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Monitoring of Expiratory Flow Rates and Lung Volumes during a High Altitude Expedition

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Abstract
Background: Data on lung volumes and changes in flow-volume spirometry at high altitude are few and do not provide comprehensive assessment of the occurring changes. This study characterizes alterations of the forced expiratory flow-volume curve (FEFV-curve) and lung volumes at increasing altitude.
Methods: FEFV-curve and lung volumes at increasing altitude were characterized by daily assessment of peak expiratory flow (PEF), forced expiratory volume in one second (FEV1), forced vital capacity (FVC) and maximal expiratory flow rates (MEF 25, 50, 75) at 25%, 50% and 75% of the FEFV-curve with a portable spirometer (turbinometric method) three times a day during an expedition to Mustagh Ata (7545m) in 15 healthy mountaineers.
Results: With increasing altitude FVC and FEV1 were reduced by up to 25% (74.8% / 74.6% of baseline) and MEF25 was reduced to 81.5% of baseline values. PEF initially increased up to 4451m and returned to baseline values above 5000m. After descent below 2000m, all values normalized within one day. There were weak negative correlations between AMSS and FEV1, FVC and PEF ($r = -0.23$, $p<0.001$).
Conclusions: We found increasing pulmonary restriction at high altitude without a marked reduction of PEF. Assessment of the FEFV-curve at high altitudes with a portable spirometer is a practical method reflecting the true field situation and may provide clinically relevant information (impending pulmonary edema).

Key words: vital capacity, forced expiratory volume, portable spirometer, high altitude

Introduction

Measurements of respiratory parameters and lung volumes at high altitude were carried out as far back as the beginning of last century. At that time the main interest was to gain knowledge about lung physiology rather than the prospect to reach even higher altitudes. Mosso in 1897 was the first to document measurements of lung volumes at an altitude above 4000 m (Mosso 1898). The prevailing misconcept at this period was that breathlessness at high altitude resulted from suppressed respiration. It was not until much later that it was recognized that the ventilatory effort was indeed increased at high altitude. Several attempts to characterize ventilatory changes occurring at high altitude have been published. The highest altitude at which measurements of lung volumes were recorded was the peak of Mt. Everest where Pizzo measured maximal voluntary ventilation and respiratory rate (Ward et al. 1995). However, data on lung volumes and changes in flow-volume spirometry at high altitude are conflicting and do not provide comprehensive assessment of the occurring changes (Basu et al. 1996, Cruz 1973, Ge et al. 1997, Pollard et al. 1996, Rupwate et al. 1990, Sellland et al. 1993, Welsh et al. 1993, Wolf et al. 1997).

There are a small number of field studies and one of their major draw-backs is that recordings were performed largely inside protected mountain shelters or huts. A report by the British 40th Anniversary Everest Expedition using a Vitalograph device (pneumotachographic method) during their ascent to Mount Everest, was not believed to have produced reliable measurements (Peacock and Jones 1997). With portable spirometers (turbinometric method) which have been developed recently, repeated measurements for the frequent assessment of changes in an authentic field situation are possible. Until now, no data are available about the time course of FEFV-curve parameters in a true field situation. It is highly probable that there is an alteration of these parameters due to exercise, the acclimatization process or intercurrent subclinical bronchopulmonary infections. Possible restrictive changes might be related to clinically relevant high altitude pulmonary edema (Cremona et al. 2002).

It was the aim of this study to characterize alterations of the forced expiratory flow-volume curve (FEFV-curve) at increasing altitude by daily assessment during an expedition to the Mustagh Ata mountain and to identify a possible correlation between altitude, high altitude-related illnesses and FEFV-curve.

Material and Methods

Fifteen mountaineers - 13 males and 2 females - agreed to participate voluntarily in the research program during an expedition organized by the Summit Club (Munich, Germany) to the Mustagh Ata mountain in Western China (7545m, Kuen Lun); informed consent was obtained from each subject.

Spirometry was performed with a portable spirometer (AsthmaMonitor AM1, Viasys Healthcare, Hoech-

berg, Germany) to measure peak flow (PEF), FEV1, FVC and maximal expiratory flow rates at 25%, 50% and 75% of the expiratory flow-volume curve (MEF25, MEF50, MEF75). The variation between repeated measurements under standardized conditions is less than 5% (precision) and meets the minimal recommendations for monitoring spirometric devices of the American Thoracic Society (1995). The measurements taken by the AsthmaMonitor are based on a turbinometric method which is not influenced by reduced air density (Pedersen et al. 1994).

To confirm the accuracy of our devices, we verified 5 of the AsthmaMonitors in a low-pressure chamber simulating altitudes up to 7000m at room temperature and after cooling the AM1 to a temperature of –10 to -12 °C. With each AsthmaMonitor a FVC - maneuver was performed using a standard 1000 ml calibration syringe according to ATS-criteria (1995). We found no significant differences in FVC measurements at different temperatures (mean FVC 1.02 l ± 0.06 SD, coefficient of variation 0.05 for warm AM1; mean FVC 1.01 l ± 0.08 SD, coefficient of variation 0.07 for cold AM1). The results of the measurement at different altitudes are given in Table 1.

Table 1. Validation of turbine flow meter (AsthmaMonitor, AM1) in a low pressure chamber at different altitudes.
Data are presented as mean (SD, standard deviation). Measurements of cold (- 10 °C) AM1 or AM1 at room temperature were not significantly different, so both measurements were pooled.

Altitude (m)	n	FVC (l, mean)	SD	Coefficient of variation
510	5	1.05	0.050	0.05
2000	5	1.05	0.014	0.03
3500	4	1.01	0.022	0.02
4500	5	1.05	0.031	0.03
5000	5	0.96	0.046	0.05
6000	5	0.98	0.020	0.02
6500	5	1.00	0.021	0.02
7000	5	1.03	0.020	0.02

The spirometer allows the permanent storing of at least 400 measurements. Every participant used his own AsthmaMonitor during the whole expedition. Three times a day each member of the group was asked to perform at least two consecutive measurements and the monitoring device (AsthmaMonitor) stored the best of these measurements according to ATS criteria (1995). Date and time of the recording permitted the correlation with the specific altitude at that time. The accuracy of the FVC maneuver was assured by regular instruction of the participants.

The measurements of the FEFV-curve were carried out in the morning, at lunch time and in the evening outside the tents in a standing position after a minimum of fifteen minutes rest, i.e. not immediately after physical exertion.

In addition, peripheral oxygen saturation and pulse rate (Oxycount, Weinmann, Hamburg, Germany), endtidal pCO_2 and respiratory rate (Capnocount, Weinmann, Hamburg, Germany) were recorded at base camp and whenever possible at the camps at high altitude.

The acute mountain sickness score (AMSS) was determined daily in the morning: headache, dizziness, appetite, nausea, fatigue were scored by each participant on a 0 – 3 scale, with 0 for no symptoms and 1, 2, 3 for mild, moderate and severe symptoms (Lake Louise Consensus (Roach et al. 1993)). A score of 3 or more was taken as AMS. None of subjects used acetazolamide or steroids during the expedition.

Baseline measurements were carried out at 508m above sea level (Islamabad, Pakistan). A few days after arrival in Western China, base camp was reached at 4451m on Mustagh Ata. Within the next 19 days camps were erected at high altitude (camp 1 at 5450m, camp 2 at 6200m, camp 3 at 6900m) and two attempts were made to reach the summit. The return route led via Kaschgar (1200m, final recordings one day after leaving the basecamp) and via Taschkurgan (3160m) back to Islamabad.

Measurements were done at different altitudes: baseline measurements in Islamabad (508 m), further measurements at base camp (4451 m) and in camp 1 to 3, but not on the summit. Additional measurements were carried out at 2235m, 2665m, 3160m, 3600m and 4000m.

Statistical Analysis

For analysis the collected data were transferred in a database and a correlation with the altitude at each time was established. To minimize the influence of missing recordings at higher altitudes, data were normalized to baseline measurements (= 100% in Islamabad). Data are then given as deviation in percent of baseline measurements. As not every participant of the expedition remained for the same number of days at each altitude (according to the different ascent profiles), the number of measurements per individual per altitude level changed.

Statistical analysis was performed using the SPSS software (version 11.0). The Spearman correlation coefficient was calculated to assess the partial correlation of the measured variables. ANOVA with least significance difference (LSD) post-hoc testing (which is not influenced by missing values) was performed to assess the influence of altitude and acclimatization (number of days at a particular altitude for each subject) on the parameters of the FEFV-curve. Data of camp 3 (6900m) were not included in these analyses due to the low number of participants taking measurements at this altitude (n = 5). Linear multivariate and logistic regression (AMSS cut-off value = 3) was used to assess a possible influence of FEFV-curve, oxygen saturation, altitude and acclimatization on AMSS. Significance was assumed with $P < 0.05$.

Results

All subjects (mean age 32 years, range 25 – 54) were healthy, well trained mountaineers with an average body mass index below 22. All apart from one had

been previously exposed to altitudes above 5000m, but none had experienced severe symptoms of acute mountain sickness (AMS), high altitude pulmonary edema (HAPE) or high altitude cerebral edema (HACE).

None of the participants had pre-existing cardiopulmonary disease, allergies or chronic disabling disease of any kind and therefore nobody had to take chronic medication. One female member of the group was a mild smoker (less than 5 cigarettes per day).

One of the subjects used nifedipine due to impending high altitude pulmonary edema on the second day after arrival at basecamp and was forced to descend to Kashgar for two days. The results of this subject were not included in the analysis of FEFV-curves on day 2 at basecamp. Some subjects used temporarily ibuprofen 400 mg/day to relieve symptoms due to acute mountain sickness. Acetazolamide or sleepings pills were not used.

Spirometry

Of 791 measurements performed, 15 values were not interpretable due to artefacts (measuring only one or two values of the FEFV-curve). A mean of 51 measurements per participant (range 19-93) was reached within an average of 20 days.

Mean results of baseline measurements (and percent predicted) were as follows: FVC 4.21 l (108 %), FEV1 3.51 l (101%), MEF75 7.25 l/s (102 %), MEF25 1.92 l/s (98%), PEF 8.8 l/s (104 %).

The altitude dependent course of FEV1, FVC, PEF and MEF75 and MEF25 are given in Table 2 (each value is shown as percent deviation from baseline). FEV1 and FVC showed a uniform pattern in relation to altitude. The values taken decreased continuously in parallel to the increase of altitude. Values at altitudes below basecamp are not included in the table as they provide no further information for these parameters. The lowest values were reached at camp 2 and 3 where values decreased to 74% of the baseline values. The recordings from camp 3 (difference in altitude 700 m) were excluded, because only nine measurements from 5 participants were existing from camp 3 (mean FVC 64%, FEV1 69%). Both, reductions in FVC and FEV1, coincided, therefore the Tiffeneau Index (FEV1 in relation to FVC) did not correlate to the changes in altitude ($r = -0.0072$, $p = 0.845$).

The PEF value followed a different course. With increasing altitude it reached a maximum up to 109% of the baseline value at an altitude between 3000 - 4000m and above 4000m slowly went back to near baseline levels (at 2235m 112.8 ± 27.4, at 2665 113.6 ± 30.2, at 3160m 113.9 ± 33.8, at 3600m 113.1 ± 35.7, at 4000m 120.5 ± 31.3; all data mean ± standard deviation).

A similar course was noted for the MEF75 values (at 2235m 108.3 ± 10.3, at 2665 106.7 ± 14.7, at 3160m 109.6 ± 17.7, at 3600m 103.4 ± 21.6, at 4000m 113.7 ± 13.8; all data mean ± standard deviation).

The reduction of the more peripheral flow rates is reflected by MEF50 and MEF25, which followed a

Table 2. Results of lung function measurements at basecamp (4451m), camp 1 (5450m), camp 2 (6200m) and after return to Kashgar (1200m).

Altitude m	day at altitude	n	FVC (% from baseline)	FEV1 (% from baseline)	PEF (% from baseline)	MEF75 (% from baseline)	MEF25 (% from baseline)
4451	1	15	86.4 (11.8)	83.0 (18.1)	110.6 (30.9)	106.2 (21.2)	81.7 (30.8)
4451	2	14	84.0 (18.5)	81.1 (21.8)	100.7 (40.8)	98.6 (22.3)	77.6 (31.7)
4451	18	15	89.6 (20.8)	86.9 (21.9)	113.8 (35.9)	104.0 (18.2)	88.5 (25.9)
4451	**day 1 to 18**	**184**	**86.5 (18.6) #**	**83.3 (19.2) #**	**106.6 (37.0)**	**101.1 (21.0)**	**87.7 (27.1)**
5450	1	12	79.1 (5.6)	75.3 (21.4)	103.3 (23.9)	102.8 (14.1)	76.3 (38.3)
5450	2	12	86.4 (12.6)	86.0 (15.2)	114.0 (27.3)	99.4 (17.8)	85.5 (35.7)
5450	8	10	67.9 (17.8)	69.5 (21.9)	112.4 (40.7)	92.9 (21.3)	67.6. (30.6)
5450	**day 1 to 8**	**68**	**81.0 (23.4)***	**78.0 (22.9)***	**106.8 (31.6)**	**94.8 (22.19**	**76.0 (35.1)**
6200	1	10	69.0 (14.6)	61.2 (16.3)	94.1 (30.9)	88.8 (10.5)	74.8 (34.4)
6200	2	8	71.6 (15.9)	76.4 (22.3)	117.1 (26.4)	113.1 (14.5)	84.6 (49.7)
6200	6	7	78.3 (17.5)	75.4 (22.4)	105.4 (29.5)	97.2 (25.4)	90.5 (44.5)
6200	**day 1 to 6**	**32**	**74.8 (18.6) §**	**74.6 (21.0)§**	**105.5 (28.9)**	**92.4 (26.0)**	**81.5 (36.2)**
1200	1 †	15	101.4 (13.3)	102.1 (11.7)	110.1 (29.9)	103.4 (8.5)	101.2 (29.7)

Data are presented as mean (standard deviation) and as percent deviation of baseline measurements at 508m. There was no significant difference between the first, the second and the last day at a given altitude (n = number of subjects; ANOVA, LSD post-hoc testing). Data of all measurements at a given altitude were pooled (n in bold letters, including the days not shown in the table) to compare baseline and altitude values of lung function parameters (ANOVA, LSD post-hoc testing): # denotes $p < 0.01$ for comparison of baseline to basecamp values; * denotes $p < 0.05$ for comparison of basecamp to camp 1 (5450m) or camp 2 (6200m); § denotes $p < 0.01$ for comparison of basecamp to camp 1 (5450m) or camp 2 (6200m).
† denotes measurements were done after return to Kashgar at 1200m.

similar course to FEV1. At an altitude above 4000m a gradual reduction of these parameters was found and at an altitude of 6200 m values decreased to 81% (MEF25) of the baseline.

For all FEFV-curve values we found no significant effect of the acclimatization process. However, values from day 1 or day 2 at a given altitude are generally lower than those of the last day at this altitude.

All respiratory parameter returned to baseline within one day after the descent from base camp to Kaschgar (1200m).

Clinical Symptoms and AMSS

The Acute Mountain Sickness Score was highest on arrival at base camp (median AMSS 1.5, range 0 - 10; five subjects showed scores of 3 or more) and clinical symptoms ameliorated during the process of acclimatization. Due to deteriorating weather conditions only 8 members of the expedition reached the summit. Apart from one participant who had to be treated for impending high altitude lung edema on the second day after arrival at base camp (lung function results have been excluded from analysis of day 2 at basecamp), and had to descend to lower altitude for two days, all other members with mild to moderate acute mountain sickness could be treated symptomatically (with ibuprofen 400 mg daily).

There were weak, although significant correlations between AMSS and FEV1 ($r = -0.23$, $p = 0.008$), AMSS and FVC ($r = -0.25$, $p = 0.003$) and AMSS and PEF ($r = -0.21$, $p = 0.01$). Nevertheless, neither multivariate nor logistic regression analysis revealed a significant influence of lung function parameters on AMSS. We found no correlation between AMSS and gas exchange parameters. As expected, only altitude was a significant factor for the prediction of AMS score (regression equation: AMSS = 0.21 + 0.32 * (altitude / 1000); $p = 0.031$, 95% CI for coefficient B = 0.03 – 0.60).

Oxygen Saturation, Pulse Rate and Endtidal CO_2

With increasing altitude SaO_2 and $etCO_2$ declined (each $p < 0.01$) and PR and respiratory rate (RR) were raised (each $p < 0.01$, Table 3). There was a significant positive correlation between SaO_2 and FVC ($r = 0.31$, $p<0.01$), low FVC-values being correlated with low SaO_2-values.

Discussion

Our data show that with increasing altitude maximum forced expiratory volume was reduced by up to 25%. This can be interpreted as increasing restrictive changes during ascent to an altitude of 6200 meters.

In our population, we were able to demonstrate that the decline in FEV1 and FVC returned rapidly (within one day) to normal baseline values, when the subjects descended to low altitude. Increasing ventilatory restriction has been observed also by others. However, their series included a substantially lower number of measurements, were taken in protected shelters and did not include mountaineers with daily exercise (Welsh et al. 1993, Pollard et al. 1996, Jaeger et al. 1979, Rupwate et al. 1990, Dramise et al. 1976, Mansell et al. 1980).

Mansell et al. observed an increase of PEF (20 %) at 5300 m in seven subjects (Mansell et al. 1980)[18]. FVC decreased in some subjects by more than one liter, although the mean decrease was not significant. They plotted a typical FEFV-curve with patterns similar to our observations. They found higher flow rates during early expiration and lower flow rates during late expiration. The decrease in vital capacity was judged to be due to an increase in TLC and RV with formation of interstitial lung edema and increase in closing capacity.

Jaeger et al. showed a decrease in FVC of 6.7% in 25 subjects after a 72 h stay at an altitude between 3000 and 4300 m (Jaeger et al. 1979)[16]. They found no radiographic signs of interstitial pulmonary edema, although the values for thransthoracic impedance, vital capacity, closing capacity and the transpulmonary pressure-volume relationship were consistent with an increase in lung fluid volume.

Dramise et al. found a decline of FVC of 6.7 %, together with an increase of TLC and RV (Dramise et al. 1976). The return to baseline values took approximately three days, this is somewhat longer than in our study.

Rupwate et al. reported an initial increase of FEV1 at altitudes between sea level and 3500 m followed by a slow decrease up to one liter at an altitude of 6799 m (Rupwate et al. 1990). As a Wright's mini-peak flow meter was used for measurements of the PEF-values, an underestimation of the values is possible (Pedersen et al. 1994).

The observed fall in FVC (and its rapid resolution with better oxygenation at low altitude) might be due

Table 3. Results of respiratory measurements at increasing altitude.

Altitude (m)	Oxygen Saturation SaO_2 (%)	Heart rate (x/min)	endtidal CO_2 (mmHg)	Breathing frequency (x/min)
508 (Islamabad)	96.7 (2.1)	63.1 (8.7)	n.a.	n.a.
4451 (basecamp)	84.9* (6.2)	89.5* (14.8)	29.0 (4.0)	13.7 (3.4)
5450 (camp 1)	75.3* (8.0)	95.8* (13.7)	27.5 (3.9)	14.3 (2.7)
6200 (camp 2)	64.0* (4.0)	99.6* (16.9)	21.2* (4.5)	15.7* (4.6)
6900 (camp 3)	62.6* (4.0)	108.3* (7.6)	15.5* (2.1)	22.0* (1.2)

Data are presented as mean (standard deviation). * $P < 0.01$ for the comparison <2000m versus >4000m (LSD post-hoc test). N.a. denotes measurements not available at this altitude.

to increased intrathoracic fluid volume, probably caused by sub-clinical interstitial lung edema of the whole group (Jaeger et al. 1979)[16] (Cremona et al. 2002)[12]. This is strengthened by the fact that the ascent profile of the expedition was very fast (up to 6900 meters in less than two weeks); therefore the time for acclimatization was quite short. Even though the time dependent change of lung function parameters at a given altitude (i. e. basecamp) was not significant, there was an obvious trend toward higher values to the end of the stay at basecamp or high camps suggesting removal of interstitial or alveolar lung water due to the acclimatization process.

Corroborating the hypothesis of increased intrathoracic fluid volume, the mean oxygen saturation at camp 2 (6200 meters) was very low (63.9%) and the mean heart rate raised to 100 beats per minute. Furthermore, one subject showed severe high-altitude lung edema on the second day in basecamp and the fall in FVC in this subject was even more pronounced (up to 51% of baseline). After staying in Kashgar (1200m) for two days, this subject recovered completely with normalization of his FVC (increase to 95% of baseline after one day below 2000 m). Therefore, a pronounced reduction of FVC or FEV1 could be correlated to impending high altitude pulmonary edema (HAPE). This phenomenon should be assessed in a higher number of subjects prone to develop HAPE, as simple spirometry could be used as a predictor of imminent lung edema.

A note of caution should be applied when interpreting our spirometric data at high altitude as factors such as reduced muscle strength and reduced voluntary effort after strenuous exercise at high altitude or heavy winds may have affected single measurements.

Another reason for increased intrathoracic fluid volume is increased intrathoracic blood volume caused by pulmonary venoconstriction. We have no measurements to confirm or reject this possibility. Welsh et al. (Welsh et al. 1993)[9] demonstrated a partial recovery of the FVC in the first thirty minutes after decompression of the hypobaric chamber which they contributed mainly to the decrease in pulmonary venous blood volume.

The increase of PEF at high altitude is known to be due to the lower gas density (Kryger et al. 1978)[19]. However, and in contrast to others, we found a reduction of PEF and MEF75 back to baseline values at altitudes above 5000m. The differences between our results at altitudes higher than 5000 meters and those of other observers could be explained by increasing bronchial obstruction due to the prolonged exposure to cold and exercise in our subjects, reflecting the true field situation during an expedition (Giesbrecht 1995)[20]. The decrease in PEF-values could reflect the opposing forces of higher flow rates due to reduced gas densitiy and increase of airway obstruction due to the cold environment. Measurements in heated tents or warm pressure chambers are therefore likely to miss the effect of the mountain situation and are difficult to compare.

We found only weak correlations between PEF, FEV1, FVC and AMSS, with low values for parameters of FEFV-curve being associated with higher values of AMSS. When adjusted for altitude effects, the strength of the correlation did not improve. In addition, oxygen saturation was not correlated with AMSS when adjusted for altitude effects. This might be due to the overall low score of total AMSS during this expedition. This is also reflected by the relatively low significance of altitude effects in the multivariate and logistic regression model.

Conclusions

Our data show that the assessment of the flow-volume-curve at high altitudes with a hand-held portable spirometer is a valuable and practical method reflecting the true field situation more accurately. The type of spirometry used provided additional information (sub-clinical lung edema, peripheral obstruction) which may be clinically relevant. Whether spirometry can be used as a predictor of impending HAPE is not clear, further studies in larger cohorts are needed.

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Authors' contributions: RF carried out the field measurements during the expedition and drafted the manuscript. SML participated in the design of the study and helped to draft the manuscript. AB participated in the design of the study and helped to draft the manuscript. RMH conceived of the study, and participated in its design and coordination. All authors read and approved the final manuscript.

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