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EEG, ECG and oxygen concentration changes from sea level to a simulated altitude of 4000 m and back to sea level

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ABSTRACT

In order to describe how high altitude affects the body during a one night stay at 4000 m experiments were performed in a hypobaric chamber and compared to a study on Dachstein (mountain in Austria, 2700 m). Ten subjects had to perform a reaction time task at different altitudes. The EEG and ECG were recorded simultaneously. Additionally, the oxygen saturation of the blood was measured at different altitudes and the subjects filled out a Lake Louise questionnaire that describes the degree of altitude mountain sickness (AMS). After elevation from 134 m to 4000 m in the hypobaric chamber heart-rate increased from 68.9 bpm to 81.6 bpm, RMSSD (root mean square of squared differences of adjacent heart beat intervals) decreased from 54.3 ms to 33.3 ms, the LF/HF ratio increased from 2.5 to 3.9 and oxygen saturation decreased to 82.7% after 11 h at 4000 m altitude. The Lake Louise Score (LSS) reached 3.4 after one night at 4000 m. EEG beta activity between 14 Hz and 18 Hz was attenuated at 4000 m and also after return to 134 m. The results indicate that the subjects were not able to adapt to 4000 m within 12 h in the hypobaric chamber. Even after 1 h after the return to 134 m all parameters are still affected from the night at 4000 m altitude. ECG and EEG changes are in line with results obtained at 2700 m height at Dachstein.

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Even with little subjective awareness of the reduced amount of oxygen at an altitude of 2700 m, the cardiovascular and central nervous system are already affected [8]. A study on the Dachstein showed that the heart-rate (HR) increased from 990 m altitude to 2700 m altitude in a group of 10 subjects [8]. Additionally, heart-rate variability (HRV) parameters were decreased significantly. Furthermore, with the increase in altitude, the sympathetic system becomes more active compared to the parasympathetic system. These effects were also shown in a study in a hypobaric chamber at 5000 m altitude [2] in long-term exposure studies to altitudes above 4000 m [1,6,9] and in mountaineers at 2700 m and 3700 m [10].

The Lake Louise Score (LLS) was created in Canada and is a simplified and standardized scoring system that allows diagnosis and quantification of mountain sickness in altitude research [15]. Currently, it is widely used by mountaineers and trekkers because it is short, has a simple format and is easy to complete. The LLS is sensitive enough to detect altitude mountain

sickness (AMS), but the very specific scoring system also avoids over-diagnosis.

Due to the reduced atmospheric pressure at high altitude, the alveolar oxygen pressure and therefore the arterial oxygen saturation (SpO₂) is reduced. The resulting lack of oxygen seen at tissue level is regarded as a crucial starting mechanism in the development of high altitude illness in hitherto healthy persons [4]. It can easily be measured non-invasively by pulse oximetry and serves as an objective indicator of oxygen delivery to the tissue. Some studies have shown that SpO₂ at rest correlates with high altitude symptomatology even at higher altitude and with high altitude performance [16,20].

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 [13]. 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. A right hand finger movement produces an ERD in the left hemisphere close to electrode position C3 of the international 10/20 electrode system. The recovery phase from the movement starts with movement offset and typically lasts between 1 s 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

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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 [13].

Event-related EEG changes were also analyzed in the Dachstein study. The subject had to press a button as fast as possible after a light flashed. This button press was analyzed with the ERD/ERS methods. The study showed that the ERD in the alpha range stayed almost constant between 990 m and 2700 m, but the ERS (beta rebound) decreased from +42% at 990 m to +12% at 2700 m. This study showed for the first time that the post-movement beta ERS is significantly attenuated at the high altitude compared to the low altitude measurement [8].

Both self-paced finger movement and electrical median nerve stimulation are terminated by a beta rebound of similar magnitude and latency [12]. Corticospinal excitability was shown in the reaction time movement tasks to be significantly reduced in the first second after EMG offset [3]. These findings support the hypothesis that beta ERS could be related to an idling or deactivated state [13,14], or even active immobilization of the motor cortex [17]. 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 [7,18]. 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 [8].

The ERD indicates the activated cortical areas involved in the processing of cognitive information and production of motor behavior. An enhancement of ERD can be found in Parkinson patients [5], in subjects with lower IQs [11] or during increased task complexity [19]. ERD changes in the alpha band, however, were not significant when the results from 2700 m and 990 m in the Dachstein study were compared. One explanation can be the relatively low altitude of 2700 m. The present study therefore investigates the changes of ERD at higher altitudes. A higher ERD could be expected with increasing altitude. Furthermore, the changes of the beta rebound will be investigated with the increasing altitude above 2700 m. Also the HR might increase more, and the HRV parameters decrease more at 4000 m compared to 2700 m.

Furthermore here we will investigate the changes of the ECG, EEG, LLS and SpO₂ parameters not only with increasing altitude, but also during a 12 h stay at 4000 m and after the return to sea level. The trend of the parameters over the night will show the adaptation level of the subjects. If highest HR, LLS, LF/HF ratio and lowest HRV values are found at the beginning of the night at 4000 m then the subject already adapted over the night. If these values are found after one night at 4000 m then the body is still adapting to the high altitude. Also the beta rebound can be used as marker for the adaptation. It will be investigated if the beta rebound shows normal values at the beginning or at the end of the night at 4000 m.

The hypothesis is that the parameters show higher changes after the night at 4000 m compared to the beginning of the night at 4000 m and the return to 134 m.

In June 2006 10 healthy subjects (all male, 21–33 years) participated in the experiment in the hypobaric chamber in Königsbrück/Germany. All subjects participated the first time in the experiment and were right handed. The subjects were informed of potential risks, where after a written consent was obtained.

A hypobaric chamber in Königsbrück was used to simulate the altitude. An elevation of 4000 m was reached after 1 h and kept for 12 h. The descent was also done within 1 h. The experiments were performed at four measurement points: MP1, at 134 m (between 1 day and 1 h before the ascent); MP2, 1 h after arrival at 4000 m; MP3, after 11 h at 4000 m; MP4, 1 h after the descent to 134 m. The

oxygen saturation and LLS was additionally measured after 5 h at 4000 m (MP2').

First EEG and ECG were recorded while the subjects performed a reaction time task. Each measurement lasted about 6 min. The subjects were sitting in front of the flashing light and were instructed not to move during the experiment so as to ensure high data quality. A Pocket PC-based recording system g.MOBilab (g.tec medical engineering GmbH, Graz, Austria) was used to acquire one ECG (Einthoven I) and two bipolar EEG channels (2.5 cm posterior and anterior to C3 and C4) with 256 Hz and 16 Bits. Gold electrodes with abrasive gel were used for a high signal quality.

The reaction time experiment consists of trials with 5 s length (plus a 0–2 s random interval to avoid adaptations). 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 finger was placed beside the button on the table. The blinking sequence of the green light (50 times) and the red light (10 times) is randomly distributed.

Then SpO₂ was measured with a portable measurement device with recording function (Palm Sat 2500, Nonin Medical) which was fixed on the index finger.

Finally the Lake Louise questionnaire was filled out. The questionnaire contains 8 questions about headache, gastrointestinal symptoms, fatigue/weakness, dizziness, difficulty sleeping, mental status, ataxia and peripheral oedema. AMS is present if there was an altitude rise AND the person has headache AND at least another symptom AND a total score equal or above 5 is reached (Min: 0; Max: 25).

The ECG recordings were used to calculate the SDNNindex (mean of the standard deviation of 30-s segments; describes the variability due to cycles shorter than 30 s), SDANN (standard deviation of the averages of RR intervals of all 30-s segments), RMSSD (square root of the mean squared difference of successive RR intervals).

The power spectrum (PS) was calculated to distinguish three components: very low frequency (VLF): <0.04 Hz, low frequency (LF): 0.04–0.15 Hz and high frequency (HF): 0.15–0.4 Hz. 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 LF/HF ratio describes the balanced behavior of both components. Fig. 1 shows the PS for subject S9 for the two different MPs.

The acquired EEG data was split into 6 s trials (2 s before the button press and 4 s afterwards). Then a visual artifact inspection and trial removal was performed and this yielded to 38–50 trials for each recording. Then the EEG data was first bandpass filtered in the specific frequency band. The power in the alpha band (8–13 Hz) was averaged from 1.5 s to 3 s, and in the beta band (14–18 Hz) from 3 s to 5 s. The same was performed in the reference interval (0.5–1.5 s). Then all trials were averaged and an ERD coefficient was calculated. A sign test for paired samples is used to check if the differences between the MPs are significant. Additionally ERD/ERS maps are shown in Fig. 1 for subject S9.

Table 1 gives the grand average of the MeanHR and HRV parameters at the four measurement points. The HR increased from MP1 to the highest values at MP3. Finally it decreased again at MP4. The HRV parameters RMSSD, SDANN and SDNNindex show the same behavior. The parameters decrease from MP1 to MP2 and decrease even further in MP3. At MP4 the value increases again.

The LFnorm and LF/HF parameters increase from MP1 to MP3 and return to almost their normal values in MP4. The HFnorm component does the opposite; it decreases from MP1 to MP3 and increases again in MP4. All parameters showed a significant difference between MP1 and MP3.

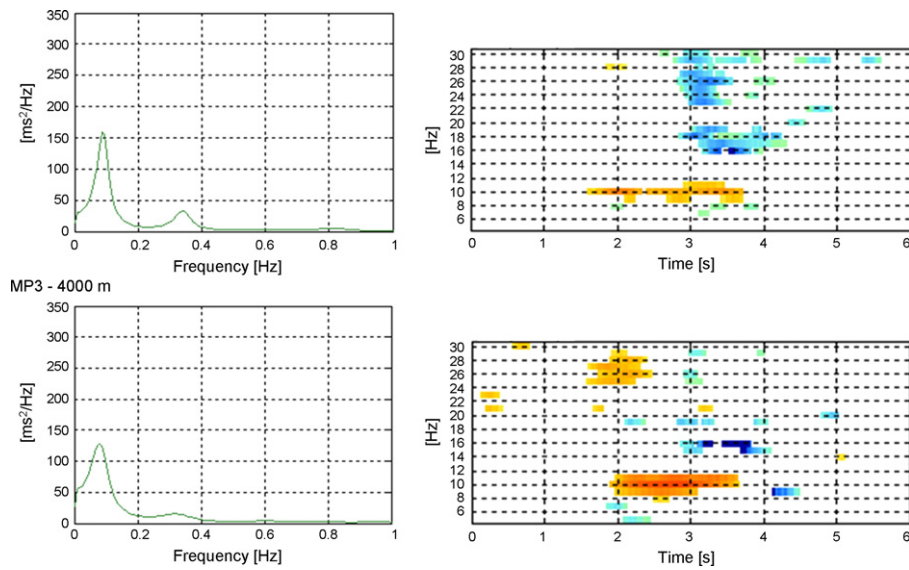


Fig. 1. Left: Power spectrum of RR intervals of subject S9. The activity of the high frequency band and of the low frequency is plotted. The transition from MP1 to MP3 clearly shows an increase of the LF/HF ratio. Right: ERD-map of subject S9. At MP1 the ERS can be found between 16 and 30 Hz and lasts from second 3 to 4.2. At MP3 the ERS appears smaller. The ERD appears between 8 and 12 Hz and lasts from second 1.6 until 3.7 at MP1 and from second 1.9 until 3.7 at MP3. The button was pressed at second 2.

The LLS starts with an obvious value of 0 at MP1 and increases with each MP to 3.4 in MP3. The oxygen saturation starts with 97.5% at MP1 and decreases to 82.7% at MP2'. In MP3 it increases again to 84.9%. MP1 is significantly different from MP2' and

MP3 in both cases. The reaction time between the onset of the green light and the button press increased from MP1 to MP3 and decreased again in MP4, but the changes were not significant. All subjects pressed 50 times correctly the button after the green

Table 1

Grand average (for all subjects) of HR and HRV parameters, Lake Louis Score, oxygen saturation (SpO₂) and reaction time with standard deviation for Königsbrück and Dachstein

	MP1	MP2	MP2'	MP3	MP4
Königsbrück	0 h–134 m	1 h–4000 m	5 h–4000 m	11 h–4000 m	13 h–134 m
Heart-rate					
MeanHR (bpm)	68.9 ± 9.3	80.3 ± 11.5		81.6 ± 8.8*	77.2 ± 8.5
HRV time domain					
RMSSD (ms)	54.3 ± 32.8	40.4 ± 15.1		33.3 ± 13.2 [†]	36.8 ± 16.1
SDANN (ms)	34.9 ± 5.3	29.8 ± 3.8		28.7 ± 5.6**	30.4 ± 5.0
SDNNindex (ms)	84.0 ± 27.0	73.4 ± 17.0		68.6 ± 23.5 [†]	68.9 ± 13.9
HRV frequency domain					
LFnorm (n.u.)	54.1 ± 15.6	57.2 ± 18.0		67.2 ± 10.8*	55.9 ± 20.9
HFnorm (n.u.)	31.7 ± 10.4	27.2 ± 14.2		20.2 ± 8.9*	26.8 ± 11.3
LF/HF (1)	2.5 ± 3.1	2.8 ± 1.9		3.9 ± 1.6*	2.7 ± 1.9
LLS	0 ± 0	0.5 ± 0.7	1.2 ± 1**	3.4 ± 3.7*	
SpO ₂	97.5 ± 0.5	97.2 ± 0.6	82.7 ± 6.8***	84.9 ± 4.5***	
Reaction time (s)	0.51 ± 0.09	0.52 ± 0.08		0.59 ± 0.12	0.55 ± 0.11
Green lights correct 50	50	50		50	50
Red lights pressed	0	0		0	1
Dachstein	0 h–900 m	1 h–2700 m			
Heart-rate					
MeanHR (bpm)	69.1 ± 12.6	80.4 ± 15.4**			
HRV time domain					
RMSSD (ms)	33.0 ± 28.5	14.8 ± 6.4**			
SDNNindex (ms)	58.1 ± 23.8	41.1 ± 12.2*			
HRV frequency domain					
LFnorm (n.u.)	51.1 ± 18.3	65.4 ± 19.3**			
HFnorm (n.u.)	35.1 ± 14.3	25.0 ± 14.1*			
LF/HF (1)	2.1 ± 1.98	4.4 ± 4.1 [†]			

* p < 0.05.

** p < 0.01.

*** p < 0.001.

Table 2
Grand average (for all subjects) of EEG activity at 134 m and 4000 m

	MP1: 0 h–134 m	MP2: 1 h–4000 m	MP3: 11 h–4000 m	MP4: 13 h–134 m
EEG activity				
8–13 Hz, C3, 0.5–1.5 s	7.99 **	6.82+ **	8.65 *	12.64**
8–13 Hz, C3, 1.5–3 s	4.11 **	4.10 *	5.00 *	6.52**
ERD (%)	–48	–36	–35	–43
14–18 Hz, C3, 0.5–1.5 s	1.04 +	0.88 +	1.04 **	1.69+
14–18 Hz, C3, 3–5	1.68	1.34 +	1.21 **	1.99
ERS (%)	+61	+52	+16	+17

An asterisk on the right hand side of a number shows that the changes are significant compared to 0 h–134 m. An asterisk under a number shows that the changes are significant compared to 13 h–134 m. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

light and only 1 subject pressed once the button after the red light.

Table 2 shows the grand average EEG activity in the alpha (8–13 Hz) and beta (14–18 Hz) frequency range at the 4 MPs for the reference and action intervals. The alpha activity in the reference interval (0.5–1.5 s) decreases from MP1 to MP2, increases to MP3 and increases further in MP4 on channel C3. MP1 and MP4 are significantly different. In the action interval (1.5–3 s) the alpha activity decreases from MP1 to MP2 and increases from MP2 to MP4. Again MP1 and MP4 show a significant difference. MP4 is significantly different from all other MPs.

In the beta band (14–18 Hz) and reference interval (0.5–1.5 s) the activity decreases from MP1 to MP2 and increases again until MP4. But the differences are not significant. In the action interval (3–5 s) the beta activity is decreasing from MP1 to MP3 and finally increasing in MP4. A significant difference between MP1 and other MPs could not be found. But MP3 and MP4 are significantly different.

The lower beta rebound, higher HR, lower HRV measures, a high sympathetic activity and lower parasympathetic activity, a higher LLS and lower SpO₂ were found during a one night sleep at 4000 m. All parameters together describe very nicely the effect of the reduced amount of oxygen to the human body at 4000 m and also during the adaptation process to this altitude.

Table 1 shows also the grand averages for 10 subjects of the Dachstein study. The Dachstein and Königsbrück groups had a very similar HR (69.1 bpm and 68.9 bpm) at the base stations (900 m and 134 m) and also at the top stations (2700 m and 4000 m) (80.4 bpm and 80.3 bpm). Of special interest is that the HR did not increase much further after 11 h at 4000 m (81.6 bpm). The changes at Dachstein were significant with a p -value < 0.01 . In Königsbrück only the changes from MP1 to MP3 were significant ($p < 0.05$). It was expected that the higher amplitude of 4000 m causes a higher change of the HR which was not the case. An explanation could be the different age distribution of the subjects. At Dachstein the subjects were between 25 and 57 years and in Königsbrück the subjects were between 21 and 33 years old and at Dachstein the measurements were made with people attending a ski opening, in Königsbrück trained subjects from the German army participated. Even after a whole night at 4000 m the HR was in the same range as at 2700 m, but this time significantly different from the measurements at MP1. Very interesting is the still high HR at MP4 which shows the influence of the lack of oxygen, the stress in the hypobaric chamber and the reduced sleep during the last night.

At Dachstein and in Königsbrück LFnorm increased from 51.1 and 54.1 (base station) to 65.4 and 57.2 (2700 m and MP2). HFnorm decreased from 35.1 and 31.7 (base station) to 25.0 and 27.2 (2700 m and MP2). Therefore the parameters are again very similar at the base station compared to the top stations (2700 m and MP2).

LFnorm increased further to 67.2 and HFnorm decreased further to 20.2 at MP3. But now the changes of the LFnorm and HFnorm parameters describe very nicely the additional effect of spending the whole night at 4000, which was not clearly seen in the HR. Also the recovery at MP4 can nicely be seen by an increase of the parasympathetic activity and a decrease of the sympathetic activity. Increased sympathetic and decreased parasympathetic nervous system activity were also found in an early phase of acclimatization above 4000 m by Hughson et al. and Farinelli et al. [6,9] and during exercise at 3500 m in a hypobaric chamber [21]. In the current study additionally the changes over a one night sleep were investigated and quantified.

The difference of the oxygen saturation between base station and MP1 is not very high because of the short time interval spent at 4000 m. But during the night the oxygen saturation drops down to only 82.7% at MP2' after 5 h and it increases again slightly to 84.9% after 11 h at MP3. The LLS increase from base station to MP1 and further during the night. Very interesting is that the highest LLS was reached at MP3. Therefore the subjective awareness of high altitude symptoms might be delayed compared to SpO₂.

At Dachstein it was found the first time that the post-movement beta ERS was significantly attenuated at the high altitude of 2700 m compared to the low altitude measurement at 990 m. At the base station the maximum beta ERS was around +42%, at the TOP station it was only +12%. In Königsbrück the beta bandpower at the base station increased by +61%. At MP2 the beta rebound was already reduced to +52% and at MP3 it was only +16%. Also at MP4 the beta rebound was still only 17%. This shows the immediate effect of the lack of oxygen on event-related EEG beta activity. Very interesting is that the activity decreased only slightly within the first hour at 4000 m but very much after 11 h. Even after some recovery time the beta activity was still very low. This shows again that the beta rebound can be seen as marker of the high-altitude effects on the central nervous system and the first time that the beta rebound describes also the response of the brain to the lack of oxygen in the air over a whole night. It can be expected that with successful adaptation, the beta rebound will return to the values found at sea level. In the current study the adaptation time of 11 h to 4000 m and even after 1 h at sea level after the night was not long enough. Therefore in future studies the investigation of several days at this altitude will be interesting.

It must be noted that the changes in the beta range were not significant (except MP3 versus MP4), but the trends are in line with the Dachstein study and only 10 subjects were analyzed. With only 10 subjects it is very difficult to get significant results; therefore in future studies more subjects are planned.

At Dachstein ERD changes in the alpha band were not significant when results from 2700 m and 990 m were compared. An

explanation was the relatively low altitude of 2700 m. Therefore changes of ERD in the alpha range were also investigated at 4000 m and a higher ERD was expected with increasing altitude. But the ERD becomes even smaller with increasing altitude as shown in Table 2. At MP4 the ERD reached almost its initial value. In literature increased ERD values were found e.g. during increased task complexity, in Parkinson patients or in subjects with lower IQs. The reaction time increased with the altitude, but all subjects responded to the green lights correctly in all 50 trials and did not press the button after the red flashes (except one case). As conclusion can be stated that the task complexity at 4000 m does not increase for the subjects. This is now proven twice at Dachstein and in Königsbrück.

Interesting is also that the oxygen saturations at MP1 and MP2 were very similar, but the ECG and EEG parameters already reacted. The body reacts at high altitude with hyperventilation to compensate the lack of oxygen. At MP4 the ECG parameters started already to return to their initial values. Interestingly the alpha and beta bandpower values increased above the initial values at MP1. This shows that the central nervous system is still influenced although SpO₂ returns normally within 1–2 min to full saturation when the sea level altitude is reached. Hence the result of this study at high altitude might also be important for studies performed at sea level. Therefore the air exchange rate in the lab where the experiment is performed must be controlled because the lack of oxygen could also effect the result of the studies in e.g. HRV and EEG. This might be especially important for long sleep studies in small rooms.

Summarizing the physiological parameters after the night at 4000 m showed higher changes than at the beginning at 4000 m and return to 134 m altitude. This is also in line with the experience of high altitude mountaineers which mostly get ill during the night and not at the beginning.

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