Resting energy expenditure in children in a pediatric intensive care unit: comparison of Harris-Benedict and Talbot predictions with indirect calorimetry values


ABSTRACT  The use of prediction equations has been recommended for calculating energy expenditure. We evaluated two equations that predict energy expenditure, each of which were corrected for two different stress factors, and compared the values obtained with those calculated by indirect calorimetry. The subjects were 55 critically ill children on mechanical ventilation. Basal metabolic rates were calculated with the Harris-Benedict and Talbot methods. Measured resting energy expenditure was 4.72 ± 2.53 MJ/d. The average difference between measured resting energy expenditure and the Harris-Benedict prediction with a stress factor of 1.5 was −0.98 MJ/d, with an SD of 1.56 MJ/d and limits of agreement from −4.12 to 2.15; for a stress factor of 1.3 the average difference was −0.22 MJ/d, with an SD of 1.57 MJ/d and limits of agreement from −3.37 to 2.93. The average difference between measured resting energy expenditure and the Talbot prediction with a stress factor of 1.5 was −0.23 MJ/d, with an SD of 1.36 MJ/d and limits of agreement from −2.95 to 2.48; for a stress factor of 1.3, it was 0.42 MJ/d, with an SD of 1.24 MJ/d and limits of agreement from −2.04 to 2.92. These limits of agreement indicate large differences in energy expenditure between the measured value and the prediction estimated for some patients. Therefore, neither the Harris-Benedict nor the Talbot method will predict resting energy expenditure with acceptable precision for clinical use. Indirect calorimetry appears to be the only useful way of determining resting energy expenditure in these patients. Am J Clin Nutr 1998;67:74–80.

KEY WORDS  Energy metabolism, critical illness, artificial respiration, pediatric intensive care unit, indirect calorimetry, Harris-Benedict equation, Talbot equation, prediction equation, resting energy expenditure

INTRODUCTION

The metabolic response of critically ill pediatric (1–3) and adult (4, 5) patients is characterized by increased energy expenditure. This increase has traditionally been calculated by using formulas and correcting the values for injury factors (6–9). These injury factors were calculated originally from studies done on patients in whom resting energy expenditure (REE) was measured with indirect calorimetry. The measured values were then compared with values obtained from formulas or tables. The most commonly used formula to predict basal metabolic rate in adult patients is the Harris-Benedict equation (10). Talbot developed a table to calculate basal metabolic rate in children (11). Although formulas and predictive equations modified by injury factors have been reported (12–14), others have shown that energy expenditure cannot be calculated accurately in critically ill and mechanically ventilated patients in this manner (15–17).

The use of indirect calorimetry to measure energy expenditure allows for a more accurate monitoring of the patient’s energy needs and decreases the risks associated with underfeeding or overfeeding (18, 19). The technique of indirect calorimetry was used in many of the early studies of burn patients (20, 21) but until recently could be used only as a research tool. Problems with routine use of the technique included the handling of collection bags for expired gases, limitations on the use of high inspired oxygen concentrations, and technical difficulties in performing the technique in patients on mechanical ventilation. The development of portable, accurate devices for measuring oxygen consumption (VO₂) at the bedside permits individualized patient nutritional planning (22). The purpose of this study was to evaluate the use of two well-known predictive equations, corrected for stress factors of 1.5 and 1.3, in the calculation of REE in mechanically ventilated pediatric patients and to compare the values obtained with measured indirect calorimetry values by using methods comparison analysis as described by Bland and Altman (23).

SUBJECTS AND METHODS

Mechanically ventilated patients admitted to the pediatric intensive care unit of Texas Children’s Hospital were evaluated. The protocol was approved by the Institutional Review Board for clinical research as described by Bland and Altman (23).
For each test, mean values of predicted \( \text{VO}_2 \) and \( \text{VCO}_2 \) were computed according to the formulas used by Damask et al (25) and these values were compared with the mean values measured by the metabolic unit. The respiratory quotient (\( \text{VCO}_2/\text{VO}_2 \)) was calculated for each test and compared with the predicted value. Relative error was calculated as \( [(\text{measured value} - \text{predicted value})/\text{measured value}] \times 100 \). All measurements were corrected to standard temperature and pressure of dry gas. The results of this validation study are shown in Appendix B.

**Clinical studies**

A total of 45 patients were ventilated with the Servo 900C (Siemens) and 10 patients were ventilated with the Puritan-Bennett 7200 (Puritan-Bennett, Carlsbad, CA). Patients with uncuffed endotracheal tubes were placed in the best position to minimize air leaks; for patients with cuffed tubes the cuff was inflated. Any breath values with a difference > 10%, reflecting an airway leak, were not included in the calculation. Only studies for which ≥80% of the breaths were free of leaks were considered. The total number of breaths varied in relation to the respiratory rate of the patient. No minimum number of breaths was defined to calculate the measured REE.

Before the study, each patient was suctioned to clear secretions from the trachea. An average REE was then obtained and recorded. After a steady state period, the measurement was performed over 15–20 min. Steady state was defined by a variation between 5% and 10% in the average value for \( \text{VO}_2 \) and \( \text{VCO}_2 \) over a 5-min period (26). All measurements were performed between 0700 and 1400. Basal metabolic rate was calculated with use of the Harris-Benedict equation and the Talbot table, each corrected for stress factors of 1.5 and 1.3. Nutritional assessment was performed by calculating the ratio of actual weight to ideal weight-for-height (27). With use of this criterion, undernutrition was defined as a weight-for-height ratio < 0.90 and obesity as a weight-for-height ratio > 1.20. Source and amounts of energy intake were also recorded. To determine the effect of nutritional status and food intake on energy expenditure, comparisons were made between malnourished and obese patients, and among patients receiving enteral nutrition, intravenous fluids, or parenteral nutrition.

Age; weight; length of stay in the intensive care unit; duration of mechanical ventilation at the time of the study; use of catecholamines, sedatives, or muscle relaxants; and Pediatric Risk of Mortality (PRISM) score were all recorded. The PRISM score is a measure of severity of illness and is composed of physiologic and laboratory information on 14 variables (28). It is based on the observation that the amount and extent of physiologic dysfunction are related to the patient’s mortality risk. For each variable, a point is provided that directly reflects the contribution of that instability to mortality risk. A score of zero represents zero risk of mortality.

Ventilator data included were \( \text{FiO}_2 \), PEEP, tidal volume (\( V_t \)), ventilator rate (IMV), peak inspiratory pressure (PIP), and mean airway pressure (Paw). The ratio of the partial pressure of oxygen (\( \text{PaO}_2 \)) to \( \text{FiO}_2 \) (\( \text{PaO}_2/\text{FiO}_2 \)) was also calculated.

**Statistical analysis**

The methods comparison procedure described by Bland and Altman (23) was used to compare measured REE with Harris-Benedict and Talbot values, each corrected for stress factors of
1.5 and 1.3. Differences (Δ) between methods for individual patients were used to estimate 1) the relation between the difference and energy expenditure, for which regression analysis was used; 2) the bias (Δ) or mean difference between formula values and REE, for which paired t tests were used; and 3) the limits of agreement (Δ ± 2 SD), which indicate how much error might be expected when one of the formulas is used instead of measured REE. Descriptive variables are reported as means ± 1 SD. A value of $P < 0.05$ was considered significant.

**RESULTS**

Fifty-five patients (31 males and 24 females) participated in the study. The patients’ mean age was 5.7 ± 5.9 y and their mean weight was 21 ± 18 kg. A total of 43 patients had a medical condition: 10 had adult respiratory distress syndrome, 9 had viral pneumonia, 20 had bacterial pneumonia, and 4 had other conditions. Twelve patients were classified as postsurgical, with an average length of stay in the intensive care unit of 6 ± 4 d after the surgical procedure (five patients had intestinal surgery, three patients had liver transplants, two patients were twins who underwent separation, one patient had repair of a cardiac anomaly, and one patient had neuroblastoma resection). The mean length of stay in the intensive care unit for the patients at the time of the study was 12 ± 9 d, with a mean time on mechanical ventilation of 11 ± 9 d. The mean PRISM score on the day of study was 11 ± 5 points.

A total of 26 patients received at least one catecholamine (dopamine, dobutamine, or epinephrine); measured REE in these patients was not significantly different from that in patients who did not receive catecholamines (0.30 ± 0.13 compared with 0.29 ± 0.13 MJ·kg⁻¹·d⁻¹, respectively). Twenty-two patients received one sedative drug and 33 patients received two different sedatives, with no significant differences in energy expenditure between the two groups (0.26 ± 0.09 compared with 0.32 ± 0.15 MJ·kg⁻¹·d⁻¹, respectively). Sixteen patients received muscle relaxants and had a significantly lower mean measured REE than 39 patients who did not receive these drugs (0.21 ± 0.06 compared with 0.33 ± 0.14 MJ·kg⁻¹·d⁻¹, respectively; $P < 0.005$).

The patients’ average body temperature during the measurements was 37.4 ± 0.7 °C; three patients had body temperatures ≥38.5 °C and four patients had body temperatures ≤36.5 °C. The mean ventilator measurements were as follows: FiO₂, 0.45 ± 0.11; PEEP, 7 ± 4 cm H₂O; $V₁$, 12 ± 7 mL/kg; IMV, 17 ± 8 breaths per minute; PIP, 48 ± 16 cm H₂O; Paw, 12 ± 5 cm H₂O; and PaO₂/FiO₂, 204 ± 102.

The average ratio of actual weight to ideal weight-for-height for all patients was 1.09 ± 0.35. Thirteen patients were classified as undernourished (weight-for-height ratio: 0.84 ± 0.06), 31 patients as normally nourished (weight-for-height ratio: 1.63 ± 0.48), and 11 patients as obese (weight-for-height ratio: 1.00 ± 0.07). The measured REE in undernourished patients was 0.39 ± 0.16 MJ·kg⁻¹·d⁻¹ and was significantly different from that in normally nourished patients (0.28 ± 0.11 MJ·kg⁻¹·d⁻¹; $P < 0.01$) and obese patients (0.24 ± 0.11 MJ·kg⁻¹·d⁻¹; $P < 0.05$).

The average energy intake was 0.21 MJ·kg⁻¹·d⁻¹. Sixteen patients received oral feedings providing 0.32 ± 0.15 MJ·kg⁻¹·d⁻¹, 10 patients received intravenous fluids providing an energy intake of 0.05 ± 0.03 MJ·kg⁻¹·d⁻¹ only, and 29 patients received total parenteral nutrition providing 0.22 ± 0.12 MJ·kg⁻¹·d⁻¹. The measured REE in relation to source of food intake was not significantly different among the three groups: 0.33 ± 0.14, 0.29 ± 0.15, and 0.28 ± 0.12 MJ·kg⁻¹·d⁻¹ in patients fed orally, receiving intravenous fluids, and receiving total parenteral nutrition, respectively. Patients with a carbohydrate intake > 10 g·kg⁻¹·d⁻¹ ($n = 13$) had significantly higher energy expenditures than did patients with intakes < 10 g·kg⁻¹·d⁻¹ ($n = 42$): 0.38 ± 0.14 compared with 0.27 ± 0.12 MJ·kg⁻¹·d⁻¹, respectively, ($P < 0.01$).

The measured REE values and the Harris-Benedict and Talbot values corrected for stress factors of 1.5 and 1.3 are listed in Table 1. The methods comparison analysis (Table 2) showed that the Harris-Benedict equation corrected for a stress factor of 1.5 overestimated REE; in addition, the difference between the measured REE value and the Harris-Benedict value increased with basal metabolic rate ($P < 0.02$). The Harris-Benedict equation corrected for a stress factor of 1.3 also overestimated REE and the difference between measured REE value and the Harris-Benedict value increased with basal metabolic rate ($P < 0.001$) (Figure 1). The use of the Talbot table overestimated REE with a stress factor of 1.5 and underestimated REE with a stress factor of 1.3. For these two comparisons, the difference between measured REE and the Talbot value did not change significantly with basal metabolic rate.

**DISCUSSION**

The purpose of this study was to evaluate the use of formulas modified by accepted stress factors of 1.5 and 1.3 in critically ill pediatric patients on mechanical ventilation and to compare the values derived by use of these formulas with measured REE values by indirect calorimetry. The results showed clinically unacceptable differences between measured REE and predicted values for all four methods. The bias was significantly different in Harris-Benedict (with the stress factor of 1.5) and Talbot (with the stress factor of 1.3) predictions. In addition, for the Harris-Benedict equation the difference between measured REE and the predicted value increased as basal metabolic rate increased. This suggests that the Harris-Benedict equation overestimates energy expenditure in infants and small children.

The measurement of REE in critically ill patients on mechanical ventilation is important to adequately assess nutritional needs. Several studies found large differences between measured energy expenditure and values obtained with various predictive

**TABLE 1**

<table>
<thead>
<tr>
<th>Metabolic data</th>
<th>REE (MJ·kg⁻¹·d⁻¹)</th>
<th>Prediction, stress factor = 1.5 (MJ/d)</th>
<th>Prediction, stress factor = 1.3 (MJ/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harris-Benedict</td>
<td>4.72 ± 2.53</td>
<td>5.70 ± 2.04</td>
<td>4.94 ± 1.77</td>
</tr>
<tr>
<td>Talbot</td>
<td>4.29 ± 2.40</td>
<td>4.95 ± 2.77</td>
<td>4.29 ± 2.40</td>
</tr>
</tbody>
</table>

$\bar{x} \pm SD; n = 55$. REE, resting energy expenditure measured by indirect calorimetry. Harris-Benedict equation (10); Talbot table (11). (MJ × 1000)/4.184 = kcal.
equations (1–3). Formulas or tables, such as those of Harris-Benedict (10) and Talbot (11), have been used in critically ill patients to calculate energy expenditure and have been corrected for stress or injury factors (eg, for elective surgery, multiple fractures, severe infection, and burns). These factors range from 1.0 to 2.7, as reported by several authors (1, 29, 30).

Several studies of critically ill children (1, 7) and adults (13, 15) on mechanical ventilation, in whom energy expenditure was measured by indirect calorimetry, reported wide differences between the measured values and the Harris-Benedict or Talbot predicted values. These differences ranged from 40% to 100% above the basal metabolic rate and persisted in a range from 10% to 15% even after injury factors were applied.

The use of predictive equations based on regression analysis techniques has also been reported (7, 12–14). With this approach, energy expenditure is measured by indirect calorimetry or another method and compared with basal metabolic rate calculated by Harris-Benedict or Talbot equations with use of regression analysis. Other factors (eg, diagnosis of sepsis, presence of catecholamines, body temperature, and minute ventilation) have been included as independent variables to increase the accuracy of the estimated energy expenditure.

Even more important are bias and variability. Bias is the systematic distortion of a statistical result (31). In ideal conditions, the bias for a methods comparison pair should be zero, meaning that, on average, there is no difference in predicted energy expenditure calculated by a method, such as the Harris-Benedict equation or Talbot table, and measured energy expenditure calculated by indirect calorimetry. Variability is measured by calculating the SD of individual differences for all patients (SD$_\Delta$). The SD$_\Delta$ represents the reproducibility with which one can predict energy expenditure in an individual patient. For $\approx$95% of individual patients, energy expenditure calculated with a formula corrected for a stress factor is within $\pm$2 SD$_\Delta$ of the energy expenditure measured by indirect calorimetry. If this interval indicates differences that are not clinically tolerable, then the method is not useful.

The results of the present study differ from the findings of a report of 20 postsurgical infants (mean age: 14 mo) by Chwals et al (3). These authors found that measured energy expenditure was on average 53% lower compared with predicted energy expenditure. Our results showed on average a 53% higher measured REE in relation to predicted energy expenditure. These differences may be explained on the basis of a reduced metabolic response after injury (postsurgical condition) and the use of medications (narcotics and muscle relaxants). Note that the patients in the study by Chwals et al were younger than the patients in the present study (mean age: 70 mo). Also, the use of a specific equation to predict energy expenditure may modify the results. In the study by Chwals et al, predicted energy expenditure values based on age from Passmore were used (32); in the present study, predicted values based on weight and sex from Talbot were used.

The results of two other studies, one by Tilden et al (1) and the other by Phillips et al (2), were similar to those of the present study in relation to the degree of hypermetabolism, with values for the ratio of measured energy expenditure to predicted energy expenditure of 1.5 and 1.3, respectively. Both studies included mainly posttrauma head-injury patients as opposed to patients with adult respiratory distress syndrome, viral pneumonia, bacterial pneumonia, and postsurgical conditions as in the present study.

One component of energy expenditure is diet-induced thermogenesis. The magnitude of this effect depends on the amount and type of substrate ingested (33). In this study, there was no significant difference in REE indexed to body weight among the patients, regardless of the source of food intake (ie, oral formula, intravenous fluids, or total parenteral nutrition). This may be explained by the fact that all patients received on average only 80% of their measured energy expenditure as energy intake. Therefore, the effect of diet-induced thermogenesis on energy expenditure may not be completely apparent in this patient population. Although the patients fed orally had a higher (but not significantly so) REE indexed to body weight than did the rest of the patients, this group of patients had a significantly lower PRISM score than did the patients not fed orally, implying a less

![Average of REE and Harris-Benedict prediction value (MJ/d)](image)

**FIGURE 1.** Average of resting energy expenditure (REE) and Harris-Benedict prediction value (with a stress factor of 1.3) versus the difference between the two methods. The solid horizontal line is the estimated bias ($\Delta$). The dashed horizontal lines are $\pm$2 SD.
severe condition. Note that patients who received > 10 g carbohydrate·kg⁻¹·d⁻¹ had significantly higher energy expenditures, as was mentioned by other authors (33, 34).

Body weight can also affect energy expenditure. Reduced energy expenditure has been described during periods of fasting, in malnourished patients (33), and in obese patients (35). Our results showed on average a significantly higher energy expenditure per kilogram body weight in malnourished patients, which may be explained by the fact that the average age of the undernourished patients was significantly lower (3 y) than that of the obese patients. Energy expenditure per kilogram body weight of obese patients (average age: 10 y) was significantly lower than that of malnourished patients, probably as a reflection of decreased metabolic activity of the fat tissue and a lower energy expenditure in this group of older children.

Other factors that have been reported to influence energy expenditure in critically ill patients are catecholamines (36), narcotics (37), and muscle relaxants (38). Catecholamines increase VO₂ and consequently energy expenditure. In this study, neither VO₂ nor energy expenditure was significantly higher in patients who received catecholamines, most likely reflecting the influence of other factors that counteracted the action of catecholamines in increasing VO₂.

The use of sedatives and pain medications also has been said to decrease energy expenditure in critically ill patients. Measured REE in patients who received more than one drug, either a sedative or a narcotic, was not significantly different from that in patients who received only one drug. Finally, patients who received muscle relaxants at the time of the measurements had a 35% lower energy expenditure per kilogram body weight than did patients who did not receive paralytic agents; however, PRISM scores in these patients were not significantly different, implying that the lower energy expenditure could be secondary to the reduction of muscle metabolic activity and not explained by differences in severity of illness.

It is important to know the energy needs of critically ill patients to provide enough energy to avoid underfeeding but not an excess of the requirements for maintaining metabolic homeostasis, resulting in overfeeding. The results of this study show that the use of formulas with correction factors to estimate the energy needs of critically ill patients on high ventilator support (mean values: FiO₂ 0.45, PIP: 48 cm H₂O; and Paw: 12 cm H₂O) will either overestimate or underestimate energy needs depending on the method or stress factor used.

Studies in adults (39) and children (40) have shown that malnutrition is associated with increased morbidity and mortality. Among the complications associated with malnutrition are depressed immune function and increased susceptibility to infections, poor wound healing secondary to insufficient protein intake, loss of muscle mass resulting in alteration of respiratory function, and inability of mechanically ventilated patients to be weaned off ventilation. As malnutrition becomes severe, organ dysfunction results, leading to higher morbidity and mortality.

With respect to overfeeding, the deleterious effects include respiratory compromise, hepatic dysfunction, and an increased risk of mortality. The delivery of excess carbohydrate to critically ill patients causes increased VCO₂ resulting in a respiratory quotient > 1.0 (lipogenesis) and an increase in energy expenditure; this has been reported in several studies in adults (18) and infants (41). In this study, 20% of the patients had a respiratory quotient > 1.0. These patients had significantly higher carbohydrate intakes (11 compared with 7 g·kg⁻¹·d⁻¹) and significantly higher energy intakes in proportion to measured energy expenditure (1.10 compared with 0.72) than did patients with a respiratory quotient < 1.0. Several authors reported fatty liver infiltration, intrahepatic cholestasis, and abnormal liver function in patients who were overfed (42, 43). In a retrospective study of 24 postoperative patients by Vo et al (44), the mortality rate was significantly greater in the overfed group than in the group who received an energy intake equal to measured energy expenditure (40% compared with 28%, P < 0.05).

It is important to mention the limitations of this study. First, the population of subjects studied was composed of critically ill patients on high ventilatory support with a mean PRISM score of 11 and an average length of stay in the intensive care unit of 12 d at the time of the study. In a study by Sachdeva et al (45) that included 598 patients admitted to the pediatric intensive care unit who were both intubated and nonintubated, the mean PRISM score was 9 and the mean length of stay was ≈ 5 d. Even though this study showed that measured energy expenditure is more accurate than the use of predictive equations, it is possible that the use of these equations to estimate energy needs may be more accurate in less severely ill, nonintubated, pediatric patients with short lengths of stay in the intensive care unit, the possibility of overfeeding or underfeeding therefore being less likely. The second limitation of this study relates to the conditions under which the in vitro validation study was done. Although the results of the in vitro validation study were in an acceptable range for high ventilator settings (mean FiO₂ 0.60, mean PIP: 44 cm H₂O, and mean Paw: 20 cmH₂O; Appendix B), ventilator settings for 13% of the subjects studied were outside the settings used in the in vitro study.

The differences between the methods shown in this study indicate that neither the Harris-Benedict nor the Talbot method will predict REE within acceptable clinical limits in critically ill pediatric patients requiring mechanical ventilation. Therefore, the only way to determine REE for individual patients in this population is by indirect calorimetry.

REFERENCES

APPENDIX A
Relative error for oxygen consumption (\(\dot{V}O_2\)), carbon dioxide production (\(\dot{V}CO_2\)), respiratory quotient (RQ), and volume from the Douglas bag comparison\(^1\)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Relative error</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\dot{V}O_2)</td>
<td>(-0.4 \pm 1.5)</td>
</tr>
<tr>
<td>(\dot{V}CO_2)</td>
<td>(-1.1 \pm 1.1)</td>
</tr>
<tr>
<td>RQ</td>
<td>(-0.6 \pm 0.8)</td>
</tr>
<tr>
<td>Volume</td>
<td>(1.0 \pm 1.8)</td>
</tr>
</tbody>
</table>

\(^1\)\(\bar{x} \pm SD; n = 8\). The relative error was calculated as: \(\frac{(\text{measured value} - \text{Douglas bag value})}{\text{measured value}} \times 100\).

APPENDIX B
Relative error for oxygen consumption (\(\dot{V}O_2\)), carbon dioxide production (\(\dot{V}CO_2\)), and respiratory quotient (RQ) using a tidal volume of 250 mL\(^1\)

<table>
<thead>
<tr>
<th>PEEP (cm H(_2)O)</th>
<th>PIP (cm H(_2)O)</th>
<th>(\text{FiO}_2)</th>
<th>(\dot{V}O_2)</th>
<th>(\dot{V}CO_2)</th>
<th>RQ</th>
<th>(\dot{V}O_2)</th>
<th>(\dot{V}CO_2)</th>
<th>RQ</th>
<th>(\dot{V}O_2)</th>
<th>(\dot{V}CO_2)</th>
<th>RQ</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0.21</td>
<td>0.21</td>
<td>0.40</td>
<td>0.40</td>
<td>0.60</td>
<td>0.60</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>23 ± 4</td>
<td>4 ± 2</td>
<td>0.20 ± 0.8</td>
<td>2.7 ± 0.7</td>
<td>2.5 ± 0.8</td>
<td>1.16 ± 1.9</td>
<td>0.20 ± 0.9</td>
<td>2.1 ± 1.9</td>
<td>0.61 ± 3.0</td>
<td>-3.9 ± 1.6</td>
<td>-5.1 ± 3.2</td>
</tr>
<tr>
<td>5</td>
<td>37 ± 1</td>
<td>11 ± 1</td>
<td>-0.26 ± 1.2</td>
<td>2.1 ± 1.0</td>
<td>2.4 ± 0.9</td>
<td>1.25 ± 2.0</td>
<td>-0.16 ± 1.2</td>
<td>-2.6 ± 1.8</td>
<td>0.03 ± 3.4</td>
<td>-3.8 ± 1.2</td>
<td>-4.8 ± 3.3</td>
</tr>
<tr>
<td>10</td>
<td>41 ± 1</td>
<td>16 ± 1</td>
<td>-0.39 ± 1.3</td>
<td>2.5 ± 0.8</td>
<td>2.9 ± 1.4</td>
<td>1.76 ± 2.1</td>
<td>0.43 ± 0.9</td>
<td>-2.5 ± 2.0</td>
<td>0.42 ± 3.1</td>
<td>-2.9 ± 1.0</td>
<td>-4.3 ± 3.2</td>
</tr>
<tr>
<td>15</td>
<td>44 ± 1</td>
<td>20 ± 1</td>
<td>0.23 ± 1.4</td>
<td>2.9 ± 1.0</td>
<td>2.7 ± 1.4</td>
<td>2.02 ± 1.8</td>
<td>0.81 ± 0.9</td>
<td>-2.3 ± 1.7</td>
<td>0.90 ± 3.0</td>
<td>-2.4 ± 1.1</td>
<td>-4.2 ± 3.0</td>
</tr>
</tbody>
</table>

\(^1\)\(\bar{x} \pm SD; n = 9\). \(\text{FiO}_2\), fraction of inspired oxygen; PEEP, positive end expiratory pressure; PIP, peak inspiratory pressure; Paw, mean airway pressure. The relative error was calculated as: \(\frac{(\text{measured value} - \text{predicted value})}{\text{measured value}} \times 100\).