are needed using direct measurement of REE to allow for a better understanding of the energy expenditures of patients. It is also appropriate to control as many variables as possible when evaluating a given metabolic event. Such quantitative data are rewarding. However, such predictive equations are appropriate in estimating the energy needs of patients whose REE measurements are not possible. It would also be of interest to know how the investigators determined the total daily caloric needs from their quantitative 24-h REE measurement. If they assumed an activity factor with their measured REE, they may be no better off than using predictive equations that hold for normal states.

With these considerations, it seems reasonable to use the data that have been published in terms of general responses of various patient groups and adjusting those requirements on a logical basis (with a consideration of other parameters such as nitrogen balance) to assure that the clinician is meeting the patient's energy and protein needs on a daily basis.

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In reply:—We strongly disagree that the article "Resting metabolic rate of the critically ill patient: Measured *versus* predicted" presents a distorted view of the relationship between measures and predicted metabolic rates and, as such, dilutes the use of predicted (resting energy expenditures (REEs) in patient care.

It is important to realize that this paper focuses specifically on the ability of two commonly used predictive equations (Aub-Dubois and Harris-Benedict) to predict REE in critically ill patients. Since these equations are derived from studies performed on normal subjects, it is not unexpected that it is rather difficult to predict accurately the resting metabolic rate of these patients. This is due to the many complex interacting factors that influence these extremely ill patients' metabolic rate. This article thus points out the limitations of predicted metabolic rates in accurately predicting patient needs.

Dr. Long has previously proposed that the caloric intake of patients be estimated by tempering the Harris-Benedict equation with an activity factor and an injury factor. These factors have been derived from studies performed on spontaneously breathing, noncritically ill patients. Whether these recommendations are applicable to intensive care unit (ICU) patients still must be determined. In fact, recent work by our group has found the activity factor in ICU patients to be about 5%.[1] Dr. Long's recommendation is to add up to 20% for activity. Long *et al.* also recommend adding injury factors to the Harris-Benedict equation. These factors are derived from work performed by Kinney[2] and Long *et al.*[3] in patients who were spontaneously breathing and who by and large had single system disease, for example patients convalescing from total hip replacements, gastrectomies, and cystectomies as well as burn patients. Whether these data are directly applicable to mechanically ventilated, sedated, critically ill patients with multiple system organ failure needs to be determined. It may be that predictive formulae could be used as the basis of estimating the needs of these patients, but more work is needed to determine if and how activity and injury factors are to be added to these formulae. We agree that ideally the effects of the many variables that alter the energy expenditure in critically ill patients should be controlled for. However, practically, that is quite difficult because these patients usually have many intercurrent treatments. It is not uncommon for a mechanically ventilated septic patient to be heavily sedated. Mechanical ventilation and sedation both decrease metabolic rate, while sepsis usually increases but may also decrease metabolic rate.

We agree with Dr. Long that there are many problems with measuring oxygen consumption and carbon dioxide production when elevated oxygen concentrations are being used. We have studied this problem in detail and have developed special validation and calibration procedures.[4] We disagree with Dr. Long's statement that in major injury, REE values are increased by as much as 100%. Only burned patients show such increase, while most studies have shown more modest increases, *i.e.*, at the maximum, 40–50%.

Figure 1 in our article demonstrates the relationship of measured to predicted REE. The reason that the instrument used to obtain each measurement is shown is to

demonstrate a lack of systematic error by each instrument. Of course if the correlation of the data from each cart is calculated, the regression may be different and that is due to the large variability in the data. Thus, any small group of patients may cause a slightly different correlation than the total data. We have evaluated patients using both carts and found a variation less than 5%.

In conclusion, we feel that this article merely points out the fact that in ICU patients there is a large variability in metabolic rate that makes prediction difficult. There is great need to examine further the various factors that influence this variability so that predictive methods, such as those used in spontaneously breathing patients with some success, may be developed. It is important to remember that a prediction is just an educated guess.

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Pulmonary Edema Following Low-dose Naloxone Administration

To the Editor:—Although early studies suggested that naloxone (Narcan™) reversal of high-dose narcotic anesthesia was not accompanied by significant cardiovascular changes,[1–3] more recent reports describe complications following naloxone administration to surgical patients. Responses ranging from severe hypertension[4–6] to tachycardias,[7] ventricular arrhythmias,[6,8] acute pulmonary edema,[9–11] and in some cases cardiac arrest[8,12] have been reported. Suggestions for the mechanism of these sporadically occurring complications have centered around centrally mediated catecholamine responses to narcotic reversal.[13]

Initial reports described complications arising in patients with preexisting cardiac disease who had received a high dose of narcotic. This resulted in recommendations to give smaller, incremental doses of naloxone when narcotic reversal was required.[9,10] Subsequently, Prough *et al.*[11] reported two cases of pulmonary edema following administration of 100 μg and 500 μg naloxone to two young healthy males undergoing minor surgical procedures. In light of these case reports and despite package inserts suggesting doses of 100–200 μg, it has been our practice to administer naloxone in aliquots of only 40 μg, and to separate doses by 3 to 5 min. We report here a case of pulmonary edema requiring intubation and positive pressure ventilation following a total naloxone dose of just 80 μg administered over 5 min.

REPORT OF A CASE

The patient, a 19-yr-old weighing 95 kg, was scheduled for incision and drainage of a five cm neck abscess. He had no previous medical history and, specifically, no history of drug abuse. Computed tomographic imaging of the neck and physical examination demonstrated no evidence of airway compromise.

The patient was anesthetized at 8:00 AM following a fast of approximately 10 h. He received sodium thiomylal 500 mg in divided doses, 200 μg fentanyl and 2 mg of metocurine followed by 120 mg succinylcholine for intubation, and a total of approximately 1,300 ml of lactated Ringer's solution. At the end of the 65-min procedure, the patient was breathing spontaneously. He was suctioned and extubated and was transferred to the recovery room receiving oxygen at 6 l/min by mask. In the recovery room, his initial respiratory rate of 16 breaths/min fell to 3–4/min, and he recieved two iv doses of 40 μg naloxone separated by 5 min. Almost immediately following the second dose, he was noted to be in considerable respiratory distress, with an oxygen saturation, measured by pulse oximeter, of less than 80%. He began to produce copious amounts of pulmonary edema fluid, which continued for more than 12 h despite reintubation and positive pressure ventilation with up to 15 cm of PEEP. Chest roentgenogram was consistent with pulmonary edema. A pulmonary artery catheter was placed, demonstrating a pulmonary artery wedge pressure of 12–14 mmHg, a systemic vascular resistance of 900 dyn · s^{-1} · cm^{-5}, and a cardiac output greater than 6 l/min. Arterial blood pressures remained at 130–140/70–80 mmHg throughout the patient's hospital stay. After 14 h, the patient was extubated, but he continued to cough up small amounts of edema fluid and maintained oxygen saturations of approximately 90% while breathing 40% oxygen administered by mask. Normal oxygen saturations were not obtained until more than 40 h after the naloxone administration. The patient suffered no lasting ill effects and was discharged home on the fourth hospital day.