Going to extremes of lung volume

VOLUME CHANGES IN THE LUNG are the basis for life-sustaining respiration, yet in many regards, the lung is overengineered and the mechanical limits are most often not approached (11). For some activities, however, the entire volume range of the thorax and lung is necessary; for example, consider childbirth. But what determines the extreme limits of lung volume? Common wisdom (14, 15, 17, 20) holds that total lung capacity (TLC) is achieved because the respiratory muscles distort the chest wall to a maximal volume that is resisted by lung recoil. On the other hand, residual volume (RV) is achieved by the respiratory muscles distorting the chest wall toward a minimal volume that, in adults, is limited by airway closure. Is the conventional wisdom regarding the determinants of maximal and minimal lung volume correct; or are there exceptions?

In the current issue of the Journal of Applied Physiology, Loring and colleagues (19) explore the mechanical effects of glossopharyngeal insufflation (GI) and exsufflation (GE) on the respiratory system. They studied four competitive breath-hold divers measuring lung volumes (TLC and RV) before and after GI and GE. Additionally, they measured pressure-volume characteristics of the respiratory system during these maneuvers. The results of the present study are provocative in several respects but most importantly further substantiate similar findings made by Lindholm and Nyren (18) and Seccombe et al. (24) that challenge conventional wisdom regarding the lungs’ ability to tolerate extremes of volume and pressure.

Why are GI and GE, and thereby alterations in TLC and RV, advantageous to the breath-hold diver? Glossopharyngeal breathing was first described in the 1950s in postpoliomyelitic patients with severe respiratory muscle weakness (9). These patients were observed swallowing air into their lungs, allowing them to significantly increase their vital capacity. The technique of GI entails filling the lungs to TLC and then forcing air out against a closed glottis. The air is compressed within the oropharynx and then forced into the lungs. This process is then repeated several times to achieve a new, maximal TLC. GE represents the opposite extreme. While at maximal end expiration (RV), air is forced from the lungs into the oropharynx followed by glottic closure. The breath is then exhaled (or used to help equalize ear canal pressures), further decreasing RV (18). Duration of breath-hold time is one important factor that may be improved by glossopharyngeal breathing. GI allows for a greater volume of oxygen to be stored in the lungs under greater pressure and in turn allows a longer breath hold (19, 23). In addition, physiologists have long believed that the maximal breath-hold depth is determined by compression of thoracic gas as dictated by Boyle’s law \( P_1V_1 = P_2V_2 \), where \( P \) is pressure and \( V \) is volume. If a diver attempted to descend to a depth that resulted in TLC compression below RV, “thoracic squeeze” would result with the risk of lung rupture (27). Yet current world records in breath-hold diving greatly exceed predictions based on this model (13). One explanation may be glossopharyngeal breathing. GE may allow breath-hold divers to increase their diving depths by repeatedly deforming the chest to low volumes, thereby reducing the outward recoil of the chest at very low volumes. This would reduce thoracic squeeze and make the divers more like dolphins, whose chest collapses impressively at depth, without developing high transthoracic pressure (4).

The authors of the present study (19) suggest that TLC can be increased beyond normal limits with GI to an extent limited by a “sensation of fullness” rather than by a summation of mechanical forces. The magnitude of TLC achieved after GI is impressive but variable, with one diver increasing their calculated dimensional TLC by 2.85 liters to a new maximal gas volume of an additional 4.16 liters and a second diver able to increase TLC by only several hundred milliliters (19). Interestingly, all divers had greater than predicted baseline TLC, which in the past has been attributed to training or more likely selection of mechanical characteristics important for performance of these feats (1, 6, 8, 10, 25). However, it is also possible that repeatedly increasing the lung beyond TLC alters “fullness sensation” and thereby establishes a new larger endpoint for TLC.

Whereas TLC is felt to be determined by a summation of opposing forces, RV is said to be determined by airway closure in adults (17, 20) and can also be altered by glossopharyngeal breathing (18, 19). As was seen with the alterations in TLC after GI, changes in RV after GE were variable between divers. Three of the four divers were able to dramatically decrease their RV (0.31 to 0.45 liter), while the fourth diver was able to make only modest changes (0.09 liter). These reductions in RV were confirmed by chest computed tomography. The ability to decrease RV may result from physiological differences inherent in the individuals’ lung or may result from training; it is equally likely that both factors play a role.

Increasing TLC and decreasing RV may be advantageous maneuvers for breath-hold divers, but altering lung volumes
Invited Editorial

832

beyond conventional “set points” results in large swings in transpulmonary (Pt) pressures. The present study (19) measures these extremes in pressure, and, like the variability in maximal and minimal attainable lung volume, there is a range of tolerable pressures. Diver 1 is able to generate only modest increases in Pt over that observed at TLC (19). In contrast, diver 4 generates nearly a fourfold increase in Pt after GI with intrapulmonary pressures up to 109 cmH2O! Interestingly, diver 1 sustained a complication of the maneuver (pneumomediatinum), despite having the lowest recorded Pt after GI of all the subjects tested (19). All four subjects had a lower than predicted Pt at TLC (19), further suggesting that their baseline lung mechanical characteristics may be altered by repeated GI and GE maneuvers (7).

How high can Pt increase (or decrease) and still be safe? Common wisdom suggests that Pt beyond 30 cmH2O is to be avoided or barotrauma or in extreme cases pleural rupture can occur. Clinicians caring for mechanically ventilated patients attempt to achieve airway pressures <35 cmH2O to prevent barotrauma (3). The ability to tolerate high pulmonary pressures varies between mammals. What determines the ability of the lung to resist rupture at high pressure? One explanation is the structural thickness of the pleura. Some species of mammals are intolerant of high airway pressures and have thin visceral pleura (e.g., rabbits), whereas others with thick pleural membranes such as bats (17) or mice (26) can tolerate airway pressures much higher than 35 cmH2O without negative consequences. The results of the present study (19) suggest that in humans, the maximal tolerable Pt is unknown and almost certainly varies between individuals as three of the four divers were able to tolerate markedly elevated Pt without ill effect while one of the four divers sustained a pneumomediatinum presumably related to high Pt. The authors (19) comment that physiological findings in healthy subjects cannot always be easily applied to patients with lung disease and therefore may limit the clinical applicability of their findings. Nonetheless, this study demonstrates that at least some individuals are able to tolerate Pt far greater than what has previously imagined.

What accounts for the increased TLC observed in these breath-hold divers (Fig. 1)? Glossopharyngeal breathing may simply increase TLC by overdistension of the respiratory system beyond the physical point possible by contraction of the respiratory muscles. In this regard, it might be of great interest to determine which part of the chest wall is further distorted. Second, it is known that as TLC is approached there is increased surface electrical activity within the respiratory muscles (2, 21), and while it serves to increase thoracic dimensions, contraction of the respiratory muscles also stiffens the chest wall. In asthma it is speculated that chest wall stiffening may inhibit maximum TLC (5, 15) by a reflex mechanism. The divers may have dis inhibition this reflex control of the limits of chest wall excursion either by happenstance or training. Lastly, divers (1, 8, 25) and swimmers (6) are known to have large lungs and attendant lower lung compliance. Previously, Mills et al. (22) showed that inhalation to TLC followed by glottic closure and relaxation with a repeated inspiratory effort increased TLC further, presumably because of stress relaxation and reduced lung recoil.

What accounts for the decrease in RV that the divers can achieve (Fig. 1)? There are really only two possibilities. First, the closing volume of the divers may be below RV since closing volume is variable between subjects and is age dependent (12, 16). It is possible that the negative intra-airway pressure developed by GE merely distorts the chest wall to a new lower minimal configuration. Since not all airways close at RV, GE maneuvers may simply cause nondependent, open regions of the lung to expire further. Alternatively, the combination of higher intrathoracic (alveolar) pressure coupled with the negative airway pressure from GE results in airway reopening, air escaping, and a lower RV.

The study of Loring et al. (19) is a good example of what we learn when the lung is subjected to extremes in performance, whether that be exercise, altitude, gravity, or, in this case, ocean depths. It also serves to remind us that the lung is a truly remarkable organ that we still do not fully understand. Here we learn that the common wisdom about the extremes of lung volume and pressure apparently are not correct. Indeed, as Leith and Mead (17) pointed out in 1967, “In some subjects, strength, motivation, learning, and fatigue may play a part...” in determining the minimal size of RV and, it appears, TLC as well.

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