Alveolar gas exchange during simulated breath-hold diving to 20 m

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Linér MH, Ferrigno M, Lundgren CEG. Alveolar gas exchange during simulated breath-hold diving to 20 m. Undersea & Hyperbaric Med 1993; 20(1):27-38.—Alveolar gas exchange, as affected by changes in pulmonary blood flow, was studied in five subjects performing breath holds lasting 75 s at the surface and during compression to 20 m in a hyperbaric chamber. After reaching the maximal depth, \( V_O_2 \) started to increase, compared to control, reaching a maximum of \( 346 \pm 66 \) (se) ml (STPD) \( \cdot \) min\(^{-1} \cdot m^2 \) (body surface area) at 50 s, i.e., early in the ascent; it exceeded the 50-s surface breath-hold value by \( 214 \pm 9 \) ml \( \cdot \) min\(^{-1} \cdot m^2 \). During descent, \( C_O_2 \) was absorbed from the alveoli into the blood, initially at \( 140 \pm 24 \) ml \( \cdot \) min\(^{-1} \cdot m^2 \); during ascent \( C_O_2 \) was transferred back into the lungs. These changes reflected compression and expansion of lung air. The increase in \( V_O_2 \) during the dives, which are not steady states, may be explained by an increasing cardiac output at depth. An augmented cardiac output had earlier been observed under identical conditions and explained by a drop in transthoracic pressure, enhancing venous return. Upon surfacing, the \( P_A_O_2 \) was about 20 mmHg lower than after surface breath holds, reflecting the effects of changes in cardiac output.

pulmonary circulation, apnea, respiration, hypoxia, immersion, cardiac output, oxygen stores

Alveolar gas exchange in humans during breath-hold diving has been studied by several authors. However, with the exception of one investigation done in a dry hyperbaric chamber (1), most of these studies were performed at sea (2-5) or in submarine escape training towers (6, 7), with few subjects and under conditions making difficult the control of relevant factors such as depth-time profile, water temperature, and degree of physical exertion. In particular, water temperature may play a role in the so-called dive response, influencing the degree of bradycardia (e.g., 8, 9) and peripheral vasoconstriction (e.g., 10). Moreover, earlier analyses of pulmonary gas exchange in the course of breath-hold dives have lacked information about concurrent hemodynamic changes that might have influenced experimental results. For example, the rebreathing technique for alveolar gas sampling throughout a breath hold (cf 1) may alter cardiac output (11), and this might also affect alveolar gas exchange. In the absence of ventilation, pulmonary gas concentrations become particularly dependent on lung blood flow and mixed venous gas concentrations.
Submersion is known to redistribute blood into the thorax, thereby increasing venous return and consequently cardiac output (e.g., 12, 13). Breath-hold diving has also been shown to influence cardiac output. Cardiac performance during breath holding at the surface and during simulated diving to 20 m in a hyperbaric chamber has recently been investigated in this laboratory (14, 15). The experiments were performed in a thermonutral environment with resting subjects, in both the non-submersed (dry) and the submersed (wet) condition. Breath holding at a large lung volume decreased cardiac index (CI), probably due to the increase in intrathoracic pressure (14) which may have impeded venous return. Conversely, CI at depth was increased, compared to breath holding at the surface (15). This was apparently due to a fall in intrathoracic pressure (relative to ambient pressure) during diving, which would be likely to restore venous return. The combination of the opposite effects of breath holding and compression to 20 m left cardiac performance unchanged at depth, compared to the eupneic surface control.

In light of the hemodynamic changes just described, the present study was designed to correlate alveolar gas exchange with the changes in cardiac output caused by breath-hold diving and to isolate the effects of breath holding, diving (compression), and submersion. Experiments were performed in both the dry and the wet condition, in a thermonatical environment (to minimize temperature-dependent circulatory changes), and with resting subjects. Exertion was avoided to mimic the conditions of deep breath-hold diving in which the divers descend and ascend pulled by mechanical devices (16). The CI values, obtained about 1 yr earlier under identical experimental conditions in the same subjects, were employed in the interpretation of gas exchange in the present experiments.

METHODS

The subjects were five healthy male nonsmokers. They gave their informed consent as required by the Institutional Review Board for Human Experimentation. Their mean age was $24 \pm 3.5$ (SD) yr, height $179.8 \pm 7.0$ cm, weight $76.4 \pm 9.8$ kg, body surface area $1.95 \pm 0.13$ m$^2$, vital capacity (VC) $5.80 \pm 0.69$ liter, and residual volume (RV) $1.46 \pm 0.22$ liter. They were certified scuba divers.

Four series of experiments were performed: breath holds at the surface and breath-hold dives, simulated in a hyperbaric chamber; these experiments were made both in the dry and the wet condition. In the dry experiments the subjects, wearing swim trunks, sat upright; wet experiments were initiated, while sitting in water to the chin, by gently tilting the head under the surface. Water temperature was kept at $35.0^\circ \pm 0.4^\circ$C, which is thermonutral for the naked resting human (17). During the dry dives, the subjects were protected by a light bathrobe against short-lasting temperature changes induced by pressure changes. The temperature underneath the bathrobe was always perceived as comfortable.

At the end of each breath hold or breath-hold dive, sampling of expired gas for analysis by mass spectrometer (model MGA 1100, Perkin Elmer Corp., Pomona, CA) was performed at the mouth, with a probe inserted in a mouthpiece. In the dive experiments, the rate of the sample flow was set by a needle valve close to the mouthpiece, and the sample was passed out through a tube penetrating the chamber wall and to an outside flowmeter, as described in detail elsewhere (18). Before each
sampling, the mass spectrometer was calibrated against standard mixtures to ensure an accuracy of ± 0.05%. The concentrations of oxygen and carbon dioxide in the expired gas were recorded on a strip-chart recorder, as well as digitally sampled. The partial pressures of expired gases were derived from their concentrations and the ambient pressure. Correction factors for alveolar gas pressure reflecting chest–lung recoil were obtained for different lung volumes from standard pressure–volume curves for the dry and the wet condition (19, 20). Lung volumes were computed using the principle of conservation of the mass of nitrogen. Specifically, for the surface breath holds, the lung volume at the end of the breath hold was calculated using the nitrogen concentration (at the end of breath hold) and the initial nitrogen volume, derived from the known starting lung volume and nitrogen concentration. For the dives, the initial nitrogen volume was reduced by the amount of nitrogen estimated to have been taken up by the blood. This amount was derived from changes in nitrogen pressure related to ambient pressure, cardiac output values in identical dives (15), and nitrogen solubility in the blood. Typically, in the longest lasting dives with a duration of 75 s, the nitrogen uptake was computed to average about 100 ml. For calculations of partial pressures and volumes of oxygen and carbon dioxide in the lungs, the lowest concentration of the former and the highest concentration of the latter at end exhalation were used.

The rates of \( \text{O}_2 \) and \( \text{CO}_2 \) exchange (\( \dot{V}_{\text{O}_2}, \dot{V}_{\text{CO}_2} \)) were calculated from the differences in the volumes of \( \text{O}_2 \) and \( \text{CO}_2 \) in the lungs at two consecutive sampling times. The resulting \( \dot{V}_{\text{O}_2} \), or \( \dot{V}_{\text{CO}_2} \) was arbitrarily assigned a time that was halfway between the two sampling times used. For example, \( \dot{V}_{\text{O}_2} \) calculated between the sampling times 15 and 25 s was assigned the time 20 s.

A rolling-seal spirometer was used for VC determinations, and RV measurements were made with a \( \text{N}_2 \)-dilution technique (21). Oxygen uptake and \( \text{CO}_2 \) elimination during breathing at rest were determined from expired gas composition and volume. Esophageal pressure, in both the dry and the wet mode, was obtained with an esophageal balloon, referenced, in the latter condition, to a point 14 cm below the sternal notch according to a technique described earlier (15). Esophageal pressure recordings were made to ensure that the subjects were relaxing their respiratory muscles during breath holding, and were used, during the dives, as a guide for the prevention of thoracic squeeze (15).

Measurements were obtained after dry and wet breath holds and breath-hold dives lasting 0, 15, 25 (wet only, for technical reasons), 35, 45 (wet only), 55, and 75 s. Comparisons of \( \text{O}_2 \) and \( \text{CO}_2 \) partial pressures at end exhalation in the dry and wet conditions were made by means of two-way analysis of variance (ANOVA). One factor was the combination of time with either dry or wet breath holds and breath-hold dives; the other factor was the difference between subjects. The data from one subject were excluded from this analysis because his expired volumes at 20 m in the dry condition were below predicted anatomic dead space volume. No differences were found among \( \text{PO}_2 \) values and \( \text{PCO}_2 \) values in the dry and in the wet condition, respectively. Further statistical evaluation of the results was made with another two-way ANOVA, without interaction. In this analysis, one factor consisted of a combination of time with either breath holding at the surface or breath-hold diving, whereas the other factor was the difference between subjects. Specific pair-wise comparisons were tested using statistical contrasts, i.e., the difference between two means, and their levels of significance were not adjusted for multiple comparisons.
PROCEDURES

The subjects were encouraged to breathe normally, avoiding hyperventilation between breath holds, and to relax their respiratory muscles during breath holds. In preparation for each breath hold, the subject, using a noseclip and a mouthpiece, exhaled to RV and inhaled, from a rubber bag, a volume of fresh air corresponding to 80% of his VC (measured in dry conditions). The subject then held his breath for a predetermined time, either at the surface or while being compressed in the hyperbaric chamber. Breath-hold experiments were performed in triplicate by each subject. The first three breath holds were not included in the results because of possible physiologic changes related to the so-called short-term training effect on breath-hold duration (22). Each breath hold was followed by a 5-min rest period.

Gas samples were taken at the end of breath holds or breath-hold dives of different duration, as illustrated in Fig. 1. To complete the dive profile, seven breath-hold experiments were required. To reduce bias from order effects the sequence of the breath holds or breath-hold dives was varied.

RESULTS

All data reported are means ± SE of the means of three experiments in each of the five subjects, and differences noted below were all significant at the $P < 0.01$ level. Furthermore, only data from the submersed experiments are shown because they were not statistically different from results obtained in the dry condition.

![Diagram](image)

**FIG. 1**—Timing (horizontal axis) of alveolar gas sampling events (circles); each circle represents the end of a surface breath hold or a breath-hold dive (depth on vertical axis).
ALVEOLAR GAS IN SIMULATED BREATH-HOLD DIVING

The partial pressures of O₂ (P_{AO₂}) and CO₂ (P_{ACO₂}) in end-expiratory air are shown in Fig. 2. At the surface, the P_{AO₂} slowly decreased from 130.0 ± 3.0 mmHg at the beginning of breath holding to 88.7 ± 4.0 mmHg at 75 s. During breath-hold diving, the P_{AO₂} increased from 127.1 ± 3.1 mmHg at 0 s, before leaving the surface, to 339.6 ± 7.8 mmHg at 25 s on arrival at 20 m. At the bottom the P_{AO₂} slowly decreased to 296.6 ± 11.8 mmHg at 45 s, immediately before starting the ascent. At 75 s, on arrival at the surface, the P_{AO₂} had dropped precipitously to 67.0 ± 6.3 mmHg, 21.7 mmHg lower than the P_{AO₂} at 75 s of breath holding at the surface. All the P_{AO₂} values during breath-hold diving, except for the 0 s value, were significantly different from the corresponding surface values. The P_{ACO₂} during breath holding at the surface first increased rapidly from 30.2 ± 2.8 mmHg at 0 s to 38.2 ± 2.5 mmHg at 15 s, then more slowly to 46.5 ± 1.9 mmHg at 75 s. In the course of breath-hold diving, the P_{ACO₂} rose from 31.5 ± 2.6 mmHg at 0 s to 47.8 ± 1.9 mmHg at 15 s at 10 m; then to 52.0 ± 2.0 mmHg at 25 s on arrival at 20 m. While at the bottom, the P_{ACO₂} remained unchanged, at 35 s being 10.0 mmHg higher than the corresponding surface value. Finally, during ascent the P_{ACO₂} decreased first to 46.8 ± 1.8 mmHg at 55 s when the subjects had reached 10 m, then, more slowly, to 44.2 ± 2.2 mmHg at 75 s on arrival at the surface. The surface values were consistently lower (by an average of 2.3 mmHg) than the corresponding values of breath holding at the surface. All other P_{ACO₂} values during breath-hold diving, except for the 0 s value, were higher than the corresponding surface values.

![Graph](image-url)

**FIG. 2—**Oxygen and carbon dioxide pressures in alveolar gas samples vs. time during wet surface breath holds and wet breath-hold dives. In this and the following figures, data points are means ± SE of measurements, in triplicate, in each of five subjects.
The volumes of \( \text{O}_2 \) and \( \text{CO}_2 \) in the lungs are shown in Fig. 3. In the following, changes in gas volumes are expressed in milliliters STPD and reflect transfer of gas molecules from the alveoli to the lung tissue and blood, or vice versa. The total \( \text{VO}_2 \) during breath-hold diving was 494 ml (395.2 ml/min), which was 161 ml larger than during breath holding at the surface (333 ml, 266.4 ml/min). The total \( \text{VO}_2 \) at the surface was very similar to the one expected based on control \( \text{VO}_2 \) (333 ml measured vs. 348 ml calculated). The \( \text{CO}_2 \) content in the lungs decreased during the descent phase of the breath-hold dives by 115 ml, was unchanged at the bottom, and increased during ascent, resulting in a net increase of 61 ml. This was 45 ml less than the increase in \( \text{CO}_2 \) volume during surface breath holds.

The slopes of the lines in Fig. 3 are proportional to the rates of \( \text{O}_2 \) and \( \text{CO}_2 \) exchange. These rates, corrected for body surface area, are shown in Fig. 4. For the purpose of interpretation of the current data, \( \text{Cl} \) values obtained in earlier studies on the same subjects, under identical breath holding and breath-hold diving conditions (14, 15), are also shown. The \( \text{VO}_2 \) and \( \text{VCO}_2 \) are shown in Fig. 4 as positive values for gas transfer from the alveoli, and as negative values for gas transfer to the alveoli. The control, pre-breath hold, \( \text{VO}_2 \) was 143 ± 4 ml (STPD) \text{min}^{-1} \text{m}^{-2} \) of body surface area, and during breath holding at the surface it was 180 ± 15 ml \text{min}^{-1} \text{m}^{-2} \) at 7.5 s and 126 ± 16 ml \text{min}^{-1} \text{m}^{-2} \) at 65 s, these values not being significantly different from each other or from the control value. During the early phase of breath-hold diving, \( \text{VO}_2 \) did not change significantly, being 152 ml \text{min}^{-1} \text{m}^{-2} \) at 20 m, 30 s into the dive; at 40 s, while still at 20 m, an increase in \( \text{VO}_2 \) started which reached its maximum of 346 ± 66 ml \text{min}^{-1} \text{m}^{-2} \) at 50 s, early during ascent; in the late phase of ascent, at 65 s, the \( \text{VO}_2 \) decreased to 140 ± 29 ml \text{min}^{-1} \text{m}^{-2} \).

**FIG. 3**—Volumes of oxygen and carbon dioxide in the lungs during wet surface breath holds and wet breath-hold dives vs. time.
ALVEOLAR GAS IN SIMULATED BREATH-HOLD DIVING

The peak in $\dot{V}O_2$ observed at 50 s during the breath-hold dives exceeded the 50-s value at the surface by 214 ml · min$^{-1}$ · m$^{-2}$.

In the eupneic control situation, the $\dot{V}CO_2$ was 127 ± 9 ml · min$^{-1}$ · m$^{-2}$ and the initial value at 7.5 s during the surface breath holds was about the same, at 119 ± 12 ml · min$^{-1}$ · m$^{-2}$; it then decreased so as to become negligible from 30 s and throughout the remainder of the breath holds. In the breath-hold dives, during the first 20 s, $CO_2$ left the alveoli at a rate of about 140 ml · min$^{-1}$ · m$^{-2}$; whereas at 20 m there was no net $CO_2$ exchange; finally, after leaving the bottom, $CO_2$ moved into the alveoli at an accelerating rate reaching 218 ± 13 ml · min$^{-1}$ · m$^{-2}$ at 65 s late in the ascent.

DISCUSSION

The patterns of alveolar gas exchange during breath holding and breath-hold diving in this study are in general agreement with the results of previous investigations (1–7). However, as discussed in the introduction, experimental conditions in those studies varied widely, making comparisons difficult.

Once ventilation has been interrupted and the airway is closed, the body becomes a closed system in terms of gas exchange. Changes in pulmonary blood flow will greatly influence gas exchange in the alveoli, and the rate and distribution of blood flow to various tissues will also affect $\dot{V}O_2$ and $\dot{V}CO_2$. 

FIG. 4—Top, exchange of oxygen from (positive values) or to the lungs (negative values) during wet surface breath holds and wet breath-hold dives vs. time. Bottom, exchange of carbon dioxide during wet surface breath holds and wet breath-hold dives vs. time. Bars show CIs obtained under identical conditions in other studies (14, 15).
Breath holding

Oxygen uptake did not change significantly in the course of breath holding at the surface, even though numerical values showed a decline in four of the five subjects. A falling rate of \( \dot{V}_{O_2} \) was shown by Lanphier and Rahn (23) in breath holding during mild exertion and by Hong et al. (24) during breath holds of up to 240 s; in both studies breath holding was combined with intermittent rebreathing. Exercise in the former study, and longer duration in the latter study, might have made the tendency for \( \dot{V}_{O_2} \) to decline more evident than in our study. Although Lanphier and Rahn (23) assumed a constant cardiac output, they recognized that a decrease in pulmonary blood flow in the course of breath holding might have contributed to the fall in \( \dot{V}_{O_2} \). This hypothesis is supported by the 20–25% decrease in CI measured earlier in our breath-holding subjects (14). The decrease in cardiac output due to breath holding might also help to explain the time course of CO\(_2\) transfer in the alveoli. When normal elimination of CO\(_2\) is blocked, its tension will rise in all body compartments. The rate of rise of the CO\(_2\) tension will depend on the CO\(_2\) production, the buffering capacity of blood and various tissues, and the rate and distribution of blood flow. In in the surface breath-hold experiments, the \( P_{ACO_2} \) increased rapidly in the first 15 s, and then more slowly (Fig. 2). The rate of CO\(_2\) transfer from the blood into the alveoli decreased so as to become negligible by 30 s and throughout the remainder of the breath holds (Fig. 4). No appreciable reversal of CO\(_2\) flow was noted in our experiments at the surface. Similar findings have been reported in the literature. In 1959, Mithoefer (25) described the CO\(_2\)-concentrating effect of the progressive decrease in lung volume caused by O\(_2\) uptake in breath holds after O\(_2\) breathing. A few years later, Lanphier and Rahn (1) pointed out that the Haldane effect will cause a further elevation of alveolar and arterial Pco\(_2\) in the breath-holding subject, once the \( P_{ACO_2} \) equals or exceeds the mixed venous Pco\(_2\) (P\(v_{CO_2}\)). The same authors also gave a partial explanation for the decline with time of the rate of P\(ACO_2\) increase: the decrease in P\(AO_2\) and \( \dot{V}_{O_2} \) in the course of breath holds would reduce both the Haldane effect and the concentrating effect of lung shrinkage (1).

The net effects of hemodynamic changes on CO\(_2\) exchange during breath holding are difficult to predict. Several, at times opposing, factors are involved. For example, a reduced systemic blood flow should tend to increase tissue Pco\(_2\) and hence P\(v_{CO_2}\). On the other hand, it will lead to increased oxygen extraction which, because of the Haldane effect, will tend to decrease P\(v_{CO_2}\). Early in a breath hold a decrease in lung blood flow will decrease the rate of rise in P\(ACO_2\). Once the concentrating effect described earlier elevates the P\(ACO_2\) above the P\(v_{CO_2}\), a slower blood flow will, by itself, tend to accelerate the rise in P\(ACO_2\). Furthermore, any effects of circulatory changes on \( \dot{V}_{CO_2} \) would tend to be attenuated by the large capacity of the body for storing CO\(_2_2\).

A decreased pulmonary blood flow might also explain why \( \dot{V}_{CO_2} \) at the beginning of the breath holds was not different from the \( \dot{V}_{CO_2} \) measured in the eupneic control situation. A faster CO\(_2\) transfer might have been expected in the first few seconds of the breath holds because of the dilutional effect of the large initial inspiration which, in our experiments, produced a P\(ACO_2\) of 30.2 mmHg at 0 s. However, we demonstrated that a reduction in cardiac output was already present in the first few seconds of breath holding (14). Therefore, this might have prevented a rise in \( \dot{V}_{CO_2} \).
ALVEOLAR GAS IN SIMULATED BREATH-HOLD DIVING

Compression

The remarkable differences in alveolar gas exchange during breath-hold diving, compared to breath holding at the surface, have traditionally been ascribed to the effects of changing ambient pressure on the alveolar gas tensions (1–3). Little attention has been paid to the possibility that changes in cardiac output, also related to ambient pressure variations, might affect alveolar gas transfer during the breath-hold dives.

In the present dives, oxygen uptake was the parameter that differed the most from previous studies or theoretical analyses. There were no significant changes in $\dot{V}O_2$ during descent, but after 30 s, $\dot{V}O_2$ started to increase and reached its maximum at 50 s, i.e., during the early phase of ascent. At this time, it exceeded the 50-s value of the surface breath holds by 214 ml $\cdot$ min$^{-1}$ $\cdot$ m$^{-2}$. Later in the ascent, $\dot{V}O_2$ decreased toward the corresponding surface value. As a result of the time course of $\dot{V}O_2$, total $O_2$ uptake during the dives was 161 ml larger than during breath holds at the surface (Fig. 4). Furthermore, enhancement of $\dot{V}O_2$ at depth was reflected by lower $P_{A}O_2$ values at 75 s; at the end of the dives $P_{A}O_2$ had dropped by 67 mmHg, i.e., 21.7 mmHg lower than at the end of the surface breath holds. It has been suggested (1) that the increased $P_{A}O_2$ at depth may augment blood oxygenation. In the present dive experiments, this can only have played a small role in increasing the $\dot{V}O_2$ above that recorded in the surface breath holds. This is so because, judging from the $P_{A}O_2$, the arterial blood would have remained well-saturated throughout the duration of the surface breath holds. Certainly the increase in $P_{A}O_2$ would have caused some transient increase in $\dot{V}O_2$ due to more $O_2$ being loaded in physical solution. If the average $P_{A}O_2$, throughout a breath-hold dive is estimated to have been about 220 mmHg, this would have resulted in an extra $O_2$ load of about 0.35 ml $\cdot$ 100 ml$^{-1}$ blood, for a total additional uptake of only about 25 ml over 75 s (assuming an average pulmonary blood flow of 5.9 liter $\cdot$ min$^{-1}$ (cf 15)).

The difference in total $O_2$ uptake and in time course of $\dot{V}O_2$ between dives and breath holds might be explained by differences in hemodynamic changes. A fall in intrathoracic pressure was probably responsible for the 29.5% higher cardiac output at depth compared to surface breath holds (15), as illustrated in Fig. 4. The resulting increase in pulmonary blood flow was, in all likelihood, partially responsible for the accelerated $\dot{V}O_2$ at the bottom and during the first phase of ascent. It may be calculated that over the duration of a dive, the pulmonary blood flow was higher by about 1.0 liter than during the surface breath holds. This would account for an extra oxygen uptake of 50 ml, given a normal mixed venous oxygen concentration. However, as will be explained in the following, venous oxygen was probably lower than normal.

The increase in $\dot{V}O_2$ by 162%, early during ascent, was considerably larger than the 29.5% increase in cardiac output. This can probably be explained as follows: Early in the breath hold the intrathoracic pressure was high, leading to reductions in cardiac output and peripheral blood flow which may have increased oxygen extraction from the blood; with the reduced and eventually negative intrathoracic pressure during descent, venous blood was redistributed into the chest. The amount of this blood may approach about 1.0 liter (26, cf 12) and since it is likely to be low in $O_2$ content, it would contribute to an increase in $O_2$ uptake from the lung. A similar finding was made by Loeppky and Luft (27) when studying the effect of posture on
oxygen stores of the body. They could explain only about 33% of the increase in blood oxygen stores by the increase in cardiac output when changing from head up to supine position. The rest of the change in the blood oxygen stores was attributed to redistribution of peripheral blood flow and venous blood volume. Furthermore, Mekjavic and Bligh (28) reported a transient increase in \( \dot{V}O_2 \) by about 120% [calculated from fig. 3 in (28)] during the first minutes of head-out immersion in water of 40°C. Corresponding increase in cardiac output was only 41% as reported by Choukroun and Varene (29).

The increase in \( \dot{V}O_2 \) during compression did not occur until the later part of the bottom stay. This can be explained by several factors. The dives were started after inhalation to a large lung volume, which increased intrathoracic pressure as did Valsalva maneuvers occasionally performed during compression for the purpose of clearing the ears. The increased transthoracic pressure probably hindered venous return during descent. The time required for the venous blood to reach and pass through the lungs might have been another factor delaying the rise in \( \dot{V}O_2 \). Later in the ascent, \( \dot{V}O_2 \) fell abruptly. The rapidly decreasing \( PAO_2 \) certainly played a major role in this fall, although a concomitant decrease in pulmonary blood flow probably contributed (Fig. 4) (15). It should also be noted that there may be some error in the time profile of the \( \dot{V}O_2 \) changes because it is (for technical reasons) based on relatively few data points. With the exception of a study by Schaeffer and Carey (7) in which the subjects exercised during the ascent, an increase in \( \dot{V}O_2 \) early in the ascents apparently has not been reported previously. This may be because sampling was not performed frequently enough or significant transients in cardiac output were lacking, either because the dives were shallower and/or exercise and rebreathing were performed (1, 2, 4).

As for \( CO_2 \) exchange in the alveoli during the breath-hold dives, the pattern of changes is in good agreement with the results reported in the literature (1). During descent, breath holding and compression will augment the \( PA_{CO_2} \), eventually reversing the normal direction of \( CO_2 \) exchange between the blood and the alveoli. Early in the descent an increase in cardiac output will increase the rate of rise in \( PA_{CO_2} \), although it will also buffer the increase in \( PA_{CO_2} \) that would be caused by compression of the gas in the lungs. Then, as the \( PA_{CO_2} \) exceeds \( P_{CO_2} \), the increase in cardiac output will slow the rise in \( PA_{CO_2} \). At 3 atm abs there was no \( CO_2 \) exchange, indicating that the \( CO_2 \) pressure of the alveolar air was the same as that of the pulmonary capillary blood. During ascent, the \( PA_{CO_2} \) decreased, normalizing the direction of \( CO_2 \) transport across the alveolar membrane. This exchange was probably enhanced by high venous return and cardiac output early in the ascent. Yet the resulting \( CO_2 \) content of the lung was lower by 45 ml after breath-hold diving than after surface breath holding. Carbon dioxide retention in this situation has also been observed by others (6).

**Submersion**

An interesting finding in this study is the lack of difference in gas exchange between non-submersed and submersed conditions. This is in spite of the blood redistribution (12) and the 30–40% larger CI during submersion compared to non-submersion (14, 15). In contrast to this is the large increase in oxygen uptake early during ascent,
ALVEOLAR GAS IN SIMULATED BREATH-HOLD DIVING

attributed to hemodynamic changes secondary to ambient pressure changes. The difference is that diving was a dynamic situation, whereas submersion was a steady state in our experiments. In a steady state condition, VO₂ is dependent only on metabolic oxygen consumption. In contrast, an increase in cardiac output will cause a transient increase in the O₂ uptake in the lungs (without change of O₂ utilization), increasing the venous oxygen content (30).

In summary, the alveolar exchange of O₂ and CO₂ is greatly influenced by compression during breath-hold diving. This influence apparently depends to a great extent on the increase in venous return, cardiac output, and intrathoracic blood pooling due to shrinkage of the lung and decreased transthoracic pressure, secondary to the compression. In contrast to this is the fact that the higher cardiac output during steady state submersion does not alter gas exchange.

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