# BLOOD OXYGEN SATURATION AND HEART RATE DURING EXERCISE A MONTH AFTER A HIGH-ALTITUDE ALPINIST EXPEDITION

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Original scientific paper
UDC 576.8.095:613.11:796.015.576

**Abstract:**

Some alterations, reached during acclimatization on a high-altitude alpinist expedition, return towards pre-altitude levels at different rates during the deacclimatization phase after returning to normoxia. The aim of the study was to ascertain whether three specific adaptation characteristics – alterations in body weight (BW), arterial blood oxygen saturation ($SaO_2$) and heart rate (HR), showed that deacclimatization was completed a month after having reached the summit of Gasherbrum II. Four experienced alpinists (age 45±8 years, BW 75±5 kg and body height 171±9 cm) were tested in the conditions of normoxia and normobaric hypoxia prior to and following a month of deacclimatization after a high-altitude climbing expedition. The results showed that BW was still decreased by about 8 kg (p<.05) a month after reaching the summit. Data obtained during exercise with similar absolute intensity of 202±28 W showed that $SaO_2$ rose from 89±1 to 91±1% (p<.05) during testing in conditions of hypoxia after the high-altitude expedition. When the data were analysed at the same relative intensity of 2.7±.2 W/kg, the $SaO_2$ showed an even higher increase to 92±1 % during testing in conditions of hypoxia (p<.05). HR remained similar during testing at a similar absolute intensity in conditions of normoxia (153±19 $min^{-1}$ pre- vs 154±15 $min^{-1}$ post-expedition) and hypoxia (155±21 $min^{-1}$ pre- vs 158±20 $min^{-1}$ post-expedition), however, at the reduced BW during post-expedition testing. HR decreased significantly (p<.05) during similar relative intensity in conditions of hypoxia (155±21 $min^{-1}$ pre- vs 145±19 post-expedition). HR decreased systematically in all subjects in spite of the fact that it was not significantly different during testing in normoxic conditions. The results showed that deacclimatization, which was observed during submaximal intensity exercise in normoxic conditions and hypoxia, was not completed even a month after the high-altitude alpinist expedition.

***Key words:*** *normoxia, hypoxia, high altitude, exercise, deacclimatization*

## Introduction

Hypoxia and its dramatic influence on the performance of alpinists are the main research interest in the field of high-altitude climbing (Åstrand, Rodahl, Rahl, & Strom, 2003; Hornbein & Schoen, 2001). The decrease of a partial pressure of oxygen ($PO_2$) in the air also reduces the partial pressure in arterial blood ($PaO_2$) and thus the saturation of oxygen in arterial blood ($SaO_2$). Hypoxia occurs due to the reduced delivery of oxygen to the tissues. The physical and mental performance of climbers during high-altitude expeditions therefore dramatically decreases. Acclimatization has been recognized as the single most important factor of successful climbing in such conditions (Åstrand, et al., 2003; Hornbein & Schoen, 2001). The acclimatization process significantly improves performances during exercise at a high altitude. The organism dramatically responds to this situation by an increased $V_E$, which enhances blood saturation with oxygen in spite of the decreased $PaO_2$ (Vogel & Harris, 1967; Lahiri & Cherniak, 2001; Smith, Dempsey, & Hornbein, 2001). The classical concept suggests that cardiovascular system also improves its functioning through several adaptations (Roach, 2000; Vogel & Harris, 1967; Groover, & Bartsch, 2001), which leads to an improved oxygen delivery and uptake in muscles affected by hypoxia. In contrast, the reduction of muscle tissue mitochondria has been found (Hoppeler, Voght, Weibel, & Fluck, 2003) after high-altitude alpinist expeditions and in natives who have lived in a high-altitude environment for generations.

During deacclimatization, alpinists return from a high altitude hypoxia to their native sea-level normoxia. Several functions respond to increased $PO_2$ quickly - $PaO_2$ and $SaO_2$ increase (Åstrand, et al., 2003). Increased blood haemoglobin and erythrocytes cause an increase in blood oxygen content ($CaO_2$). Therefore, $V_E$ during resting and submaximal exercise initially decreases rapidly, followed by slower dynamics (Åstrand, et al., 2003). Some readaptations seem to be relatively slow, for example, structural changes in the muscles (Green & Sutton, 2001; Hoppeler & Voght, 2001). Deacclimatization is therefore not a simple reverse process of acclimatization (Hornbein & Schoen, 2001). Some exercise responses retain and remain different even after a longer period after acclimatization to a high altitude and/or are quickly reacclimatized if subjects are again introduced to altitude (Beidleman, et al., 1997). Deacclimatization may be observed in two possible ways: during normoxia at sea level and during a reintroduction to altitude (hypoxia). This reintroduction has two possible ways acute hypoxia testing after a certain period of deacclimatization or reacclimatization to hypoxia after a certain period of deacclimatization. The aim of our study was to ascertain the phenomenon more precisely amongst a group of alpinists who participated in the Gasherbrum II high-altitude alpinist expedition. They experienced extreme physiological, environmental and psychological stress whose effects can persist for a long period of time and influence the basic responses of the organism during exercise. The response during deacclimatization may be different in a study at sea-level normoxia or reintroduction to altitude hypoxia (reacclimatization). Therefore, both experimental designs were selected in our study. Heart rate (HR) increases because of hypoxia, which enhances the sympatoadrenal system activity (Hainsworth, Drinkhill, & Rivera-Chira, 2007; Roach, 2000), but decreases because of the reduced sympatoadrenal activity and influence on the parasympathetic neural activity during the acclimatization phase (Beidleman, et al., 1997; Boushel, et al., 2001) and due to the possible training effect on the cardiovascular system (Åstrand, et al., 2003). The existence of the training effect can be observed by measuring the HR during exercise in conditions of normoxia prior to and following a month of deacclimatization. By observing the HR during exercise and in hypoxic conditions it is possible to estimate whether some acclimatization effects are still present in addition to the training effect. These are two reasons why HR was selected as a variable of interest. High altitude exposure causes severe hypoxia - decrease of blood oxygen saturation ($SaO_2$). The effect of acclimatization is the opposite process: during hypoxic conditions $SaO_2$ increases. We have measured $SaO_2$ to estimate the possible differences occuring in hypoxic conditions before and after the expedition. We tested the hypothesis that a certain significant difference between the values after a month of deacclimatization and the values measured before the high-altitude expedition should exist in body weight (BW), HR, and $SaO_2$ during exercise, which may indicate that a certain level of acclimatization still existed.

## Methods

Four experienced alpinists (age 45±8 years, BW 75±5 kg and body height 171±9 cm) voluntarily participated in an experiment with the approval of the National Ethics Committee. All reached the summit of Gasherbrum II (8,032 m) on the same day.

A week before their departure, all subjects completed the initial testing protocol in normobaric hypoxia, after sleeping a night in the same hypoxic conditions. For this purpose, sleeping and testing were carried out in a room with controlled environment conditions (“a nitrogen house”). Each subject participated in four incremental tests on a cycle ergometer (Figure 1). Nobody had regularly performed any kind of cycling training.

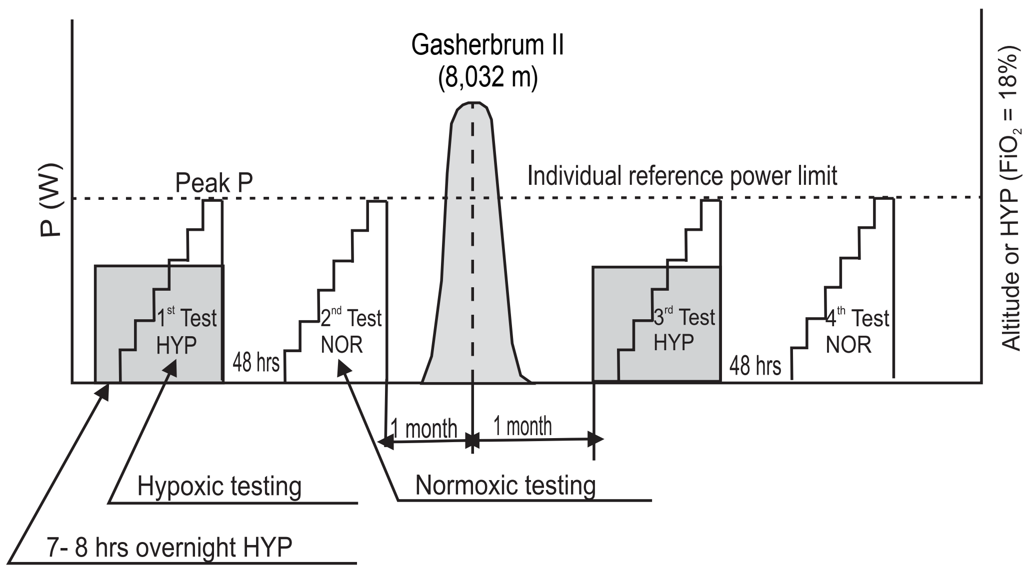


*Figure 1. A schematic presentation of experiment. See text for explanations.*

The first test was a continuous incremental test on a cycle ergometer in conditions of ambient normobaric hypoxia (HYP) ($FiO_2$=18%) (Figure 1). The test was performed after an overnight sleep in the same ambient conditions (HYP). It consisted of 4 minute steps of the exercise starting at 60 W and increasing by 40 W until voluntary fatigue. Subjects were quite motivated for testing and none of them needed additional encouragement from laboratory personnel during testing. The peak exercise intensity, which was reached during presented conditions before the expedition (PRE-HYP), was selected as the individual reference power limit (IRPL) for the three other incremental tests at which exercise should be stopped. After two days, the subjects performed the same test but in conditions of normoxia (PRE-NOR) (Figure 1). The duration of the expedition in the ascending phase (acclimatization) was one month (Figure 1). In this period, the alpinists climbed to the base camp at an altitude of 5,100 m within seven days. Over the next three weeks they finally acclimatized by preparing altitude camps and reached the summit after that. The descending phase (deacclimatization) lasted 10 days, including a few days of trekking over a small region, while the final two weeks were spent travelling and relaxing until the final testing (Figure 1). Therefore, this was carried out about a month after reaching the summit of Gasherbrum II.

The third test was performed one month after reaching the summit of Gasherbrum II (Figure 1). This test was a repetition of the first test in conditions of hypoxia, also after a night sleeping in conditions of normobaric hypoxia (POST-HYP). The fourth test was a repetition of the second one in normoxic conditions (POST-NOR) (Figure 1).

During the tests the blood gas $PO_2$ was measured by an ABL 5 analyser (Radiometer, Denmark). Capillary blood samples were obtained by a micro puncture of the hyperemied ear lobe. Arterial blood $O_2$ saturation ($SaO_2$) was measured by a TrueSat Pulse Oxymeter (Patex-Ohmeda, Lousville, USA). Heart rate (HR) was measured by Vantage NV heart rate meters (Polar, Finland) by averaging signals every 10 s.

The analysis of the data consisted of: 1) the individual data analysis (logical observations) of differences and/or similarities was used for the assessment of possible effects of high-altitude living and climbing, and 2) of a paired *t*-test applied to ascertain any statistically significant differences by Sigma Plot 11 and Sigma Stat 3 (Systat Software Inc, Germany).

## Results

The alpinists reached the peak absolute intensity of 202±29 W (2.7±.2 W/kg) during the initial testing in conditions of normobaric hypoxia (PRE -HYP) (Table 1). One subject reached 240 W, one 210 W and two 180 W. These were their individual reference power limits used in further tests. The $SaO_2$ reached 89±1% during hypoxic testing ($PO_2$ ranged between 8-9 kPa) (Figure 2). HR reached

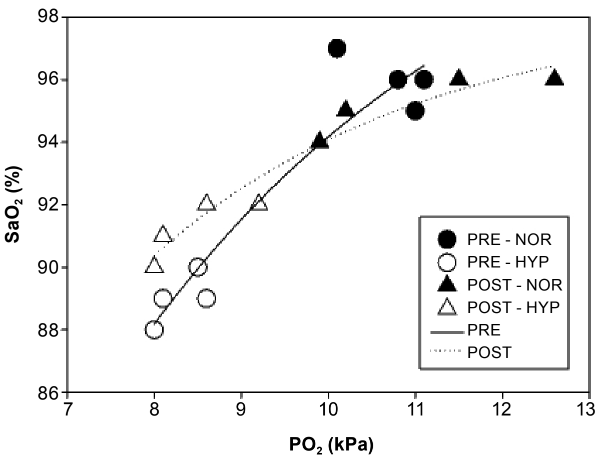


*Figure 2. The dependence of arterial oxygen saturation ($SaO_2$) on its partial pressure ($PO_2$) at a similar absolute exercise intensity on a cycle ergometer. Data before the high-altitude expedition in conditions of normoxia (PRE-NOR) did not differ from $SaO_2$ after the expedition (POST-NOR). In contrast, values during the hypoxic testing after the expedition (POST-HYP) showed a clear enhancement in comparison to $SaO_2$ before the expedition (PRE-HYP).*

*Table 1. Values achieved at a similar absolute intensity during an incremental testing protocol before and after a high-altitude alpinist expedition*

| | P (W) | Pr (W/kg) | HR ($min^{-1}$) | $PO_2$ (kPa) | $SaO_2$ (%) |
|---|---|---|---|---|---|
| PRE – NOR | 202±29 | 2.7±.2 | 153±19 | 10.7±.5 | 96±1 |
| PRE – HYP | 202±29 | 2.7±.2 | 155±21 | 8.3±.3 * | 89±1 * |
| POST – NOR | 202±29 | 3.0±.1 † | 154±15 | 11.0±1.2 | 95±1 |
| POST – HYP | 202±29 | 3.0±.1 †† | 158±20 | 8.5±.5 * | 91±1 * †† |

* denotes p<.05 between values measured PRE-NOR and PRE-HYP
** denotes p<.05 between values measured POST-NOR and POST-HYP
† denotes p<.05 between values measured PRE-NOR and POST-NOR
†† denotes p<.05 between values measured PRE-HYP and POST-HYP

was 153±19 min$^{-1}$ (Table 1). One of the subjects reached lower HR than the others (Figure 3).

During testing in normoxic conditions (PRE-NOR) two days after the first testing, all the subjects reached their individual target intensity as expected. In these conditions the values of $SaO_2$ (94-97%) were higher than the values during PRE-HYP testing by about 7% (p<.05) (Table 1, Figure 2). HR achieved was 155±21 min$^{-1}$, which is comparable to the values from the PRE-HYP testing (Table 1). For the subjects who achieved a lower HR during PRE-HYP testing a similar phenomenon could be observed during the PRE-NOR test. Three subjects showed a clear trend of decreasing their HR in normoxic conditions at the individual reference power limit in comparison to the results obtained during the previously made test in hypoxic conditions (Figure 3).

A month after the high-altitude expedition, all the subjects showed a significant reduction of their BW by about 8 kg (p<.05). Their BW reached 67±19 kg instead of 75±10 kg, which they had before the expedition (Table 1). Due to this phenomenon a similar absolute exercise intensity (P) became the higher relative exercise intensity (Pr), by about 0-3 W/kg (p<.01) (Table 1). All subjects reached their individual target exercise intensity in conditions of normobaric hypoxia (POST-HYP) during the third test. Therefore, their exercise performance on the cycle ergometer remained similar to their performance before the expedition. For $SaO_2$ (91±1%) increased values (p<.01) were obtained at the similar absolute exercise intensity in conditions of normobaric hypoxia (POST-HYP) compared to the PRE-HYP values (Table 1). Each subject reached higher values (Figure 2). In contrast, no alterations were observed for HR either during testing in normoxic or hypoxic conditions (Figure 3).

At the similar relative exercise intensity (Pr) of about 2.7 W/kg HR was significantly reduced (p<.05) during testing in conditions of normobaric hypoxia compared to the HR values before (155±21 min$^{-1}$) and after the expedition (145±19 min$^{-1}$) (Table 2). The pattern of changes of all individual data showed a similar decrease of HR (Figure 4). Comparison of HR during normoxic testing before (PRE-NOR) and after (POST-NOR) the expedition showed a similar pattern as during hypoxic testing, however, without reaching a level of significance (Table 2, Figure 4). $SaO_2$ increased (p<.05) during testing at Pr in hypoxic conditions (92±1%

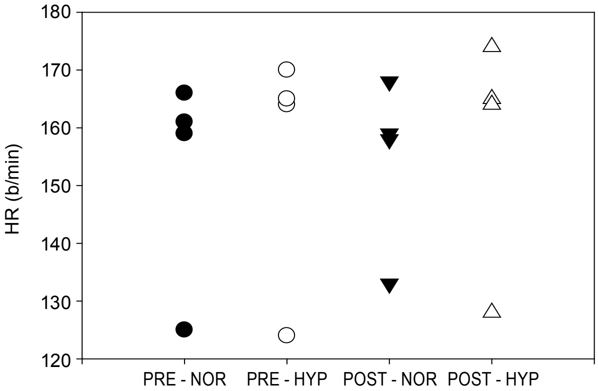


*Figure 3. The high-altitude alpinist expedition did not affect HR in conditions of normoxia (PRE-NOR vs POST-NOR). During hypoxic testing, the picture was similar. It should be considered that these alterations occurred at the similar absolute exercise intensity, but at the reduced BW.*

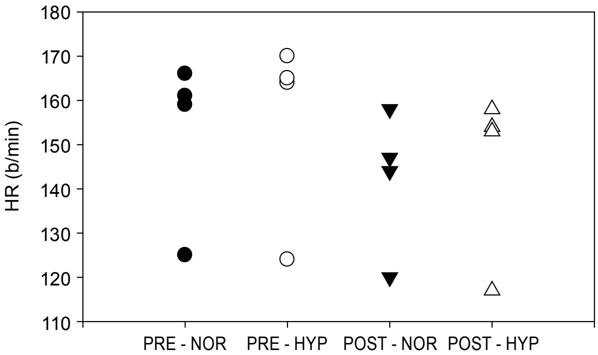


*Figure 4. Individual values at the similar relative exercise intensity on a cycle ergometer. The expedition decreased HR in all subjects during testing in conditions of normoxia (PRE-NOR vs POST-NOR). During hypoxic testing, all subjects significantly (p<.05) decreased their HR (PRE-HYP vs POST-HYP).*

*Table 2. Values achieved at the similar relative exercise intensity in incremental testing before and after the high-altitude alpinist expedition.*

| | P (W) | Pr (W/kg) | HR (min$^{-1}$) | $PO_2$ (kPa) | $SaO_2$ (%) |
|---|---|---|---|---|---|
| PRE – NOR | 202±29 | 2.7±.2 | 153±19 | 10.7±.5 | 96±1 |
| PRE – HYP | 202±29 | 2.7±.2 | 155±21 | 8.3±.3 | 89±1 * |
| POST – NOR | 175±26 | 2.6±.2 | 142±16 | 11.5±1.1 | 96±2 |
| POST – HYP | 175±26 | 2.6±.2 | 145±19 †† | 8.5±.5 | 92±1 * †† |

* denotes p<.05 between values measured PRE-NOR and PRE-HYP
** denotes p<.05 between values measured POST-NOR and POST-HYP
† denotes p<.05 between values measured PRE-NOR and POST-NOR
†† denotes p<.05 between values measured PRE-HYP and POST-HYP

POST-HYP) in comparison to the values reached before the expedition (89±1% PRE-HYP) (Table 2, Figure 5).

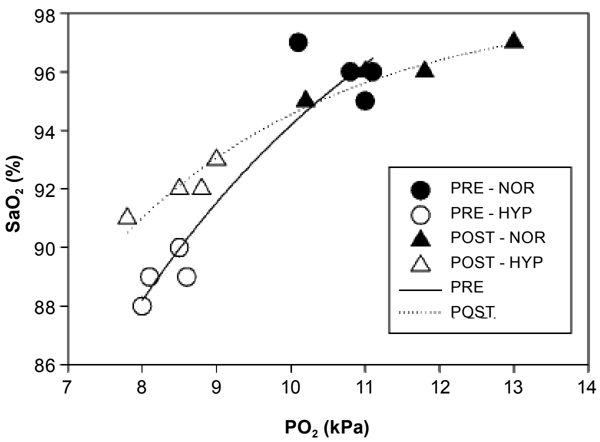


*Figure 5. The dependence of arterial oxygen saturation ($SaO_2$) on its partial pressure ($PO_2$) at a similar relative exercise intensity on a cycle ergometer. Data before the high-altitude expedition in conditions of normoxia (PRE-NOR) did not differ from $SaO_2$ after the expedition (POST-NOR). In contrast, values during hypoxic testing after the expedition (POST-HYP) showed a clear enhancement in comparison to $SaO_2$ before the expedition (PRE-HYP).*

## Discussion

The main finding of this study is that deacclimatization was still not completed one month after the high-altitude alpinist expedition. A still increased blood oxygen saturation during hypoxic exercise as well as reduced BW supported this conclusion. Unchanged HR reached at the similar submaximal absolute intensity in spite of the reduced BW and decreased HR under similar relative exercise intensity loadings may also support the previous conclusion.

In available literature it is very rare that the period of deacclimatization in combination with detraining is observed for such a long period as one month, as was the case in our study. With this experiment we sought to discover whether certain physiological alterations, caused by hypoxia, may persist a month after the high-altitude expedition. In addition, hypoxia (reacclimatization) which was reintroduced after the period of deacclimatization and included a night before testing (in these conditions) has not yet been used in any experimental design. With this experiment we tried to trigger early reacclimatization. This seems to be interesting especially in the simulated situations: re-exposure to hypoxic conditions following the initial acclimatization and deacclimatization phases. These situations are frequent during military operations and alpine rescue activities in high-altitude mountains.

High-altitude alpinist expeditions have been characteristically accompanied by significant reduction in BW (Boyer & Blume, 1984; Hoppeler & Voght, 2001). This phenomenon occurs dramatically during high altitude exposure due to several factors as reported by Boyer and Blume (1984). Acute dehydration occurs in the first phase because of loss of appetite (Boyer & Blume, 1984). Additionally, interstitial malabsorption of carbohydrates and fats lead to enhanced catabolism (Boyer & Blume, 1984). At extreme altitude, significant muscle wasting occurs irrespective of body composition (Bales, et al., 1993). Nitrogen balance shows that nitrogen excretion increases due to increased oxidation of glycine as a function of altitude exposure (Schols & Westerterp, 2002). Smaller muscle mass may provide an advantage for oxygen transport via an increase in capillary density associated with smaller muscle fibres (Green & Sutton, 2001). However, due to a reduced mitochondria density (Hoppeler & Voight, 2001), it is controversial whether the result of these alterations is an adaptation which improved oxygen delivery and uptake during hypoxia or not. We can assume that reduced BW was one important factor which reduced endurance performance soon after returning to sea level. It seems that even a partial regain of BW had a significant effect on the restoration of endurance performance during the month of deacclimatization. Therefore, the dynamics of regaining BW and endurance performance in the process of returning to the pre- a high-altitude expedition levels seem to be different.

Similarly, $SaO_2$ increased during hypoxic testing at similar absolute and relative exercise intensities, when it was compared prior to and following one month of deacclimatization from the high-altitude alpinist expedition. Therefore, two possible influences may affect a higher oxygenation of blood at a similar $PaO_2$. The increase of blood $O_2$ content due to an increased haemoglobin concentration and haematocrit (Åstrand, et al., 2004) could be the first influence. However, we do not know whether any significant alterations that had occurred during the high-altitude alpinist expedition in haemoglobin concentration and haematocrit persisted during the month of deacclimatization. The second influence could be related to the reacclimatization phase during overnight sleep in normobaric hypoxia conditions preceding the hypoxic testing. It can be speculated that overnight reacclimatization a month after the high-altitude expedition could be enhanced in relation to the pre- a high-altitude expedition due to an enhanced hypoxic ventilatory response (HVR), which caused blood alkalosis and increased blood oxygen saturation in hypoxic conditions (Smith, et al., 2001)

Heart rate (HR) values were similar before the high-altitude alpinist expedition and after one month of deacclimatization when it was observed during normoxic and hypoxic testing at the similar absolute exercise intensity in spite of the approximately 8 kg reduction of BW. Because the increased relative exercise intensity has not been accompanied by an adequate increase in HR values, we have assumed that a certain training effect of climbing still existed. This assumption can be also used in the explanation of HR decrease at the similar relative exercise intensity when comparing data before and after the alpinist expedition. Results gained by the testing under normobaric hypoxia conditions did not show any different HR alterations in comparison to those gained by the testing under normoxic conditions when they were compared before and after the expedition. Therefore, we assume that acclimatization was not a part of the phenomenon any more.

Because the main limitation of the study is the low number of subjects involved (four), the conclusions presented here need to be viewed with caution. Nevertheless, our results clearly support the idea of the importance of body mass reduction in the enhancement of certain physiological responses. This idea should be seriously incorporated in the discussion of endurance performance during deacclimatization at similar relative and absolute exercise intensities. It should also be incorporated in the explanation of HR results obtained during exercise after the high-altitude expedition. The second limitation of the study was the application of capillary blood sampling for the $PO_2$ measurements. As can be observed from the $PO_2$ values shown in Figure 2 and Figure 5, capillary blood tends to show lower values than arterial blood due to a possible mixing of venous capillary blood and extracellular fluid with arterial blood in the samples obtained during exercise in spite of the hyperemied ear lobe. As regards the accurate measurements of $PO_2$, it is necessary to obtain arterial blood samples. However, this deficiency does not significantly influence the results nor the meaning of this study.

In conclusion, it has been previously shown that exercise response, reached during acclimatization, returns towards pre-altitude levels at different rates during deacclimatization. Our results showed that deacclimatization, which was observed during sub-maximal intensity exercise in normoxic conditions and hypoxia, had not been completed even one month after the high-altitude alpinist expedition. Increased blood oxygen saturation during hypoxic exercise showed acclimatization effect. Reduced body weight showed a complex and prolonged effect of acclimatization, exercise training and changed nutrition. Heart rate, which was unaffected by the increased relative exercise intensity (due to the reduced body weight) in normoxic conditions showed that certain training effects still existed. Similar HR observed during testing in hypoxic and normoxic conditions showed that acclimatization effect was not a part of the phenomenon any more.

## References


Antoni, H. (1984). Function of the heart. In R.F. Schmidt & G.Thews (Eds.), *Human Physiology* (pp. 358-396). Berlin: Thieme.

Åstrand, P.O., Rodahl, K., Rahl, H.A., & Strom, S.B. (2003). *Textbook of work physiology* (pp. 441-445). Champaign, IL: Human Kinetics.

Bales, B., Hackney, A.C., Coyne, J.T., Shaw, E., McAninch, G., Kramer, A., & Brownsberger, R. (1993). Mountaineering sojourn: Effects on body composition of prolonged exposure to high altitude in a cold environment. *Journal of Wilderness Medicine*, *4*, 32-39.

Beidleman, B., Muzo, S.R., Pack, P.B., Fulco, L.C., Lyons, T.P., Haty, R.W., & Cymerman, A. (1997). Extreme responses after altitude acclimatization are retained during reintroduction to altitude. *Medicine and Science in Sports and Exercise*, *19* (11), 1588-1595.

Boushel, R., Calbet, J-A. L., Radegram, G., Sandergaard, H., Wagner, P.D., & Saltin, B. (2001). Parasympathetic neural activity accounts for the lowering of exercise heart rate at high altitude. *Circulation, 104,* 1785-1791.

Boyer, S.J., & Blume, F.D. (1984). Weight loss and changes in body composition at high altitude. *Journal of Applied Physiology*, *57*(5), 1580-1585.

Green, H.J., Sutton, J.R. (2001). The effects of altitude on skeletal muscle. In T.H. Hornbein & R.B. Schoene (Eds.), *High altitude – An exploration of human adaptation* (pp. 443-492). New York: Marcel Dekkerr.

Groover, R.F., & Bartsch, P. (2001). Blood. In T.H. Hornbein & R.B. Schoene (Eds.), *High altitude – An exploration of human adaptation* (pp. 493-523). New York: Marcel Dekkerr.

Hainsworth, R., Drinkhill, M.J., & Rivera-Chira, M. (2007). The autonomic nervous system at high altitude. *Clinical Autonomic Research, 17,* 9-13.

Hoppeler, H., & Voght, M. (2001). Muscle tissue adaptation to hypoxia. *The Journal of Experimental Biology, 204,* 3133-3139.

Hoppeler, H., Voght, M., Weibel, E., & Fluck, M. (2003). Response of skeletal muscle mitochondria to hypoxia. *Experimental Physiology, 88,* 111-119.

Hornbein, T.H., & Schoene, R.B. (2001). *High altitude – An exploration of human adaptation*. New York: Marcel Dekkerr.

Lahiri, S., & Cherniak, N.S. (2001). Cellular and molecular mechanisms of $O_2$ sensing with special reference to the carotid body. In T.H. Hornbein & R.B. Schoene (Eds.), *High altitude – An exploration of human adaptation* (pp. 101-130). New York: Marcel Dekkerr.

Roach, R.C. (2000). Cardiovascular regulation during hypoxia. In B. Saltin, R. Boushel, N. Secher & J. Mitschell (Eds.), *Exercise and circulation in health and disease* (pp. 177-294). Champaign, IL: Human Kinetics.

Schols, A.M.W., & Westerterp, K.R. (2002). Hypoxia, nitrogen balance and body weight. *European Journal of Applied Physiology*, *20*, 252-253.

Smith, C.A., Dempsey, J.A., & Hornbein, T.F. (2001). Control of breathing at high altitude. In T.H. Hornbein & R.B. Schoene (Eds.), *High altitude – An exploration of human adaptation* (pp. 139-173). New York: Marcel Dekkerr.

Vogel, J.A., & Harris, C.W. (1967). Cardiopulmonary response of a resting man during early exposure to high altitude. *Journal of Applied Physiology*, *22*(6), 1124-1128.

Vogel, J.A., Hansen, J.E., & Harris, C.W. (1967). Cardiovascular responses in man during exhaustive work at sea level and high altitude. *Journal of Applied Physiology*, *23*(4), 531-539.

Vogt, M., Puntschart, A., Geissen, J., Zuleger, C., Billeter, R., & Hoppeler, H. (2001). Molecular adaptations in human skeletal muscle to endurance training under simulated hypoxic conditions. *Journal of Applied Physiology*, *91*, 173-182.

Submitted: July 5, 2009
Accepted: October 1, 2009

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# RAZINA ZASIĆENOSTI KRVI KISIKOM I FREKVENCIJA SRCA TIJEKOM VJEŽBANJA MJERENE MJESEC DANA NAKON ALPINISTIČKE EKSPEDICIJE NA VELIKU NADMORSKU VISINU

Neke promjene fizioloških parametara, postignute aklimatizacijom tijekom alpinističke ekspedicije na veliku nadmorsku visinu, u fazi “deaklimatizacije” na povratku u normalne visinske uvjete, različitim se tempom vraćaju na razinu kakva je bila prije odlaska na ekspediciju. Cilj je ovog istraživanja bio utvrditi da li tri specifične karakteristike adaptacije na nadmorsku visinu – promjene u tjelesnoj težini, zasićenost arterijske krvi kisikom i frekvencija srca – pokazuju da je proces aklimatizacije završio mjesec dana nakon osvajanja vrha Gasherbrum II. Četiri iskusna alpinista (u dobi od 45±8 godina, tjelesne mase 75±5 kg i tjelesne visine 171±9 cm) testirana su u uvjetima normoksije i normobarične hipoksije prije alpinističke ekspedicije na veliku nadmorsku visinu i mjesec dana nakon povratka s ekspedicije. Rezultati su pokazali da je mjesec dana nakon osvajanja vrha tjelesna težina još uvijek bila niža za oko 8 kg (p<,05). Podaci prikupljeni tijekom vježbanja sličnim apsolutnim intenzitetom od 202±28 W u uvjetima hipoksije nakon ekspedicije pokazuju da se zasićenost arterijske krvi kisikom povećala sa 89±1 na 91±1% (p<,05). Analiza podataka zasićenosti arterijske krvi kisikom pri istom relativnom intenzitetu rada od 2.7±.02 W/kg pokazala je još veće povećanje (na 92±1%) tijekom testiranja u uvjetima hipoksije (p<,05). Frekvencija srca ostala je ista tijekom testiranja pri istom apsolutnom intenzitetu u uvjetima normoksije (153±19 o/min prije u odnosu na 154±15 o/min nakon ekspedicije) i hipoksije (155±21 o/min prije u odnosu na 158±20 o/min nakon ekspedicije), ali ne smijemo zaboraviti smanjenu tjelesnu težinu nakon ekspedicije. Frekvencija srca opala je statistički značajno (p<,05) tijekom vježbanja jednakim relativnim intenzitetom u uvjetima hipoksije (155±21 o/min prije u odnosu na 145±19 o/min nakon ekspedicije). Frekvencija srca se sustavno snizila u svih ispitanika, unatoč činjenici da se u uvjetima normoksije nije statistički značajno razlikovala. Rezultati su pokazali da “deaklimatizacija”, koja se procjenjivala vježbanjem submaksimalnim intenzitetom u uvjetima normoksije i hipoksije, nije završila čak ni mjesec dana nakon povratka s alpinističke ekspedicije na veliku nadmorsku visinu.

***Ključne riječi:*** *normoksija, hipoksija, velika nadmorska visina, vježbanje, deaklimatizacija*