Inspiratory Work with and without Continuous Positive Airway Pressure in Patients with Acute Respiratory Failure

Jeffrey A. Katz, M.D.,* and James D. Marks, M.D.†

To compare the effects of continuous positive airway pressure (CPAP) with those of ambient end-expiratory pressure (T-tube) on lung mechanics and blood gas exchange, transpulmonary pressure ($P_{trans}$), tidal volume ($V_t$), respiratory frequency, and arterial oxygen and carbon dioxide tensions were measured in 16 spontaneously breathing patients recovering from acute respiratory failure. These variables were measured during breathing through a T-tube with 18, 12, and 6 cmH2O CPAP; and again during breathing through a T-tube. During all levels of CPAP, mean effective lung compliance ($C_{el}$) was higher and mean total pulmonary power during inspiration lower than during breathing through a T-tube before CPAP ($P < 0.05$). The data obtained at the level of CPAP producing maximum $C_{el}$ (optimum CPAP) were grouped and compared with values obtained during breathing through a T-tube. Mean total pulmonary power of inspiratory muscles during breathing through a T-tube before CPAP (0.7 ± 0.14 kg·m·min⁻¹) decreased during optimum CPAP (0.44 ± 0.07 kg·m·min⁻¹) and increased during breathing through a T-tube after CPAP (0.63 ± 0.12 kg·m·min⁻¹). Mean $V_t$ was higher (557 ± 63 ml vs. 474 ± 47 ml) and frequency lower (17.5 ± 1.6 breaths/min vs. 22.5 ± 2.5 breaths/min) during optimum CPAP than during breathing through a T-tube before CPAP, and inspiratory time was significantly longer. Mean minute ventilation was also lower during optimum CPAP (8.7 ± 0.8 l/min) than during breathing through a T-tube (9.6 ± 0.8 l/min); $P_{aCO2}$ did not change significantly. Mean alveolar-to-arterial oxygen pressure difference decreased significantly during optimum CPAP. The authors conclude that CPAP, when adjusted to the appropriate levels, improves lung mechanics in patients recovering from acute respiratory failure. Continuous positive airway pressure reduces total pulmonary power during inspiration and at the same time improves oxygen and carbon dioxide exchange. In these respects, it is preferable to breathing through a T-tube without CPAP. (Key words: Lung; compliance; function; respiratory failure. Ventilation: continuous positive-pressure breathing; failure; work of breathing.)

During weaning from mechanical ventilation, the physiologic effects of continuous positive airway pressure (CPAP) include an improvement in pulmonary oxygen exchange and an increase in functional residual capacity (FRC). The increase in FRC at low levels (5 cmH2O) of CPAP, as compared with spontaneous breathing at ambient end-expiratory pressure (through a T-tube), has been related to the abilishment (by the endotracheal tube) of an expiratory resistance produced by the glottis. The increased FRC with CPAP may improve lung mechanics and decrease the work of breathing. A decrease in the work of breathing during administration of CPAP has been demonstrated in babies but only suggested in adults.

This study compares, in adults, the inspiratory muscular work of breathing at various levels of CPAP with the inspiratory work of breathing through a T-tube. In addition, the timing, flow, and volume of each breath are compared.

**ABBREVIATIONS**

$C_{dyn}$ = dynamic lung compliance

$C_{el}$ = effective lung compliance

CPAP = continuous positive airway pressure

FRC = functional residual capacity

EEP = end-expiratory pressure

$P(A-O)_{aw}$ = alveolar-to-arterial oxygen pressure difference

$(\int P \cdot dT_t/T_t)$ = mean transpulmonary pressure during inspiration

$(\int P \cdot dT_t/T_t)\bar{V}_min$ = total pulmonary power (work per minute) during inspiration

PEEP = positive end-expiratory pressure

$P_{w}$ = transpulmonary pressure

$P_{p}E$ = minimum expiratory transpulmonary pressure

$P_{p}E'$ = expiratory transpulmonary pressure at instant of zero flow

$P_{p}I$ = maximum inspiratory transpulmonary pressure

$P_{p}I'$ = inspiratory transpulmonary pressure at instant of zero flow

$T_e$ = length of expiration

$T_i$ = length of inspiration

$T_{TOT}$ = length of the total respiratory cycle

$V$ = air flow

$\bar{V}_t$ = peak inspiratory flow

$V_E$ = peak expiratory flow

$V_{min}$ = minute ventilation

$V_t$ = tidal volume

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for breathing during CPAP and breathing through a T-tube.

Materials and Methods

The study was approved by the Committee on Human Research, and informed consent was obtained for each patient.

We studied 13 male and three female patients (18–70 yr of age; mean 45 yr) who had acute respiratory failure. All patients had undergone endotracheal intubation; 13 were being weaned from mechanical ventilation. Respiratory failure followed multiple trauma in five patients, head trauma in four, major laparotomy in three, severe pulmonary aspiration in two, and pulmonary edema and pneumonia in the remaining two. All patients were able to sustain a spontaneous tidal volume (V_T) of 300 ml and a maximum inspiratory pressure of at least −20 cmH_2O during airway occlusion. For two patients, chest roentgenograms were normal; the others revealed varying degrees of atelectasis and pulmonary infiltrates.

Continuous positive airway pressure was delivered using both continuous and demand flows from an air/oxygen blender regulated by an Emerson® demand valve. We adjusted the pressure regulator of the demand valve to maintain a constant flow of gas (30–60 l/min) at the selected end-expiratory pressure. In addition, this valve increases the flow rate (to as much as 100 l/min) during inspiration, on demand, to match the patient’s inspiratory flow. Gas flow to the patient passed through a Bird® humidifier and a disposable ventilator circuit. Exhalation occurred through an Emerson® positive end-expiratory pressure (PEEP) assembly. Thus, CPAP was ensured by the valve during inspiration and by the water column during exhalation.

The apparatus for breathing through a T-tube received its air/oxygen mixture from a Venturi® nebulizer. From the nebulizer, a corrugated plastic tube with a small reservoir limb open to ambient pressure led to the patient’s airway. A constant flow of fresh gas was delivered to prevent rebreathing. During the study, patients were in Fowler’s position (i.e., truncal elevation of 30–40 degrees), and no narcotics or sedatives were administered.

Measurements

We measured transpulmonary pressure (P_{w}) with a Statham PM131TC® differential pressure transducer (precalibrated with a water manometer) using pleural pressure estimated by the esophageal balloon method and airway pressure measured at the tracheal tube. Esophageal pressure was measured with a nasogastric esophageal balloon (National Catheter Corporation, Argyle, New York), 10 cm long and 3.8 cm in circumference. The tip of the balloon was placed in the mid to lower third of the esophagus by passing the empty balloon into the stomach and inflating it with 0.5 ml of air. Using continuous recording of esophageal pressure, we verified that the balloon was in the stomach by observing a positive deflection of esophageal pressure during inspiration. The balloon then was withdrawn to a position in the esophagus where the cardiac oscillations were minimal and the end-expiratory esophageal pressure most negative. This position was found to be 36–44 cm from the tip of the balloon to the nares. In addition, the position was verified on chest roentgenogram, using a radiopaque marker in the tip of the balloon. The balloon was evacuated of all air and inflated with 2 ml of air; then 1.5 ml was withdrawn, leaving a residual volume of 0.5 ml.

Air flow (V) was measured between the tracheal tube and the circuit connection with the use of a heated Fleisch #2 pneumotachograph; with the use of this instrument, the air flow/pressure relationship was found to be linear over the range of 10–90 l/min. Tidal volume was obtained by integration of the flow signal of the pneumotachograph, which was calibrated with a 500-ml syringe. The frequency responses of the pneumotachograph and pressure transducer were adjusted using the methods of Macklem.

Transpulmonary pressure, flow, and tidal volume were recorded on a Gould® recorder. Inspired oxygen fraction was measured by mass spectrometry (Perkin-Elmer®) and adjusted to 0.5. Arterial oxygen and carbon dioxide partial pressures (P_{aO2} and P_{aCO2}) were measured using a Corning 175® blood-gas analyzer and were corrected to the patient’s temperature. Measurements were taken in the last minute of five 15-min stages. During Stage 1 (“before CPAP”), patients breathed through a T-tube. During Stages 2, 3, and 4, patients breathed with 18, 12, and 6 cmH_2O CPAP, respectively. During Stage 5 (“after CPAP”), patients once again breathed through a T-tube. Before Stage 1, the trachea was suctioned to remove any sputum and the lung was hyperinflated. After Stage 5, in 10 patients, the esophageal balloon was evacuated and inflated with 0.5 ml of air to verify the reproducibility of esophageal pressure and volume over time. Transpulmonary pressure, flow, and tidal volume were compared before and after deflation and inflation of the esophageal balloon.

Calculations

Breath-by-breath analysis of pulmonary mechanics was accomplished by inspecting each breath recorded on the

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Dynamic lung compliance \( (C_{dyn}) \), \( V_T/(P_{IP}-P_{IP}E') \) was derived from the transpulmonary pressure difference between inspiration and expiration at the instant of zero flow, reflected on the pneumotachygraph tracing (fig. 1). Effective lung compliance \( (C_{eff}) \), \( V_T/(P_{IP}-P_{IP}E) \), was derived from the difference between the maximum inspiratory and minimum expiratory transpulmonary pressures (fig. 1). Because effective lung compliance includes both elastic and resistive forces expended in inflating the lung, total pulmonary work during inspiration can be approximated from this measurement.¹⁰

Total pulmonary power (work per minute) during inspiration \( (\int P \cdot dT_I/T_I) \) was determined using a planimeter to measure the area under the transpulmonary pressure tracing \( (\int P \cdot dT_I, \) fig. 1), dividing by \( T_I \) to obtain the mean pressure during inspiration and multiplying by \( V_{min} \). Studying patients with and without lung disease, McIlroy and Eldridge¹⁰ used this method to obtain values that were very close to the measured total work \( (\int P \cdot dV) \), where \( P \) is the mean transpulmonary pressure with respect to volume and \( dV \) is the tidal volume.

Elastic pulmonary power during inspiration \( (0.5[P_{IP}-P_{IP}E][V_{min}]) \) was computed by multiplying the difference in \( P_{IP} \) between inspiration and expiration at points of zero flow by the minute ventilation and dividing by 2. Non-elastic pulmonary power was computed by subtracting the elastic power from the total pulmonary power. As FRC and lung recoil pressures increase with CPAP \( (V_T \) being held constant for simplicity), total elastic work done on the lung during each inspiration increases markedly, although most of the work is done by the external equipment (fig. 2). That done by the inspiratory muscles decreases if lung compliance increases. We were interested in the muscular part of the inspiratory elastic work, measured by the procedures just described.

Data were analyzed using two-way analysis of variance; differences between groups were detected using the Newman-Keuls test.

**Results**

**Effect of CPAP on Lung Mechanics**

Expiratory transpulmonary pressures \( (P_{IP}E \text{ and } P_{IP}E') \) decreased progressively with each decrease in CPAP of 6 cmH₂O \( (P < 0.001) \) (table 1). During the patients' breathing through T-tubes after CPAP (Stage 5), \( P_{IP}E \) and \( P_{IP}E' \) were significantly greater than during breathing through a T-tube before CPAP (Stage 1) \( (P < 0.025) \). However, values were returning to pre-CPAP levels. During all levels of CPAP, mean effective lung compliance was higher and total pulmonary power during inspiration lower than during the patients' breathing through T-tubes.
before CPAP \( (P < 0.05) \) (table 1). Mean total pulmonary power and effective compliance did not differ significantly among the levels of CPAP. The level of CPAP that resulted in maximum effective compliance varied from patient to patient. Maximum effective compliance occurred at 6 cm\(H_2O\) CPAP in seven patients, at 12 cm\(H_2O\) CPAP in four patients, and at 18 cm\(H_2O\) CPAP in five patients. Because of this variation, and in order to group data for all patients at the optimum CPAP level, the data obtained at the level of CPAP coinciding with maximum effective compliance were grouped and compared with values obtained during the patients' breathing through T-tubes before and after CPAP.

During optimum CPAP, dynamic and effective lung compliances were significantly larger \( (P < 0.025) \) (fig. 3) and total pulmonary power during inspiration significantly less \( (P < 0.01) \) (fig. 4) than during the patients' breathing through T-tubes before CPAP. The decrease in total pulmonary power was caused primarily by a decrease in nonelastic power (fig. 4). Although mean elastic pulmonary power did not decrease significantly, the response varied—decreasing in 11 patients, increasing in three patients, and unchanged in two patients. No significant differences were found between dynamic lung compliance and total pulmonary power during the patients' breathing through T-tubes either before or after CPAP (figs 3 and 4). However, during the patients' breathing through T-tubes after CPAP (Stage 5), mean effective lung compliance was significantly larger \( (P < 0.05) \) than during breathing through a T-tube before CPAP (fig. 5).

**EFFECT OF CPAP ON TIDAL VOLUME, FLOW RATE, AND BREATHING PATTERN**

Several generalizations can be made when the mean data are plotted on a schematic spirogram (fig. 5). Points on the ordinate represent the mean tidal volume. The abscissa represents the length of inspiration \( (T_i) \) and expiration \( (T_e) \). Slopes of the lines connecting corresponding points to the left of the ordinate give the mean inspiratory flow rate, whereas slopes to the right of the ordinate give mean expiratory flow rates.

During optimum CPAP, tidal volume is higher \( (557 \pm 65 \text{ ml, } P < 0.05) \) than during breathing through a T-tube before CPAP \( (474 \pm 47 \text{ ml}) \) (fig. 5). No change occurred in either peak or mean inspiratory flow rates during CPAP, compared with breathing through a T-tube. Therefore, the increase in tidal volume was associated with a significantly longer \( T_i \) \( (P < 0.01) \) (fig. 5). During optimum CPAP, \( T_e \) was significantly longer than during the patients' breathing through T-tubes \( (P < 0.01) \); the result was a significantly decreased mean expiratory flow rate \( (P < 0.05) \) (fig. 5). Peak expiratory flow was also significantly \( (P < 0.01) \) lower during CPAP than during the patients' breathing through T-tubes (table 2). The ratio of \( T_i \) to \( T_{ Tot} \) \( (T_i/T_{ Tot}) \) during the patients' breathing through T-tubes before CPAP \( (0.37 \pm 0.02) \) decreased during optimum CPAP \( (0.33 \pm 0.02, P < 0.05) \) and increased during breathing through a T-tube after CPAP \( (0.36 \pm 0.02) \).

Mean respiratory frequency during the patients' breathing through T-tubes before CPAP \( (22.5 \pm 2.5 \text{ breaths/min}) \) decreased during optimum CPAP \( (17.5 \pm 1.6 \text{ breaths/min}, P < 0.05) \) and increased during the patients' breathing through a T-tube after CPAP \( (20.9 \pm 1.9 \text{ breaths/min}) \). Although the mean decrease in re-
Table 1. Pulmonary Mechanics and Blood Gas Exchange in 16 Patients with Acute Respiratory Failure Who Breathed with Various Levels of CPAP and Ambient Airway Pressure

<table>
<thead>
<tr>
<th>Stage</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>P &lt; 0.05*</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEP (cmH2O):</td>
<td>0</td>
<td>18</td>
<td>12</td>
<td>6</td>
<td>0</td>
<td>a, b, e-i</td>
</tr>
<tr>
<td>PpO (cmH2O):</td>
<td>10 ± 1.2</td>
<td>16.7 ± 1.0</td>
<td>13.8 ± 1.0</td>
<td>11 ± 1.0</td>
<td>10 ± 1.0</td>
<td>a, b, e-i</td>
</tr>
<tr>
<td>PpE (cmH2O):</td>
<td>-2.4 ± 1.3</td>
<td>7.3 ± 1.1</td>
<td>4.9 ± 1.1</td>
<td>2.0 ± 1.3</td>
<td>-0.5 ± 1.3</td>
<td>a-j</td>
</tr>
<tr>
<td>Ppl (cmH2O):</td>
<td>5.4 ± 0.9</td>
<td>14.1 ± 1.0</td>
<td>10.8 ± 1.0</td>
<td>8.1 ± 1.0</td>
<td>6.6 ± 0.8</td>
<td>a-c, e-j</td>
</tr>
<tr>
<td>PEEP (cmH2O):</td>
<td>-0.5 ± 1.1</td>
<td>8 ± 1.1</td>
<td>5.5 ± 1.1</td>
<td>2.9 ± 1.2</td>
<td>0.8 ± 1.1</td>
<td>a-j</td>
</tr>
<tr>
<td>Cdyn (ml/cmH2O)</td>
<td>37 ± 5</td>
<td>54 ± 6</td>
<td>56 ± 7</td>
<td>55 ± 7</td>
<td>46 ± 8</td>
<td>a-d</td>
</tr>
<tr>
<td>Cdyn (ml/cmH2O)</td>
<td>77 ± 10</td>
<td>80 ± 8</td>
<td>94 ± 10</td>
<td>96 ± 10</td>
<td>85 ± 12</td>
<td>b, c</td>
</tr>
<tr>
<td>Total inspiratory pulmonary power (kg · m · min⁻¹)</td>
<td>0.70 ± 0.14</td>
<td>0.50 ± 0.07</td>
<td>0.48 ± 0.08</td>
<td>0.48 ± 0.08</td>
<td>0.63 ± 0.12</td>
<td>a-c</td>
</tr>
<tr>
<td>Elastic power (kg · m · min⁻¹)</td>
<td>0.29 ± 0.04</td>
<td>0.27 ± 0.03</td>
<td>0.24 ± 0.03</td>
<td>0.23 ± 0.03</td>
<td>0.29 ± 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Nonelastic power (kg · m · min⁻¹)</td>
<td>0.41 ± 0.10</td>
<td>0.23 ± 0.04</td>
<td>0.24 ± 0.05</td>
<td>0.25 ± 0.06</td>
<td>0.34 ± 0.08</td>
<td>a-c</td>
</tr>
<tr>
<td>P(A−a)O₂ (mmHg)</td>
<td>170 ± 13</td>
<td>155 ± 12</td>
<td>154 ± 12</td>
<td>160 ± 12</td>
<td>169 ± 12</td>
<td>i</td>
</tr>
<tr>
<td>PaCO₂ (mmHg)</td>
<td>41 ± 1</td>
<td>43 ± 2</td>
<td>43 ± 2</td>
<td>42 ± 2</td>
<td>42 ± 2</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SE; n = 16.
* a = Stage 1 versus 2; b = Stage 1 versus 3; c = Stage 1 versus 4; d = Stage 1 versus 5; e = Stage 2 versus 3; f = Stage 2 versus 4; g = Stage 2 versus 5; h = Stage 3 versus 4; i = Stage 3 versus 5; j = Stage 4 versus 5. Data were analyzed using two-way analysis of variances; differences were determined using the Newman-Keuls test.

Respiratory frequency during optimum CPAP was 5 breaths/min, four patients had marked decreases of more than 10 breaths/min.

These typical effects of CPAP are shown in figure 6 for a representative patient who had multiple trauma, laparotomy, and acute respiratory failure. During 18 cmH2O CPAP (the level that resulted in maximum effective compliance), inspiratory and expiratory times were longer than during the patient's breathing through a T-tube. End-expiratory transpulmonary pressures were substantially higher during CPAP, and the transpulmonary swing in pressure between inspiration and expiration decreased at a slightly larger tidal volume, indicating an increase in both effective and dynamic lung compliances.

**Effect of CPAP on Ventilation and Blood-Gas Exchange**

Minute ventilation decreased from 9.6 ± 0.8 l/min during the patients' breathing through T-tubes to 8.7 ± 0.6 l/min during the patients' breathing with optimum CPAP (P < 0.05); PaCO₂ did not change significantly (fig. 7). Also, the mean alveolar-to-arterial oxygen partial pressure difference decreased significantly (P < 0.05) (from 170 ± 13 mmHg during the patients' breathing through T-tubes to 155 ± 13 mmHg during optimum CPAP).

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**Fig. 3.** Dynamic (Cdyn) and effective (Ceff) lung compliances during breathing through a T-tube and with optimum CPAP. Mean ± SE, n = 16. *P < 0.05 compared with breathing through a T-tube before CPAP. †P < 0.05 compared with breathing through a T-tube after CPAP.
EFFECT OF DEFLECTING AND REINFLATING THE ESOPHAGEAL BALLOON

During the patients' breathing through T-tubes after CPAP (Stage 5), transpulmonary pressures and effective and dynamic lung compliances were not affected significantly by deflation and reinflation of the esophageal balloon (table 3).

Discussion

The principal finding of this study is a decrease in the total pulmonary power during inspiration with CPAP in patients recovering from acute respiratory failure. The level of CPAP at which $C_{el}$ was greatest (optimum CPAP) varied from patient to patient. The calculated total pulmonary power during breathing with CPAP includes only work done by the subject and not the contribution of the CPAP system. The mean total pulmonary power during the patients' breathing through T-tubes before CPAP was 59% greater than during optimum CPAP. Mean total pulmonary power during the patients' breathing through T-tubes was approximately twice the normal value for healthy volunteers whose tracheas were not intubated. The increased level of work in our patients during breathing through a T-tube is consistent with their decreased dynamic lung compliance and increased $V_{min}$. During optimum CPAP, dynamic lung compliance increased, but inspiratory elastic pulmonary power did not decrease significantly. The observed decrease in total pulmonary power resulted from a decrease in the nonelastic component and, in 11 patients, a corresponding decrease in pulmonary elastic power. We assume that pulmonary elastic power did not change significantly, despite an increase in dynamic lung compliance, because the effects of increased tidal volume (and elastic work) offset the effects of decreased respiratory frequency.

An increase in FRC would decrease pulmonary resistance, thus decreasing nonelastic inspiratory work. Although pulmonary resistance was not calculated, a decrease is suggested during CPAP, because the difference between $P_{tp}$ and $P_{tp}'$ decreased while peak and mean inspiratory flow rates did not change.

CPAP may decrease inspiratory work in another way. If expiratory muscle activity sets FRC below the relaxation volume of the respiratory system during CPAP, then the contribution of CPAP to inspiration would increase and that of inspiratory muscles would decrease. Urbschelt et al. found that positive-pressure breathing evoked abdominal expiratory muscle activity in normal subjects whose tracheas were not intubated. Bishop et al. found that the increase in end-expiratory lung volume was less than that predicted by the passive pressure–volume relaxation curve. These two studies indicate that expiratory muscle activity may oppose an increase in FRC during breathing with CPAP.

Gherini et al. demonstrated a 45% decrease in total inspiratory work per minute in athletes (tracheas not intubated) given 10–20 cmH$_2$O CPAP. Functional residual capacity increased progressively with increments of CPAP, whereas dynamic lung compliance decreased progressively. However, with increasing levels of CPAP, FRC increased above the volume of the relaxed thoracic cage, and active expiratory work occurred as the pleural pressure increased above ambient levels.

We illustrated the effects of CPAP on lung mechanics by grouping data from the CPAP level producing maximum effective compliance. As indicated earlier, effective compliance includes both elastic and resistive forces that must be overcome to inflate the lungs. By grouping the data at optimum CPAP, the differences in variables (lung compliance, inspiratory pulmonary power, tidal volume, and respiratory frequency) between breathing through a
T-tube and with optimum CPAP are greater than the difference in variables during breathing through a T-tube and with any level of CPAP.

Evaluating our data and those of other authors, we believe that the mechanical and ventilatory responses to breathing with CPAP may depend on the initial value for FRC. When this is normal, further (i.e., hyperinflation) would decrease lung compliance, thereby increasing elastic work and \( O_2 \) cost of breathing. In healthy subjects breathing at high lung volumes, Roussos et al.\(^{17} \) found enhanced inspiratory muscle fatigue. The maximal active force a muscle can develop is a function of its length and

**Table 2. Timing, Volume, Peak, and Mean Air Flow in 16 Patients with Acute Respiratory Failure Who Breathed with Various Levels of CPAP and Ambient Airway Pressure**

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>( P &lt; 0.05 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEP (cmH(_2)O):</td>
<td>0</td>
<td>18</td>
<td>12</td>
<td>6</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>( T_I ) (s)</td>
<td>1.08 ± 0.07</td>
<td>1.17 ± 0.08</td>
<td>1.17 ± 0.07</td>
<td>1.15 ± 0.06</td>
<td>1.12 ± 0.06</td>
<td>a, b</td>
</tr>
<tr>
<td>( T_E ) (s)</td>
<td>2.32 ± 0.51</td>
<td>3.03 ± 0.69</td>
<td>2.86 ± 0.59</td>
<td>2.65 ± 0.63</td>
<td>2.24 ± 0.7</td>
<td>a, b, g, i</td>
</tr>
<tr>
<td>( T_I/T_R )</td>
<td>0.37 ± 0.02</td>
<td>0.33 ± 0.02</td>
<td>0.33 ± 0.02</td>
<td>0.35 ± 0.02</td>
<td>0.36 ± 0.02</td>
<td>a, b, f, g, i</td>
</tr>
<tr>
<td>Respir. freq. (breaths/min)</td>
<td>22.5 ± 2.5</td>
<td>18.3 ± 1.9</td>
<td>18.6 ± 1.9</td>
<td>19.3 ± 1.7</td>
<td>20.9 ± 1.9</td>
<td>a-c</td>
</tr>
<tr>
<td>( V_T ) (ml)</td>
<td>474 ± 47</td>
<td>544 ± 64</td>
<td>523 ± 53</td>
<td>513 ± 47</td>
<td>517 ± 43</td>
<td>a</td>
</tr>
<tr>
<td>( V_T/T_I ) (ml/s)</td>
<td>440 ± 26</td>
<td>460 ± 26</td>
<td>448 ± 27</td>
<td>448 ± 26</td>
<td>464 ± 25</td>
<td>NS</td>
</tr>
<tr>
<td>( V_T/T_E ) (ml/s)</td>
<td>266 ± 27</td>
<td>225 ± 22</td>
<td>250 ± 24</td>
<td>243 ± 23</td>
<td>270 ± 24</td>
<td>a, b, g, i</td>
</tr>
<tr>
<td>( V_I ) (l/min)</td>
<td>36 ± 2</td>
<td>37 ± 2</td>
<td>36 ± 2</td>
<td>36 ± 2</td>
<td>37 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>( V_E ) (l/min)</td>
<td>29 ± 2</td>
<td>21 ± 1</td>
<td>21 ± 1</td>
<td>24 ± 2</td>
<td>29 ± 2</td>
<td>a-c, f, j</td>
</tr>
</tbody>
</table>

*Values are mean ± SE; \( n = 16 \).*

\( a \) = Stage 1 versus 2; \( b \) = Stage 1 versus 3; \( c \) = Stage 1 versus 4; \( d \) = Stage 1 versus 5; \( e \) = Stage 2 versus 3; \( f \) = Stage 2 versus 4; \( g \) = Stage 2 versus 5; \( h \) = Stage 3 versus 4; i = Stage 3 versus 5; j = Stage 4 versus 5. Data were analyzed using two-way analysis of variances; differences were determined using the Newman-Keuls test.
OPTIMUM CPAP

Fig. 6. Effects of 18 cmH2O CPAP on inspiratory and expiratory timing, flow rates, changes in transpulmonary pressure, and tidal volume for a representative patient (see text for details).

Fig. 7. Minute ventilation ($V_{\text{min}}$) and arterial carbon dioxide partial pressure ($P_{a\text{CO}_2}$) during breathing through a T-tube and with optimum CPAP.

geometric configuration. As lung volume increases, inspiratory muscles shorten and the diaphragm flattens, thus decreasing the maximal force developed.\textsuperscript{18}

In contrast, when the initial FRC is below normal because of processes causing regional atelectasis, further lung distension may be advantageous. For example, our patients had abnormal lung function and probably decreased FRC. These conditions were indicated by dynamic lung compliances that were 55% of normal and by the presence of pulmonary infiltrates on chest roentgenogram. During breathing at optimum CPAP, dynamic lung compliance increased and total pulmonary power decreased. Although we did not measure indices of inspiratory muscle fatigue, $T_1/T_{TOT}$ decreased during optimum CPAP. This decrease would allow the inspiratory muscles a longer period of time to recover and possibly would decrease the risk of fatigue.\textsuperscript{19,20}

The ventilatory response to breathing with CPAP also may depend on starting lung volume and the subsequent lung distention with CPAP. In healthy volunteers with nonintubated tracheas (who were seated within a body plethysmograph in which pressure was lowered to equivalent levels of transpulmonary pressure), Bishop et al.\textsuperscript{15} found the ventilatory response to 5, 10, or 15 cmH2O CPAP to be an increase in $V_{\text{min}}$ achieved as a result of both increased $V_T$ and increased respiratory frequency. A similar and progressive increase in $V_{\text{min}}$ was observed by Urbschleit et al.\textsuperscript{14} in volunteers (tracheas not intubated).
Table 5. Transpulmonary Pressures, Tidal Volume, and Compliances in 16 Patients with Acute Respiratory Failure after Inflation and Deflation of the Esophageal Balloon

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th><em>p</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ppl (cmH₂O)</td>
<td>9.2 ± 1.0</td>
<td>9.0 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>P₂ (cmH₂O)</td>
<td>6.0 ± 0.7</td>
<td>5.4 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>P₃ (cmH₂O)</td>
<td>-0.3 ± 1.2</td>
<td>-0.9 ± 1.2</td>
<td>NS</td>
</tr>
<tr>
<td>P₄ (cmH₂O)</td>
<td>0.84 ± 1.2</td>
<td>0.14 ± 1.11</td>
<td>NS</td>
</tr>
<tr>
<td>V₁ (ml)</td>
<td>495 ± 26</td>
<td>509 ± 28</td>
<td>NS</td>
</tr>
<tr>
<td>C₁ (ml/cmH₂O)</td>
<td>50 ± 11</td>
<td>49 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Cdyn (ml/cmH₂O)</td>
<td>94 ± 20</td>
<td>93 ± 20</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SE; n = 10.

* Paired t test.

During 10 and 20 cmH₂O CPAP. At 10 cmH₂O CPAP, V̄ₘᵢᵣ decreased by 50%, whereas at 20 cmH₂O CPAP, V̄ₘᵣ increased by 300%. This increase in V₁ was believed to be related to passive lung inflation associated with a rise in mean inspiratory flow. The increased respiratory frequency was achieved mainly by reducing Tₑ independent of any change in T₁. The subsequent increase in mean expiratory flow probably results from recruitment of abdominal muscles that exists passive lung inflation. Tobin et al. studied seven patients having normal lungs who breathed spontaneously after being given mechanical ventilation for a variety of reasons. During breathing at 10 cmH₂O CPAP, V₁ and mean inspiratory flow were significantly higher, but respiratory frequency did not change.

During breathing at optimum CPAP, our patients had an increase in V₁, as shown by Bishop et al., Urbach et al., and Tobin et al. However, the increase was associated with a longer T₁ and no change in mean inspiratory flow. In contrast to normal volunteers with non-intubated tracheas and to patients with normal chest roentgenograms and intubated tracheas, our patients had decreased respiratory frequency at all three levels of CPAP. Venkatesh et al. previously demonstrated that respiratory frequency was lower during breathing with CPAP (10–25 cmH₂O) than during breathing through a T-tube in patients with acute respiratory failure.

The decrease in respiratory frequency in our patients was primarily due to prolongation of Tₑ. In contrast to flow rates during inspiration, mean peak expiratory flow decreased during CPAP. The decreased expiratory flow does not support significant recruitment of abdominal muscle activity and active opposition of end-expiratory lung volume increase during breathing with CPAP in our patients. The increased Tₑ and decreased expiratory flow are consistent with an increase in dynamic lung compliance and a decrease in elastic recoil of the lung.

With CPAP, an increase in lung compliance and a decrease in total pulmonary power during inspiration might be expected to be accompanied by a decrease in respiratory frequency and a stable or decreased V̄ₘᵣ at constant P₃. These changes would be in accord with improved lung mechanics secondary to appropriate lung inflation. However, the observed ventilatory response and changes in lung mechanics could be attributed to other factors. For example, the reduction in respiratory frequency seen with CPAP could be related to an increased resistance to exhalation through the Emerson* PEEP column. Depending, in part, on reflex gas exchange consequences, the resulting increase in expiratory time-constant might contribute to increased Tₑ, decreased mean expiratory flow, lower frequency, increased V₁, and changes in V̄ₘᵣ. Our study did not address these issues.

To minimize any additional work required to breathe from CPAP delivery systems, the change in airway pressure during inspiration should be minimal. In this study, airway pressure decreased during inspiration by approximately 2 cmH₂O as a result of decreasing the effect of flow resistance in the Emerson* PEEP assembly. Recent studies indicate that CPAP delivery systems vary widely in the amount of additional work required of a patient. Therefore, the type of CPAP delivery system might also be responsible for differences observed in breathing patterns between studies.

Clinicians usually use mechanical ventilation to treat patients who have acute respiratory failure and are euapnic but tachypneic and use PEEP to treat hypoxemia. The data from this study suggest that when adjusted to the appropriate level, CPAP decreases respiratory frequency and inspiratory pulmonary power while P₃ does not change. During optimum CPAP the decreased V̄ₘᵣ, at constant P₃, is most likely the result of the decrease in dead space ventilation and not significantly related to any change in CO₂ production. In addition, CPAP will improve pulmonary oxygen exchange.

The calculation of inspiratory pulmonary power is based on accurate measurement of esophageal and transpulmonary pressures. Our patients were in a semisitting position, and their absolute esophageal pressures were abnormally positive, presumably because of the weight of the heart and other mediastinal contents. During CPAP, as lung volume increased, any compression of the esophagus by the mediastinum might have decreased and thus changed the distribution of air within the esophageal balloon. Such a change could have artificially affected esophageal pressure and therefore our estimates of pulmonary compliance. However, Milic-Emili et al. and Sasaki et al. demonstrated that, although absolute esophageal values differed, the slope of the pressure-volume curve of the lungs was not distorted significantly over a wide range (20–80% of vital capacity) when the sitting and supine positions were compared. Their data suggest that esoph-
ageal balloon pressure would represent the overall elastic behavior of the lung. Another potential methodologic problem is that balloon air volume may be lost or redistributed over the time course of the study. We do not believe that either possibility was a problem. At the end of the study, 0.5 ml of air still remained in the balloon—removing and reinjecting it did not alter the apparent transpulmonary pressures and dynamic compliances.

Breathing with high levels of CPAP may adversely affect cardiac output, primarily by decreasing venous return. Although circulatory effects may be important, no deleterious clinical effects on circulation were observed during this study.

In conclusion, total pulmonary power during inspiration decreased in a group of patients recovering from acute respiratory failure when those patients were breathing with CPAP. This reduction was observed at all three levels of CPAP and was associated with an increase in effective compliance and in dynamic lung compliance, the latter at the two lower levels of CPAP. The level of CPAP resulting in maximum effective compliance varied from patient to patient. In contrast to normal volunteers who hyperventilate and resist passive lung inflation during positive-pressure breathing, our patients increased tidal volume, as a result of a longer inspiration, and decreased respiratory rate, primarily as a result of a longer expiration. At the maximum effective compliance, V_{min} decreased without a change in P_{aco2}. Thus, for patients recovering from acute respiratory failure who are being weaned from mechanical ventilation, CPAP not only increases FRC but also improves lung mechanics and reduces pulmonary power during inspiration. In these respects, breathing with CPAP is preferable to breathing through a T-tube.

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