

# Effects of sodium bicarbonate on voluntary face immersion breath-hold times.

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Sheard PW, Haughey H. Effects of sodium bicarbonate on voluntary face immersion breath-hold times. *Undersea Hyp Med* 2007; 34(2):91-97. The purpose of this study was to examine the effects of orally ingested sodium bicarbonate ( $\text{NaHCO}_3$ ) on voluntary breath-hold time with facial immersion. Eight non-elite breath-hold divers aged 20-41 ( $26.75 \pm 7.83$  years) were recruited to undertake two bouts of three monitored, facial-immersion breath-holds, one week apart. Subjects were randomly allocated to double-blind sessions of ingesting an experimental ( $0.3\text{g}\cdot\text{kg}^{-1}$   $\text{NaHCO}_3$ ) or placebo (8g NaCl) solution. Heart rate, pre- and post-breath-hold end-tidal gases, peripheral oxygen saturation ( $\text{SpO}_2$ ) at right index finger and maximum breath-hold times (Max-BHT) were recorded. Max-BHT was established during the third breath-hold in both trials. Max-BHT of  $168.2 (\pm 20.7)$  sec and  $145.7 (\pm 21.4)$  sec were recorded for the experimental and placebo conditions, respectively ( $P = 0.019$ ). Average bradycardic response did not differ between the experimental and placebo trials ( $P = 0.166$ ). Whilst there was a significant difference in post-breath-hold end-tidal oxygen ( $P_{\text{ET}} \text{O}_2$ ), one was not noted in end-tidal carbon dioxide ( $P_{\text{ET}} \text{CO}_2$ ) measures ( $P = 0.048$ , and;  $P = 0.530$ , respectively). There was significant difference in lowest recorded  $\text{SpO}_2$  ( $P = 0.008$ ) between the trials. It is suggested that ingestion of  $\text{NaHCO}_3$ , prior to facially immersed breath-holds, has an ergogenic effect, prolonging Max-BHT by  $\sim 8.6\%$ .

## INTRODUCTION

It has been suggested that sodium bicarbonate ( $\text{NaHCO}_3$ ) can improve the performance of short term, high intensity physical activity (1-3). While the ergogenic effects of  $\text{NaHCO}_3$  are well known, the reported side effects (gastric distress, diarrhea (3)) are often enough to discourage use during competitive efforts. It is understood to act as a pH-buffering agent in the blood (1-4) when the athlete is functioning at an anaerobic intensity. In competitive breath-hold diving the anaerobic environment is created not by intense physical activity, but through voluntary apnea. Andersson *et al.* (5) indicate an increase in plasma lactate concentration during apnea, particularly when accompanied by facial immersion. As anecdotal evidence of  $\text{NaHCO}_3$  use within the breath-hold diving community

is increasing, the present study was undertaken to determine if orally ingested  $\text{NaHCO}_3$  would act as an ergogenic agent in this unique sports activity.

## METHODS

### Subjects

Following local ethics committee approval of the project, eight non-elite breath-hold divers aged 20-41 ( $26.75 \pm 7.83$  years) were recruited. Criteria for inclusion were that they had some experience of breath-hold diving and that they could hold their breath for at least two minutes. None of the subjects were competitive breath-hold divers at the time of testing. Locally approved health questionnaires

and informed consents were completed before testing commenced. All subjects underwent a familiarization session for protocols and apparatus placement.

### Experimental design

Subjects were randomly allocated to double-blind, counterbalanced sessions of ingesting an experimental ( $0.3\text{g}\cdot\text{kg}^{-1}\text{NaHCO}_3$ ) or placebo ( $8\text{g NaCl}$ ) solution (1,2,3,4), one week apart.

### Experimental protocol

Experimental or placebo solutions were ingested one hour before testing; subjects were encouraged to drink water *ad libitum* post-ingestion to minimize gastrointestinal distress (3). Subjects were then asked to undertake their regular 'breathe-up' protocol. The breathe-up is a period of time during which breath-hold divers compose themselves physically and mentally for the upcoming immersion. There are generally two phases: the initial phase where physical relaxation and mental centering are of primary concern and a shorter second phase where pre-immersion hyperventilation becomes the focus. Fifteen minutes before immersion the subjects were asked to move from their preferred breathe-up position (supine,  $n = 7$ ; seated,  $n = 1$ ) to a seated position in preparation for facial immersion and for placement of experimental apparatus. The oxygen saturation ( $\text{SpO}_2$ ) monitor (LifePulse LP28, HME Ltd., England) was placed on the right index finger, a position chosen in preference to the ear lobe as it may better reflect limb-specific oxygen desaturation and, more practically, avoided immersion of the electronic components. The radio telemetry heart rate transmitter and recorder (Polar Vantage NV, Polar Electro, Finland) were also fitted and the breath-by-breath gas analyzer (SensorMedics, V-MAX 29C, USA) was calibrated at this point. At five minutes and 20 seconds pre-immersion

the breath-by-breath mouthpiece and nose clip were put in place. With five minutes remaining, the breath-by-breath analyzer and heart rate monitor were started simultaneously. Subjects were notified one minute before immersion and again at 30 seconds. The concluding 30 seconds consisted of a standardized series of three deep breaths with forced complete exhalations followed by a final, maximal, deep inhalation. These self-regulated breaths were the only imposed respiratory deviation from the subjects' preferred final breathe-up pattern. In contrast to Lindholm and Gennser (6), the three final breaths were not controlled to limit hyperventilation; indeed, they may have been utilized by the subjects to enhance their state of hyperventilation. The mouthpiece was removed and breath-hold timing was initiated upon facial immersion. The water temperature was maintained at  $14.5^\circ\text{C} (\pm 0.8^\circ\text{C})$  throughout all trials.

Persons experienced with safety procedures and at dealing with loss of consciousness due to hypoxia in aquatic environments were present at all times and were charged with the responsibility of eliciting a manual response to a finger tap to the wrist of the subject at 15 second intervals throughout the breath-hold. A double manual signal from the subject indicated their anticipation of the cessation of breath-hold. The breath-by-breath mouthpiece was prepared and, upon cessation of breath-hold, was inserted into the subject's mouth before their first post-immersion exhalation. With the mouthpiece in place, the subject's face was dried with a towel and expired gases were recorded during the five minutes preceding the second breath-hold. Three breath holds were performed at each testing session, with measurements continuing for three minutes following the final breath-hold. These breath-holds will be referred to as breath-holds 1, 2 and 3 during the discussion.

### Statistical analysis

Tests for normality of data (Shapiro-Wilks) and homogeneity of variance (Levene's Test) preceded the use of paired-sample *t*-tests to determine significant differences between experimental and placebo conditions at  $\alpha = 0.05$ . All analysis was undertaken with the Statistics Package for Social Sciences (SPSS Inc., V.12.0.1, Chicago, Illinois, USA).

## RESULTS

### Breath-hold time

A max-BHT of 168.2 ( $\pm 20.7$ ) sec and 145.7 ( $\pm 21.4$ ) sec were recorded for the experimental and placebo conditions ( $P = 0.019$ ; 95% CI = 4.9 to 40.0 sec), respectively, for breath-hold 3 (Figure 1, see page 88). This represents a performance increase of ~8.6%. No significant difference was recorded for max-BHT between experimental and placebo conditions across breath-holds 1 (136.9  $\pm$  24.0 versus 123.9  $\pm$  26.5) and 2 (142.0  $\pm$  26.9 versus 138.6  $\pm$  17.0).

### Pre-Breath-hold Expired Gases

Evidence of hyperventilation was present as the pre-breath-hold  $P_{ET} CO_2$  was depressed in all instances.  $P_{ET} CO_2$  values of 3.6 ( $\pm 0.41$ ) %, 3.3 ( $\pm 0.44$ ) % and 2.9 ( $\pm 0.27$ ) % were recorded for the experimental group; the placebo recorded 4.0 ( $\pm 0.37$ ) %, 3.7 ( $\pm 0.34$ ) % and 3.5 ( $\pm 0.24$ ) % for breath-holds 1 through 3. A significant difference in  $P_{ET} CO_2$  values was reached only at breath-hold 3 ( $P < 0.001$ ; 95% CI = -0.77 to -0.45%; (Figure 2, see page 88).

### Post-Breath-hold expired gases

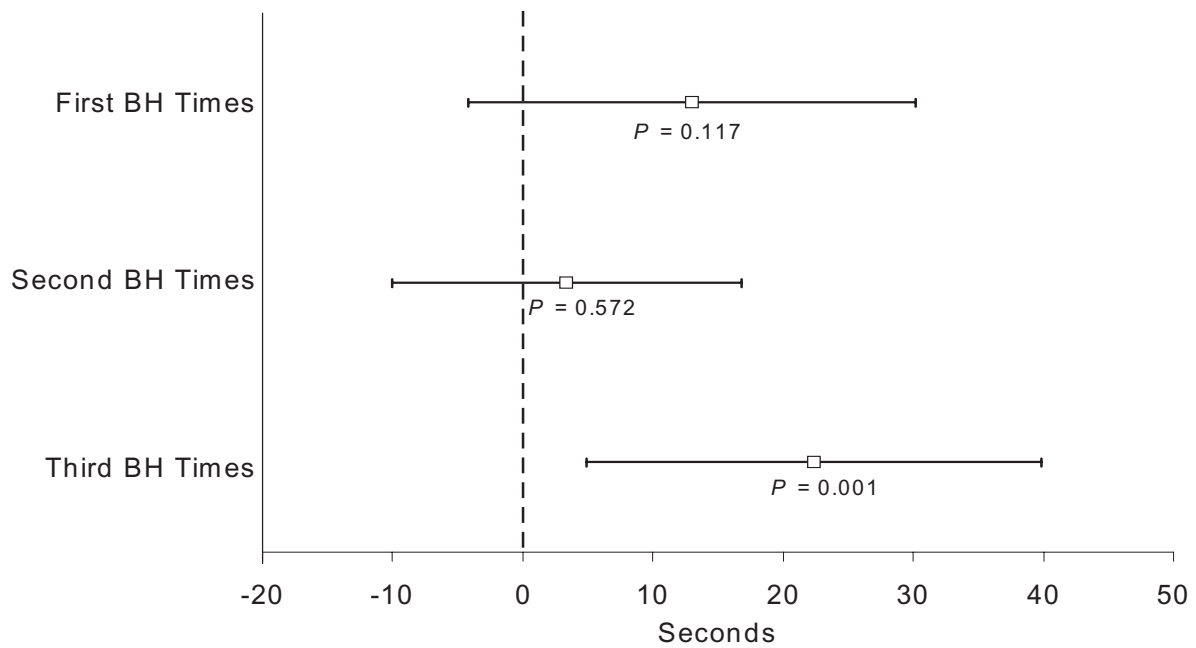
Whilst all values for  $P_{ET} CO_2$  following the three breath-holds were lower with the experimental solution than with the placebo

(6.2 ( $\pm 1.0$ ) %, 5.1 ( $\pm 1.5$ ) % and 4.6 ( $\pm 1.3$ ) % versus 6.4 ( $\pm 0.3$ ) %, 5.7 ( $\pm 1.4$ ) % and 4.9 ( $\pm 1.5$ ) %), none of the differences reached significance ( $P = 0.530$ ; 95% CI = -1.0 to 0.6%,  $P = 0.478$ ; 95% CI = -2.1 to 1.1% and  $P = 0.503$ ; 95% CI = -1.1 to 0.6%, respectively). Experimental  $P_{ET} O_2$  values were seen to rise across the three breath-holds (10.7 ( $\pm 1.9$ ) %, 12.7 ( $\pm 2.5$ ) % and 14.3 ( $\pm 1.6$ ) %) whilst the placebo values fell (12.8 ( $\pm 2.2$ ) %, 12.7 ( $\pm 2.8$ ) % and 12.5 ( $\pm 2.0$ ) %). Only at post breath-hold 1 was the difference significant ( $P = 0.048$ ; 95% CI = -3.7 to -0.1%) although at post breath-hold 3 there was a strong trend towards higher  $P_{ET} O_2$  with the  $NaHCO_3$  present ( $P = 0.095$ ; 95% CI = -0.3 to 3.6%).

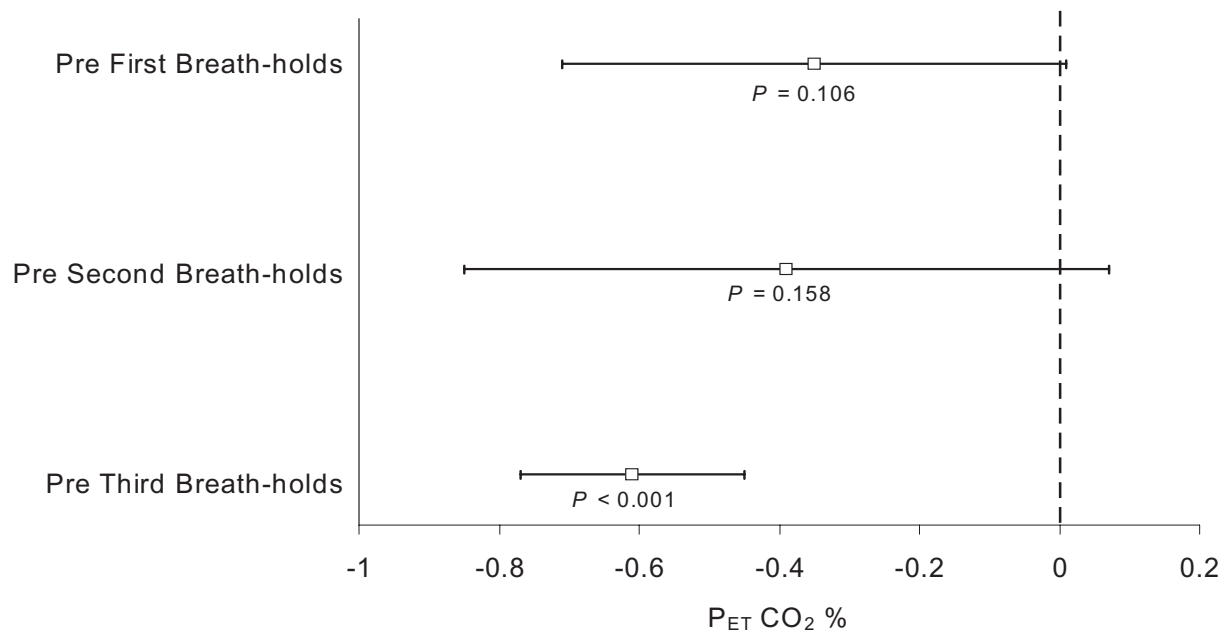
### Bradycardia

Heart rate was recorded as R-R in milliseconds, but later resolved, in Microsoft Excel®, as beats per minute (BPM) over a rolling ten-second average. Pre-immersion resting heart rate was calculated as the average heart rate during the five minutes preceding the first breath-hold, including the standardized forced exhalations. Resting heart rate was 84.9 ( $\pm 7.7$ ) BPM and 76.9 ( $\pm 9.3$ ) BPM for the experimental and placebo trials ( $P = 0.048$ ; 95% CI = -15.8 to -0.1 BPM).

Extent of bradycardia was determined as the lowest recorded heart rate (rolling 10 second average) during each of the three consecutive breath-holds. Although average bradycardic heart rates were noted to be higher during the experimental session (63.5 ( $\pm 5.3$ ) BPM, 61.1 ( $\pm 5.6$ ) BPM and 60.6 ( $\pm 5.2$ ) BPM) than the placebo session (60.1 ( $\pm 7.7$ ) BPM, 59.8 ( $\pm 7.7$ ) BPM and 58.0 ( $\pm 7.2$ ) BPM) these differences were not significant ( $P = 0.070$ ; 95% CI = -0.4 to 7.1 BPM;  $P = 0.497$ ; 95% CI = -3.2 to 5.9 BPM; and  $P = 0.093$ ; 95% CI = -0.6 to 5.8 BPM).



**Fig. 1.** Mean difference and 95% confidence intervals (CI) for experimental ( $\text{NaHCO}_3$ ) minus placebo ( $\text{NaCl}$ ) breath-hold (BH) times at each of three consecutive breath-holds.



**Fig. 2.** Mean difference and 95% confidence intervals (CI) for experimental ( $\text{NaHCO}_3$ ) minus placebo ( $\text{NaCl}$ ) pre-breath-hold  $P_{\text{ET}} \text{CO}_2$  at each of three consecutive breath-holds.

### Oxygen saturation

SpO<sub>2</sub> measured at the right index finger indicated a significant difference in lowest recorded peripheral SpO<sub>2</sub> ( $P = 0.010$ ; 95% CI = 1.4 to 7.1%) between the experimental (90.5 ( $\pm 6.5$ ) %) and placebo trials (94.8 ( $\pm 4.2$ ) %) immediately post-breath-hold 1. Post-breath-hold 2 showed a similar pattern with 84.9 ( $\pm 9.5$ ) % and 88.3 ( $\pm 8.2$ ) % ( $P = 0.026$ ; 95% CI = 0.6 to 6.3%). This trend continued to breath-hold 3: 79.0 ( $\pm 13.3$ ) % and 87.2 ( $\pm 10.9$ ) % ( $P = 0.008$ ; 95% CI = 3.3 to 13.0%).

### DISCUSSION

Although the exact biochemical mechanisms of the ergogenic effect of NaHCO<sub>3</sub> were not obtainable via the current protocol, the increased BHT post-ingestion were plainly evident. Max-BHT experimental was 168.2 ( $\pm 20.7$ ) sec and placebo 145.7 ( $\pm 21.4$ ) sec, an increased performance of ~8.6%. Despite the extension of breath-hold duration, the relationship between the use of NaHCO<sub>3</sub> and its impact on the human diving response is not clear.

Of the values measured, the pre-breath-hold P<sub>ET</sub> CO<sub>2</sub> pattern most closely matches the progression of the extension of BHT (compare Figures 1 and 2). As pre-breath-hold P<sub>ET</sub> CO<sub>2</sub> falls BHT rises. This reflects the classic pattern of hyperventilation increasing BHT (7) although the current pre-breath-hold values did not quite reach those demonstrated by Craig [ $\sim 2.8\%$ ] (7). Both BHT and pre-breath-hold P<sub>ET</sub> CO<sub>2</sub> reach significant differences between NaHCO<sub>3</sub> and NaCl protocols at the third breath-hold.

Manley's review of heart rate response to apnea (8) reflects strong evidence of bradycardia with apnea and facial immersion as a primary component of the human diving response. The bradycardic component of the diving response was demonstrated during both sessions with average decreases in intra-

breath-hold heart rates of 21.4 to 24.3 BPM (experimental) and 26.8 to 28.9 BPM (placebo). These decreases are in line with those observed in previous studies of apnea both with (5,9) and without face immersion (5,9-11). The observed differences in bradycardia noted between the testing sessions suggest that NaHCO<sub>3</sub> was actually counterproductive to the elicitation of bradycardia. Discomfort from gastric bloating and increased eructation reported by the subjects may explain the elevated resting and intra-breath-hold heart rates in the experimental group.

Peripheral vasoconstriction (12,13) is also reported in response to apnea and immersion. This, together with bradycardia, may comprise an O<sub>2</sub> conserving reflex during both static apnea (14) and during apnea accompanied by exercise (10). While we did not directly measure peripheral vasoconstriction the increase of P<sub>ET</sub> O<sub>2</sub> across the three breath-holds with NaHCO<sub>3</sub>, while the P<sub>ET</sub> O<sub>2</sub> fell across the placebo breath-holds, combined with the significantly lower right index finger SpO<sub>2</sub> during all three breath-holds with NaHCO<sub>3</sub>, suggests a change in the body's O<sub>2</sub> transport/consumption strategy with NaHCO<sub>3</sub>. The drop in SpO<sub>2</sub> noted in the current study may have been even more profound had values been measured at the ear as Lindholm and Gennser (15) have demonstrated that there is a 15.0 ( $\pm 3.5$ ) s delay ( $P < 0.001$ ; [95% CI not given]) and a  $\sim 6.2$  ( $\pm 4.0$ ) % ( $P = 0.01$ ; [95% CI not given]) higher maximum when reading SpO<sub>2</sub> at the finger compared to the ear. However, these differences may reflect changes in O<sub>2</sub> conservation strategies as moderated by exercise during apnea; subjects in the current study remained at rest.

The efficacy of NaHCO<sub>3</sub> as a buffer is suggested by the lower but non-significant difference between experimental and placebo levels of P<sub>ET</sub> CO<sub>2</sub> following all three breath-holds. It may be argued that the significantly higher consumption of O<sub>2</sub> during breath-hold 1 was

not matched by a concomitant  $\text{CO}_2$  production following  $\text{NaHCO}_3$  ingestion. Similarly, the increased use of peripheral  $\text{O}_2$  measured at the right index finger, as indicated by significantly decreased  $\text{SpO}_2$  in all experimental breath-holds, is not associated with an increased  $\text{P}_{\text{ET}} \text{CO}_2$  production following  $\text{NaHCO}_3$  ingestion;  $\text{P}_{\text{ET}} \text{CO}_2$  was seen to fall from 6.16 to 4.62%. It is postulated that the  $\text{NaHCO}_3$  acted to buffer rising carbonic acid levels ‘locking’ some of the potentially expired  $\text{CO}_2$  in the bloodstream, although this was not directly measured.

If we make the same assumption as Overgaard, *et al.* (16) that  $\text{P}_{\text{ET}} \text{O}_2$  corresponds with  $\text{P}_a \text{O}_2$  and a similar correspondence exists with  $\text{P}_{\text{ET}} \text{CO}_2$  and  $\text{P}_a \text{CO}_2$ , then we can discuss the observed  $\text{P}_{\text{ET}}$  in relation to the physiological breakpoint of the breath-hold. The physiological breakpoint (17) indicates the time at which the ability to maintain apnea can no longer override the urge to breathe and the body attempts involuntary ventilatory activity. In trained breath-holders it is not uncommon to see a series of these involuntary spasms (16) when the physiological breakpoint is reached. The conventional breakpoint (17) is the point at which apnea is broken, whether preceded by obvious physiological necessity or not. None of the subjects in the present study were seen to make, or reported any involuntary spasms. Their  $\text{P}_{\text{ET}} \text{CO}_2$  levels for both experimental and placebo sessions approached, but did not exceed, those indicated as typical (16,18) for involuntary spasms to occur.

The subjects of the present study, under the placebo protocol, maintained stable post-breath-hold  $\text{P}_{\text{ET}} \text{O}_2$  at levels above those indicated by previous studies of trained breath-hold divers (16,18) as being indicative of the  $\text{O}_2$  breakpoint. Trained divers who engage in profound hyperventilation prior to apnea (16) may reach these critical  $\text{O}_2$  levels as  $\text{P}_a \text{CO}_2$  build up, the normal breakpoint trigger, will have been delayed. In contrast to the trained

subjects of Overgaard *et al.*'s study (15), the subjects undergoing the  $\text{NaHCO}_3$  session in the present study were seen to have progressively higher post-breath-hold  $\text{P}_{\text{ET}} \text{O}_2$  despite their increasing times across the three breath-holds. This counterintuitive finding needs further investigation.

An extension of facially-immersed breath-hold time through the use of  $\text{NaHCO}_3$  has been demonstrated, but the mechanisms of action cannot be decided from the data. In light of this lack of understanding, the use of  $\text{NaHCO}_3$  cannot be recommended in breath-hold diving against the well-documented background of the risk of hypoxic loss of consciousness in aquatic environments.

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#### REFERENCES

1. Lavender G, Bird SR. Effect of sodium bicarbonate ingestion upon repeated sprints. *Br J Sports Med* 1989; 23(1):41-5.
2. Linderman J, Fahey TD. Sodium bicarbonate ingestion and exercise performance. *Sports Med* 1991; 11(2):71-7.
3. Linderman JK, Gosselink KL. The effects of sodium bicarbonate ingestion on exercise performance. *Sports Med* 1994; 18(2):75-80.
4. Gaitanos GC, Nevill ME, Brooks S, Williams C. Repeated bouts of sprint running after induced alkalosis. *J Sports Sci* 1991; 9(4):355-70.
5. Andersson J, Liner MH, Fredsted A, Schagatay E. Cardiovascular and respiratory responses to apneas with and without facial immersion. *J Appl Physiol* 2004; 96(3):1005-10.
6. Lindholm P, Gennser M. Aggravated hypoxia during breath-holds after prolonged exercise. *Eur J Appl Physiol* 2005; 93(5-6):701-7.
7. Craig AB Jr. Causes of loss of consciousness during underwater swimming. *J Appl Physiol* 1961; 16(4):583-6.
8. Manley L. Apnoeic heart rate response in humans. *Sports Med* 1990; 9(5):286-310.
9. Andersson JP, Liner MH, Runow E, Schagatay

- EK. Diving response and arterial oxygen saturation during apnea and exercise in breath-hold divers. *J Appl Physiol* 2002; 93(3):882-6.
10. Lindholm P, Sundblad P, Linnarsson D. Oxygen-conserving effects of apnea in exercising men. *J Appl Physiol* 1999; 87(6):2122-7.
  11. Lindholm P, Linnarsson D. Pulmonary gas exchange during apnoea in exercising men. *Eur J Appl Physiol* 2002; 86(6):487-91.
  12. Heistad DD, Abboud FM, Eckstein JW. Vasoconstrictor response to simulated diving in man. *J Appl Physiol* 1968; 25(5):542-9.
  13. Foster GE, Sheel AW. The human diving response, its function and control. *Scand J Med Si Sports* 2005; 15(1):3-12.
  14. Andersson J, Schagatay E. Arterial oxygen desaturation during apnea in humans. *Undersea Hyperb Med* 1998; 25(1):21-5.
  15. Lindholm P, Gennser M. Detection of hypoxemia with pulse oximetry during apnea is impaired using finger probe compared to ear lobe probe. [Abstract.] Presented at the Undersea and Hyperbaric Medical Society annual meeting Las Vegas, 2005.
  16. Overgaard K, Friis S, Pedersen RB, Lykkeboe G. The influence of lung volume, glossopharyngeal inhalation and  $P_{ET} O_2$  and  $P_{ET} CO_2$  on apnea performance in trained breath-hold divers. *Eur J Appl Physiol* 2006; 97(2):158-64.
  17. Lin YC, Lally DA, Moore TO, Hong SK. Physiological and conventional breath-hold breaking points. *J Appl Physiol* 1974; 37(3):291-6.
  18. Andersson J, Shagatay E. Effects of lung volume and involuntary breathing movements on the human diving response. *Eur J Appl Physiol Occup Physiol* 1998; 77(1-2):19-24.