

Sport Sciences and Health Research



Cardiovascular and respiratory response to ascent of the Damavand summit by classic method in elite climbers

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Article Info	Abstract				
Original Article	Background: Exposure to high altitude causes significant stress in the				
0	functioning of cardiovascular and respiratory systems.				
	Aim: The aim of this study was to investigate the changes in systolic and				
Article history:	diastolic blood pressure (BP), heart rate (HR) and pulmonary volumes during the classic ascent of Damavand 5671 m peak.				
Received: 17 March 2023	Materials and Methods: 14 healthy male climbers (age = 23.85±5.93 years,				
Revised: 20 June 2023	height = 175.5±5.91 cm, weight =71.35±8.20 kg) participated voluntarily in				
	this study. BP, HR and lung volumes were measured in basic (2000 M), BC				
Accepted: 29 June 2023	(3100 m) the first day, C1 (3650 m) the first day, BC (3100 m) the second day				
Published: 01 July 2023	after the one-night stopover, C1 (3650 m) the second day, C2 (4100 m) the				
	second day, C2 (4100 M) third day after the one-night stopover, C3 (5000				
Keywords:	m) and, peak (5671 m). Data were compared by repeated measures test at				
•	the significant level of $P \le 0.05$ and Bonferroni post hoc test.				
Ascent, blood pressure,	Results: The results of repeated measures analysis were significant for HR, systelic RP, diactelic RP, and $M(V/PC, OE)$ but not significant for EVC and				
classic method,	systolic BP, diastolic BP and MVV (P <0.05), but not significant for FVC and FEV1 (P ≥0.05). BP was high in the base camp and the first camp and				
heart rate,	gradually decreased. HR continued to increase significantly with increasing				
lung volumes.	altitude. MVV increased with increasing altitude and is reduced after the				
	one-night stopover in each height.				
	Conclusion: It can be concluded that climbing with classic method increased the				
	HR and decreased systolic and diastolic BP of climbers. It was able to induce				
	adaptation in lung volumes of climbers.				
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summit by classic method in elite climbers". Sport Sciences and Health Research. 2023; 15(2): 159-169.					
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1. Introduction

Mountaineering as a professional sport has found a special place among sports for many years. Climbing the heights is done in different methods, and each method leads to different adaptations based on the time spent at the height and the amount of height increase during the day. The term high altitude generally refers to heights more than 2000 m above sea level. One of the special characteristics of altitude is the decrease in barometric pressure, which decreases the density of oxygen and increases the density of carbon dioxide in the atmosphere; for example, at an altitude of 5000 m, the density of oxygen is 40% lower than that at sea level [1].

This decrease in pressure can be changed with atmospheric changes and barometric pressure will not be the same at the same altitude in different places [2]. Exposure to high altitude causes significant stress in the functioning of cardiovascular and respiratory systems [3].

Lungs are the first mediator between the oxygen in the environment and the body's metabolic mechanism. At rest and during activity, successful gas exchange in the capillary-alveolar membrane is necessary for tissues in order to obtain sufficient amount of oxygen and remove carbon dioxide. To achieve this goal, the mechanical function of the lungs, chest walls and respiratory muscles must move a sufficient volume of air to provide the oxygen needed by the body [4].

In addition, systemic O_2 transport is the result of arterial oxygen content (CaO₂) and cardiac output (Q). When arterial oxygen content is reduced in short-term exposure to altitude, systemic O_2 transport at rest and during submaximal exercise is maintained with higher cardiac output. Cardiac output is thus maintained to ensure oxygen delivery meets metabolic needs. The initial increase in resting cardiac output with short-term exposure to hypoxia is mostly the result of increased heart rate (HR) that is known as chronotropic response [5].

Since blood pressure (BP) affects the availability of O₂ for different organs of body, altitude can affect BP [6]. On the other hand, the increase in altitude affects the sensitivity of the respiratory centers and will lead to the possibility of a temporary inhibitory effect on the respiratory system. Therefore, breathing control is faced with problems and the possibility of changing lung volumes is aroused [7]. In addition to airway the decrease in resistance. respiratory muscle endurance may be reduced by hypobaric hypoxia; Thus, forced vital capacity (FVC) is reduced, which can be a factor in limiting exercise at high altitude [8].

Maximum voluntary ventilation (MVV) is one of the effective variables in causing muscle fatigue [9], which can affect the performance of climbers. Forced expiratory volume (FEV1) is one of the main scales of pulmonary function [10] that indicates exhalation power [11] and the resistance of airways to air flow [12].

On the other hand, for climbers, the activity of all organs and systems of the body is necessary and important during climbing activities, but we know that the survival of body cells depends on oxygen, and the respiratory and cardiovascular systems are mediators between the oxygen in the environment and the body's metabolic mechanism [8].

At high altitudes (HA), due to the decrease in the partial pressure of oxygen, a larger volume of air is needed to provide enough oxygen to the body. Due to the limited diffusion of oxygen from the air to the blood, the transfer of oxygen is damaged, that increases with the increase in altitude, and this lack of oxygen will limit the activities of mountaineers. Therefore, the respiratory and cardiovascular systems which are necessary for the functioning and life of humans at HA, are the most basic organ compared to other body systems [8].

Acute exposure to high altitude causes adaptive changes through various physiological mechanisms, which are shown by changes in blood composition and neurological, cardiovascular, renal, and metabolic functions.

Recent studies suggest that acute exposure to high altitude may affect various mechanisms of BP control, leading to increased BP and HR. BP adaptation to high-altitude are characterized by changes with duration of exposure to altitude: during the first minutes or hours of exposure to hypoxia, BP remains essentially unchanged due to chemical responses and sympathetic activity to the vasoconstrictor reflex, after this stage, the activation of the mechanisms leads to an increase in BP [13].

However, what changes in BP, HR, and pulmonary factors will be caused by longterm presence at altitude by inhibiting these mechanisms. The interactive effect of the respiratory system and changes in blood oxygen concentration should be investigated.

Physiological studies on climbers have reported that with increasing altitude, a progressive decrease in body performance and efficiency occurs. As one of the most important limiting indicators of the body's performance altitude at is called hypoxemia. Hypoxemia is the response of ventilation to hypoxia and the capacity of the lungs to transfer oxygen to the capillaries, which is possible through the measurement of respiratory factors. By measuring these variables implicitly, the

fatigue threshold of the respiratory muscles, which is one of the factors limiting the activities of climbers at altitude, is determined. During climbing to heights, the amount of oxygen that enters the blood through the lung bubbles, decreases due to the drop in barometric pressure and the decrease in the percentage of oxygen in the air. If the mountaineer is quickly faced with this reduction of oxygen, the probability of altitude sickness and damage to the respiratory system will be higher in that person [14]. To prevent these injuries, climbers use the classic method to reach the peaks.

It has been concluded that at altitudes higher than 3000 m, the average height of staying overnight at altitude should not be more than 300 m from the previous night, and every two or three days (or every 1000 m of ascent) one day of rest should be done [15].

Since the physiological effect of altitude can be different based on the amount of height, physiologists have divided altitudes into four categories: 1. Medium altitude (1500-2440 m), 2. High altitude (2440-4270 m), 3. Very high altitude (4270-5490 m), 4. Extreme altitude (5490-8848 m) [16].

In Iran, the only peak that is in the fourth category, is Damavand peak, which distinguishes it from other Iranian peaks. Based on this, it is important to investigate what effect the height of Damavand peak can have on systolic BP, diastolic BP, MVV, FVC, and FEV1 and can the classic method moderate the effects of altitude?

2. Materials and Methods

2. 1. Participants

In this semi-experimental research, the statistical population was male professional mount climbers of East Azerbaijan province, aged 20 to 35 years old.

Active mountaineering groups in East Azerbaijan Province, have an official license from the Iranian Mountaineering Federation, and carry out their annual programs based on the Federation's instructions. These groups were identified with the cooperation of the Mountaineering Committee of East Azerbaijan Province, and two groups (Babak Group and Kaveh Group) were selected randomly among 120 mountain climbing groups and 14 climbers volunteered to participate in this study. Participants were asked to complete health and activity questionnaires. Research objectives, climbing schedule, climbing methods, types of tests and measurements during the climbing were explained to the participants beforehand and a written consent was obtained from each participant. Research inclusion criteria were having at least 5 to 6 years of semi-professional mountaineering experience and 3 to 4 winter ascents above 4000 m and passing rock climbing, snow and ice courses.

2. 2. Procedure

In this study, participants and researchers ascended to Damavand summit by classic method (climbing at the top and staying overnight at the bottom; Figure 1). Climbing was carried out in a 5-day schedule. Dependent variables including pulmonary factors, BP, HR, were measured with portable digital devices in 9 stages (See Measurement stages).

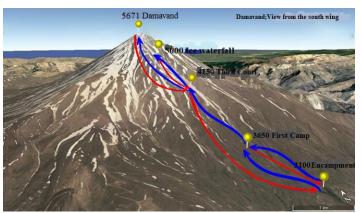


Figure 1. Ascent plan in a classic method

The ascent took place in the summer season (August) in Larijan region located in Mazandaran province from the south side of Damavand heights (5671 m). Before the start of the ascent, the load of the backpacks was controlled, and the participants were not allowed to take any drugs, especially acetazolamide and dexamethasone (in consultation with pharmacists and according to Hossein (2003)) [17]. At each stage of the night staying, the climbers were emphasized to try to get enough sleep so that their sports performance is not disturbed (but in this research, the amount

of sleep was not controlled by the researcher). The next day's work method was explained to the climbers. Altitude, barometric pressure, temperature and air condition were also controlled by precise and digital devices.

2. 2. 1. Measurement stages

The first stage. Basic conditions, at the headquarters of Mountaineering Federation in Rineh Damavand (barometric pressure= 710 mmHg, air temperature= 30°C, time of measurement=10 am on the first day).

The second stage. In Gousfand Sera where the base camp (BC) is set up (height=

3100 m, barometric pressure= 625 mmHg, air temperature= 27.8°C, time of measurement=12:30 pm on the first day).

The third stage. After climbing to the first camp (C1) (height= 3650 m, barometer pressure= 586 mmHg, air temperature= 16° C, time of measurement= 17:05 afternoon of the first day).

The fourth stage. Return to the base camp after overnight stay, at BC location (height= 3100 m, barometric pressure= 623 mmHg, air temperature= 25° c, time of measurement= 7:30 am on the second day).

The fifth stage. Moving towards C1, at the first camp site (height= 3650 m, barometric pressure= 585 mmHg, air temperature= 14° C, time of measurement= 13:30 noon on the second day).

The sixth stage. Moving towards Hosseinieh, location of the second camp, at the C2, (height= 4100 m, barometric pressure= 526 mmHg, temperature= 15° C, time of measurement= 18:30 on the second day).

The seventh stage. After overnight stay at the second camp (C2) (height= 4100 m, barometric pressure= 527 mmHg, air temperature= 13° C, time of measurement= 7:45 am on the third day).

The eighth stage. Moving to the ice waterfall, at the third camp site (C3) (height= 5000 m, barometric pressure= 464 mmHg, air temperature= 8° C, time of measurement= 2:30 p.m. on the third day).

The ninth stage. After overnight stay at the second camp (C2), move to the peak (height= 5671 m, barometric pressure= 425 mmHg, air temperature= 3° C, time of measurement= at 11:45 am on the fourth day).

2. 3. Measurements

Volunteers' weight was measured by means of a Seca weighing scale (made in Germany). In the first step, the climber stood on the scale with minimal covering. The person's weight was recorded in the specification sheet. Then, climbing shoes and clothes of the people were weighed and in each person's profile sheet was recorded. At each station, after measuring the person's weight, the weight of clothes and shoes was deducted from it. People's height was measured using a Seca height meter (made in Germany). Measurement of HR and systolic and diastolic BP, was carried out by placing a digital HR monitor around the climber's wrist.

In the next step, to measure fat percentage, fat weight and body mass index (BMI) a portable device (Body logic/body fat, Omron model) was used and the age, weight, height and sex of the climber were entered in the device by the examiner. Then the subject placed the edge of each hand on the two metal parts of the device and placed his two thumbs on the device and then by pressing the start button, the device performed the necessary measurement. Pulmonary function and its factors were measured by spirometer: In the first part called the FVC test, the person took a deep breath until the lungs were completely filled with air, followed by a strong and rapid exhalation. In the second part, which is called the MVV (liters/minute) test, the examinee performed fast inhaling and exhaling for 10 to 15 sec, and the MVV curve (liters/minute) was obtained for 1 min.

2. 4. Statistic

To analyze the collected data, descriptive statistical methods were used to obtain the mean, standard deviation, and the minimum and maximum of the data, and to compare the data, repeated measures ANOVA test with Benferroni post hoc test was used.

3. Results

Anthropometric indices and physiological variables of climbers are shown in Table 1.

The results of repeated measures ANOVA showed that there was a difference significant between data obtained in 9 stages for HR, systolic and diastolic BP of climbers. Data of FVC and FEV1 at different altitudes did not show significant differences, however, MVV showed a significant difference at different altitudes. These results are shown in Table 2.

Data showed that the HR increased as a

result of climbing to Damavand peak, but the systolic and diastolic BP decreased (Table 3). BP was high in the base camp and the first camp and gradually decreased. HR continued to increase significantly with increasing altitude. Comparing the mean values of the pulmonary factors, we found that FVC and FEV1 remained unchanged in 9 stages of measurement ($P \ge 0.05$), but MVV increased. MVV increased with increasing altitude and is reduced after the one-night stopover in each height. These results are shown in Table 3.

Table 1. Physiological and anthropometrical characteristics of participants						
Index under control	Average	Minimum	Maximum	Sandard deviation		
Age (years)	24.30	20	34	5.72		
Height (cm)	175.26	164	183	5.65		
Weight (kg)	71	61	87	8.42		
Resting HR (beats/min)	77.92	63	95	10.80		
BMI (kg/m ²)	22.51	19.6	26.1	1.57		
Body fat (%)	9.24	4.9	17.8	3.68		

Table 2. Results of repeated measurement ANOVA test for comparison of data in 9 stages during ascent by	
classic method	

classic method						
Statistics indicator	Sum of squares	Degree of freedom (Df)	Mean square	F	Р	
HR	11223.587	4.066	2760.125	18.26	0.0001*	
Systolic BP	4642.444	8	580.306	4.168	0.0001*	
Diastolic BP	4387.397	8	548.425	2.168	0.007*	
FVC	4.141	3.714	1.115	1.915	0.128	
FEV1	3.967	8	0.496	1.937	0.62	
MVV	23122.055	3.290	7027.850	3.671	0.017*	

* Significant difference between data measured at 9 stages

Table 3. Changes in variables' mean at camps with different heights

		U	1	e		
Variable/	HR	Systolic BP	Diastolic BP	FVC	FEV1	MVV
Stage	(bpm)	(mm Hg)	(mm Hg)	(L)	(L)	(L/M)
Base	79.57	146.78	85.28	3.45	3.36	164.83
1. BC	88.42	140.57	87.71	3.02	3.05	208.26
1.C1	91.50	136.85	92.21	3.39	3.41	191.96
2.BC	81.21	142.14	91.00	3.22	3.16	172.92
2.C1	98.71	138.92	78.21	3.70	3.70	180.07
1.C2	97.57	134.28	83.21	3.47	3.49	200.77
2.C2	90.50	131.21	79.35	3.45	3.36	178.85
C3	107.00	130.28	74.57	3.45	3.45	199.26
Peak	107.35	126.50	77.50	3.24	3.32	194.29

4. Discussion

Exposure to high altitude is known as exposure to hypoxia. Hypoxia refers to a decrease in the partial pressure of oxygen in the blood and tissues, and hypoxia caused by high altitude is known as hypobaric hypoxia, which is due to a decrease in atmospheric pressure that causes this decrease in partial pressure of oxygen. This decrease in partial pressure of oxygen disrupts the release of oxygen from the air to the blood and subsequently the oxygen transfers in the body.

Cardiovascular and pulmonary systems are the most basic mediating systems between the oxygen in the environment and the body's metabolic mechanism. The duration of being exposed to hypoxia may cause different responses of these body systems against the reduction of oxygen pressure. Therefore, climbers have invented different methods to climb to higher altitudes based on the duration of their stay at altitude, which is based on the experiences of climbers.

Although previous researches have examined the acute exposure and the longterm exposure of climbers to hypoxia, but the effects of classical climbing on the cardiovascular and pulmonary systems of climbers have not been investigated so far. Since each peak has its own geographical and temperature characteristics, the research conducted at similar altitudes cannot be attributed to another altitude, so we decided to explore the factors related to the cardiovascular and pulmonary systems of climbers in the classic method of climbing to Damavand peak.

The results of present study showed that there was a significant difference between HR, systolic and diastolic BP of climbers at different altitudes during the classic method of climbing. Although the lung volumes of FVC and FEV1 did not show a significant difference at different altitudes, but MVV showed a significant difference at different altitudes.

Previous studies on BP changes during acute exposure to hypobaric hypoxia at altitude done until the end of the 20th century, provided conflicting results. While some researchers observed a small increase in BP with exposure to hypobaric hypoxia, others reported a decrease or even no change of it [18].

Vogel and Harris (1967) concluded that exposure to oxygen deficiency for a short period of 1 to 4 days has little effect on systemic BP in humans [19]. Similarly, Rostrup (1998) concluded no changes in BP in response to hypobaric hypoxia [20].

On the other hand, Bestle et al. (1911) reported that exposure to hypobaric hypoxia for several days increases BP compared to sea level [21]. In a study that Veloz et al. (2020) conducted on adults in the Andean highlands, systolic and diastolic BP increased with increasing altitude [6].

Parati et al. (2014) conducted a clinical trial on 24-hour BP changes during acute and long-term exposure to altitude and in their reports, they stated that BP increases with a gradual increase in height. It remains at the same level after 3 weeks. These researchers also stated that with exposure to hypobaric hypoxia in an acute form, a systematic and significant increase in BP is seen after 7 hours during 24 hours. This increase is more evident than the increase in normal BP that is measured at rest [22].

Vittore Verrati et al. (2020) conducted a study in the Ararat heights (altitude 4200 m). They stated that acute exposure to hypobaric hypoxia causes an acute increase in systolic and diastolic BP [13]. It has been reported that acute exposure to high altitude may affect different mechanisms of BP control; these mechanisms include central nervous control and reflex sympathetic activity, arterial tension, endothelial function and blood viscosity [13].

Simpson et al. (2020) stated that the arterial baroreflex is the most important short-term controller of arterial BP, which is mainly through beat-to-beat changes in sympathetic vasomotor stimulation [3]. For example, increasing in muscle sympathetic nerve activity (MSNA) is associated with a small increase in resting arterial pressure, leading to the suggestion that baroreflex control of MSNA is impaired by chronic exposure to peripheral hypoxia. High **MSNA** is а functional adaptation mechanism that compensates for changes in vascular control mechanisms and blood volume, protects against hypotension and maintains oxygen demand at high altitude. These neural adjustments likely involve a integration of complex multiple mechanisms, the contribution of which may change as a function of duration of exposure to high altitude.

In fact, peripheral chemoreflex seems to be the main mechanism responsible for the initiation of high sympathetic vasomotor outflow during acute hypoxic exposure. However, additional mechanisms, including changes in blood volume and afferent input from pulmonary baroreceptors, contribute arterial to increased sympathetic vasomotor output during continuous exposures [3].

Based on the stated studies, acute exposure to a fixed altitude will increase systolic and diastolic BP, but the effects of long-term exposure to altitude by gradual ascent method on BP have not been investigated in studies.

In the present study, we observed that with a gradual increase in altitude and climbing in classic method, systolic and diastolic BP showed a significant decrease. By examining the data and comparing it with the normal values of systolic and diastolic BP (normal BP for systolic BP is below 120 mmHg and for diastolic BP is below 80 mmHg) [23]. We found that mountaineers, being at a high altitude in an acute form, i.e. in Federation (altitude 2000 m) and Base Camp (3100 m= BC), who were placed at that altitude with a vehicle, had higher BP than normal.

Based on the filled questionnaire, mountaineers did not have high BP disease, which shows the rapid presence at high altitude increases BP which confirms the findings of previous studies. But, what is important for us in this research that has not been noticed in previous studies, is that the gradual ascent and staying overnight at altitude (the classic method) moderates this increase in BP and brings it closer to the normal level. It can be said that at the same time that high altitude is considered the cause of BP increase, the gradual presence modulates the effect the high altitude.

Exposure to high altitude is widely considered a cardiac stressor and is associated with altered cardiac function. Our data shows a significant increase in HR during the climb to Damavand peak.

It was reported that exposure to high altitude increases HR [23]. Ascent to high altitude increases HR both at rest and during exercise compared to sea level. This increase usually appears at altitudes above 3050 m. An increase in HR is associated with an increase in BP. Tachycardia is related to the increased activity of the sympathetic system, which stimulates beta receptors in the heart. An increase in the level of plasma and urinary catecholamine accompany this response [15].

As a result of adaptation to altitudes above 3000 m, the increase in HR is maintained and 2 to 3 weeks later it decreases slightly to sea level values [5]. Our data showed that despite the gradual ascent to a high altitude, a significant increase in the HR of the climbers was seen, which indicates that in order to adapt the HR to HA, a long-term stay at altitude is needed and a gradual ascent to Damavand peak within 4 days does not create this adaptation.

In the investigation of pulmonary factors, we found that the gradual ascent to Damavand peak (5671 m) did not have a significant effect on the ventilatory factor, FVC and did not cause a change in this important factor that indicates the correct functioning of lung elasticity properties [12].

Of course, many studies in the past have pointed to the reduction of this factor at HA [<u>17</u>, <u>25</u>, <u>26</u>, <u>27</u>, <u>28</u>, <u>29</u>, <u>30</u>]. We also observed this reduction (although it was not significant) during rapid ascent to BC (3100 m), but the gradual ascent moderated this decrease and we did not observe significant changes at the peak (5671 m). Also, by examining the ventilatory factor FEV1, which is one of the main measures of lung function that indicates exhalation power [<u>11</u>] and airway resistance to air flow [<u>12</u>], no significant change was observed with gradual ascent to Damavand peak.

Previous researches stated that FEV1 increases with increasing altitude [25, 27, 30, 31] and this increase is due to the decrease in airway resistance due to the decrease in air density [25, 31], but by examining the averages of each stage, we found that rapid presence at altitude, for example, presence in BC, decreases FEV1, and the ascent of a mountaineer in the form of classic method causes an increase, and with the continuation of the process of climbing to a higher altitude and increasing

the duration of staying at altitude, we observed an adjustment. and decrease again. An interesting point is that after each stage of night staying that we had in BC and C1, we observed a decrease and adjustment in this factor. But, we observed that after each night sleep in the early hours of the increasing in altitude it increases, and then with the continuation of the ascent and the increase in the time spent at altitude, it adjusts and decreases, which leads us to the conclusion that the gradual ascent causes adaptation of the FEV1 factor and moderates the effects of rapid presence at altitude.

However, it should be noted that these changes were not significant. MVV is a practical dynamic method to measure the working capacity of respiratory muscles [32] and in fact to test the overall function of the respiratory system. This variable not only is affected by the strength of the respiratory muscles, but also by the conditions of the ventilation control systems and the resistance of the airways and tissues.

Previous researches have shown that MVV decreases after 24 hours of presence at 3110 m [25] and 4578 m [17], but Forte et al. (1997) reported a 20% increase in this index with rapid exposure at 4200 m [25]. Our investigations in this method of ascent show that rapid presence at 3100 m (BC) increased MVV and after staying overnight in BC and C1 it was moderated and decreased.

5. Conclusions

It can be concluded from the results of the present study that due to its nature (climbing to HA and staying overnight at low altitudes) and having frequent overnight stays, the classic method causes modulation in cardiovascular and respiratory function and makes climbing to higher altitudes without problems for climbers. Therefore, we suggest for climbers who intend to climb to higher altitudes (above 4000 m) to use the classic method of climbing.

Conflict of interest

The authors declared no conflicts of interest.

Authors' contributions

All authors contributed to the original idea, study design.

Ethical considerations

The authors have completely considered ethical issues, including informed consent, plagiarism, data fabrication, misconduct, and/or falsification, double publication and/or redundancy, submission, etc.

Data availability

The dataset generated and analyzed during the current study is available from the corresponding author on reasonable request.

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References

- Graham BL, Brusasco V, Burgos F, et al. "ERS/ATS standards for single-breath carbon monoxide uptake in the lung". *Eur Respir J*. 2017; 3;49(1):1600016. doi: 10.1183/13993003.00016-2016.
- [2] Apte ChV, Rao KS. "The maximum expiratory Flow- volume loop in natives of adakh and Acclimatized lowlanders". *High Alt Med Biol.* 2005; 6(3): 209-14.
- [3] Simpson LL, Steinback CD, Stembridge M, Moore JP. "A sympathetic view of blood pressure control at high altitude: New insights from microneurographic studies". *Journal of Experimental Physiology*. Dec 2020; 106(2): 377-384. doi: 10.1113/EP089194.
- [4] Baldi S, Ruffini E, et al. "Does lobectomy for lung cancer in patients with chronic obstructive pulmonary disease affect lung function

multicenter national study?". J Thorac Cordiorasc Surg. 2005 Dec; 130(6): 1616-22.

- [5] Farrell PA, Joyner MJ, Caiozzo VJ. Advanced Exercise Physiology. Ed 2, LWW 2012.
- [6] Veloz AFV, Riss AKY, de Zeeuw ChI, Arias TVC, Veloz MFV. "Blood pressure in andean adults living permanently at different altitudes". *High Altitude Medicine and Biology*. 2020; 21(4). doi: 10.1089/ham.2019.0101.
- [7] Boskabady MH, Tashakory A. et al. "Prediction equations for pulmonary function values in healthy young Iranians aged 8-18 years". *Respirology*. 2004 Nov; 9(4): 535-42.
- [8] Kim MK, Hwangbo G. "The effect of position on measured lung function in patients with spinal cord injury". J Physical Therapy Sci. 2012; 24(8): 655-7. doi: doi.org/10.1589/jpts.24.655.
- [9] Tartibian B, Sharif R. "Central and environmental". *Physiological Adaptations in Sports*. Urmia University, Jilad Publications. 2000. [in Persian]
- [10] Powers Scott K, Hawley Edvard T. Exercise Physiology: Theory and Application to Fitness and Performance. Vol 1. Translation by Tartibian B. Urmia University, Jilad Publications. 1998. [in Persian]
- [11] Mac Ardel WD, Katch FI, Katch VL. *Exercise Physiology (Energy, Nutrition and Human Performance)*. Translated by Khaledan A. Tehran: Samt Publication. 2001. [in Persian]
- [12] Joshi LN, Joshi VD. "Effect of forced breathing on ventilation function of the lung". *J Postgrad Med.* 1998; 44: 67-9.
- [13] Vittore Verrati MD, Ferrare C, Sorana D, Zambon A, et al. "Effect of high alttitude trekking on blood pressure and on asymmetric dimethylarginine and isoprostane production: Resalts from Mount Ararat expedition". *National Library of Medicine*. 2020 Aug; 22(8): 1494-1503. doi: 10.1111/jch.13961.
- [14] Herb Haltgren MD. High Altitude Medicine. United States of America, California. 1997.
- [15] Murdoch DR, Pallard A. The High-Altitude Medicine Handbook (Mountaineering). Translation by Shahbazi J, Mohaggeg Sh. Ardabil. Azar Sabalan Publication. 2001. [in Persian]
- [16] Salahi M. Ascent Methods in Mountaineering. Mountain. Issue 36. 2004. [in Persian]
- [17] Hussain MM, Aslam M. "Hypoxia and pulmonary acclimatization at 4578 m altitude: The role of acetazolamide and dexamethasone". *J Pak Med Assoc.* 2003; 53(10): 451-8.
- [18] Savonitto S, Cardellino G, Doveri G, et al. "Effects of acute exposure to altitude (3,460 m) on blood pressure response to dynamic and isometric exercise in men with systemic hypertension". *Am J Cardiol.* 1992; 70: 1493-1497
- [19] Vogel JA, Harris CW. "Cardiopulmonary responses of resting man during early exposure to high altitude". *J Appl Physiol*. 1967; 22: 1124-1128.
- [20] Rostrup M. "Catecholamines, hypoxia and high altitude". Acta Physiol Scand. 1998; 162: 389-

399.

- [21] Bestle MH, Olsen NV, Poulsen TD, et al. "Prolonged hypobaric hypoxemia attenuates vasopressin secretion and renal response to osmostimulation in men". *J Appl Physiol*. 2002; 92: 1911-1922.
- [22] Parati G, Bilo G, Faini A, et al. "Changes in 24 h ambulatory blood pressure and effects of angiotensin II receptor blockade during acute and prolonged high-altitude exposure: A randomized clinical trial". *Eur Heart J.* 2014; 35: 3113-3122. doi: 10.1093/eurheartj/ehu275
- [23] Gaiton A, Hall E. *Medical Physiology*. Translated by Sepehri H, Rastgarzadeh A. Vol 1. 2007. Second ed. [in Persian]
- [24] Jingdu T, Chuan L, Yuanqi Y, Shiyong Y, et al. "Effects of baseline heart rate at sea level on cardiac responses to high-altitude exposure". *Int J Cardiovasc Imaging*. 2020; 36(5): 799-810. doi: 10.1007/s10554-020-01769-w.
- [25] Forte VA JR, Leith De. et al. "Ventilatory capacities at sea level and high altitude". *Aviat Space Environ Med.* 1997; 68(6): 488-93.
- [26] Basu Ck, selvamurthy W. et al. "Respiratory changes during initial days of acclimatization to

increasing altitude". Aviat Space Environ Med. 1996; 67(1): 40-5.

- [27] Hashimoto F, MC Williams B, Qualls C. "Pulmonary ventilatory function decreases in proportion to increasing altitude". *Wilderness Environ Med.* 1997; 5(4): 214-7.
- [28] Compte Torrero L, Real Soriano RM, et al. "Respiratory changes during ascension to 8000 meters mountain". *Med Clin (Barc)*. 2002; 118(2): 47-52.
- [29] Deboeck G, Moraine JJ, Naeije R. "Respiratory muscle strength may explain hypoxia- induced decrease in vital capacity". *Med Sci Sports Exerc.* 2005; 37(5): 754-8.
- [30] Marinho PE, Berenguer A, et al. "Which pulmonary volume should be used in physiotherapy to obtain higher maximal inspiratory pressure in COPD patients?". *Physiother Res Int.* 2005; 10(4): 182-9.
- [31] Gautier H, Peslin R, et al. "Mechanical properties of the lungs during acclimatization to altitude". J Apple Physiol. 1982; 52(6): 1407-15.
- altitude". J Apple Physiol. 1982; 52(6): 1407-15.
 [32] Ioannis Heliopoulos, MD, Georgios P. "Maximal voluntary ventilation in myasthenia gravis". Muscle Nerve. 2003; 27(6): 715-9.