Alveolar gas composition before and after maximal breath-holds in competitive divers.

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Lindholm P., Lundgren CEG. T Alveolar gas composition before and after maximal breath-holds in competitive divers. Undersea Hyperb Med 2006; 33(6):463-467. The urge to breathe, as stimulated by hypercapnia, is generally considered to cause a breath-hold diver to end the breath-hold, and pre-breath hold hyperventilation has been suggested to cause hypoxic loss of consciousness (LOC) due to the reduced urge to breathe. Competitors hyperventilate before "Static Apnea", yet only 10% surface with symptoms of hypoxia such as loss of motor control (LMC) or LOC. We hypothesized that the extensive hyperventilation would prevent hypercapnia even during prolonged breath-holding and we also recorded breaking-point end-tidal PO_2 in humans. Nine breath-hold divers performed breath-holds of maximal duration according to their chosen "Static Apnea" procedure. They floated face down in a swimming pool (28°C). The only non-standard procedure was that they exhaled into a sampling tube for end-expiratory air, before starting the breath-hold and before resuming breathing. Breath-hold duration was 284±25 (SD) seconds. End-tidal PCO_2 was 18.9±2.0 mmHg before apnea and 38.3±4.7 mmHg at apnea termination. End-tidal PO_2 was 131.7±2.7 mmHg before apnea and 26.9±7.5 mmHg at apnea termination. Two of the subjects showed LMC after exhaling into the sampling tube; their end-tidal PAO_2 values were 19.6 and 21.0 mmHg, respectively. End-tidal CO_2 was normocapnic or hypocapnic at the termination of breath-holds. These data suggest that the athletes rely primarily on the hypoxic stimuli, probably in interaction with CO_2 stimuli to determine when to end breath-holds. The severity of hypoxia close to LOC was similar to that reported for acute hypobaric hypoxia in humans.

INTRODUCTION

Since Craig (1) showed that hyperventilation reduces the urge to breathe from hypercapnia during a breath-hold (BH), sometimes causing motivated individuals to hold the breath until loss of consciousness (LOC), BH diving instructors generally discourage hyperventilation. Even though the hypoxic ventilatory drive is also involved in the urge to breathe (2), and will stimulate breath-hold divers to resume breathing, this stimulus has been considered too week to ensure safe breath-hold diving (3). Every year recreational swimmers unaware of the dangers of hyperventilation drown. Nonetheless, athletes competing for duration in immersed breath-holding (Static Apnea) typically hyperventilate

(personal observation) before performances, yet only about 10% surface with symptoms of severe hypoxia such as loss of motor control (LMC) or rarely loss of consciousness (4).

While studies on acute hypoxia at altitude has been driven by its practical importance for aviation (5), little information is available on acute asphyxia in breath-hold diving. Therefore, this study was designed to explore the level of hypoxia commonly experienced during Static Apnea as related to the specific endpoints of LMC and LOC. We hypothesized that hyperventilation, in preparation for prolonged breath-holding by trained BH-divers, would preclude hypercapnia to reach conventional breaking-point levels (6) (in the absence of preparatory hyperventilation).

METHODS

The experimental procedure conformed with the Declaration of Helsinki and had been approved by the Institutional Review Board of the University at Buffalo, NY. The subjects gave informed consent to participate.

As part of their regular training schedule, nine male amateur breath-hold divers performed their standard apnea protocol while trying to perform apneas of maximal duration. Some of the subjects had competitive experience while others had only practiced this sport for 6 months. None of the subjects held any breath-hold diving records. Subject D was tested on two separate occasions. Subjects were 27-69 years of age, 166-198 cm in height, and 64-121 kg in weight. Vital capacities were 6.3±0.5 (range 5.7-6.9) L and barometric pressure was 740±7mmHg.

Procedure

Subjects were floating motionless face down in a swimming pool with a water temperature of 28°C. They typically wore wetsuit, a nose-clip and goggles or a facemask. Some divers wore a watch to monitor BH-duration. The divers were required to respond to a signal from their individual safety tenders every 15 sec to ensure consciousness. No attempt was made by the investigators to interfere with this schedule except that the divers were asked to exhale into a sampling tube for end-expiratory air, a few breaths before starting the breath-hold and directly after surfacing. Gas was drawn from the sampling tube within 30 seconds and analyzed for CO_2 and O_2 concentrations by a calibrated mass spectrometer (MGA 1100 Medical Gas Analyzer, Perkin-Elmer Corp., Pomona, CA). A Morgan Spiroflow Spirometer Model #131 (PK Morgan Ltd., Rainham, Gillingham, Kent, UK) was used for vital capacity (VC) measurements.

RESULTS

Breath-hold duration was 284±25 (range 241-325) sec. End-tidal PCO_2 was 18.9±2.0 (range 15.6-21.9) mmHg post-hyperventilation about 20 sec before apnea and 38.3±4.7 (range 29.5-43.4) mmHg at the termination of apnea. End-tidal PO_2 was 131.7±2.7 (range 127.5-135.1) mmHg before apnea and 26.9±7.5 (range 19.6-42.2) mmHg at the termination of apnea. Five subjects delivered samples with ET PO_2 levels below 25mmHg, and two of them had LMC, Table 1. In this table a repeat test on subject D is reported where he did not have LMC. There were two other performances (by subjects A and C) with slightly longer duration than the ones reported in Table 1 but the expired samples were contaminated by room air because the subjects failed to make a proper exhalation into the tube, probably because of hypoxia as

Table 1.

Subject	Duration (seconds)	Pre ETO2 (mmHg)	Pre ETCO2 (mmHg)	Post ETO2 (mmHg)	Post ETCO2 (mmHg)
A	256	134.2	19.7	23.6	39.5
B	287	133.2	18.4	23.0	42.5
C	287	135.1	19.6	23.1	35.5
D	298	135.1	16.7	22.4	42.7
A (LMC)	262	133.9	20.1	-	-
C (LMC)	298	134.4	18.5	-	-
D (LMC)	306	131.5	16.7	19.6	38.7
E (LMC)	272	131.6	15.6	21.0	29.5

further evidenced by LMC. No subject suffered LOC during any of the experiments.

Calculating the possible benefits of hyperventilation for breath-hold duration

Hyperventilation will reduce CO_2 but also increase oxygen stores in the lungs. This is due to a reduction in $PACO_2$ allowing an increase in PAO_2. There are also changes in blood and tissue stores of CO_2 and O_2 during hyperventilation and breath-holding but data available in the literature is not complete enough for quantitative considerations and furthermore the changes would be very small. Therefore, the following reasoning is limited to the effect in the lungs.

Physiological data for a normal male individual were used to calculate the increase lung oxygen stores (7) i.e: Weight 70 kg, total lung capacity 7 L BTPS, resting O_2 consumption 250 ml/min STPD (8). For volume conversion, BTPS*0.8259 = STPD.

Without hyperventilation:
PAO_2 100 mmHg (13.3 kPa).
Lung O_2 content: 7x0.8259x100/760 = 761 ml O_2 STPD

With hyperventilation:
PAO_2 135 mmHg (18.0 kPa).
Lung O_2 content: 7x0.8259x135/760 = 1027 ml O_2 STPD

These considerations yield a gain in "safe" BH time through hyperventilation of:
(1027 ml-761 ml)/250ml/min = 1 min and 4 sec.

DISCUSSION

Breath-hold divers managed to surface and exhale into a sampling tube for end-tidal gas after about 5 min of breath-holding with end-tidal PO_2 levels as low as 20 mmHg, with LMC, and as low as 22-23 mmHg without LMC/LOC. The divers hyperventilated extensively so as to be hypocapnic before breath-holding and were still hypocapnic or normocapnic at the termination of breath-holding. If we consider LOC instead of the urge to breathe as the end-point of a breath-hold the effect of hyperventilation to increase safe BH-duration can be ascribed to an increase in oxygen stores which can be calculated to suffice for more than one minute of extra BH time before LOC. Moreover, trained breath-hold divers have been shown to have a gradually reduced oxygen uptake from the lungs during long breath-holds (9). Note that there is typically a very tight temporal coupling between LMC/LOC, and LOC is in other settings (e.g. altitude), typically expected at a PO_2 of about 30 mmHg which is well above our present observations.

Hyperventilation for an extended duration may cause profound hypocapnia with associated neurological symptoms such as paresthesias and tetanus (10). The divers where all hypocapnic when starting the BH but, in contrast to what would be expected, neither did they report symptoms nor were any signs of hypocapnia observed. All divers were able to handle the sampling tube and exhale into it prior to breath-holding. An unanswered question is whether this lack of sensitivity to hypocapnia is due to adaptation similar to what is known to occur in mountaineers (11) although in the latter category the hypoxia exposure is more sustained which probably is a condition for the changes in bicarbonate levels of the cerebrospinal fluid which account for the acclimatization.

When the carbon dioxide content in the blood has been reduced by hyperventilation, the drive to breathe is abolished, thus making it possible to hold the breath for a period longer than the time it takes to reach critical hypoxemia (1). This is very dangerous and

accidents(sometimes fatal) occur every year (3). Nonetheless, apnea athletes almost always hyperventilate before diving. They use a mode of ventilation that consists of slow and deep breaths; approx 4-6 breaths/minute (12) (personal observation) for an extended period. Our subjects hyperventilated for approximately 6-8 minutes before each apnea including warm-ups, alternating deep slow breathing with faster deep breathing. Remarkably, these divers manage to sense when to surface to avoid LOC, a notion that suggest that they rely on the hypoxic ventilatory drive (2,13) or other cues to know when to abort breath-holding. Some divers describe that they surface when their vision changes (grey-out) while others mention some other non-specific sensation of altered mental state.

The most reliable data on the effects of acute hypoxia is available in the literature on aviation medicine. Macmillian reports a "time of useful consciousness" of 40 seconds after an explosive decompression to an altitude of 10,668 m (ambient pressure 179 mmHg) while breathing air (14). Depending on $PACO_2$ which may range between 20 and 40 mmHg, PAO_2 will be 24-19 mmHg. The interaction between PaO_2 and $PaCO_2$ in influencing LOC has been described by Ernsting (5) as follows: "*Consciousness is lost when the jugular venous oxygen tension is reduced to 17-19 mmHg. The corresponding cerebral arterial oxygen tension varies with cerebral blood flow, which itself depends upon the arterial tensions of oxygen and carbon dioxide. Thus the arterial oxygen tension that produces a jugular venous tension sufficiently low to cause unconsciousness can lie between 20 and 35 mmHg depending on the degree of hypocapnia. Accordingly, although consciousness is usually lost when the alveolar oxygen tension is reduced to 30 mmHg or below for a significant period of time, it is possible to lose consciousness with an alveolar oxygen tension as high as 40 mmHg if there is marked*

hyperventilation, or to retain consciousness at an alveolar oxygen tension as low as 25 mmHg if there is no hypocapnia." However, during apnea the level of hypoxia will not be stable, and thus the exact duration of "useful consciousness" during apnea is difficult to predict. An important point made by Ernsting is that hypocapnia impedes oxygen transport to the brain at two levels; partly by reducing brain perfusion and partly by shifting the O_2 dissociation curve of hemoglobin to the left.

There are some previous publications on ET PO_2 in breath-holding: Ferretti et al. (9) report an exhaled PO_2 of 29 and 28 mmHg after resting non-immersed apneas of 270 and 300 seconds, respectively. Hyperventilation followed by apnea during exercise has been reported to produce convulsions, ET PO_2 was measured to values between 22 and 26 mmHg (15). The same author also did BH without prior hyperventilation (and thus higher end apnea CO_2). These tests did not cause convulsions at ET PO_2 of 26 mmHg. In the classic work showing the physiological basis of ascent blackout, Lanphier and Rahn describe one subject hyperventilating and then exercising lightly on an ergometer cycle during a simulated dive to 2 ATM (10m) in a pressure chamber: "*upon ascent, his PO_2 dropped to 24mmHg, O_2 uptake ceased, and there is evidence that O_2 was being extracted from the blood. Impairment of consciousness occurred.*" (16). Field test on one diver during no-limit assisted diving has reported ET PO_2 of 24 mmHg without LOC (17). However, the severity of hypoxia close to loss of consciousness during apnea seems to be of similar intensity as in the "steady state" situation of hypobaric hypoxia during breathing.

CONCLUSIONS

The reasoning for discouraging hyperventilation before underwater swimming

seemingly does not strictly apply to Static Apnea. The obvious reason for this is that the swimming BH diver consumes oxygen at a much higher rate than the resting diver. Thus, in the swimmer, the time between hypoxic chemoreceptor stimulation forcing an ascent and arterial blood PO_2 is often short enough to cause LOC, while the Static Apnea diver who is resting has more time to react to clues for terminating the breath-hold. Furthermore, it seems as if the levels of tolerable hypoxia described for acute high-altitude exposures correspond fairly well to the tolerance to acute hypoxia due to apnea observed presently. This appears reasonable since in both cases hypocapnia or normocapnia is common.

ACKNOWLEDGMENTS

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