Neuroscience Letters 442 (2008) 123–127

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

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

Christoph Guger[a,b,*], Stefan Krausert[c], Wolfgang Domej[b,d], Günter Edlinger[a], Markus Tannheimer[c]

[a] g.tec – Guger Technologies OEG, Herbersteinstrasse 60, 8020 Graz, Austria
[b] ARGE-Alpinmedizin, Austria
[c] Department of Surgery, Military Hospital Ulm, Germany
[d] Department of Internal Medicine, University Medical Center, Graz, Austria

ARTICLE INFO

Article history:
Received 4 January 2008
Received in revised form 10 June 2008
Accepted 26 June 2008

Keywords:
High altitude
Event-related desynchronization (ERD)
Heart-rate variability
Beta oscillations
ECG
EEG

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.

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

* Corresponding author at: g.tec – Guger Technologies OEG, Herbersteinstrasse 60, 8020 Graz, Austria. Tel.: +43 7251 22240 12; fax: +43 7251 22240 39.
E-mail address: guger@gtec.at (C. Guger).

0304-3940/$ – see front matter © 2008 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.neulet.2008.06.075

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 distinguished 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.