Effects of chest wall counterpressures on lung mechanics under high levels of CPAP in humans

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Effects of chest wall counterpressures on lung mechanics under high levels of CPAP in humans. J. Appl. Physiol. 83(2): 591–598, 1997.—We assessed the respective effects of thoracic (TCP) and abdominal/lower limb (ACP) counterpressures on end-expiratory volume (EEV) and respiratory muscle activity in humans breathing at 40 cmH2O of continuous positive airway pressure (CPAP). Expiratory activity was evaluated on the basis of the inspiratory drop in gastric pressure (ΔPga) from its maximal end-expiratory level, whereas inspiratory activity was evaluated on the basis of the transdiaphragmatic pressure-time product (PTPdi). CPAP induced hyperventilation (+320%) and only a 28% increase in EEV because of a high sure-time product (PTPdi). CPAP induced hyperventilation was evaluated on the basis of the transdiaphragmatic pressure change during 0 and 40 cmH2O of CPAP, respectively. When ACP, TCP, or both were added, hyperventilation decreased and PTPdi increased (19 ± 5, 21 ± 5, and 35 ± 7 cmH2O·s−1·cycle−1, respectively), whereas ΔPga decreased (19 ± 6, 9 ± 4, and 2 ± 2 cmH2O, respectively). We concluded that during high-level CPAP, TCP and ACP limit lung hyperinflation and respiratory muscle activity and restore diaphragmatic activity.

continuous positive airway pressure; +Gz tolerance; positive pressure breathing; lung volumes; pattern of breathing; respiratory muscle activity

TO IMPROVE THE ABILITY of combat aircraft pilots to tolerate the physiological disturbances induced by accelerations with gravitational inertial forces acting from head to foot (+Gz, acceleration), counterpressure applied around the abdomen and the lower limbs by inflating anti-G pants was proposed more than 10 years ago (9). However, this failed to prevent hemodynamic disturbances with loss of consciousness when pilots were subjected to the rapid onset (>8 +Gz/s) and sustained accelerations observed with agile combat aircraft (27). Positive pressure breathing applied as continuous positive airway pressure (CPAP) has been proposed as an additional protection against such accelerations because positive intrathoracic pressure is transmitted to the vascular system, increasing systemic arterial pressure even when cardiac output is decreased (6, 8, 30). CPAP can be applied at levels of up to 90 cmH2O at 9 +Gz (11). Higher levels (110–120 cmH2O) can be used to protect pilots against acute hypoxia after accidental cockpit decompression at altitudes of up to 20 km (5). Application of CPAP levels above 35–45 cmH2O requires use of a chest garment inflated at the same pressure to avoid excessive expansion of the lungs and rib cage (1).

To our knowledge, the efforts on ventilatory mechanics of CPAP protocols presently used in aeronautics (CPAP with thoracic (TCP) and abdominal/lower limb counterpressures (ACP)) have not yet been described. The purpose of our study was to determine the changes in end-expiratory volume (EEV) and the activity of respiratory muscles induced by short periods of CPAP up to 40 cmH2O with or without TCP and/or ACP in humans. We hypothesized that in the absence of counterpressure the ventilatory pattern would be characterized by active expiration and passive inspiration to limit lung expansion but that application of TCP and/or ACP would restore inspiratory activity and reduce expiratory efforts. To check this hypothesis, we assessed the specific and synergistic effects of TCP and ACP on respiratory muscle activity and lung volumes.

METHODS

Six healthy adults (4 men and 2 women; age 36 ± 5 (SD) yr; height 175 ± 7 cm; weight 72 ± 12 kg) volunteered for this study. All were naive to the purpose of the study. The experimental protocol was approved by the Human Ethics Committee of the Henri Mondor Hospital, Créteil, France. All subjects underwent a medical evaluation before participation. Four had prior experience with CPAP up to at least 60 cmH2O. Furthermore, all study subjects attended a training session with the pressurized equipment a few days before the experiment. This session consisted of 3-min periods of CPAP at breathing pressures of 10, 20, 30, and 40 cmH2O with a 5-min recovery time between each period.

Experimental Setup

Equipment. Subjects sat in an ejection seat (type MK4, Martin Baker Aircraft, Higher Denham, Middlesex, UK). They wore an aeronautical suit and were connected to a CPAP circuit. Fitting of the garment and the verification of the CPAP were performed by the same trained technician.

The suit, as shown in Fig. 1, consisted of anti-G pants (model 830, ARZ EFA, Aérazur, Issy-les-Moulineaux, France) with five interconnected inflatable compartments (one on the anterior abdomen, one around each thigh, and one around each calf) to provide TCP and a jerkin (VHA90, Aérazur) with an inflatable chest compartment over the anterior and lateral rib cage to provide TCP. CPAP was provided through an oronasal mask (82AK, Ulmer, Les Ulis, France), which was tightened by using a harness to avoid leakage. The inspiratory circuit was connected to the chest compartment so that the pressure measured in the mask (Pmask) was equal to TCP.

To provide CPAP levels of 10, 20, 30, and 40 cmH2O at sea level, we used the device designed for the Dassault Mirage
Cardiorespiratory monitoring. Mask and jerkin pressures were measured by using pressure transducers (model H5035, ±100 cmH\textsubscript{2}O, Enertec-Schlumberger, Montrouge, France). Anti-G pants pressure was measured by using a ±500 cmH\textsubscript{2}O pressure transducer (Enertec-Schlumberger).

Flows were measured by using no. 2 Fleisch pneumotachographs (Lausanne, Switzerland) connected to the inspiratory and expiratory circuits, respectively. The pneumotachographs were connected to pressure transducers (±2 cmH\textsubscript{2}O; model CH5051, Enertec-Schlumberger) and auxiliary conditioners for pressure transducers (model CA9036, Enertec-Schlumberger). Tidal volume (V\textsubscript{T}) was calculated by integration of the flow signal (integrator model 13-G4615–70H, Gould, Cleveland, OH).

Heart rate (HR) and systolic (SBP) and diastolic arterial blood pressures (DBP) were monitored by using a fingertip blood pressure monitor (Finapres, Ohmeda, Englewood, CO). The forearm was placed on an armrest, adjusted so that the finger and the sensor were at heart level.

All monitored parameters were recorded simultaneously (OR 2300A strip-chart recorder, Yokogawa, Japan; RD 200T data magnetic recorder, TEAC) and digitized at 128 Hz (Acqknowledge software and device, Biopac Systems, Santa Barbara, CA).

EEV determination. An open-circuit N\textsubscript{2} dilution method was used to obtain EEV (12). The subject breathed pure \textit{O\textsubscript{2}}, and the total expirate was collected in a Douglas bag over 7 min. The expiratory volume (V\textsubscript{E}) and N\textsubscript{2} concentration ([N\textsubscript{2}]) of the expirate were measured (4). The fractional [N\textsubscript{2}] in the lung was assumed to be 0.81 at the onset of the dilution procedure, and a correction was made for tissue N\textsubscript{2} elimination (4). Lung volume was calculated as V\textsubscript{E} \cdot [N\textsubscript{2}]/0.81. A valve was placed on the inspiratory circuit to allow switching between the CPAP device (supplied with air) and a tank filled with 100\% \textit{O\textsubscript{2}} (at ambient pressure). Because of the characteristics of the \textit{N\textsubscript{2}} method to determine EEV, it was not necessary to maintain CPAP during the 7 min required to collect the expired gas. The requirements were that CPAP had to be maintained until the switch from air to \textit{O\textsubscript{2}} and that this switch had to occur at the volume to be measured, i.e., at EEV. V\textsubscript{E} and [N\textsubscript{2}] were measured by using a gas volumeter (OSI, Maurepas, France) and a mass spectrometer (Mediflex, VG Medical Systems, Middlewich, Cheshire, UK), respectively. To ensure that no air leaked from the room into the mask during expirate collection, the decreasing rate of fractional expiratory [N\textsubscript{2}] was monitored continuously by using spectrometry.

Respiratory muscle activity and lung mechanics. Gastric (P\textsubscript{ga}) and esophageal (P\textsubscript{es}) pressures were measured by using a probe equipped with two piezoelectric sensors (T7B/2, Gaeltec, Dunvegan, Isle of Skye, UK). We evaluated the frequency-response of these piezoelectric sensors and observed that no change in the amplitude or the phase lag of the signal occurs with frequency as high as 10 Hz. The probe was inserted through a nostril and advanced until the distal sensor was in the stomach and the proximal sensor was in the lower third of esophagus. Appropriate placement of the esophageal balloon was verified with an occlusion test. To verify that positioning of the gastric transducer was correct, gentle pressure was applied manually to the subject's stomach to observe fluctuations in P\textsubscript{ga}, and the subject was asked to drink water to verify that the sharp increase in P\textsubscript{es} due to contraction of the esophagus was not observed on the P\textsubscript{ga} tracing. The pressure probe was calibrated before the first test by using a pressure generator (Yew 2856, Yokogawa European Headquarters, Amersfoort, The Netherlands).

2000 aircraft (regulator IN 439–5, Intertechnique, Plaisir, France). This device consisted of a breathing module providing TCP and CPAP and of an anti-G module providing ACP. ACP levels were much higher than TCP and CPAP levels to simulate protection against G\textsubscript{z} accelerations (see RESULTS and Table 1).

Table 1. Mean pressures measured in the mask, and anti-G jerkin and pants

<table>
<thead>
<tr>
<th>CPAP, cmH\textsubscript{2}O</th>
<th>P\textsubscript{mask}</th>
<th>P\textsubscript{p}</th>
<th>P\textsubscript{j}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.0 ± 0.1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>0.2 ± 0.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>9.9 ± 0.1</td>
<td>9.5 ± 0.5</td>
<td>180.0 ± 1.0</td>
</tr>
<tr>
<td>20</td>
<td>19.7 ± 0.5</td>
<td>19.0 ± 0.5</td>
<td>202.0 ± 1.0</td>
</tr>
<tr>
<td>30</td>
<td>29.6 ± 1.0</td>
<td>29.0 ± 0.5</td>
<td>219.0 ± 1.0</td>
</tr>
<tr>
<td>40</td>
<td>40.0 ± 0.8</td>
<td>39.0 ± 0.5</td>
<td>240.0 ± 1.0</td>
</tr>
</tbody>
</table>

Values are means ± SE expressed in cmH\textsubscript{2}O; n = 6 subjects. P\textsubscript{mask}, pressure measured in mask; P\textsubscript{p}, pressure measured in jerkin; P\textsubscript{j}, pressure measured in pants. Control, without pressurized equipment; from 0 to 40 cmH\textsubscript{2}O continuous positive airway pressure (CPAP), with pressurized suit. For CPAP of 10 cmH\textsubscript{2}O, P\textsubscript{j} = 161 + 1.97 \cdot P\textsubscript{mask} (r = 0.99; P < 0.001).
To assign variations in $P_{ga}$ to variations in expiratory activity, changes in thoracic and abdominal volumes ($V_{ab}$) were measured by using the respiratory inductive plethysmography method (Respiritrace, Ambulatory Monitoring, Ardsley, NY). The bands were positioned under the pressurized jersey and the abdominal bladder of the anti-G pants, one around the thorax at the level of the nipples and the other around the abdomen at the level of the umbilicus. The bands were secured in place. The plethysmograph was calibrated against the integrated pneumotachometer signal with the subject breathing at rest before and after the tests, with and without the aeronautical suit, to correct any shifting of the signals. However, it has not been possible to perform the calibration at 40 cmH$_2$O of CPAP. The rib cage volume ($V_{rc}$) and $V_{ab}$ motion coefficients were obtained by using the least squares calibration method and isovolume maneuvers.

Experimental Protocol and Data Analysis

Respective effects of TCP and ACP on lung mechanics at 40 cmH$_2$O of CPAP. We evaluated the respective effects of TCP and ACP on EEV, respiratory pattern, and respiratory muscle activities at 40 cmH$_2$O of CPAP. Four tests were performed in each subject with different types of counterpressure (no counterpressure [0CP], ACP, TCP, TCP + ACP). During each test, measurements were performed during two periods, at two different levels of CPAP, i.e., 0 (control) and 40 cmH$_2$O. The sequence of administration of the different types of counterpressure was randomized. Duration of each of the eight measurement periods was 4 min. The subject was allowed 2 min for familiarization with the setup, which was sufficient for all measured variables to achieve the steady state. Measurements during the last 2 min were used for the analysis, except for EEV, which was measured after the 4-min CPAP period. After each period, the subjects rested for at least 10 min. All measurements in a given subject were completed within 2 h 30 min.

Expiratory muscle activity was evaluated as previously described (23, 26) from the changes in $P_{ga}$. On the $P_{ga}$ tracing, we measured the decrease from the maximal end-expiratory level to the minimal value ($\Delta P_{ga}$). Figure 2 illustrates this measurement in a representative subject breathing at 40 cmH$_2$O of CPAP with TCP. In this subject, most of the inspiratory effort was associated with a rise in $P_{ga}$ and an increase in abdominal cross-sectional area, indicating shortening and descent of the diaphragm. Relaxation of the diaphragm during expiration resulted in a fall in $P_{ga}$ and a decrease in abdominal diameter. After this early expiratory phase, however, $P_{ga}$ started to rise again until the end of expiration, whereas abdominal diameter continued to decrease. From the end-expiratory value, $P_{ga}$ then dropped (i.e., $\Delta P_{ga}$), whereas abdominal cross-sectional area increased. This pattern clearly indicates expiratory contraction of the abdominal muscles, and the increase in $P_{ga}$ during the expiratory phase of the breathing cycle was taken as a reflection of the direct mechanical effect of this contraction.

Diaphragmatic muscle activity was evaluated from the changes in transdiaphragmatic pressure ($P_{di}$). $P_{di}$ was set at 0.05. Post hoc comparisons were performed by using a Newman-Keuls test or a Student's $t$-test to determine differences in physiological responses to the CPAP or to equipment conditions.

RESULTS

Pressures Measured in the Aeronautical Mask, Jersey, and Pants

Pressures measured in the different parts of the equipment for each CPAP level are shown in Table 1. As expected, $P_{mask}$ and $P_{j}$ values were similar to CPAP levels. ACP was set at much higher levels determined on the basis of the CPAP level, according to the following two relationships that are used in French aircraft: $CPAP = 18 \times G_z (\geq 4 + G_z)$ and $ACP = 70 \times G_z (\geq 2 + G_z)$.

Respective Effects of TCP and ACP on Lung Mechanics at 40 cmH$_2$O of CPAP

All subjects felt more comfortable with TCP and/or ACP than with 0CP.

Absolute end-expiratory $P_{es}$ and $P_{ga}$ measured at 40 cmH$_2$O of CPAP in all counterpressure conditions are shown in Table 2. Figure 3 shows the respective effects of TCP and ACP on EEV at 40 cmH$_2$O of CPAP. With 0CP, EEV increased by only 28% over the functional residual capacity ($FRC; P < 0.05$). This change in EEV was abolished by the presence of either TCP or ACP (+7
VT remained unchanged vs. control with CPAP due to a decrease in VT, whereas respiratory rate had a significant effect on CPAP-related hyperventilation. The CPAP-related hyperventilation persisted. ACP alone limited, and changes in Vrc and Vab did not differ from control. By contrast, the respiratory rate increased and hyperventilation that was only increased by 30% vs. control, with a VT that was not different from control.

Table 4 shows respiratory drive data (TI, TE, VT/TI, VT/TE, VT/VT). At 40 cmH2O of CPAP, the increase in VT was 1.2–1.3% over FRC with TCP and ACP. When CPAP was applied with 0CP, PTPdi increased as changes in Pga decreased, with a negative correlation (r = -0.71 ± 0.28, P < 0.05), indicating progressive recruitment of the diaphragm as expiratory activity decreased. When CPAP was applied with 0CP, PTPdi decreased. TCP or ACP returned PTPdi to normal, and TCP + ACP increased PTPdi to a level above the control value with no CPAP or with 0CP.

Table 2. Absolute end-expiratory Pes and Pga under 40 cmH2O of CPAP with or without TCP and ACP

<table>
<thead>
<tr>
<th></th>
<th>CPAP</th>
<th>Pes</th>
<th>Pga</th>
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<tbody>
<tr>
<td>0CP</td>
<td>32 ± 7</td>
<td>36 ± 3</td>
<td>6 ± 3</td>
</tr>
<tr>
<td>TCP</td>
<td>36 ± 4</td>
<td>36 ± 2</td>
<td>6 ± 2</td>
</tr>
<tr>
<td>ACP</td>
<td>33 ± 3</td>
<td>36 ± 6</td>
<td>6 ± 6</td>
</tr>
<tr>
<td>TCP + ACP</td>
<td>37 ± 6</td>
<td>40 ± 9</td>
<td>6 ± 9</td>
</tr>
</tbody>
</table>

Table 3. Respiratory pattern under 40 cmH2O CPAP, with or without TCP and ACP

<table>
<thead>
<tr>
<th></th>
<th>V, l/min</th>
<th>f, breaths/min</th>
<th>VT, liters</th>
<th>ΔVrc/VT, %</th>
<th>ΔVab/VT, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>CPAP</td>
<td>Control</td>
<td>CPAP</td>
<td>Control</td>
</tr>
<tr>
<td>0CP</td>
<td>8.5 ± 3.5§</td>
<td>27.6 ± 16.3§</td>
<td>11.0 ± 3.0§</td>
<td>13.9 ± 4.1§</td>
<td>0.8 ± 0.4</td>
</tr>
<tr>
<td>TCP</td>
<td>8.6 ± 2.4§</td>
<td>24.5 ± 24.5§</td>
<td>11.4 ± 3.3§</td>
<td>16.9 ± 5.1±§</td>
<td>0.8 ± 0.2</td>
</tr>
<tr>
<td>ACP</td>
<td>8.9 ± 2.1§</td>
<td>19.4 ± 17.1*</td>
<td>12.7 ± 3.0±</td>
<td>14.3 ± 4.3±</td>
<td>0.7 ± 0.2</td>
</tr>
<tr>
<td>TCP + ACP</td>
<td>10.1 ± 1.7±</td>
<td>13.4 ± 10.9*</td>
<td>13.0 ± 2.9±</td>
<td>14.2 ± 2.8!</td>
<td>0.8 ± 0.2</td>
</tr>
</tbody>
</table>

Table 4. Respiratory pattern under CPAP with or without TCP and ACP

<table>
<thead>
<tr>
<th></th>
<th>TI, s</th>
<th>TE, s</th>
<th>VT/Te, l/s</th>
<th>VT/Te, l/s</th>
</tr>
</thead>
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<tr>
<td></td>
<td>Control</td>
<td>CPAP</td>
<td>Control</td>
<td>CPAP</td>
</tr>
<tr>
<td>0CP</td>
<td>2.0 ± 0.4c</td>
<td>2.2 ± 0.9</td>
<td>3.4 ± 1.2d,e</td>
<td>2.0 ± 0.7a</td>
</tr>
<tr>
<td>TCP</td>
<td>2.2 ± 0.7d,e</td>
<td>2.1 ± 0.8</td>
<td>3.2 ± 0.8</td>
<td>1.8 ± 0.5d,e</td>
</tr>
<tr>
<td>ACP</td>
<td>1.9 ± 0.3c</td>
<td>2.1 ± 1.0</td>
<td>2.7 ± 0.7c</td>
<td>2.2 ± 0.9a,c,e</td>
</tr>
<tr>
<td>TCP + ACP</td>
<td>1.9 ± 0.4</td>
<td>2.4 ± 0.5c</td>
<td>2.7 ± 1.3b</td>
<td>1.9 ± 0.6a,c,e</td>
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</table>
pants against high levels of acceleration is often inadequate in agile fighters, CPAP has been suggested as a means of providing additional protection during accelerations above $4 + G_z$ because CPAP is known to increase arterial blood pressure (6). CPAP can be delivered at levels of up to $40 \text{ cmH}_2\text{O}$ during accelerations above $6 + G_z$. To limit lung hyperinflation due to high-level CPAP, use of a pressurized jerkin has been proposed for flights in Mirage 2000 fighters. As shown in Table 1, ACP values were considerably higher than those for Pmask and TCP, as recommended in standard pressure schedules (11, 18, 29).

Numerous studies have demonstrated that high-level CPAP is associated with lung hyperinflation in healthy subjects (2, 3, 15, 22). In addition, the inspiratory workload was shared by the expiratory muscles, which forced the system below its equilibrium position (2, 15). When breathing against moderately high CPAP, many normal subjects strive to prevent or limit CPAP-induced chest distension (10).

Our data corroborate the findings from these studies, providing additional evidence that high-level CPAP without counterpressure in healthy subjects decreases the mechanical activity of the diaphragm and increases the expiratory activity of the abdominal muscles and that these changes fail to completely overcome lung hyperinflation. In addition, we observed that use of TCP + ACP during CPAP was effective in normalizing the lung EEV, reducing the expiratory activity of the abdominal muscles, and restoring the mechanical inspiratory activity of the diaphragm.

**Methods Used To Estimate Respiratory Muscle Efforts**

It has been previously demonstrated that, when expiratory activity is observed, abdominal muscles are responsible for most of this expiratory activity (23, 26). On the other hand, when performing expiratory efforts, normal humans cannot contract the abdominal muscles without also contracting the triangularis sterni, an important expiratory muscle of the rib cage (13). In this study, abdominal muscle activity was evaluated by measuring Pga changes during expiration. For technical reasons, including the use of CPAP and TCP, we were unable to use electromyography to assess the activity pattern of rib cage expiratory muscles. In addition, to separate inspiratory and expiratory activities of intercostal muscles, it would have been necessary to use needle or wire electrodes rather than surface electrodes. Although the contribution of tho-

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**Fig. 3.** End-expiratory volume (EEV) measured with no counterpressure (0CP) or TCP, abdominal (ACP), or thoracoabdominal (TCP + ACP) counterpressure at 40 cmH$_2$O of CPAP. Values are means ± SE; $n = 6$ subjects. Values are expressed as % control value measured at rest without equipment [functional residual capacity (FRC) = 3.39 ± 0.3 liters (dashed line)]. *Significant effect compared with control, $P < 0.05$. 1, 2, 3, and 4: Significant effect compared with 0CP, TCP, ACP, and TCP + ACP, respectively, $P < 0.05$.

**Fig. 4.** Tracings in a representative subject breathing at 40 cmH$_2$O of CPAP with 0CP, TCP, ACP, and TCP + ACP. Respiratory flow, airway pressure (Paw), transdiaphragmatic pressure (Pdi), Pga, and changes in rib cage (Vrc) and Vab compartments are measured. E, expiration; I, inspiration.
Mechanical diaphragmatic activity was evaluated on the basis of measurement of Pdi. Although we found a clear decrease in PTPdi with CPAP, the actual change in diaphragmatic activity was difficult to evaluate. Flattening of the diaphragm due to the increase in EEV that occurs during CPAP is known to decrease the pressure generated by the diaphragm for any given value of diaphragmatic tension (19, 25). This decrease in inspiratory performance with CPAP has been examined in detail by Agostoni (2), who found that Pdi became zero in conscious humans breathing at 30 cmH₂O of CPAP, despite persistence of electrical diaphragmatic activity. Nevertheless, in our study the diaphragmatic force output decreased during CPAP with 0CP, whereas it increased when pulmonary overinflation was compensated for by either type of counter-pressure studied.

Last, it could be argued that TCP during CPAP may restrict rib cage muscle action, thereby increasing the contribution of the diaphragm. However, the fact that ACP had an effect on PTPdi (Fig. 5) and EEV (Fig. 3) similar to that of TCP suggests that the increase in diaphragmatic contribution with TCP was due mainly to a reduction in overinflation.

Interpretation of Data

In keeping with a report by Ernsting (15), the ventilatory pattern observed during CPAP with 0CP was characterized by marked hyperventilation. It has been shown that instruction is important in determining the response of subjects to CPAP (19), suggesting that the stress induced by high-level CPAP may be a factor to explain hyperventilation. Nevertheless, our subjects were experienced in CPAP and were asked to remain relaxed during the tests. One explanation could be an increase in ventilatory demand due to a CPAP-induced fall in cardiac output (7), which follows the reduction in systemic venous return (20) and the increase in right ventricular afterload. Alternatively, the hyperventilation may have been produced by an increase in inspiratory activity. We found that hyperventilation was reduced by the application of TCP and/or ACP, a finding compatible with either hemodynamics or a muscle hypothesis for hyperventilation.

With regard to respiratory muscle activity, CPAP was associated with a high level of expiratory activity of the abdominal muscles (Fig. 5), a decrease in Pdi (Fig. 5), and an increase in EEV (Fig. 3), in keeping with other studies (2, 3, 15, 22, 24). As shown in Fig. 5, the mechanical inspiratory activity of the diaphragm during CPAP was inversely correlated with the mechanical activity of abdominal expiratory muscles, suggesting that during hyperinflation by CPAP the reduction in the mechanical inspiratory activity of the diaphragm was counterbalanced by an increase in the expiratory activity of the abdominal muscles, which stored elastic energy during expiration and released it at their relaxation, thereby assisting inspiratory muscles for the next inspiration (23–26). In our study, when CPAP was applied with 0CP, the increase in EEV was ~1 liter, corresponding to an effective elastance of 40 cmH₂O/l, a value much too large to be accounted for by passive characteristics of the lungs and chest wall. This suggests that expiratory activity explained the relative small increase in EEV.
The question here was to know whether chest wall counterpressure was effective in significantly limiting lung volume expansion, expiratory activity, and hyperventilation in subjects receiving CPAP. The "chest wall" includes all the parts of the body outside the lung that share the changes in volume of the lungs and can be divided into the rib cage and the abdomen (21). Each appears to move as a unit, with considerable independence of motion. For example, it is easy to expand the lung volume with either the rib cage or the abdomen and even to cause outward displacements of one of these units while one moves the other inward (21). We therefore elected to conduct separate evaluations of TCP, ACP, and total counterpressure.

TCP or ACP showed comparable efficacy in preventing increases in EEV to levels significantly above the control value (Fig. 3). In addition, either method returned the mechanical inspiratory activity of the diaphragm to a level similar to that during the control situation (no CPAP, OCP) (Fig. 5). As expected, in addition to this similar effect on the diaphragm, ACP induced a dramatic reduction in abdominal compartment and, as a result, increased the changes in the rib cage compartment. Conversely, when TCP was applied, thoracic volume and Vab changes decreased and increased, respectively. Both modes of counterpressure induced a significant reduction in the expiratory activity of the abdominal muscles, although this reduction was larger with TCP than ACP. These data suggest that TCP may restore normal mechanical diaphragmatic activity and normal EEV with less expiratory activity compared with ACP. Because the ACP compartment was inflated at high pressures, the lesser efficacy of ACP compared with TCP was surprising. One explanation may be that compliance of the abdomen is lower than that of the rib cage (16). Alternatively, the straps placed on the abdomen to secure the TCP compartment to the chest may have increased the efficacy of TCP compared with ACP (see Fig. 1).

We observed an additive effect of TCP and ACP on volumes and respiratory muscle activities: total counterpressure provided a further increase in diaphragmatic activity and further decreases in expiratory activity, EEV, and hyperventilation, compared with TCP or ACP alone. This result is in agreement with the concept that the rib cage and the abdomen are functionally separate (21) and demonstrates that counterpressure is optimally efficient only if it is applied to both mobile parts of the chest wall. With TCP and ACP, the PTPdi was above the control value (no CPAP). This increase in Pdi was largely ascribable to the decrease in thoracoabdominal compliance. Therefore, for a given inspiratory diaphragmatic displacement, the diaphragm must develop a higher tension in the presence of CPAP with TCP + ACP than in the absence of CPAP. In addition, the enhanced diaphragmatic activity compared with control may have been due in part to the persistence of ~40% hyperventilation.

Arterial SBP and DBP increased from control to 40 cmH₂O (30 mmHg) of CPAP. As previously reported, the magnitude of this increase was approximately one-half that of the CPAP level (14). A possible explanation is limitation of venous return, resulting in a decrease in cardiac output despite an increase in HR. Moreover, due to the elasticity of the lung, transmission of intravascular pressure to the vessels was not total (6). In keeping with previous studies (1, 17), arterial blood pressures further increased with TCP or ACP. Interestingly, when either type of counterpressures was applied, the magnitude of the blood pressure increase was similar to the CPAP level (30 mmHg). Furthermore, TCP + ACP had an additive effect on SBP and DBP, which further increased by 10 mmHg compared with use of TCP or ACP alone. It has been established that TCP improves intrapulmonary-to-vascular pressure transmission, whereas ACP increases venous return and therefore the cardiac preload. These hemodynamic effects may counteract hypotension in subjects exposed to accelerations.

In this study, we demonstrated that respiratory and hemodynamic disturbances observed with 40 cmH₂O of CPAP, namely, increases in V, lung volumes, expiratory muscle activity, and HR were adequately counterbalanced by an additive effect of TCP and ACP. We conclude that TCP and ACP limit lung hyperinflation and expiratory muscle activity and restore diaphragmatic activity and that these effects add to the limitation of CPAP-related hemodynamic disturbances provided by anti-G pants.

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