

Review Article

What happens to Human Lungs during Breath-Hold Diving?

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Abstract

The underwater environment is characterized by an increase of the hydrostatic pressure surrounding the body who is immersed. Each 10 meters under the sea-surface the hydrostatic pressure increases by one atmosphere. It turns that this increase can elicit some physiological and/or pathological response in terms of respiratory and subsequently cardiac homeostasis which needs to be known. Centralization of blood volume from periphery of the body, reduction of lung volumes, abnormal lung mechanics, gas exchange disorders, changes in pulmonary vascular resistances, ventilation and perfusion mismatch, sensory disorders and mental status are some of them. The entities of these reactions depend on the depth that is reached, the period of immersion, how diving is performed, if this is done in breath holding or whether it is performed by use of breathing gas mixtures with special apparatuses. Other contributing factors may include hemodynamic changes due to the water immersion, cold exposure, wearing wear suits, exertion with elevated cardiac output.

Objectives of this review is to outline relevant features derived from papers dealing mainly with the mechanisms responsible for the respiratory modifications during underwater apnea especially for the dramatic effects above the integrity of the alveolar capillary membrane. This effort has been done in order to stress much more the concept that a thorough evaluation of the pulmonary function is mandatory whenever subjects wish to perform the breath-holding apnea or where they have to get a recreational diving licence.

In order thus to get this purpose, most relevant publications, appeared in these last ten years on breath-hold diving activities, have been reviewed and their results have been pointed out.

One of the main results of these works outlines the fact that the health of recreational divers may be endangered by high extracorporeal ambient pressures with its many cardio-pulmonary effects. During breath-holding diving apnea, the reduction of static lung volumes seems at the same time associated with a peripheral vasoconstriction (maximum in the muscle and skin organs) and derived consequent redistribution of the blood volume at the thoracic level ("blood shift") and, in particular, from the areas of the lung bases to the middle and apical regions. The redistribution of ventilation and blood flow inside the lungs may be responsible for the significant increase observed in the diffusion of carbon monoxide along the alveolar capillary membrane within the first two minutes from an apnea dive, followed by a progressive reduction in the following minutes lower than baseline values. The increase in the diffusion of the CO within the first minutes may suggest persistence of the "blood shift" (effect of increasing the pulmonary capillary blood volume). Another intriguing aspect of the health status during apnea is the fact that pulmonary gas exchange may be deteriorated in the course of repeated diving as result of the occurrence of interstitial edema at the level of the alveolar-capillary membrane.

From these evaluations it turns out that there is a need to have a more accurate evaluation of the lung function, especially when people are going to apply for getting the licence for recreational activities related to breath-holding dive.

It could be hypothesized that the evaluation of diffusion capacity along the alveolar capillary membrane may be added to the respiratory work-up during the procedures to declare a person fit to breath-hold diving.

Keywords: Breath-hold diving; Lung function; Pulmonary edema; Gases; Diffusion of carbon monoxide

INTRODUCTION

Free diving is an athletic and recreational activity; during this process many organs and their functions can be altered, mainly due to the effects of water pressure. The consequence of this environment is the centralization of blood volume from the periphery of the body, abnormal lung mechanics, sensory disorders and mental status [1,2]. The entities of these stresses and changes depend on the depth that is reached, the period of immersion but also on the modalities of diving. These can be of two types: the breath holding (apnoea) method or whether it is performed by use of breathing apparatuses and gas mixtures;

both influence the thoracic and lung mechanics. The main effects are on static lung volumes and on the hemodynamic, mainly due to changes in distribution of pulmonary blood flow, with a gravitational redistribution of blood and subsequently increase in pulmonary resistances [1,2].

The aim of this review is to outline the results and the considerations attained from literature on the effect of breath-hold diving on the cardio-thoracic system.

METHODS

In this concise review the most relevant published works on these topics are reviewed and their main results are evidenced.

RESULTS AND DISCUSSION

The underwater apnoea technique is also responsible for the changes in respiratory gases such as oxygen and carbon dioxide, at the level of alveolar capillary membrane. During apnoea, the increase in hydrostatic pressure results in important effects either on the respiratory mechanics and on the cardiovascular system. In fact, the changes of static lung volumes seems to be associated with a peripheral vasoconstriction (maximum in the muscle and skin organs) and consequent redistribution of blood volume at the thoracic level ("blood shift"). In particular, the areas of the middle and apical regions of the lungs are mainly interested. Really, the "blood shift" occurs already with the simple immersion in water up to the neck, resulting in external compression and reduction of the vital capacity. In addition, when the depth obtained is at the level of 30 meters, it could be postulated that the vital capacity approximates the residual volume and that the increase in central blood volume be about 700ml [1,2]. This accumulation of blood in the thorax appears to be a physiological mechanism that compensates for the reduction in the volume of pulmonary air, preventing the implosion of the rib cage over the residual lung volume. This is usually reached at a depth of about 30-40 meters. On the one hand, this reactive preserving mechanism allows apneists to dive at much greater depths (current depth reached in apnoea state is greater than 200 meters). It also involves an important stimulus to the loading of the pulmonary capillary up to their possible and potential limit of breakage, resulting in oedema and alveolar hemorrhage (capillary stress failure) [3,4]. In normal physiological condition, as that of a orthostatic position, the pulmonary capillary pressure has a value of approximately 2,26 KPa (17mmHg) at the bases of the lungs and 1,33 KPa (10mmHg) on average, due to the action of gravity. In immersion, it is estimated that there could be an increase over 10mmHg of pulmonary transmural pressure at the level of pulmonary arteries [5]. The critical value of pulmonary capillary pressure, in the event of rupture of the endothelial wall, is estimated at 5,33 KPa (40mmHg) [3]. This fact becomes a condition of "shear stress" on the capillary wall, rupture of epithelial layer, progressive increasing thinning of endothelium, destruction of the basal membrane. The final consequence could be the rupture of the endothelium, with leakage of blood and proteinaceous material in the interstitial and alveolar spaces [6,7,23]. The increased pulmonary capillary pressure, in addition to the effect of the hydrostatic pressure, can also be due to a possible state of acute and protracted hypoxia-induced deep immersion, similarly to what happens in high altitude [8]. This results in an immediate inflammatory response, mediated by oxygen sensors deployed along the pulmonary vessels, increasing the sensitivity to the decreased partial pressures of oxygen, at the alveolar level. The impaired endothelial function is followed by the increase of vascular permeability, most likely due to reactivity to the oxygen mediated factors [8].

More recently, few authors have studied individual susceptibility to immersion pulmonary edema and they concluded that subjects susceptible to pulmonary edema may have lower lung density, significant lower mass and fewer interlobular septa than subjects resistant [9]. It has been questioned whether the extensive breath hold diving could impose high pulmonary stress by performing voluntary lung hyperinflation maneuvers,

hyperinflating lung up to 50% of total lung capacity. Some authors concluded that the glossopharyngeal insufflation does not permanently impair pulmonary distensibility nor ventilatory flows and volumes. This observation need more extensive evaluation, since it is possible that who performs voluntary hyperinflation, in the course of his life may be at risk to develop pulmonart emphysema. Anyway this type of breath hold preparation may be responsible of a blunted response to elevated CO₂ concentrations, thus suggesting a training effect of CO₂ tolerance due to breath hold rather than being an inherited phenomenon [10]. More recently, recommendations, such as limiting the number of maneuvers and performing lung packing, should be evoked to avoid injuries secondary to use of glossopharyngeal insufflation [11].

A study of pulmonary imaging (MRI) has finally shown uneven distribution of pulmonary perfusion, with areas of greater blood supply more susceptible to the stress of the capillary wall and pulmonary edema [12]. This phenomenon is also relevant for the deterioration of lung function. Redistribution of uneven pulmonary blood may impair normal relationship, i.e. ventilation-perfusion (V_A/Q) ratio, causing a shunted blood in low ventilated areas of the lungs resulting in arterial hypoxemia.

Many reports are present in the literature about pulmonary oedema in swimmers and divers with breathing apparatus, as well as cases about episodes of haemoptysis and alveolar haemorrhage occurred during spearfishing [13]. On the female triathletes pulmonary edema was triggered by cold water immersion and exertion [14]. Another intriguing hypothesis raised from a European group is that oxidative stress in diving after repetitive dives may account for the potential damage to lungs [15]. Since hyperoxia can elicit oxidative stress, being breath-hold diving transiently associated with hyperoxia followed by hypoxia and build-up carbon dioxide, chest wall compression and significant hemodynamic changes, it has been demonstrated a circulating increase in nitric oxide after repetitive dives. Consequently, interaction of hypoxic induced superoxide anions and nitric oxide may reduce the production of protective thiols [15].

Recently it has been observed in elite apnoeists divers who can overcome the depth of 30 meters, the onset of significant signs and symptoms which resolve within 24-48 hours after surfacing. The symptoms may include profuse asthenia, cough, metallic ionic taste and chest tightness. However when there is frank hemoptysis, this is usually indicative of pulmonary capillary stress beyond the point of respiratory compromise. Imaging studies have highlighted aspects in such circumstances where parenchymal areas are covered by cottony floccoses plugs, typical of involvement of alveolar bleeding proteic material [16]. Although the pathophysiological phenomena described are completely reversible at the end of the acute phase, the functional long term sequelae cannot be excluded with the acute repeated stresses of hypoxia and pressure over time.

In addition to haemodynamic effects described above, the underwater apnoea practice also involves variations of the relationships between respiratory gases, such as oxygen and carbon dioxide, in blood and the alveolar level [17]. By the law of Dalton it is known that there is an increase in hydrostatic pressure

during apnoeic dive, resulting in an increase of the alveolar pressure of respiratory gases. This is associated with a decrease in blood CO₂ contents, maintenance of high levels of alveolar pO₂, despite continuous oxygen consumption. It is only during the ascent that the decreasing partial alveolar oxygen pressure and the rapid arterial oxygen depletion, along with progressive decrease of hydrostatic pressure, dramatically increase the risk of loss of consciousness [18].

From the literature it is known that a residence time in apnea for almost twenty seconds at a depth of 10 meters is more likely to increase the alveolar pressure of CO₂ and O₂ to values of 5, 6 KPa and 19,6 KPa respectively (42 and 147 mmHg). By prolonging the breath hold time, a reduction of the alveolar pO₂ to 3,99 KPa (30 mmHg) is observed after 5 minutes [19]. It also true that specific adjustments in the course of repeated extreme apnea-dives are not fully understood, whereas it is well described a modified ventilatory response to exercise in breath hold divers in a such way that elite breath hold divers present signs of a metabolic shift from aerobic to anaerobic energy supply, decreased chemosensitivity during exercise and a distinct ventilatory-response pattern during exercise that differs scuba divers and controls [20].

Recent studies have evaluated the alveolar-capillary distress and the changes in pulmonary capillary blood volume resulting from breath-hold freediving to a depth of 10 or 30 meters. This has been done, simply, by measuring the lung diffusion through alveolar-capillary membrane of the lung with carbon monoxide (CO) [16]. The authors found an increase of the diffusion of CO immediately after diving up to 30 meters: this result has been interpreted as consequence of the persistence of the "blood shift" in a such way that the increase of the pulmonary capillary blood inside the capillaries may be captured by the CO pathway. It turns out that to have more red blood cells windowing by one side of the alveolar capillary membrane may be the signal that the subject is at risk to develop a frank hemorrhage during diving in breath-holding at 30 meters depth. Finally, another interesting result was to find values of lung diffusion, measured almost thirty minutes after diving, lower than those obtained before diving: this feature may suggest the occurrence in initial damage at the level of alveolar capillary membrane (interstitial edema).

It could be hypothesized, in turn, that the process of pulmonary edema may transit from a very early phase, where red blood cells are going to window from the blood side of the alveolar capillary membrane, to a late phase where the dramatic effect in endothelial rupture with the consequence of pulmonary edema and pulmonary hemorrhage occur [3, 23]. Further experiments, applying DLNO/DLCO breathing mixture, before and after apnoeic diving conditions showed that repeated hypoxic stimuli in depth may increase the CO pathway [21] and this could be another element in the intriguing scenario of pulmonary edema during breath-hold diving.

CONCLUSIONS

It turns out from these studies that either physicians, recreational and professional divers should be very well informed and concern about the physical and physiological changes of breath-hold diving and the associated risks to health [22].

Particularly, the physicians proposed to declare a person able to dive in apnea should perform accurate evaluation in terms of pulmonary gas exchange functions.

In conclusion, the effects of lung immersion during apnea are several in terms of gas exchange and mechanics although many advances have been made in these last 10 years. Hopefully in next future others will be addressed. One of this entails the study of the alveolar-capillary diffusion process which allowed to outline the probable mechanism supporting the ventilation-perfusion mismatch and the damage of the alveolar-capillaries during freediving, at different depths. Going away from these observations, it would be interesting to think at the the study of lung diffusion in the functional pulmonary work-up usually followed, just to see the basal integrity of alveolar capillary membranes when a person who wish to dive needs to be licensed.

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