

Effect of training on the development of exercise-induced arterial hypoxemia in volleyball players

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Authors' Contribution: A –Study design; B –Data collection; C –Statistical analysis; D –Manuscript Preparation; E – Funds Collection.

Abstract

Purpose: The purpose of this study was to examine the effect of volleyball training on the development of exercise-induced arterial hypoxemia during incremental exercise in male competitive volleyball players.

Material: Eight male amateur volleyball players (age 21 ± 1.3 years) participated in a 6-week volleyball training program three times a week in the pre-season preparatory period. All participants were students of the Faculty of Sport Sciences. Before and after the training period, all players performed an incremental treadmill test to determine maximal oxygen uptake (VO_{2max}), and oxyhemoglobin saturation (SaO_2) was continuously measured using a pulse oximeter during the test. Maximal values of minute ventilation (VE_{max}), respiratory exchange ratio (RER_{max}), ventilatory equivalent for oxygen (VE/VO_2) and carbon dioxide (VE/VCO_2) were determined. Exercise-induced arterial hypoxemia (EIAH) was defined as a SaO_2 decreased by at least 4% ($\Delta SaO_2 \leq -4\%$) from resting level.

Results: All the players exhibited exercise-induced arterial hypoxemia before ($\Delta SaO_2 = -8.8 \pm 3.3\%$) and after ($\Delta SaO_2 = -8.31.5\%$) the training period. SaO_2 was significantly decreased from $97.6 \pm 1\%$ at rest to $88.7 \pm 2.7\%$ at exhaustion before the training period, and from $97.2 \pm 1.1\%$ at rest to $88.8 \pm 2.1\%$ at exhaustion after training period ($p < 0.001$). There was no significant difference in resting and lowest SaO_2 values by comparison between the before and after training ($p > 0.05$). There were no significant changes in VO_{2max} , VE_{max} , RER_{max} , VE/VO_2 and VE/VCO_2 after training period ($p > 0.05$).

Conclusions: The results of this study showed that volleyball players with a history of anaerobic training may exhibit EIAH, but that 6-week volleyball training has no effect on the degree of exercise-induced arterial hypoxemia.

Keywords: desaturation, pulse oximetry, oxyhemoglobin saturation, team sports athletes

Introduction

Maximal oxygen uptake (VO_{2max}) is one of the most important factors determining aerobic endurance performance [1]. It has been shown that the respiratory system may be a factor limiting VO_{2max} in healthy athletes [2]. Many athletes have been shown to experience exercise induced arterial hypoxemia in a normoxic environment [3, 4]. This negative finding accompanied by decreased partial pressure of oxygen in arterial blood may contribute to local muscle fatigue [5]. It has been shown that when the fraction of inspired oxygen increased during exercise, the decrease in oxyhemoglobin saturation (SaO_2) prevented, the endurance time to exhaustion and VO_{2max} increased [6, 7].

Exercise-induced arterial hypoxemia (EIAH) manifests as decreased partial pressure of oxygen in arterial blood (PaO_2) or decreased SaO_2 below the pre-exercise level [2]. EIAH is classified as mild (93–95% SaO_2), moderate (88–93% SaO_2), and severe ($< 88\%$ SaO_2) [2]. The mechanism underlying of EIAH is still not clearly understood. Several potential causes of EIAH including inadequate alveolar hyperventilation, oxygen diffusion limitations, ventilation-perfusion inequality, intra- and extra-pulmonary shunts, low pulmonary capillary blood transit time and interstitial edema or an interaction among these factors have been proposed [2, 8, 9, 10].

Studies investigating EIAH mostly focused on endurance athletes and reported that EIAH is more common in athletes with high aerobic capacity [2, 3, 10, 11]. The finding of negative correlation between SaO_2 and VO_{2max} supported the suggestion that improving aerobic capacity by physical training may be associated with the occurrence of EIAH during strenuous exercise [12]. On the other hand, we and others have recently demonstrated that EIAH can occur in anaerobic trained athletes and non-endurance sportsmen (relatively low VO_{2max}) [13, 14].

A few researchers investigated effects of physical training on EIAH, however, showed different results [12, 14, 15]. Low ventilator chemoresponsiveness may be induced by physical training and it has been suggested that it may contribute to less hyperventilation and therefore EIAH during strenuous exercise in trained subjects [10, 12, 14]. It has been shown that physical training-induced increases in VO_{2max} were accompanied by more severe EIAH [12, 14]. On the contrary, Dominelli et al. showed that VO_{2max} increased after physical training, but the degree of EIAH was at the same level as before the training program [15]. To our knowledge, there is no previous study examining the effects of volleyball training on EIAH during incremental exercise.

The purpose of this study was to examine the effect of volleyball training on the development of exercise-induced arterial hypoxemia during incremental treadmill exercise in male competitive volleyball players.

Material and Methods

Participants

Eight male volleyball players (mean \pm SD; age 21 ± 1.3 years, height 181.4 ± 3 cm, body mass 71 ± 4.7 kg) who played in Erciyes University volleyball team volunteered to participate in the present study. All participants were students of the Faculty of Sport Sciences. All players had trained and competed regularly in volleyball for at least 4 years. Measurements were performed following the approval of the Ethics Committee and carried out in accordance with the Declaration of Helsinki. All testing and training procedures were fully explained, and written informed consent was obtained for each participant.

Experimental Design

The experimental protocol consisted of baseline testing, a 6-week training intervention, and post-testing. Players performed a volleyball training program three times per week for 6 weeks at the beginning of the pre-season preparatory period. All training sessions were conducted at the same time of day on Monday, Wednesday and Friday of each consecutive week. One week before the start of the 6-week training period and two days after its completion all players performed an incremental treadmill test. During the study, the players were not allowed to perform any additional strength and conditioning training that would affect the results of the study.

Data Collection

Incremental running test was performed on a motorized treadmill (h/p/Cosmos Quasar med, Nussdorf-Traunstein, Germany). Throughout all tests, expired air was measured online using a breath-by-breath cardiopulmonary exercise testing system (Quark PFT Ergo, Cosmed Srl, Rome, Italy). During the incremental testing period, heart rate (HR) was monitored continuously using a wireless HR monitor (S610i, Polar, Finland) and was synchronized to ventilatory signals. Before each test, ambient conditions were measured, and the gas analysers and turbine flowmeter were calibrated with known certified gas concentrations (16 %O₂, 5 %CO₂, and balanced N₂) and a 3 L calibration syringe, respectively, following the manufacturer's instructions.

Breath-by-breath data was smoothed using a five-step average filter and then reduced to 15 s stationary averages. Maximal oxygen uptake (VO_{2max}), maximal values of minute ventilation (VE_{max}) and respiratory exchange ratio (RER_{max}), ventilatory equivalent for oxygen (VE/VO₂) and carbon dioxide (VE/VCO₂) were determined during the incremental treadmill test.

SaO₂ was measured continuously and recorded every 15 s during the incremental treadmill test, using a finger pulse oximeter (Spiropalm 6MWT; COSMED, Rome, Italy). For most accurate readings, the sites were vigorously cleaned with alcohol and gauze pads. EIAH was assumed to have developed when SaO₂ decreased by at least 4 % ($\Delta\text{SaO}_2 \leq -4\%$) from the baseline values [2]. Delta SaO₂ (ΔSaO_2) was calculated as the difference between rest and maximal exercise values.

Incremental Treadmill Test

Before test, the players performed a standardized warm-up consisting of a 5 minutes run at their own pace followed by about 3 minutes of stretching. Players started running at 7 km/h with speed increments of 1 km/h every minute until they could no longer keep pace. All players were given strong verbal encouragement throughout the test to elicit their best performance.

Achievement of VO_{2max} was considered as the attainment of at least two of the following criteria: 1) a plateau in VO₂ despite increasing speed, 2) a respiratory exchange ratio (VCO₂/VO₂) above 1.10, and 3) a HR within 10 beats per minute of age-predicted maximum HR ($220 - \text{age}$). The VO_{2max} was defined as the highest 15 s VO₂ value reached during the incremental test and expressed as a relative value (milliliters per minute per body mass; ml/kg/min). VE/VO₂, VE/VCO₂, VE_{max} and RER_{max} were expressed as the highest 15 s average value obtained during the last stage of the incremental exercise test.

Training Program

A single training session lasted approximately 120 minutes (comprising warm-up, main and cool-down periods). The warm-up period consisted of jogging, different types of running and accelerations, submaximal jumps, mobility exercises, full body stretching and specific volleyball warm-up drills with the ball, and lasted 20-25 minutes. Each training session ended with a 10-15 minutes cool-down consisting of walking and stretching. The main part of the volleyball session consisted of on-court skills training and the game-based drills including small-sided games and real-game volleyball drills. On-court skills training included serving, passing, and setting in small groups, spiking, blocking and digging drills, as well as skills-based conditioning drills such as lateral movement and blocking, lateral movement and dig drill, moving off of the net and retrieving a ball. Training sessions concluded with high-intensity game-based drills to work on offensive and defensive strategies and individual tactics. The game-based drills included small-sided games such as 3 vs. 3 and 4 vs. 4, where the volleyball court was divided into two smaller courts, and 6 vs. 6 real-game volleyball drills.

Statistical Analysis:

Data are reported as mean \pm standard deviation (SD). Statistical significance was accepted at $p < 0.05$. The normality distribution of the data was checked with the Shapiro-Wilk test. Within-group changes before and after the 6-week training period was compared using paired t-test for normally distributed data, and Wilcoxon matched-pair signed-rank test for non-normally distributed data. To allow a better interpretation of the results, effect sizes were also calculated using Cohen's d [16]. Effect sizes were interpreted as negligible ($d \geq 0.2$), small ($0.2 \leq d \leq 0.5$), medium ($0.5 \leq d \leq 0.8$) or large ($0.8 \geq d$). SPSS version 21 was used for all analyses (SPSS Inc., Chicago, IL).

Results

Table 1 shows the players' responses to incremental treadmill test. All the players exhibited EIAH (as defined by $\Delta\text{SaO}_2 \leq -4\%$) before and after the training program (Figure 1). SaO_2 was significantly decreased from $97.6 \pm 1\%$ at rest to $88.7 \pm 2.7\%$ at exhaustion before the training program ($p < 0.001$, $d = 5.5$), and from $97.2 \pm 1.1\%$ at rest to $88.8 \pm 2.1\%$ at exhaustion after training program ($p < 0.001$, $d = 5.3$). There was no significant difference in resting $\text{SaO}_2\%$ and ΔSaO_2 values by comparison between the before and after training program ($p > 0.05$, $d = 0.4$). The lowest $\text{SaO}_2\%$ values occurred at or near $\text{VO}_{2\text{max}}$ in both exercise tests with no significant difference the before and after training program ($p > 0.05$, $d = 0.04$) (Figure 1).

Figure 2 shows the changes in SaO_2 plotted as a percent of the relative work rate (i.e. $\text{VO}_{2\text{max}}\%$). EIAH

was begun at $70.4 \pm 21.1\%$ of maximum work rate (i.e. $\text{VO}_{2\text{max}}\%$) before the training program and at $70.9 \pm 18.1\%$ of maximum work rate after the training program. There were no significant differences in the exercise intensity of began to experience EIAH after training program compared to before ($p > 0.05$, $d = 0.03$). There were no significant changes in $\text{VO}_{2\text{max}}$, VE_{max} , RER_{max} , VE/VO_2 and VE/VCO_2 after training program compared to before ($p > 0.05$) (see Table 1).

Discussion

In the present study, the incremental treadmill test caused EIAH in all volleyball players, and this response was found to be similar after the 6-week volleyball training program. In addition, volleyball training had no statistically effect on $\text{VO}_{2\text{max}}$, VE_{max} , RER_{max} , VE/VO_2 and

Table 1. Results of the incremental treadmill test of the volleyball players before (pre) and after (post) the 6-week training period

Variables	Pre- training	Post- training	p	d
ΔSaO_2 (%)	8.8 ± 3.3	8.3 ± 1.5	0.7	0.2
$\text{VO}_{2\text{max}}$ (ml/kg/min)	50.3 ± 3.1	51.3 ± 3.3	0.9	0.3
VE_{max} (L/dak)	155.4 ± 7.7	160.6 ± 10.8	0.07	0.5
RER_{max}	1.16 ± 0.05	1.2 ± 0.06	0.2	0.7
VE/VO_2	41.3 ± 4.2	42.4 ± 4.6	0.4	0.2
VE/VCO_2	37.7 ± 2.9	36.6 ± 3.6	0.2	0.3

Note: Values are means \pm SD; $\text{VO}_{2\text{max}}$ = maximal oxygen uptake, RER_{max} = maximal respiratory exchange ratio, VE_{max} = maximal minute ventilation, VE/VO_2 = ventilatory equivalent for O_2 at maximal exercise intensity, VE/VCO_2 = ventilatory equivalent for CO_2 at maximal exercise intensity, ΔSaO_2 = difference between rest and maximal exercise values of oxyhemoglobin saturation.

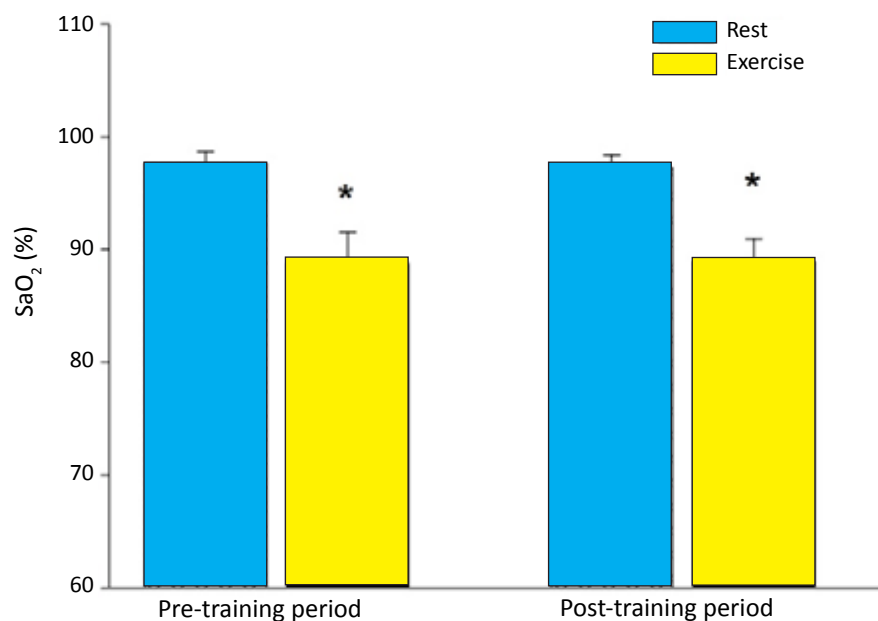


Figure 1. * Significantly different from pre-training (baseline) values. SaO_2 values at rest and maximum work rate during incremental treadmill test before (pre) and after (post) the 6-week training period.

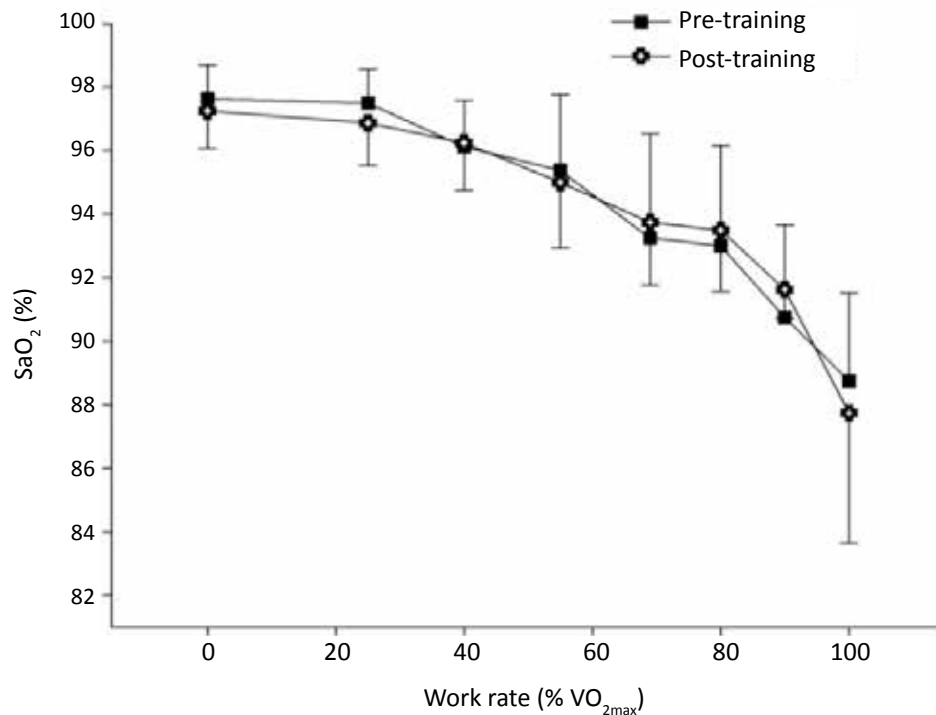


Figure 2. Mean values for the changes in the percent of oxyhemoglobin saturation (SaO₂) at different percentages of relative work rate during incremental treadmill test before (pre) and after (post) the 6-week training period.

VE/VCO₂. These findings suggest that anaerobic trained athletes may exhibit exercise-induced arterial hypoxemia, but volleyball training has no effect on the degree of exercise-induced arterial hypoxemia.

We found that EIAH occurred in all subjects before and after the training period. On the other hand, previous studies have observed that EIAH did not occur in all trained athletes [4, 17]. In particular, EIAH has been reported to occur mostly in endurance athletes with a VO_{2max} greater than 60 ml/kg/min [3, 10, 11, 17]. Powers et al. found that 52% of the highly trained athletes occurred EIAH during incremental cycle exercise test, whereas none of the untrained or moderately trained subjects developed EIAH [17]. A more recent study has shown that EIAH during incremental treadmill exercise occurs in 70% of well-trained endurance athletes [4]. Differences in measured SaO₂ may be explained by differences in exercise modality and types of protocols used [18, 19]. It has been shown that a greater drop in the SaO₂ during treadmill running compared with cycle ergometry exercise in the same subjects [20]. Mucci et al. showed that non-endurance sportsmen developed EIAH after an eight-week supra-maximal interval-training program [14]. In our recent study, we showed that EIAH during incremental treadmill exercise occurred at similar level in both aerobic and anaerobic trained athletes with different aerobic fitness levels [13]. It appears that studies examining EIAH have primarily focused on endurance athletes [11, 17, 21]. The more severe occurrence of EIAH in elite athletes may be due to the trained athletes being able to push their physical capacity to the limit during

maximal exercise rather than aerobic capacity level.

Pulse oximetry has been widely used in the literature to determine EIAH and has been recognized as a valid and reliable tool for continuously monitoring SaO₂ % during exercise [22, 23]. Similar to previous studies using pulse oximetry, in our study, the SaO₂ % values decreased in the range between 85% and 92% during incremental exercise [3, 24, 25]. SaO₂ decreased 8.8% and 8.3% from rest before and after training period, respectively, similar to previous studies performed during incremental treadmill exercise [13, 26]. In our study, volleyball players were considered to have developed EIAH when SaO₂ fell at least 4 % ($\Delta\text{SaO}_2 \leq -4\%$) from the baseline values [2]. During the incremental treadmill exercise, we followed the time course of SaO₂ from rest state to exhaustion. EIAH developed at the about 70.4 and 70.9 % of maximum work rate (i.e. VO_{2max} %) before and after the training program, respectively, and peaks at or near maximal exercise intensity. There was no difference in the work rate of began to experience EIAH after training program compared to before. Our findings are consistent with studies in the literature showing that EIAH begins to occur at submaximal exercise in some subjects and usually peaks at or near maximal exercise intensity during the incremental exercise [13, 21, 27]. Inadequate hyperventilation and widened alveolar-to-arterial oxygen difference are the most likely mechanisms for EIAH occurs at moderate intensity workloads [2, 9, 21, 27].

There are limited studies in the literature investigating the effect of training on EIAH [12, 13, 15]. Interval training has been shown to increase VO_{2max}, while decreasing

SaO₂ more during heavy exercise [12, 14]. It is suggested that the more severe occurrence of EIAH after training is caused by less alveolar hyperventilation response [12, 14]. In these studies, the decrease in VE/VO₂ after the training period compared to before was interpreted as less hyperventilation response developed with training [12, 14]. Mucci et al. associated the development of EIAH after 8 weeks of interval training with a decrease in VE/VO₂ despite an increase in maximal ventilation during exercise [14]. The decreased hyperventilatory response during strenuous exercise may be related to the lower adaptation of the ventilator system to physical training [12]. It has been suggested that less hyperventilation during strenuous exercise after training period may be related to adaptations in ventilator chemo-responses [12]. Endurance athletes endowed with low ventilator chemo-responses have been reported to breathe less than non-athletes at similar exercise intensities [28]. The low chemoresponsiveness of the respiratory system has been shown to contribute to less hyperventilation and arterial hypoxemia during strenuous exercise [10, 29]. Granger et al. suggested that chemoreceptor sensitivity to carbon dioxide may play a role in the development of EIAH in aerobically trained athletes, partially explaining the variability in EIAH occurrence and severity [10]. VO_{2max} did not change after 6 weeks of volleyball training in our study. In addition, it may be suggested that less hyperventilation response did not develop with volleyball training, as there were no significant changes in VE/VO₂ and VE/VCO₂. As a matter of fact, no difference was found in the degree of EIAH in volleyball players after the training program compared to before. The ability of the neuromuscular system to produce power during intense exercises such as various sprints, jumps and high-intensity court movement is the most important factor determining athletic performance in volleyball [30]. Volleyball training may not include physiological adaptations to improve the aerobic capacity of athletes.

On the other hand, exercise-induced arterial hypoxemia has been suggested to not necessarily worsen with aerobic training [15]. There are some researchers showing that aerobic training did not increase the severity

of EIAH [15]. Dominelli et al. showed that female runners' VO_{2max} increased after five months of endurance training, but the degree of EIAH remained similar during the maximal exercise test [15]. Some researchers reported that arterial hypoxemia during strenuous exercise was accompanied by less hyperventilation response and lower alveolar oxygen partial pressure [21, 31]. On the contrary, others concluded that the increase in alveolo-arterial oxygen pressure difference (A-aDO₂) plays a major role in the occurrence of arterial hypoxemia during strenuous exercise and ventilation contributes less [32]. It has been reported that the increase in A-aDO₂ during strenuous exercise is mainly due to oxygen diffusion limitation caused by the shortening of the pulmonary capillary blood transit time when cardiac output reaches its maximum in trained athletes [21, 11]. One of the possible causes of the increase in A-aDO₂ is the increase in ventilation-perfusion mismatch as a result of the greater increase in minute ventilation volume compared to perfusion during exercise [9]. In addition, the development of interstitial pulmonary edema due to a high cardiac output during exercise contributes to the enlargement of A-aDO₂ by decreasing PaO₂ [2]. However, there are very few longitudinal studies examining the specific effect of aerobic or anaerobic training on EIAH. Our data represent a rather novel finding that could be of considerable importance for showing occurrence of EIAH in volleyball players, but that volleyball training has no effect on the severity of EIAH.

Conclusions

The results of this study showed that volleyball players with a history of anaerobic training may exhibit exercise-induced arterial hypoxemia, but that 6-week volleyball training has no effect on the degree of exercise-induced arterial hypoxemia. Further research is needed to investigate the effects of training on exercise-induced arterial hypoxemia in different team sport branches.

Conflict of interest

Authors declare no conflict of interest.

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