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Apnea: A new training method in sport?

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SUMMARY

The physiological responses to apnea training exhibited by elite breath-hold divers may contribute to improving sports performance. Breath-hold divers have shown reduced blood acidosis, oxidative stress and basal metabolic rate, and increased hematocrit, erythropoietin concentration, hemoglobin mass and lung volumes. We hypothesise that these adaptations contributed to long apnea durations and improve performance. These results suggest that apnea training may be an effective alternative to hypobaric or normobaric hypoxia to increase aerobic and/or anaerobic performance.

Introduction

Recent observations of several of the physiological responses to apnea [1] and apnea training in breath-hold divers (BHDs) suggested this technical note. Apnea durations clearly increase with training. Perhaps less well known are the findings that apnea training also increases hematocrit (Hct), erythropoietin (EPO) concentration, hemoglobin (Hb) mass, and lung volumes [2–5]. In addition, blood acidosis and oxidative stress were shown to be reduced after three months of apnea training [6,7]. Therefore, why not encourage apnea training for athletes? As these effects are advantageous in several sports, would they not offer an alternative means to improve performance? We hypothesise that apnea training may be an effective alternative to hypobaric or normobaric hypoxia to increase aerobic and/or anaerobic performance.

Hypoxia effects

The major determinant of aerobic performance is the capacity to deliver oxygen to the tissues [8]. An increase in the total amount of erythrocytes, as reflected by increased Hct and Hb mass, is mediated by the glycoprotein hormone EPO, which is predominantly synthesized by the kidneys in response to chronic hypoxia [9] and to some extent (10–15% of total production) by the liver. EPO stimulates the proliferation and maturation of red blood cell precursors in bone marrow, increasing oxygen delivery to muscle and thereby enhancing sports performance [9]. Low tissue oxygenation (hypoxic or ischemic conditions) results in a stabilization of the transcription factor hypoxia-inducible factor (HIF)-1α, which increases EPO secretion and the expression of EPO receptor [10]. The use of recombinant human erythropoietin (rhEPO) or its analogues (i.e., darbepoetin alfa, NESP) is banned by the World Anti-Doping Agency (WADA), as well as by most major sports authorities, and their detection leads to sanctions [11]. Thus, alternative methods have been developed to augment endogenous EPO by "natural" means. Altitude-training to ensure a hypoxic environment optimizes the stimuli needed to improve oxygen delivery while avoiding the detraining effects associated with chronic hypoxia [12,13]. However, many controlled studies have failed to provide evidence of improved aerobic performance at sea level after an altitude-training sojourn [14,15]. The "living high, training low" method, proposed by Levine and Stray-Gundersen, is widely used by endurance athletes [16]. It consists of combining resting exposure to hypoxia with normoxic training. The results of this type of training on total Hb mass [17,18] and sea-level performance are conflicting, as they depend mainly on the conditions of the hypoxia exposure (length of exposure, number of repetitions, etc.) [14]. Furthermore, any training effect vanishes rapidly (two weeks), as the newly formed red cells disappear within a matter of days due to neocytolysis. Brief exposures to normobaric hypoxia, as a stimulus for improved performance is another method, though still under debate.

Apnea training effects

The splenic contraction effect

Apnea training may well be a future training method. Splenic contraction has been described in marine mammals as improving

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oxygen transport, through an increase in circulating erythrocytes. Its consequence is a prolonged dive without injuries. In humans, repeated apneas (five, in general) induce splenic contraction. This increases Hct and Hb (both between 2% and 5%) independently of hemoconcentration [19] and reduces arterial oxygen desaturation, thereby prolonging the apnea duration [3,19–22]. Repeated apneas are known to induce hypoxemia in the spleen and kidney, increasing respectively Hct and Hb and serum EPO concentrations [2,23]. These findings support the idea that apnea could be an effective stimulus of aerobic performance. Similar results were observed in obstructive sleep apnea patients [24] and, moreover, this apnea response was abolished in splenectomized volunteers [19,20]. Although the response of these two mechanisms is different (i.e., splenic contraction and EPO production), their actions may be synergistic. First, the splenic contraction develops quickly after three or four apneas separated by two minutes of recovery and is associated with a transient increase in Hb concentration. The amplitude of the spleen volume reduction after repeated apneas, with or without face immersion, varies widely (20–46%) depending on the rate of change in oxygenation [3,19,22,25–27]. The rapidity of the splenic contraction after simulated apneas strongly suggested a centrally-mediated feed-forward mechanism rather than the influence of slower peripheral triggers [19]. These spleen and Hb responses may be trainable. Indeed, the increase in apnea duration across serial apneas tended to be greater after apnea training [28], and trained BHDs were found to have a more marked increase in Hb (+5%) during apneas than untrained subjects and elite cross-country skiers [1,23]. Second, DeBruijns et al. [2] recently observed that repeated apneas increased EPO concentration by 24%, with the peak value reached 3 h after the last apnea and a return to baseline 2 h later. EPO production induced by high altitude was previously shown to peak about 3 h after the end of altitude exposure, with a half-life of about 5 h [29]. These results agreed with a previous altitude study showing a 24% EPO increase after 6 h at an altitude of 1,780 m [27]. The rapid reduction in tissue oxygen levels that occurs during apneas has been suggested to stimulate enhanced EPO production [25]. The decreased kidney blood flow induced by apneic vasoconstriction would result in local ischemic hypoxia, stimulating kidney EPO production. Similarly, obstructive sleep apnea increases the levels of EPO ($\times$1.6) and Hb (+18%) [24]. Given these findings, it is reasonable to assume that the intensive training of elite BHDs (e.g., 5–6 h/day, 6 days/week for 6 months) would provide sufficient stimulation to increase erythopoiesis, thereby increasing apnea times. The lower SaO_2 decrease found in trained divers after repeated apneas may account for the reduced oxygen delivery because of the diving response (bradycardia and vasoconstriction) and/or an increase in oxygen content [1].

Long term-effects

Another important consideration is the persistence of the performance gains. Most of the altitude exposure studies reported short-term effects (i.e., weeks). Repeated apneas increase Hct but this increase disappears within 10 min after the last apnea [22,26]. To boost performance in sports like swimming, repeated apneas would thus need to be performed just before entering the pool. Furthermore, splenic contraction might also have a role in the fast adaptation to altitude or other ambient hypoxic conditions, serving as a compensatory mechanism by bridging the gap between non-acclimatization and altitude-induced polycythemia [23]. Repeated apneas might be an easy method to boost immediate performance. The effects of repeated apneas on spleen and endogenous EPO may also constitute an alternative to using rhEPO or its analogues. In addition, comparison of resting Hb mass in elite BHDs and untrained subjects showed a 5% higher Hb mass in the BHDs, and the BHDs also showed a larger relative increase in Hb after three apneas (2.7%). The long-term effect of apnea training on Hb mass might be implicated in elite divers' performances. Recently, it has been found that after a 3-month apnea training program, the forced expiratory volume in 1 s was higher (4.85 ± 0.78 vs. 4.94 ± 0.81 L, $p < 0.05$), with concomitant increases in the maximal oxygen uptake, arterial oxygen saturation, and respiratory compensation point values during an incremental test [30]. Swimming performance was not improved (clean velocity and time on 50 m); however, stroke rate was decreased and stroke length and the index of coordination (IdC) based on the time gap between the propulsive phases of each arm [31], were increased. These results indicate that apnea training improves effectiveness at both peak exercise and submaximal exercise and can also improve swimming technique by promoting greater propulsive continuity. In addition to increasing EPO and provoking splenic contraction, apnea training has been hypothesized to modify muscle glycolytic metabolism. An improvement in muscle buffer capacity [6,7,32] would reduce blood acidosis and post-apnea oxidative stress [6]. Delayed acidosis would also be advantageous for exercise performance. Finally, trained BHDs exhibit high lung volumes [15]. Apnea training might be interesting to improve respiratory muscle performance [15], thereby delaying the respiratory muscle fatigue during prolonged and maximal exercise.

Preconditioning effects

Elite BHDs are able to maintain very long apnea, inducing severe hypoxaemia without brain injury or black-out. It has thus been hypothesized that they develop protection mechanisms against hypoxia, as well as a decrease in overall oxygen uptake [33]. Hypoxia preconditioning refers to a period of hypoxia followed by a period of time during which there is a protection against asphyxia [34]. The BHD's apnea response could be the result of an enhancement of the 'diving reflex' but also a hypoxia preconditioning effect. Greater cerebral blood flow (CBF) increase was described during long apnea in elite BHDs than in controls and interpreted as a protection of the brain against the alteration of blood gas [33]. The CBF increase observed in BHDs could be the result of an increased capillary density in the brain as has been described after a prolonged hypobaric hypoxia exposure [35]. These results suggest that apnea training per se provides hypoxic preconditioning, increasing hypoxemia and ischemia tolerance [33].

Conclusions

The physiological responses to apnea training exhibited by elite breath-hold divers may contribute to improving sports performance. These adaptations may be an effective alternative to hypobaric or normobaric hypoxia to increase performance. Further experimental research of the apnea training effects on aerobic and/or anaerobic performance are needed to confirm this theory.

Conflicts of interest statement

None declared.

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