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ORIGINAL ARTICLE

Ultrasound lung "comets" increase after breath-hold diving

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Abstract The purpose of the study was to analyze the ultrasound lung comets (ULCs) variation, which are a sign of extra-vascular lung water. Forty-two healthy individuals performed breath-hold diving in different conditions: dynamic surface apnea; deep variable-weight apnea and shallow, face immersed without effort (static maximal and non-maximal). The number of ULCs was evaluated by means of an ultrasound scan of the chest, before and after breath-hold diving sessions. The ULC score increased significantly from baseline after dynamic surface apnea ($p = 0.0068$), after deep breath-hold sessions ($p = 0.0018$), and after static maximal apnea ($p = 0.031$). There was no statistically significant difference between the average increase of ULC scores after dynamic surface apnea and deep breath-hold diving. We, therefore, postulate that extravascular lung water accumulation may be due to other factors than (deep) immersion alone, because it occurs during dynamic surface apnea as well. Three mechanisms may be responsible for this. First, the immersion-induced hydrostatic pressure gradient applied on the body causes a shift of peripheral venous blood towards the thorax. Second, the blood pooling effect found during the diving response Redistributes blood to the pulmonary vascular bed. Third, it is possible that the intense involuntary diaphragmatic contractions occurring during the "struggle phase" of the breath-hold can also produce a blood shift from the pulmonary capillaries to the pulmonary alveoli. A combination of these factors may explain the observed increase in ULC scores in deep, shallow maximal and shallow dynamic apneas, whereas shallow non-maximal apneas seem to be not "ULC provoking".

Keywords Apnea · Extravascular lung water · Edema · Hypoxia · Static · No-limits · Underwater swimming

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Introduction

Capillary stress failure inducing lung water accumulation may occur in otherwise healthy individuals as a result of exposure to environmental stress (West and Mathieu-Costello 1995a), as well as during underwater immersion (Koehle et al. 2005; Slade et al. 2001).

The prevalence of lung water accumulation (acute pulmonary edema) during immersion was reported and studied previously during scuba-diving or swimming (Hampson and Dunford 1997; Pons et al. 1995; Weiler-Ravell et al. 1995; Wilmshurst et al. 1989). The occurrence of pulmonary edema in swimmers and divers was reported for the first time by Wilmshurst and co-workers. These

investigators reported up to 7 episodes of pulmonary edema in 11 individuals whilst scuba-diving or swimming (Wilmshurst et al. 1989).

Some anecdotal case-reports appeared in breath-hold divers as well (Boussuges et al. 1999; Fitz-Clarke 2006; Kalemoglu and Keskin 2006). Later, it has been shown that early after breath-hold diving sessions, there is an obvious reversible increase in ultrasound lung comets (ULCs) (Frassi et al. 2008) which are a sign of extra-vascular lung water accumulation and a surrogate measure of pulmonary edema (Picano et al. 2006).

Acute pulmonary edema occurs when the pulmonary capillary permeability is increased (non-cardiogenic), when the pulmonary capillary hydrostatic pressure exceeds the plasma oncotic pressure (cardiogenic), or both (West and Mathieu-Costello 1995a). The exact nature of the stress experienced by scuba divers and immersion victims is not clear but may be due to raised pulmonary capillary pressure.

In swimmers and SCUBA divers, an increased transalveolar pressure gradient due to a combination of factors has been implicated in the pathogenesis of the condition. The final common pathway appears to be "stress failure" or in another way a sort of "hyperfiltration" of pulmonary capillaries manifested by leaks in the capillary endothelial layer and the alveolar epithelial layer such as can be found in non-diving-related conditions such as cardiac failure (West and Mathieu-Costello 1995b).

Ferretti reviewed data from others regarding radiologic findings in apnea divers under water, including a substantial reduction in lung volume, an elevation of the diaphragm, and an engorgement of the lung blood vessels (Ferretti 2001). Indeed, in breath-hold divers, the centralization of blood volume from the periphery to the intrapulmonary vessels leads to engorgement and over-distention of pulmonary vessels and an elevation of the capillary pulmonary pressure, which may lead to a fluid shift from the capillary lumen into the alveolar interstitium and/or to alveolar hemorrhage (Muth et al. 2005).

Very recent data recorded at depth during breath-hold diving by means of a submersible Doppler-echocardiograph showed signs of impaired diastolic left ventricular filling (Marabotti et al. 2009), similar to the hemodynamic pattern observed in constrictive/restrictive heart diseases. This situation could be due to the combined effects of chest volume reduction (Ferrigno et al. 1997) and intrathoracic blood content increase, thus contributing to capillary hyperfiltration.

As reported above, an increase in extravascular lung water may be evaluated by means of an ultrasound scan of the lung, mirrored by appearance of ULCs (Picano et al. 2006). This echographic sign detectable with cardiac probes along the intercostals spaces consists of multiple comet-tails fanning out from the lung surface (Lichtenstein et al. 1997) (see, Fig. 1). A patient ULC score denoting the extent of the extravascular fluid of the lung can be obtained by summing the number of ULCs on the chest (Frassi et al. 2007). The presence of a few scattered comets can be a normal variant, especially in the lower intercostal spaces. Below or equal to the threshold of five comets found in the whole scanning field, the signal is considered to be an insignificant clinical noise (Picano et al. 2006).

The aim of this study was to analyze the ULCs variations present after different breath-hold performances: first, shallow immersion with consistent effort (Dynamic apnea in swimming pool; mono-fin swimming), second, deep apnea diving with minimal effort (no-limits); and third, shallow, face immersed without effort (maximal and non-maximal static surface apnea in a swimming pool).

Methods

Subjects

Forty-two healthy individuals aged 35 ± 10 years (mean ± SD); (2 women and 40 men) volunteered to participate in this study. All subjects were healthy breath-hold divers, participating in different apnea events or competitions, although not all of them were highly trained. None of the subjects reported a medical history of cardiovascular events except. The subjects were chosen within the participants that were not performing "lung packing" (glossopharyngeal lung insufflations), since this maneuver can interfere with some parameters involved in our investigation.

Subjects with more than five comets before the breath-hold diving session were excluded from the study. The protocol was submitted to our institutional Academic Ethical Committee which gave its approval. Before participating, all subjects gave their written informed consent.

Protocol and measurements

Measurements were made before and after breath-hold diving sessions held in four different conditions: dynamic surface apnea, deep breath-hold diving (variable-weight apnea, "no-limits"), static surface maximal, and non-maximal apnea.

Dynamic surface apnea was performed with monofin. After a deep inhalation the athletes swam as far as possible horizontally under water (just below the surface in a 1.5 m depth pool); the mean distance covered was 137 ± 30 m and the duration of the swims 113 ± 26 s (mean ± SD) ($n = 15$).

Depth variable-weight apnea (no-limits freediving) consisted of using a weighted sled for descent and, attached

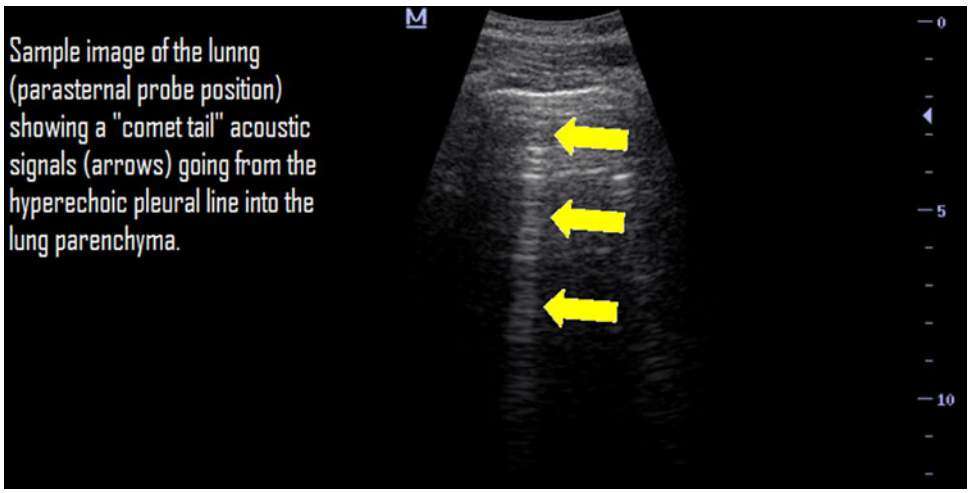

Fig. 1 Ultrasound lung comets (ULCs) image

to the sled, an air balloon which was inflated by means of an air tank providing a lift for the ascent to the surface. The mean depth and duration of the dives were 50 ± 10 m (mean ± SD) and 120 ± 30 s (mean ± SD), respectively ($n = 19$). Dynamic surface apnea requires heavy physical effort; while no-limits free-diving requires little physical activity and is focused on the ability to cope with the high water pressure at depth and with descending and ascending as quickly as possible while breath-holding (Muth et al. 2005).

Static shallow breath-hold dives were performed in two ways. One time, they made several maximal apneas to reach a total time of at least 5 min (300 s); the mean time reached was 318 ± 21 s (mean ± SD) (maximal static apnea) ($n = 8$). On another day, the same subjects made 10 successive apneas of 30 s with 30 s of rest between each one (non-maximal static apnea) ($n = 8$). Those two types of breath-holding required absolutely no physical activity and subjects were well relaxed. However, during the "struggle phase" of maximal apneas, 7 out of 8 of the subjects underwent intense involuntary diaphragmatic contractions.

The chest echographic examination was performed by the same investigator on each subject in supine position, before and within 10 min after each breath-hold diving sessions. The investigator was blinded as to the apnea type performed by each subject tested. All ultrasound scans were performed with two-dimensional sonography machine (Mindray M5, Mindray Medical International Limited, Shenzhen, China), with a cardiac sectorial probe (2.5–3.5 MHz). The subjects were studied in a quiet, temperature controlled room.

The methods previously described by Picano et al. (2006) were used: ultrasound scanning of the anterior and lateral chest was obtained on the right and left hemithorax, from the second to the fourth (on the right side to the fifth) intercostal space, and from the parasternal to axillary line. In each intercostal space, the number of ULCs (Fig. 1) was noted. The sum of ULCs yielded a score denoting the extent of the extravascular fluid of the lung. Zero was defined as a complete absence of ULCs on the investigated area (Frassi et al. 2007).

Statistical analysis

The increase in the number of ULCs was calculated in two ways: first, the number of "new" ULCs was calculated as the difference between ULCs before and after each apnea session. This analysis was performed for all experimental groups.

Then, the proportion of subjects with ULCs above $n = 5$, before and after each apnea session was calculated and analysed.

After normality testing of the sample with Kolmogorov–Smirnov test, a non-parametric approach was decided (Mann–Whitney and Wilcoxon signed rank tests). For the proportions statistics, the Fisher's exact test was chosen. The significance level was set at a p value <0.05.

Results

One subject in the dynamic shallow diving group was excluded because at baseline, more than 5 ULCs were measured. The ULC score increased significantly from baseline to post exercise after dynamic shallow breath-hold diving sessions ($p = 0.0068$; Fig. 2) and after deep breath-hold diving sessions ($p = 0.0018$; Fig. 3).

The comparisons of the baseline number of comets in both experimental groups showed no statistical difference ($p = 0.52$), neither after exposure ($p = 0.10$).

The ULCs score increased for 68.75% ($n = 11$) of subjects after dynamic shallow breath-hold diving sessions and for 68.42% ($n = 13$) of subjects after deep variable-weight

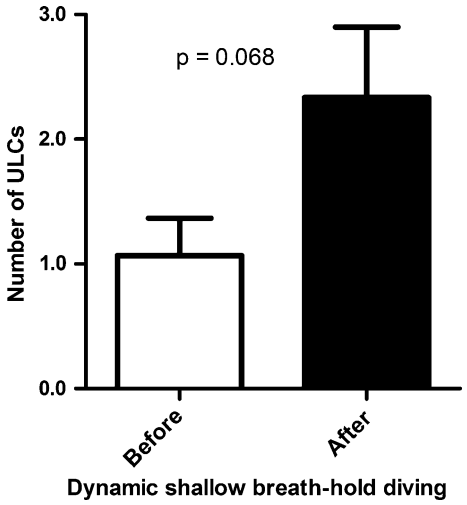

Fig. 2 Evolution of the ULCs scores from baseline to post exposure after dynamic shallow breath-hold diving (1.5 m depth pool and mean distance: 138 ± 26 m) (mean ± SEM). Significant increase of number of ULCs after breath-hold diving sessions ($n = 15$) (mean ± SEM)

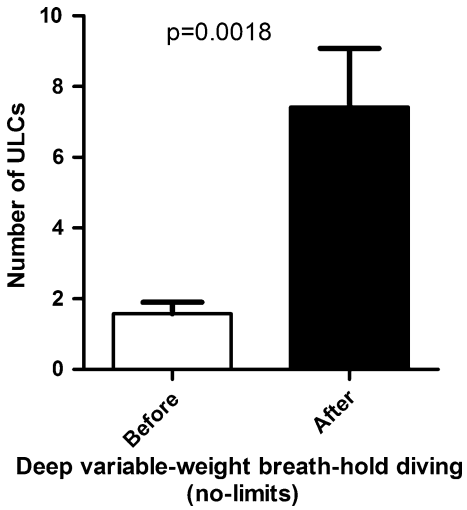

Fig. 3 Evolution of ULCs scores from baseline to post exposure after deep variable-weight breath-hold diving (deep: 50 ± 10 m) (mean ± SEM). Significant increase of ULCs score breath-hold diving sessions ($n = 19$) (mean ± SEM)

breath-hold diving sessions. The Fisher's exact test indicated no significant difference of proportions between the two groups ($p > 0.5$).

Neither was a significant difference found between the number of subjects who had a number of ULCs above 5 after depth variable-weight breath-hold diving sessions and dynamic shallow breath-hold diving sessions ($p > 0.5$).

Concerning static breath-hold diving, as illustrated in Figs. 4, 5, the comparison of the baseline numbers of comets in both experimental groups (maximal and non-maximal) showed no statistical difference ($p = 0.50$).

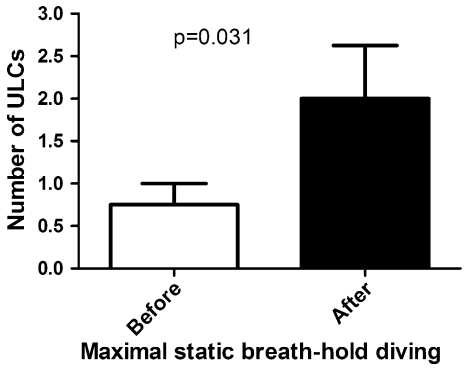

Fig. 4 Evolution of the ULCs scores from baseline to post exposure during several maximal static shallow breath-hold diving (a total of 318 ± 21 s). Significant increase of number of ULCs after breath-hold diving sessions (Wilcoxon signed ranked test $p = 0.031$) ($n = 8$) (mean ± SEM)

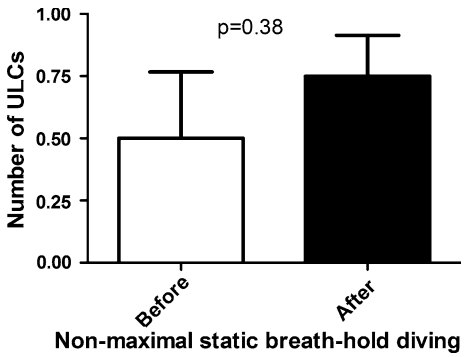

Fig. 5 Evolution of the ULCs scores from baseline to post breath-hold values during several non-maximal static shallow breath-hold diving (10 times 30 s of apnea). No Significant difference before and after breath-hold diving sessions ($p = 0.35$) ($n = 8$) (mean ± SEM)

The number of ULCs increased significantly during maximal apnea tests ($p = 0.03$; Fig. 4), contrary to non-maximal apnea values where the difference between the baseline and the post-test values was non-significant ($p = 0.37$; Fig. 5).

Discussion

Methodology

The non-invasive nature, easy and fast applicability of chest echography have important advantages. The ULCs process has been used and approved in several experimental studies before (Agricola et al. 2005; Bedetti et al. 2006; Frassi et al. 2007; Frassi et al. 2008; Jambrik et al. 2004; Picano et al. 2006). To find the signs of ULCs in the present study, we used the methods previously described by Picano et al. (2006), which consist in scanning only anterior chest compared to another recent study where data

were derived from scans of anterior and posterior sites (Frassi et al. 2008). It would have been interesting to also explore the posterior chest in order to expand the mapping of the chest and thus increase the probability of finding ULCs. But it would have doubled the scanning time and made difficult to scan each subject within 10 min after the breath-hold diving sessions.

Results

After different breath-hold performances (breath-hold mono-fin swimming—dynamic surface apnea—and deep variable-weight—"no-limits apnea"), the ULC scores increased significantly for most subjects, as observed in a previous study in top-level breath-hold divers (Frassi et al. 2008). Thus, it seems clear that breath-hold diving has a significant effect on extra-vascular lung water accumulation, which can be interpreted as a primitive form of pulmonary edema. However, the precise causative factor is not yet clear.

Nevertheless, the main finding of the present study is that there was no significant difference between the two groups in terms of the increase in ULCs resulting from each of the two types of dives. Thus, the average increase of ULCs score after depth breath-hold diving was similar to the average increase of ULCs after dynamic shallow breath-hold diving. This suggests that we cannot assert that the accumulation of extravascular lung water during breath-hold diving sessions is only due to the depth of the immersion.

A similar recent study (Liner and Andersson 2008) performed an analysis of clinical symptoms and spirometric data after breath-hold diving. The subjects had to perform several contests during the measurements: dynamic shallow, fin assisted and fin unassisted, fin assisted and fin unassisted deep breath-hold, and finally pulling themselves down and up along the diving line. All those exercises are quite strenuous and effort demanding. The authors found a significant difference in the clinical outcomes and spirometric data after the deep dives rather the shallow ones.

Our study protocol is quite different, and this could explain the apparent discrepancy of our data. The "no-limit" deep apnea is less effort demanding than any of the exercises imposed by Liner et al. Moreover, in their study, a majority of the divers (15/19) was performing glossopharyngeal breathing ("lung packing"), which in itself may provoke pulmonary damage (Muth et al. 2005). Our static data (Figs. 4, 5) illustrate that, for the same subjects, in a non-effort setting, maximal breath-holding provokes ULCs whereas the same total duration of apnea (but non-maximal) did not significantly increase ULC scores.

In opposition to the measurements taken by Liner et al., ULCs can be considered a more sensitive, be it semi-quantitative indication of lung edema; indeed, since a small amount of interstitial fluid will generate a significant "comet tail" image, ULCs can not be taken as an indicator of significant pulmonary oedema.

Mechanisms

In our results, we found that the number of ULCs increased significantly during maximal static surface breath-hold diving sessions but non-significantly during non-maximal static shallow breath-hold diving sessions. On the other hand, a relatively short (2 min) deep breath-hold dive without significant effort or maximal duration of apnea, provokes a very significant increase. It seems thus that depth, physical effort and maximum breath-hold can all induce this phenomenon.

Several fundamental mechanisms may be invoked to explain this.

Immersion

First, as described in right-heart catheterized subjects sitting immersed to the neck, immersion induces hemodynamic changes including the "cardiac tamponade pattern". The hydrostatic pressure gradient applied on the body causes an immediate shift of peripheral venous blood towards the thorax (Arborelius et al. 1972).

In this case, ULCs would be expected to increase in all groups, independent of depth, effort of apnea duration.

Moreover, during deep breath-hold diving the intrathoracic volume is decreased (Boyle's Law) favouring fluid shifts into the interstitial pulmonary space (Muth et al. 2005, Fitz-Clarke 2006).

Diving response

Second, during breath-hold diving sessions, different cardiovascular modifications called "diving response" occur to the apneist in order to save up oxygen (Delapille et al. 2002). This diving response can be induced by voluntary or involuntary breath-holding and reinforced by the face immersion in cold water. Inputs primarily from trigeminal receptors on the upper part of the face are integrated by the respiratory and cardiovascular in the brainstem. Later, inputs from aortic and carotid chemoreceptors help maintain the diving response. Those inputs generate neural signals to the lung, heart, and arterioles that include both sympathetic and parasympathetic divisions of the autonomic nervous system (Hiebert and Burch 2003).

Accordingly, an extreme peripheral vasoconstriction is associated with a dramatic increase in arterial blood

pressure (Heusser et al. 2009). The consequent stimulation of arterial baroreceptors causes an extreme drop of heart rate. Bradycardia is not compensated by a higher stroke volume, with a consequent decrease in cardiac output (Ferrigno et al. 1997; Marabotti et al. 2009). This decrease, however, is not such as to undermine perfusion to vital organs. A redistribution of blood volume occurs, reducing circulation to the muscles and other tissues while maintaining blood flow to the brain and heart (Ferretti and Costa 2003). This redistribution of blood volume leads to an increasing flow of the pulmonary bloodstream can induce an engorgement of pulmonary vessels and an increase of the capillary pulmonary pressure. And as shown by Muth et al. (2005), a pressure elevation in pulmonary capillaries may lead to a fluid shift from the capillary lumen into the alveolar interstitium causing an extravascular lung water accumulation.

In this case, the same "universal" increase in ULCs would have been observed in all groups.

Struggle phase (diaphragmatic and intercostal muscular contractions)

Another possible explanation for this phenomenon refers to the intense involuntary diaphragmatic contractions in breath-hold divers during the so-called "struggle phase" at the end of dives. These involuntary contractions progressively increase in frequency and intensity, creating a negative intrathoracic pressure (Kiyan et al. 2001).

It has been shown in previous studies that forceful inspiratory effort against an obstructed extrathoracic airway (in patients with obstructive sleep apnea) allows the markedly negative intrapleural pressure to be transmitted almost completely to the perivascular interstitium, thereby facilitating fluid movement into this compartment (Ferretti and Costa 2003; Timby et al. 1990).

Furthermore, a more recent study has demonstrated that each involuntary breathing movement coincides with an abrupt increase in inferior vena cava flow. This supports the view that involuntary breathing movements related decreases in intrathoracic pressure are at least partly involved in the restoration of the venous return (Palada et al. 2008).

As referred to, in our study, the participants performing "lung packing" were excluded. This practice is known to be able, by itself, to cause alveolar damage leading to increased fluid permeability (Lindholm and Lundgren 2009). Moreover, if dives would have begun on a large lung volume, substantial intrathoracic blood pooling may have been prevented because the elastic chest recoil is likely to counteract pulmonary vascular engorgement and thus reduce any tendency for abnormal extravasation of water. And finally, the inspiratory efforts during the struggle phase may not have generated much negative pressure if they occurred close to or above total lung capacity where the inspiratory muscles are at a mechanical disadvantage.

This mechanism could be consistent with the fact that maximum duration breath-hold (dynamic surface apnea and maximal static apnea) provoked significant increases of ULC scores (as opposed to non-maximal static breath-holds); however, the increase observed during deep breath-hold is less likely to be explained by this mechanism, the struggle phase being limited or absent (owing to the shorter duration and the higher partial pressure of oxygen at depth).

Combination of factors

We may speculate that the increased venous return (immersion plus physical effort) adding to the diving response contributes to the pressure elevation in pulmonary capillaries, leading to a fluid shift into the pulmonary alveoli. The struggle phase contributes certainly to this venous return, however, depending on the type of breath-hold diving, the relative importance of all these phenomena will vary.

For example, heavy middle ear equalization efforts performed with trunk movements or using abdominal strain at depth could also provoke similar diaphragmatic and capillary wall stress, since it has been observed that during these maneuvers, intrathoracic pressure variations do occur (Balestra et al. 1998); anecdotal evidence of this may be that some divers, who developed other equalizing techniques to avoid the phenomenon [such as "mouth fill" or even sinuses and middle ear flooding (Germonpre et al. 2008)], seem to suffer less from pulmonary symptoms after their dives.

Conclusions

In conclusion, we have reported that the variation of ULCs after different types of breath-hold diving is not solely attributable to depth or effort alone, but to a combination of several factors: hemodynamic changes induced by the hydrostatic pressure gradient applied on the body, the diving response and the intense involuntary diaphragmatic, trunk and abdominal contractions occurring during the struggle phase. We also suggested that some heavy equalization efforts (Valsalva like maneuvers) can also provoke such pulmonary stress.

References

Agricola E, Bove T, Oppizzi M, Marino G, Zangrillo A, Margonato A, Picano E (2005) "Ultrasound comet-tail images": a marker of pulmonary edema: a comparative study with wedge pressure and extravascular lung water. Chest 127:1690–1695

Arborelius M Jr, Ballidin UI, Lilja B, Lundgren CE (1972) Hemodynamic changes in man during immersion with the head above water. Aerosp Med 43:592–598

Balestra C, Germonpre P, Marroni A (1998) Intrathoracic pressure changes after Valsalva strain and other maneuvers: implications for divers with patent foramen ovale. Undersea Hyperb Med 25:171–174

Bedetti G, Gargani L, Corbisiero A, Frassi F, Poggianti E, Mottola G (2006) Evaluation of ultrasound lung comets by hand-held echocardiography. Cardiovasc Ultrasound 4:34

Boussuges A, Pinet C, Thomas P, Bergmann E, Sainty JM, Vervloet D (1999) Haemoptysis after breath-hold diving. Eur Respir J 13:697–699

Delapille P, Verin E, Tourny-Chollet C (2002) Ventilatory adaptations for breath holding in divers. Rev Mal Respir 19:217–228

Ferretti G (2001) Extreme human breath-hold diving. Eur J Appl Physiol 84:254–271

Ferretti G, Costa M (2003) Diversity in and adaptation to breath-hold diving in humans. Comp Biochem Physiol A Mol Integr Physiol 136:205–213

Ferrigno M, Ferretti G, Ellis A, Warkander D, Costa M, Cerretelli P, Lundgren CE (1997) Cardiovascular changes during deep breath-hold dives in a pressure chamber. J Appl Physiol 83:1282–1290

Fitz-Clarke JR (2006) Adverse events in competitive breath-hold diving. Undersea Hyperb Med 33:55–62

Frassi F, Gargani L, Gligorova S, Ciampi Q, Mottola G, Picano E (2007) Clinical and echocardiographic determinants of ultrasound lung comets. Eur J Echocardiogr 8:474–479

Frassi F, Pingitore A, Cialoni D, Picano E (2008) Chest sonography detects lung water accumulation in healthy elite apnea divers. J Am Soc Echocardiogr 21:1150–1155

Germonpre P, Balestra C, Musimu P (2008) Passive flooding of paranasal sinuses and middle ears as a method of equalisation in extreme breath-hold diving. Br J Sports Med

Hampson NB, Dunford RG (1997) Pulmonary edema of scuba divers. Undersea Hyperb Med 24:29–33

Heusser K, Dzamonja G, Tank J, Palada I, Valic Z, Bakovic D, Obad A, Ivancev V, Breskovic T, Diedrich A, Joyner MJ, Luft FC, Jordan J, Dujic Z (2009) Cardiovascular regulation during apnea in elite divers. Hypertension 53:719–724

Hiebert SM, Burch E (2003) Simulated human diving and heart rate: making the most of the diving response as a laboratory exercise. Adv Physiol Educ 27:130–145

Jambrik Z, Monti S, Coppola V, Agricola E, Mottola G, Miniati M, Picano E (2004) Usefulness of ultrasound lung comets as a nonradiologic sign of extravascular lung water. Am J Cardiol 93:1265–1270

Kalemoglu M, Keskin O (2006) Hemoptysis and breath-holding diving. Mil Med 171:606–607

Kiyan E, Aktas S, Toklu AS (2001) Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest 120:2098–2100

Koehle MS, Lepawsky M, McKenzie DC (2005) Pulmonary oedema of immersion. Sports Med 35:183–190

Lichtenstein D, Meziere G, Biderman P, Gepner A, Barre O (1997) The comet-tail artifact. An ultrasound sign of alveolar-interstitial syndrome. Am J Respir Crit Care Med 156:1640–1646

Lindholm P, Lundgren CE (2009) The physiology and pathophysiology of human breath-hold diving. J Appl Physiol 106:284–292

Liner MH, Andersson JP (2008) Pulmonary edema after competitive breath-hold diving. J Appl Physiol 104:986–990

Marabotti C, Scalzini A, Cialoni D, Passera M, L'Abbate A, Bedini R (2009) Cardiac changes induced by immersion and breath-hold diving in humans. J Appl Physiol 106:293–297

Muth CM, Ehrmann U, Radermacher P (2005) Physiological and clinical aspects of apnea diving. Clin Chest Med 26:381–394, v

Palada I, Bakovic D, Valic Z, Obad A, Ivancev V, Eterovic D, Shoemaker JK, Dujic Z (2008) Restoration of hemodynamics in apnea struggle phase in association with involuntary breathing movements. Respir Physiol Neurobiol 161:174–181

Picano E, Frassi F, Agricola E, Gligorova S, Gargani L, Mottola G (2006) Ultrasound lung comets: a clinically useful sign of extravascular lung water. J Am Soc Echocardiogr 19:356–363

Pons M, Blickenstorfer D, Oechslin E, Hold G, Greminger P, Franzeck UK, Russi EW (1995) Pulmonary oedema in healthy persons during scuba-diving and swimming. Eur Respir J 8:762–767

Slade JB Jr, Hattori T, Ray CS, Bove AA, Cianci P (2001) Pulmonary edema associated with scuba diving: case reports and review. Chest 120:1686–1694

Timby J, Reed C, Zeilender S, Glauser FL (1990) "Mechanical" causes of pulmonary edema. Chest 98:973–979

Weiler-Ravell D, Shupak A, Goldenberg I, Halpern P, Shoshani O, Hirschhorn G, Margulis A (1995) Pulmonary oedema and haemoptysis induced by strenuous swimming. BMJ 311:361–362

West JB, Mathieu-Costello O (1995a) Stress failure of pulmonary capillaries as a limiting factor for maximal exercise. Eur J Appl Physiol Occup Physiol 70:99–108

West JB, Mathieu-Costello O (1995b) Vulnerability of pulmonary capillaries in heart disease. Circulation 92:622–631

Wilmshurst PT, Nuri M, Crowther A, Webb-Peploe MM (1989) Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. Lancet 1:62–65