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Resting Metabolic Rate of the Critically Ill Patient: Measured versus *Predicted*

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Critically ill patients requiring mechanical ventilation are particularly susceptible to malnutrition. A knowledge of the energy requirements of these patients is essential in designing nutritional regimens. This study examines 45 resting energy-expenditure measurements performed in a group (n = 40) of postoperative, critically ill patients who were hemodynamically stable, noncomatose, and receiving mechanical ventilation. It examines in particular to what degree the resting energy expenditure of such patients can be predicted using the Harris-Benedict and Aub-Dubois formulae. Resting energy expenditure was measured using indirect calorimetry. There was only a moderate correlation between measured resting energy expenditure and that predicted using the Harris-Benedict (r = 0.57) and Aub-Dubois (r = 0.59) formulae. There was little correlation between the ratio of the measured to the predicted (Harris-Benedict) resting energy expenditure and age, or the ratio of actual to ideal body weight and body weight. The measured resting energy expenditure differed widely (70–140%) from predicted, reflecting the many complex factors that influence these patients' metabolic rate. The role of standard predictive formulae in such patients is as an arbitrary reference point to be used to define hypermetabolism (measured greater than predicted) and hypometabolism (predicted greater than measured). (Key words: Carbon dioxide: production. Metabolism: carbon dioxide production; oxygen consumption; respiratory quotient. Oxygen: consumption.)

MALNUTRITION IS A common finding among hospitalized patients.[1] One patient group particularly susceptible to malnutrition is the critically ill patient population requiring mechanical ventilation.[2,3] Designing effective nutritional support regimens for such patients is often a difficult task because of the complex nature of their illness and the dearth of knowledge about their caloric and substrate requirements. The dietary regimen must provide enough calories to replete body cell mass but must also avoid excessive caloric loads. Inadequate nutritional intake may result in a variety of complications, including respiratory muscle weakness,[4] decreased host defenses,[5] and cardiac abnormalities.[6] Excessive nutritional intakes, especially of carbohydrates, have been associated with increases in metabolic rate,[7] carbon dioxide production, and respiratory dysfunction.[8] Further knowledge of the metabolic requirements of the critically ill patient group will lead to a more precise formulation of nutrient requirements.

Studies in this patient population are often complicated by the heterogeneous nature of the type and severity of illness. This study examines the energy expenditure of a discrete subgroup of critically ill patients: hemodynamically stable, noncomatose, postoperative patients receiving mechanical ventilation. It explores to what degree the energy expenditure of such patients can be predicted using commonly used formulae and attempts to examine some of the factors that may influence the energy expenditure of such patients. This is especially important because most studies of the energy expenditure of critically ill surgical patients have been performed in spontaneously breathing patients.[7,9,10] The critically ill, mechanically ventilated patient differs in important respects from spontaneously breathing patients. The former patients tend to be more severely ill, often are massively fluid overloaded, and often receive large amounts of sedatives and muscle relaxants while receiving mechanical venti-

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TABLE 1. Harris-Benedict and Aub-Dubois Formulae

Type	Formula
Harris-Benedict	
Male	REE (kcal/day) = 66.473 + 13.7516 (wt) + 5.0033 (ht) − 6.755(a)
Female	REE (kcal/day) = 655.0955 + 9.5634 (wt) + 1.8496 (ht) − 4.6756(a)

Aub-Dubois ($kcal \cdot m^{-2} \cdot h^{-1}$)

Age (yr)	Male	Female
14–16	46.0	43.0
16–18	43.0	40.0
18–20	41.0	38.0
20–30	39.5	37.0
30–40	39.5	36.5
40–50	38.5	36.0
50–60	37.5	35.0
60–70	36.5	34.0
70–80	35.5	33.0

REE (kcal/24 h) = BSA (m^2) × kcal/m^2/h (from table) × 24 h.

REE = resting energy expenditure; wt = weight; ht = height; a = age.

lation. Both mechanical ventilation and drugs frequently used in the intensive care setting have been shown to alter metabolic rate substantially.[11–14] Such factors can either stimulate or depress metabolic rate to a greater degree than is found in spontaneously breathing patients.

Methods

Forty postoperative patients receiving mechanical ventilation had their resting energy expenditures (REEs) measured; 30 on one occasion and 5 on two occasions.

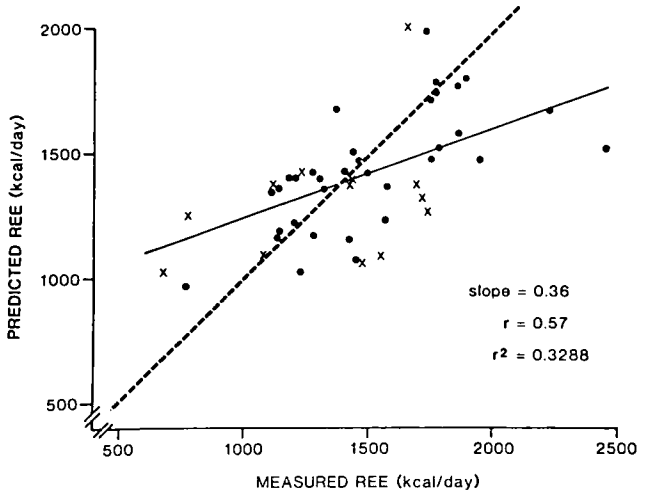

FIG. 1. Measured resting energy expenditure (REE) is plotted *versus* REE predicted using the Harris-Benedict equation ● = measurements made using the Beckman® MMC I; X = measurements made using the Horizon® MMC.

All were hemodynamically stable and not comatose. All were located in the Surgery-Anesthesiology Intensive Care Unit (ICU) of the Columbia-Presbyterian Medical Center. The mean age of the subjects was 65.7 ± 18.8 (SD) yr and ranged from 18–91 yr.

REE was determined by measuring oxygen consumption, ($\dot{V}_{O_2}$), carbon dioxide production ($\dot{V}_{CO_2}$), and respiratory quotient (RQ) during periods of rest. The measurements were made using either a Beckman® Metabolic Measurement Cart I (MMC) (Beckman Instruments, Fullerton CA) or a Horizon® Metabolic Measurement Cart (Sensormedics, Anaheim, CA). Both instruments consist of a polargraphic oxygen analyzer, an infrared carbon dioxide analyzer, and a turbine for measuring expired volume. Measurements of inspired and expired oxygen and carbon dioxide concentration along with those of minute ventilation are entered into a microprocessor wherein calculations of oxygen consumption and carbon dioxide production are performed. Both machines were validated using the method of Damask *et al.*[15] The air and oxygen intakes of the Bournes Bear I® respirators were provided with gas blended by a single Bennett A-O-I® air–oxygen blender to assure a constant fractional inspired O_2 concentration (FI_{O_2}).[16] The O_2 and CO_2 analyzers, as well as the volume turbine of the Beckman® MMC, were calibrated frequently. The patients were ventilated with an FI_{O_2} that ranged between 0.35–0.45.

Frequent measurements of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ were performed in all the patients over a 6–8 h period. The Horizon® system continuously measured $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ and provided 3-min averages. The Beckman® MMC I measured $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ for 4 min every 15 min. A log was kept of the activity state of the patients during the metabolic measurements. Resting was defined as lying motionless with eyes open and responsive to surrounding events.[17] During each 6–8 h measurement session, at least two to three periods of resting were observed. The $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values during these periods were then averaged to provide mean resting $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values.

The mean resting $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ were then used to calculate REE using the Weir method.[18] REE was predicted from each subject's height, age, current weight, and sex using both the Harris-Benedict (H-B)[19] and the Aub-Dubois[20] formulae (table 1).

Measured and predicted values for REE were expressed as kilocalories per day (kcal/day). In addition, REE, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ were normalized for body surface area (m^2) and body weight (kg). The ideal body weight of each patient was established using the Metropolitan Life Actuarial Tables (1959), and the ratio of actual to ideal body weight was then calculated.

The relationship of the measured REE to actual body weight, body temperature, age, and the ratio of actual to ideal body weight was calculated using simple regression

analysis. In addition, the relationship of measured REE to sex, age, height, weight, and body temperature was determined using multiple linear regression analysis, and the coefficient of determination (r^2) was then calculated. The correlation between the ratio of measured to predicted REE and age as well as the correlation of measured to predicted REE to the ratio of actual to ideal body weight was also determined.

This study was approved by the Committee on Human Investigations of Columbia University Health Sciences.

Results

There was only a moderate correlation between measured REE and REE predicted using the H-B equation (figs. 1 and 2). The relationship between measured REE and that predicted using the Aub-Dubois formulae was r = 0.59 (slope 0.38). The ratio of measured *versus* predicted REE (H-B) is plotted *versus* age (fig. 3). The correlation between the ratio of measured *versus* predicted REE (H-B) and the ratio of actual to ideal body weight was r = 0.17; the correlation with body temperature was r = 0.16. Multiple regression analysis of the ratio of measured to predicted REE to body temperature, age, and the ratio of actual to ideal body weight revealed a coefficient of determination (r^2) of 0.03.

There was little correlation between measured REE and either weight (r = 0.35), body surface area (r = 0.38), body temperature (r = 0.17), or the ratio of actual to ideal body weight (r = 0.11). Also, age had poor correlation (r = 0.11) with measured REE. Multiple regression analysis revealed an r^2 of 0.43 in the relationship of measured REE to height, weight, sex, age, and body temperature.

$\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and REE normalized for body weight (kg) and body surface area (m^2) can be found in table 2.

RQ is plotted *versus* nutritional intake in figure 4. The majority of the patients were receiving 5% dextrose and had an RQ that ranged from 0.71 to 0.83.

Discussion

Recent development of instrumentation designed to measure oxygen consumption and carbon dioxide production in mechanically ventilated patients has made possible the systematic exploration of the metabolic demands for such patients. The advent of parenteral nutrition has made possible the provision of complete caloric requirements in such patients; therefore, there has been much interest in accurately measuring the energy expenditure of the critically ill patient so as to be able to adjust the supply of nutrients to the demand. The present study is an initial attempt to examine the metabolic rate of the critically ill patient. This study was limited to a noncomatose, hemodynamically stable group of postoperative patients who were being mechanically ventilated with oxygen concentrations of 35–45%. The study population contains mainly older patients (fig. 3) because such patients tend to have more prolonged and complicated postoperative ICU courses than the younger trauma patients (mainly penetrating trauma victims) seen in our hospital.

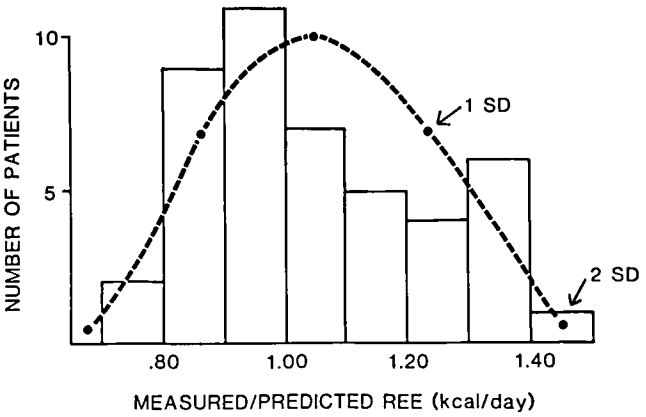

FIG. 2. The distribution of the ratio of measured resting energy expenditure (REE) to that predicted using the Harris-Benedict equation.

The usual method of calculating the caloric demands of an individual is to use one of the standard formulae such as the H-B,[19] Aub-Dubois,[20] or Kleiber[21] equations. The results obtained from these equations, although labeled as the basal metabolic rate, are in reality estimates

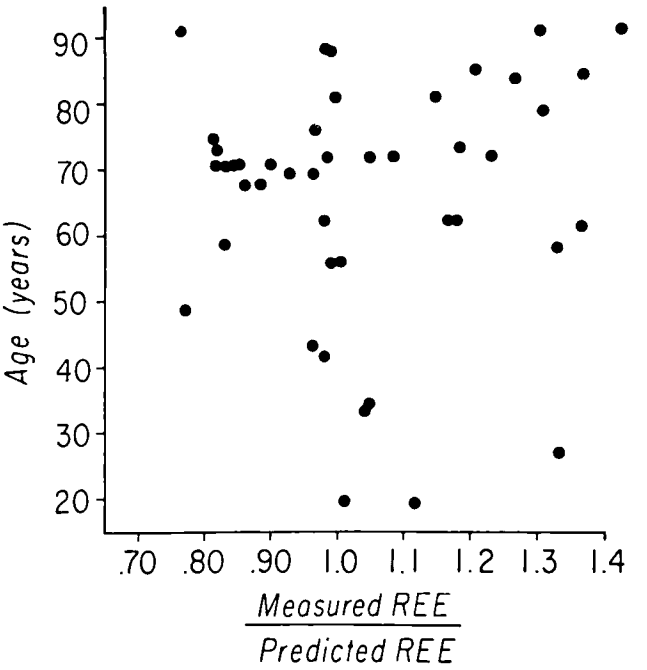

FIG. 3. The ratio of measured resting energy expenditure (REE) to that predicted using the Harris-Benedict equation is plotted *versus* age.

TABLE 2. Metabolic Measurements Corrected for Body Weight and Surface Area (n = 45)

	$\dot{V}_{O_2}$ (ml/min)	$\dot{V}_{CO_2}$ (ml/min)	REE (kcal/day)	REE (kcal/day)	
				Males (n = 25)	Females (n = 20)
Per kg	3.0 ± 0.7	2.4 ± 0.5	20.9 ± 4.8	22.6 ± 4.2	18.8 ± 4.7
Per body surface area (M^2)	118.7 ± 23.0	92.9 ± 16.8	819.5 ± 158.3	870 ± 134	753 ± 166

Mean ± SD.

of REE, due to the conditions of the original measurements.[22] To these estimates of REE, a factor to account for activity is usually added (*e.g.*, 10–20% for a sedentary hospital patient), as are factors for the patient's clinical state (*e.g.*, sepsis, injury, nutritional depletion).[23] One must then decide if the goal of nutritional support is the maintainance or repletion of lean body tissue. Energy requirements for the latter are greater than for the former. Previous studies have demonstrated that for patients with diseases such as cancer,[24,25] inflammatory bowel disease,[26] and obesity,[27] standard predictive formulae did not uniformly predict metabolic rate. Patients with inflammatory bowel disease weighing less than 90% of their ideal body weight had significantly increased energy requirements on a per-kilogram weight basis compared with those weighing more than 90% of ideal body weight.[26] In patients with cancer, both hypometabolism (measured less than predicted REE) and hypermetabolism (measured greater than predicted REE) have been reported.[24,25] In a study of 200 clinically stable patients and 72 healthy control subjects, Feurer *et al.*[22] observed that the measured REE was overestimated or underestimated by greater than 10% *via* the H-B predictors in 40% of the patients but only 20% of the 72 healthy control subjects studied.

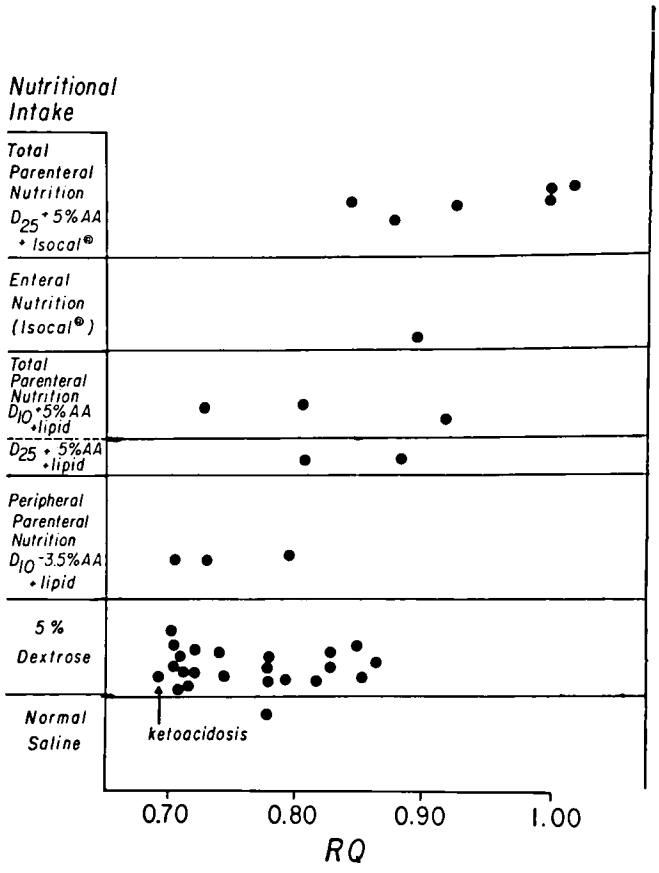

FIG. 4. Nutritional intake is plotted *versus* respiratory quotient (RQ).

Review of the original work of Harris and Benedict revealed that 88% of the normal subjects had predictions within 10% of measured REE and that 95% of the subjects were within 14% of measured REE.[22] Boothby showed 92% of normal subjects were within ±10% of predicted REE and 99% were within 15% of predicted REE.[28,29] Therefore, if a patient's energy expenditure is outside the range of ±15% of predicted REE, the discrepancy is likely due to disease and/or therapy or an abnormality in body composition or metabolism.[22]

The present study demonstrated that the ability to predict metabolic demand in the postoperative patient is poor. Predictions of energy expenditure based on the H-B equation correlated only modestly (r = 0.57) with the measured values (fig. 1). The same was true with Aub-Dubois formulae. This is not surprising because these predictive formulae are based on data from healthy subjects. Their importance in the care of the critically ill lies not in their ability to predict caloric requirements accurately but in their use as arbitrary reference points to assess changing levels of metabolism. Our patients had a rather wide range of measured to predicted (H-B) ratios, from 70–140%. Carlsson *et al.*[30] observed similar (70–126%) variability in a group of infected and traumatized patients being mechanically ventilated. The reason for this wide range is not immediately evident. In the present study, it was not related to either age, the ratio of actual to ideal body weight, or body temperature. There are various factors other than those previously noted that can influence the metabolic rate of such patients. These include the amount of sedation administered, nutritional

intake,[7] the clinical condition (sepsis often increases REE while nutritional depletion leads to reduced energy expenditure), and the amount of water retention that has occurred. The latter factor is particularly important because water does not contribute to metabolic rate and the predictive formulae all include weight or body surface area (which is derived from weight) as a variable. This point is especially important because extracellular fluid can change within hours or days to a greater degree than can either fat or lean body mass. The importance of accumulated extracellular water can be demonstrated through one of the subjects in this study who had gained approximately 10 kg of fluid during her ICU stay of 6 days. Her measured REE was 1123 kcal/day. The predicted (H-B) value using her actual weight was 1353 kcal/day. Yet, if 10 kg is subtracted, it is 1277 kcal/day. Therefore, the ratio of measured to predicted REE changed from 0.83 to 0.88.

Current methods cannot be used to predict metabolic rate with precision. Measures of body cell mass may be a better parameter for prediction of metabolic demand, as has been proposed by Spanier and Shizgal.[31] However, other factors, such as the effects of disease states and medication, may result in poor predictability even when body cell mass is used. As noted in the present study, there was little correlation between such variables as age, body temperature, or ratio of actual to ideal body weight to the measured REE. This is not unexpected, since others have noted that no single variable is sufficient for predicting metabolic rate. Keys *et al.*[32] observed that the commonly cited effect of age on the metabolic rate of adults (*i.e.*, that metabolic rate decreases as age increases) is less than that accounted for by the predictive formulae. The predictive formulae thus may overestimate the rather small effect of age on metabolic demand. In addition, in the current study the ability of multiple variables to predict metabolic rate was only fair, as demonstrated by an r^2 of 0.43 when multiple regression analysis was performed between measured REE and height, weight, age, sex, and body temperature. At the present time, direct measurement is the only reliable way of determining the metabolic rate of the critically ill patient.

In order to analyze further the results of this study and compare them with results obtained by others, the $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and REE were divided by body weight and body surface area (table 2). It is important to note the rather large SDs indicating that there is much variability in the data. This is consistent with the findings of other investigators studying acutely ill patients. Quebbeman *et al.*[33] noted that stressed male and female patients had REEs of 838 ± 118.0 (SD) kcal · day^{-1} · m^{-2} and 866.1 ± 121.5 kcal · day^{-1} · m^{-2}, respectively. These SDs are similar in magnitude to those found in our study (table 2). Feurer *et al.*[22] noted that male patients had a mean resting energy expenditure of 22.4 ± 3.8 (SD) kcal · day^{-1} · kg^{-1} while female patients had a mean of 21.2 ± 4.6 kcal · day^{-1} · kg^{-1}. Normal subjects, however, had a tighter distribution: 23.2 ± 2.4 kcal · day^{-1} · kg^{-1} for males and 23.1 ± 2.7 kcal · day^{-1} · kg^{-1} for females. The wider distribution of patient data reflects the many factors that can affect their energy expenditures, as were noted before. Despite the wide distribution of energy expenditure values, there are two important observations to be made from the data. One is that these patients were not profoundly hypermetabolic as a group. Predicted (H-B) REE was 1412 ± 253 (SD) kcal/day and measured REE was 1466 ± 306 kcal/day, a difference of 3.8%. These results are similar to those of Baker *et al.*,[34] who observed that, unlike the spontaneously breathing septic patients studied by Askanazi *et al.*[7] whose metabolic rates averaged 14–15% above predicted rates, septic patients receiving mechanical ventilation had metabolic rates that averaged only 4.6% above predicted (H-B) rates. Carlsson *et al.*[30] observed that in 14 infected/traumatized, mechanically ventilated patients, measured REE was 94 ± 4% of predicted rates. The reason for this apparent difference in the metabolic rate of spontaneously breathing and mechanically ventilated patients may be due to a number of factors, including: 1) greater fluid retention by ICU patients, which would lead to falsely elevated predictions; 2) the fact that these patients are not breathing spontaneously; the work of breathing, which in normal subject consists of about 3% of REE, may be as much as 25% of the REE of a spontaneously breathing, critically ill patient[11]; 3) the profound illness of these patients; and 4) the sedated and relatively motionless state of these patients. This is emphasized further when one examines figure 2, which reveals that a large proportion of the postoperative critically ill patients studied had metabolic rates below predicted rates. Thus, from these results and those of Baker *et al.*,[34] it appears that hypermetabolism may not be as prevalent in the ICU population as one would predict from observations made in spontaneously breathing patients. Similar results have been reported by Hunker *et al.*,[35] who noted that eight out of ten patients receiving mechanical ventilation had measured REEs below predicted rates. These patients were heavily sedated and/or paralyzed. Bartlett *et al.*,[36] however, found that almost all the critically ill patients they studied were hypermetabolic, which they defined as having a $\dot{V}_{O_2}$ greater than 130 ml · min^{-1} · m^{-2}. This was not the case in our study, where the mean $\dot{V}_{O_2}$ was 118.7 ± 23.0 ml · min^{-1} · m^{-2}. The discrepancy may be due to the fact that the Bartlett group used a volumetric ventilator-respirometer, while the present study and that reported by Baker[34] and Hunker[35] used modifications of the Douglas bag technique.

Measurement of metabolic rate in mechanically ventilated patients is a difficult and often arduous task, for

both technical and clinical reasons. Because the physiologic range of these measurements, especially the RQ, is very narrow, (0.70–1.2) exacting, reproducible technique and precision are needed. In addition the complexity of the respiratory plumbing and the potential error from small leaks make vigilance essential. When small amounts of air leak into the system in cases where an elevated $F_{I_{O_2}}$ is in use, the prominent error is a decrease in RQ. The RQ is important for two reasons. One is to determine net substrate utilization on any given diet. In the study reported here, the majority of patients were receiving 5% dextrose (100–450 kcal/day) and thus were in a state of semistarvation. Their RQ ranged from 0.70–0.82. Net substrate utilization for energy was mainly endogenous fat oxidation with a lesser degree of protein oxidation. A respiratory quotient of 0.80 indicates some carbohydrate utilization from either the 5% dextrose solution or endogenous glycogen stores. An RQ of 0.70 reflects utilization of fat as the sole fuel for oxidation. The formation of ketones from fat has an RQ of 0 because no carbon dioxide is produced and only oxygen is consumed. If these are oxidized during the period of measurement, the net effect is as if the precursor (fat) has been oxidized (RQ = 0.7). However, if the ketones are excreted in the urine, the RQ is decreased because inspired oxygen is lost without CO_2 being produced. Thus, patients with ketosis or ketoacidosis who are excreting large amounts of ketones may have an RQ slightly below 0.70, as is demonstrated by the patient with diabetic ketoacidosis in the present study. There were three instances of RQs of 1.0 or above. In all three instances the patients were in the process of being switched from enteral to parenteral nutrition and their total intake (enteral plus parenteral) was about twice the measured REE. In all instances their carbohydrate intake alone was in excess of REE. An RQ of above 1.0 indicates net lipogenesis, *i.e.*, the conversion of carbohydrate to fat—a process that has an RQ of 8.0.[7] It is important to remember that in humans this process is not very efficient and that the highest observed respiratory quotients are about 1.2.

The other reason that measurements of the RQ are important is that they can also serve as a quality control technique. Because the physiologic range is narrow, small errors in either or both $\dot{V}_{O_2}$ or $\dot{V}_{CO_2}$ will cause significant alterations in RQ. Measurements of whole body RQ that fall outside the range of 0.67 to 1.25 should lead one to suspect an error in the measurement technique. Thus, in any report of gas exchange data, the RQs should be reported and correlated with nutrient intake.

To summarize, currently, many nutritional support regimens are tailored to provide a caloric intake of 20% to 50% above metabolic demand. The findings of this study indicate that if metabolic demand is estimated but not measured, patients may well receive a caloric intake that is either above or below requirements by 50% or more. This is especially true when using the often recommended method of designing a nutritional support regimen, namely, adding values for the energy cost of physical activity and the energy cost of injury and illness to the estimated (H-B) energy expenditure.[37,38] This may result in the significant overfeeding, especially of those critically ill patients with REEs below predicted rates. Critically ill patients, with their already compromised respiratory and cardiovascular systems, are even more vulnerable to the complications of overfeeding, as demonstrated by Covelli *et al.*[8] and more recently by Dark *et al.*[39] and others[40] than are the spontaneously breathing patients these recommendations are based on.

Clinicians designing nutritional regimens based on predicted rate rather than measured metabolic rate must be aware that the estimation of metabolic rate in postoperative mechanically ventilated, critically ill patients using established formulae is often inaccurate. More studies using direct measurement of REE are needed to allow for a better understanding of the energy expenditures of such patients. The role of the standard predictive formulae, especially the H-B equation, appears to be an arbitrary reference point whose reliability in the critically ill patient should be questioned.

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References

1. Bistrian BR, Blackburn GL, Hallowell E, Heddle, R: Protein status of general surgical patients. JAMA 230:858–60, 1974
2. Driver AG, LeBurn M: Iatrogenic malnutrition in patients receiving ventilatory support. JAMA 244:219, 1980
3. Boles JM, Garre MA, Youinov PY, Mialon P, Menez JF, Jouquar J, Miossec PJ, Pennec Y, Menn GLE: Nutritional status in intensive care patients: Evaluation in eighty-four unselected patients. Crit Care Med 11:87–90, 1983
4. Arora NS, Rochester DF: Respiratory muscle strength and maximal voluntary ventilation in undernourished patients. Am Rev Resp Dis 126:5–8, 1982
5. Kahan BD. Nutrition and host defense mechanisms. Surg Clin North Am 61:557–70, 1981
6. Heymsfield SB, Bethel RA, Ansley JD, Gibbs DM, Felner JM, Nutter DO: Cardiac abnormalities in cachectic patients before and during nutritional repletion. Am Heart J 95:584–594, 1978
7. Askanazi J, Carpentier YA, Elwyn DH, Nordenstrom J, Jeevanandam M, Rosenbaum SH, Gump FE, Kinney JM: Influence of total parenteral nutrition on fuel utilization in injury and sepsis. Ann Surg 191:40–6, 1980
8. Covelli HD, Black JW, Olsen MS, Beekman JF: Respiratory failure predicted by high carbohydrate loads. Ann Intern Med 95: 579–81, 1981
9. Elwyn DH: Nutritional requirements of adult surgical patients. Crit Care Med 8:9–20, 1980
10. Kinney JM: The application of indirect calorimetry to clinical

studies. Assessment of Energy Metabolism in Health and Disease. Edited by Kinney JM. Columbus, Ross Laboratories. 1980, pp 42–48

11. Field S, Kelly SM, Macklem PT: The oxygen cost of breathing with cardiorespiratory disease. Am Rev Respir Dis 126:9–13, 1982
12. Bursztein S, Taltelman V, DeMy Henoere S, Michelson M, Dahan E, Cepstein R, Edelman D, Melamed Y: Reduced oxygen consumption in catabolic states with mechanical ventilation. Crit Care Med 6:162–64, 1978
13. Dempsey DT, Guenter P, Mullen JL, Fairman R, Crosby LO, Speilman G, Gennarelli L: Energy expenditure in acute trauma to the head with and without barbiturate therapy. Surg Gynecol Obstet 160:128–34, 1985
14. Rodriguez J, Weissman C, Damask M, Askanazi J, Hyman AI, Kinney JM: Morphine and postoperative rewarming in the critically ill. Circulation 68:1238–46, 1983
15. Damask MC, Weissman C, Askanazi J, Hyman AI, Rosenbaum SH, Kinney JM: A systematic method for validation of gas exchange measurements. ANESTHESIOLOGY 57:213–218, 1982
16. Browning JA, Lindberg SE, Turney SF, Chodoff P: The effects of fluctuating $F_{I_{O_2}}$ on metabolic measurements in mechanically ventilated patients. Crit Care Med 10:82–5, 1982
17. Weissman C, Kemper MC, Damask M, Askanazi J, Hyman AI, Kinney JM: The effect of routine intensive care interactions on metabolic rate. Chest 86:815–18, 1984
18. Weir JB: New method for calculating metabolic rate with special reference to protein metabolism. J Physiol (Lond) 109:1–6, 1949
19. Harris JA, Benedict FG: Standard basal metabolism constants for physiologists and clinicians, A Biometric Study of Basal Metabolism in Man. Philadelphia. JB Lippincott. 1919. pp 223–250
20. Aub JC, Dubois EF: The basal metabolism of old men. Arch Intern Med 19:823, 1917
21. Kleiber M: The fire of life: An introduction to animal energetics. Huntington, Robert E. Krieger Publishing. 1975
22. Feuer ID, Crosby LD, Mullen JL: Measured and predicted resting energy expenditure in clinically stable patients. Clin Nutr 3: 27–34, 1984
23. Long CL, Schaffel N, Geiger JW: Metabolic response to injury and illness: Estimation of energy and protein needs from indirect calorimetry and nitrogen balance. JPEN 3:452–6, 1979
24. Knox LS, Crosby LO, Feurer ID, Buzby GP, Miller CL, Mullen JL: Energy expenditure in malnourished cancer patients. Ann Surg 197:152–62, 1983
25. Lindmark L, Bennegard K, Eden E, Ekman L, Schorsten T, Svaninger G, Lundholm K: Resting energy expenditure in malnourished patients with and without cancer. Gastroenterology 87:402–8, 1984
26. Barot LR, Rombeau JL, Steinberg JJ, Crosby LO, Feurer ID, Mullen JL: Energy expenditure in patients with inflammatory bowel disease. Arch Surg 116:460–62, 1981
27. Feurer ID, Crosby LO, Buzby GP, Rosato EF, Mullen JL: Resting energy expenditure in morbid obesity. Ann Surg 197:17–21, 1983
28. Boothby W, Sandiford I: Summary of the basal metabolism data on 8614 subjects with especial reference to the normal standards for the estimation of basal metabolic rate. J Biol Chem 54: 783–803, 1922
29. Boothby WM, Berkson J, Dunn HL: Studies of the energy expenditure of normal individuals: A standard for basal metabolism with a nomogram for clinical application. Am J Physiol 3:468–83, 1936
30. Carlsson M, Nordenstrom J, Hendstierna G: Clinical implications of continuous measurement of energy expenditure in mechanically ventilated patients. Clin Nutr 3:103–110, 1984
31. Spanier AH, Shizgal HM: Caloric requirements of the critically ill patient receiving intravenous hyperalimentation. Am J Surg 133:99–104, 1977
32. Keys A, Taylor HL, Grande F: Basal metabolism and age of adult man. Metabolism 22:579–87, 1973
33. Quebbeman EJ, Ausman RK, Scheider TC: A re-evaluation of energy expenditure during parenteral nutrition. Ann Surg 195: 282–6, 1982
34. Baker JP, Detsky AS, Stewart S, Whitwell J, Marliss EB, Jeejeebhoy KN: Randomized trial of total parenteral nutrition in critically ill patients: Metabolic effects of varying glucose–lipid ratios as the energy source. Gastroenterology 87:53–9, 1984
35. Hunker FD, Burton CW, Hunker EM, Durham RM, Krumdieck CL: Metabolic and nutritional evaluation of patients supported with mechanical ventilation. Crit Care Med 8:628–32, 1980
36. Bartlett RH, Dechert RE, Mault JR, Ferguson SK, Kaiser AM, Erlandson EE: Measurement of metabolism in multiple organ failure. Surgery 92:771–8, 1982
37. Long CL, Schaffel N, Gieger JW, Blakemore WS: Metabolic response to injury and illness: estimation of energy and protein needs from indirect calorimetry and nitrogen balance. JPEN 3:452, 1979
38. Caldwell MD, Kennedy-Caldwell C: Normal nutritional requirements. Surg Clin North Am 61:489–507, 1981
39. Dark D, Pingleton SK, Kerby GR: Hypercapnia during weaning: A complication of nutritional support. Chest 88:141–143, 1985
40. Herve P, Simonneau G, Girard P, Cerrina J, Mathieu M, Duroux P: Hypercapnic acidosis induced by nutrition in mechanically ventilated patients: Glucose *versus* fat. Crit Care Med 13:537–40, 1985