

# Acute mountain sickness: predictors of climbers' performance at high altitudes

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None

**Objective:** To determine the relationship between climber variables (lactic acid levels, blood pressure, heart rate, respiratory frequency, and peripheral oxygen saturation [SaO<sub>2</sub>]) and their physical performance and development of acute mountain sickness (AMS) on the Lullailaco volcano (6739 m) in Argentina.

**Material and methods:** As 8 climbers ascended the mountain, we measured lactic acid and other clinical variables, including SaO<sub>2</sub>. We also assessed visual acuity (Pelli-Robson chart), AMS (Lake Louise scoring). SaO<sub>2</sub> and heart rate were assessed at rest and on a 6-minute walk test.

**Results:** Lactic acid levels of 2 mmol/L or more at 5000 m predicted worsening of a climber's condition at higher altitudes, progression to AMS, and declining performance (positive predictive value, 66.7%; negative predictive value, 100%; precision, 87.5%). Values on performance of the 6-minute walk test at 5000 m showed that a change of 20 beats/min in heart rate and a 10% change in SaO<sub>2</sub> predicted worsening condition at higher altitudes (specificity, 100%). Likewise, a Lake Louise score of 4 or more at 5000 m also predicted worsening climber condition and AMS.

**Conclusions:** Clinical variables and laboratory findings for mountain climbers in base camps can predict their performance at higher altitudes. A lactic acid level of 2 mmol/L or more, a change in SaO<sub>2</sub> of more than 10% after the walk test and a Lake Louise score of 4 or more predicted worsening of clinical signs. [Emergencias 2011;23:276-282]

**Key words:** Predictors. Clinical variables. Acute mountain sickness.

## Introduction

Little is known about physical performance at high altitude, but it seems that as from 5,000 m above sea level (asl), hypobaric hypoxia has a detrimental effect on gas exchange and on enzyme and mitochondrial metabolism<sup>1,2</sup>. It leads to accelerated ventilation and decreased exercise performance, loss of weight and muscle mass and the possibility of developing acute mountain sickness (AMS) with subsequent cerebral edema<sup>3,4</sup>.

The diagnosis of AMS is based on the Lake Louise consensus criteria which include headache and other symptoms of altitude, and which provides a severity score<sup>5</sup>.

Lactic acid (LA) is related to energy production capacity, both in physiological processes (exercise)

and in pathological processes (sepsis, trauma, shock, etc.)<sup>6-8</sup>. Little is known about the relationship between endpoints, such as heart rate (HR), and LA levels, and even less in the context of high altitude sports. HR increases with effort up to a certain point, known as the HR deflection point, and then stabilizes. Hypobaric hypoxia causes a sustained increase in ventilation. This initial increase in ventilation is achieved by an increase in tidal volume and peak flow, while maintaining a constant respiratory rate (RR). Increased HR occurs as a late response to severe hypoxia. Arterial oxygen saturation (SaO<sub>2</sub>) correlates to hypoxia with greater sensitivity<sup>9</sup>.

Measuring blood levels of LA in athletes, despite the existence of many studies, remains a controversial issue. According to Conconi et al.

these levels constitute a very important diagnostic and prognostic tool in sport<sup>10</sup>. Despite this assertion, LA has for years been considered as the waste product of glycolysis which has the effect of blocking the synthesis of adenosine triphosphate (ATP) causing muscle fatigue. However, new studies have changed the conception of LA as a product harmful. In-depth studies have been carried out on the production of LA or lactate and cellular mechanisms for its removal and transportation. These mechanisms allow reversible conversion of lactate to pyruvate, either in the cytoplasm of the cell where it was produced or in other body cells (muscle groups, heart, liver, etc.). Thus lactate is today considered a fuel. The transport and re-use of lactate are known as the lactate shuttle. This process is important in intense exercise, since it eliminates the residual lactate and acts as a glucose saver during the whole process.

In recent years, lactate turnover studies have shown that, during intense exercise, LA increases up to a certain level; it reaches a high concentration relative to LA at rest, but then stabilizes. Thus the rate of production and removal reach a balance. It is also necessary to point out that lactate clearance capacity is exponentially related to aerobic power, i.e. oxygen consumption.

The primary objective of this study was to determine the association between levels of LA at different heights with clinical symptoms and the development of AMS, and their relationship to other clinical parameters such as heart rate (HR), respiratory rate (RR) and peripheral oxygen saturation (SaO<sub>2</sub>). Secondary objectives were to relate HR and peripheral SaO<sub>2</sub> values at rest with those obtained after the 6-minute walking test at different altitudes, to determine whether AMS could be predicted at higher altitudes and detect incipient symptoms of AMS using clinical scores and visual tables of contrast sensitivity.

## Method

The study included 8 male volunteers, non-smokers, who were experienced mountain climbers. Their objective was to reach the top of the 4th highest volcano in the world: Lullullaico, 6,739 meters (m) high, located in Argentina on the border with the Republic of Chile. All were experienced in climbing to 5,000 m without significant complications. One of them lived at 3,500 m. (Subject No. 5). All signed informed consent to participate in the study.

Initial measurements were obtained at 3,500m



**Figure 1.** Pelli Robson chart.

(Pueblo Tolar Grande) and taken as baseline. Altitudes were determined by GPS (GarminTrex Summit). LA determinations were performed at temperatures above 15° C in a closed environment at rest at 3,500 m, 5,000 m (base camp) and on returning from the top (immediately after exercise) to the base camp by photometric enzyme reflectance (Accutrend Plus®, Roche Products). The peripheral capillary blood samples were obtained from the finger tip using disposable lancets, after confirming the absence of local circulation disorders (cooling or freezing).

Data collection was completed with clinical measures (HR, RR), SaO<sub>2</sub> (pulse oximetry), visual acuity (Pelli Robson contrast sensitivity letter chart) and Lake Louise clinical score of AMS (Figure 1, Table 1). The Pelli Robson table is used by ophthalmologists to detect incipient retinal pathologies based on visual acuity findings. At high altitudes, papilledema and retinal microbleeds may appear. The chart consists of 6 columns and 8 rows of letters of the same size but with decreasing contrast from left to right and top to bottom, so the letter at the bottom right is the faintest and therefore the most difficult to see. Contrast sensitivity is considered normal if the subject can see rows 7 and 8, slightly impaired if only up to rows 5 and 6 only, moderately im-

**Table 1.** Lake Louis score for acute mountain sickness (AMS)

Symptoms	Points
Headache	0 No headache
	1 Mild
	2 Moderate
	3 Severe, incapacitating
Gastrointestinal symptoms	0 None
	1 Poor appetite or nausea
	2 Mild nausea &/or vomiting
	3 Severe nausea &/or vomiting
Fatigue and/or weakness	0 Not tired or weak
	1 Mild fatigue/weakness
	2 Moderate fatigue/weakness
	3 Severe fatigue/weakness
Vertigo/Dizziness	0 Not dizzy
	1 Mild dizziness
	2 Moderate dizziness
	3 Severe dizziness, incapacitating
Sleep disturbances	0 Slept as usual
	1 Did not sleep as well as usual
	2 Woke many times, poor night's sleep
	3 Could not sleep at all
Mental Alterations	0 No change
	1 Lethargy /lassitude
	2 Disoriented/confused
	3 Stupor/semiconsciousness
	4 Coma
Ataxia (walking on a line heel to toe)	0 Normal gait
	1 Maneuvres to maintain balance
	2 Steps off the line
	3 Falls down
	4 Inability to stand
Edema	0 Absent
	1 One location
	2 Two or more locations

paired up to rows 3 and 4 only, and severely impaired if the subject cannot see the letters below the first two rows at the top of the chart<sup>11,12</sup>.

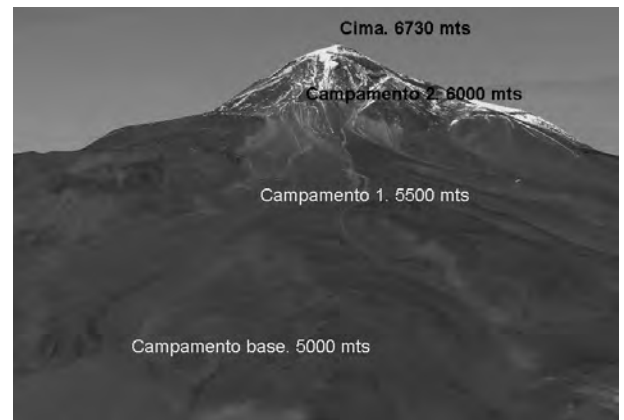
All these variables were measured at different altitudes (Figure 2): Pueblo Tolar Grande (3,500 m), Llullaillaco volcano's base camp (5,000 m), Camp 1 (5,500 m), Camp No. 2 (6000 m) and the pre-summit (6,400 m).

The 6-minute walk test was performed on the flat at 3,500m and 5,000m: SaO<sub>2</sub> and HR were measured at rest immediately after the walk<sup>13,14</sup>.

All data were recorded in individual files. Medical equipment used consisted of: electronic blood pressure (BP) and HR (Omron®) instruments, digital infrared oxymeters (Foximeter®), capillary blood LA-measuring instrument (Accutrend Plus®, Roche) strips (BM Lactate®), and plastified Pelli Robson contrast charts. The Lake Louise AMS score was also recorded.

Lake Louis score, agreed by consensus in 1993, accurately assess the severity of AMS (Table 1). The sum of the points increases with severity: 1 to 3 points (mild), 4 to 6 points (moderate) and 7 points or more (severe AMS).

Continuous variables were compared with the Wilcoxon test, given the non-normal distribution. Cat-

**Figure 2.** Llullaillaco volcano and location of the camps.

egorical variables (discrete and ordinal) were analyzed with chi-square test and Fisher test. The strength of associations was evaluated using the Mantel-Haenszel test and the odds ratio (OR) was obtained with 95% confidence intervals (CI95%). Bivariate study was conducted to assess the association of variables. Given the small sample size and low number of events, multivariate analysis was not performed. Statistical analysis was carried out using STATA.

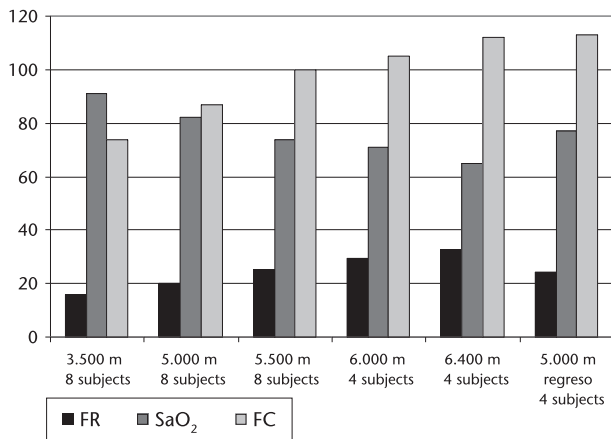
## Results

All climbers (mean age 35 years, range 26-44 years) reached 5,500 m (Camp 1), only 4 subjects reached 6,400 m high (pre-summit) and only one reached the summit of the volcano Llullaillaco, after spending 11 days at more than 3,500 m. Figure 3 shows the mean values of HR, RR, SaO<sub>2</sub> at corresponding altitudes.

Mean SaO<sub>2</sub> decreased with lower atmospheric pressure (expressed as hPa) at progressively higher altitude (3,500 m: 645 hPa, 5,000 m: 559 hPa; 5,500 m: 519 hPa; 6,000 m: 380 hPa; 6,400 m: 360 hPa) from an average 91% at 3,500 m to 65% at 6,400 m. In contrast, resting RR increased from an average of 15 breaths per minute at 3,500 m to 32 breaths per minute at 6,400 m.

Comparative data for LA levels, Lake Louis score, Pelli-Robson chart test and the 6-minute walk test at altitudes of 3,500 m and 5,000 m are shown in Tables 2 and 3.

Of the 8 climbers, subjects 7 and 8 descended to accompany subjects 2 and 6 who could not continue after reaching 5,500 m due to AMS symptoms (mild headache and vomiting) and a Lake Louis score of  $\geq 4$ . They were treated with intravenous dexamethasone and metoclopramide before descending.



**Figure 3.** Mean values of heart rate (HR) respiratory rate (RR) and oxygen saturation (SaO<sub>2</sub>) at different altitudes.

At 5,000 m, LA  $\geq 2$  mmol/l showed a sensitivity and specificity of 100% and 83.3% respectively to predict poor outcome of the clinical state at greater altitudes, AMS progression and worsening of athletic performance with a positive predictive value (PPV) of 66.7%, negative predictive value (NPV) of 100%, likelihood ratio of 6, accuracy 87.5% and  $p = 0.019$ . Relating LA and AMS  $\geq 4$ , we also found a significant association ( $p = 0.035$ ). The OR of having LA  $\geq 2$  mmol/l and an AMS score  $\geq 4$  was 1.23 (CI 95% 1.12 to 1.47), i.e., those with the LA  $\geq 2$  at 5,000 m had a 23% risk increase of suffering moderate AMS compared to those who did not have this value of LA. The area under the ROC curve was 0.91.

We analyzed the 6-minute walk test evaluating the changes ( $\Delta$ ) in SaO<sub>2</sub> and HR; at 5,000m we observed a HR  $\Delta \geq 20$  lpm and SaO<sub>2</sub>  $\Delta \geq 10\%$  with 100% sensitivity and specificity to predict poor outcome and progression of AMS at altitudes above 5,000 m (PPV 100%, NPV 100%, 100% accuracy,  $p = 0.11$  for HR and  $p = 0.088$  for SaO<sub>2</sub>). Those who had a  $\Delta$  SaO<sub>2</sub>  $\geq 10$  had

an OR of 1.15 (15% increased risk) of having AMS  $\geq 4$  compared to those who did not have that change, with a statistically significant difference (95% CI 1.06 to 3.33).

We found that a Lake Louise score of  $\geq 4$  at 5,000 m identified those who would have a poor outcome at higher elevations, with worsening of AMS and worsening of athletic performance (sensitivity, specificity, PPV and NPV of 100%,  $p = 0.021$ ). At 5,500 m, the baseline HR, RR and SaO<sub>2</sub> showed no significant alterations, unlike the walking test, for the development of AMS ( $p = NS$ ).

Regarding the data on visual acuity obtained using the Pelli Robson chart, no significant differences between climbers were found, not even in those with moderate AMS who were obliged to descend earlier.

Table 4 shows the clinical values, the Lake-Louis score and the Pelli Robson chart values of the 4 subjects who reached the maximum height of 6,400 m (no walk test) measured at 3,500, 5,000, 6,000 and 6,400 m.

At 6,400 m, the average HR was 112.5 beats per minute, RR was 32.5 breaths per minute, SaO<sub>2</sub> was 65% and Lake Louise AMS score was 3.75. On returning to base camp (5,000 m) no significant differences were observed in SaO<sub>2</sub> values, HR or RR compared to baseline measurements taken at the same height in the days before the ascent. Neither did we observe LA values  $> 1$  mmol/l in these subjects.

## Discussion

The Lake Louise consensus established the definition of AMS as the presence of headache in a non-acclimatized person ascending to an altitude of 2,500 m plus the presence of one or more of the following symptoms: gastrointestinal disorder (anorexia, nausea, vomiting), insomnia, dizziness,

**Table 2.** Results of 6-minute walk test at 3,500 meters

Subject No.	SaO <sub>2</sub> baseline	SaO <sub>2</sub> 6 min	HR baseline	HR 6 min	Change in SaO <sub>2</sub>	Change in HR	LA	Lake Louis score	Pelli Robson score
1	93	86	78	102	7	24	0.8	1	8
2	89	86	72	98	3	26	0.8	1	8
3	97	87	86	110	3	24	2.2	1	8
4	93	84	84	115	9	31	1.8	0	8
5	94	87	60	67	7	37	0.8	0	7
6	92	83	90	100	9	10	0.8	0	8
7	90	84	55	100	6	45	0.8	0	8
8	93	86	70	114	7	44	0.8	1	8
Mean $\pm$ SD	92.6 $\pm$ 2.4	85.3 $\pm$ 1.5	74.3 $\pm$ 12.4	100.7 $\pm$ 15.1	6.4 $\pm$ 2.3	30.1 $\pm$ 11.7	1.1 $\pm$ 0.5	0.5 $\pm$ 0.5	7.8 $\pm$ 0.3

SD: standard deviation; SaO<sub>2</sub>: arterial oxygen saturation, HR: heart rate, LA: lactic acid.

**Table 3.** Results of 6-minute walk test at 5,000 meters

Subject No.	SaO <sub>2</sub> baseline	SaO <sub>2</sub> 6 min	HR baseline	HR 6 min	Change in SaO <sub>2</sub>	Change in HR	LA	Lake Louis score	Pelli Robson score
1	84	77	86	100	7	14	0.8	2	8
2	80	70	100	120	10	20	2.4	4	8
3	84	80	85	91	4	6	2	2	7
4	84	77	85	102	7	17	0.8	3	8
5	82	76	76	80	6	4	0.8	0	8
6	85	75	90	110	10	20	3.9	5	8
7	81	74	75	92	7	17	0.8	1	8
8	83	79	100	107	4	7	0.8	1	8
Mean ± SD	82.8 ± 1.7	76 ± 3.1	87.1 ± 9.4	100.2 ± 12.5	6.9 ± 2.3	13.1 ± 6.5	1.5 ± 1.5	2.2 ± 1.6	7.8 ± 0.3

SD: standard deviation; SaO<sub>2</sub>: arterial oxygen saturation, HR: heart rate, LA: lactic acid.

lassitude or fatigue<sup>5</sup>. One or more of these symptoms usually occur between 6 and 10 hours of ascent. Cerebral edema, due to altitude and defined by the presence of ataxia and/or altered state of consciousness, represents the final stage of AMS. Some associated signs include papilledema, retinal hemorrhage and paralysis of the facial nerves due to increased intracranial pressure. AMS may progress over hours or days to death. Therefore, in ascents over 5,000 m, it is very important that all clinical and exploratory data be properly assessed by trained staff to avoid disastrous outcomes.

Respiratory distress, nausea, vomiting and headache are common indicators of altitude sickness between 2,500 and 5,000 m, though rarely seen in climbers who die at more than 8,000 m. This is because those people who have these early symptoms usually return earlier<sup>15-19</sup>.

In this study we evaluated a wide range of clinical parameters, visual charts, LA and AMS scores that have not ever been measured all together at these Andean altitudes. All the climbers were living below 1,200 meters above sea level except for subject N°5, and he was the only one who managed to reach the summit at 6,730 m. The maximum altitude reached by the rest of the team was 6,400 m. In the present study, measurements made at 5,000 m proved to be predictors of clinical outcome at greater altitudes. Lake Louis AMS score proved to be easy to measure (it does

not include the clinical measures of BP, HR, RR or SaO<sub>2</sub>) and a score ≥ 4 (moderate AMS) showed high sensitivity and specificity for climber status and predicted poor outcome in ascents to higher altitudes (NPV 100%).

A more complex assessment of AMS and its progression was performed evaluating the delta of SaO<sub>2</sub> and HR with the 6-minute walk test at 5,000 m. This test to measure the decrease of SaO<sub>2</sub> was conducted by Decker in 1989 with 13 patients with chronic lung disease<sup>13</sup>. We tailored the test for use with our volunteers, adding HR, in order to detect incipient subclinical pulmonary edema. Climbers with an SaO<sub>2</sub> delta ≥ 10% showed a 15% increased risk of developing moderate AMS, suggesting that this parameter measured at 5,000 m may be useful to alert climbers of poor clinical evolution at greater altitudes (NPV 100%), although our sample size was insufficient to show statistically significant differences.

However, the same cannot be said about measurements performed at 3,500 m, which yielded no significant data regarding resting values of SaO<sub>2</sub>, HR, RR, the 6-minute walk test or the values of LA, as described in previous studies<sup>9</sup>.

It is important to plan properly the risk of AMS in an ascent to over 5,000 m, since at this point AMS appears in 30-68% of athletes<sup>20</sup>. A descriptive study of 14,138 climbers ascending Mount Everest between 1921 and 2006 reported a mor-

**Table 4.** Results of measurements at different altitudes (3,500, 5,000, 6,000 and 6,400 meters) of the 4 climbers who reached 6,400 meters

Subject N°	HR (lpm)				RR (rpm)				SaO <sub>2</sub> (%)				Lake Louise score				Pelli Robson score			
	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400	3500/5000/6000/6400		
1	78	86	110	115	14	23	30	32	93	84	78	65	1	2	4	5	8	8	8	8
3	86	85	104	115	17	26	31	34	97	84	76	67	1	2	4	4	8	7	8	7
4	84	85	115	120	18	27	32	34	93	84	64	63	0	3	4	5	8	8	8	8
5	60	76	91	100	15	25	25	30	94	82	68	65	0	0	4	1	7	8	8	8
Mean	77	83	105	112.5	18.5	19.3	29.5	32.5	92.5	83.5	71.5	65	0.5	1.75	4	3.75	7.75	7.75	8	7.75

HR: heart rate, SaO<sub>2</sub>: arterial oxygen saturation.

tality rate of 1.3% (192), of whom 58.8% died from injuries associated with cognitive disorder and/or ataxia and 8.8% severe AMS<sup>21</sup>.

Based on previous studies, in climbs over 2,500 m it is recommended that non-acclimatized people should ascend no more than 400 m at a time<sup>20</sup>. It is worth repeating that, in our study population, subject N°5 was a climber who lived at 3,500 m. He was one of the leaders of the expedition, much like a Sherpa guide in the Himalayas. His Lake Louis score was always between 0 and 1, even at the maximum height, and delta HR and SaO<sub>2</sub> were 4 and 6 respectively. This reinforces the importance of slowly acclimatizing the body before attempting to reach higher camps and the summit; the greater the acclimatizing time, the better the performance.

Altitude-induced cerebral edema is known to generate papilledema and in asymptomatic climbers, retinal microbleeds are formed at great heights<sup>22</sup>. In this study we assessed possible retinal disorder by means of a Pelli Robson contrast sensitivity chart used by ophthalmologists to detect emerging retinal disease. We found no differences between climbers who reached 6,400 m and those who did not. We believe this was because none of the volunteers had symptoms of cerebral edema or retinal disorders. We should have performed ophthalmic fundus assessment to certify normal Pelli Robson results.

In our study at 5,000 m, an LA  $\geq$  2 mmol/l had a sensitivity and specificity of 100% and 83.3%, respectively, to predict poor outcome at higher altitudes. The odds ratio of having an LA value  $\geq$  2 mmol/l and suffering AMS was 1.23, i.e., climbers with an LA  $\geq$  2 at 5,000 m had a 23% higher risk of moderate AMS. This result differs from those obtained by Michael Grocott on Everest, where the subjects studied had no significant hyperlactatemia (mean LA 2.2 mmol)<sup>23</sup>. One explanation could be that on Everest there is a long process of acclimatization of 45-60 days, much longer than normal for the mountains of the Andes. A second explanation is that at higher altitudes (over 7,000 m) anaerobic metabolism does not contribute substantially to energy production. At such altitudes, the anaerobic glycolytic pathway is limited due to the decrease of the enzyme lactate dehydrogenase (LDH) in the muscle and this means that the two end products of glycolysis, pyruvic acid and hydrogen, do not complete the reaction and are converted by this enzyme into LA<sup>24</sup>.

We conclude that base-camp measurements of clinical and laboratory parameters in elite climbers may allow predicting poor outcome at greater heights. At 5,000 m, LA  $\geq$  2 mmol/l and Lake Louis score  $\geq$  4 significantly predicted poor outcome at greater heights. In contrast, baseline or resting HR, RR and SaO<sub>2</sub> values showed no predictive value in this study regarding poor outcome or moderate-severe AMS above 5,000 m.

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## Mal agudo de montaña: parámetros predictivos de un mejor rendimiento en montañismo de altura

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**Objetivo:** En el volcán Llullaillaco (6.739 m) situado en Argentina, se determinó la asociación entre los niveles de ácido láctico (AL), presión arterial (PA), frecuencia cardiaca (FC), frecuencia respiratoria (FR) y saturación O<sub>2</sub> periférica (SaO<sub>2</sub>) con el rendimiento físico y el desarrollo de mal agudo de montaña (MAM).

**Métodos:** A 8 escaladores se les midió el AL en sangre y los parámetros clínicos durante su ascenso al volcán. Se completó con datos de oximetría, agudeza visual (Pelli Robson), puntuación clínica de MAM de Lake Louise y test de caminata a los 6 minutos.

**Resultados:** A los 5.000 m un AL  $\geq 2$  mmol/l predijo mala evolución del estado clínico a mayores alturas, progresión a MAM y empeoramiento del rendimiento deportivo (valor predictivo positivo 66,7%, valor predictivo negativo 100%, exactitud 87,5%). Un cambio en la FC  $\geq 20$  lpm y en la SaO<sub>2</sub>  $\geq 10\%$  en el test de caminatas a 5.000 m de altura tenían una sensibilidad y especificidad del 100% para evidenciar mala evolución en campamentos de altura. Así mismo se comprobó que una puntuación de Lake Louise  $\geq 4$  a los 5.000 m determinaba una mala evolución en alturas mayores por acentuar el MAM.

**Conclusión:** La medición de parámetros clínicos y de laboratorio en los escaladores en campamentos bases podría predecir la evolución a mayores alturas. A los 5.000 m un AL  $\geq 2$  mmol/l, un cambio en SaO<sub>2</sub>  $> 10\%$  en el test de caminata y una puntuación de MAM de Lake Louise  $\geq 4$  predicen de forma significativa una mala evolución clínica. [Emergencias 2011;23:276-282]

**Palabras clave:** Parámetros predictivos. Parámetros clínicos. Mal agudo de montaña.