Transpulmonary pressures and lung mechanics with glossopharyngeal insufflation and exsufflation beyond normal lung volumes in competitive breath-hold divers

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Loring SH, O’Donnell CR, Butler JP, Lindholm P, Jacobson F, Ferrigno M. Transpulmonary pressures and lung mechanics with glossopharyngeal insufflation and exsufflation beyond normal lung volumes in competitive breath-hold divers. J Appl Physiol 102: 841–846, 2007. First published November 16, 2006; doi:10.1152/japplphysiol.00749.2006.—Throughout life, most mammals breathe between maximal and minimal lung volumes determined by respiratory mechanics and muscle strength. In contrast, competitive breath-hold divers exceed these limits when they employ glossopharyngeal insufflation (GI) before a dive to increase lung gas volume (providing additional oxygen and intrapulmonary gas to prevent dangerous chest compression at depths recently greater than 100 m) and glossopharyngeal exsufflation (GE) during descent to draw air from compressed lungs into the pharynx for middle ear pressure equalization. To explore the mechanical effects of these maneuvers on the respiratory system, we measured lung volumes by helium dilution with spirometry and computed tomography and estimated transpulmonary pressures using an esophageal balloon after GI and GE in four competitive breath-hold divers. Maximal lung volume was increased after GI by 0.13–2.84 liters, resulting in volumes 1.5–7.9 SD above predicted values. The amount of gas in the lungs after GI increased by 0.59–4.16 liters, largely due to elevated intrapulmonary pressures of 52–109 cmH2O. The transpulmonary pressures increased after GI to values ranging from 43 to 80 cmH2O, 1.6–2.9 times the expected values at total lung capacity. After GE, lung volumes were reduced by 0.09–0.44 liters, and the corresponding transpulmonary pressures decreased to −15 to −31 cmH2O, suggesting closure of intrapulmonary airways. We conclude that the lungs of some healthy individuals are able to withstand repeated inflation to transpulmonary pressures far greater than those to which they would normally be exposed.

esophageal balloon; barotrauma; pneumomediastinum; chest wall; elastic recoil pressure

MOST HEALTHY HUMANS rarely reach their maximal or minimal lung volumes, except for scientific or medical purposes. Competitive breath-hold divers are exceptional in this regard; in the practice of their sport, they achieve extraordinarily large and small lung volumes. Before each dive, they inhale to maximal lung volume (total lung capacity, TLC) and then employ glossopharyngeal insufflation (GI), which is a pumplike action of the cheeks, tongue, pharynx, and larynx (6), to fill their lungs beyond TLC by up to 2–3 liters (11, 16, 20, 21). By employing this maneuver, they can increase oxygen stored in the lungs (and, therefore, their breath-hold duration) and provide additional intrapulmonary gas to reduce dangerous chest compression (11) that can result in hemoptysis and pulmonary edema (7) during dives that have recently exceeded 150 m in depth (www.aida-international.org). During descent to such depths, divers perform glossopharyngeal exsufflation (GE) maneuvers (11) to draw air from the lungs into the pharynx to equalize pressure in the middle ear at a time when lung volumes are so low that expiratory muscles are ineffective (11). In training to use GE at depth, while at the surface, they exhale forcibly to their minimal lung volume (residual volume, RV) and use GE to extract a few hundred milliliters of gas from the lungs. The practice of GI and GE therefore exposes competitive divers to lung volumes above TLC and below RV, which are the usual limits considered physiological (and therefore presumably safe) for normal lungs, thus providing physiologists with an opportunity to investigate the elastic properties of the respiratory system at both extreme lung volumes. Recently, Lindholm and Nyren (11) studied the effects of GI and GE on the maximal and minimal amounts of gas in the lungs in five divers, and Seccombe et al. (20) studied the effects of GI on the increases in lung gas volume and airway pressures during voluntary relaxation. However, these investigators did not measure the corresponding transpulmonary pressures across the lung itself. The only previous report of transpulmonary pressures after GI (21) concluded that these pressures were not greater after GI, a finding inconsistent with conventional pulmonary mechanics. To explore the range of transpulmonary pressures attained at extraordinarily high and low lung volumes, we measured pressure-volume characteristics of the respiratory system after GI and GE in elite competitive breath-hold divers. Our results suggest that the lungs of some healthy individuals are able to withstand repeated inflation to volumes and transpulmonary pressures far greater than those previously reported.

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METHODS

Four elite breath-hold divers gave written informed consent for the study, which had been approved by the Human Research Committee at Brigham and Women’s Hospital, and agreed to publication of their athletic records (Table 1). All were healthy nonsmokers, used to performing GI and GE maneuvers in training and competition.

Lung volumes. Lung volume at functional residual capacity (FRC) was measured by multiple breath helium dilution in accordance with recommended techniques (4), and TLC was calculated as FRC plus the inspiratory capacity. Duplicate measurements of TLC were averaged and reported at body temperature and pressure saturated (BTPS). Spirometry procedures and equipment consistent with American Thoracic Society recommendations (2) were used to measure the forced expiratory vital capacity (FVC) during exhalations from TLC and from TLC augmented by GI (TLCGI); the difference was used to calculate the amount of gas (expressed as a volume at BTPS) in the lungs at TLCGI. However, the intrapulmonary gas pressure was elevated at TLCGI, reducing actual (Euclidian) volume of the intrapulmonary gas. At any state of inflation, we denote these actual volumes as VL. We calculated VL following GI (as a corrected volume, denoted cTLCGI) using the airway (= alveolar) pressure measured at TLCGI (see below) to correct for gas compression using the formula

\[ cTLCGI = TLCGI(BTPS) \times \frac{(Patm - PH2O)}{(Patm + Pao - PH2O)} \]

where Patm is atmospheric pressure, PH2O = 64 cmH2O is the vapor pressure of water at 37° C, and Pao is pressure at the airway opening referenced to atmospheric pressure.

RV is routinely calculated as the FRC volume determined by helium dilution minus the expiratory reserve volume measured spirometrically. However, RV reduced by GE (RVGE) could not be determined in this way, because subjects could not perform GE maneuvers while connected to a mouthpiece. Furthermore, although we could have estimated the difference between RV and RVGE, we were concerned that the measurement error of the inhaled vital capacity would be comparable to the actual difference between RV and RVGE. We therefore used thoracic computed tomography (CT) scans obtained at both RV and RVGE to obtain VL directly (without pressure correction). Spiral CT scans of the chest taken at RV and RVGE were transferred to our workstation and analyzed using custom software. We determined the area of the lung on each slice using a density mask, manually including any dense structures within the lung, but not including air in the trachea or mainstem bronchi. Using tracheal air volume and ventricular blood as benchmarks, we computed tissue density, air volume, and tissue volume for each slice. Figure 1 shows CT scans at both RV and RVGE to obtain VL.

Transpulmonary pressure-volume curves. Static pressure-volume curves of the lung (P-Vl. curves) were obtained in the seated position using Pao and esophageal pressure (Pes). The 10-cm-long esophageal balloon was passed through the nose to position its tip 40 cm from the nares and inflated with 0.5 ml of air (8). Proper positioning of the balloon was confirmed by finding negligible change in transpulmonary pressure during respiratory efforts made against an occlusion. Airflow was measured with a pneumotachometer (Fleisch no. 1, Rusch OEM Medical, Duluth, GA), and flow was integrated to provide a volume signal that was calibrated with a 3-liter syringe. Multiple static P-Vl. curves of the lungs were measured during interrupted deflations from TLC or TLCGI, and during interrupted inflations from RVGE. Subjects kept the glottis open as an investigator opened or occluded the airway. (For consistency, all data reported are static values measured during airway occlusion, and the transiently greater maximum values of transpulmonary pressure seen at the end of inflation to TLC are not plotted.) To partly compensate for stress-relaxation of the lung during the 15–30 s required for GI before performing Pt-Vl. curves from TLCGI, subjects held their breath for a similar period at TLC when performing Pt-Vl. curves. As noted above, lung volumes in the Pt-Vl. curves from TLC and TLCGI were corrected for gas compression. For example, during deflation from TLCGI, we calculated the BTPS volume of gas in the lung at each point as TLCGI/BTPS reduced by the measured exhaled volume and then corrected that BTPS volume by Pao to obtain actual lung gas volume. Vl. Pt-Vl. curves made during inflation from RVGE were plotted without pressure correction from volumes determined by CT because the negative pressures measured at the airway at these low volumes were assumed not to reflect pressures in alveolar gas.

RESULTS

Lung volumes. Lung volumes at TLC and RV and the effects of GI and GE are summarized in Table 2. GI increased the amount of gas (BTPS) in the lungs by 0.59–4.16 liters (7–47%) as indicated by the increase in the FVC. Figure 2 shows that after GI, Vl. increased from 0.13 to 2.84 liters, and cTLCGI ranged from 114% to 181% of the predicted normal TLC. Figure 1 shows that GE reduced lung size and increasing the apparent lung density, reducing the volume of gas in CT scan slices. GE reduced Vl. below RV by 0.09–0.44 liter (8–26%).
Pressures. Figure 3 shows static PL-VL curves during interrupted exhalations from TLC and TLCGI and inhalation from RVGE, with the predicted normal value of PL at TLC during airway occlusion (5) indicated for each subject by an arrow. Static deflation curves from TLC were relatively consistent, whereas deflation curves from TLCGI varied in the peak pressure achieved because of variations in the volume of GI. Thus Fig. 3 shows a representative curve from TLC for each subject plotted with the curve from TLCGI with the greatest maximal PL and the curve from RVGE with the most negative minimal PL. Whereas PL was near or slightly less than predicted at TLC, PL was substantially increased by GI. Static PL values at TLCGI ranged from 43 to 80 cmH2O.

After exhalation to RV with subsequent GE, PL was substantially negative (i.e., $P_{aO_2}/H_11002$), with values at RVGE of $10 \text{ to } 30 \text{ cmH}_2\text{O}$. Table 2 summarizes PL, $P_{aO_2}$, and Pes at TLCGI and RVGE. Because it is nearly impossible to voluntarily completely relax respiratory muscles at extreme lung volumes, these values of Pao and Pes may not reflect the passive elastic characteristics of the chest wall or respiratory system. Pao values at TLCGI

<table>
<thead>
<tr>
<th>Lung volumes</th>
<th>Diver 1</th>
<th>Diver 2</th>
<th>Diver 3</th>
<th>Diver 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLC</td>
<td>8.46</td>
<td>5.91</td>
<td>9.78</td>
<td>8.88</td>
</tr>
<tr>
<td>TLC predicted</td>
<td>7.55</td>
<td>5.23</td>
<td>6.83</td>
<td>8.31</td>
</tr>
<tr>
<td>TLCGI (BTPS)</td>
<td>9.05</td>
<td>6.85</td>
<td>13.59</td>
<td>13.04</td>
</tr>
<tr>
<td>cTLCGI</td>
<td>8.59</td>
<td>6.47</td>
<td>12.35</td>
<td>11.72</td>
</tr>
<tr>
<td>RV</td>
<td>1.70</td>
<td>1.03</td>
<td>2.02</td>
<td>1.45</td>
</tr>
<tr>
<td>RVGE</td>
<td>1.25</td>
<td>0.94</td>
<td>1.71</td>
<td>1.14</td>
</tr>
</tbody>
</table>

Table 2. Volumes and pressures at volume extremes with and without GI or GE

Pressures. Figure 3 shows static Pt-VL curves during interrupted exhalations from TLC and TLCGI and inhalation from RVGE, with the predicted normal value of Pt at TLC during airway occlusion (5) indicated for each subject by an arrow. Static deflation curves from TLC were relatively consistent, whereas deflation curves from TLCGI varied in the peak pressure achieved because of variations in the volume of GI. Thus Fig. 3 shows a representative curve from TLC for each subject plotted with the curve from TLCGI with the greatest maximal Pt and the curve from RVGE with the most negative minimal Pt. Whereas Pt was near or slightly less than predicted at TLC, Pt was substantially increased by GI. Static Pt values at TLCGI ranged from 43 to 80 cmH2O.

After exhalation to RV with subsequent GE, Pt was substantially negative (i.e., $P_{aO_2}/H_11002$), with values at RVGE of $-10 \to -30 \text{ cmH}_2\text{O}$. Table 2 summarizes Pt, Pao, and Pes at TLCGI and RVGE. Because it is nearly impossible to voluntarily completely relax respiratory muscles at extreme lung volumes, these values of Pao and Pes may not reflect the passive elastic characteristics of the chest wall or respiratory system. Pao values at TLCGI
were substantially positive, ranging from 52 to 109 cmH2O, and those at RVGE were negative, ranging from −59 to −90 cmH2O. Intrathoracic pressure estimated by Pes ranged from 9 to 29 cmH2O at TLCGI and from −28 to −61 cmH2O at RVGE.

**DISCUSSION**

Among mammals, lung size scales with body mass over seven orders of magnitude (23), and in adult humans, lung volume scales linearly with skeletal size (1, 22); this relationship forms the basis of prediction formulas that specify normal lung volumes for an individual. All of our subjects had larger than average lungs, with TLCs greater than predicted; in *diver 3*, TLC exceeded the 95% confidence interval for the normal range. In *divers 3* and *4*, GI increased Vt, to 181% and 141% of the predicted TLC, which are 7.9 and 4.9 SDs greater than predicted. Clearly, the lungs of *divers 3* and *4* were inflated to volumes far greater than those commonly measured in normal subjects, consistent with previous reports.

GI increased the amount of gas in the lungs by up to 47%. Of this increase, approximately one-half was accounted for by an increase in actual lung volume, the remainder being accounted for by intrapulmonary gas compression at TLCGI. For example, in *diver 4*, the increase in actual (Euclidian) lung gas volume at TLCGI was 2.84 liters, whereas the total additional volume of gas insufflated (expressed as a volume at BTPS) was 4.16 liters. Gas compression thus increased oxygen stored in the lungs by 0.26 liter BTPS, enough to sustain the diver for nearly 1 additional minute at resting oxygen consumption. In *divers 1–4*, gas compression during GI accounted for approximately 79%, 41%, 33%, and 32% of the increase in gas and oxygen stored at TLCGI, respectively, similar in some subjects to the 31% reported by Seccombe et al. (20).

TLC is normally determined by the balance between the expiratory (inward) recoil of the lungs and chest wall and the inspiratory pressure exerted by the respiratory muscles (14). Thus the three factors limiting inhalation as lung volume increases are the rapidly decreasing effectiveness of the inspiratory muscles, the increasing elastic recoil of the lung, and the increasing elastic recoil of the relatively compliant passive chest wall. By contrast, cTLCGI appears to be limited not by mechanics of the lung, chest wall, or respiratory muscles, but by sensation. The pressures that can be generated by the buccal cavity far exceed those measured after GI, so the GI maneuver is capable of increasing lung volume far beyond cTLCGI. All of our subjects continued GI until they felt “full enough,” describing a sensation of pressure or fullness in the chest, and we conclude that it is this sensation that limited the volume at cTLCGI. However, compliance of the lungs (i.e., the tangent slope of the Pt-Vt curve) also became very low near TLCGI. For example, *diver 4*, who had a lung compliance of 340 ml/cmH2O near FRC, had a tangent compliance of only 23 ml/cmH2O near cTLCGI. By contrast, the chest wall, although not sufficiently relaxed to measure a true passive compliance, contributed only 27% of the total transpulmonary pressure (= Pao) at cTLCGI in this subject.

At the other extreme, RV is determined in young healthy people by the ability of expiratory muscles to overcome the inspiratory (outward) elastic recoil of the chest wall, which is relatively stiff near RV. In such young subjects, decreases in airway pressure can further reduce lung volume (10). This was apparently true of our subjects, in whom GE was able to reduce lung volume below RV. In older persons, RV is limited by the lung’s ability to empty due to functional airway closure, and sustained expiratory efforts produce a highly negative Pt. In this situation, a negative pressure at the airway is ineffective at reducing lung volume further (10). This was apparently the case in our subjects at RVGE, at which volume a highly negative Pt was sustained statically, suggesting closure of intrapulmonary airways (3). It may be that RVGE in our subjects was the minimal gas volume of the lungs. When the lungs are deflated to the point of airway closure, the subsequent reinflation curve may exhibit a downward convexity and variably higher recoil pressures than inflation curves from higher volumes. Indeed, the inflation Pt-Vt curves from RVGE in three of four subjects were relatively inconsistent and variable and exhibited downward convexity (increasing compliance with increasing volume) that is usually interpreted as evidence of airway reopening (recruitment) on reinflation (Fig. 3). As expected, inflation curves from RVGE in *divers 1*, *3*, and *4* also showed greater elastic recoil (higher Pt) near FRC than the deflation curves, consistent with the pressure-volume hysteresis of the lung.

Unlike lung volume, Pt in mammals at TLC does not vary appreciably with species, body size, or lung size (5, 9, 17). The Pt at TLCGI in our subjects exceeded those predicted according to normative data (5) by approximately 3, 5, 8, and 9 SD, suggesting that the healthy lung in some individuals is able to withstand stresses far greater than those encountered normally at maximal lung volume. Currently, medical practice is to limit the transrespiratory pressure (Pao) in patients on mechanical ventilation to about 35 to 45 cmH2O to prevent lung damage from overdistension, or “barotrauma,” and patients on mechanical ventilation are considered at risk of alveolar rupture when Pao exceeds this range (e.g., 18). However, we know of no systematic exploration of the tolerable limits of Pt in healthy individuals. Our findings suggest that at least some healthy lungs are able to withstand repeated sustained inflation to transpulmonary pressures much higher than those previously thought to be injurious.

Caution is in order, however, as *diver 1* apparently developed a small collection of air in the intrathoracic tissues, a pneumomediastinum (13), which was noted on a CT scan of his chest. This pneumomediastinum was asymptomatic and too small to be detected with a chest roentgenogram. Such small collections of air are occasionally noted in healthy athletes, especially following strenuous physical activity (15) and do not require treatment. It is possible that this pneumomediastinum was caused by increased lung stress during GI maneuvers, either those practiced as part of our study or those done previously. Among our subjects, this diver had the smallest increase in lung volume with GI, and the lowest maximal Pt at TLCGI, raising the possibility that his physiological characteristics may have limited his performance in this respect. Although some of our healthy subjects attained extraordinarily high transpulmonary pressures without apparent ill effects, the finding of a pneumomediastinum in one of our subjects demands caution before applying our findings to patients with lung disease.

This study has several limitations. We studied only four subjects, who differed markedly in the volumes they insufflated or exsufflated during GI and GE. In addition, whereas
TLC and RV are determined mechanically and are relatively reproducible within an individual, cTLC(GI) appears to be limited only by sensation, and RV(GE) may be limited by airway closure. Indeed, all of our subjects exhibited a range of volumes and pressures after both GI and GE maneuvers. Thus cTLC(GI) and RV(GE) should not be thought of as uniquely determined for an individual. To characterize fully the effects of GI and GE on lung volumes, we had to combine methods of volume measurement, using both helium dilution with subsequent spirometry to measure TLC and cTLC(GI) and analysis of thoracic CT scans to resolve small differences between RV and RV(GE). Neither method was sufficient by itself. We have investigated CT volume measurement in a cohort of 40 subjects at our hospital and found this method to yield estimates of TLC not significantly different from those obtained by helium dilution or plethysmography.

The only previous report of Pt during GI maneuvers by Simpson et al. (21) concluded “...buccal pumping itself does not carry a risk of pulmonary barotraumas.” This conclusion was based on their finding that Pt at cTLC(GI) was less than at TLC (29 vs. 32 cmH2O), despite a large increase in lung volume from 9.28 to 11.01 liters. One would expect Pt to increase with increasing lung volume, and their paradoxical finding apparently resulted from an attempt to measure transpulmonary pressure at a time when the glottis was closed. This is apparently the case at “maximum transpulmonary pressure” in their Fig. 2. In addition, their Fig. 3, showing an exhalation from TLC(GI), demonstrates an initial increase in Pt followed by a progressive decrease with exhalation, consistent with an esophageal contraction beginning before and extending into early exhalation. Peristaltic esophageal contractions, which are sometimes induced by respiratory maneuvers, increase esophageal pressure and would cause an apparent decrease in maximal Pt at cTLC(GI). We avoided using data affected by esophageal contraction, and our Pt-Vl curves were smoothly monotonic from cTLC(GI) through the normal physiological range.

Several previous studies have explored the limits of airway pressure to which the lungs can be exposed without rupture and leakage. Malhotra and Wright (12) studied the mechanical limits of lung inflation in cadavers, some of which had their chest and abdomen bound with inelastic tape. In those studies, the maximum pressure in the lungs was limited by air leakage, and they found that binding the chest allowed greater pressures to be attained, implying that binding prevented lung rupture. Similarly, Schaefer et al. (19) found that anesthetized animals decompressed from depth could withstand pressures in the lungs of 100 cmH2O before developing interstitial emphysema (free air in the soft tissues of lungs and mediastinum), whereas animals whose chest was bound could withstand up to 240 cmH2O before developing emphysema. These studies emphasize the potential role of the chest wall in limiting lung expansion (i.e., by raising intrathoracic pressure that compresses the lungs and thereby reducing Pt). Our subjects may have limited the expansion of their lungs somewhat by using expiatory muscles to raise intrathoracic pressure, although our recordings of Pes during GI maneuvers do not indicate forceful expiratory efforts; therefore, most of the transrespiratory pressure was borne by the lung.

Our findings suggest that repeated sustained overinflation with GI causes stress-relaxation of the lungs. The two Pt-Vl curves from cTLC(GI) with the highest Pt values (divers 3 and 4) exhibit lower elastic recoil pressures near TLC than curves initiated from TLC, suggesting that lung stretch reduces elastic recoil pressure (Fig. 3). The question arises whether repeated inflations to supernormal volumes can cause structural change in divers’ lungs, reducing elastic recoil pressures at physiological TLC. The characteristic Pt values at TLC in our subjects were slightly less than the average pressures at TLC reported by Colebatch et al. (5) in 124 healthy nonsmokers [27 ± 6 and 25 ± 5 cmH2O (mean ± SD) for 30-year-old men and women, respectively]. Diver 1, who showed the pneumomediastinum on CT, had the highest Pt at TLC, suggesting that he may have lacked structural adaptations that reduce elastic recoil and allow extreme lung inflation to be achieved without damage.

A principle of parsimony, widely applied in biology, holds that biological structures are built to withstand only those stresses to which they are likely to be subjected in the performance of their function. Examples include the apparent matching of structural features in the ventilatory and circulatory aspects of the lung within the overall hypothesis of symmorphosis (24) and the compromise of competing design criteria in the pulmonary microcirculation to both facilitate gas exchange and protect against stress failure (25). This report is the first to demonstrate that the lungs of some elite breath-hold divers can withstand transpulmonary pressures and volumes far greater than those to which lungs would normally be exposed. The ability of these divers’ lungs to withstand these extreme volumes and transpulmonary pressures may reflect unusual genetic predisposition, structural adaptations to repeated lung distension, or both. Alternatively, these abilities may actually reflect the truly extraordinary capacity of the normal human body to exceed limits based on conventional wisdom.

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