Diving response and arterial oxygen saturation during apnea and exercise in breath-hold divers

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Andersson, Johan P. A., Mats H. Linér, Elisabeth Rünöw, and Erika K. A. Schagatay. Diving response and arterial oxygen saturation during apnea and exercise in breath-hold divers. J Appl Physiol 93: 882–886, 2002.—This study addressed the effects of apnea in air and apnea with face immersion in cold water (10°C) on the diving response and arterial oxygen saturation during dynamic exercise. Eight trained breath-hold divers performed steady-state exercise on a cycle ergometer at 100 W. During exercise, each subject performed 30-s apneas in air and 30-s apneas with face immersion. The heart rate and arterial oxygen saturation decreased and blood pressure increased during the apneas. Compared with apneas in air, apneas with face immersion augmented the heart rate reduction from 21 to 33% (P < 0.001) and the blood pressure increase from 34 to 42% (P < 0.05). The reduction in arterial oxygen saturation from eupneic control was 6.8% during apneas in air and 5.2% during apneas with face immersion (P < 0.05). The results indicate that augmentation of the diving response slows down the depletion of the lung oxygen store, possibly associated with a larger reduction in peripheral venous oxygen stores and increased anaerobiosis. This mechanism delays the fall in alveolar and arterial PO2 and, thereby, the development of hypoxia in vital organs. Accordingly, we conclude that the human diving response has an oxygen-conserving effect during exercise.

breath-holding; hypertension; vasoconstriction; oxygen conservation; bradycardia; hypoxia. During exercise, the diving response is powerful enough to override the exercise tachycardia for the period of apnea (3, 4, 34, 36). The cardiac output is reduced throughout apneas during exercise, largely due to the bradycardia, whereas the systemic vascular resistance increases (5). Cold-water face immersion augments the apneic bradycardia also during exercise (4, 36).

Even though the existence of the human diving response is well established, this may not be synonymous with oxygen conservation (20). Some earlier studies suggest an oxygen-conserving effect (7, 37, 38), whereas others do not (15, 16, 29). A close relationship between the magnitude of the diving response and the breath-holding time has been observed among groups of subjects differing in their diving experience (30). This could indicate an oxygen-conserving effect of the diving response. Andersson and Schagatay (1) showed that the augmented diving response observed during apneas with face immersion in resting subjects was accompanied by higher arterial oxygen saturations compared with during apneas in air and concluded that this reflects an oxygen-conserving effect of the human diving response. Lindholm et al. (21) found concordant results during dynamic exercise using apnea and rebreathing as a model to obtain two conditions with and without a diving response. A higher oxygen uptake was observed during the rebreathing periods with no diving response present. However, they concluded that the work of breathing could have caused the higher oxygen uptake in the rebreathing condition (21). Because there is a stronger diving response during apneas with cold-water face immersion, a possible oxygen-conserving effect of the diving response could be revealed if instead apneas in air and apneas with face immersion during exercise were compared.

The aim of this investigation was to study the diving response and arterial oxygen saturation during apneas in air and apneas with face immersion in cold water during dynamic exercise. We hypothesized that with an augmented diving response during apneas with face
immersion, the resulting nadir arterial oxygen saturation is higher than during apneas in air.

METHODS

Subjects. A group of eight healthy, male subjects volunteered and gave their informed consent to participate in the protocol, which had been approved by the research ethics committee at Lund University. Their mean age was 26 yr (range: 18–29 yr), height 183 cm (173–195 cm), weight 82 kg (73–108 kg), and vital capacity sitting upright 6.1 liters (5.6–6.7 liters). Seven of the subjects were trained breathhold divers or underwater rugby players, involved in breath-hold diving at least 2 h/wk, i.e., Class B according to Schagatay and Andersson (30). The eighth subject did not practice breath-hold diving to the same extent (Class C). In addition to their breath-hold diving, the frequency of physical exercise averaged 4 h/wk (0–9 h/wk). All subjects were nonsmokers. Subjects reported to the laboratory after at least 2 h without any heavy meal or caffeine-containing beverages.

Experimental protocol. First, the probes of the noninvasive instruments were attached and the vital capacity was measured in subjects sitting upright on a cycle ergometer (Monark 829 E, Monark Exercise, Varberg, Sweden). A container used for face immersion was positioned on a shelf in front of the ergometer. The subject’s arms could rest on both sides of the container. By just flexing the neck, the entire face, including the chin and forehead, could be immersed with maintained body position during the exercise. Also during the apneas without face immersion the neck was flexed so that the tip of the nose was just over the water surface. This ensured that the body was in virtually the same position during all apneas. When stable cardiovascular data were observed, recordings began. After an additional 2-min period of rest on the cycle ergometer, the subject performed upright, steady-state, dynamic leg exercise at a constant workload of 100 W for ~50 min (Fig. 1). During the exercise, the subject performed a total of eight apneas, alternating between apnea with face immersion and apnea with the face in the air above the water surface. Exercise began 5 min before the first apnea and continued until 5 min after the last apnea. The breath-holding time was predetermined to 30 s in both conditions, and the apneas were spaced by 5 min. Four subjects began with an apnea with face immersion and four with an apnea with the face in air. The water temperature was 10.0°C (9.4–10.5°C) and the ambient air temperature 24.3°C (23.9–25.0°C).

Before each apnea, the subject exhaled to his residual volume and inhaled, from a rubber bag, a volume of air equivalent to 80% of the individual sitting vital capacity. On command from the experimenter, the face was lifted and the apnea was terminated after 30 s with a maximal exhalation.

Measurements. Before the test, an electrocardiogram was recorded and checked for anomalies (Cardisuny 501, lead II, Fukuda ME Kogoyo, Tokyo, Japan). The vital capacity was measured with a spirometer (Micro Plus, Micro Medical, Rochester, UK). During the experiment, the heart rate was continuously recorded with a heart rate monitor (Polar Vantage NV, Polar Electro Oy, Kempele, Finland). The arterial blood pressure was continuously recorded with a photo-plethysmometer with the cuff on the left middle finger (Finapres 2300, Ohmeda, Madison, WI). It was previously reported that it is possible to accurately record changes in mean arterial blood pressure with the Finapres during both exercise and apnea (17, 27). The left hand was positioned at the same level relative to the heart throughout the whole experiment. Skin capillary blood flow in the left thumb was continuously recorded with a laser-Doppler flowmeter (Advanced Laser Flowmeter 21, Advance, Tokyo, Japan). Arterial hemoglobin oxygen saturation (SaO2) was continuously recorded with an earlobe pulse oximeter (Biox 3700e, Ohmeda, Madison, WI). All of these parameters were recorded from 2 min before the beginning of exercise until 2 min after the end of exercise.

Data analysis. A control value for each parameter was calculated as an average value from the period 90–30 s before each apnea. For heart rate, mean arterial blood pressure, and skin blood flow, an average value was calculated for each 5-s period during each apnea and also for the last 10 s of apnea. For SaO2, an average value was calculated for each 5-s period from the beginning of apnea until 30 s after apnea, and also for the last 10-s period ending with the nadir SaO2 value. The relative change from control for each 5- and 10-s period was calculated. For each subject, an individual mean value from the three last apneas in each condition was calculated for all parameters, and a mean ± SE of the eight individual means was calculated. The apneic values were compared with control, and the apneas in air were compared with apneas with face immersion using paired t-test. The level used for accepting significance was F < 0.05.

RESULTS

The heart rate gradually decreased from the control level during both apneas in air and apneas with face immersion (P < 0.001, Fig. 2), but the reduction of heart rate was slower during the apneas in air. At the end of apneas in air, the heart rate was higher than at the end of apneas with face immersion, 82 ± 6 vs. 70 ±
6 beats/min ($P < 0.001$). The mean decrease from control in heart rate during the last 10 s of apneas in air was $-21 \pm 4$ vs. $-33 \pm 4\%$ during apneas with face immersion ($P < 0.001$). During steady-state exercise before apneas, the control heart rate was slightly lower before apneas in air, $107 \pm 2$ beats/min, compared with before apneas with face immersion, $110 \pm 2$ beats/min ($P < 0.05$).

The arterial blood pressure increased during apneas ($P < 0.001$), and the change from control was more pronounced during the apneas with face immersion (Fig. 2). The increase from control in mean arterial blood pressure during the last 10 s of apneas was $34 \pm 4\%$ for apneas in air vs. $42 \pm 6\%$ for apneas with face immersion ($P < 0.05$). The control mean arterial blood pressure did not differ before apneas in air and apneas with face immersion, $111 \pm 4$ vs. $112 \pm 5$ mmHg [not significant (NS)]. The skin blood flow was reduced during apneas, both during apneas in air and apneas with face immersion ($P < 0.05$). The mean decrease from control in skin blood flow during the last 10 s of apneas was $-29 \pm 6\%$ for apneas in air and $-39 \pm 5\%$ for apneas with face immersion (NS). The control skin blood flow did not differ before apneas in air and apneas with face immersion.

The $\text{SaO}_2$ began to decrease toward the end of apneas, and it reached a minimum value $15$ s after the end of apneas in both conditions ($P < 0.001$ compared with control, Fig. 2). The nadir $\text{SaO}_2$ value corresponds to the end of apnea but is delayed due to the circulation time between the lungs and earlobe. The value from the 10-s period ending with the nadir $\text{SaO}_2$ value thus corresponds to the last 10 s of apnea. The reduction in $\text{SaO}_2$ from control during this period was larger for apneas in air, $-6.8 \pm 0.8\%$, than for apneas with face immersion, $-5.2 \pm 0.5\%$ ($P < 0.05$). The control $\text{SaO}_2$ was $97 \pm 0.2\%$ before both apneas in air and apneas with face immersion (NS).

**DISCUSSION**

This study shows that during dynamic exercise in trained breath-hold divers, the diving response is induced by apnea and augmented by face immersion in cold water. The augmented response is accompanied by a smaller reduction in $\text{SaO}_2$. This indicates that the human diving response may slow down the depletion of the lung oxygen store and thus have an oxygen-conserving effect during apneas in exercising subjects.

The heart rate reduction is more pronounced when apnea is combined with cold-water face immersion as a result of stimulation of cold receptors located in the area innervated by the ophthalmic nerve, i.e., forehead and eye region (18, 26, 32). This effect is thus observed both during rest and during exercise (4, 31, 35). The
slightly higher heart rate before apneas with face immersions is attributed to an anticipatory response (2).

Apneas during dynamic exercise cause a reduction in cardiac output that is closely related to the reduction in heart rate (5). Consequently, the cardiac output would be lower during apneas with face immersion than during apneas in air. It is unlikely that an increase in stroke volume would have compensated for the heart rate reduction and maintained the cardiac output in the present experiments (5, 11, 35). Rather, it has been shown that during breath holding with a large lung volume, the high intrathoracic pressure impedes venous return and reduces the stroke volume and consequently the cardiac output (12, 24). Therefore, the increased blood pressure and reduced skin blood flow during apneas in the present study indicate an intense arterial vasoconstriction (5). Limb muscle blood flow would be especially reduced by the vasoconstriction and consequent redistribution of blood flow during steady-state exercise (10, 21). As the increase in mean arterial blood pressure was greater during the apneas with face immersion, the peripheral vasoconstriction seems to be augmented by cold-water face immersion.

Sterba and Lundgren (35) also observed more pronounced changes in cardiac output, forearm blood flow, and blood pressure during apneas in cold water (20°C) than in thermoneutral water (35°C) during light, dynamic exercise.

The $\text{SaO}_2$ was reduced more during apneas in air than during apneas with face immersion, which confirms our previous findings in resting subjects (1). This difference should also apply to the alveolar $\text{PO}_2$, and the lung oxygen store would thus be larger during apneas with face immersion. The smaller reduction in $\text{SaO}_2$ thus indicates that the oxygen uptake from the lung was slower during apneas with face immersion than during apneas in air. This is probably due to a reduced cardiac output and a redistribution of peripheral blood flow.

It has been shown that during breath holding, a change in cardiac output results in a corresponding change in oxygen uptake from the lung (23, 24). Also during eupnea, a decrease in cardiac output is associated with a reduced oxygen uptake from the lung and thus with an increase in the arteriovenous oxygen difference and a reduced venous oxygen store (9, 22). However, a preferentially peripheral vasoconstriction and peripheralization of venous blood volume, the latter caused by the high intrathoracic pressure, accompany the drop in cardiac output during apnea. This will prolong the turnover time for tissue (venous) oxygen stores (22, 24). Taken together, the reduced cardiac output and redistribution of peripheral blood flow will preserve the lung oxygen store at the expense on the tissue oxygen stores (in peripheral venous blood). This mechanism slows down the fall in alveolar and arterial $\text{PO}_2$, thereby delaying the development of hypoxia in the heart and brain. Longer apneic durations are thus possible with an augmented diving response. Consequently, this represents an oxygen-conserving effect of the human diving response.

With maintained tissue oxygen consumption, an augmented diving response during apneas with face immersion would thus result in a reduced venous oxygen store and relatively larger lung oxygen store than during apneas in air. However, because the diving response is characterized by a peripheral vasoconstriction that reduces tissue blood flow, tissue oxygen consumption may also be reduced, thereby causing a reduced depletion of both the venous and lung oxygen stores. Affected tissues could to some extent be forced to derive energy from high-energy phosphates, from aerobic metabolism with tissue oxygen stores, and from anaerobic metabolism with lactic acid formation (10, 11). Ferretti et al. (10) found lactic acid accumulation in elite divers performing breathing-hold dives requiring a metabolic power output of ~20–30% of the individual maximal oxygen uptake. Several of our subjects also stated that they experienced symptoms of lactic acid accumulation, e.g., weakness in legs, at the end of or after apneas in both conditions. The low levels of metabolic demand in the former (10) and the present study are normally not associated with lactic acid accumulation during eupnea. Lactic acid accumulation would therefore support the view that there is a substantial muscle vasoconstriction during apneas and indicate an additional oxygen-conserving effect of the human diving response through an increase in anaerobiosis. A shift from aerobic to anaerobic metabolism has been demonstrated during prolonged dives in diving mammals (19, 28, 35), and it is believed that circulatory changes largely determine the magnitude of this shift (6). The cardiovascular responses observed in humans resemble those in diving mammals and may have similar, although less significant, effects.

In conclusion, during steady-state, dynamic leg exercise in trained breath-hold divers, the more pronounced diving response during apneas with cold-water face immersion than during apneas in air was associated with a higher $\text{SaO}_2$. This indicates a slower depletion of the lung oxygen store when the diving response is augmented, and, thereby, the available oxygen is preserved for vital organs. Thus we suggest that the human diving response has an oxygen-conserving effect during exercise through reductions in cardiac output and peripheral blood flow, reducing the oxygen uptake from the lung during apnea.

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REFERENCES


