

# TECHNIQUES AND PROCEDURES

## Body Composition and Nutrition Support in Pediatrics: What to Defend and How Soon to Begin

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**ABSTRACT:** Specialized nutrition support is initiated during hospital care on the assumption that sparing the mobilization of body energy reserves is advantageous to recovery. The combined effects of disease and undernutrition on body cell mass, organ function, immune responsiveness, and wound healing are well documented in adults. Children cannot survive a fast as long as adults because of their lesser stores of energy substrates relative to the rate of energy expenditure. The present contribution attempts to estimate the rates of compositional losses for infants and prepubertal children on the basis of available data and reasonable metabolic assumptions. The lesson that emerges from this exercise is one of a very critical need for the early initiation of nutrition support for infants and children. The analysis suggests that an acute risk of protein depletion exists for children of all ages. Especially for infants, the empirical wisdom that "the absence of evidence is not evidence of absence" should be invoked to support early nutrition intervention. This work is submitted for critical review and revision to establish a consensus on the timeline of pediatric morbidity or mortality from semistarvation or starvation.

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Specialized nutrition support is initiated during hospital care on the assumption that sparing the mobilization of body energy reserves is advantageous to recovery. The recent A.S.P.E.N. Guidelines<sup>1</sup> update previous advice for the clinical management of adult patients. These Guidelines also

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provide, for the first time, a comprehensive clinical practice document for pediatric nutrition support (Sections V to VIII). The strength of the evidence supporting each recommended practice is stated. Each pediatric guideline is supported by literature specific to infants or children; citations from the adult literature are not carried over for support. Thus the pediatric sections also serve to highlight, by weak or absent evidence, areas where additional knowledge is needed. How soon to initiate nutrition support to prevent the sequelae of malnutrition is one such area (reference 1, Section V).

The combined effects of disease and undernutrition on body cell mass, organ function, immune responsiveness, and wound healing are well documented in adults. Functional consequences are expected after 2 weeks of semistarvation, and death from absolute starvation occurs within 60 to 70 days.<sup>2,3</sup> This may be an overly generous timeline; 40 days is perhaps a more appropriate cutoff for a reasonable expectation of survival.<sup>4</sup> In any case, nutrition support is recommended when 7 days of inadequate nutritional intake have elapsed (reference 1, Section II).

For children, malnutrition frequently accompanies disease.<sup>1,5,6</sup> No timeline corresponding to that for the adult sequelae is provided in the pediatric rationale (reference 1, Section V). Holliday<sup>7</sup> notes that fasting is not a lack of energy substrates *per se*, but is a condition where body fat and muscle protein, rather than food, provide the energy substrates. He further notes that children cannot survive a fast as long as adults because of lesser stores of energy substrates relative to the rate of energy expenditure. A closer examination of this latter premise suggests that limitations on survival include an acute risk of protein depletion for children of all ages. Beyond infancy, surviving chronic undernutri-

tion of significant degree is further threatened by limited fat reserves. The present contribution attempts to estimate the rates of compositional losses that lead to physiologic dysfunction or fatality for infants and prepubertal children on the basis of available data and reasonable metabolic assumptions. The analysis is submitted for critical review and revision toward establishing a consensus on the timeline of pediatric morbidity or mortality from semistarvation or starvation. The author fully recognizes that substantial individual variability exists and, therefore, presents "textbook case scenarios" for consideration.

#### BODY COMPOSITION AND ENERGY RESERVES IN CHILDREN

Potential body energy stores in infants and children, as in adults, consist of fat and protein. Triglyceride stores are virtually synonymous with the fat mass (FM) whereas the total body protein is variably dispersed among the tissues comprising the fat free mass (FFM). The available body energy stores are those fractions of energy stores that can be mobilized for catabolism; ie, the nonesterified fatty acids (loosely bound to plasma albumin for transport) and glycerol released from the FM stores and the amino acids liberated from the so-called "labile" portion of total body protein. The mobilization of protein from the FFM to meet the demands of gluconeogenesis is not uniform and preferentially depletes the subcompartment of skeletal muscle mass as is evidenced by the classic "wasting" phenomenon of starvation.

The sizes of the fat and skeletal muscle compartments in healthy infants and prepubertal children have not been catalogued for convenient reference. To that end, Table 1 and Figure 1 are presented. These models are derived by collapsing reliable data<sup>8</sup> into age groupings as single sex profiles. The skeletal muscle mass (SMM) is independently estimated using the body weight ranges together with published norms for creatinine excretion.<sup>9</sup> Adult reference data<sup>10,11</sup> are shown for comparison.

The fourth column in Table 1 lists the SMM in absolute wet weight. The specific protein content (SM Prot) is estimated as 22% of that weight.<sup>12</sup> SM Prot is listed separately in the final column of the table to illustrate the proportion of the total body protein that is housed in the SMM subcompartment of the FFM. The remaining body protein resides in nonmuscle organs and tissues, including skeletal structures. The time course for the mobilization of SM Prot relative to other available protein stores to satisfy gluconeogenic demands is unknown, but no sparing of SM Prot appears to occur on empirical grounds. The compartments shown in Figure 1 correspond to a four-compartment scheme in which the nonmuscle and skeletal compartments within the FFM could also be separated.<sup>7,11</sup> They are combined in the present figure for simplicity. The point here is to emphasize the relative proportions of fat and muscle in healthy children at differing ages.

The expected normal body composition has been recast in Figure 2 to illustrate the reserves of energy consistent with the analysis that follows in sections II and III. Most of the triglyceride stored within the fat mass is considered as available. The "available protein" is dispersed throughout the FFM. The "Noncaloric Mass," ie, the remainder of the FFM that cannot provide energy is comprised of about half of the total protein (see below) plus water and minerals. The very limited stores of glycogen have been ignored for simplicity.

These compositional and energetic estimates represent the "healthy normal" starting points. Under-nutrition before hospitalization, whether acute or chronic, will deplete compartment sizes and a portion of the available energy stores. The fat mass and the SMM are useful body compartments that can be readily measured clinically in pediatric practice to track the erosion of available energy stores in individual cases. The following analysis will show that (1) muscle wasting may signal a far greater overall protein depletion in infants and toddlers and (2) that fat reserves are less appreciable in older children compared with adults.

Table 1. Body composition of healthy children: "What to defend" with nutritional support

Age (years)	Body weight (kg)	Fat mass (kg)	Skeletal muscle mass (kg)	Total body protein (kg)	Skeletal muscle protein (kg)
0.5	7-8	2.0	1.5-1.8	0.9-1.0	0.3-0.4
1	9-10	2.2	2-2.3	1.2-1.3	0.4-0.5
2-3	12-15	2.5	3-4.5	1.7-2.2	0.6-0.9
4-5	16-19	2.5-3.0	5.5-6.7	2.4-3.0	1.1-1.4
6-7	20-23	3.0-4.0	7-8	3.3-3.8	1.4-1.6
8-10	25-33	3.5-6.5	9-12.2	3.8-5.5	1.8-2.5
Adult	50-70	14	17-28	10-13	3.8-6.2

Data grouped and rounded; weight, fat and total protein from Foman et al<sup>8</sup>; skeletal muscle estimated from weight and creatinine excretion Holliday<sup>9</sup>; and adult data from Blackburn et al<sup>10</sup> and Cunningham.<sup>11</sup>

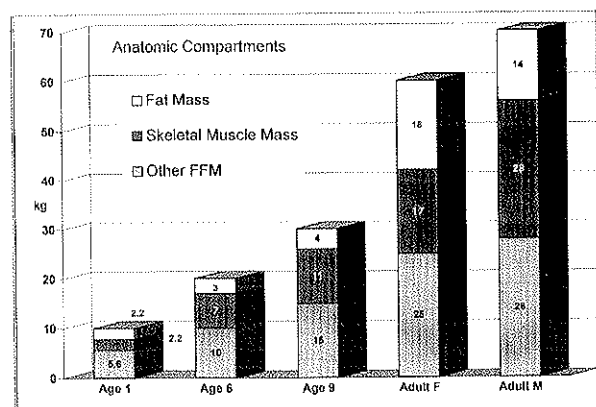


Figure 1. Approximations of anatomic compartment sizes within the body composition of infants, prepubertal children, and adults. Data from Table 1 and references 8 through 11.

#### COMPONENTS OF MINIMAL ENERGY EXPENDITURE

Two general assumptions regarding energy expenditure are required to predict the rate of depletion of energy reserves. First, a minimal energy expenditure, represented by the predicted normal basal metabolic rate (BMR), is assumed. Second, an estimate of the specific requirement for glucose is needed because this drives the requirement for protein catabolism. These two assumptions permit an estimation of a maximal energy reserve capacity from a starting point of normal body composition as above. This scenario is then impacted by the magnitude of increase in the actual metabolic energy expenditure (MEE) above the BMR for activity, healing, or hypermetabolic states as well as by any depletion of energy stores before illness or injury.

Estimates for BMR as published by the World Health Organization<sup>13</sup> and by the Federation of American Societies for Experimental Biology<sup>14</sup> range from 50 to 60 kcal/kg for children of various ages. Measured energy expenditures of sick and low-birth-weight newborns requiring nutrition support<sup>15-17</sup> also fall within this range. Table 2 lists the BMR ranges corresponding to the age groupings in Table 1. The obligatory need for glucose is shown to be a substantial component of the BMR in Table 2. It is based on the data of Holliday<sup>7</sup> for gluconeogenesis during fasting.

#### THE EFFECTS OF STARVATION ON ENERGY STORES

*Protein Wasting.* Any deficit in glucose triggers obligatory nitrogen wasting. The minimal rate of loss during starvation can be calculated as the mobilization of available protein for gluconeogenesis to

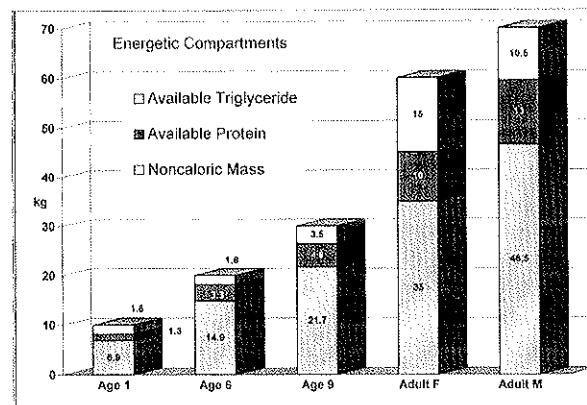


Figure 2. Approximations of Energetic Compartments available for catabolism in infants, prepubertal children, and adults.

meet the glucose requirements listed in Table 2. Two assumptions are made: first, that gluconeogenesis is only approximately 60% efficient at producing 3-carbon skeletons for glucose;<sup>20</sup> and, second, as noted, that only about half of the total body protein is available for mobilization, based on adult data.<sup>18,19</sup> Table 2 estimates the survival time during absolute starvation as the number of glucose days that are available from protein catabolism. The model seems reasonable because the end point for adults is 70 days, which agrees with the A.S.P.E.N. timeline.<sup>1</sup>

The accelerated rate of protein depletion in starving pediatric patients (Table 2) is striking, but not well appreciated. Infants are shown to be in imminent danger during any sustained period of fasting because of the very high glucose demands of their nervous system.<sup>7</sup> Protein wasting in these infants, as well as that in children younger than 3 years, could be clinically difficult to document given that the SMM comprises a smaller proportion of their anatomic composition (Table 1, Figure 1). Children ages 3 to 10 years remain at very high risk, but wasting should be more easily detected in these children because their SMM is larger and skeletal muscle protein constitutes half of their total body protein (Table 1). The assessment of SMM should give early evidence of their wasting.

One basic tenant for pediatric clinical care becomes evident in this analysis. The capacity to survive, estimated in glucose days (ie, the availability of gluconeogenic protein stores to cover obligate glucose needs) will be lengthened in acute or chronic undernutrition in proportion to the provision of glucose equivalents (eg, glucose or its precursors such as glycerol, lactate, or gluconeogenic amino acids) by nutrition support. Electrolyte replacement fluids that contain dextrose should be properly viewed as one step in nutrition support because some of the

demand for glucose equivalents is met exogenously, and the capacity to survive is prolonged by offsetting protein catabolism. Unfortunately, several constraints impact on an exact estimation of the magnitude of "protein sparing" by exogenous carbohydrates. The constant infusion of dextrose signals a "fed" metabolic state in which fatty acid mobilization is dampened by circulating insulin. As a result, much of the plasma glucose could satisfy facultative organ energy needs rather than obligate glucose demands. The degree of insulin resistance would regulate this partitioning. In addition, much of the intermediary metabolism of healing appears to utilize glucose as a preferential fuel substrate. Finally, the loss of SMM secondary to disuse atrophy and bed rest will liberate amino acids for catabolism despite the provision of exogenous substrates.

The range of protein sparing to be expected from the delivery of glucose or glucose equivalents can be somewhat approximated using the equations below. A minimum sparing involves all tissues equally utilizing this exogenous glucose (Eq 1). A maximum sparing occurs when only the normal glucose requirement (Table 2) plus the energy expended for healing are competing for the delivered glucose equivalents (Eq 2). A more exact clinical judgement for an individual case would require some knowledge of substrate partitioning. Measurement and interpretation of the respiratory quotient<sup>19</sup> or the plasma level of nonesterified fatty acids would be informative. Values reflecting the fed state, RQ > 0.9 or FFA < 0.3 mmol, would signal that equation 1 is more appropriate:

#### Minimum sparing

$$= (\%BMR \text{ as Glucose}/MEE) \times \text{Delivered grams} \quad (1)$$

where %BMR as Glucose is from Table 2 and MEE is the actual metabolic energy expenditure expressed as a ratio of the normal BMR in Table 2.

#### Maximum sparing

$$= \text{delivered grams} - [(MEE - BMR)/3.7] \quad (2)$$

where (MEE - BMR) quantifies the additional kilocalories for healing that are fueled by glucose as monohydrate at 3.7 kcal/g.

The impact of feeding on protein reserves is then approximated as follows:

#### Net glucose requirement

$$= \text{grams glucose required} - (\text{Min to Max})$$

and

#### Protein reserve (glucose days)

$$= \text{Prot (Mcal)}/[\text{net glucose required} \times 0.0067]$$

For example, if 40 g/d of dextrose were given to a 6-mo-old infant whose MEE was  $1.2 \times BMR$ , then minimum sparing (Eq 1) =  $50/1.2 \times 40 = 17$  g/d and the maximum sparing (Eq 2) =  $40 - (480 \text{ kcal} - 400 \text{ kcal})/4 = 20$  g/d. The net glucose required remains high at 30 to 33 g/d (ie, = grams glucose required - [17 to 20]) and the protein reserve is extended only to 8.5 to 9 days (=  $1.9/[30 \text{ or } 33 \times 0.0067]$ ). Clearly, this level of nutrition management is only slightly less devastating than starvation.

The timeline for mortality from starvation that results from the present analysis calls for close attention in pediatric practice, especially because a best-case scenario beginning with normal body composition has been assumed. In all cases, the adult model fails as a reasonable yardstick of appropriate risk for pediatric practice. Undernutrition depletes body protein stores much more rapidly in infants and children. The initiation of significant nitrogen-sparing nutrition support is shown to be an immediate and continuous component of quality care in pediatrics.

*Fat Catabolism.* The depletion of body fat reserves is less life-threatening than the sequelae of nitrogen wasting in adults. It is often assumed that the limited fat stores of infants and young children will place them at a relatively greater risk. Again, no quantitative guidelines are available. The data in Table 1 are used to derive estimations of the rates of fat depletion shown in Table 2 when reasonable assumptions are applied. First, it is assumed that the available fat stores are those above a critical minimal body fat. A conservative, but arbitrary, minimum of 5% of body weight was chosen for the present model. Second, gluconeogenesis provides some offsetting as fatty acid equivalents from the 2-carbon de-aminated skeletons of the estimated 40% of the amino acids liberated but not useable for gluconeogenesis. Third, it is assumed that all of the BMR that is not satisfied by gluconeogenesis must be fueled by fat mobilization, either as fatty acids, glycerol, or ketone bodies. The loss of ketones through breath and urine is ignored in the present model, but would serve to lower the survival estimates to some degree.

Application of these three assumptions to the data in Table 1 yields the estimates of the available fat stores and the corresponding "kcal days" of survival time catalogued in the final two columns of Table 2.

Some perhaps surprising observations result from the analysis. The model again appears to be valid when judged by expectations for adult males.<sup>1</sup> However, adult females are shown to be favored for survival. This is readily understandable when their lower BMR per unit body weight and their larger fat mass per unit of body weight (Fig 1) are considered. Infants through 1 year of age are also favored for survival, rather than at a heightened risk as is often presumed. This follows from their high glucose re-

Table 2. Body energy reserves of the healthy child: "How soon to begin" with nutritional support

Age (years)	BMR (kcal)	Glucose required		Protein reserve		Available fat	
		g/d	%BMR	Mcal	Glucose days	Mcal	kcal days
0.5	350-450	50	50	1.9	6	14-15	105-110
1	500-550	75	60	2.5	5	15-15.8	180-190
2-3	675-875	85	45	3.5-4.5	6-8	16-17.1	63-52
4-5	850-950	95	40	4.5-6	7-10	15-18.5	41-44
6-7	950-1025	100	40	6.5-7.5	10-12	18-25.7	43-57
8-10	1050-1250	110	20	7.5-11	10-15	20-43	26-47
Adult	1400-1800	70-90	20	20-25	70	95-135	70-150

1 Mcal = 1,000 kcal; original source data grouped and rounded; BMR estimated from reference 13; glucose requirement for the brain from reference 7; adult glucose requirement as 20% of BMR; available protein reserve is assumed to be 50% of total body protein from Table 1 with its utilization assumed to provide 60% glucose and 40% acetate; glucose days provide all of the glucose requirement via gluconeogenesis at 60% efficiency (references 19, 20); kcal days calculated as providing the remainder of BMR until body fat is reduced to 5% of body weight.

quirement, 50% to 60% of BMR and the spill-over of fatty acid equivalents from protein catabolism during gluconeogenesis. Fat need only provide a net 15% to 30% of the BMR during a fast. The mobilization of essential fatty acids from fat stores will, therefore, be slower in starving infants, and this may present an independent risk for deficiency.

Children ages 3 to 10 years are shown to be at an extreme risk for fat depletion. The survival time estimated from fat stores abruptly falls after age 2 years. This heightened fat catabolism combined with the accelerated loss of available TB protein places children at the highest overall risk from starvation. At ages 3 to 10 years the proportion of TB Prot that is SM Prot is also much increased (Table 1). Children are often presumed to be at lessening risk with increasing age, but the opposite is revealed by the energy balance analysis.

#### SUMMARY

The lesson that emerges from this exercise is one of a very critical need for the early initiation of nutrition support for infants and children. Tissue wasting to meet energetic demands proceeds much more rapidly in children of all ages than for adults. Intervention can, and should, be justified purely on the metabolic grounds illustrated herein.

For toddlers and older children, anthropometric assessment techniques can serve to accurately document the degree of depletion of the fat mass and the skeletal muscle mass when compared with compositional expectations provided in Table 1. Indeed, marked weight loss itself would be an indicator of significant compositional losses among these children.

However, for infants, the lesser proportion of total body protein (TB Prot) residing in skeletal muscle protein (SM Prot) together with a lesser ability to mobilize fat stores to supply energy demands will

serve to mask ongoing and potentially devastating effects of undernutrition on the TB Prot pool when these assessments are used. For wasting in infants then, the empirical wisdom that "the absence of evidence is not evidence of absence" can be invoked to begin early nutrition intervention.

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