Features of glossopharyngeal breathing in breath-hold divers

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Seccombe, Leigh M., Peter G. Rogers, Nghí Mai, Chris K. Wong, Leonard Kritharides, and Christine R. Jenkins. Features of glossopharyngeal breathing in breath-hold divers. J Appl Physiol 101: 799–801, 2006. First published May 11, 2006; doi:10.1152/japplphysiol.00075.2006.—One technique employed by competitive breath-hold divers to increase diving depth is to hyperinflate the lungs with glossopharyngeal breathing (GPB). Our aim was to assess the relationship between measured volume and pressure changes due to GPB. Seven healthy male breath-hold divers, age 33 (8) [mean (SD)] years were recruited. Subjects performed baseline body plethysmography (TLCPre). Plethysmography and mouth relaxation pressure were recorded immediately following a maximal GPB maneuver at total lung capacity (TLC) (TLCGPB) and within 5 min after the final GPB maneuver (TLCPost). Mean TLC increased from TLCPre to TLCGPB by 1.95 (0.66) liters and vital capacity (VC) by 1.92 (0.56) liters (P < 0.0001), with no change in residual volume. There was an increase in TLCPost compared with TLCPre of 0.16 liters (0.14) (P < 0.02). Mean mouth relaxation pressure at TLCGPB was 65 (19) cmH2O and was highly correlated with the percent increase in TLC (R = 0.96). Breath-hold divers achieve substantial increases in measured lung volumes using GPB primarily from increasing VC. Approximately one-third of the additional air was accommodated by air compression.

We hypothesize that the increase in measured lung volume due to GPB, as previously reported, is the result of increasing VC, with no effect on RV, and is primarily the result of gas compression. We wished to measure TLC acutely after cessation of GPB to confirm that the measured volume increase was primarily due to gas compression, with an immediate return to baseline levels.

METHODS

Subjects. Seven male competitive breath-hold divers who had previously practiced the technique of GPB were recruited. The subjects were nonasthmatic, were not current smokers, and did not have known or previous cardiac or lung disease.

Baseline “pre” lung function. Sitting spirometric tests and single breath transfer factor for carbon monoxide were measured to confirm normal lung function. Baseline body plethysmography (TLCPre) (Sensormedics Vmax, Yorba Linda, CA) was then performed. All lung function tests were performed according to American Thoracic Society and American Association for Respiratory Care criteria (1, 3, 4) with predicted values derived from the recommendations of the European Community for Coal and Steel (8).

GPB plethysmography. Sitting body plethysmography was recorded immediately following a maximal GPB maneuver at TLC (TLCGPB). Once placed in the body plethysmograph, with tracing initiated by tidal volume, the subjects were instructed to come off the mouthpiece to perform the typical maneuver that allowed them to achieve what they believed to be their maximal lung volume with GPB, nose clip in situ. Then, without air leak, they returned to the mouthpiece and performed a slow expiratory VC maneuver to RV. Pans against a closed shutter for thoracic gas volume (VTG) measurements were performed after a small inhalation (ERV1), immediately post-VC. This technique was replicated for TLCPre and sitting plethysmography within 5 min of the final maneuver (TLCPost). TLC was calculated by: TLC = VTG – ERV1 + VC.

If they usually performed “warm-up” stretching and GPB, we allowed time for them to do so after the baseline measurement (no more than 10 min).

Mouth relaxation pressure. The subjects were cognizant of the aims of measurement, and careful attention was given to obtaining open-glottis mouth relaxation pressure (P0mouth). The pressure plateau was recorded at TLCGPB with a fluid-filled catheter inserted through pursed lips with no air leak. The catheter was connected to a physiological pressure transducer (Spacelab Program module, Spacelabs, Redmond, WA), and measurements were repeated until two maximal reproducible efforts (within 2 cmH2O) were obtained. The pressure transducer was calibrated by use of a water manometer and was zeroed at the level of the subject’s mouth before each measurement.

Because all lung volume measurements are performed assuming barometric pressure (P0ambient), the application of Boyle’s Law allowed us to estimate the compressive effect of the TLCGPB lung volume.
compared with the open-glottis TLC\textsubscript{PRE} lung volume. Any measured volume above TLC\textsubscript{PRE} that was not due to compression of air can be assumed to be causing distention of the lung (TLC\textsubscript{Distended}). Distention refers to the increased volume occupied by the lung in the hyperinflated and compressed state.

Boyle’s law states that for a mass of gas with temperature held constant the pressure (P) is inversely proportional to the volume (V). Therefore the pressure-volume product in compressed state (P\textsubscript{1}V\textsubscript{1}) equals the product in atmospheric state (P\textsubscript{2}V\textsubscript{2}) with constant body temperature. Thus:

\[(\text{absolute}) P_{\text{Mouth}} \times \text{TLC\textsubscript{Distended}} = P_{\text{Baro}} \times \text{TLC\textsubscript{GPB}}\]

Therefore,

\[
\text{TLC\textsubscript{Distended}} = \left( \frac{P_{\text{Baro}}}{P_{\text{Baro}} + P_{\text{Mouth}}} \right) \times \text{TLC\textsubscript{GPB}}
\]

The percent change in volume from TLC\textsubscript{PRE} values attributable to gas compression can then be estimated by

\[
\frac{\text{TLC\textsubscript{GPB}} - \text{TLC\textsubscript{Distended}}}{\text{TLC\textsubscript{GPB}} - \text{TLC\textsubscript{PRE}}} \times 100
\]

"Post" plethysmography. Sitting plethysmography was repeated within 5 min of the final GPB maneuver (TLC\textsubscript{POST}).

All V\textsubscript{TG} curves and TLC, VC, and RV calculations were later verified by a second scientific officer who was not in attendance at testing. The body plethysmograph was calibrated before each testing session.

Supplemental O\textsubscript{2}, resuscitation equipment, and medical personnel were available at all times.

The study was reviewed, approved, and conducted in accordance with the principles of the World Medical Association Declaration of Helsinki 2000, and this included the provision of fully informed consent. The Ethics Committee was Central Sydney Area Health Service Human Research Ethics Committee (Concord Raptapartion General Hospital zone).

Statistical analysis. Results were expressed as means (SD). A two-tailed repeated-measures ANOVA was performed to analyze the change in measured lung volumes from TLCPRE to TLC\textsubscript{GBP} and TLCPRE to TLC\textsubscript{POST}. Significance was determined by use of a pairwise comparison and was considered significant if \(P < 0.02\). The relationship between mouth relaxation pressure at TLC\textsubscript{GBP} and change in measured lung volume, from TLCPRE to TLC\textsubscript{GBP}, was determined by use of a correlation analysis.

RESULTS

Seven male breath-hold divers were studied. The demographic and lung function data and breath-hold diving history of the study subjects are shown in Table 1. Baseline lung function in all subjects was within normal limits.

There were increases in intrathoracic gas volume from TLCPRE to TLC\textsubscript{GBP}, returned almost to resting values at TLC\textsubscript{POST}, less than 5 min later. Mean percent increase in TLC from TLCPRE to TLC\textsubscript{GBP} was 24\% (range 15–35) and in VC was 30\% (range 22–44). Mean (SD) values for lung volume measurements and statistical significance are presented in Fig. 1.

Average measured mouth relaxation pressure at TLC\textsubscript{GBP} was 65 (19) cmH\textsubscript{2}O. The correlation of recorded mouth relaxation pressure at TLC\textsubscript{GBP} vs. the percent recorded volume change from TLCPRE to TLC\textsubscript{GBP} is shown in Fig. 2.

The mean increase in measured volume (from TLCPRE to TLC\textsubscript{GBP}) attributable to air compression was calculated as 31
lung. This requires substantial pressure given the mouth relaxation pressure to overcome recoil pressures and distend the rapidly stiffening lung. The mean recorded increase in TLC from TLC\textsubscript{PRE} to TLC\textsubscript{GPB} was 1.95 liters (range 1.23–3.01). The mean volume due to air compression was 31\% of this measured volume increase and equates to 610 ml (BTPS). Therefore, the mean increase in TLC that is distention of the lung is estimated to be 1.34 liters (BTPS).

### DISCUSSION

This study has measured the substantial increases in lung volumes that breath-hold divers can achieve using GPB as described previously (13, 14, 17). This was primarily from an increase in measured VC, with no change in RV. From average recorded mouth relaxation pressure measured at TLC\textsubscript{GPB}, we estimate that 31\% of the additional air was accommodated by air compression.

Conventionally TLC is the point of maximum inflation. This volume is reached when maximal inspiratory muscle tension is exerted. The increasing muscular disadvantage against an increasing chest wall and more importantly lung recoil is said to determine TLC (16).

To increase lung volume above normal TLC, GPB must overcome recoil pressures and distend the rapidly stiffening lung. This requires substantial pressure given the mouth relaxation pressures measured here range from 40 to 90 cmH\textsubscript{2}O. GPB above TLC is limited by “discomfort” and reported to improve with training (W. Steyn, personal communication). This raises the possibility that recoil pressure at high lung volumes may be altered transiently by lung overdistension. This view is consistent with our finding that TLC\textsubscript{POST} of all subjects measured in the 5-min period after GPB was increased outside expected normal intrasubject variation (1).

The concept of hysteresis explains the recoil pressure difference at a given lung volume depending on the volume history (i.e., during inflation or deflation) on a breath-by-breath basis (10). However, it is not inconceivable that, when lungs are distended by GPB beyond the normally obtained maximum inflation (i.e., TLC), the time course for lung and/or chest wall recoil pressure recovery may be prolonged. Addressing this question, a previous report on a single elite breath-hold diver did not find a change in pulmonary compliance between normal and TLC\textsubscript{GPB}; however, they reported difficulty in measuring transpulmonary pressures during the GPB maneuver (17). Extrapolating the volume-pressure relationship of Rohrer and subsequent authors (2, 10, 15, 16) above 100\% VC (equivalent to 50 cmH\textsubscript{2}O) suggests that the greater proportion of the increased mouth relaxation pressure measured here, averaging 65 (19) cmH\textsubscript{2}O, is generated by lung recoil pressure and a lesser proportion by chest wall recoil.

The measured volume increase was closely correlated with recorded mouth relaxation pressure in the TLC\textsubscript{GPB} state. The reporting of the volume reduction that occurs as a result of the air compression during relaxation against an obstruction is not new (2). From our subjects’ results, we estimated that this air compression accounted for only a 31\% reduction of our measured volume increase from TLC\textsubscript{PRE} to the TLC\textsubscript{GPB} state. Thus 69\%, or a mean of 1.34 liters (BTPS), we estimate to be due to the volume distension of the lung. The proportional distribution between the thorax and abdomen distension is yet to be determined and is subject to blood volume redistribution (11).

Methodology limitations resulted in the volume and mouth relaxation pressure measurements being recorded independently. Both measurements were highly reproducible. This small group of athletes represents a substantial portion of the competitive breath-hold divers nationally. Their results demonstrated consistent trends in all variables measured.

Our initial hypothesis that the measured increase in TLC due to GPB resulted primarily from an increased VC with no change in RV was supported. However, gas compression made a smaller contribution to this volume increase than we predicted. Importantly, we failed to support our hypothesis that there would be no difference between baseline TLC and that acutely after ceasing GPB. The elevated TLC once ceasing GPB suggests that there has been some transient distention of the lung.

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### REFERENCES