Correcting Data for Body Size May Confound Results

To the Editor:

We read with interest the article by Gallagher et al (June 2005) comparing cardiorespiratory fitness in patients with morbid obesity vs those with established heart failure. The authors found that obese subjects had maximum oxygen uptake (VO₂max) values very similar to those of nonobese heart failure subjects. We were surprised to find the published VO₂max values were expressed in “milliliters per kilogram per minute,” after correction for body weight, rather than the absolute uncorrected VO₂max (milliliters per minute).

For the same absolute value of VO₂max (milliliters per minute), subjects with greater body mass would have smaller corrected VO₂max (milliliters per kilogram per kilogram). In the absence of absolute VO₂max (milliliters per minute) or body weight data in the published article, it is possible to estimate the surrogate VO₂max by assuming height was not significantly dissimilar between the groups; thus, multiplying by body mass index can give a rough indication of the VO₂max of each group (Table 1).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Obese</th>
<th>Heart Failure</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>47.8</td>
<td>30.1</td>
<td>33.8</td>
</tr>
<tr>
<td>VO₂max per kilogram</td>
<td>17.8</td>
<td>16.5</td>
<td>21.3</td>
</tr>
<tr>
<td>Body mass index × VO₂max</td>
<td>850.8</td>
<td>496.7</td>
<td>719.9</td>
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</table>

These data suggest that the obese group may have the highest VO₂max, the opposite of the impression given by VO₂max per kilogram. We propose that uncorrected VO₂max (milliliters per minute) and weight data should be presented to avoid misleading information and conclusions.

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To the Editor:

Maximal oxygen consumption when measured absolutely (milliliters per minute) is a reflection of total body energy expenditure. In those individuals with a larger body habitus, higher absolute values are obtained based solely on having larger muscle mass. Clinicians will generally divide this absolute value by body weight in kilograms to allow for a more equitable comparison between individuals of variable sizes. Thus, when we express oxygen consumption in milliliters of oxygen per kilogram body weight (milliliters per kilogram per minute), we utilize this variable in an effort to compare the three groups, using the best single index of physical work capacity or cardiorespiratory fitness. Using the uncorrected oxygen consumption in milliliters per minute would have not allowed these important inferences on cardiorespiratory fitness.

As previous studies have identified a low level or aerobic fitness as an independent risk factor for all-cause and cardiovascular mortality, we believed our data reflected aerobic conditioning best when considered in this fashion. Additionally, by using oxygen consumption in milliliters per kilogram per minute, we were better able to compare our data with those standards previously reported for healthy individuals and for those with heart failure. In general, it has been reported that a maximum oxygen consumption of < 10.0 mL/kg/min signifies a poor prognosis in heart failure, with only a 50% 1-year survival rate. In addition, a maximum oxygen consumption of < 14.0 mL/kg/min is often used as a signal to consider cardiac transplantation. There are no such heuristics with uncorrected oxygen consumption.

Lastly, by reporting our data in milliliters per kilogram per minute, we are better able to relate the functional capacity to daily activities and/or exercise tolerance. Thus, when comparing our data to the metabolic costs of traditional activities of daily living (ie, walking or gardening), one is better able understand the significant aerobic impairment we observed in the obese.

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References

CO₂ Can Be Good for You!

To the Editor:

The study of the effects of noninvasive positive pressure ventilation to reduce chronic compensated CO₂ retention in COPD patients ignores the possible adaptive advantage of “resetting” the PCO₂ to a higher level than normal. As experts in the mechanics of COPD have argued in the past, chronic compensated CO₂ retention will allow for CO₂ homeostasis at a lower level of alveolar, and thus minute, ventilation. This may result in decreased dyspnea during exercise. Of course, these patients also received lower level of alveolar, and thus minute, ventilation. This may result in decreased dyspnea during exercise. Some time ago, we reported on a group of patients with very advanced COPD and quite high PCO₂ levels, ranging from 75 to 110 mm Hg (mean, 90 mm Hg) with partial bicarbonate compensation: mean HCO₃⁻ of 45 mEq/L and pH 7.32. Of course, these patients also received long-term nasal oxygen. All were functional to a remarkable degree in view of severe airflow obstruction: mean FEV₁, 0.41 L (range, 0.31 to 0.67 L). One patient worked daily as a road inspector! Mean survival was 17 months. I have also had many patients gain remarkable relief from dyspnea while receiving oxygen and exercise during pulmonary rehabilitation. It is as if their brain adjusts to the work of breathing by “living” at a high but compensated PCO₂. Thus, in my view, we should not focus on just one physiologic manifestation of COPD, and we must sometimes be reminded about the wisdom of nature.

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To the Editor:

We are grateful for the comments of Dr. Petty regarding our article in CHEST (August 2005) dealing with noninvasive positive-pressure ventilation (NPPV) treatment in stable patients with hypercapnic COPD. We agree with the statement that chronic compensated CO₂ retention reflects an adjustment mechanism that will allow for CO₂ homeostasis at a lower level of minute ventilation, thus resulting in decreased dyspnea, particularly if long-term oxygen therapy (LTOT) is applied. We also agree that this compensatory mechanism reflects the wisdom of nature. Nevertheless, chronic hypercapnia indicates long-term failing of the respiratory pump. Accordingly, COPD patients with higher PaCO₂ levels have a lower ventilatory reserve, and this has been suggested to be a limiting factor both during daily living and particularly during exacerbation.

Recently, we have shown that controlled nocturnal NPPV therapy is capable of increasing tidal and minute ventilation during the 3 subsequent hours of daytime spontaneous breathing and of sustaining increased tidal and minute ventilation until reconnection to the ventilator in the evening. Here, augmented alveolar ventilation caused a decrease of PaCO₂ followed by respiratory alkalosis that allowed renal elimination of elevated bicarbonate levels. This indicates reversion of the adjustment mechanism of chronic hypercapnia and may provide beneficial clinical effects in the addition to the positive effects gained by LTOT. Accordingly, health-related quality of life (HRQL) was reported to be higher in stable patients with hypercapnic COPD to whom NPPV leading to a decrease of PaCO₂ was administered in the addition to LTOT when compared to LTOT alone. Further, improvements of HRQL have been shown to be dependent on the ability of NPPV to lower bicarbonate levels following PaCO₂ reduction.

In stable patients with COPD, survival has yet not been shown to be improved when NPPV is applied in addition to LTOT compared to LTOT alone. However, studies that formed these results have used low ventilator settings that were not sufficient enough to increase alveolar ventilation and consecutively to decrease PaCO₂ values. As discussed in our article, NPPV using higher inspiratory pressures in a controlled mode is well-tolerated and capable of markedly improving hypercapnia in these patients. Survival rates were higher than previously reported. Since COPD is known to be one of the most important causes of chronic morbidity and mortality worldwide, we strongly think that the existing data regarding the potential benefits of NPPV that is aimed at more aggressively reducing PaCO₂ levels justify further controlled outcome studies.

In summary, COPD patients with chronic ventilatory failure metabolically and clinically adapt to chronic hypercapnia. LTOT further decreases dyspnea, increases PaO₂, and may thereby rest the exhausted respiratory pump, which could in part explain the improved prognosis. Moreover, NPPV aimed at a maximal reduction of PaCO₂ has been suggested to reverse this adjustment mechanism of chronically tolerated hypercapnia by augmentation of alveolar ventilation, thus decreasing PaCO₂ and bicarbonate levels. This has been suggested to provide additional clinical benefits regarding improvements of HRQL and prolongation of life, which need to be estimated in a more quantitative fashion following controlled trials.

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