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

Beaumont, Maurice, Damien Lejeune, Henri Marotte, Alain Harf, and Frédéric Lofaso. 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 PTPdi. CPAP hyperventilation was evaluated on the basis of the transdiaphragmatic pressure (CPAP). Expiratory activity was evaluated on the basis of the transdiaphragmatic pressure-time product (PTPdi). When ACP, TCP, or both were added, hyperventilation decreased and PTPdi increased (19 ± 5, 21 ± 5, and 35 ± 7 cmH2O·s⁻¹·cycle⁻¹ during 0 and 40 cmH2O of CPAP, respectively). We concluded that during high-level CPAP, TCP and ACP limit lung hyperinflation and respiratory muscle activity and restore diaphragmatic activity.

To our knowledge, the effects 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 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 ACP 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 Ullis, 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.
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 Gz accelerations (see RESULTS and Table 1).

Cardiorespiratory monitoring. Mask and jerkin pressures were measured by using pressure transducers (model H5035, ±100 cmH2O, Enertec-Schlumberger, Montrouge, France). Anti-G pants pressure was measured by using a ±500 cmH2O 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 cmH2O; model CH5051, Enertec-Schlumberger) and auxiliary conditioners for pressure transducers (model CA9036, Enertec-Schlumberger). Tidal volume (VT) 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 N2 dilution method was used to obtain EEV (12). The subject breathed pure O2, and the total expirate was collected in a Douglas bag over 7 min. The expiratory volume (VE) and N2 concentration ([N2]) of the expirate were measured (4). The fractional [N2] in the lung was assumed to be 0.81 at the onset of the dilution procedure, and a correction was made for tissue N2 elimination (4). Lung volume was calculated as VE *[N2]/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% O2 (at ambient pressure). Because of the characteristics of the N2 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 O2 and that this switch had to occur at the volume to be measured, i.e., at EEV. VE and [N2] 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 [N2] was monitored continuously by using spectrometry.

Respiratory muscle activity and lung mechanics. Gastric (Pga) and esophageal (Pes) pressures were measured by using a probe equipped with two piezoelectric sensors (S7B/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 Pga, and the subject was asked to drink water to verify that the sharp increase in Pga due to contraction of the esophagus was not observed on the Pga tracing. The pressure probe was calibrated before the first test by using a pressure generator (Yew 2856, Yokogawa European Headquarters, Amersfoort, The Netherlands).

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

<table>
<thead>
<tr>
<th>CPAP, cmH2O</th>
<th>Pmask</th>
<th>Pj</th>
<th>PP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.0 ± 0.1</td>
<td>0.2 ± 0.0</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td></td>
<td>0.2 ± 0.0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>9.9 ± 0.1</td>
<td>9.5 ± 0.5</td>
<td>18.0 ± 1.0</td>
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<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>
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</table>

Values are means ± SE expressed in cmH2O; n = 6 subjects. Pmask, pressure measured in mask; Pj, pressure measured in jerkin; PP, pressure measured in pants. Control, without pressurized equipment; from 0 to 40 cmH2O continuous positive airway pressure (CPAP), with pressurized suit. For CPAP of 10 cmH2O, Pj = 161 + 1.97 * Pmask (r = 0.99; P < 0.001).
To assign variations in $P_{ga}$ to variations in expiratory activity, changes in thoracic and abdominal volumes (Vab) were measured by using the respiratory inductive plethysmography method (Respitrace, 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 securely taped 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 (Vrc) and Vab 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 ($P_{di}$). $P_{di}$ pressure-time product (PTP$_{di}$) was used as an estimate of the metabolic work or the oxygen consumption of the diaphragm (28).

We computed PTP$_{di}$ as the area subtended by the $P_{di}$ above the end-expiratory baseline divided by inspiratory time (TI), as described previously (28). To assign variations in $P_{ga}$ to variations in expiratory activity, changes in Vrc and Vab were measured as described above.

From the flow tracings, we measured TI, expiratory time (TE), respiratory frequency, and minute ventilation (V) as the product of respiratory frequency times Vt.

From the Finapres recordings, we measured HR, SBP, and DBP during inspiration and expiration.

Statistics

All data were analyzed with commercially available software (PCSM plus, Deltasoft, Grenoble, France) by using a two-way (CPAP, type of counterpressure) analysis of variance. The critical significance level of $P$ 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, Jerkin, 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 x $G_{2}$ (>4 + $G_{2}$) and ACP = 70 x $G_{2}$ (>2 + $G_{2}$).

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 OCP.

Absolute end-expiratory Pes 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 OCP, 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/TE). Ti remained unchanged vs. control with CPAP treatment due to a decrease in VT, whereas respiratory rate had a significant effect on CPAP-related hyperventilation. The CPAP-related hyperventilation persisted. ACP alone decreased VT, Ti, and changes in Vrc and Vab did not differ from control. By contrast, the respiratory rate increased and limited, and changes in Vrc and Vab did not change. There was a further decrease in abdominal contribution to VT with ACP compared with 0CP. TCP + ACP had an additive effect on the CPAP-related hyperventilation that was only increased by 30% vs. control, with a VT that was not different from control.

Table 3 shows V,t respiratory rate, VT, and changes in VT recruited from rib cage and abdominal compartments (ΔVrc/VT, ΔVab/VT). At 40 cmH2O of CPAP with 0CP, there was marked hyperventilation mainly because of an increase in VT, with a predominant change in thoracic volume. With TCP, the increase in VT was limited, and changes in Vrc and Vab did not differ from control. By contrast, the respiratory rate increased and the CPAP-related hyperventilation persisted. ACP alone had a significant effect on CPAP-related hyperventilation due to a decrease in VT, whereas respiratory rate did not change. There was a further decrease in abdominal contribution to VT with ACP compared with 0CP. TCP + ACP had an additive effect on the CPAP-related 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). Ti remained unchanged vs. control with CPAP levels up to 40 cmH2O under all counterpressure conditions except TCP + ACP, which was associated with an increase in Ti. The increase in VT/TI due to CPAP with 0CP was limited by TCP and even more so by ACP, and was even below control (not significant) with TCP + ACP.

TE was decreased by CPAP under all counterpressure conditions.

Figure 4 shows representative tracings from a subject receiving CPAP under all counterpressure conditions studied. With 0CP, no variations in Pdi were seen in five of the six study subjects. Compared with 0CP, breathing with TCP + ACP was characterized by a high Pdi and by a Pga signal indicating a considerably lower level of expiratory muscle activity.

Figure 5 shows that during CPAP with various conditions of counterpressure the 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.

Respective Effects of TCP and ACP on Arterial Blood Pressures at 40 cmH2O of CPAP

Arterial SBP and DBP increased from control to 40 cmH2O of CPAP (Table 5). These increases were more marked when counterpressures were applied. The magnitude of the increase in SBP or DBP seen with TCP or ACP corresponded to the level of CPAP (30 mmHg). HR increased by ~30% from control to 40 cmH2O of CPAP in all counterpressure conditions except TCP + ACP, during which HR was identical to the control value (Table 5).

DISCUSSION

Protection against +Gz accelerations is mainly obtained by inflation of anti-G pants to limit pooling of blood in the lower limbs and thereby improving venous blood return. Because the protection provided by anti-G...
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 cmH$_2$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 strove 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-

![Graph](image-url)
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 expiratory 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

Fig. 5. Effect of 0CP, TCP, ACP and TCP + ACP, respectively, at 40 cmH₂O of CPAP on diaphragmatic activity assessed on the basis of diaphragmatic pressure-time product (PTPdi; A) and end-expiratory Pga decay (ΔPga; B). In addition PTPdi was matched against ΔPga (C). During CPAP, PTPdi was low with 0CP, normal with TCP or ACP, and high with TCP + ACP, and there were significant differences between all types of counterpressure (P < 0.05), except between TCP and ACP. In addition, during CPAP, a significant negative correlation was observed in the 6 subjects between PTPdi and ΔPga (r = −0.71 ± 0.28, P < 0.05). ○, □, ●, ○, and star: 0CP, TCP, ACP, and TCP + ACP, respectively. ○, Control; solid symbols, 40 cmH₂O of CPAP.
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 alveolar 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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