water than left-ventricular systolic function of the heart. The lower-than-predicted TLC levels they observed may in part relate to the technique used for TLC measurement. In patients with cardiac disease and elevated wedge pressures, Ries et al measured the lowest TLC values by gas dilution techniques (mean = 86.7 percent of predicted), intermediate TLCs by plethysmography (90.5 percent predicted) and normal TLC by radiographic techniques (98.0 percent). Such differences are in accord with airway compression and "gas-trapping" and reduction of airspaces within the lung secondary to increased fluid in interstitial and alveolar spaces.

Does this study shed light on the interesting but neglected question of what causes the noises we describe as rales or crackles? Is it the rapid changes in tissue tension as compressed small airways expand? . . . the sudden equalization of gas pressures as closed airways open? . . . or the bubbling and movement of fluid within airways? The safest conclusion from the study of Siegel and colleagues is that rales are more likely to be present in patients with lower Dco values, probably because these are the patients with more fluid within the lung. Whether the origin of the rales was compressed airways or fluid within airways and alveoli cannot be determined from this study. A less likely, but more entertaining possibility is that patients with low Dco levels are "predisposed" to have "leakier" vessels and therefore more likely to have more severe pulmonary edema and rales, as suggested by Frand et al in their study of pulmonary edema in patients who had heroin overdoses.

A better understanding of the acoustic basis of rales will probably require investigations under more controlled experimental conditions than is readily available for clinical studies, conditions which would allow for semi-simultaneous measurements of ventricular function, lung water, radiographic findings, measures of pulmonary function, and descriptions of rales under standardized control of location, posture, breathholding, volume history and other relevant variables.

As is the case with a number of quantitative laboratory tests used in medicine (plasma prothrombin times, serum levels of cholesterol or alpha,-antitrypsin), caution must be used in clinically applying the conclusions of studies regarding Dco because of differences between measurement techniques used in individual laboratories and differences between predictive normal values. In their recent report on Dco measurements of normal subjects from Spain, Boca et al noted clinically important differences between predictive values: the percentage of healthy adults with Dco values below the lower limit of normal ranged from 0 to 60 percent when results were calculated from five different prediction equations (all of which were published within the last ten years).

Even larger discrepancies between prediction values for Dco may occur secondary to whether or not predicted values are adjusted for smoking history, as was done in this study. We expect that in the future, differences in Dco values measured in different laboratories or predicted by various equations will become negligible as laboratories and investigators adopt standardized test methodologies, but until these differences are known to be negligible, caution must be used in applying published results using limits of normal for the Dco to clinical decision making.

Differences in predicted values may also in part be responsible for the discordance in the literature whether Dco is reduced or within normal limits in patients with CHF. Variances in how CHF is defined, physiologic adaptions to chronic CHF, and how patients are selected for studies are additional key factors which are likely to explain differences in the literature on findings in patients with CHF. These variables also add complexity to the challenges of making sense of diagnostic tests when a patient has more than one process causing dyspnea.

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REFERENCES

Things That Go Around Come Around, Even in the Intensive Care Unit

Nutritional support (NS) continues to be enthusiastically accepted by many physicians who treat
patients in the intensive care unit (ICU), my concerns notwithstanding.¹ The rationales for employing NS include the fact that such patients are already, or are in danger of becoming, malnourished and the association between malnutrition and a poor clinical outcome. Malnutrition is also associated with respiratory-related phenomena (eg, impairments in ventilatory response to hypoxia,² vital capacity,³ and maximal voluntary ventilation⁴). Tests measuring these functions may improve if NS is provided.⁴

In the old days (1960s and 1970s) NS was called "hyperalimentation," and large numbers of calories were provided. Excessive caloric supply has important negative implications for patients with respiratory disease, as CO₂ is produced when unused carbohydrates are stored as fat.⁵ Current therapy seeks to replace only those calories burned. Formulæ (Harris-Benedict) can be used to predict these requirements, but concerns over potential inaccuracies in critically ill patients led to the introduction of subjective fudge factors.⁶ The quest for even more precision spawned the use of metabolic carts, which measure actual energy expenditures by indirect calorimetry.

These carts are expensive and, in the hands of the less experienced, often unreliable. In 1987, Liggett and colleagues described a simpler technique for those patients who had thermodilution pulmonary catheters in place.⁷ In this issue of Chest, (see page 682) Liggett and Renfro bring us full circle by showing that the unmodified predictions of the Harris-Benedict equations correlate with the energy requirements measured by the pulmonary catheter technique. Do we now have an inexpensive guideline which will not require any more sophisticated equipment than pencil and paper?

Unfortunately, this will not always be the case. Liggett and Renfro did observe that, in patients with sepsis, caloric needs increased by 20 percent. Patients with extensive burns (and perhaps other forms of severe trauma) will also have larger requirements and need some fudge factor adjustment. However, for most medical (as opposed to surgical) ICU patients, the observations of Liggett and Renfro will obviate the need for more expensive monitoring technology.

In their discussion the authors note that it is "essential to follow nutritional parameters such as nitrogen balance to ultimately determine appropriate caloric therapy." This statement raises an important question: what are we trying to accomplish by giving NS to ICU patients? Do we want to replace energy being burned, or do we want to restore positive nitrogen balance? These goals are not the same. Positive nitrogen balance will not occur unless there is adequate nitrogen and excess (more than resting energy expenditure) caloric supply. This requirement appears to be at least 140 to 150 percent of the resting energy expenditure⁸⁻¹⁰ or at least 40 calories/kg/day.¹¹⁻¹⁴ amounts equivalent to those provided in the not-so-good old days. Coming full circle by again giving excess calories will undoubtedly bring about the very respiratory complications we have been trying to avoid.

More importantly, what does the patient want us to accomplish? If I were that patient I would not be worried about weight loss. In fact, I would look forward to feeling better and then eating to put the weight back on. Similarly, nitrogen balance would not matter to me, as the prospective randomized controlled trials (PRCTs) of NS have shown that improving nitrogen balance does not necessarily improve clinical outcome.¹⁵⁻¹⁶ My concerns would be whether or not I was going to live, how much more suffering I was going to have and for how long, and perhaps even how much this was all going to cost me.

The rationales for NS, while intuitively appealing, do not prove that it is good for patients. The association between malnutrition and a poor prognosis does not mean that malnutrition has a causative relationship. Making a phenomenon better does not necessarily make a patient better, as we have already seen for nitrogen balance. Efficacy can only be proven with PRCTs. A few such studies have been published. Parenteral NS did not improve outcome in infants with respiratory distress syndrome¹⁷ and it may have even worsened the postoperative course of cardiac patients on respirators.¹⁸ Supplemental oral feedings did not change pulmonary function (as measured by formal testing) in ambulatory patients with chronic obstructive pulmonary disease¹⁹⁻²² even though it did favorably affect body weight and anthropometric measurements.²⁰⁻²² (One of these unblinded studies did show that an improvement occurred in the subjective symptoms of "general well-being" and "breathlessness").²¹ In general, PRCTs demonstrating efficacy of NS in pulmonary disease still do not exist. Wouldn't it be comforting to have some?

In the meantime (a time which has already been longer than I had planned in 1984), many medical patients will continue to receive NS in the ICU. The article by Liggett and Renfro has provided information to allow physicians to be more cost-efficient in delivering it. I would remind those doctors about the dangers of trying to put these patients into positive nitrogen balance. More importantly, I must come around again and make the appeal to randomize half of the patients to a group which does not receive NS.

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A Sound Idea

Accurate clinical staging of lung cancer remains a challenging task. Prior to the late 1970s, plain chest radiography and bronchoscopy, sometimes supplemented by scintigraphic examinations or mediastinoscopy, were the mainstay of the staging evaluation. The primary difficulty lay in determining the status of regional lymph nodes. As computed tomography (CT) became available for routine use, the overall accuracy of clinical staging of lung cancer rose to 85-95 percent, depending upon the criteria employed in assessing nodal involvement. Certainly, this level of precision is acceptable, particularly as it surpasses the accuracy achieved in the clinical staging of many other solid tumors. Why, then, are improved staging modalities for lung cancer necessary?

The ever-increasing number of patients with bronchogenic carcinoma, the leading cause of cancer-related deaths in the United States, mandates improved methods of management. Recent trials of neoadjuvant chemotheraphy, with or without irradiation, exemplify the likely future of therapy for patients with regionally advanced disease. Techniques which permit accurate noninvasive staging are essential if precise assessment of disease response is to be achieved. Part of the difficulty with CT in determining nodal status lies in its dependence on size as a criterion for metastatic involvement. In addition to regional variability in the size of normal lymph nodes, other anatomic structures can also interfere with accurate identification and measurement of nodes, particularly those in the hilum and aortopulmonary window. Magnetic resonance imaging (MRI) has the potential to overcome some of these difficulties, but the present technology necessitates long acquisition times which result in motion artifacts and poor spatial resolution. Many of the problems with CT and MRI may be overcome through the use of ultrasonography.

The original initiative to use sound pulses in measuring distance apparently arose from the tragedy of the ocean liner Titanic, following which a variety of techniques were suggested to permit recognition of objects at distance.1 The threat of submarines to surface shipping during World War I led to the development of transducers capable of generating ultrasound which were subsequently used for depth measurement and the detection of fish shoals. Further refinements during World War II allowed the first true commercial use of ultrasound by Firestone for non-destructive materials testing.2 The inevitable jump to medical diagnostics and therapeutics took but a few years, with the original report of human soft tissue...