Energy expenditure and rib cage-abdominal motion in chronic obstructive pulmonary disease

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ABSTRACT: The resting energy expenditure (REE) was measured by indirect calorimetry in 10 patients with chronic obstructive pulmonary disease (COPD) in stable clinical state and in 10 normal subjects. In order to avoid artefactually increased values, REE was obtained from prolonged measurements in steady state using a ventilated hood, without facial apparatus. The REE of COPD patients was significantly increased to 117% of predicted basal metabolic rate and to 125% of the control group values. Rib cage and abdominal movements were measured in COPD patients by inductance plethysmography and expressed with three indices: rib cage contribution to tidal volume (RC/VT), compartmental contribution to tidal volume (so RC/VT), and maximal compartmental amplitude/tidal volume ratio (MCA/VT). No correlation was found between REE and any of the indices of rib cage and abdominal motion. We conclude that the REE is increased in patients with COPD in stable clinical state, but is not related to the degree of abnormal rib cage-abdominal motion.

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Malnutrition is known to be frequently associated with chronic obstructive pulmonary disease (COPD). It has been shown to be more severe in hospitalized [1] than in ambulatory patients [2], and more severe in patients with, than without, acute respiratory failure [3]. According to dietary histories, the weight loss of COPD patients does not seem to be mainly the consequence of a reduced caloric intake [1, 2]. Therefore, a hypermetabolic state has been postulated to explain the development of malnutrition in these patients. Short-term measurements, using a mouthpiece to collect expired air, gave increased values of oxygen consumption in COPD patients [2, 4]. However, by the stimulating effect of the mouthpiece [5, 6] and the short time allowed for equilibration, such conditions of measurement may prevent the subjects from attaining their true level of resting energy expenditure (REE). Therefore, the first aim of this study was to determine if COPD patients present an increased REE, using standard methods of calorimetry.

The most likely cause of an increased energy expenditure in COPD is currently thought to be the respiratory muscles [7]. Indeed, they face a high load and they work in unfavourable mechanical conditions because of hyperinflation. As a consequence, patients with COPD often manifest abnormal respiratory muscle recruitment and some degree of chest wall distortion. Therefore, the second aim of this study was to evaluate the relationship between REE and different indices of rib cage and abdominal motion.

Subjects

Ten male patients with COPD, defined by the presence of chronic airflow obstruction and permanent dyspnoea on exertion, were studied. They were all in stable clinical state at the time of the study. The results of their lung function tests, including lung volumes as measured by body plethysmography (Erich Jaeger, Würzburg, FRG), are presented in table 1. Ten normal male subjects previously studied under the same experimental conditions in our laboratory were included as control group for the metabolic part of the study [8]. The physical characteristics of the two groups of subjects are presented in table 2. The protocol had been approved by the Hospital Ethical Committee.

Methods

For each subject, the body fat content was estimated by measuring skinfold thickness at four sites - bicipital, tricipital, subscapular, suprailiac - with a caliper.

Keywords: Calorimetry; chest wall mechanics; COPD; energy expenditure; respiratory muscles; work of breathing.
Cambridge, Maryland, content from body weight.

open-circuit indirect calorimetry, as previously described

IBW: ideal body weight. BMI: body mass index

Mean 49.4 176.1 68.5 97.8

Mean 57.7 172.1 60.8 90.9

Mean 49.4 176.1 68.5 97.8

IBW: ideal body weight. BMI: body mass index

(Lange caliper, Cambridge Scientific Industries Inc., Cambridge, Maryland, U.S.A.) [9, 10]. The lean body weight (LBW) was calculated by subtracting the fat content from body weight.

Energy expenditure was determined by computerized open-circuit indirect calorimetry, as previously described

[11]. A transparent plastic ventilated hood was placed over the subject's head and made airtight around the neck. To avoid air loss, a negative pressure was maintained in the hood. Ventilation was measured by a pneumotachograph (Hewlett-Packard 47303 A, USA). A sample of the air flowing in and out of the hood was continuously collected for analysis. The oxygen content was measured by a paramagnetic analyser (Magnos 4G, Hartmann & Braun, Frankfurt, FRG) and carbon dioxide content by an infrared analyser (Uras 3G, Hartmann & Braun, Frankfurt, FRG) whose accuracy was tested with calibration gases before each measurement. The system was characterized by a short response time (90% of the response after 3 min). The stability of the overall system was tested over periods of 4–5 hours and showed less than 1% variability. The oxygen consumption, the carbon dioxide production, and the respiratory quotient (RQ) were calculated. Taking into account Haldane's correction and the caloric equivalent of oxygen as determined by the RQ, the energy expenditure was calculated.

In all subjects, calorimetry was performed at 8 am after an overnight fast. In patients, oral theophylline was discontinued for 36 hours and inhaled adrenergics for 12 hours before the measurements. The subjects were lying supine with the head elevated at 30 degrees and placed in the hood. They were asked to remain completely quiet and they did not watch television or listen to radio. The experimenters ensured that the subjects did not move or sleep. After initiating the measurements, time was allowed for energy expenditure to stabilize. Resting energy expenditure (REE) was then determined over a 20 minute period of steady state.

The REE of COPD patients was analysed in two ways. First, it was expressed in kcal-min⁻¹ and compared to predicted basal metabolic rate by a paired t-test [12]. Second, the REE was expressed in kcal per 24 h per kg of lean body weight and was compared to that of the control group by an unpaired t-test.

After completion of the study, it was decided to perform thyroid tests in the COPD patients to rule out the possibility of hyperthyroidism in this group of subjects. By that time, one subject had died. Total T3 and T4,
T3 uptake, and TSH were measured in the remaining nine patients.

The movements of the rib cage and abdomen were measured in the COPD patients by respiratory inductance plethysmography (Respitrace, Ardsley, NY, USA) while the subjects were lying supine. The signals of rib cage (RC) and abdomen (AB) were displayed on an oscilloscope as the Konno-Mead diagram [13] and recorded along with their summation (SUM) on a paper recorder (Hewlett-Packard 7758A, USA). The inductance plethysmograph was calibrated according to the isovolume method [13]. The respiratory movements were analysed breath by breath during a five minute period and expressed with three indices [14-16]:

1. Rib cage contribution to tidal volume ratio (RCNT): This was computed by taking the extent of RC excursion between the points of minimal and maximal deflection of the SUM (VT) signal, dividing by the SUM (VT), and multiplying by 100.

2. Variability of compartmental contribution (SD RC/VT): The breath by breath variation in the relative contribution of RC and AB to tidal volume was determined by calculating the standard deviation of the RC/VT ratio.

3. Maximal compartmental amplitude to tidal volume ratio (MCA/VT): The maximal compartmental amplitude (MCA) is the sum of the absolute values of the RC and AB excursions, irrespective of their timing to the SUM (VT) signal. When the trough to peak amplitudes of the RC and AB signals are in phase, the ratio MCA/VT is equivalent to unity. When the trough to peak amplitude of the RC and AB signals are out of phase with each other, the ratio exceeds unity.

All reported values are mean±sd.

Results

Nutritional assessment

The two groups of subjects were not significantly different for age, height, weight, percent of ideal body weight, body mass index and body fat content (table 2). However, the COPD patients tended to be leaner, with six of them weighing less than 90% of ideal body weight.

<table>
<thead>
<tr>
<th>Subject</th>
<th>measured kcal·min⁻¹</th>
<th>predicted kcal·min⁻¹</th>
<th>measured %</th>
<th>kcal·24h⁻¹·kg·LBW⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1</td>
<td>1.10</td>
<td>1.10</td>
<td>100.0</td>
<td>29.54</td>
</tr>
<tr>
<td>2</td>
<td>1.52</td>
<td>1.07</td>
<td>142.1</td>
<td>36.24</td>
</tr>
<tr>
<td>3</td>
<td>1.10</td>
<td>0.99</td>
<td>111.1</td>
<td>36.41</td>
</tr>
<tr>
<td>4</td>
<td>1.16</td>
<td>0.95</td>
<td>122.1</td>
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</tr>
<tr>
<td>5</td>
<td>1.23</td>
<td>0.88</td>
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<tr>
<td>6</td>
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<td>0.93</td>
<td>110.8</td>
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<tr>
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<td>0.94</td>
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<td>0.97</td>
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<tr>
<td>9</td>
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<td>1.05</td>
<td>117.1</td>
<td>31.54</td>
</tr>
<tr>
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<td>1.27</td>
<td>1.10</td>
<td>115.5</td>
<td>28.79</td>
</tr>
<tr>
<td>Mean</td>
<td>1.163</td>
<td>0.998</td>
<td>116.7</td>
<td>32.79</td>
</tr>
<tr>
<td>sd</td>
<td>0.162</td>
<td>0.077</td>
<td>14.8</td>
<td>4.38</td>
</tr>
</tbody>
</table>

| Control |                     |                      |            |                      |
|---------|---------------------|----------------------|------------|                      |
| 11      | 1.19                | 1.21                 | 98.3       | 26.78                |
| 12      | 1.28                | 1.34                 | 95.5       | 25.96                |
| 13      | 0.97                | 1.12                 | 86.6       | 25.87                |
| 14      | 1.04                | 1.15                 | 90.4       | 27.73                |
| 15      | 0.95                | 1.12                 | 84.8       | 24.00                |
| 16      | 0.86                | 0.98                 | 87.8       | 23.82                |
| 17      | 0.99                | 1.01                 | 98.0       | 27.95                |
| 18      | 0.95                | 0.94                 | 101.1      | 29.11                |
| 19      | 0.97                | 1.03                 | 94.2       | 25.87                |
| 20      | 1.02                | 1.03                 | 99.0       | 26.23                |
| Mean    | 1.022               | 1.093                | 93.6       | 26.33                |
| sd      | 0.124               | 0.120                | 5.8        | 1.66                 |

Fig. 1. - Individual values of resting energy expenditure in COPD patients and in normal subjects.

Energy expenditure

The REE of COPD patients was 1.163±0.162 kcal·min⁻¹, corresponding to 116.7% of predicted basal metabolic rate (p<0.01). Expressed per mass of lean
body weight (LBW), the REE was 32.8±4.4 kcal·24h⁻¹·kgLBW⁻¹ in COPD patients and 26.3±1.7 kcal·24h⁻¹·kgLBW⁻¹ in normal subjects (fig 1, table 3). The REE of COPD patients was therefore 125% of REE of the normal subjects (p<0.001).

Thyroid function
Total T3 and T4, T3 uptake, and TSH were normal in the nine COPD patients.

Rib cage-abdominal motion (COPD patients)
On average, the rib cage contributed 54.4% to Vr, however with wide individual differences, ranging from 19.5% to 70.7%. The standard deviation of RC/Vr, expressing the variability of compartmental contribution, ranged from 5.9% to 19.7%. The MCA/Vr was between 1.00 and 1.06 in nine subjects, and was 1.67 in one. No correlation was found between the individual values of REE and any of the indices of rib cage-abdominal motion (figs 2, 3, and 4).

Discussion
In the past, an increase in energy expenditure was indirectly suggested in COPD patients by two types of experiment.

First, the oxygen cost of breathing during hyperpnoea has been repeatedly found increased in COPD patients relatively to normal subjects [17-22]. The method consists of measuring oxygen consumption at rest and at different levels of hyperpnoea. The oxygen cost of breathing is given by the slope of the relationship between oxygen consumption and ventilation. This method presents several problems and limitations. The oxygen cost of breathing is calculated for hyperpnoea and can only be evaluated by extrapolation for resting breathing. Moreover, the accuracy of the method is affected by several factors. Hyperpnoea cannot be sustained for a long period, so that measurements are of short duration. With the open-circuit method, the inspired-expired oxygen difference falls to very low values during hyperpnoea, so that analytical errors produce large errors in oxygen consumption. With the closed-circuit method, the oxygen consumption is given by the slope of the spirometric trace which can easily be affected by fluctuations in FRC [23].

Second, increased levels of oxygen consumption have also been reported at rest in COPD patients [2, 4, 24]. However, these results were obtained by short-term measurements of oxygen consumption using a mouthpiece. The use of a mouthpiece is likely to affect the measurements of REE for two reasons. Resting minute ventilation has been shown to increase when subjects breathe through a mouthpiece [5, 6]. Moreover, the discomfort of the mouthpiece prevents prolonged measurements, so that a true basal steady state is less certainly attained.

In order to minimize artefactual increments in REE, we used a ventilated hood and thereby avoided the use of any facial apparatus. The conditions of our study
were very close to those required for measuring basal metabolic rate (BMR). We use the term resting energy expenditure (REE) because the subjects were not studied shortly after awakening in their bed. However, the conditions allow comparison of the results with predicted values of BMR [12].

Under these experimental conditions, we found that REE was significantly increased to 117% of predicted BMR in this group of COPD patients in stable clinical state. This result is strikingly close to that reported in another COPD patients by Goldstein et al., while our study was underway [25]. These authors used similar methods to ours and found that REE was 116% of predicted BMR in their group of patients.

We were concerned that referring the REE of this group of COPD patients to predicted BMR might not be appropriate. Indeed, the predicted values of BMR are based on normal subjects, whereas the nutritional status of several of our patients was below normal. Consequently, their body composition might have been altered, with a relatively increased lean body mass. Since energy expenditure is closely related to lean body weight, it could possibly be expected to find a slight increase of REE in these subjects relative to subjects of normal body composition. Therefore, the REE was expressed in kcal per 24 h per kg of LBW and was compared between COPD patients and normal subjects. Our results confirm that the REE of COPD patients is significantly increased, by 25%, relatively to normal subjects.

Among the possible causes of increased REE in COPD patients, the most likely appears to be an extra energy expenditure of the respiratory muscles [7] In COPD, they not only face a higher load, but also work in unfavourable mechanical conditions because of hyperinflation. Their resting length being shorter than optimal, they need a greater activation to produce a given tension. In severe hyperinflation, the diaphragm is flattened and develops only little transdiaphragmatic pressure despite high muscular tension. To compensate for this reduced diaphragmatic efficacy, the inspiratory rib cage muscles are recruited and are responsible for some degree of chest wall distortion, which further increases the respiratory load.

There is at present no reliable indicator of energy expenditure of respiratory muscles. The work of breathing, as estimated from pleural pressure swing and tidal volume [26], is poorly correlated with the oxygen consumption of respiratory muscles in the presence of an increased load [27]. The reason is that it does not account for isometric contraction, velocity of shortening, duration of contraction, distortion of the rib cage, or work exerted on the abdomen [27]. Even when the latter factor is taken into account by estimating the work of breathing from separate rib cage and abdominal volume-pressure tracings [28], the correlation with oxygen consumption of respiratory muscles is weak [27]. In normal subjects, the diaphragm pressure-time index (TTd = Pdi/Pdi, - T/T, ) appears as a better indicator [27]. However, it has not been tested in COPD patients who recruit their inspiratory rib cage muscles markedly and, to a certain degree, their expiratory muscles as well.

In recent years, several indices have been developed to quantify the movements of the rib cage and abdomen and their degree of asynchrony or paradox [14–16]. The rib cage contribution to tidal volume ratio (RC/VT) is a reflection of the loss of diaphragm efficacy and of the degree of recruitment of inspiratory rib cage muscles. The standard deviation of this index (SD RC/VT) reflects the variability of compartmental contribution, rib cage and abdomen, to tidal volume and increases with "respiratory alternans". Finally, the maximal compartmental amplitude to tidal volume ratio (MC/VT) is a global index of asynchrony and paradox between the rib cage and abdomen.

These indices reflect the incoordination of breathing movements and indirectly the load faced by the respiratory muscles, as well as their operating conditions. For instance, the indices so RC/VT and MC/VT have been shown higher in unsuccessful than in successful weaning trials from mechanical ventilation [16]. Although they do not provide an estimation of the actual work of breathing, we hypothesized that they might show a correlation with the REE. The individual values of rib cage contribution (RC/VT) and variability in compartmental contribution (SD RC/VT) covered a wide span but showed no correlation with those of REE. Furthermore, REE was increased despite little asynchrony and paradox (MC/VT) in the majority of subjects. This lack of correlation may be interpreted in different ways. It is possible that these indices, although expressing the mechanical action of the respiratory muscles on the chest wall, do not reflect reliably the degree of tension developed in the different muscles. Furthermore, these indices do not account for the possible contraction of non respiratory muscles. Finally, it could be envisaged that part of the increased REE of COPD patients might originate from metabolic processes unrelated to respiratory muscle function.

References