Effect of gravity and posture on lung mechanics

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Bettinelli, D., C. Kays, O. Bailliart, A. Capderou, P. Techoueyres, J. L. Lachaud, P. Vaida, and G. Miserocchi. Effect of gravity and posture on lung mechanics. J Appl Physiol 93: 2044–2052, 2002.—The volume-pressure relationship of the lung was studied in six subjects on changing the gravity vector during parabolic flights and body posture. Lung recoil pressure decreased by ~2.7 cm H2O going from 1 to 0 vertical acceleration (Gz), whereas it increased by ~3.5 cm H2O in 30° tilted head-up and supine postures. No substantial change was found going from 1 to 1.8 Gz. Matching the changes in volume-pressure relationships of the lung and chest wall (previous data), results in a decrease in functional residual capacity of ~580 ml at 0 Gz relative to 1 Gz and of ~1,200 ml going to supine posture. Microgravity causes a decrease in lung and chest wall recoil pressures as it removes most of the distortion of lung parenchyma and thorax induced by changing gravity field and/or posture. Hypergravity does not greatly affect respiratory mechanics, suggesting that mechanical distortion is close to maximum already at 1 Gz. The end-expiratory volume during quiet breathing corresponds to the mechanical functional residual capacity in each condition.

IN A PREVIOUS STUDY (3), we evaluated how changes in the gravity vector (Gz) obtained during parabolic flights affect chest wall mechanics. In the present work, we show data gathered in the same subjects previously studied to describe how similar changes modify the elastic properties of the lung. The knowledge of the elastic features of the chest wall and of the lung allows a full mechanical analysis of the respiratory system. In particular, coupling the volume-pressure curve of the lung and of the chest wall allows the mechanical definition of functional residual capacity (FRC), corresponding to the resting point of the respiratory system. This volume represents the end of expiration during quiet breathing at 1 Gz; indications from previous studies did not allow clarification of how gravity-dependent changes would influence the end-expiratory point (10, 11, 18, 30) in relation to changes in elastic properties of the respiratory system and of its two main components: the chest wall and the lung. This study also integrates previous information on how changes in gravity influence other aspects of the respiratory function, such as regional ventilation and perfusion (17, 20, 26, 33, 34) and diffusion capacity (32, 35, 37).

METHODS

Parabolic flights. All experiments were done during three European Space Agency (ESA)-Centre National d’Etudes Spatiales (CNES) campaigns of parabolic flights in the period between October 1999 and April 2000. Each campaign included 3 flight days; an Airbus A300 aircraft was used; and each flight lasted 2.5–3 h and included 30 parabolas (90 parabolas per campaign). During the parabolic flight, the vertical acceleration vector Gz changes relative to steady horizontal flight corresponding to the 1-Gz phase: during pull-up, an acceleration of 1.8 Gz is reached; subsequently, reducing engine thrust allows the aircraft to enter a free-falling parabolic trajectory generating a 0-Gz phase, and, finally, during pull-out another 1.8-Gz phase is reached. All three phases lasted ~20 s.

Subjects. Respiratory variables (lung volume and esophageal pressure) were obtained during steady horizontal flight and during short periods of microgravity and hypergravity in five male (age: 53 ± 2 yr; weight: 74 ± 3 kg; height: 174 ± 1 cm) and two female (age: 42 ± 6 yr; weight: 52 ± 5 kg; height: 165 ± 4 cm) subjects. The same subjects were also studied in ground experiments in sitting and supine posture by use of the same equipment. The subjects were members of the experimental team, were nonsmokers, were in good health, and had no report of pulmonary disease. The subjects were trained to perform the respiratory maneuvers (see below); furthermore, they took part in previous parabolic flight campaigns and were well accustomed to the challenge of abruptly changing Gz several times during each flight.

Experimental equipment and system. Subjects were sitting in a body plethysmograph made of wood (empty volume of 360 liters) equipped with a pneumotachograph and transducers to measure pressure in the box and at the mouthpiece (Pm); the mouthpiece was also provided with an electromagnetic shutter. We also performed some parabolas with subjects off the transducers to evaluate the dependence of transducer signals from acceleration. To minimize the effect of changes in aircraft accelerations on both transducers, they...
were oriented along the aircraft’s transverse axis. Lung volumes were measured by flow integration. Esophageal pressure (Pes) was derived from a pressure transducer mounted on a Gaeltec CTO-2 catheter, 2 mm external diameter (12). Transducer sensitivity was 5 µV-V⁻¹-mmHg⁻¹; the linear pressure range was ±300 mmHg. Subjects advanced the catheter through the nose until the proper positioning of the esophageal probe was reached on the basis of preliminary experiments aiming at determining the best recording site. On average, the approximate location of the esophageal recording site was ~15 cm below the jugular notch, which roughly corresponds to the apex of the lung. The location of the esophageal transducer was chosen on the basis of a minimal cardiac artifact and a stable pressure signal.

The pneumotachograph response was linear for flow rates compatible with the respiratory maneuvers performed; the maximum error was ~5% at high flow rates (~3 l/s).

All signals were acquired through a system made of an analog-to-digital card (Digimérie; 50 Hz/channel) and stored in a PC-DOS “home-made” program allowing on-line plethysmograph pressure conversion into pneumotachograph flow and volume by integration and displaying on a video screen present lung volume, pressure variables, and Gz. The software also allowed processing of an off-line preliminary data analysis.

All the data were also stored on an analog tape recorder as a backup, as were the live comments from the researchers during the flight.

Calibration. Before takeoff, calibration of the plethysmograph was done by use of a 2-liter syringe. A syringe volume control was made for each subject during the flight. Pressure transducer calibration for body box pressure and Pm was carried out by using a water manometer. Cabin pressure tends to decrease during the ascending phase relative to level flight and to increase during the descending phase. In terms of volume signal, the plethysmograph would overestimate lung volume during the ascending phase and underestimate lung volume during the descending phase. Cabin pressure was manually corrected during the parabola, and over 30 parabolas we checked that the overall change in lung volume during the 0-Gz phase due to mismatch in pressure correction averaged ~0.029 ± 0.27 liters, 0.6% of vital capacity (VC) (a nonsignificant underestimate).

Gaeltec transducers were calibrated by using a special calibration chamber in which pressure could be set by water manometers; sensitivity of transducers and zero drift at atmospheric pressure were carefully noted. Sensitivity of the transducer was independent of temperature, whereas zero drift was slightly dependent on temperature. Zero value corresponding to body temperature was obtained on withdrawing the probe at the end of each experimental session. These zero values were then used to correct Pes previously recorded.

Protocols for in-flight experiments. Subjects were sitting inside the plethysmograph and breathing through the mouthpiece. During 0 Gz exposure, there was a tendency for the subject to float up in the air because of the changing trajectory of the aircraft; to counteract such an inertia-dependent phenomenon, the subject was kept strapped at the thighs and feet; other loose bands around the arms kept them in a natural position parallel to the chest. During level flight, a check was performed to ensure regular recording of all variables. The time frame for data acquisition during respiratory maneuvers started in the last minute of level flight and lasted 2 min as follows: level flight (1 Gz, 1 min), pull-up (1.8 Gz, 20 s), injection (0 Gz, 20 s), pull-out (1.8 Gz, 20 s).

Subjects were instructed to perform different respiratory maneuvers within each phase, as shown by the experimental record of Fig. 1. After few control breaths, thoracic gas volume (TGV) was measured close to the end-expiratory volume; mouth pressure (Pm) and vertical acceleration (Gz) during the respiratory maneuvers started in the last minute of level flight and lasted 2 min as follows: level flight (1 Gz, 1 min), pull-up (1.8 Gz, 20 s), injection (0 Gz, 20 s), pull-out (1.8 Gz, 20 s).

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Fig. 1. Experimental records of lung volume, mouth pressure (Pm), esophageal pressure (Pes), and vertical acceleration (Gz) during the respiratory maneuvers performed at 1, 1.8, and 0 Gz. Relevant phases are pointed out: 1) panting maneuver to measure total gas volume; 2) inspiration at established lung volume; followed by 3, slow expiration maneuver to determine the volume-pressure curve of the lung.
phase 3. Lung volume and Pes recorded during slow expiration allowed determination of the volume-pressure relationships of the lung. During such maneuvers, the alveolar pressure may be considered atmospheric and, therefore, Pes = \(-P_t\), where Pes represents an estimate of pleural surface pressure (Ppl) and \(P_t\) is the elastic recoil pressure of the lung.

2) VC maneuver (not shown in Fig. 1). The subjects inspired up to TLC and expired relatively rapidly down to RV.

Protocol for ground experiments. Ground experiments were performed on the same subjects adopting the same general protocols and equipment in sitting, supine posture, and 30° tilted head-up relative to supine. The change in posture was obtained by leaning the plethysmograph backward; this implied that legs remained as in the sitting posture.

Data analysis. TGV, computed from Boyle’s law is given by

\[ TGV = \frac{Pc}{V}\frac{\Delta V}{\Delta Pm} \]

where \(Pc\) is in-flight cabin pressure and \(\Delta V/\Delta Pm\) is the ratio of change in thoracic volume to change in alveolar pressure during panting maneuvers. This ratio was inferred as the slope of the linear regression between the two variables. Before the regression was executed, the parameters affecting the volume of the panting maneuvers were subtracted so we generally obtained regression coefficients near 0.99, ensuring an accurate TGV measurement.

Lung volume recorded throughout the time frame also displayed a drift due to increasing temperature inside the plethysmograph. Digital reading of lung volumes was obtained after correction for the volume drift between two successive TGV values, assuming a linear drift with time. Pes data were corrected for the zero drift on withdrawal of the catheter at the end of the session. A “moving average filter” was employed to reduce high-frequency noise in the pressure records.

At 1.8 Gz, whenever possible, we preferred data gathered during the pull-up phase because the Gz vector remained more steady.

At least five lung volume-pressure relationships were obtained in each subject at 1, 1.8, and 0 Gz, in supine and in 30° tilted head-up position. For each condition, the Pes values corresponding to the same lung volume were averaged. A statistical approach was adopted to appropriately characterize the dependence of volume-pressure curves on gravity vector and posture. We adopted a generalized linear regression model (19) that represents a powerful tool to evaluate the dependence of an experimentally measured variable on multiple factors and therefore allows a simultaneous comparison of several regressions.

More specifically, we considered Pes (p) as depending on volume (V), the experimental condition, and an error term (\(e\)) as defined by

\[ p = \hat{A} - \hat{I}V + \hat{B} - \hat{I} + e \quad (1) \]

where \(\hat{A}\) and \(\hat{B}\) are the matrices of slope and intercept coefficients of the model, determined by the multiple linear regression method, and \(I\) is the “dummy variable” that takes into account the gravity and/or posture condition. The program uses a recursive algorithm based on minimizing the sum-of-squares corrected by the reciprocal of the standard deviation squared of the scatter points (weighted-least-squared regression, Ref. 19). This correction was necessary because of statistic heteroscedasticity of the samples. Statistical tests (Student’s t-test) were executed to verify the significance of the differences in slope (reciprocal of lung compliance) and/or in position (intercept) of the lung volume-pressure curves by changing gravity and posture.

A further statistical analysis was carried out to verify the significance of VC, total lung capacity (TLC), RV, and FRC by ANOVA for repeated measures followed by the Student-Newman-Keuls posttest (95% confidence interval).

RESULTS

Figure 2 and Table 1 summarize the data of RV, VC and TLC (\(=RV + VC\)). No significant differences were found in RV; VC was significantly decreased in supine and 30° tilted head-up postures (\(~550\) ml, \(~10%\) VC) relative to 1 Gz on ground; the decrease in VC accounted for a similar decrease in TLC.

Figure 3 reports the average volume-pressure curves of the lung from all the subjects in the various conditions. The relationship was displaced to the right going from 1 Gz (pooling data on ground and in-flight) to 0 Gz. No substantial change was observed going from 1 to 1.8 Gz, whereas a leftward shift occurred in supine and 30° tilted head-up postures. The changes in the position of the volume-pressure curve are reflected in the corresponding changes in the Pes values estimated at 40% VC that are reported in Fig. 4A (and Table 2). Relative to 1 Gz (pooled on-ground and in-flight data), Pes became significantly less subatmospheric at 0 Gz (by \(~2.7\) cmH\(_2\)O), whereas it became significantly more subatmospheric in supine and 30° tilted head-up postures (by \(~3.5\) cmH\(_2\)O). No change was observed going from 1 to 1.8 Gz. Lung compliance (Fig. 4B and Table 3), calculated from 20 to 40% VC, was found to significantly decrease, relative to 1 Gz (pooled on-ground and in-flight data) only in supine posture.

Figure 5 displays the lung volume-pressure curves of the subjects obtained in the various conditions revealing individual differences. In subject F, 0 Gz exposure did not result in a clear rightward shift of the lung volume-pressure curve. Furthermore, in subject D, no...
difference in compliance was observed between 1 Gz (pooled on-ground and in-flight data) and supine and 30° tilted head-up postures.

In one subject, we compared in-flight results obtained by the classic esophageal balloon technique and by the Gaeltec transducer probe. No differences were found when superimposing the lung volume-pressure curves obtained by the two techniques. This finding confirms that the two methods provide similar results, as previously found (31), also considering the intrinsic noise of the Pes due to heart rate. We also compared the volume-pressure curves at 1 Gz obtained with the catheter at different times, up to 1 h apart, and found

### Table 1. RV, VC, and TLC of the subjects in the various conditions

<table>
<thead>
<tr>
<th>Subject</th>
<th>0 Gz</th>
<th>1 Gz</th>
<th>1.8 Gz</th>
<th>On Ground</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>2,190 ± 10(2)</td>
<td>2,250 ± 30(3)</td>
<td>2,270 ± 50(6)</td>
<td>2,960 ± 10(3)</td>
</tr>
<tr>
<td>B</td>
<td>1,660 ± 40(3)</td>
<td>1,830 ± 40(3)</td>
<td>1,810 ± 60(3)</td>
<td>1,660 ± 20(4)</td>
</tr>
<tr>
<td>C</td>
<td>3,080 ± 10(4)</td>
<td>3,100 ± 10(4)</td>
<td>3,040 ± 180(6)</td>
<td>2,570 ± 80(2)</td>
</tr>
<tr>
<td>D</td>
<td>1,750(1)</td>
<td>1,790 ± 10(2)</td>
<td>2,060 ± 160(3)</td>
<td>2,500 ± 30(7)</td>
</tr>
<tr>
<td>E</td>
<td>3,040 ± 50(3)</td>
<td>2,510 ± 40(3)</td>
<td>2,630 ± 100(3)</td>
<td>2,190 ± 90(3)</td>
</tr>
<tr>
<td>F</td>
<td>2,290 ± 80(3)</td>
<td>2,900 ± 170(3)</td>
<td>2,680 ± 340(2)</td>
<td>2,940 ± 40(5)</td>
</tr>
<tr>
<td>Means ± SE</td>
<td>2,350 ± 240</td>
<td>2,900 ± 220</td>
<td>2,410 ± 180</td>
<td>2,470 ± 200</td>
</tr>
</tbody>
</table>

Values are means ± SE for no. of measurements given in parentheses. RV, residual volume; VC, vital capacity; TLC, total lung capacity; Gz, vertical acceleration. *Significance relative to 1 Gz (on ground), 1-way ANOVA for repeated measures.

![Fig. 3. Average volume-pressure lung curves for the 6 subjects studied at 1 (solid line), 1.8 (dotted line), 0 Gz (short dashed line), supine (dash-dotted line), and 30° tilted head-up (long dashed line). At 1 Gz, pooled data for in-flight and on-ground experiments are shown. Volume is expressed as % of VC. Bars are SE. Ppl, pleural surface pressure.](image1)

![Fig. 4. A: average Ppl at 40% VC at 1, 1.8, 0 Gz, and supine and 30° tilted head-up postures. B: average lung compliance in the volume range 20–40% VC at 1, 1.8, 0 Gz, and supine and 30° tilted head-up postures. Bars denote SE. **Significant differences (1-way ANOVA for repeated measures) relative to 1 Gz (on-ground and in-flight pooled data).](image2)
Table 2. Esophageal pressure of each subject measured on volume-pressure curve at 40% VC and differences relative to 1 Gz

<table>
<thead>
<tr>
<th>Subject</th>
<th>1 Gz</th>
<th>2 Gz</th>
<th>Δ/2-1 Gz</th>
<th>0 Gz</th>
<th>Δ(0-1 Gz)</th>
<th>tilted 30°</th>
<th>Δ(tilted 30°-1 Gz)</th>
<th>supine</th>
<th>Δ(supine-1 Gz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>-4.4</td>
<td>-4.0</td>
<td>+0.4</td>
<td>-1.5</td>
<td>+2.9</td>
<td>-8.4</td>
<td>-4.0</td>
<td>-9.0</td>
<td>-4.6</td>
</tr>
<tr>
<td>B</td>
<td>-2.9</td>
<td>-3.9</td>
<td>-1.0</td>
<td>0.0</td>
<td>+2.9</td>
<td>-7.2</td>
<td>-4.3</td>
<td>-7.7</td>
<td>-4.8</td>
</tr>
<tr>
<td>C</td>
<td>-6.8</td>
<td>-7.2</td>
<td>-0.4</td>
<td>-3.9</td>
<td>+2.9</td>
<td>-9.2</td>
<td>-1.1</td>
<td>-7.9</td>
<td>-1.1</td>
</tr>
<tr>
<td>D</td>
<td>-8.1</td>
<td>-10.2</td>
<td>-2.1</td>
<td>-4.6</td>
<td>+3.5</td>
<td>-9.2</td>
<td>1.1</td>
<td>-9.6</td>
<td>-1.5</td>
</tr>
<tr>
<td>E</td>
<td>-9.0</td>
<td>-9.7</td>
<td>-0.7</td>
<td>6.8</td>
<td>-2.2</td>
<td>-17.4</td>
<td>-8.4</td>
<td>-16.8</td>
<td>-7.8</td>
</tr>
<tr>
<td>F</td>
<td>-9.5</td>
<td>-7.8</td>
<td>+1.7</td>
<td>-7.7</td>
<td>-1.8</td>
<td>-10.3</td>
<td>-0.8</td>
<td>-9.2</td>
<td>+0.3</td>
</tr>
<tr>
<td>Means ± SE</td>
<td>-6.8 ± 1.1</td>
<td>-7.1 ± 1.1</td>
<td>-0.4</td>
<td>-4.1 ± 1.2</td>
<td>-2.7</td>
<td>-10.5 ± 1.8</td>
<td>-3.7</td>
<td>-10.0 ± 1.4</td>
<td>-3.3</td>
</tr>
</tbody>
</table>

Values are given in cmH2O. *Significance relative to 1 Gz, 1-way ANOVA for repeated measures.

no difference, indicating that the response characteristic of the miniaturized probe remains constant over time.

DISCUSSION

This paper provides data on how lung recoil pressure is affected by varying the gravity vector by means of parabolic flights and body position. The present results integrate previous data (3) gathered from the same subjects concerning the effect of changing gravity on the mechanical properties of the chest wall. Adding the present to the previous data allows a mechanical analysis of the respiratory system when exposed to changing posture and gravity, in particular to the microgravity environment that characterizes spaceflights.

Static lung volumes. Despite some intra- and interindividual variability, on the average no significant difference in RV, VC, and TLC was found on comparing 1 Gz in-flight to 1 Gz on-ground data (Fig. 2). This finding suggests that the mechanical properties of the lung are not altered by repeated exposure to changing gravity vector between 0 and 1.8 Gz. The effects of changing lung volume and body position are similar to those reported in previous studies, in particular as far as the decrease in VC observed in supine posture (1, 11), reflecting a corresponding decrease in TLC (Fig. 2). We also observed no difference in RV after acute switching to supine posture, in line with previous studies (11, 18, 30). However, our finding of no change in RV after acute exposure to microgravity contrasts with the significant decrease (~300 ml) observed in sustained microgravity (11). A possible explanation of the difference might reside in the progressive increase in intrathoracic and intrapulmonary blood volume in sustained microgravity.

Volume-pressure curves of the lung. As shown in Fig. 3, the volume-pressure relationship of the lung seems to be affected by changing either Gz or posture. In Fig. 6, we attempt an interpretation of the observed changes based on the following considerations. In the gravity field, lung distortion results in a vertical gradient of alveolar size, and the alveoli in the less dependent regions being more inflated (15); accordingly, a vertical gradient of tissue recoil pressure also exists, determining a vertical gradient of Ppl (27). The indirect evidence of such deformation is given by the shape of the washout curves of inert gases, in particular the change in slope from phase III to phase IV in head-up (4, 6, 9) and supine postures (17, 21). The elastic properties of the lung are commonly described by its volume-pressure relationship; however, one should observe that total lung volume is plotted as a function of the washout curves of inert gases, in particular the change in slope from phase III to phase IV in head-up (4, 6, 9) and supine postures (17, 21). The elastic properties of the lung are commonly described by its volume-pressure relationship; however, one should observe that total lung volume is plotted as a function of the washout curves of inert gases, in particular the change in slope from phase III to phase IV in head-up (4, 6, 9) and supine postures (17, 21). Therefore, in microgravity, one may assume that all lung regions should be more uniformly expanded; as a consequence, regional and total lung volume are the same percentage of VC. Accordingly, a one-to-one correspondence between regional volume and regional pleural pressure should define more accurately the intrinsic elastic properties of the lung (curve labeled “Regional Lung Volume” in Fig. 6). The differences between the regional percentage expansion and the overall lung volume for similar pleural pressure reflect lung distortion.

Table 3. Lung compliance (volume range 20–40% VC)

<table>
<thead>
<tr>
<th>Subject</th>
<th>1 Gz</th>
<th>1.8 Gz</th>
<th>0 Gz</th>
<th>tilted 30°</th>
<th>supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.328</td>
<td>0.249</td>
<td>0.391</td>
<td>0.299</td>
<td>0.186</td>
</tr>
<tr>
<td>B</td>
<td>0.142</td>
<td>0.120</td>
<td>0.155</td>
<td>0.128</td>
<td>0.117</td>
</tr>
<tr>
<td>C</td>
<td>0.498</td>
<td>0.467</td>
<td>0.537</td>
<td>0.484</td>
<td>0.251</td>
</tr>
<tr>
<td>D</td>
<td>0.560</td>
<td>0.455</td>
<td>0.536</td>
<td>0.484</td>
<td>0.313</td>
</tr>
<tr>
<td>E</td>
<td>0.318</td>
<td>0.200</td>
<td>0.331</td>
<td>0.237</td>
<td>0.160</td>
</tr>
<tr>
<td>F</td>
<td>0.174</td>
<td>0.187</td>
<td>0.221</td>
<td>0.160</td>
<td>0.189</td>
</tr>
<tr>
<td>Means ± SE</td>
<td>0.337 ± 0.068</td>
<td>0.296 ± 0.058</td>
<td>0.362 ± 0.065</td>
<td>0.262 ± 0.063</td>
<td>0.203 ± 0.028*</td>
</tr>
<tr>
<td>Means normalized</td>
<td>0.00702</td>
<td>0.00623</td>
<td>0.00768</td>
<td>0.00643</td>
<td>0.00486</td>
</tr>
</tbody>
</table>

Values are expressed in 1/cmH2O of each subject in the various conditions. The last row shows the values of compliance at 1, 1.8, 0 Gz, supine, and 30° tilted head-up expressed as % of VC. *Significance relative to 1 Gz, 1-way ANOVA for repeated measures.
At 0 Gz, the end-expiratory Ppl value is -1.9 cmH\textsubscript{2}O, corresponding to -30% regional VC (Fig. 6, ■). At 1 Gz, the end-expiratory Ppl is -6.4 cmH\textsubscript{2}O, corresponding to -40% VC (Fig. 6, ●). Note that, at this same value of Ppl, the regional volume would be 55% VC (Fig. 6, ○). In supine posture, the end-expiratory Ppl value is -3.8 cmH\textsubscript{2}O, corresponding to only 15% VC (Fig. 6, ●); at this Ppl value, the regional volume would be as high as 35% regional VC (Fig. 6, △). Switching from 0 to 1.8 Gz would cause changes in percentage total lung volume similar to those found when going from 0 to 1 Gz because of similarity of the volume-pressure curve of the lung (Fig. 3) and end-expiratory Ppl values (Table 4). In particular, at 1 Gz (and also at 1.8 Gz,) the lower total lung volume compared with regional midthoracic volume (40 vs. 55% VC, respectively) suggests that alveolar units below the esophageal pressure recording site (~15 cm below the lung apex) must be less inflated. This finding is in line with the increase in slope of phase III and IV of the washout curves of inert gases going from 0 to 1 Gz in upright posture (17, 26). Previous data (26) showed that the hypergravity condition, relative to 1 Gz, induced a further increase in the slope of phases III and IV of the washout curves for Ar and N\textsubscript{2}, an increase in cardiogenic oscillation of O\textsubscript{2}, CO\textsubscript{2}, 

Fig. 5. Individual volume-pressure lung curves of the 6 subjects studied at 1 (solid line), 1.8 (dotted line), 0 Gz (short dashed line), supine (dash-dotted line), and 30° tilted head-up (long dashed line). Volumes are expressed as % of VC. At 1 Gz, pooled data for in-flight and on-ground experiments are shown.

Fig. 6. Effect of lung distortion on the volume-pressure curve. At 1 Gz and in supine posture (solid and dash-dotted line, respectively), alveolar size decreases from top to bottom, and the ordinate refers to %VC of total lung volume. In microgravity (dashed line), the gravitational unloading largely abolishes the vertical gradient in alveolar volume, and therefore the regional and total lung volume are the same percentage of VC; the end-expiratory (end-exp) point is indicated (●). At the end-expiratory pleural pressures in 1 Gz and supine posture, total lung volume (● and ▲, respectively) corresponds to a smaller percentage of VC relative to regional volume after gravitational unloading (○ and ▼, respectively).
and closing volume, indicating a further increase in uneven distribution of blood perfusion and regional lung volume, namely greater expansion of apical alveoli and greater collapse at the base of the lung. Because end-expiratory pleural pressure, and therefore regional lung volume, are similar at 1 and 1.8 Gz, one may hypothesize that the increase in nonuniformity of regional lung volume involves the most dependent region of the lung.

In supine posture, the overall end-expiratory volume is about half of the regional volume in microgravity (15 vs. 35% VC, respectively), suggesting a greater degree of lung distortion. In particular, this indicates that in supine posture, the degree of deflation of the regions below the esophagus (dorsal regions) is greater than that occurring at 1 and 1.8 Gz in upright posture (basal regions). This is consistent with a further increase in slopes of phase III and IV in washout curves (17) and a larger closing volume (21) when comparing supine to upright at 1 and 1.8 Gz.

The lung compliance in the volume range 20–40% VC was only significantly decreased in the supine posture compared with 0, 1, and 1.8 Gz, and 30° tilted head-up posture. These findings are in line with previous observations on dynamic lung compliance on changing Gz (10, 18) and posture from supine (22, 23) to upright on supine (22, 23) and on static lung compliance (2, 36).

Other factors could be considered to affect differently the volume-pressure curve of the lung on changing Gz or posture. Despite a relatively homogeneous lung expansion (17, 26) and blood perfusion (17, 20, 33, 34), an increase in pulmonary blood volume has been documented (32, 35) in weightlessness and, in turn, this was shown to influence the alveolar geometry (5). The latter should not impact greatly on lung recoil, because surface tension is physiologically relatively low in the volume range 20–40% VC (16). An increase in blood volume was shown to reduce lung recoil at lung volume below 25% VC, although changing blood volume has little effect on static lung recoil pressure (13, 14). Furthermore, lung recoil pressure also depends on airways smooth muscles contraction (7, 8, 24, 25); however, it seems unlikely that changes in bronchomotor tone are so rapid to be in phase with abrupt changes in Gz.

### Volume-pressure curves of the respiratory system

Because the lung and the chest wall are arranged in parallel, the total pressure exerted by the respiratory system (Prs) in relaxed conditions is given by $\text{Prs} = \text{P}_{\text{pl}} + \text{P}_{\text{w}} \equiv \text{Palv}$, where $\text{P}_{\text{pl}}$ and $\text{P}_{\text{w}}$ are the recoil pressure of the lung and chest wall, respectively, and Palv is the alveolar pressure. Both lung and chest wall recoil pressures are derived from Ppl by appropriate respiratory maneuvers. Respiratory mechanics assume that local Ppl recording can be representative for the whole structure being analyzed, either the lung or the chest wall; therefore, one considers the volume-pressure curves of lung and chest wall as reflecting their average “functional” mechanical behavior.

Figure 7 presents the average volume-pressure curves of the lung and of the chest wall obtained for the same subjects in a previous study (3). The lung and chest wall volume-pressure curves cross at the resting volume of the respiratory system (FRC) where one has $\text{P}_{\text{w}} = -\text{P}_{\text{pl}} = \text{P}_{\text{alv}}$ and therefore Palv = 0.

Going from 1 to 0 Gz results in a strong expiratory effect on the volume-pressure curve of the chest wall (3) in the volume range below 40% VC (rightward shift). Because of the concomitant rightward shift of the volume-pressure curve of the lung, the resting volume decreases from 41 to 29% VC (~580 ml). Note that FRC would drop to ~17% VC (a further loss of ~500 ml) if the lung volume-pressure curve were not displaced to the right at 0 Gz. Therefore, the reduction in lung recoil pressure at 0 Gz has an inspiratory effect on lung-chest wall interaction. The decrease in resting volume of the respiratory system going from 1 to 0 Gz found in the present study, on the basis of a mechanical analysis of chest wall-lung coupling, compares well with the average decrease in end-expiratory volume of ~390 ± 150 ml reported in previous studies (10, 11, 18, 30).

### Table 4. Volume and pressure at the end of expiration and at FRC (% VC) obtained as crossing point of lung and chest wall volume-pressure curves of each subject

<table>
<thead>
<tr>
<th>Subject</th>
<th>1 Gz</th>
<th>2 Gz</th>
<th>0 Gz</th>
<th>Tilted 30°</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>FRC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>%VC</td>
<td>cmH2O</td>
<td>%VC</td>
<td>cmH2O</td>
<td>%VC</td>
<td>cmH2O</td>
</tr>
<tr>
<td>A</td>
<td>37.8</td>
<td>-4.0</td>
<td>36.7</td>
<td>-3.3</td>
<td>35.0</td>
</tr>
<tr>
<td>B</td>
<td>49.8</td>
<td>-5.6</td>
<td>47.6</td>
<td>-5.6</td>
<td>42.3</td>
</tr>
<tr>
<td>C</td>
<td>42.8</td>
<td>-6.9</td>
<td>41.1</td>
<td>-7.7</td>
<td>23.7</td>
</tr>
<tr>
<td>D</td>
<td>33.5</td>
<td>-7.5</td>
<td>24.8</td>
<td>-8.4</td>
<td>15.8</td>
</tr>
<tr>
<td>Means ± SE</td>
<td>41.0 ± 3.5</td>
<td>6.0 ± 0.8</td>
<td>37.64 ± 6.3</td>
<td>1.1 ± 0.4*</td>
<td>16.1 ± 2.6*</td>
</tr>
</tbody>
</table>

End of expiration:

<table>
<thead>
<tr>
<th>%VC</th>
<th>cmH2O</th>
<th>%VC</th>
<th>cmH2O</th>
<th>%VC</th>
<th>cmH2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>37.8</td>
<td>-4.1</td>
<td>34.6</td>
<td>-3.2</td>
<td>32.0</td>
</tr>
<tr>
<td>B</td>
<td>51.3</td>
<td>-4.9</td>
<td>51.9</td>
<td>-6.7</td>
<td>43.1</td>
</tr>
<tr>
<td>C</td>
<td>44.8</td>
<td>-7.1</td>
<td>42.4</td>
<td>-7.3</td>
<td>19.5</td>
</tr>
<tr>
<td>D</td>
<td>36.3</td>
<td>-8.7</td>
<td>37.0</td>
<td>-12.3</td>
<td>37.7</td>
</tr>
<tr>
<td>E</td>
<td>25.2</td>
<td>-6.2</td>
<td>20.7</td>
<td>-6.4</td>
<td>12.4</td>
</tr>
<tr>
<td>F</td>
<td>34.1</td>
<td>-7.2</td>
<td>29.6</td>
<td>-6.4</td>
<td>22.4</td>
</tr>
<tr>
<td>Means ± SE</td>
<td>38.3 ± 4.5</td>
<td>6.4 ± 0.8</td>
<td>36.0 ± 5.3</td>
<td>-7.1 ± 1.5</td>
<td>27.9 ± 5.9*</td>
</tr>
</tbody>
</table>

Ppl, pleural surface pressure; FRC, functional residual capacity. *Significance relative to 1 Gz, 1-way ANOVA for repeated measures.
The data presented in Fig. 7 allow critical reconsideration of the frequently proposed similarity between supine posture and microgravity exposure. In fact, although the changes in chest wall mechanical behavior are qualitatively similar (rightward shift of the curve and increase in compliance), the changes in overall elastic properties of the lung are opposite. Going from upright (1 Gz) to supine posture results in an expiratory effect on the volume-pressure curve both of the chest wall and of the lung, causing FRC to drop to as low as ~16% VC (~1,200 ml), as previously documented (11, 22, 23).

The volume-pressure curve of the chest wall is minimally affected by hypergravity as exposure to 1 Gz generates a loading on the chest wall that already brings its compliance close to its minimum, as described in the previous study (3). Because the volume-pressure curve of the lung is not significantly affected by hypergravity, no significant changes in FRC were observed when in the shift from 1 to 1.8 Gz. Our conclusions are in line with some data based on flowmeter measurement (18) but not with others based on flowmeter measurement and inductive plethysmography revealing an increase of ~200 ml (10, 30).

Lung volumes corresponding to the mechanical FRC are reported in Fig. 8 and in Table 4 to be compared with the end-expiratory lung volumes measured by the panting maneuver (TGV) on changing Gz and posture. As one can appreciate, there is a very good matching between the two estimates, suggesting that 1) despite the limitation concerning the coupling of total lung volume to esophageal pressure, the mechanical analysis appears still valid for discussing the interaction between lung and chest wall, and 2) subjects remain essentially relaxed at end expiration during quiet breathing despite abrupt changes in either Gz or posture.

A comment is due concerning the modifications in mechanical properties as observed during transient changes in gravity environment during parabolic flights. The immediate change in lung volume, reflecting the mechanical equilibrium point between chest wall and lung, suggests that major mechanical changes occur with a short time constant. This is also confirmed by the relative reproducibility of the measurements throughout the parabolas during the flight. One cannot exclude that additional gradual changes may occur with a long time constant, although the similarity in inert gas washout curves in short and sustained microgravity would suggest that this effect may be negligible.

We used an innovative system to measure esophageal pressure consisting of a thin catheter mounting miniaturized pressure transducers. The placement of the probe via nasopharyngeal route was easily tolerated by the subjects and was found much less uncomfortable than the esophageal balloon. Therefore we believe that this method might represent a useful tool to study respiratory mechanics in transient and sustained microgravity.

Interaction between Ppl and pulmonary interstitial pressure. Some speculation is due concerning pulmonary interstitial fluid dynamics in microgravity. At end expiration, the hydraulic pressure in the pulmonary interstitium is physiologically subatmospheric (~10 cmH2O), reflecting a complex interaction between microvascular and/or interstitial fluid exchanges and parenchymal forces (29). Pulmonary interstitial pressure was found to become more subatmospheric with decreasing (more negative) Ppl (28); for this reason, increasing lung volume should lead to an increase in microvascular filtration. Microgravity is a potential cause of interstitial edema in the lung because of capillary recruitment that, in turn, increases microvascular filtration. Because in microgravity Ppl values are less negative during the respiratory activity (end-expiratory volume decreases by ~600 ml), this should be considered a protective factor countering the potential edematous condition due to increased capillary perfusion.

In summary, microgravity causes a decrease in lung recoil pressure because it removes most of the distortion of lung parenchyma induced by changing gravity field and/or posture. The rightward shift of the lung and chest wall volume-pressure curves in microgravity results in a decrease in FRC (~580 ml). Conversely,
lungs recoil pressure increased in supine posture; the leftward shift of the lung volume-pressure curve combined with the rightward shift of the chest wall curve results in a much larger decrease in FRC (−1,200 ml) compared with microgravity. Hypergravity does not greatly affect the volume-pressure curve of the lung and of the chest wall, indicating that mechanical distortion is close to maximum already at 1 Gz. The end-expiratory volume during quiet breathing corresponds to the mechanical FRC in each condition.

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