

# EFFECT OF NUTRITIONAL SUPPORT ON ROUTINE NUTRITION ASSESSMENT PARAMETERS AND BODY COMPOSITION IN INTENSIVE CARE UNIT PATIENTS

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**OBJECTIVES:** To determine whether routine nutrition assessment parameters and body composition change after nutritional support in intensive care unit (ICU) patients and whether the changes, if any, are related to cumulative energy and fluid balances.

**DESIGN:** A prospective study.

**SETTING:** A university teaching hospital.

**PATIENTS:** Forty-five mechanically ventilated medical and surgical patients admitted to the ICU who received nutritional support for 7 days (group 1) and 9 patients of this group who received nutritional support for 3 weeks or longer (group 2).

**INTERVENTIONS:** Enteral and parenteral nutritional support prescribed on the basis of metabolic cart measurements of energy expenditure.

**OUTCOME MEASURES:** Routine nutrition assessment, including determinations of weight, serum albumin and prealbumin, and lymphocyte count and body composition, including measurements of body cell mass, extracellular fluid and body fat, determined from bioelectric impedance analysis.

**RESULTS:** In group 1 patients, weight, albumin and prealbumin levels, and extracellular mass changed, but there was no change in lymphocyte count, body cell mass or body fat. Changes in weight and extracellular mass were slightly related to cumulative fluid balance; changes in albumin and prealbumin levels were not related to cumulative energy or fluid balance. The findings were similar for group 2 patients.

**CONCLUSIONS:** Changes in routine nutrition assessment parameters and body composition are slightly affected by fluid balance but not by energy balance; thus, they are not specific indicators of the adequacy of nutritional support in ICU patients. Improved nutrition assessment parameters are required to better monitor the response to nutritional support in critically ill patients.

**OBJECTIFS :** Déterminer si les paramètres d'évaluation de l'alimentation de routine et la composition du corps changent après une alimentation de soutien chez des patients aux soins intensifs et si les changements, s'il en est, sont liés aux bilans énergétique et hydrique cumulatifs.

**CONCEPTION :** Étude prospective.

**CONTEXTE :** Hôpital d'enseignement universitaire.

**PATIENTS :** Quarante-cinq patients en médecine et en chirurgie ventilés mécaniquement et admis aux soins intensifs qui ont reçu une alimentation de soutien pendant 7 jours (groupe 1) et 9 patients de ce groupe qui ont reçu une alimentation de soutien pendant 3 semaines ou plus (groupe 2).

**INTERVENTIONS :** Alimentation de soutien entérale et parentérale prescrite en fonction de mesures métaboliques de la dépense d'énergie.

**MESURES DES RÉSULTATS :** Évaluation de l'alimentation de routine, y compris calcul du poids, albumine sérique, préalbumine et numération lymphocytaire, et composition du corps, y compris mesures de la

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masse cellulaire corporelle, des fluides extracellulaires et du taux d'adiposité corporelle, établis à partir d'une analyse d'impédance bioélectrique.

**RÉSULTATS :** Chez les patients du groupe 1, le poids, les taux d'albumine et de préalbumine et la masse extracellulaire ont changé, mais la numération lymphocytaire, la masse cellulaire corporelle ou le taux d'adiposité corporelle n'ont pas changé. On a établi un lien ténu entre les changements de poids et de masse corporelle et le bilan hydrique cumulatif; on n'a établi aucun lien entre les changements des taux d'albumine et de préalbumine et le bilan énergétique ou hydrique cumulatif. Les résultats ont été semblables chez les patients du groupe 2.

**CONCLUSIONS :** Les changements des paramètres de l'évaluation de l'alimentation de routine et de la composition du corps sont affectés légèrement par le bilan hydrique, mais non par le bilan énergétique. C'est pourquoi ils ne constituent pas des indicateurs spécifiques de la suffisance de l'alimentation de soutien chez les patients aux soins intensifs. Il faut améliorer les paramètres d'évaluation de l'alimentation afin de mieux surveiller la réaction à l'alimentation de soutien chez les patients en phase critique.

**P**atients admitted to the intensive care unit (ICU) usually receive nutritional support after full resuscitation and cardiopulmonary stability have been achieved. Energy intake, protein, water, electrolytes, minerals and trace elements are prescribed according to estimates of metabolic requirement.

Once nutritional support is started in general ward patients, routine nutrition assessment includes measurements of anthropometric features (weight, triceps, skin-fold thickness and mid-arm muscle circumference), "visceral" protein levels (albumin, prealbumin and transferrin) and immune function (cutaneous reactivity to recall antigens and lymphocyte count). Increases in these parameters as well as accretion of nitrogen (positive nitrogen balance) indicate adequacy of nutritional support.

In ICU patients, however, these routine measurements have limited usefulness because they are technically difficult to perform and because factors other than the adequacy of nutritional support influence the variable. For example, determination of nitrogen balance by 24-hour collection of body fluids for urea can be difficult if the patient has massive diuresis or diarrhea or is anuric. Also, because tissue edema is common in ICU patients, anthropometric measurements are inadequate indicators of response to nutritional support. Similarly, "vis-

ceral" protein levels and measures of immune function are affected not only by nutritional support but also by large fluid intake for resuscitation and by a generalized inflammatory state. Therefore, other methods of assessing nutritional support are required for these patients.

Body-composition analysis is an investigational method of assessing nutritional status and response to nutritional support. Neutron activation and tritium dilution space have been used to evaluate total body nitrogen and total body water.<sup>1</sup> Since potassium is confined to the intracellular space, body cell mass has been estimated by measuring exchangeable potassium by radioisotope dilution of potassium-42.<sup>2</sup> These methods of body-composition analysis are not widely available. Body composition has more recently been determined from bioelectric impedance analysis with the use of a commercially available system (RJL Systems Inc., Detroit), which provides a convenient and painless bedside estimate of lean body mass and body fat.<sup>3,4</sup> Joidin, Trott and Shizgal<sup>5</sup> and Shizgal<sup>6</sup> further refined body-composition analysis by using bioelectric impedance methods to three compartments: body cell mass, extracellular mass and body fat. These formulations consider extracellular mass separately, since Shizgal included edematous patients in his studies of postoperative and septic patients. We used bioelectric im-

pedance analysis and Shizgal's formulations to determine body composition.

In our study we investigated the utility of serial determinations of routine nutritional parameters and body-composition analysis to assess nutritional support in ICU patients. We asked two specific questions: first, do the nutrition assessment variables, weight, albumin, prealbumin, lymphocyte count, body cell mass, extracellular mass and body fat, change after 7 days or after 3 weeks of nutritional support in ICU patients? Second, are changes in the nutrition assessment variables related to cumulative energy balance or to cumulative fluid balance?

Ethics approval was given by St. Paul's Hospital and the University of British Columbia. The study was conducted in accordance with the principles for human experimentation as defined in the Declaration of Helsinki.<sup>7</sup>

## METHODS

### Patients

We studied 45 intubated, mechanically ventilated medical and surgical patients admitted to the ICU of St Paul's Hospital, Vancouver, between January 1991 and February 1992, who received at least 7 days of nutritional support (group 1). Nine of these patients received 3 or more weeks of nutritional support (group 2). We

recorded age, sex, APACHE II score,<sup>8</sup> diagnoses, subjective global assessment (SGA) score<sup>9</sup> and hospital survival.

### Protocol

Nutritional support was started once patients were hemodynamically stable on instructions from the attending ICU team. We recorded the number of days from ICU admission that patients had not been fed. Patients were assessed by the ICU dietitian, and individual prescription was made for kilojoules based on a metabolic cart measurement; protein requirements were estimated as 1.0 to 1.9 g/kg body weight daily, depending on the severity of the illness and renal and hepatic function. The preferred nutritional support was enteral feeding of commercially available standard and elemental enteral products with additional modular glucose, fat or protein delivered by a gastric or enteral feeding tube. Parenteral nutrition consisted of glucose, lipid and amino acids with added electrolytes, minerals and trace elements, and was provided if the gut was not usable. We did not prescribe specialized amino acids (branched chain, glutamine or arginine) or fatty acids (omega-3). Insulin was prescribed as needed. Enteral feeding was started at 25 mL/h of full strength feed and increased to the target rate over 2 to 3 days. Parenteral nutrition was started at the target rate.

The following measurements were made at baseline, prior to nutritional support, then once weekly: resting energy expenditure, using a metabolic cart, weight, serum albumin and prealbumin levels, lymphocyte count and body composition (body cell mass, extracellular mass, body fat), using bioelectric impedance analysis and formulas described earlier.<sup>5,6</sup> Daily fluid balance and energy intake were recorded from nursing records. Cu-

mulative fluid balance was determined as the sum of daily fluid balances for 7 days in group 1 and for 3 or more weeks in group 2. Daily energy balance was determined as daily recorded energy intake minus 1.1 times the resting energy expenditure, measured once for each week of nutritional support. The 10% factor above the measured resting energy expenditure is to account for activity, to give a reliable estimate of 24-hour energy expenditure in critically ill patients.<sup>10</sup> Cumulative energy balance was recorded as the sum of daily energy balances for 7 days in group 1 and for 3 or more weeks in group 2.

### Measurement instruments

The measurement instruments and their accuracy were as follows. Weight was measured by an in-bed scale (Century CC bed; Hill-Rom, Batesville, Ind.), which uses piezoelectric load cells and according to the manufacturers has an accuracy of  $\pm 1\%$  in the range from 45 to 180 kg. Serum levels of albumin and prealbumin, and lymphocyte count were measured with automated instruments in the hospital laboratory: the Ektachrome 700XR (colorimetric method; Eastman Kodak, Rochester, NY), the Array 360 Systems (antibody binding and light scatter method; Beckman Instruments, Brea, Calif.) and the STKS (volume, conductivity and light scatter method; Coulter Electronics, Miami) respectively. These instruments are calibrated with the use of standard solutions and have a 1% to 3% coefficient of variation for measurements according to the manufacturer's specifications and routine calibrations in our hospital laboratory.

The metabolic cart system of indirect calorimetry used to determine resting energy expenditure was a Deltatrac Metabolic Monitor (Datex Instru-

ments, Helsinki, Finland). We have previously validated this cart for accuracy of measurement of oxygen consumption and carbon dioxide production (from which energy expenditure is calculated) in vitro<sup>11</sup> and in critically ill, mechanically ventilated patients.<sup>12</sup> The overall error of the metabolic cart is less than 2% in vitro. Resting energy expenditure was determined as the mean of 20 to 30 minutes of values obtained for each minute and had a coefficient of variation less than 10%.

Bioelectric impedance analysis was performed with the BIA-101A (RJL Systems, Inc., Detroit). The instrument applies an almost unnoticeable, painless 800- $\mu$ A current of 50 kHz between electrodes placed on the dorsum of the hand and foot and measures resistance and reactance to the current. This instrument has a coefficient of variation of  $\pm 1\%$  in vitro according to the manufacturer. We have determined that the coefficient of variation for three readings over 1 hour in ICU patients is about 5% (unpublished data). From readings of resistance and reactance, regression equations have been validated for lean body mass and body fat ( $r = 0.84$ ,  $p < 0.0001$ ), and for body cell mass and extracellular mass ( $r = 0.93$ ,  $p < 0.0001$ ) by multiple isotope dilutions for exchangeable sodium and potassium.<sup>5,6</sup>

### Analysis

To determine whether nutrition assessment variables changed over 7 days in group 1, measures of weight, albumin level, prealbumin level, lymphocyte count, body cell mass, extracellular mass, and body fat obtained at baseline were compared with those obtained after 7 days by means of a paired *t*-test. For the nutrition assessment variables that changed significantly ( $p < 0.05$ ) after 7 days of nutri-

tional support we related change in the variable to 7-day energy balance and 7-day fluid balance by linear regression. Similarly, for group 2 patients we compared nutrition assessment variables at baseline and after 3 or more weeks of ICU nutritional support by a paired *t*-test, then performed linear regression to relate the change in these variables to the energy balance and fluid balance. In addition, for this group, change in weight was assessed by means of a repeated measures analysis of variance.

Post-hoc analyses were performed to determine whether nutritional state, severity of illness or route of feeding affected the observed response in nutritional parameters. To address these questions, changes in serum prealbumin measurements from baseline to day 7 were assessed with the use of the paired *t*-test in the following subgroups: nutritional state as indicated by SGA,<sup>9</sup> severity of illness as indicated by APACHE II score<sup>8</sup> and the use of enteral versus parenteral nutrition. Also we calculated the ratio of extracellular mass to body cell mass as an estimate of the nutritional index proposed by Tellado and colleagues,<sup>13</sup>

which related nutritional status to mortality.

Data are reported as means (and standard deviations). Statistical analyses were performed with the Systat version 5.1 for Macintosh (Systat Inc, Evanston, Ill.).

## RESULTS

The mean (and SD) age of the 45 patients in group 1 was 58 (17) years. There were 27 men and 18 women. The APACHE II score was 18 (7). Twenty of the 45 patients died. Patient diagnostic groups are given in Table I. SGA scores<sup>9</sup> were A (not malnourished) in 16 patients, B (slightly malnourished) in 9 patients and C (clearly malnourished) in 20 patients. The number of days of no feeding from the time of ICU admission was 3 (2). Enteral feeding alone was given in 26 patients, parenteral feeding alone in 13 patients, and both enteral and parenteral feeding in 6 patients. Patients who were fed enterally had a mean increase in the serum prealbumin level of 37 (59) mg/L, compared with 37 (34) mg/L in patients fed parenterally. The 7-day fluid balance was -8 (6944) mL,

and the 7-day energy balance was -9731 (12 743) kJ.

The nine patients in group 2 had a mean (SD) APACHE score of 14 (8); five patients survived and four died. Six patients were studied for 3 weeks, two patients for 4 weeks and one patient for 7 weeks. The cumulative 3-or-more-weeks energy balance was -22 957 (20 525) kJ, and the cumulative 3-or-more-weeks fluid balance was 8481 (4887) mL.

The changes in nutrition variables over the 7-day support period in group 1 are shown in Table II and Fig. 1. The variables that changed significantly were weight, serum albumin level, serum prealbumin level and extracellular mass. There was no significant change in the lymphocyte count, body cell mass or body fat.

Of the nutrition variables that changed in group 1, there was no correlation of change in weight, level of serum albumin or prealbumin, or extracellular mass with cumulative 7-day energy balance (all  $r < 0.1$ , all  $p < 0.5$ ), and no correlation of change in albumin ( $r = 0.17$ ,  $p < 0.3$ ) or prealbumin level ( $r = 0.28$ ,  $p < 0.07$ ) with cumulative 7-day fluid balance. How-

**Table I**

**Demographic Characteristics by Diagnostic Group of 45 Patients in the Intensive Care Unit (ICU) Who Received Nutritional Support for 7 Days**

Diagnostic group	Mean (SD) age, yr	Mean (SD) APACHE score	No./total no. (%) of survivors	Mean (SD) 7-d fluid balance, mL	Mean (SD) 7-d energy balance, kJ
Septic respiratory failure without intra-abdominal disease (pneumonia, ARDS, COPD, Ca lung, renal failure, CHF, postop CABG, postop AVR, MVR, ITP)	58 (12)	19 (7)	5/14 (36)	2007 (8292)	-2759 (2720)
Septic respiratory failure with intra-abdominal disease (abscess, cholecystitis, IBD, ischemic bowel, hepatitis, trauma, ruptured AAA, GI hemorrhage, GI fistula, GI Ca, pancreatitis)	62 (16)	19 (8)	10/16 (62)	-2181 (5647)	-1817 (3593)
Nonseptic respiratory failure (Guillain-Barré syndrome, ALS, intracranial hemorrhage, overdose, asthma)	57 (21)	18 (7)	10/15 (67)	149 (6066)	-2379 (2759)

ARDS = adult respiratory distress syndrome, COPD = chronic obstructive lung disease, Ca = carcinoma, CHF = congestive heart failure, CABG = coronary artery bypass grafting, AVR = aortic valve replacement, MVR = mitral valve replacement, ITP = idiopathic thrombocytopenia, IBD = inflammatory bowel disease, AAA = abdominal aortic aneurysm, GI = gastrointestinal, ALS = amyotrophic lateral sclerosis

ever, there was a slight correlation of change in weight ( $r = 0.37, p < 0.01$ ) and extracellular mass ( $r = 0.38, p < 0.01$ ) with 7-day fluid balance (Fig. 2). When the single apparent outlier in the correlation of change in weight was removed, there remained a slight correlation of change in weight with 7-day fluid balance ( $r = 0.4, p < 0.01$ ).

The changes in nutrition variables over the 3 weeks or more of nutritional support in group 2 are shown in Table III. There was significant change in mean weight, serum prealbumin level and extracellular mass. However, the change in weight was not significant by repeated measures analysis of variance ( $p < 0.25$ ). There was no change in the serum albumin

level, the lymphocyte count, the body cell mass or body fat.

Of the nutrition variables that changed in group 2, there was no significant correlation of change in weight ( $r = 0.6, p < 0.06$ ), prealbumin level ( $r = 0.14, p < 0.7$ ) or extracellular mass ( $r = 0.43, p < 0.3$ ) with cumulative 3-or-more-weeks energy balance, and no correlation of change in the serum prealbumin level ( $r = 0.56, p < 0.12$ ) with cumulative 3-or-more-weeks fluid balance. There was a correlation of change in weight ( $r = 0.77, p < 0.02$ ) and extracellular mass ( $r = 0.84, p < 0.01$ ) with cumulative 3-or-more-weeks fluid balance.

Post-hoc analyses of the effects of nutritional state, severity of illness and route of feeding on the observed re-

sponse in nutritional parameters showed that: (a) clearly malnourished patients (SGA group C) had an increase in the serum prealbumin level of 30 (52) mg/L, which was not different from the change in prealbumin level of 45 (55) mg/L in less malnourished patients (SGA groups A and B) ( $p < 0.4$ ); (b) patients who were severely ill (APACHE II score 20 or greater) had an increase in the serum prealbumin level of 37 (56) mg/L, which was not significantly different from the increase in less severely ill patients of 39 (53) mg/L ( $p < 0.9$ ); (c) patients who were fed enterally had an increase in the serum prealbumin level of 37 (59) mg/L compared with 37 (34) mg/L for patients fed parenterally ( $p < 0.9$ ). Also patients who had an increased prealbumin level of more than 10 mg/L in response to nutritional support had an increased death rate (13 of 27 [48%]) compared with patients who did not have an increase in prealbumin level (7 of 18 [39%]).

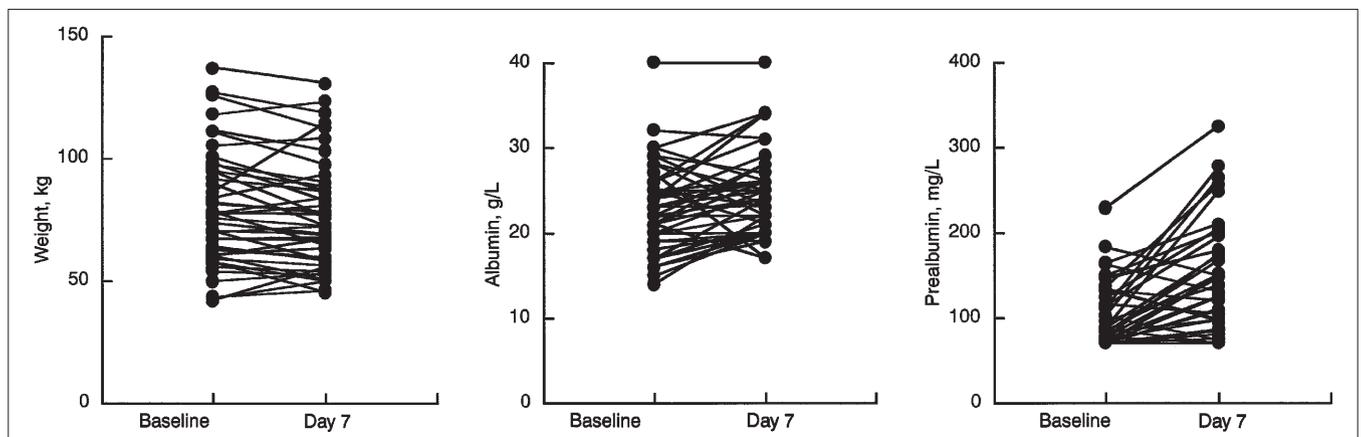
The effect of nutritional status on the death rate was assessed according to the ratio of extracellular mass to body cell mass as proposed by Tellado and colleagues.<sup>13</sup> This ratio was 1.71 (0.55) at baseline and 1.59 (0.55) after 7 days of ICU nutritional support. Eighteen of the 20 patients who died

**Table II**

**Mean (SD) Changes in Nutrition Variables From Baseline in 45 Patients in the ICU Who Received Nutritional Support for 7 Days**

Variable	Baseline		7 d		<i>p</i> value
Weight, kg	79	(23)	75	(24)	< 0.05
Serum albumin, g/L	23	(5)	24	(5)	< 0.03
Serum prealbumin, mg/L	99	(41)	139	(66)	< 0.01
Lymphocyte count, $\times 10^9/L$	1.2	(1.0)	1.2	(0.8)	< 0.6
Body cell mass, kg*	25	(11)	24	(8)	< 0.11
Extracellular mass, kg	38	(8)	35	(9)	< 0.01
Body fat, kg	16	(12)	18	(11)	< 0.1

\*28 patients had < 2 kg change, 8 patients had increase of > 2 kg and 9 had decrease of > 2 kg.



**FIG. 1.** Baseline and day 7 values of weight, serum albumin and prealbumin.

had a ratio less than 2.5, which is the cutoff value for survival suggested by Tellado and colleagues.<sup>13</sup>

DISCUSSION

The main finding from our study is that ICU patients who receive nutritional support for 7 days have significant changes in weight, serum albumin and prealbumin levels and extracellular mass but no significant changes in lymphocyte count, body cell mass or body fat. These changes did not correlate with the cumulative 7-day energy balance. Changes in albumin and prealbumin levels were not related to the cumulative 7-day fluid balance; however, changes in weight and extracellular mass correlated slightly with cumulative 7-day fluid balance. Similarly, weight, prealbumin level and extracellular mass changed significantly in ICU patients who received nutritional support for 3 or more weeks. In this group of patients, changes in weight and extracellular mass were related to cumulative fluid balance but not to cumulative energy balance. We conclude that changes in routine nutrition as-

essment parameters and body composition are slightly affected by fluid balance, so that they are not specific indicators of the adequacy of nutritional support in ICU patients.

Our findings support the contention that assessment of the efficacy of nutritional support in ICU patients is difficult and that routine nutrition assessment parameters do not accurately indicate the success of nutritional support. Change in weight was slightly related to the cumulative fluid balance but not to nutritional support, a not unexpected finding in these patients because of the large fluid vol-

umes used in resuscitation and because tissue edema decreases specificity of body weight as an indicator of muscle mass. We found increases in albumin and prealbumin levels in ICU patients but no significant relationship to either cumulative energy balance or fluid balance over 7 days or 3 or more weeks. We speculate that other factors affect these increases, such that changes in albumin and prealbumin lose specificity as indices of nutritional status in ICU patients. In particular, inflammation and sepsis are known to depress synthesis of albumin and prealbumin,<sup>14,15</sup> and albumin is diluted or

Table III

Mean (SD) Changes in Nutrition Variables From Baseline in Nine Patients in the ICU Who Received Nutritional Support for 3 Weeks or Longer

Variable	Baseline	7 d	p value
Weight, kg	66 (14)	61 (12)	< 0.05
Serum albumin, g/L	24 (4)	27 (3)	< 0.18
Serum prealbumin, mg/L	104 (34)	184 (75)	< 0.01
Lymphocyte count, 2 10 <sup>9</sup> /L	0.8 (0.5)	1.2 (0.8)	< 0.3
Body cell mass, kg	19 (3)	20 (4)	< 0.8
Extracellular mass, kg	36 (8)	32 (6)	< 0.02
Body fat, kg	11 (9)	10 (7)	< 0.5

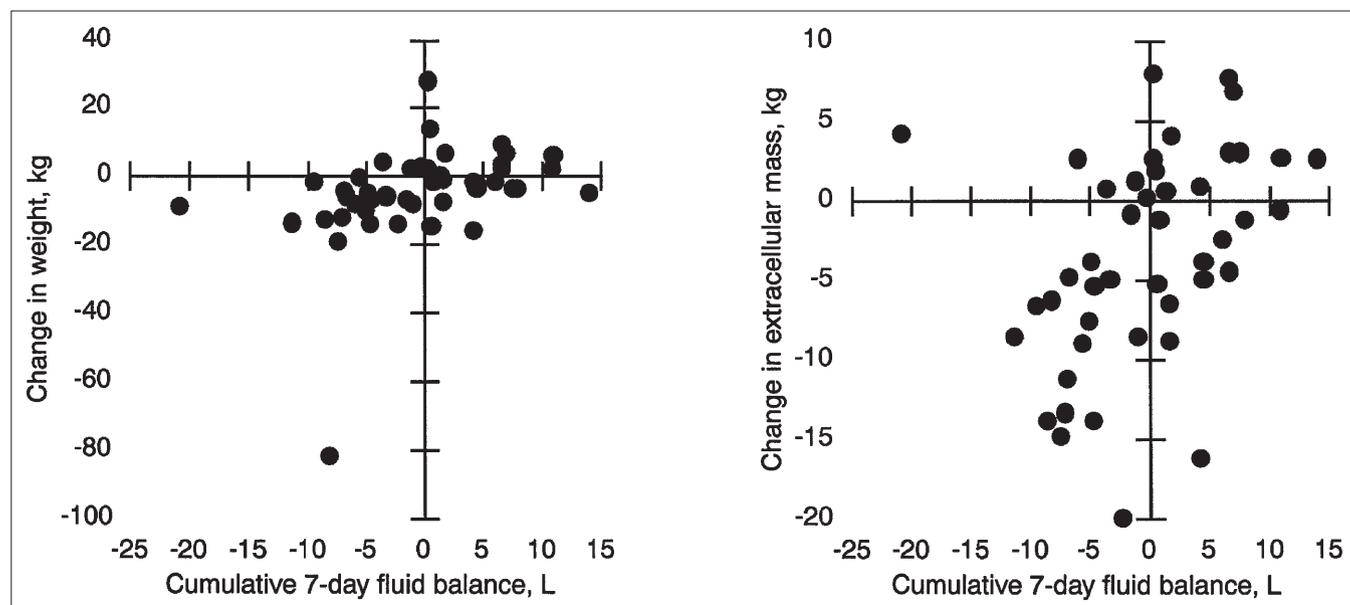


FIG. 2. Plots of change in weight and change in extracellular mass versus 7-day fluid balance.

lost to the extravascular space in the presence of endothelial injury in surgical patients.<sup>16</sup> The values were low normal or below normal for albumin and prealbumin and likely reflect both dilution and depressed synthesis from inflammation and sepsis. Although others<sup>17</sup> have found increases in lymphocyte count in response to nutritional repletion, we did not find such changes over 7 days or over 3 or more weeks in ICU patients. We speculate that the presence of inflammation, sepsis and dilution could decrease the sensitivity of the lymphocyte count as an index of nutritional status.

We found that extracellular mass, determined through bioelectric impedance analysis and the use of Shizgal's formulations, changed in ICU patients after 7 days and after 3 or more weeks of nutritional support. Also, changes in extracellular mass were slightly related to cumulative fluid balance. Although the correlation is slight, this finding ties in with the large fluid intakes, tissue edema and diuresis to which ICU patients are subject and that likely affect extracellular mass more than body cell mass or body fat, both of which did not change in our study. Also, the mean baseline value for extracellular mass of 38 kg was higher than the range of normal values (27 to 32 kg) and could result from tissue edema. In contrast, mean baseline values for body cell mass (25 kg) and body fat (16 kg) were within the normal range (23 to 29 kg and 11 to 27 kg respectively). Lastly, the accuracy of bioelectric impedance in detecting change in extracellular mass is somewhat supported by the demonstration that total body water, estimated from bioelectric impedance analysis, is closely related to values for total body water in a study of surgical patients, determined using tritium dilution space.<sup>4</sup>

That body cell mass did not change

significantly in either group could have resulted from preservation of body cell mass by nutritional support and nitrogen retention in surgical patients.<sup>13</sup> Alternatively, it could result from lack of power of the data to detect significant change in the group mean. The sensitivity of bioelectric impedance analysis to detect change in body composition may be estimated from our determination of a 5% coefficient of variation for bioelectric impedance analysis from three measurements conducted over 1 hour in ICU patients (unpublished data). Since the mean baseline body cell mass was 25 kg, changes in body cell mass of more than two standard deviations, or approximately 2 kg, should be detectable by this technique. Our data have a 95% power to detect a change of 2 kg. Of the 45 patients studied over 7 days, 28 had changes in body cell mass of less than 2 kg, 8 had an increase of more than 2 kg, and 9 had a decrease of more than 2 kg (Table II). Therefore, body cell mass did not change in the majority of individual patients, corresponding to absence of change of the group mean value. Interestingly, post-hoc regression of change in body cell mass on change in cumulative fluid balance showed a slight correlation ( $r = 0.32$ ,  $p < 0.03$ ), indicating that body cell mass is also slightly affected by cumulative fluid balance. Lastly, limitations of current nutrient preparations and techniques in critically ill patients could account for our not finding differences in nutritional assessment parameters.

Post-hoc subgroup analysis was also performed to determine whether prior nutritional state, severity of illness and use of enteral versus parenteral nutrition affected the response of nutritional markers in our study patients. Patients who were clearly malnourished (SGA group C) had a mean

increase in prealbumin level of 30 (52) mg/L, which was not different from that of patients who were less malnourished (SGA groups A and B) and who had an increase in prealbumin level of 45 (55) mg/L. Prior nutritional status, severity of illness and use of enteral versus parenteral nutrition apparently did not by themselves affect the response of nutritional parameters. Patients who had increased prealbumin levels of more than 10 mg/L in response to nutritional support had a 48% death rate compared with a 39% death rate in patients whose prealbumin level did not increase. Therefore, the response of nutritional parameters to feeding was not associated with improved outcome in our patients.

The ratio of exchangeable sodium to exchangeable potassium has been referred to as the nutritional index by Tellado and colleagues,<sup>13</sup> and a cut-off value of the nutritional index of more than 2.5 was related to death in surgical patients. Since the ratio of exchangeable sodium to exchangeable potassium relates total body water and extracellular mass to body cell mass, we were able to estimate this nutritional index from our data: the mean ratio was 1.71 at baseline and 1.59 after 7 days of ICU nutritional support. In spite of the low values for the ratio of extracellular mass to body cell mass, 20 of our 45 patients died, and 18 of them had values for this ratio of less than 2.5, so the ratio did not accurately predict mortality in this small sample of ICU patients. We speculate that this was because the outcome was strongly affected by other comorbid factors, such as infection and cardiac, pulmonary, renal, hepatic, neurologic and hematologic dysfunction, rather than by the direct influence of nutritional status alone.

We conclude that changes in currently available nutrition assessment

parameters in ICU patients do not accurately indicate the success of nutritional support. Changes in weight and extracellular mass are slightly related to cumulative fluid balance; changes in albumin and prealbumin levels are not related to cumulative energy balance or cumulative fluid balance; and lymphocyte count, body cell mass and body fat do not change significantly. Cumulative fluid balance influences nutrition assessment variables, and we suspect that inflammation and sepsis also influence them, although these factors were not evaluated in this study. At present, we provide nutritional support in our critically ill patients according to measured energy expenditure and estimated requirements for protein, electrolytes and minerals. We suggest that improved methods of assessing nutritional support are required to evaluate the traditional goals of such support, namely, provision of enough nutritional substrate to preserve body cell mass and organ function, and to evaluate therapeutic modulation of the immune response.

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