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Effects of hypoxia on interval moderate exercise

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ABSTRACT

There is a controversy in the published scientific data whether extended training at altitude increases performance at sea level. The effect of hypoxia at rest and on the response to interval moderate exercise was determined in six healthy male individuals during an incremental 3×5 min exercise cycle test (5 min recovery) at sea level and in a hypobaric chamber (10000 feet/3100 m altitude). Ventilation rate (VE), breathing frequency (BF), heart rate (HR), cardiac output (Q), blood lactate (La_{bl}) and % of arterial oxygen saturation (SaO_2) were measured. Blood samples were drawn at rest and at the end of each exercise bout. Hypoxia led to a significant increase in VE during exercise (81.7 vs. 62, 87 vs. 66, 89 vs. 64 ml/l, for the three exercise bouts, respectively $p < .05$). There was also a significant increase in BF (11.3 vs. 10, 29 vs. 25, 32 vs. 24, 33 vs. 25, $p < .05$), HR (73 vs. 62, 153 vs. 138, 161 vs. 138, 166 vs. 137 b/min, $p < .05$), Q (4.8 vs. 4, 13.1 vs. 12.5, 14.6 vs. 12.2, 15.5 vs. 12.8 l/min, $p < .05$) and La_{bl} (0.41 vs. 0.35, 3.8 vs. 2.6, 4.7 vs 3, 5.9 vs 2.9 mmol/l, $p < .05$) at rest and during exercise. Hypoxia lowered SaO_2 at rest and exercise (99.3 vs 98.6, 98.1 vs 95.1, 98.5 vs 95.3, 98.3 vs 95.5%, $p < .05$). The results suggest that there is a hypoxic augmentation of the cardiorespiratory variables measured. Also we concluded that exercise potentiated the acute ventilatory response to hypoxia by increasing VE, breathing frequency, heart rate, cardiac output, blood lactate and decreasing SaO_2.

KEY WORDS: *hypoxia, lactate, breathing frequency, ventilation rate, heart rate, cardiac output, arterial oxygen saturation.*

INTRODUCTION

Altitude training is frequently used by competitive athletes in a wide range of sports in the belief that it will improve sea level performance (4). However, the published scientific data on performance increases at sea level after extended training at altitude are contradictory. While a number of studies have reported an improvement in sea level work performance and maximal oxygen uptake following exposure to high altitude, others have observed no change (10). When training is performed under hypoxic conditions, it induces muscular and systemic adaptations which are either absent or found to a lesser degree after training under normoxic conditions (13). Terrados et al. (12) demonstrated that when hypoxia is combined with exercise, significantly greater increases occur in oxidative enzyme activity and myoglobin than when the same training is performed in normoxia. Thus, it seems that training in hypoxic conditions may increase the «stimulus adaptation» and thereby magnify the normal sea level responses to training.

Conversely, altitude induced hypoxia may reduce the intensity at which athletes can train resulting in a relative deconditioning (Brosnan et al., 2000). Acute mountain sickness, problems with acclimatization and detraining due to decreased intensity are believed to influence the effectiveness of altitude training (13). One of the major factors that can reduce the potential beneficial effect of altitude training is the reduction in training workload. Due to this reduction in aerobic power, athletes, and especially elite ones, may not reach and sustain their normal training workloads during their stay at altitude (9). It has been proposed that interval training undertaken at even moderate altitude (2500m) would result in lower absolute work rates and/or speeds, with lower heart rates (HR) and blood lactate concentrations compared with those at sea level. Indeed, investigations that compared submaximal exercise of the same relative intensity reported higher heart rates, a reduced training pace and higher blood lactate concentrations Labl for exercise under hypoxic vs. normoxic conditions (4).

A reduction in environmental oxygen at high altitude induces hypoxemia in skeletal muscle, which, in turn, causes the limitation in exercise performance, although the cause-and-effect relationship for muscle-hypoxia limiting performance is debated (2). Ascent to high altitude is accompanied by an increase in minute ventilation (VE) and a decrease in arterial O_2 saturation (SaO_2) at rest (2). The increase in VE is caused by increases in tidal volume and respiratory frequency (14). Katayama et al. (8) have also reported that a sojourn at high altitude leads to increases in resting hypoxic ventilatory responses (HVR) accompanied by increases in pulmonary ventilation and SaO_2 at rest. Moreover, Engelen et al. (5) showed that hypoxia, which as it was said before reduces the percentage of O_2 in the arterial blood, reduces both peak O_2

uptake (VO_2 peak) and the lactic acidosis threshold. Indeed, several studies showed that all these metabolic responses are potentiated during exercise in a hypoxic environment. Nakajono et al. (11) showed that there was a 10.7% increase in VE during exercise in hypoxia compared to normoxia). Hogan et al. (7) have previously reported that during an incremental maximal test performed under hypoxic conditions (17% O_2) Labl was elevated at moderate-to high power output (200 w) compared with normoxia.

Therefore, the aim of the present investigation was to document the effects of reduced inspired percentage of O_2 (simulated moderate altitude at 10000feet/3100m) on indicators such as ventilation rate (VE), breathing frequency (BF), blood lactate concentration (Labl), arterial oxygen saturation (SaO_2), cardiac output (Q) and heart rate (HR) during rest and interval moderate exercise sessions that involved efforts of short duration (5 min) in a group of six healthy individuals. On the basis of the results of previous studies we expected an increase in VE, SaO_2, BF and Labl during both rest and exercise in the hypoxic environment. In addition, it was hypothesized that an increase in HR and Q during rest and exercise will be observed in the hypoxic environment compared to normoxia.

METHODS

Subjects

Six healthy non-smoking men with no history of cardiorespiratory diseases volunteered to participate in this study. Their mean age, body mass and height was 24 ± 2 years, 70 ± 3 kg and 170 ± 5 cm, respectively. Before giving their written consent to participate in the experiment, they were informed of the nature, the potential risks involved and the benefits of the study. Prior to the commencement of the main experiments, all subjects took part in a habituation session in order to familiarize themselves with the laboratory environment and testing procedures. The experiment received the approval of the Ethical Committee of the University. The subjects served as their own control by participating in two separate experimental trials.

Experimental Procedures

The experimental protocol required each subject to visit the laboratory three times. At each visit they cycled two identical exercise tests on an electronically braked cycle ergometer (Monarch). The order of the tests was counterbalanced and double blinded. All tests were performed with the subjects in the

upright position on the cycle ergometer. Seat and handlebar heights were held constant for each subject for all the tests. Before the experiment starts, subjects were familiarized with the equipment used in this experiment at sea level and the hypobaric chamber (3100m altitude). The tests were performed while subjects were breathing room air (21% O_2) or breathing hypoxic gas mixture equivalent of that at 10000 feet (3100 m) altitude. Before the intermittent exposure to altitude, the moderate exercise test was conducted at sea level.

Incremental exercise tests

Subjects were instructed to maintain their normal diets and to abstain from alcohol, caffeine and tobacco and from taking any medication before and during the experimental trials period. All tests started with 10 min at rest with subjects breathing room air. The subjects started exercising at unloaded cycling for 5 min for warming up. Then, they performed 5min exercise bouts on the cycle ergometer at a power output of 200 W repeated three times with a 5 min break between each bout on two separate occassions; under normoxic and hypoxic (10000 feet altitude) environment. The tests were conducted at the same time of day (in the morning), the same day of the week and care was taken to ensure that the exercise procedures and environmental conditions (ambient temperature 22-23℃ and relative humidity 50-55%) did not differ during the two trials.

Measurements

All measurements were taken at rest and during the last minute of each exercise bout both in normoxia and hypoxia condition. Percentage of arterial O_2 saturation (SaO_2) was monitored during the two interval exercise sessions using Criticom dynamap (Criticom, Australia). Ventilation rate (VE) and breathing frequency (BF) were measured using an online SensorMedics 2900 gas analysis system (SensorMedics, Netherlands). Heart rate (HR) and cardiac output (Q) were measured using a portapres system (Polar PE 3000, Finland). Capillary blood was drawn at rest and at the end of each bout for the determination of Labl using a lactatepro system (Lactatepro, UK).

Statistical analyses

The effects of simulated altitude exposure at rest and during the incremental moderate exercise on Labl, HR, Q, VE, BF, and SaO_2 were analysed using a two-way (sea level-altitude) ANOVA with repeated measures (rest and three interval exercise bouts). Values are expressed as means. Specific mean

comparisons of interest were evaluated by using a priory planned contrasts. Statistical significance was accepted when $p < .05$. SPSS data analysis software was used for the statistical analysis of the data.

RESULTS

All subjects completed the prescribed interval exercise sessions both in normoxia and hypoxia. Tables 1-6 display the Means ± SD from all six variables measured (VE, BF, Q, HR, Labl and SaO_2) during rest and at the end of each of the three bouts, at sea level and in hypoxia. The main effect of altitude for each set of intervals calculated by using a two-way ANOVA with repeated measures and the Mauchly's test of sphericity has been checked to ensure that the assumption of sphericity has been met.

As anticipated, hypoxia (10000 feet altitude) led to significant changes in all variables measured. Also, there was a significant main effect to all variables in transition from rest to exercise.

Ventilation rate

VE was significantly higher in hypoxia compared to normoxia [$F(1,5) = 41.3$, $p < .05$]. Moreover, VE was significantly higher during exercise in hypoxia compared to normoxia (81.7 vs. 62, 87 vs. 66, 89 vs. 64 ml/l, for the three exercise bouts, respectively). Also, there was a significant increase in VE between bout 1 and bout 2 and over time (between the first and third bout) which was observed only in the hypoxia condition (Figure1).

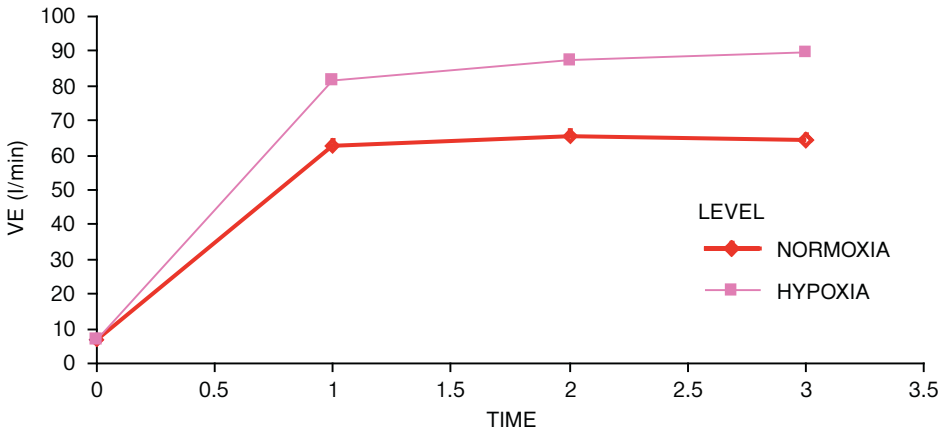

Figure 1: *Average VE (l/min) at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

Breathing frequency

BF was significantly higher in hypoxia compared to normoxia, [$F(1,5) = 379$, $p < .05$] both at rest and after each of the three work bouts. In contrast, there was no difference between BF during the three exercise bouts, either over time (Figure 2).

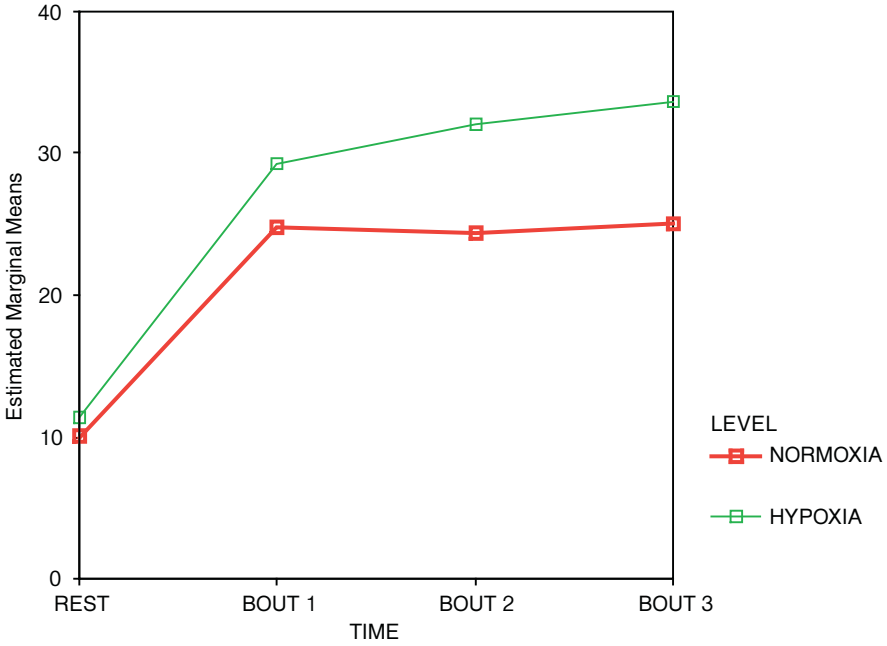

Figure 2: *Average breathing frequency at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

Cardiac output/Heart rate

Q was significantly higher in hypoxia compared to normoxia [$F(1,5) = 252$, $p < 0.05$]. HR was significantly higher in hypoxia too [$F(1,5) = 73.8$, $p < 0.05$]. Q was significantly higher in hypoxia at rest and during exercise and a significant difference between Q over time (between first and third bout) was also found. HR was also higher in hypoxia at rest and during exercise and it continued to increase during exercise at altitude. (Figure 3 and 4).

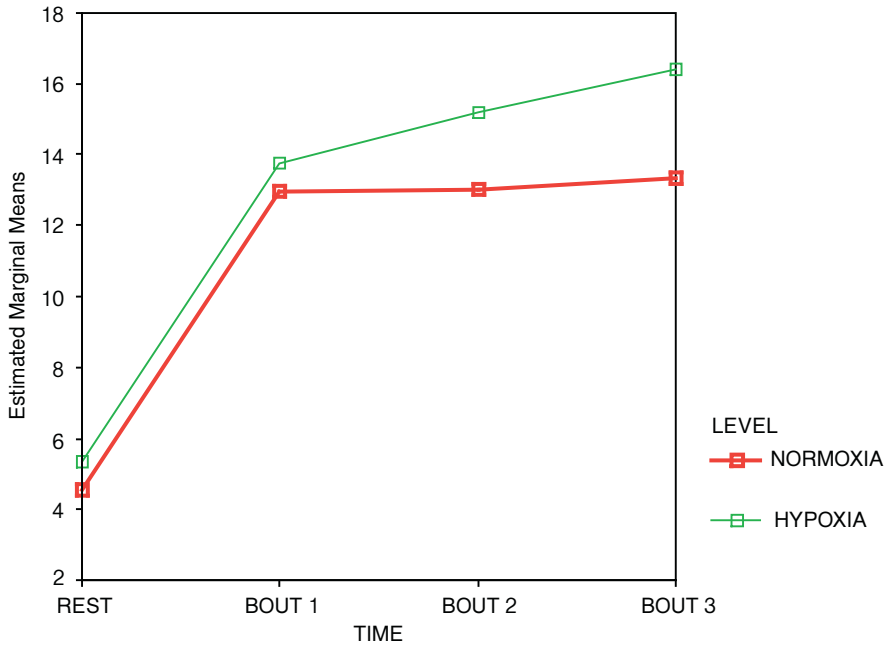

Figure 3: *Average cardiac output (l/min) at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

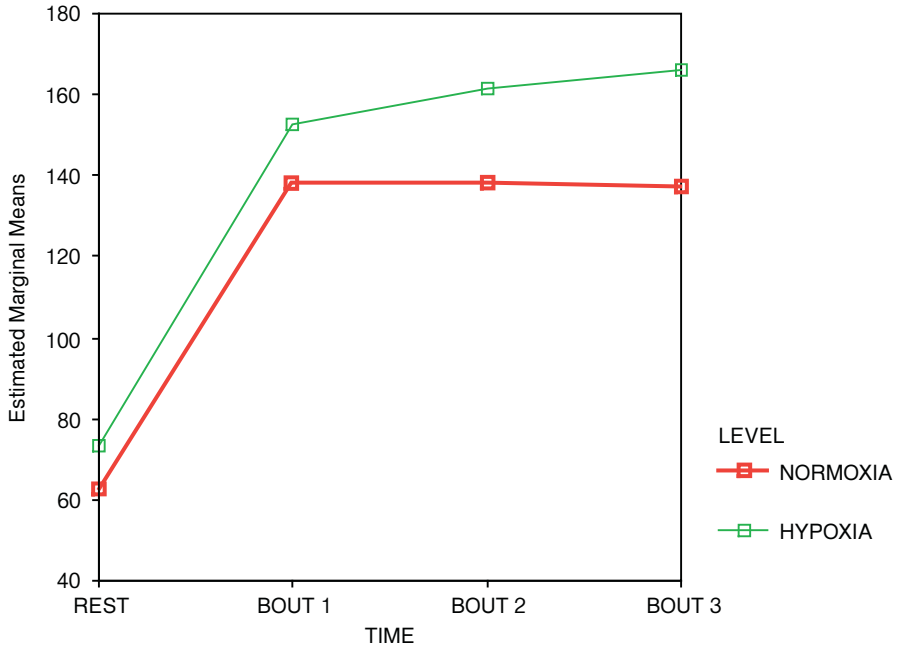

Figure 4: *Average heart rate at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

Lactate

Hypoxia resulted in a significant increase in [La]bl at rest and at the end of each exercise bout. Labl was also significantly higher in hypoxia compared to normoxia. [$F(1,5) = 152$, $p < 0.05$] (Figure 5).

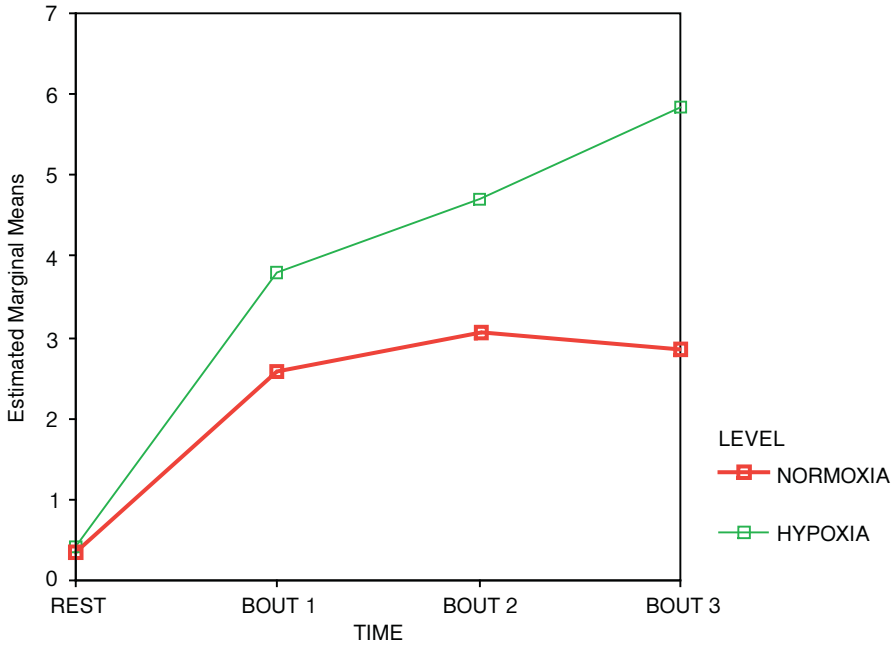

Figure 5: *Average La_{bl} at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

Arterial O_2 saturation

SaO_2 was significantly lower under hypoxia than normoxia, [$F(1,5) = 95.6$, $p < 0.05$ at rest and after the end of each exercise bout. A significant decrease between transitions from rest to exercise was observed. SaO_2 declined progressively over time during exercise in hypoxia (Figure 6).

The environmental condition (sea level vs. altitude) – exercise stage (rest, bout 1, bout 2, bout 3) interaction was significant in all variables measured, indicating that the effect of rest and exercise bouts differed in hypoxia compared to normoxia.

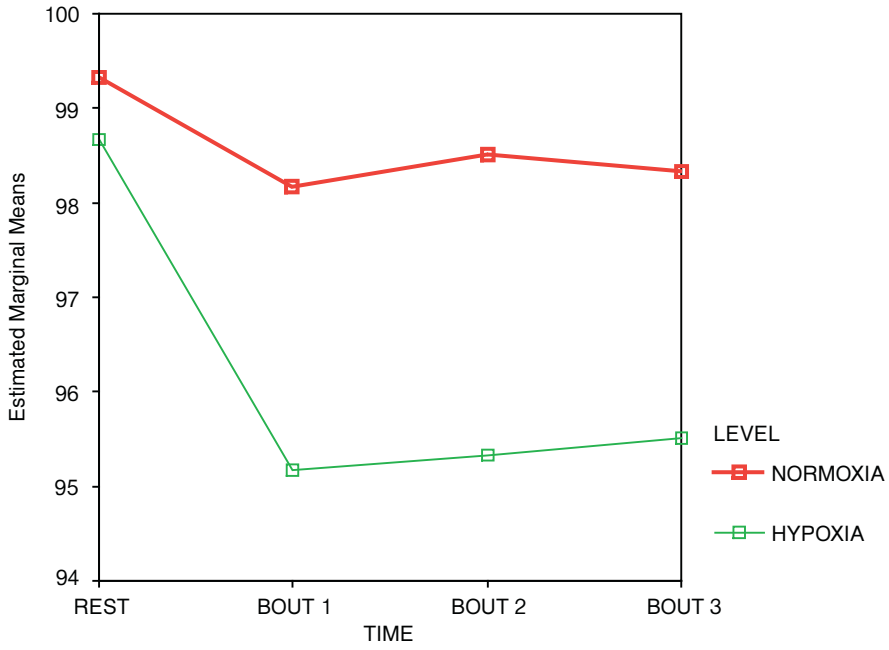

Figure 6: *Average arterial oxygen saturation at rest and during the three exercise bouts in normoxia and hypoxia, $p < .05$ ($n = 6$).*

Table 1. *Venilation rate (VE) means ± SD at normoxia and hypoxia condition.*

$[La]_{bl}$ (mmol/l)	Sea level	138.80 + 4.39	138.80 + 4.39	138.80 + 4.39	138.80 + 4.39
		Rest	**Bout 1**	**Bout 2**	**Bout 3**
VE(1/min)	Sea level	6.80 ± 0.70	62.90 ± 5.00	65.80 ± 3.70	64.40 ± 3.40
	Altitude	6.90 ± 0.50	81.50 ± 8.10	87.20 ± 5.60	89.60 ± 3.50

Table 2. *Breathing frequency (BF) means ± SD at normoxia and hypoxia condition.*

		Rest	**Bout 1**	**Bout 2**	**Bout 3**
BF	Sea level	10.00 ± 1.40	24.80 ± 2.50	24.30 ± 1.60	25.00 ± 1.40
	Altitude	11.30 ± 1.60	29.30 ± 1.20	32.00 ± 1.40	33.70 ± 0.80

Table 3. *Cardiac output (Q) means ± SD at normoxia and hypoxia condition.*

		Rest	Bout 1	Bout 2	Bout 3
Q (l/min)	Sea level	4.00 ± 0.50	12.50 ± 0.50	12.20 ± 0.60	12.80 ± 0.40
	Altitude	4.80 ± 0.30	13.10 ± 0.40	14.60 ± 0.50	15.50 ± 0.60

Table 4. *Heart rate (HR) means ± SD at normoxia and hypoxia condition.*

		Rest	Bout 1	Bout 2	Bout 3
HR	Sea level	63.00 ± 5.00	138.00 ± 8.00	138.00 ± 4.00	137.00 ± 2.00
	Altitude	73.00 ± 6.00	152.00 ± 8.00	161.00 ± 5.00	166.00 ± 5.00

Table 5. *Blood Lactate ($[La]_{bl}$) means ± SD at normoxia and hypoxia condition.*

		Rest	Bout 1	Bout 2	Bout 3
$[La]_{bl}$ (mmol/l)	Sea level	0.35 ± 0.16	2.58 ± 0.36	3.06 ± 0.39	2.85 ± 0.29
	Altitude	0.41 ± 0.14	3.80 ± 0.46	4.70 ± 0.53	5.83 ± 0.39

Table 6. *Arterial oxygen saturation (SaO_2) means ± SD at normoxia and hypoxia condition.*

		Rest	Bout 1	Bout 2	Bout 3
SaO_2 (%)	Sea level	99.30 ± 0.50	98.20 ± 0.80	98.50 ± 0.50	98.30 ± 0.50
	Altitude	98.70 ± 0.50	95.20 ± 0.70	98.30 ± 0.80	95.50 ± 1.10

DISCUSSION

In the present study we found that ventilation rate, breathing frequency, cardiac output, heart rate, Labl and SaO_2 increased significantly in the hypoxic condition (10000 feet altitude) in contrast with normoxia. The earliest and most obvious response and adaptation of the sojourner to high altitude is an increase in VE and breathing frequency accompanied by elevating arterial oxygenation (8). Bender et al. (2) demonstrated that ascent to high altitude is accompanied by an increase in VE. Katayama et al. have also reported that a sojourn at high altitude leads to increases in resting hypoxic ventilation responses (HVR) accompanied by increases in pulmonary ventilation and SaO_2 at rest (8). On the other hand, this study showed a significant increase in VE during exercise in hypoxia compared to normoxia, accompanied by a significant increase in breathing frequency at rest and during exercise in hypoxia. Those results agree with Ward et al. (14) who showed that during a background of moderate exercise, introduction of hypoxia caused VE to increase from 30.84 to 56.44 l/min-1. This increase was also associated with an increase in tidal volume and respiratory frequency as seen at rest but with much larger magnitudes. In addition, Nakajono et al. (11) showed that there was a 10.7% increase in VE in hypoxia during exercise.

This increasing ventilation in hypoxia may be advantageous for performance at altitude and prevents acute mountain sickness and high-altitude pulmonary edema. Several other studies have indicated that hypoxic ventilatory responses as indexes of ventilatory chemosensitivity to hypoxia correlate with ventilatory response to exercise in normoxia and that HVR correlate with ventilation and SaO_2 during hypoxic exercise (8). In this study, SaO_2 decreased both in hypoxia and normoxia at the transition from rest to exercise. This decrease was significantly higher at altitude condition. This finding agrees with the results from a study conducted by Bender et al. (2) who demonstrated that in the transition from rest to moderate exercise SaO2 tends to decrease in acute hypobaric hypoxia. Moreover, Brosnan et al. (4) have reported a higher decrease in SaO_2 under hypoxia than normoxia after each of the three 10min endurance work bouts (95.4 vs. 92.9, 96.1 vs. 93, 96.1 vs. 94.2%).

During moderate exercise, hypoxia results in slower O_2 uptake kinetics, which suggests that the elevated HR does not completely compensate for the reduced SaO_2 to maintain normal O_2 delivery to the contracting muscles during the exercise transition (5). The primary effect of hypoxia on HR seen in this study is an increase in the baseline level during rest. This is possibly due to an increase in circulating catecholamines resulting from spillover of the greater sympathetic nerve activation that has been reported to take place under hypoxic conditions. It might also result from peripheral reflexes that stimulate cardiac output when the arteriovenous O_2 content difference decreases

(5). This study has also reported a significant higher HR and Q at rest and during exercise in hypoxia compared to normoxia and a continuous increase in HR and Q in exercise due time. This increase in Q is probably due to an increased HR both at rest and during exercise in hypoxia condition, and not a decrease in stroke volume as exercise was of moderate intensity and not intensive enough to cause decreases in stroke volume. Relevant to this, Wolfel et al. (15) found that HR and Q were increased in exercise during acute exposure to altitude. Moreover, a study conducted by Galbet et al. (6) demonstrated that hypoxia causes a 17% decrease in peak Q due to equal reductions in both peak HR and stroke volume during incremental cycle ergometer exercise to exhaustion. All these date collectively demonstrate a) the importance of increased SaO_2 in establishing the driving force for O_2 diffusion from the capillaries to the mitochondria and b) the circulatory responses to acute hypoxia appear to be insufficient to fully compensate for the reduced SaO_2 (5).

As it was reported before, Hogan et al. (7) have previously demonstrated that during an incremental maximal test performed under hypoxic conditions (17% O_2), [La]bl was elevated at moderate to high power output (>200 w) compared with normoxia. In this study [La]bl was significantly higher in hypoxia during rest and exercise and continued increasing in hypoxia over time during exercise. These results reach an agreement with the findings from a study conducted by Bouissou et al. (3) which have also concluded that exercise in hypoxia leads to an increased [La]bl accumulation. Due to the slower O_2 uptake kinetics, referred above, the O_2 deficit is greater in hypoxia than normoxia, implying greater reliance on anaerobic sources of high-energy phosphates (e.g. phosphocreatine and anaerobic glycolysis with lactate production). Thus, the slowed VO_2 kinetics with hypoxia is associated with a greater rise in blood lactate concentration (5). Balsom et al determined the effects of simulated altitude (3000 m, 562 mmHg) on repeated high-intensity cycle sprints. These researchers proposed that the lower power output and higher levels in the hypoxic condition were due to a decreased O_2 availability and an increased reliance on O_2-independent glycolysis for ATP synthesis (1). This would also suggest an increased metabolism as an energy source during hypoxic exercise.

In conclusion, the results of this study confirmed the hypothesis showing that after short-term intermittent exposure to acute hypoxia in a hypobaric chamber (equivalent to 3100 m altitude), VE, BF, HR, Q, Labl and SaO_2 increased significantly during exercise and rest compared to normoxia, except from VE which had a lower value during rest in hypoxia. This study has also suggested that a low exercise-induced hyperventilatory response is a significant mechanism in the arterial desaturation observed during hypoxic exercise. The increased values of the variables measured during hypoxia suggest that

enhanced hypoxic ventilatory chemosensitivity is the main reason for the enhanced metabolism at altitude. The slowing in VO_2 kinetics during exercise is correlated with an enhanced production of lactate during exercise. The baseline HR during rest in hypoxia is elevated presumably due to elevated circulating catecholamines. All these changes may be interpreted as acclimatization to altitude. Exercise seems to have potentiated the acute ventilatory response to hypoxia by modifying all the cardiorespiratory variables measured. The monitoring of the cardiorespiratory responses during moderate exercise in hypobaric hypoxia may be used to detect the first stages of acclimatization to altitude.

CONCLUSION

Taking into account the weaknesses of these classes, the criticism that they have being facing and the prospect of the administration of closing then down, a number of studies were initiated which tried to establish their weaknesses and make recommendations about their future.

These studies firstly argue that even though the Classes fell short in their formation of the Greek Olympic team, their contribution to the Greek athletic system should not be undervalued because: a) a lot of valuable experience has being gained from the functioning of these Classes, b) the teachers-coaches who are involved in spotting athletic talents, the teachers-coaches who are employed in these classes, along with the whole organization of the system at a local and national level have made a positive contribution to Greek sports, c) even though most of the best pupils-talents that were spotted did not enrol in the Classes of Athletic Facilitation, nevertheless since they had been marked out representatives of clubs were able to get in contact with their parents and enrol them in the athletic clubs, d) they made a minor contribution to the solution of the problem of unemployment of Physical Education teachers since thousands of teachers-coaches found a permanent or part time job there.

Secondly these studies make certain recommendations for the reorganisation of these Classes so that the institution can survive. In short all of them recommend the closing down of the Classes and so that they can be replaced by specialized Athletic Schools. The process will start from the major cities and if it proves successful be expanded to smaller towns. However even though those studies seem to make a positive contribution to the solution of the problem, they see the problem only as a «technical» one, ignoring the wider issues associated with it. It seems that they are lacking a broader theory concerning the process of implementing changes in athletics.

Every proposed change aiming at the improvement of the Classes has to take into account at least three parameters. a) The weaknesses of the Class-

es as has been mentioned above, b) the existing climate in athletics in Greece in the post Olympic era, and c) the impact of what is called in the literature the «multiple streams framework». i.e. (i) the changing values, beliefs and ideas, (ii) the changes in organizational infrastructure and response dependency, (iii) the relative strength of lobby/interest group activity, and (iv) the significance of influential individuals. So an in-depth research on the above variables «b» and «c» is needed in order for a new, comprehensive and viable plan to be created which will give the Classes a chance to survive by making them more attractive for the pupils in order to meet the expectations of the people who established them twenty years ago.

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