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Effects of a fast cable car ascent to an altitude of 2700 meters on EEG and ECG

Christoph Guger[a,b,*], Wolfgang Domej[a,c], Gerhard Lindner[a], K. Pfurtscheller[a], Gert Pfurtscheller[d], Günter Edlinger[b]

[a] *ARGE-Alpinmedizin, Austria*
[b] *g.tec — Guger Technologies OEG, Herbersteinstr. 60, A-8020 Graz, Austria*
[c] *Department of Internal Medicine, University Medical Center, Graz, Austria*
[d] *Institute of Human-Computer Interfaces, University of Technology Graz, Austria*

Abstract

In the Eastern Alps, the Dachstein massif with a height of almost 3000 m is an ideal location for investigating the effects of changes in altitude on the human body. Within a few minutes, a cable car facilitates an ascent from 1702 to 2700 m above sea level, where the partial pressure of oxygen is about 550 mmHg (as compared to 760 mmHg at sea level). In this study, 10 healthy subjects performed a reaction time task at 990 m and 2700 m in altitude. The subjects were instructed to perform a right hand index finger movement as fast as possible after a green light flashed (repeated 50 times). The corresponding electrocardiogram (ECG) and the electroencephalogram (EEG) were recorded. From the ECG heart rate and heart rate variability measures in the time and frequency domain were calculated. An event-related desynchronization/synchronization (ERD/ERS) analysis was performed with the EEG data. Finally, the EEG activity and the ECG parameters were correlated.

The study showed that with the fast ascent to 2700 m the heart rate increased and the heart rate variability measures decreased. The correlation analysis indicated a close relationship between the EEG activity and the heart rate and heart rate variability. Furthermore it was shown for the first time that the beta ERS in the 14–18 Hz frequency range (post-movement beta ERS) was significantly reduced at high altitude. Very interesting also is the loss of correlation between EEG activity and cardiovascular measures during finger movement at high altitude. The suppressed post-movement beta ERS at the altitude of 2700 m may be interpreted as results of an increased cortical excitability level when compared with the reference altitude at 990 m above sea level.

Keywords: High altitude; Cortical excitability; Event-related desynchronization (ERD); Heart rate variability; Beta oscillations

With increasing altitude, the concentration of oxygen in the air remains constant but its partial pressure drops. This means that the number of oxygen molecules in the blood and tissues of the body is also reduced. Normal air pressure is 760 mm of mercury at sea level with an oxygen concentration of 21%. At 3000 m, the pressure drops to about 550 mmHg, and at the summit of Mont Blanc (4800 m) the partial pressure of oxygen is about half of that at sea level. A common way of simulating high altitude is to use hypobaric chambers that change the oxygen concentration of the air, e.g. between 9.5 and 15% to simulate an altitude of 2700–6500 m. But in reality the concentration of oxygen remains constant and therefore in the Eastern Alps, the Dachstein massif with a height of almost 3000 m is an ideal location for investigating the effects of changes in altitude on the human body. The BASE station of the cable car is at 1702 m and the TOP station at 2700 m. The ascent to the TOP station lasts about 7 min. Hence, changes in EEG (electroencephalogram) and ECG (electrocardiogram) activity caused by the rapid ascent and drop of oxygen partial pressure can be investigated without the strenuous exercise of an ascent by foot.

Up to now several studies have been conducted to investigate the autonomic regulation of heart rate (HR) during gradual high altitude acclimatization [1,6,9,11,12]. Others used hypobaric chambers to describe the effect of acute exposure to simulated altitude on heart rate variability (HRV) [2,23]. During exposure to acute hypoxia, increased sympathetic activity results in an elevated cardiac output to compensate for

* Corresponding author. Tel.: +43 316 675106 12; fax: +43 316 675106 39.
E-mail address: guger@gtec.at (C. Guger).

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the reduced arterial oxygen content. Because the HRV analysis of the ECG signal in the time and frequency domains can be used for non-invasive investigation of autonomic cardiovascular regulation and sympathovagal interaction [21], each QRS complex (depolarization of the heart) is detected in the ECG signal and the distance from one to the previous is calculated and termed RR interval. Then the power spectrum of this time series can be estimated to describe the parasympathetic and sympathetic system [1,2,6,11,12,23]. The power spectrum in the frequency range 0.04–0.15 Hz is normally referred as the low frequency (LF) component, and that in the range of 0.15–0.4 Hz as the high frequency (HF) component. The latter is mainly modulated by the parasympathetic system and the former by the parasympathetic and sympathetic system [12,21]. However, if the LF component is divided by the total power of the power spectrum then it can be seen to be mainly modulated by the sympathetic system [21]. The ratio LF/HF describes the balanced behavior of the sympathetic and parasympathetic systems.

Bernardi et al. [1] measured 10 healthy sea-level dwellers upon arrival at 4970 m and found an increased sympathetic activity at high altitude. This increased sympathetic activity resulted in a power increase in the low frequency component (slow oscillations) and an increase in the LF/HF ratio. Compared to sea level, acute high altitude exposure decreased the mean RR interval from 1002 ms (standard deviation: ±45) to 775 ± 57 ms. The LF power increased from 47 to 65 ms, and the HF power decreased. Another study showed an increase of the LF/HF ratio from 1.6 to 3.2, caused by a change in elevation from sea level to an altitude of 5000 m in a hypobaric chamber [2]. Kanai et al. [12] investigated the changes of HR and HRV at altitudes of 2700 m and 3700 m in tourists. The HR increased from 70 beats per minute (bpm) to 78 bpm and 89 bpm. The LF/HF ratio was not significantly different at 2700 m compared to that of sea level, but was significantly increased at 3700 m (after a 6 h ascent).

The EEG can also be used to investigate the effects of fast altitude changes on the central nervous system. In the first stage of hypobaric hypoxia (at 3000 m) the spontaneous alpha activity is decreased [15]. In a further stage starting at 5000 m, theta activity is enhanced in the anterior areas, and strong suppression of alpha in the posterior areas of the brain occurs [15].

Beside the investigation of the altitude dependent changes in spontaneous EEG, it is of interest to study event-related EEG changes which can be observed in motor, sensory and cognitive tasks. It is well known that the execution of movement is accompanied by a desynchronization of mu and central beta rhythms over the corresponding cortical representation areas [16]. The movement-related power decrease in a specific frequency band (event-related desynchronization, ERD) can be found in EEG traces measured over the sensorimotor areas during, e.g. finger movements. A right hand finger movement produces an ERD in the left hemisphere close to electrode position C3 of the international 10/20 electrode system. Similarly, a left hand movement results in an EEG desynchronization over the right hemisphere close to C4. The recovery phase from the movement starts with movement offset and typically lasts between 1 and 2 s. In this phase, the mu rhythm slowly returns to its resting state while bursts of short-lasting oscillations in the beta band occur after the movement offset. The peak of such a post-movement beta event-related synchronization (ERS) occurs at about 0.6–1 s after movement offset [17]. In contrast to an ERD, an ERS indicates a power increase in a specific frequency band. The post-movement beta ERS (beta rebound) occurs mainly on the contralateral side (with respect to the movement side) but also with a smaller amplitude on the ipsilateral side. It displays a strict somatotopic organization and coincides with a reduced corticospinal excitability [22]. An ERD in the alpha band is characteristic of perceptual, judgment and memory tasks. The power decrease is greater with increased task complexity or attention [3,13,20].

Townes et al. showed that sensory/motor functions are not inhibited at 2700 m, but the time required to learn a new task is increased [22]. In the literature no task related EEG investigations at high altitude can be found. Furthermore, evidence of quantification of the correlation between changes of EEG activity and ECG parameters is not available. Therefore, the current study has the following aims: (i) to investigate the effects of a fast cable car ascent (within a few minutes) on both the autonomic and the central nervous system and (ii) to show that changes in EEG and HRV measures occur also without the strenuous exercise of an ascent by foot. EEG and HRV changes in subjects during a well-defined workload shall be investigated at altitudes of 990 m and 2700 m.

In October 2003, six healthy subjects (three male, three female, 28–57 years) and in March 2004, four healthy subjects (two male, two female, 26–34 years), participated in the experiment (all right handed). All subjects were participating in the study for the first time. ECG and EEG were recorded while the subjects performed a psycho-physiological experiment (reaction time task). The measurements were performed (i) 30 min prior to the cable car ride at an altitude of 990 m (BASE) and (ii) 1 h after the cable car ride at 2700 m (TOP). Each measurement lasted about 6 min. The study was approved by the local human ethics committee and the subjects were informed of potential risks, whereafter written consent was obtained. All measurements were performed inside of buildings in an extra room (room temperature was 20 °C). After arrival at a new altitude, the subjects rested for 1 h prior to the experiment. The subjects sat in a chair in a pre-defined body position in front of the flashing light. They were instructed not to move during the experiment so as to assure high data quality.

A pocket PC-based EEG and ECG recording system g.MOBIlab (g.tec — medical engineering GmbH, Graz, Austria) was used for biosignal acquisition and to display the reaction time paradigm. One channel of ECG (Einthoven I) and two bipolar EEG channels were recorded. The EEG electrodes were mounted 2.5 cm posterior and 2.5 cm anterior to electrode positions C3 and C4 of the international

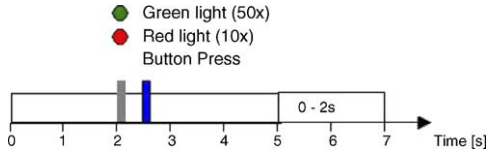

Fig. 1. Timing of the paradigm. At 2 s of the trial a green or a red light flashes. If the green light flashes the subject has to press the button as fast as possible. The subject is not allowed to press the button if the red light flashes.

10/20 system. The EEG ground electrode was mounted on the forehead. Signals were recorded at a sampling frequency of 256 Hz and with a resolution of 16 bits.

The protocol of the reaction time experiment is illustrated in Fig. 1. At second two a green or red light flashes for 200 ms. The subject's task is to press a button with the right index finger as fast as possible in response to a green flash but refrain in the case of a red flash. The duration of the trial is 5 s. To avoid adaptations of the subject, each trial is followed by a random interval of 0–2 s. The blinking sequence of the green light (50 times) and the red light (10 times) is randomly distributed. A complete experiment consists of 60 trials.

The EEG and ECG analysis was performed with the g.BSanalyze biosignal analysis software package (g.tec — medical engineering GmbH, Graz, Austria).

From the ECG recordings the QRS complexes were detected and the results were visually inspected to avoid incorrectly identified QRS complexes. Then the time interval from the R-peak of one QRS complex to that of the previous was calculated and termed the RR interval. Non-normal beats such as extra systoles are not considered.

The simplest time-domain variable calculated is the mean heart rate (MeanHR) in bpm. More complex measurements can be divided into two classes: (i) those derived from direct measurements of the heart rate: the SDNN index is the mean of the standard deviation of 30-s segments and describes the variability due to cycles longer than 30 s; (ii) those derived from the RR interval difference between consecutive intervals: the NN50 is the number of intervals which differ by more than 50 ms from the previous interval. pNN50 is the NN50 divided by the total number of NN intervals. RMSSD is calculated as the square root of the mean squared difference of successive RR intervals.

Power spectrum (PS) analysis provides the basic information on how power is distributed as a function of frequency. The tachogram of an RR series is non-uniformly sampled because of the different length of each RR interval. Therefore, in this study the tachogram was resampled at 2 Hz to obtain an equidistant sampled sequence. Thereafter the first 128 data points were detrended and a Hanning window was applied. From the resulting signal the power spectrum was calculated. Then the data window was shifted by 64 samples and the next PS was calculated in the same way. This was repeated after the end of the data set was reached where after the spectra of all segments were averaged. Three main spectral components were distinguished: (i) very low frequency (VLF): <0.04 Hz, (ii) low frequency (LF): 0.04–0.15 Hz and (iii) high frequency (HF): 0.15–0.4 Hz. The unit of these parameters is ms^2. For normalization the LF (Lfnorm) and HF (Hfnorm) components are divided by the total power minus the VLF component. This minimizes the effect of the total power on LF and HF.

The EEG data set was split into epochs of 6 s. Each epoch contained the data for 2 s prior to the finger movement and 4 s thereafter. The EEG data were visually controlled for artifacts. If the subject was not responding to the green light the trial was removed. If the subject responded to the red light, the trial was also removed. It was never necessary to remove more than two trials for these reasons. Trials containing eye, muscle or movement artifacts were excluded, resulting in about 35–45 trials for each subject. Each EEG channel was band-pass filtered into bins of 2 Hz bandwidth from 4 to 30 Hz and a step size of 1 Hz (i.e. the first bin was from 4 to 6 Hz, the next from 5 to 7 Hz, etc.) The band pass filtered signal was squared and averaged over all trials. Thereafter samples were averaged over time to smooth the data and reduce the variability. ERD and ERS values were defined as relative power decrease and increase with respect to a resting period measured in the pre-movement period from 0.5 to 1.5 s (reference period). For details on the quantification of ERD/ERS see [16]. A bootstrap algorithm was used to find only the significant changes ($p < 0.01$). These significant ERS and ERD values are shown in the time–frequency map (see Fig. 2).

The EEG band-power was calculated in the alpha (8–13 Hz) and beta (14–18 Hz) frequency ranges. It was averaged in the reference interval from 0.5 to 1.5 s, in the action interval for the alpha activity from 1.5 to 2.5 s, and in the action interval for the beta activity from 2.5 to 4 s. This was done for both electrodes in each trial before calculating the mean of all trials and taking the logarithm thereof. This yielded four parameters (reference-alpha, action-alpha, reference-beta, action-beta) at the BASE station and at the TOP station. The differences were then calculated between the TOP and BASE station. These band-power difference

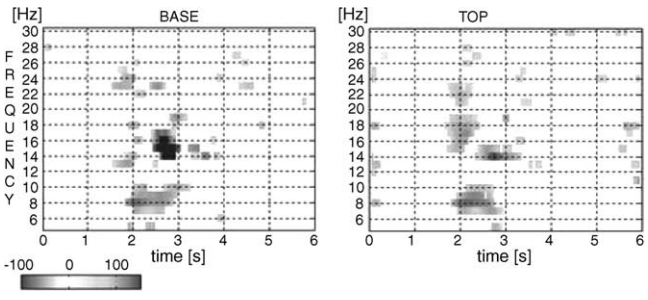

Fig. 2. Event-related desynchronization (ERD) and event-related synchronization (ERS) analysis of the EEG data of subject 1 (left side: BASE station, right side: TOP station). The subject performed the finger movement at 2 s. Relative power decrease (ERD) is color coded red and relative power increase is color coded blue. The ERD occurs in the alpha band (8–13 Hz) starting approximately at 1.5 s (0.5 s before the actual movement onset) and lasting until second 3 s. Post-movement beta ERS (14–18 Hz) starts at 2.5 s and lasts for about 1 s.

Table 1
Grand average (for all subjects) of HR and HRV parameters at the BASE and TOP stations

	BASE station	TOP station	*p*-value
Heart rate			
MeanHR (bpm)	69.1 ± 12.6	80.4 ± 15.4	<0.002
HRV time domain			
RMSSD (ms)	33.0 ± 28.5	14.8 ± 6.4	<0.002
pNN50 (%)	9.6 ± 22.94	0.9 ± 4.7	<0.002
SDNN index (ms)	58.1 ± 23.8	41.1 ± 12.2	<0.05
HRV frequency domain			
Lfnorm (n.u.)	51.1 ± 18.3	65.4 ± 19.3	<0.002
Hfnorm (n.u.)	35.1 ± 14.3	25.0 ± 14.1	<0.05
LF/HF [1]	2.1 ± 1.98	4.4 ± 4.1	<0.05

A paired sign test yielded significant differences for all parameters shown.

scores were created for each of the two electrodes C3 and C4. Similarly, difference scores of heart rate and heart rate variability were created by subtracting the parameters from the BASE station from the parameters from the TOP station. From these the correlation coefficient was calculated.

Grand averages of HR and HRV parameters for 10 subjects in the time and frequency domains are given in Table 1. With the ascent, the MeanHR increased by 11.3 bpm while the RMSSD, pNN50 and SDNN index decreased. The frequency domain parameters indicate that the LF component becomes more prominent with the ascent and that the HF component is reduced resulting in the LF/HF ratio increasing from 2.1 to 4.4.

ERD/ERS time courses in the alpha (8–13 Hz) and in the beta (14–18 Hz) frequency range were calculated for each subject and both EEG channels. Grand average ERD/ERS time courses for 10 subjects are given in Fig. 3 for channel C3.

To perform an analysis of significance, the power in the alpha band was averaged from 1.5 to 2.5 s, and in the beta band from 2.5 to 4 s. The same was performed for the reference interval (0.5–1.5 s). Then all trials were averaged and the ERD coefficient was calculated. This was done for each subject. Statistical analysis of all 10 subjects with a sign test for paired samples showed that there are no significant changes in the alpha band. However, the ERD changes in the beta band were significant ($p < 0.05$) for channel C3. No significant changes were found for channel C4.

The results from the correlation analysis indicate that changes in the alpha and beta range over C3 are positively correlated to the MeanHR (+0.79 and +0.89, $p < 0.01$) in the reference period. Additionally, the band-power in the alpha range of both electrodes calculated in the action period is correlated to the MeanHR. The band-power in the reference and action periods of the alpha range is found to have a negative correlation to all HRV time domain measures over C3 while in the beta range a correlation can only be found in the reference period. Lfnorm shows a high correlation over C3 and C4 in the alpha range while in the beta range only the band-power in the reference period is correlated. The negative correlation of Hfnorm in the reference and action periods

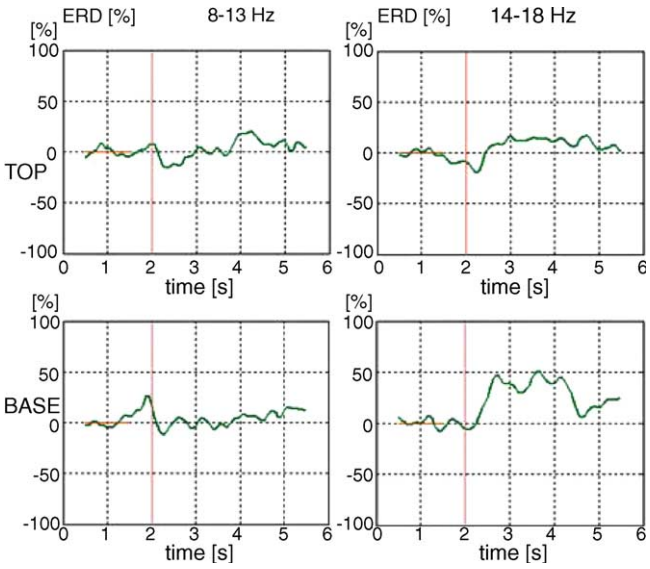

Fig. 3. Grand average (for all 10 subjects) ERD/ERS time courses for electrode C3. The top row represents the results at the TOP station, the lower row the results at the BASE station. At the TOP station the alpha ERD has a value of about −20% after 2.2 s while at the BASE station the alpha ERD has a value of approximately −15%. The beta ERS has a value in the region of +12% at the TOP station and about +42% at the BASE station.

is also interesting. LF/HF did not show a significant relation (Table 2).

The study showed that a passive ascent to an altitude of 2700 m affects both the central and autonomic nervous system. Changes were found in ERD/ERS, heart rate, heart rate variability measures and correlation between EEG and ECG measures.

It was shown for the first time that the post-movement beta ERS (beta rebound) is significantly attenuated at the high altitude compared to the low altitude measurement. Both self-paced finger movement and electrical median nerve stimulation are terminated by a beta rebound of similar magnitude and latency [16]. Corticospinal excitability was shown in the reaction time movement tasks to be significantly reduced in the first second after EMG offset [4]. These findings support the hypothesis that beta ERS could be related to an idling or deactivated state [17], or even active immobilization of the motor cortex [18]. Such a beta rebound originates mainly in the pre-central localized motor cortex and is attenuated or even suppressed during activation of the motor cortex [8,19,20]. The beta ERS can therefore be seen as an inverse marker for the excitability level of motor cortex neurons with an attenuation when the excitability level is increased. The suppressed post-movement beta ERS at the altitude of 2700 m may therefore be interpreted as a result of an increased cortical excitability level when compared with the reference altitude of 990 m. At an altitude of 2700 m essential sensory/motor functions are not inhibited, but the length to learn a new task is already increased [22].

The ERD indicates the activated cortical areas involved in the processing of cognitive information and production

Table 2
Correlation coefficients of band-power in the alpha (8–13 Hz)/beta (14–18 Hz) ranges for the reference and action intervals and heart rate and heart rate variability parameters

	Correlation coefficients							
	Alpha range (8–13 Hz)				Beta range (14–18 Hz)			
	Reference period (0.5–1.5 s)		Action period (1.5–2.5 s)		Reference period (0.5–1.5 s)		Action period (2.5–4 s)	
	C3	C4	C3	C4	C3	C4	C3	C4
Heart rate								
MeanHR	+0.79**	+0.63	+0.87**	+0.67*	+0.89**	+0.62	+0.42	+0.21
HRV time domain								
RMSSD (ms)	−0.85**	−0.46	−0.79**	−0.54	−0.69*	−0.71*	−0.41	−0.17
pNN50 (%)	−0.71*	−0.30	−0.68*	−0.39	−0.47	−0.66*	−0.29	−0.09
SDNN index (ms)	−0.67*	−0.29	−0.79**	−0.46	−0.65*	−0.56	−0.26	+0.03
HRV frequency domain								
Lfnorm (n.u.)	+0.73*	+0.74*	+0.44	+0.77**	+0.76*	+0.59	+0.34	+0.28
Hfnorm (n.u.)	−0.85**	−0.72*	−0.7*	−0.78**	−0.87**	−0.70*	−0.39	−0.30
LF/HF [1]	+0.20	+0.53	+0.01	+0.38	+0.47	+0.03	+0.15	−0.15

For the correlation analysis the difference between each parameter at the BASE and the TOP station was calculated.
* $p < 0.05$.
** $p < 0.01$.

of motor behavior. An increased ERD can be the result of a larger neural network involved in the task of information processing. Such an enhancement of ERD can be found during increased task complexity [20], in Parkinson patients [5] or in subjects with lower IQs [14]. ERD changes in the alpha band, however, were not significant when comparing the results from the TOP to the BASE station.

Hughson et al. and Farinelli et al. studied the effect of long-term exposure to an altitude above 4000 m [6,11]. Both found increased sympathetic and decreased parasympathetic nervous system activity in the early phase of the acclimatization. Yamamoto et al. reported the same effect caused by exercise in a hypobaric chamber which simulated an altitude of 3500 m [23]. The increased sympathetic activity under hypoxia was also found by measuring the muscle sympathetic nerve activity [9]. The present experiment showed that the mean heart rate increased and the heart-rate variability decreased with the increasing altitude. The HFnorm component was lower at the BASE station, indicating that the parasympathetic system, which is coupled to the HFnorm components, was less active. If the LF component is expressed in normalized units it basically represents the sympathetic system. This implies that the sympathetic system was more activated at the higher altitude [21]. These results are in accordance with earlier studies performed at a 5000 m height [1,2]. It is interesting to note that even an altitude of 2700 m causes the changes in HRV. Furthermore, the changes occurred without the strenuous exercise of an ascent by foot.

Foster and Harrison investigated the relation of cortical electrical activity and cardiovascular response during different emotional states of their subjects [7]. The results showed that changes in magnitude of alpha (8–13 Hz), low beta (13–21 Hz), and high beta (21–32 Hz) EEG activity at frontal and temporal sites are correlated with changes in cardiovascular measures (heart rate, diastolic and systolic blood pressure). In the present study it was also possible to show a relationship between EEG and ECG activity. It is interesting to note that only the heart rate and Lfnorm are positively correlated to the band-power changes in the alpha and beta range. Heart rate variability parameters in the time domain and Hfnorm always showed a negative correlation. It is also important to note the correlation in the reference period between band-power changes (alpha and beta) and cardiovascular changes. The band-power in the beta frequency range during the action period, however, was not correlated to the ECG parameters. This is exactly the same EEG frequency range where the suppressed post-movement ERS was found. Therefore, during the period where the cortex shows an increased excitability level there seems to be no correlation to the cardiovascular system.

A limitation of the study is that all 10 subjects performed the first experiment at the low altitude and the second one at high altitude. This could introduce some order effects i.e. the subjects could be more familiar with the task or could be more tired the second time. For this reason a very simple experiment with a simple finger movement was selected. A more complex type of movement would have enhanced the training effect. The tiredness can also be considered to play a minor role because all subjects performed the experiments between 8 and 12 a.m. and each experiment lasted only 6 min. All subjects started with the experiment at low altitude because they should not have any adaptation effects to high altitude.

There are three main advantages of carrying out the experiments at the Dachstein compared to a hypobaric chamber: (i) at Dachstein the partial pressure changes and not the oxygen saturation, (ii) subjects are available for longer experiments (up to days) and (iii) it is easier to recruit subjects.

Bernardi [1] showed that high altitude induces sympathetic activation in sea-level natives and lower sympathetic

activity in high-altitude natives. This effect was also seen despite long-term acclimatization at sea level by the high-altitude natives, thus indicating a persisting high-altitude adaptation which might be genetic. Thus HRV parameters seem to be a marker for the level of adaptation to high altitudes.

Studies showed also decreased finger-tapping speed, reduction of short-term memory and aphasic deficits [10] after hypoxic exposure to an altitude of 8850 m in a chamber simulation. A decrease in performance in a neuropsychometric test was also found in mountaineers who climbed above 8500 m 2–10 months after having returned to low altitude. The study showed EEG and magnetic resonance imaging (MRI) abnormalities [10]. The findings support the presumption that non-life threatening hypoxia may damage the brain at extreme altitudes. The present study showed that even at an altitude of 2700 m, which is a relatively low altitude, the EEG is effected. What is happening at the cellular level during hypoxia is not clear.

Even under little subjective awareness of the reduced amount of oxygen at an altitude of 2700 m, the beta rebound and HRV measures were significantly reduced. Even in a simple motor task such as brisk finger movement, changes in EEG can be observed. ERS data indicates a higher cortical excitability level at the TOP station compared to the BASE station. The fact that the changes were caused by a passive ascent to high altitudes is interesting. Further studies are planned to investigate the changes of ERD/ERS and HRV over several hours and days when subjects adapt themselves to the high altitude. The parameters might be useful as markers for the adaptation level to high altitudes.

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