rapidly decaying radioactive isotopes of potassium, a technique for the indirect measurement of  $K_e$  was developed. This technique is based on the fact that in all tissues, except for bone, the ratio of the sodium plus potassium content divided by the water content is a constant and is equal to the ratio of the sum of exchangeable sodium ( $\text{Na}_e$ ) plus  $K_e$  divided by TBW, i.e.,

$$\frac{\text{Na}_e + K_e}{\text{TBW}} = \frac{\text{Na} + \text{K}}{\text{H}_2\text{O}} = R$$

Thus,

$$K_e = R \times \text{TBW} - \text{Na}_e$$

Total body water and  $\text{Na}_e$  are easily measured by standard isotope dilution using tritiated water and  $^{22}\text{Na}$ , respectively. The constant,  $R$ , is determined by measuring the sodium, potassium, and water content of a sample of whole blood. The validity of this measurement was experimentally verified both in the experimental animal and in a group of patients [12]. The indirect measurement of  $K_e$  was identical to the measurement obtained simultaneously by either carcass analysis or by  $^{42}\text{K}$  dilution.

Total body potassium also can be measured by means of a whole body counter. The whole body counter measures the total mass of  $^{40}\text{K}$ , a naturally occurring radioactive isotope of potassium which comprises 0.012 percent of all naturally occurring potassium. The whole body counter is an expensive installation. In addition, its calibration remains a difficult problem, since the  $^{40}\text{K}$  count detected by the whole body counter is a function of both total body potassium and the geometric configuration of the subject. The geometric configuration of the individual determines the degree of self-absorption of the emitted gamma rays and thus the percentage of emitted  $^{40}\text{K}$  gamma rays detected. In the majority of installations, the body counter is calibrated by injecting  $^{42}\text{K}$  into a group of volunteers and measuring  $^{42}\text{K}$  with the whole body counter. Potassium-42 is used, as it emits a gamma ray with an energy similar to the gamma emitted by  $^{40}\text{K}$ . The counting efficiency (i.e., counts detected/counts injected) is correlated with weight and/or height. The resultant regression is then used to determine the counting efficiency arising from the geometry of the individual and thus correct the  $^{40}\text{K}$  count. The error introduced by the use of this regression may be considerable, especially since the background count is usually large relative to the  $^{40}\text{K}$  count. With some total body counters the background count may also be a function of body weight. Thus, although the reproducibility of the whole body potassium de-



termination is usually excellent, the accuracy is often poor because of the low  $^{40}\text{K}$  count, the large background count, and the difficulty associated with determining the  $^{40}\text{K}$  counting efficiency. The low  $^{40}\text{K}$  counts also necessitate long counting times, which may pose a problem with ill patients.

The body cell mass (BCM) can be calculated from  $K_e$  based on the assumption that the average potassium to nitrogen ratio in tissue is 3 mEq/gm. Thus,

$$\text{Total body nitrogen} = \frac{K_e}{3} \text{ (gm)}$$

$$\text{Total body protein} = \frac{6.25}{3} K_e \text{ (gm)}$$

$$\text{Body cell mass} = \frac{4 \times 6.25}{3} K_e = 8.33 K_e \text{ (gm)}$$

Because the BCM is related to  $K_e$  by a simple constant, the calculation of the BCM results in an approximation of its absolute size, but does not yield additional compositional information.

The body cell mass can also be calculated from the intracellular water volume (ICW), by assuming that the average water content of the body cell mass is 70 percent, i.e.,

$$\text{BCM} = \frac{\text{ICW}}{0.70}$$

This approach is much less accurate, since ICW cannot be measured directly, but is calculated as the difference between TBW and the extracellular water volume (ECW). The accuracy of ICW determination is therefore dependent on the errors associated with both the TBW and ECW measurements. The accuracy of the TBW measurement is excellent, since the TBW space is well defined anatomically and is measured by a tracer, usually tritiated water, which does not "leak" out of the TBW space. The equilibrated concentration of the tracer is used to calculate the TBW volume. In contrast, the majority of the tracers used to measure the ECW space leak out

of the ECW at varying rates. The volume is therefore calculated from the kinetics of the disappearance curve, as defined by the slope and the intercept. The ECW volumes obtained with the larger and less diffusible tracers, such as mannitol and insulin, are smaller than those obtained with the ions, such as sodium, bromide, and sulfate. The ECW volume is in part, therefore, a function of the distribution volume of the tracer used. Since the ECW is not well defined anatomically, biologic validation of an ECW measurement is impossible. The inaccuracies of the ICW measurement, therefore, result from the difficulties associated with the ECW determination and not from the TBW measurement. As a result, the ICW is seldom used to calculate the body cell mass.

The body cell mass can also be calculated from total body nitrogen (TBN) from

$$\text{BCM} = \text{TBN} \times 6.25 \times 4$$

Because body nitrogen does not exist in well-defined "pools," TBN cannot be measured by isotope dilution. However, neutron activation analysis has been used to measure TBN. This involves the irradiation of the subject with neutrons from either a cyclotron or from a plutonium source [3, 5]. When any material is irradiated with neutrons, nuclear changes result, with the production of gamma rays specific for the substance that captured the neutron. In the case of nitrogen a 10.83 Mev gamma ray is produced. By using large sodium iodine crystal detectors, a measure of TBN can be obtained. In one study involving 190 measurements in 65 patients, an excellent correlation was obtained between TBN and  $K_e$  [3].

## EXTRACELLULAR MASS

The extracellular mass (ECM) is that component of the fat-free mass which exists outside the cells and is composed of both fluids and solids. The fluid component of the extracellular mass consists of plasma, and interstitial and transcellu-