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Invited Editorial

The pressure to understand the mechanism of lung compression and its effect on lung function

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THE HUMAN BREATH-HOLD DIVING depth record is currently 214 m, whereas the deepest recorded dive in a marine mammal exceeds 1,500 m. These ultra-deep diving feats defy our current understanding of respiratory physiology and lung mechanics. In breath-hold diving marine mammals, Brown and Butler (2) concluded that the lungs must collapse as trans-thoracic differences exceeding 101 kPa would cause tissue failure. The physical mechanism of lung compression and how the atelectatic alveoli are again recruited during ascent are currently not understood and are of great clinical importance. Pulmonary medicine would benefit by understanding how breath-hold diving affects lung mechanics and gas exchange. Furthermore, providing scientifically tested safety guidelines to the growing number of competitive breath-hold divers would help prevent serious injury during these events. The study by Lindholm et al. (8) in the *Journal of Applied Physiology* is important because it is one of a few attempts to advance our understanding of pulmonary physiology during breath-hold diving. It is also the first study to measure the effect of simulated deep diving on lung function.

Early work on human divers suggested that compression below residual volume (RV) would result in negative intrathoracic pressures. The negative pressure, commonly called the squeeze, would eventually result in pulmonary edema formation. The squeeze depth was estimated by Boyle's law as the ratio between the diver's total lung capacity (TLC) and RV. This calculation suggested that lung squeeze would occur at ~34-m depth (3). As human divers continued to descend deeper without apparent problems, Craig (3) suggested that redistribution of peripheral blood into the thoracic space would help reduce lung volume and prevent extreme negative intrathoracic pressures. However, as the diver descends deeper, the increasing vascular engorgement could eventually lead to capillary stress failure and edema formation (12).

The difficulty in measuring lung function during breath-hold diving has limited most experimental studies on alveolar collapse to diving marine mammals (7) or theoretical models (4, 6). A few experimental studies have attempted to determine the depth of complete alveolar collapse. By measuring inert gas exchange during diving, it was suggested that alveolar collapse and termination of gas exchange occurs between 30-m and 70-m depth in marine mammals (5, 10). However, a model of inert gas exchange suggested that alveolar collapse probably occurs significantly deeper than 70 m (4). The modeling results agree with experimental data in the harbor seal and California sea lion in which the pulmonary shunt was only ~60% at 70 m (7). It was further estimated that complete alveolar collapse would not occur until a depth of ~170 m (7). A recent modeling effort on the human respiratory system suggested that complete atelectasis would occur at ~235 m (6). Even though mathematical models are useful research tools, they are merely an abstraction of reality, and they need appropriate experimental data to verify their predictions. In addition, to utilize respiratory adaptations in marine mammals for human medicine, it is necessary to understand the similarities and differences in respiratory function under pressure between humans and other animals.

Professional human breath-hold divers practice simulated deep diving and lung squeeze at shallow depths in a swimming pool. By exhaling and performing glossopharyngeal exsufflation (GE), they achieve prediving lung volumes below RV. This procedure is followed by a dive to shallow depths (3–6 m), and it is believed that this practice increases compression tolerance and thoracic flexibility. However, any beneficial effects are merely anecdotal, and, other than reports of minor hemoptysis, the physiological effects of this training procedure have never been confirmed. For this reason, Lindholm et al. (8) measured lung function and diffusion capacity in human breath-hold divers before and after a 20-min training session with dives to 6 m. In addition, laryngoscopy was performed to determine the location of any hemorrhages that would lead to hemoptysis. When corrected for the diving-induced increase in hemoglobin (1), the pulmonary diffusion capacity, as measured by single-breath diffusion capacity for carbon monoxide (CO), was not affected by the training procedure. However, the authors were not able to measure the diffusion rate at different O_2 partial pressures. Therefore, their results could have been affected by the competitive binding of O_2 and CO to hemoglobin, and it was concluded that this needs to be resolved in future studies. The forced vital capacity (FVC) and forced expiratory volume during 1 s (FEV_1), on the other hand, decreased by 5.4% and 10.0%, respectively. As the subjects dove in thermoneutral water, the authors concluded that the reduction in expiratory flow was not caused by bronchoconstriction seen during face immersion in cold water. Two subjects experienced hemoptysis, and laryngoscopy indicated that the bleeding originated below the vocal cords. The hemoptysis and reduction in expiratory flow suggested diving-induced obstruction of the lower airways. On the basis of these results and a previous study showing tracheal compression during GE (9), the authors concluded that diving to shallow depths after full exhalation and GE may cause mechanical stress, resulting in edema formation of the conductive airways (8).

Future studies should be encouraged to understand the interaction between lung compression and reexpansion of atelectatic alveoli. Applying a positive pulmonary pressure of ~30 mmHg is clinical practice for reinflating a human atelectatic lung. Marine mammals, on the other hand, appear able to

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collapse and open the alveoli with relative ease. Understanding the reason for these differences may provide novel treatments for the atelectatic lung without the risk of formation of emboli. What effect does lung compression have on gas exchange and pulmonary shunt? This is particularly important in humans in whom a significant portion of the available O_2 stores are located in the respiratory system. The study by Lindholm et al. (8) is a step in the right direction to determine the physiological boundaries of human respiratory performance.

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