

Pulmonary edema after competitive breath-hold diving

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Linér MH, Andersson JP. Pulmonary edema after competitive breath-hold diving. *J Appl Physiol* 104: 986–990, 2008. First published January 24, 2008; doi:10.1152/jappphysiol.00641.2007.—During an international breath-hold diving competition, 19 of the participating divers volunteered for the present study, aimed at elucidating possible symptoms and signs of pulmonary edema after deep dives. Measurements included dynamic spirometry and pulse oximetry, and chest auscultation was performed on those with the most severe symptoms. After deep dives (25–75 m), 12 of the divers had signs of pulmonary edema. None had any symptoms or signs after shallow pool dives. For the whole group of 19 divers, average reductions in forced vital capacity (FVC) and forced expiratory volume in the first second (FEV₁) were –9 and –12%, respectively, after deep dives compared with after pool dives. In addition, the average reduction in arterial oxygen saturation (SaO₂) was –4% after the deep dives. In six divers, respiratory symptoms (including dyspnea, cough, fatigue, substernal chest pain or discomfort, and hemoptysis) were associated with aggravated deteriorations in the physiological variables (FVC: –16%; FEV₁: –27%; SaO₂: –11%). This is the first study showing reduced spirometric performance and arterial hypoxemia as consequences of deep breath-hold diving, and we suggest that the observed changes are caused by diving-induced pulmonary edema. From the results of the present study, it must be concluded that the great depths reached by these elite apnea divers are associated with a risk of pulmonary edema.

apnea; hemoptysis; hypoxemia; dynamic spirometry; pulse oximetry

THE BLOOD-GAS BARRIER OF THE lung is very thin, allowing effective gas exchange, and at the same time strong enough to prevent failure, which would cause plasma or blood to enter the alveolar spaces (30). The barrier function can, in fact, be compromised by conditions that increase the pulmonary capillary pressure (“stress failure”), and pulmonary edema has been reported in humans after such conditions as high-altitude exposure, land-based exercise, scuba diving, and endurance swimming at the surface (1, 16, 28, 30, 34). While these conditions have been relatively well studied, symptoms and signs of pulmonary edema have not been studied systematically in association with deep diving in breath-hold divers.

During breath-hold diving, according to Boyle’s law, the volume of air in the lungs is reduced in direct proportion to the increase in pressure with depth. It has been assumed (2) that the ratio of total lung capacity (TLC) to residual volume (RV) determines the depth limit (TLC/RV = maximum diving depth, in atmospheres of pressure). Any reduction in lung volume below the RV was believed to result in a lung squeeze (pulmonary barotrauma of descent) and would potentially be harmful. Competitive breath-hold divers have since long surpassed the limit posed by TLC/RV (23). Possible explanations

include a pre-dive increase in TLC by glossopharyngeal breathing, also known as “lung packing” or “buccal pumping” (18, 24, 27). After a maximum inspiration, a mouthful of air is taken with the glottis closed. Thereafter, the air in the mouth is compressed using the oral and pharyngeal muscles, the glottis opened, and the air forced into the lungs. These maneuvers, being repeated in rapid succession, can substantially increase the volume of air in the lungs. In addition to a pre-dive increase in TLC, a redistribution of blood from the periphery to the intrathoracic blood-containing structures, a “blood shift,” allows the RV to decrease below normal (6, 26). An increase in intrathoracic blood volume of ~700 ml is observed during mere head-out immersion (3), and the effect is further pronounced by the depth-dependent compression of the lungs (6, 7, 26). Nevertheless, there have been a few case reports describing divers experiencing problems related to pulmonary edema or frank hemorrhage (4, 5, 10, 15, 29), even when diving to what seems to be within the earlier-mentioned theoretical depth limit. Furthermore, using a protocol involving breath-hold dives to a depth of 6 m with a starting lung volume below RV, thus simulating much deeper depths, reductions in performance in dynamic spirometry tests, indicative of pulmonary edema, have been observed (17). Therefore, it is still a viable assumption that overdistension of blood vessels in the thorax and a relative underpressure in the airways during deep breath-hold diving could cause pulmonary edema or hemorrhage.

In the present study, we investigated dynamic spirometry and arterial oxygen saturation (SaO₂) in competitive breath-hold divers after both deep dives in the sea and horizontal underwater swimming in a pool. We sought to elucidate to what extent pulmonary edema, secondary to lung squeeze, occurs in this population. We also wanted to reveal whether any signs or symptoms of pulmonary edema after deep breath-hold diving are associated with significant arterial hypoxemia. We hypothesized that reduced performance on a dynamic spirometry test, together with reduced SaO₂, would be observed after deep breath-hold dives, but not after pool dives, indicating pulmonary edema and an impaired diffusion capacity of the blood-gas barrier.

METHODS

The experiments were conducted during an international breath-hold diving competition in Sweden, in August 2006, with approval from the research ethics committee at Lund University. Out of the 41 divers in the competition, 19 volunteered to participate in the study (15 men/4 women). They all claimed to be healthy and free of any medication, and they had to present a recent approved physical

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examination to the competition organizer. Their mean age was 31 yr (range: 17–42 yr), height 183 cm (163–194 cm), and weight 76 kg (55–96 kg). Their history of breath-hold diving averaged 5 yr (0.5–18 yr) with a personal maximum diving depth at 53 m (26–83 m). Current training routines involved breath-hold training at 4.3 h/wk (1.5–15 h/wk) and physical training at 4.3 h/wk (1–12.5 h/wk). Nine of the subjects had previously (1–20 times) experienced symptoms normally related to pulmonary edema following diving to depths ranging between 20 and 75 m.

Protocol. The divers competed in both “dynamic apnea” (DYN), in which the maximal horizontal distance underwater is attempted in a pool, and in deep diving in the sea (DIVE), involving an attempt to retrieve a tag from the bottom of a line set to a predetermined depth. On separate days, the divers competed in both fin-assisted and fin-unassisted categories in DYN and DIVE, as well as a category in which the diver pulled him- or herself down and up along the diving line. Results from the longest and deepest achievements of each diver were chosen for subsequent analysis. Before their competition dives, divers typically performed a number of submaximal “warm-up” dives. All observed divers performed pre-dive hyperventilation. Diving was performed after a countdown by the competition judges. After surfacing, a special “surface protocol” (removing face mask, giving OK sign, and saying “I am OK”) had to be completed within 20 s in order for the dive to be approved. During the 20-s period, judges watched carefully for any signs of hypoxia that could lead to disqualification of the dive. During the competition, water temperature was 28°C in the pool and 20°C in the sea (at the surface). Normally, there is a thermocline at varying depths at the competition site. Divers have reported that the water temperature was 12°C at 60 m. Air temperature was 27°C at the pool and 21°C at the sea.

On the first day of the competition, before any diving began, the divers were familiarized with dynamic spirometry, and control measurements were performed. Before DYN and DIVE, many of the divers used glossopharyngeal inhalation to increase their lung volume, and dynamic spirometry was also done following this maneuver. SaO_2 was also recorded in all divers. As soon as possible after their competition dives, the divers exited the water into a specially designated boat or into a poolside laboratory area, and within 15 min dynamic spirometry was performed and SaO_2 recorded again.

Measurements. The forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), FEV_1 -to-FVC ratio (FER), and peak expiratory flow (PEF) were measured with a spirometer (Micro Plus, Micro Medical, Rochester, UK). During the measurements, the subject was sitting down, wearing a wet suit, and using a nose clip. Immediately after the spirometric measurement, a pulse oximeter was used for measuring SaO_2 (TuffSat, Datex-Ohmeda, Madison, WI). The finger probe was held in place for at least 1 min, and the quality of the signal was ensured. The values following deep diving (post-DIVE) were compared with values following diving in the pool (post-DYN) using paired *t*-test. The level used for accepting significance was $P < 0.05$.

RESULTS

All results are presented as group means (SD) from 19 subjects, unless otherwise stated.

Diving performances. The mean depth reached by the competitive breath-hold divers when diving in the sea was 48 m (SD 16) (range: 25–75 m), and these dives lasted 118 s (SD 36) (53–190 s). In the pool, diving distance and time were 105 m (SD 25) (48–150 m) and 102 s (SD 23) (61–150 s), respectively.

Spirometric values and SaO_2 . As previously shown in trained breath-hold divers, the control spirometric values (Table 1) revealed a larger than predicted FVC [116% (SD 11), $P < 0.001$], whereas the FEV_1 did not differ from predicted [105% (SD 13)], with predicted values derived from Quanjer et al. (25). Glossopharyngeal inhalation was used by 15 of the 19 divers, and these divers increased their FVC from 6.3 (SD 1.0) to 7.4 liters (SD 1.3) with the technique, i.e., an increase by 18%.

After the DYN dives, no divers experienced symptoms or presented signs related to pulmonary edema. Spirometric and pulse oximetry values did not differ from the control, pre-dive values (Table 1). However, in 12 of the divers, the FVC or FEV_1 were reduced by at least 5% post-DIVE compared with post-DYN (Fig. 1). For the whole group of divers, DIVE reduced the FVC and FEV_1 by 9 and 12%, respectively, compared with the values measured after DYN (Table 1). The FER was not affected by DIVE, whereas there was a tendency for a reduced PEF following DIVE. When measured within the first 15 min after DIVE, the SaO_2 of 7 of the subjects was $<95\%$ (Fig. 2), defined as hypoxemia (33). During measurements after DYN, no subject had SaO_2 values $<96\%$. For the whole group, the SaO_2 was 3.6% lower after DIVE than after DYN (Table 1). All of the divers negated having aspirated water during the dives.

Of the 19 volunteering divers, 6 experienced significant respiratory symptoms after DIVE. These divers had been diving to depths between 41 and 75 m (mean 61 m). The symptoms included fatigue ($n = 2$), dyspnea ($n = 2$), cough ($n = 1$), substernal chest pain or discomfort ($n = 5$), and hemoptysis ($n = 3$). Upon chest auscultation on five of these six divers, basal crepitations or expiratory wheezings were noted in four. In this subgroup with typical symptoms and signs of pulmonary edema, changes in the physiological measurements were aggravated compared with in the whole group of divers. The FVC and FEV_1 were reduced by 16 and 27%, respectively, compared with after DYN ($P < 0.05$). As for the whole group of divers, the FER was not significantly reduced by DIVE (-14% , $P = 0.12$), while there was a tendency for a reduced PEF (-21% , $P = 0.07$). The post-DIVE SaO_2 was between 73 and 94% in these subjects (mean 88%). It should be added that, in the diver with the lowest saturation value, no spirometric measurements were performed directly after diving

Table 1. Spirometric values and arterial oxygen saturation measured before diving, after pool dives, and after deep dives in the sea

	FVC, liters	FEV_1 , liters	FER, %	PEF, l/min	SaO_2 , %
Control	6.1 (1.2)	4.6 (0.9)	76 (8)	527 (106)	98 (1)
Post-DYN	6.0 (1.2)	4.6 (0.9)	76 (8)	519 (87)	98 (1)
Post-DIVE	5.5 (1.2)	4.0 (1.1)	73 (13)	474 (111)	95 (7)
	$P < 0.01$	$P < 0.01$	$P = 0.13$	$P = 0.06$	$P < 0.05$

Values are means (SD) for 19 subjects. Post-DYN, after pool dives; Post-DIVE, after deep dives in the sea; FVC, forced vital capacity; FEV_1 , forced expiratory volume in the first second; FER, FEV_1 -to-FVC-ratio; PEF, peak expiratory flow; SaO_2 , arterial oxygen saturation. *P* values refer to comparisons between Post-DYN and Post-DIVE.

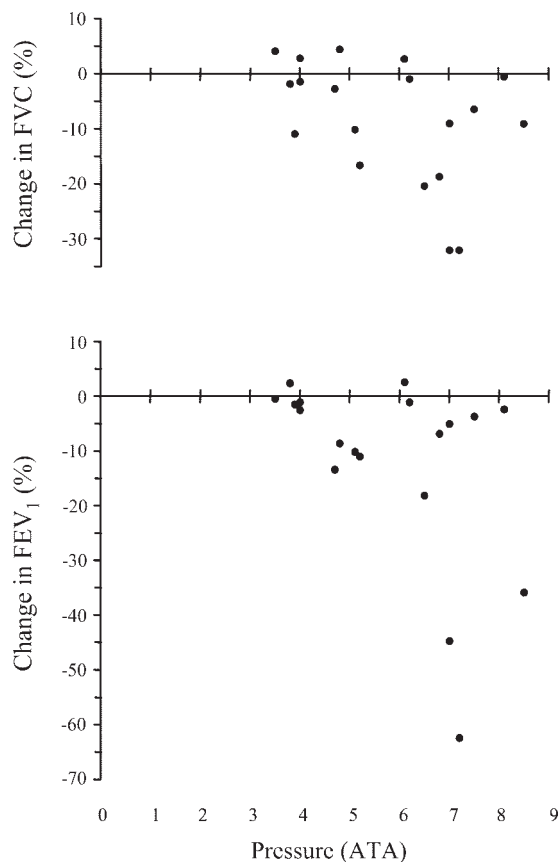


Fig. 1. Changes in forced vital capacity (FVC) and forced expiratory volume in the first second (FEV₁) after deep dives compared with after pool dives vs. the maximal absolute pressure to which the diver was exposed during deep diving.

due to severe discomfort and O₂-breathing because of the symptoms. Instead, this diver's post-DIVE spirometry measurement was performed 17 h after the dive (FVC: -6%; FEV₁: -4%). At the same time, the SaO₂ was 97% in this diver.

DISCUSSION

This is the first study showing reduced spirometric performance and arterial hypoxemia as consequences of deep breath-hold diving, and we suggest that the observations are caused by diving-induced pulmonary edema. These findings are in accordance with previous observations of reduced spirometric performance after simulated deep dives (17). In the present study, symptoms and signs related to pulmonary edema were observed in competitive breath-hold divers after deep dives, but not after pool dives performed just below the surface, in accordance with the notion that the effects are depth related. In divers experiencing symptoms of pulmonary edema, aggravated reductions in performance in the dynamic spirometry test and mild to severe hypoxemia were found. From the results of the present study, it must be concluded that the great depths reached by these elite apnea divers are associated with a risk of pulmonary edema.

The pathophysiology leading to the formation of pulmonary edema in breath-hold diving depends on an increase in intrathoracic blood volume and hence increased pulmonary capillary transmural pressure. Pulmonary capillary stress failure

represents a continuum of conditions from a low-permeability, hydrostatic type of pulmonary edema at slightly elevated capillary pressures, to a high-permeability form of edema or even frank hemorrhage at high capillary pressures (31, 32). During a breath hold at or near the surface with a large lung volume and relaxed respiratory muscles, the intrathoracic pressure is increased, relative to ambient, due to the elastic inward recoil of the chest wall (7). The increased intrathoracic pressure hinders venous return, resulting in decreased pulmonary blood flow and cardiac output, as well as decreased intrathoracic blood volume (7), cardiovascular changes that would not bring about pulmonary edema. Supporting this view was the lack of symptoms and signs of pulmonary edema after the shallow pool dives in the present study. In contrast, when a breath-hold diver descends to depth during a dive, the lung gas volume decreases due to increased ambient pressure, according to Boyle's law (pressure \times volume = constant). When the lung gas volume decreases, the intrathoracic pressure drops, relative to ambient pressure, due to decreased elastic inward recoil of the chest wall (7). This depth-related drop in intrathoracic pressure results in increased venous return, pulmonary blood flow, and cardiac output, as well as increased intrathoracic blood volume compared with breath holding at the surface (7, 20). Most likely, these changes cause the pulmonary capillary transmural pressure to increase (20). Measurements referred to here (7, 20) were performed in association with breath-hold dives to a depth of 20 m, where the subject's lung gas volume at depth was above RV. Diving to a depth where the lung gas volume is even lower would result in an even larger intrathoracic blood volume (6, 26), and a redistribution of as much as 1.5 liters of blood into the thorax has been discussed (8, 23). Diving in relatively cold water, as during the competition reported in the present study, most likely augments the intrathoracic blood pooling (21). The increase in intrathoracic blood volume compensating for decreased intrathoracic gas volume at depth is probably one contributing explanation to the fact that the theoretical depth limit has long since been surpassed by competitive breath-hold divers (23, 26). However, it stands to reason that this large volume of blood would encompass a considerable overdistension of the intrathoracic blood vessels and increased pulmonary capillary transmural pressure (11). Whether such deep dives are potentially harmful has not been studied systematically until now, although a few previous case reports describe breath-hold divers experiencing hemorrhage from the airways after deep dives (4, 5, 15, 29). In addition, Lindholm et al. (17) studied experienced breath-hold divers during breath-hold dives to a depth of 6 m. Dives were

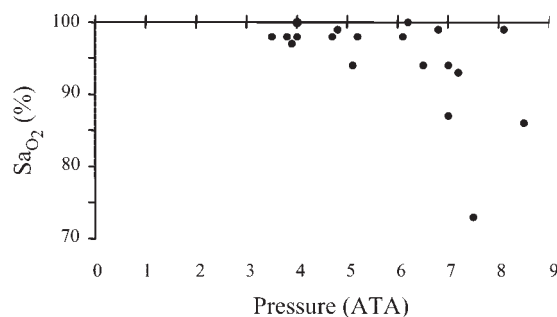


Fig. 2. Arterial oxygen saturation (SaO₂) after deep dives vs. the maximal absolute pressure to which the diver was exposed during deep diving.

begun with a starting lung volume below RV, using glossopharyngeal exhalation (18), thus simulating much deeper depths. After these dives, reductions in FVC and FEV₁ of 5 and 9%, respectively, were observed and attributed to the presence of edema in the lower airways (17). These results are comparable to the changes observed in the present study.

A comparison with other situations reported to have caused pulmonary edema may be of interest here. Immersion has been shown to be associated with pulmonary edema, in both swimmers and scuba divers (1, 16, 28). During head-out immersion, the water pressure on the body and the negative-pressure breathing increase the venous return and, consequently, pulmonary blood flow and cardiac output, as well as the intrathoracic blood volume and pulmonary arterial pressure (3). Adir et al. (1) found restrictive spirometric changes in a group of endurance swimmers with pulmonary edema, similar to the changes in the present study, namely a drop in FVC (−7%) and FEV₁ (−9%), but no change in FEV₁/FVC. In addition, the SaO₂ was decreased (−10%) in the swimmers with pulmonary edema in that study (1).

Radiographic imaging, computerized tomography, or magnetic resonance imaging could have been used to verify that the findings were caused by pulmonary edema (34). The reported symptoms (fatigue, dyspnea, cough, substernal chest pain or discomfort, and hemoptysis) are not exclusively found in association with pulmonary edema; e.g., hemoptysis could be caused by blood originating from blood vessels in the sinuses, upper or lower airways (17, 18), or the alveoli (4). Nevertheless, the restrictive pattern of changes in dynamic spirometry performance and the arterial hypoxemia after deep diving in the present study support the conclusion that the findings were caused by pulmonary edema. Such pulmonary restriction ascribed to pulmonary edema has previously been observed in other situations (1, 9, 14, 22). Alternative explanations for the reduced FVC and FEV₁ include a large intrathoracic blood volume, caused by immersion and diving, which could have the potential to cause restrictive changes. However, changes in intrathoracic blood volume during emersion are fast (19, 21). Thus a greater intrathoracic blood volume during postdive measurements than during control measurements is unlikely to have contributed to the spirometric observations. Atelectasis due to compression of alveoli during descent to depth can also be excluded as a contributing factor to the observations of the present study, as all alveoli should reopen during ascent (12) or during the maximal inspiration of the dynamic spirometry measurement. Any relative respiratory muscle fatigue, caused by the competitive performances, is not believed to be of any importance for the postdive restrictive pattern, as pool dives of comparable durations did not affect the spirometric performance.

The RV of the competitors in the present study was, for logistical reasons, not measured. However, for the sake of discussion, we adopted RV values from a normal values formula (13). For calculating the lung gas volume at the start of breath-hold dives, we used the anthropometrically predicted RV and the vital capacity measured during dynamic spirometry, performed in association with the control measurements, with glossopharyngeal inhalation, if pertinent. So calculated, the competitors in the present study reach RV during a dive, by an average, at a depth of 40 m (5 ATA). It is interesting to note that the present change in FVC and FEV₁ (Fig. 1), as well as

SaO₂ (Fig. 2), after a dive seems to be more affected by dives deeper than ~40 m than shallower breath-hold dives.

It could be that some individuals can dive to greater depths without being affected; e.g., the diver in the present study diving to 71 m had no signs of pulmonary edema. This diver had a vital capacity after glossopharyngeal inhalation of 9.5 liters and an anthropometrically predicted RV of 1.6 liters (TLC: 11.1 liters). Thus, with this volume of gas in the lungs at the beginning of the dive, at 71 m the gas volume would have been compressed, due to the Boyle's law effect alone, to 1.4 liters. Assuming that he would attempt a dive to 142 m, the lung gas volume would have been compressed to 0.7 liters. This is within the theoretical reduction in lung gas volume below RV of 1.0–1.5 liter that has been discussed as a consequence of the so-called "blood shift" (8, 23). However, it is not established whether or not the blood-containing structures of the chest could accommodate this volume of blood without any ill effects. It is noteworthy that the reduction in lung volume from the surface to 71 m (9.7 liters) is much larger than the further reduction from 71 to 142 m (0.7 liters), showing that additional gas compression and need for blood redistribution and chest compression decrease as depth increases.

Glossopharyngeal inhalation, i.e., overinflation of the lungs, could be a risk factor for pulmonary barotrauma (8, 23). West et al. (32) state that both an increase in capillary pressure and high states of lung inflation are important factors for stress failure of pulmonary capillaries. In the present study, no relationship between employment of predive glossopharyngeal inhalation and symptoms or signs could be observed, but this does not exclude the possibility that such a relationship exists.

The incidence of pulmonary edema during deep breath-hold diving has not been studied previously. Fitz-Clarke (10), acting as event physician, reported that one diver was affected by pulmonary edema during a competition in which 57 divers participated. However, it seems that not all divers in that competition were observed for detection of signs of pulmonary edema, and, therefore, also milder cases, not reported to the event physician by the affected diver, may have occurred. In the present study, 12 of the 19 volunteer divers (of the total 41 divers competing) had signs that we attribute to pulmonary edema. Furthermore, six divers were markedly affected, experiencing symptoms and showing more severe signs of pulmonary edema. We do not believe that any of the divers who did not volunteer for the measurements were markedly affected by pulmonary edema, but we cannot exclude the possibility that some of these divers were also affected to some extent. Nevertheless, the actual number of observations of pulmonary edema-related symptoms and signs leads us to the conclusion that pulmonary edema is not a rare consequence of deep breath-hold diving in humans.

In conclusion, we attribute the findings after deep breath-hold dives in the present study, i.e., symptoms, reduced performance on the dynamic spirometry test (restrictive pattern), decreased SaO₂, and findings upon chest auscultation, to pulmonary edema caused by lung squeeze and increased intrathoracic blood volume. Elite apnea divers, when diving to deep depths, are at risk of developing a depth-dependent pulmonary edema or even frank alveolar hemorrhage. These findings were obtained in a situation in which the divers were performing a limited number of dives (a few "warm-up dives" and one

competition dive). It remains to be investigated whether repeated diving to shallower depths, e.g., spear fishing, can provoke similar changes.

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REFERENCES

1. Adir Y, Shupak A, Gil A, Peled N, Keynan Y, Domachevsky L, Weiler-Ravell D. Swimming-induced pulmonary edema: clinical presentation and serial lung function. *Chest* 126: 394–399, 2004.
2. Agostoni E. Limitation to depths of diving: mechanics of chest wall. In: *Physiology of Breath-hold Diving and the Ama of Japan*, edited by Rahn H and Yokoyama T. Washington, DC: National Academy of Sciences, National Research Council, 1965, p. 139–145. (Publication no. 1341)
3. Arborelius M Jr, Balldin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. *Aerospace Med* 43: 592–598, 1972.
4. Boussuges A, Pinet C, Thomas P, Bergmann E, Sainy JM, Vervloet D. Haemoptysis after breath-hold diving. *Eur Respir J* 13: 697–699, 1999.
5. Boussuges A, Succo E, Bergmann E, Sainy JM. Intra-alveolar hemorrhage. An uncommon accident in a breath holding diver [in French]. *Presse Med* 24: 1169–1170, 1995.
6. Craig AB Jr. Depth limits of breath hold diving (an example of Fennology). *Respir Physiol* 5: 14–22, 1968.
7. Ferrigno M, Hickey DD, Linér MH, Lundgren CEG. Simulated breath-hold diving to 20 meters: cardiac performance in humans. *J Appl Physiol* 62: 2160–2167, 1987.
8. Ferrigno M, Lundgren CEG. Breath-hold diving. In: *Bennett and Elliott's Physiology and Medicine of Diving* (5th ed.), edited by Brubakk AO and Neuman TS. Edinburgh, UK: Saunders, 2003, p. 153–180.
9. Fischer R, Lang SM, Bergner A, Huber RM. Monitoring of expiratory flow rates and lung volumes during a high altitude expedition. *Eur J Med Res* 10: 469–474, 2005.
10. Fitz-Clarke JR. Adverse events in competitive breath-hold diving. *Undersea Hyperb Med* 33: 55–62, 2006.
11. Fitz-Clarke JR. Computer simulation of human breath-hold diving: cardiovascular adjustments. *Eur J Appl Physiol* 100: 207–224, 2007.
12. Fitz-Clarke JR. Mechanics of airway and alveolar collapse in human breath-hold diving. *Respir Physiol Neurobiol* 159: 202–210, 2007.
13. Grimby G, Söderholm B. Spirometric studies in normal subjects. III. Static lung volumes and maximum voluntary ventilation in adults with a note on physical fitness. *Acta Med Scand* 173: 199–206, 1963.
14. Hosenpud JD, Stibolt TA, Atwal K, Shelley D. Abnormal pulmonary function specifically related to congestive heart failure: comparison of patients before and after cardiac transplantation. *Am J Med* 88: 493–496, 1990.
15. Kiyan E, Aktas S, Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. *Chest* 120: 2098–2100, 2001.
16. Koehle MS, Lepawsky M, McKenzie DC. Pulmonary oedema of immersion. *Sports Med* 35: 183–190, 2005.
17. Lindholm P, Ekborn A, Gennser M. Pulmonary squeeze and haemoptysis after breath-hold diving. An experimental study in humans. In: *XXIX Annual Scientific Meeting of the European Underwater and Baromedical Society*, edited by Jansen EC, Risby Mortensen C, and Hyldegaard O. Copenhagen, Denmark: European Underwater and Baromedical Society, 2003, p. 38.
18. Lindholm P, Nyren S. Studies on inspiratory and expiratory glossopharyngeal breathing in breath-hold divers employing magnetic resonance imaging and spirometry. *Eur J Appl Physiol* 94: 646–651, 2005.
19. Linér MH. Tissue gas stores of the body and head-out immersion in humans. *J Appl Physiol* 75: 1285–1293, 1993.
20. Linér MH, Linnarsson D. Intrapulmonary distribution of alveolar gas exchange during breath-hold diving in humans. *J Appl Physiol* 78: 410–416, 1995.
21. Lundgren CEG. Immersion effects. In: *The Lung at Depth*, edited by Lundgren CEG and Miller JN. New York: Dekker, 1999, p. 91–128.
22. Mason NP, Barry PW, Pollard AJ, Collier DJ, Taub NA, Miller MR, Milledge JS. Serial changes in spirometry during an ascent to 5300 m in the Nepalese Himalayas. *High Alt Med Biol* 1: 185–195, 2000.
23. Muth CM, Ehrmann U, Radermacher P. Physiological and clinical aspects of apnea diving. *Clin Chest Med* 26: 381–394, 2005.
24. Örnthagen H, Schagatay E, Andersson J, Bergsten E, Gustafsson P, Sandström S. Mechanisms of “buccal pumping” (“lung packing”) and its pulmonary effects. In: *XXIV Annual Scientific Meeting of the European Underwater and Baromedical Society*, edited by Gennser M. Stockholm, Sweden: National Defence Research Establishment, 1998, p. 80–83.
25. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal Official Statement of the European Respiratory Society. *Eur Respir J Suppl* 16: 5–40, 1993.
26. Schaefer KE, Allison RD, Dougherty JH Jr, Carey CR, Walker R, Yost F, Parker D. Pulmonary and circulatory adjustments determining the limits of depths in breathhold diving. *Science* 162: 1020–1023, 1968.
27. Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins CR. Features of glossopharyngeal breathing in breath-hold divers. *J Appl Physiol* 101: 799–801, 2006.
28. Slade JB Jr, Hattori T, Ray CS, Bove AA, Cianci P. Pulmonary edema associated with scuba diving: case reports and review. *Chest* 120: 1686–1694, 2001.
29. Strauss MB, Wright PW. Thoracic squeeze diving casualty. *Aerospace Med* 42: 673–675, 1971.
30. West JB. Invited review: Pulmonary capillary stress failure. *J Appl Physiol* 89: 2483–2489, 2000.
31. West JB, Mathieu-Costello O. Stress failure of pulmonary capillaries: role in lung and heart disease. *Lancet* 340: 762–767, 1992.
32. West JB, Tsukimoto K, Mathieu-Costello O, Prediletto R. Stress failure in pulmonary capillaries. *J Appl Physiol* 70: 1731–1742, 1991.
33. Williams AJ. ABC of oxygen: assessing and interpreting arterial blood gases and acid-base balance. *BMJ* 317: 1213–1216, 1998.
34. Zavorsky GS. Evidence of pulmonary oedema triggered by exercise in healthy humans and detected with various imaging techniques. *Acta Physiol (Oxf)* 189: 305–317, 2007.