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Arterial Blood Gases During Diving in Elite Apnea Divers

Abstract

Elite apnea divers have considerably extended the limits of dive depth and duration but the mechanisms allowing humans to tolerate the compression- and decompression-induced changes in alveolar gas partial pressures are still not fully understood. Therefore we measured arterial blood gas tensions and acid-base-status in two elite apnea divers during simulated wet dives lasting 3 : 55 and 5 : 05 minutes, respectively. Arterial pO_2 followed the compression- (from 13.8/16.9 kPa before the dive to 30 kPa at the start of the bottom time) and decompression-induced (from 13.7/21.0 kPa to 3.3/4.9 kPa immediately after surfacing) variations of ambient pressure, while the arterial pCO_2 remained within the physiologic range (3.0/3.9 kPa before diving vs. 5.7/5.9 kPa at the end of the bottom time), probably due to the CO_2 storage capacity of the blood. These findings may help to explain why humans can sustain deep and long apnea dives without major increases in respiratory drive.

Key words
Buccal pumping · alveolar gas exchange · breath-holding

Introduction

During the last decade, elite apnea divers have extended the limits of dive duration to 5 – 8 min during "static apnea" and dive depth to 81 and 152 msw for unassisted dives with constant weight and assisted dives, respectively [1]. This development renewed the interest in the physiology of extreme apnea diving, prompting a number of studies on circulatory changes and alveolar gas composition as well as energetics of deep breath hold diving [6]. In particular, interest focussed on the regulation of tissue blood flow and metabolism as well as breath-hold induced bradycardia [7]. Furthermore, studies have been undertaken on alveolar gas partial pressures in apnea divers, both during simulated as well as free diving [3 – 5, 8 – 12, 14, 15, 19, 20], but up to now data on arterial blood gas partial pressures are only available from short-term dives to shallow depths [14, 18]. Data obtained from short breath-hold times, however, may not necessarily predict results during extreme breath-hold diving. According to Boyle's and Dalton's law, the increase of ambient pressure during descent affects the partial pressures of the respiratory gases. Consequently, hyperoxia and pronounced hypercarbia should theoretically occur during the descent and the bottom time, while profound hypoxia may be expected after surfacing. Applying Boyle's law alone, however, does not account for the effects on the arterial blood gas tensions, in particular pCO_2, of the increase in pulmonary blood flow which occurs during compression due to the reduction in intrapulmonary gas volume. In fact, Linér et al. [10] demonstrated that during dry apnea dives to a simulated depth of 20 msw alveolar pCO_2 only reached 6.7 kPa although the compression to 3 atm of ambient pressure would have raised the alveolar CO_2 tension to about 13.3 kPa [10]. Therefore we measured arterial blood gas partial pressures and acid-base-status in two elite apnea divers during simulated dives in the wet tank of a hyperbaric chamber.

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Table **1** Anthropometric data and personal records of the elite divers

Diver	*Age* [years]	*Height/weight* [cm]/[kg]	*TLC* without/with buccal pumping* [l]	*RV* [l]	*Personal records depth/breath-hold time* [m]/[min]
H. M.	24	190/86	10.0/13.0	1.62	60/7:02
S. P.	30	182/67	8.0/11.9	1.85	50/6:04

*Note the increase in TLC after the *"buccal pumping"* manoeuvre.

Material and Methods

Subjects

After approval by the ethical committee of the Ulm University Medical School and detailed explanation, two members of the German apnea national team gave their written informed consent to volunteer for the investigation, which was conducted according to the principles of the "Declaration of Helsinki". The two well-trained divers were in good physical condition as demonstrated by their routine training performance with daily apnea dives to more than 30 msw and breath-hold times of more than 3 – 4 minutes. The anthropometric characteristics and their personal records are listed in Table **1**.

Prior to the dives lung functions were measured in a bodyplethysmograph (Master Screen Body, Jaeger, Hoechberg, Germany) estimating the subjects total lung capacity (TLC), vital capacity (VC) and residual volume (RV) with and without a breathing technique known as "buccal pumping". The technique of "buccal pumping", which is fairly common among these athletes allows to deliberately overinflate the lungs [11,19].

Arterial cannulation and blood sampling

A radial artery cannula was placed to collect arterial blood. Before insertion, the patency of the radial and ulnar arteries of the non-dominant arm was confirmed using the Allen test. After topical local anesthesia with Emla® and local infiltration anesthesia with 1 % lidocaine, the radial artery hand was cannulated percutaneously under aseptic conditions using a modified Seldinger technique and a 18-gauge Teflon catheter. The catheter was attached to a short length of tubing and flushed intermittently with heparinized saline from a 20 ml syringe connected to the end of the tubing. The catheter was secured with a self-adhesive cannula fixation dressing. An additional occlusive dressing, which gently encompassed the forearm and prevented water from getting to the skin, was placed over the insertion site. The arterial cannulation sites were monitored for two days after the study. Except for a small hematoma in one of the subjects, no complications from the use of arterial cannulas were noted.

During the study blood samples were withdrawn using a system of disposable three-way stopcocks and heparinized syringes. At least 5 ml of blood were first withdrawn into a 20 ml discard syringe to clear the dead space of the catheter and tubing consisting of a series of three-way stopcocks. Then a blood sample was withdrawn into a heparinized 2 ml syringe mounted on one stopcock. After each sample the dead space of the arterial cannula and the tubing was flushed with physiologic saline from another syringe mounted on a stopcock. Blood sampling underwater had been practised before using a dummy, and therefore the time lag between the start of the dead space aspiration and the actual blood sampling was always <15 seconds.

The dives were performed in the wet tank (water temperature 24 – 26 °C) of the pressure chamber of the German life guard association (DLRG, Deutsche Lebens-Rettungs-Gesellschaft, Berlin, Germany) and consisted of exposures to an ambient pressure of 3 atm. For this purpose each subject dove to the bottom of the wet tank, while the hyperbaric chamber was simultaneously pressurised to 2.2 atm to yield a simulated depth of 20 msw. After two "warm up" dives each individual volunteer performed one single dive with blood sampling. Prior to this dive the subjects hyperventilated for one minute according to their usual daily training routine and overinflated their lungs using the "buccal pumping" manoeuver [7,13]. Descent and ascent times were 1min each with a linear pressure increase over time, bottom times were 2 and 3 min, respectively, depending on the diver's individual breath-hold capacity, resulting in total dive times of 3 : 51 and 5 : 05 min, respectively.

Arterial blood samples were taken prior to the dive before preparation for breath-hold, during head-out immersion immediately before diving, at the beginning of the bottom time, during continued breath-holding at the end of the bottom time immediately before ascent, right after surfacing just before breath hold was broken, and 10 and 30 minutes after the dive when the subjects rested in a sitting position. Blood gas analyses were performed immediately (those taken during the dive immediately after surfacing) using an automated self-calibrating blood gas analyzer (ABL 725, Radiometer, Copenhagen, Denmark). Since only one single dive in just two subjects could be investigated, a statistical analysis was not performed.

Results

The buccal pumping manoeuvre allowed the two divers to increase their TLC by 3.0 and 3.9 litres, respectively (Table **1**). The arterial pO_2 values before diving were 16.9 and 13.8 kPa. At the beginning of the bottom time arterial pO_2 had increased to 30.1 and 30.0 kPa and subsequently fell to 21.0 and 13.7 kPa, respectively, at the end of the bottom time. The lowest values of 4.9 and 3.3 kPa were seen after surfacing immediately before breath-hold was broken. Due to moderate hyperventilation the arterial pCO_2 values immediately before the dive were 3.0 and 3.9 kPa and increased during ascent to 4.6 and 5.8 kPa, respec-

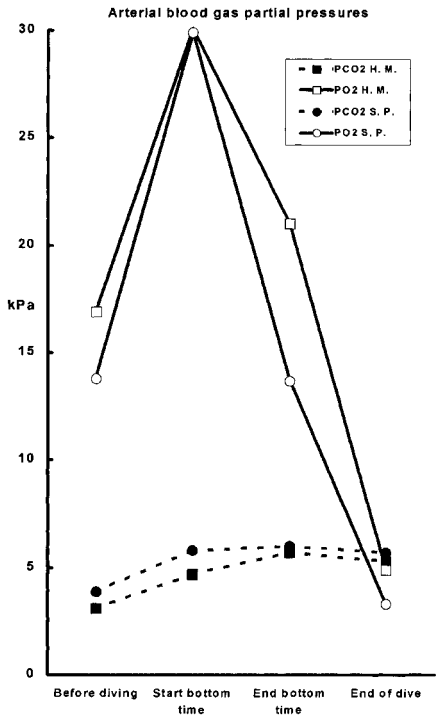

Fig. **1** Arterial blood gas partial pressures in two elite apnea divers during a breath-hold dive to 20 msw. Measured pO_2- and pCO_2-values are represented by closed and open symbols, respectively, connected by closed or dashed lines. The pO_2-data show a marked compression-induced increase and decompression-related decrease during the dive. In contrast, the changes in pCO_2 are moderate and far less affected by variations in ambient pressure.

tively, reaching the highest values (5.7 and 5.9 kPa, respectively) at the end of the bottom time. Immediately before breath-hold was broken the arterial pCO_2 was 5.3 and 5.7 kPa (Fig. **1**). As shown in Fig. **1**, the results of the model calculations approach the measured PO_2 and PCO_2-values. Because of the decreased arterial pCO_2 the arterial pH prior to the dive was alkalotic (7.53 and 7.47) and decreased to the normal physiologic range (7.39 and 7.38) while neither standard bicarbonate nor base excess were affected (data not shown).

Discussion

The present study was conducted to characterize the kinetics of arterial blood gas tensions during simulated wet apnea dives to 20 msw in an 8 m deep tank of a hyperbaric chamber. The key findings were that 1) the arterial pO_2 values showed a substantial pressure-induced increase at depth and a decompression-induced decrease during ascent, while 2) the arterial pCO_2 remained within the normal physiologic range.

The high arterial pO_2 values at depth as well as the rapid fall of the arterial pO_2 at the end of the dive, the latter resulting from the ascent-induced decrease of ambient pressure together with the oxygen consumption over a prolonged period of time, are in good agreement with the results obtained during previous investigations on apnea dives to shallower depths [14,17]. By contrast, at first glance the only moderate increase in arterial pCO_2 seems to contradict the conjecture that the descent-induced compression of alveolar gas should result in pronounced hypercarbia and CO_2 retention during deep and prolonged apnea diving [3,4,11,15,19]. It should be noted, however, that measurements of blood gas tensions both during short-term dives to shallow depths in the professional Korean apnea divers [18] as well as in aquatic mammals (free diving Weddell seals) [17], agree with our observations: arterial pCO_2 values did not increase to the levels predicted from the depth-induced increase in alveolar pressure in these studies either. Our results on arterial pO_2 and pCO_2 suggest that a theoretical estimation solely based on Boyle's law, which predicts linear in- or decrease of alveolar partial pressures for the respiratory gases as a result of compression and decompression, respectively, does not sufficiently explain the time course of arterial blood gases during apnea dives, the resulting predictions being particularly erroneous in the case of the pCO_2. To satisfactorily explain the measured data, the blood solubilities of the respiratory gases together with the diving-related increased pulmonary blood flow have to be considered as well. In fact, Linér et al. demonstrated that during dry apnea dives to a simulated depth of 20 msw alveolar pCO_2 only reached 6.7 kPa although the compression to 3 atm of ambient pressure would have raised the alveolar CO_2 tension to about 13.3 kPa [10]. This reasoning is supported by the concept of separate blood/lung (R_{BL}) and metabolic (R_{met}) gas exchange [16] demonstrating that during steady state changing the ventilation causes opposite changes of arterial pCO_2 and pO_2 of similar magnitude [$\Delta\, pCO_2 \approx R_{met} \times \Delta\, pCO_2$]. Apnea as an extreme form of hypoventilation ($R_{BL} < R_{met}$) will therefore result in a much higher O_2 uptake from the lung than CO_2 output into the lung. Thus, a substantial rise in arterial pCO_2 is prevented by the large amount of this highly soluble gas taken up by the blood with increasing ambient pressure, which thereby blunts the compression effect on the blood gas tension. By contrast, the blood solubility of O_2 is much less than that of CO_2. Therefore, a pronounced increase in arterial pO_2 can be expected during compression such as demonstrated by our investigation. Moreover, the compression-related rise in intrathoracic blood volume [7] affiliated with increased pulmonary blood further contributes to the buffering effect of gas uptake on pCO_2. Finally, during ascent an increase in arterial pCO_2 is blunted by the Haldane effect of the reduced hemoglobin oxygen saturation on the CO_2-dissociation curve.

The observed increase in vital capacity due to the buccal pumping manoeuver increases the oxygen store of the body and thus may help to extend the apnea time. On the other hands according to the assumption that maximum diving depth is where total lung capacity (TLC) is reduced to the residual volume (RV), this manoeuver may assume importance for the maximum reachable diving depth, as an increase in TLC will affect the TLC/RV ratio which would result (even according to this simplified hypothesis) in a deeper diving depth [20]. The increase in vital capacity which can be reached after a deep inspiration by this voluntarily overinflation was reported earlier as 22 – 39 % [7,13]. These data are well in line with the findings in this study.

Conclusion

This study confirms the prediction derived from measurements of alveolar gas partial pressures during simulated diving that during breath-hold diving a compression-induced increase in arterial pCO_2 is blunted to a large extent by the CO_2 capacity of the blood volume and the CO_2 storage in the rapidly perfused tissues while arterial pO_2 increases in parallel to the rise in ambient pressure. Consequently, deep- and long-term apnea dives do not lead to a major increase in CO_2-induced respiratory drive.

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