

Changes in Spirometric Parameters and Arterial Oxygen Saturation During a Mountain Ascent to Over 3000 Meters

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OBJECTIVE: To ascertain whether climbing a mountain over 3000 meters high produces any alterations in ventilation, whether such alterations are modified by acclimatization, and whether they correlate with changes in arterial oxygen saturation (SaO₂) or the development of acute mountain sickness (AMS).

SUBJECTS AND METHODS: The following parameters were measured in 8 unacclimatized mountaineers who climbed Aneto (3404 m) and spent 3 days at the summit: forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁), airway response to inhaled terbutaline, SaO₂, and the symptoms of AMS.

RESULTS: At the summit, mean (SD) FEV₁ declined by 12.3% (5.7%) and mean FVC by 7.6% (6.7%) while the ratio of FEV₁ to FVC remained normal. The means for both parameters were higher on the following day. No airway response to bronchodilator treatment was observed. The restriction disappeared entirely on descent. At the peak, SaO₂ increased progressively as the climbers became acclimatized. During the ascent, FEV₁ correlated with SaO₂ ($r=0.79$). One participant who suffered from AMS had a ratio of FEV₁ to FVC less than 70% and the worst SaO₂ during the 3 days on the summit. Obstruction preceded the AMS symptoms, did not respond to bronchodilator treatment, and disappeared when the climber descended.

CONCLUSIONS: The mountaineers who climbed over 3000 meters presented restriction that correlated with hypoxemia. This restriction did not respond to bronchodilator treatment, improved with acclimatization, and disappeared on descent. One person with AMS presented obstruction that did not respond to terbutaline and disappeared on descent.

Key words: Spirometry. Arterial oxygen saturation. Acute mountain sickness. Acclimatization.

Cambios espirométricos y en la saturación arterial de oxígeno durante la ascensión a una montaña de más de 3.000 metros

OBJETIVO: Averiguar si en la ascensión a una montaña de más de 3.000 m se produce alguna alteración ventilatoria, si ésta se modifica por la aclimatación y si se relaciona con los cambios en la saturación arterial de oxígeno (SaO₂) o con la aparición de síntomas de mal de montaña agudo (MAM).

SUJETOS Y MÉTODOS: En 8 montañeros no aclimatados que ascendieron a la cumbre del Aneto (3.404 m) y permanecieron 3 días en ella medimos: la capacidad vital forzada (FVC), el volumen espiratorio forzado en el primer segundo (FEV₁), la respuesta a la inhalación de terbutalina, la SaO₂ y los síntomas de MAM.

RESULTADOS: Al llegar a la cumbre disminuyeron el FEV₁ ($12,3 \pm 5,7\%$) y la FVC ($7,6 \pm 6,7\%$) con la relación FEV₁/FVC% normal. Al día siguiente aumentaron ambos parámetros. No hubo respuesta al tratamiento broncodilatador. La restricción se corrigió totalmente al descender. La SaO₂ en la cumbre aumentó progresivamente con la aclimatación. Durante la ascensión el FEV₁ se correlacionó con la SaO₂ ($r = 0,79$). Un participante con MAM presentó FEV₁/FVC menor del 70% y la peor SaO₂ durante la estancia en la cima. Esta obstrucción precedió a los síntomas, no cedió con tratamiento broncodilatador y se corrigió con el descenso.

CONCLUSIONES: Los montañeros que ascienden a montañas de más de 3.000 m presentan una restricción que se correlaciona con la hipoxemia, no mejora con el tratamiento broncodilatador, se alivia con la aclimatación y desaparece con el descenso. Un sujeto con MAM sufrió una obstrucción que no respondió a la terbutalina y desapareció con el descenso.

Palabras clave: Espirometría. Saturación arterial de oxígeno. Mal agudo de montaña. Aclimatación.

Introduction

Little is known about the changes that occur in pulmonary airflows and volumes when people climb high mountains. Most of the published studies on this

intriguing subject report that a restrictive pattern is found at high altitudes, although some authors observed no changes.¹⁻¹³ A number of possible explanations for this restrictive defect have been proposed, including an increase in pulmonary vascular flow, interstitial, and/or alveolar edema, muscle fatigue, and changes in pulmonary volumes brought about by air trapping and bronchoconstriction.¹⁴⁻¹⁹

To date, most studies have been carried out on subjects in hypobaric chambers, on subjects who have traveled up

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a mountain by mechanical means, or else on mountain expeditions of long duration. While hypobaric chambers and mountain locations with access via mechanical transport are ideal for complex studies of hypoxic conditions, such studies do not take into account the effect of the factors that may be inherent in the process of climbing the mountain on foot. Studies carried out on long expeditions do not have this shortcoming, but they involve conditions that make it much more difficult to adhere strictly to a study protocol.

Many field studies have been carried out during approaches to base camps or similar locations during which the climbers acclimatize gradually. However, this type of expedition has little in common with the high mountain activities of the immense majority of European mountaineers, who generally climb mountains of between 2500 meters (the lower limit of high altitude) and 4808 (the height of Mont-Blanc) over a weekend or during a holiday.

The objective of this study was to ascertain whether a typical weekend ascent of a mountain over 3000 meters high produces any alterations in ventilation, whether such alterations are modified by acclimatization, and whether they correlate with changes in arterial oxygen or the appearance of the symptoms of acute mountain sickness.

Subjects and Methods

Eight unacclimatized mountaineers drove from Valencia to Benasque, where they spent the night. The following day they climbed to the summit of Aneto (3404 meters) and they then camped for 2 nights at 3350 meters near the Puente de Mahoma. The group comprised 2 women and 6 men, and the mean (SD) age was 32.5 (6.6) years. Spirometry and pulse oximetry were performed on all 8 mountaineers in Valencia (elevation 20 m), in Benasque (1138 m), at the La Renclusa mountain refuge (2140 m), at the Portillon Superior (pulse oximetry only, 2870 m), at the summit of Aneto (3404 m), on arrival at the high mountain campsite (3350 m), and on the second and third days spent at this camp. In order to provide data for comparison with the results obtained during the ascent, the measurements were repeated in the same places on the way down.

On the second day spent at high altitude, the subjects inhaled 1.5 mg of terbutaline in the form of a dry powder and spirometry was repeated 15 minutes later in order to detect the presence of any bronchostriction that might respond to bronchodilator treatment.

Atmospheric pressure and temperature were measured with a portable Module 950 Alt 6000 combination barometer and thermometer (Casio Computer Company, Tokyo, Japan).

Spirometry was performed with a VM1 Ventilometer (Clement Clarke International, Edinburgh, United Kingdom), a device equipped with a pressure transducer that can measure peak expiratory flow rate and a microprocessor that digitizes the signal. The parameters studied were forced vital capacity (FVC) expressed in liters, forced expiratory volume in 1 second (FEV₁) in liters, peak expiratory flow rate (PEFR) in liters per minute, and the ratio of FEV₁ to FVC. Spirometry was performed with the subjects standing and in accordance with the recommendations of the Spanish Society of Pulmonology and Thoracic Surgery (SEPAR).²⁰ Results were also then expressed as percentages of the reference values.²¹

The results found in the course of our study led us to suspect that the lower density and viscosity of the air at high altitude had caused the spirometer we used to underestimate the volumes measured.²² A second ascent was undertaken in order to investigate this effect and obtain the data necessary to correct the results of the first expedition. The destination on this occasion was the summit of Veleta (3396 m) in the Cordillera Penibética. A 3-liter calibration syringe (Sibelmed, Barcelona, Spain) was used to perform 5 measurements of volume on different flows at known altitudes including the summit. We used this data to create a regression equation that would allow us to quantify the percent underestimation of volume depending on elevation. The equation for the percent underestimation of volume (%) was $0.2454 + 0.0044 \times \text{meters}$ (elevation in meters). The variability of the measurements increases with decreasing atmospheric pressure (the coefficient of variation at 3396 meters is 1.7%), but this effect is too slight to create problems when the correction formula is applied.²³

Arterial oxygen saturation (SaO₂) was measured with a CSI 503 SpO₂T portable pulse oximeter (Criticare Systems Inc, Waukesha, Wisconsin, USA), a device with a resolution of 1% and an accuracy of 2%. The measurements were performed at rest, with the individual in a sitting or standing position. The SaO₂ value was accepted when the screen showed a reasonably stable result; when the measurement fluctuated, the result was taken to be the mean of several figures.

We tested the accuracy of the pulse oximeter before using it in the mountains by comparing its findings with results of arterial blood gas analysis in our respiratory function research laboratory at Hospital La Fe (intraclass correlation coefficient of 0.86). In addition, to make sure that this device functioned properly at low temperatures we tested it during one afternoon at -24°C in an industrial cold room (Friomer, Valencia, Spain).

The diagnosis of mountain sickness was confirmed when a subject scored 3 points or higher on the Lake Louise scoring system.²⁴⁻²⁷

Statistical Analysis

The results of our study are expressed as means (SD). The relationships in the values obtained at different elevations and the groups established according to acclimatization were analyzed using repeated measures analysis of variance. The relationship between spirometric data and SaO₂ was analyzed using linear correlation (the Pearson coefficient). Differences were considered significant when the *P* value was less than .05.

Results

Atmospheric pressure was as follows: in Valencia, 100.9 kPa; in Benasque, 89.5 kPa on the ascent and 88.8 kPa on the descent; in La Renclusa, 78.7 kPa on the ascent and 78.5 kPa on the descent; and at the Portillon Superior it was 71.7 kPa on both occasions. During our stay in the vicinity of the summit of Aneto, atmospheric pressure was 67.6 kPa on the first day, 67.8 kPa on the second, and 67.4 kPa on the third.

Spirometric Parameters

All of the participants had normal spirometry in Valencia. The table and Figure 1 show how the spirometric variables and SaO₂ evolved over the course of the study.

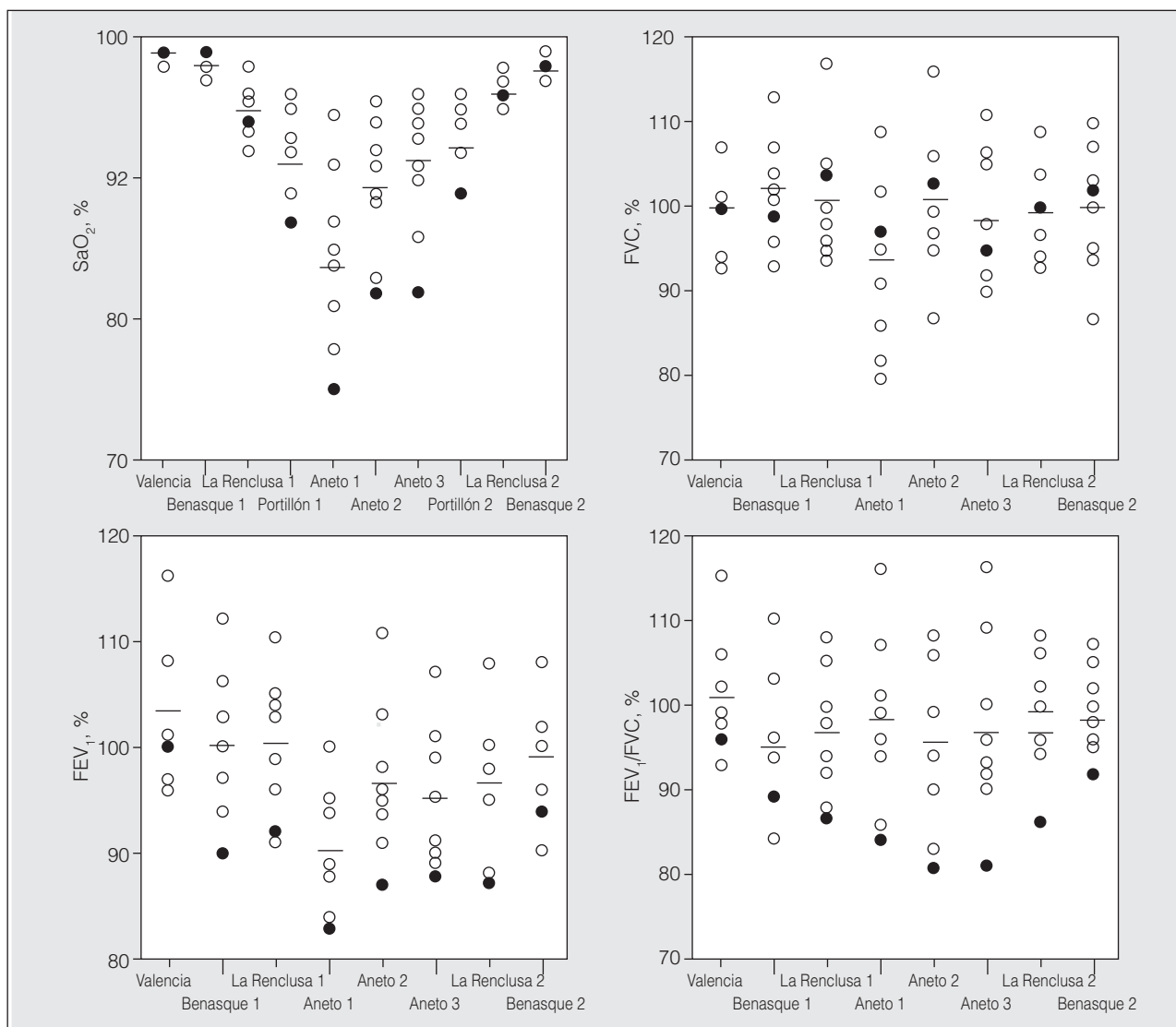


Figure 1. Spirometry and arterial oxygen saturation (SaO_2) in Valencia and during the ascent in Benasque (Benasque 1), La Renclusa (La Renclusa 1), Portillón Superior (Portillón 1), on arrival at the summit of Aneto (Aneto 1), on the second day near the summit (Aneto 2), on the third day near the summit (Aneto 3), and during the descent in Portillón Superior (Portillón 2), La Renclusa (La Renclusa 2), and Benasque (Benasque 2). The black circles indicate the values from the subject affected by mountain sickness. The horizontal line represents the arithmetic mean for the group. SaO_2 indicates arterial oxygen saturation; FEV_1 , forced expiratory volume in 1 second; and FVC, forced vital capacity.

The values recorded on arrival at the summit of Aneto were significantly lower than those recorded at sea level. The decrease was 12.3% (5.7%) in FEV_1 and 7.6% (6.7%) in FVC. The 9.1% (6%) decrease in PEFR was not statistically significant, and the ratio of FEV_1 to FVC remained normal with a value of 78.4% (9%).

As pulmonary flows and volumes increased after 24 hours at 3350 meters, the differences between these values and the results obtained in Valencia were no longer statistically significant. At this time, the inhalation of terbutaline 1.5 mg did not produce any appreciable change. The increases, expressed as percent changes, were 1.9% (2.6%) for FVC, 1.3% (1%) for FEV_1 , and 1.5% (3.2%) for PEFR.

On the third day spent near the summit, we observed a slight limitation in the spirometric parameters, but the change was only significant in FEV_1 . Although the results obtained during the descent tended to be lower than those observed at the same elevations during the ascent, the differences were not significant.

Arterial Oxygen Saturation

SaO_2 values decreased progressively with altitude. The mean value on arrival at the summit of Aneto was 84.4% (6.5%). During the days spent on or near the summit—and particularly during the first 24 hours— SaO_2 improved as a result of acclimatization (Table and Figure 1).

TABLE
Lung Function Parameters Recorded at Different Times and Altitudes*

Parameter, Unit	Altitude and Times of Measurement							
	20 m	1138 m, Ascent	2140 m, Ascent	3355 m, 1st Day	3355 m, 2nd Day	3355 m, 3rd Day	2140 m, Descent	1138 m, Descent
FVC, mL	5005 (551)	5098 (677)	5056 (624)	4630 (650)	5058 (753)	4899 (564)	4915 (536)	4980 (634)
FVC, % pred	100 (5)†	102 (6)†	101 (8)†	93 (10)	101 (8)†	98 (8)	99 (6)	100 (7)
FEV ₁ , mL	4061 (387)†	3950 (512)†	3895 (271)	3554 (300)	3821 (379)	3759 (410)	3816 (384)	3920 (324)
FEV ₁ , % pred	103 (7)†	100 (7)†	100 (7)†	90 (6)	97 (7)†	95 (7)†	97 (7)	99 (7)†
PEFR, L/s	9.96 (1.58)	9.59 (1.48)	9.80 (1.47)	9.01 (1.19)	9.43 (1.38)	9.36 (1.66)	9.56 (1.41)	9.86 (1.55)
PEFR, % pred	107 (11)	104 (10)	106 (10)	98 (10)	101 (10)	101 (11)	104 (10)	107 (10)
FEV ₁ /FVC, %	81.2 (6.5)	75.9 (7.6)	77.4 (7.1)	78.4 (8.9)	76.9 (8.7)	77.5 (9.6)	77.6 (6.7)	78.8 (5.2)
FEV ₁ /FVC, % pred	101 (7)	95 (8)	97 (8)	98 (11)	96 (11)	97 (11)	97 (8)	98 (6)
SaO ₂ , %	98.8 (0.5)†,‡	98.1 (0.8)†,‡	95.1 (0.8)†,‡,§	84.4 (6.5)§	89.4 (4.8)†,§	90.9 (4.8)†,§	96.1 (1.1)†,‡	98 (0.5)†,‡

*% pred indicates that values are percentages of reference values; FEV, forced vital capacity; FEV₁, forced expiratory volume in 1 second; PEFR, peak expiratory flow rate; and SaO₂, arterial oxygen saturation. All results are mean (SD).
†Significantly different from the measurement recorded at the summit on the first day, $P < .05$.
‡Significantly different from the measurement recorded at the summit on the second and third days, $P < .05$.
§Significantly different from the measurement recorded at 20 meters, $P < .05$.

A significant correlation (Figure 2) was observed between the percent decrease in FEV₁ during the ascent and the SaO₂ value on arrival at the summit ($r=0.79$). A correlation was also found between SaO₂ on arrival at the summit and the value observed on the third day before leaving the summit ($r=0.75$).

Acute Mountain Sickness

Two individuals had persistent headache during their stay on the summit, but only one of them fulfilled the diagnostic criteria for acute mountain sickness according to the Lake Louise scale (he also suffered nausea and reduced performance on effort). This participant had a decrease in FEV₁ of 17% and in PEFR of 10%, and his ratio of FEV₁ to FVC remained below 70% during his whole stay on the summit (Figure 1). This airflow limitation preceded the appearance of symptoms, did not respond to inhalation of terbutaline (FEV₁ only increased by 2%, FVC by 4%, and PEFR

by 5%), and clearly disappeared on descent. This individual had the worst SaO₂ on arrival at the summit (75%), at the end of his stay on the summit (82%), and during the whole period the group remained above 2500 meters. The symptoms of acute mountain sickness disappeared on descent.

Discussion

Our results show that unacclimatized individuals are affected by a decline in pulmonary airflows and volumes when they ascend to over 3000 meters. The impairment observed is restrictive, characterized as it is by a decline in FVC while the ratio of FEV₁ to FVC remains within normal reference limits.

The 7.6% decrease in FVC observed on arrival at the summit of Aneto may seem excessive when compared with the results of other studies.^{3,6,8} However, these differences can probably be explained by the fact that we took the measurements immediately upon arrival at the summit and our subjects were unacclimatized mountaineers who had just made a considerable effort. Moreover, the decline we observed in FEV₁ and FVC values was predicted by a study undertaken by Hashimoto et al.¹³

The interpretation of spirometric findings measured at high altitude is complicated.^{5,28,29,32} It is possible that the increase one might have expected in PEFR was offset in our study by the bias introduced by our spirometer. However, neither that bias nor the corrective equation we applied to the data undermine the credibility of the differences between the spirometric results obtained over the 3-day period at the same altitude using the same spirometer.

In our opinion, the changes observed may have been secondary to subclinical pulmonary edema.^{4,6,14,16} This problem appears to be somewhat more common in subjects affected by acute mountain sickness.^{24,26,27,31}

The decrease in FVC may, as various authors have suggested, be related to an increase in pulmonary vascular flow caused by hypoxia.^{6,15,16} However, the

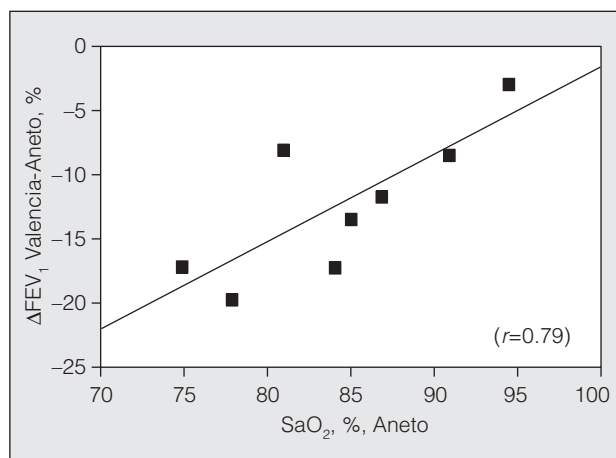


Figure 2. Linear relationship (r , the coefficient of correlation) between the percent decrease in forced expiratory volume in 1 second (FEV₁) between Valencia and arrival at the summit of Aneto— Δ FEV₁ Valencia-Aneto (%)—and arterial oxygen saturation (SaO₂) on arrival at the summit.

results of the study we carried out during the University of Valencia's Gasherbrum II expedition would argue against this hypothesis.¹

Another possible explanation is muscle fatigue resulting from the effort made by the climbers in hypoxic conditions.^{10,11} This mechanism has never been demonstrated, and its existence is contradicted by the results of our research cited above.¹ While the participants in the present study were tired when they reached the summit of Aneto, as one would expect after climbing one of the high Pyrenean peaks, their physical state was far from exhaustion. The subjects rested for a brief period before spirometry was performed; this interval was more than 15 minutes for the first individual tested. We do not, therefore, think that the spirometric changes observed can be attributed to muscle fatigue.

It has been reported in the literature that hypoxia, hypobarica, and hypocapnia can cause bronchospasm.^{18,19} However, this phenomenon has not been observed in a number of studies carried out in hypobaric chambers and at high altitudes,^{1-6,12,13} and neither did we observe any response to inhaled terbutaline on the summit of Aneto. Even the mountaineer affected by mountain sickness, who had an obstructive ventilatory defect, did not improve after inhaling this β_2 -agonist. This finding is consistent with our hypothesis that the cause of the airflow limitation is airway edema.

The way altitude-related spirometric changes evolve on acclimatization has been analyzed by various researchers. Some authors have reported a return to normality^{1,9,11} or a trend towards improvement that is not statistically significant.³ In other studies, FVC did not return to normal on acclimatization, and pulmonary volumes only recovered when the subjects descended^{2,8,13} or 2³ or 7⁶ days after the descent. In our study, we observed marked recovery of pulmonary flows and volumes during the first day the mountaineers spent in the vicinity of the summit; the slight limitation that still persisted on the third day disappeared completely on the descent.

It is known that physical effort can give rise to alterations in pulmonary volumes^{32,33} and facilitate high-altitude pulmonary edema.^{24,27} It is not surprising that our results do not coincide with those published by Selland et al⁷ and Ge et al,⁴ who observed no significant spirometric changes in healthy individuals, since the former used a hypobaric chamber and in the latter study the participants were transported to high altitude. The participants in those studies made no physical effort whatsoever.

To our knowledge, this is the first time that this decrease in pulmonary volumes has been observed in the Pyrenees, and also the first time that such rapid recovery has been observed during the early phase of acclimatization. Although of some importance, therefore, this is an ephemeral phenomenon that we have been able to observe by studying unacclimatized individuals who actually climbed to the summit on foot.

The practical interest of our finding is obvious, since this field study reflects the real conditions faced every year by tens of thousands of mountaineers who climb the higher peaks of the Pyrenees and the Alps.

Several researchers have studied the relationship between spirometric parameters and acute mountain sickness.^{4,5,7,35-37} In line with previous findings, the only individual affected by acute mountain sickness in our study presented an obstructive ventilatory defect. It is very possible that the altered spirometric pattern found in individuals with acute mountain sickness may represent an early sign of high-altitude pulmonary edema.

Analysis of the relationship between spirometric changes and hypoxemia revealed that the participants in our study who had the lowest SaO₂ values also had the greatest reduction in FEV₁ during the ascent. This finding supports our hypothesis, also proposed by Bärtsch et al,³⁸ that both these phenomena are caused by pulmonary edema. The relationship we found between SaO₂ measured on arrival at the summit and the same variable measured at the end of the stay in that vicinity is an indication that individual idiosyncrasies may be found in adaptation to altitude.

Our conclusion is that unacclimatized mountaineers who climb to peaks above 3000 meters are affected by a restrictive ventilatory defect revealed by decreases in FVC and FEV₁ while the ratio of FEV₁ to FVC remains within normal reference limits. This restriction *a*) does not respond to bronchodilator treatment; *b*) correlates with the decrease in SaO₂; *c*) resolves to a large degree on acclimatization, especially during the first 24 hours spent at high altitude; and *d*) disappears entirely on descent. We believe that this restriction could be caused by subclinical pulmonary edema. In the single individual who had acute mountain sickness an obstructive defect was observed that *a*) preceded the development of headache; *b*) did not respond to inhaled terbutaline; *c*) persisted throughout the entire stay at high altitude; and *d*) disappeared on descent. This obstruction may also occur in other individuals who have acute mountain sickness.

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