Cerebral Oxygenation At High Altitude: Reproducible Data After Acclimatisation At 5050 Meters During A Common Trekking Tour In The Nepal Himalayas

I Hadolt, G Litscher, V Sattelmeyer

Citation

Abstract
Trekking at high altitudes (>2500 m above sea level) demands adequate acclimatisation to hypobaric hypoxia. When adaptation is insufficient, health is threatened by acute mountain sickness (AMS), high altitude cerebral edema and high altitude pulmonary edema, respectively.

Regional cerebral oximetric values ($rSO_2$) were measured in 27 adults (8 f, 19 m; mean age $\pm$ SD group A: 39.5 $\pm$ 11.2 years; group B: 44.8 $\pm$ 14.9 years) during two different ascending routes to an altitude of 5050 m using an INVOS 5100 cerebral oximeter. The results showed reproducible data in both groups (49.0 $<$ mean $rSO_2$ $<$ 55.0). There was a significant ($p < 0.05$; ANOVA, Tukey test) decrease compared to baseline values at a level of 133 m.

Further studies are necessary to obtain data from the acute phase of hypobaric hypoxia and from persons with AMS.

INTRODUCTION

High altitude, defined as heights between 2500 and 5300 m above sea level, can become dangerous to health because of increasing hypobaric hypoxia, especially when acclimatisation has been insufficient [1]. At an altitude of 5000 m oxygen partial pressure is reduced to about one-half compared to sea level and peripheral oxygen saturation ($SaO_2$) in the healthy human body is reduced to values of about 75% [2]. It is possible, that such decreases in $SaO_2$ influence cerebral oxygenation, and may be an important key in developing acute mountain sickness (AMS) and high altitude cerebral edema (HACE).

To our knowledge, only five studies concerning regional cerebral oxygenation ($rSO_2$) at high altitude measured with transcranial near infrared spectroscopy (NIRS) exist. As expected, all authors found a decrease in $rSO_2$, however the decrease was less pronounced than the changes in $SaO_2$, which is supposed to be an unknown compensation mechanism of brain [3,4,5,6,7]. Nevertheless, results have to be interpreted carefully because of dissimilar measurement conditions (different altitudes, kind and rate of ascent, geographical location, temperature) and methods (Critikon 2020 system, INVOS 3100 and 5100 system). Based on the common opinion that the rate of ascent is a main factor for developing AMS [4], the goal of this study was to evaluate, whether various routes, with similar rates of ascent result in different or reproducible changes in brain oxygenation data.

For the first time, this present study seeks measured $rSO_2$ from two different trekking groups, who started from the same location (2850 m) and time, but ascended to an altitude of 5050 m using two different routes and returned back within 22 days (Fig. 1).

Figure 1
Fig. 1: Expedition routes of the 22-day trekking tour in the Nepal Himalayas.
METHODS AND MATERIALS

NIRS is a non-invasive method to measure rSO$_2$, which is based upon the principles of light absorption and transmission in oxygenated and desoxygenated haemoglobin in cerebral vessels. Light of defined wave-length is emitted through the skull and the spectral absorption of cerebral tissue can be determined by detecting the returned light [9]. NIRS has been used successfully in research and clinical treatment to observe brain function during surgery and in critical care patients [10].

In our study, an INVOS 5100 cerebral oxymeter (Somanetics Corp., Troy, USA) was used to measure alterations in rSO$_2$. Each measurement was performed in sitting position (Fig. 2). First, skin was cleaned with alcohol before sensors were applied to the left and right side of forehead according to the manufacturer's recommendations. To minimize influences from extracerebral light, the forehead was covered with a black cloth during measurement. Data was recorded after a three-minute resting period.

Figure 2
Fig. 2: Healthy volunteer during measurement of rSO (regional cerebral oxygen saturation) at high altitude (5050 m).

Two groups of hobby mountaineers (group A; Island Peak group; n=15; 4 female, 11 male; mean age + SD: 39.5 + 11.2 years) (group B; Gokyo group; n=12; 4 female, 8 male; mean age + SD: 44.8 + 14.9 years) were investigated during different trekking routes in the Khumbu region of Nepal. Fifty-three days before starting the trekking tour, rSO$_2$-values from all volunteers were measured near sea level (133 m, Herxheim, Germany) in order to obtain NIRS- and medical control data (blood samples, blood pressure, electrocardiogram, lung function analysis, transcranial and extracranial Doppler sonography).

The study “Neuromonitoring Silberpyramide 2002” was approved by the ethics committee of the University of Graz (12-055 ex 01/02) and written informed consent was obtained from all volunteers.

The two groups started at an altitude of 2850 m and were measured the day after arrival at an altitude of 5050 m. Duration of ascent was 12 days. During these 12 days, group A had reached a maximum altitude of 5357 m and group B had climbed up to a maximum of 6000 m (compare Fig. 1).

Each group was accompanied by a sherpa team and yaks for alimentation and transport of equipment. Twice a day the volunteers were checked by the expedition doctor (AMS score, pulse oximetry, haematocrit and blood glucose three times during the entire trek). Only data from healthy participants were included in the study.

STATISTICAL ANALYSIS

The data were tested with analysis of variance using the software SigmaStat 2.03 software (Jandel Scientific Corp., Erkrath, Germany). In addition the post hoc analysis Tukey test was used. Changes were considered significant at a p-value < 0.05.

RESULTS

Figure 3 shows the baseline data (133 m) of left and right rSO$_2$ values and the data at an altitude of 5050 m of both groups. Group A (rSO$_2$ left: 70.5 + 6.2 %; rSO$_2$ right: 71.7 + 9.0 %) had higher baseline values compared to group B (rSO$_2$ left: 63.8 + 6.7 %; rSO$_2$ right: 65.1 + 6.5 %) (n.s.).
Figure 3
Fig. 3: Regional cerebral oxygen saturation (rSO) from the left and right frontal area at different altitudes (133 m, 5050 m) in two healthy volunteer groups (A, B).

At an altitude of 5050 m the values from both groups were reduced significantly (p<0.05). The values in group A (rSO<sub>2</sub> left: 54.7 + 11.6 %; rSO<sub>2</sub> right: 55.0 + 9.4 %) were less reduced than those in group B (rSO<sub>2</sub> left: 49.0 + 6.5 %; rSO<sub>2</sub> right: 50.2 + 7.3 %). With regard to the different baseline data and the percentual reduction of rSO<sub>2</sub>, the decrease was almost the same (compare Fig. 3).

DISCUSSION
With increasing altitudes, the partial pressure of inspired oxygen decreases. This requires sufficient acclimatisation of the human body at high altitudes to manage low oxygenation without health risks. The symptoms of altitude related disorders like AMS, including HACE and high altitude pulmonary edema (HAPE) can be verified with AMS score and treatment, however, the accurate pathophysiology regarding development of these disorders has not yet been clarified [1].

The present study deals with the effect of hypobaric hypoxia on the brain. To this day, neither commonly recognized values regarding the status of brain oxygenation at high altitudes before and after acclimatisation, nor knowledge of oxygen decrease in brain correlated symptoms in AMS or HACE exist. These could be important factors for the development of such disorders. Further it is very important to recognise AMS as early as possible, due to the very difficult rescue conditions of sick people at high altitudes. Rate of ascent is the main risk for developing AMS [1]. Only common recommendations based on experience concerning ascend velocity exist, however no facts with reproducible measurements are available.

Considering these aspects, we chose study conditions like those performed by thousands of trekking tourists in the Himalaya region every year. Further, we wanted to get reproducible data from different groups in order to get common recognised values in literature. Compared with the rSO<sub>2</sub> measurements by other authors [1, 3, 4, 5, 6], we found an important difference in our results [1]: The decrease in our rSO<sub>2</sub> values was much more pronounced than those of values measured by other authors. This fact could be a consequence of measurement conditions such as rate of ascent (active versus passiv ascent in different time manner) and geographical location (Himalaya or Ands). One study by Imray et al. [6] was also performed in the Himalaya region at an altitude of 5005 m and the conditions of ascent and temperature were similar. However, these authors also found much more higher rSO<sub>2</sub> values compared to our results. In our opinion one of the reasons could lie in the different technical equipment, which has to work at during low temperatures to minus 10 °C [11]. This demands the implementation of temperature compensation and signal quality index which is realized in the INVOS 5100 oxymeter, but not in the Critikon 2020 system.

The reduction of rSO<sub>2</sub> to such low values is remarkable, because our volunteers had no symptoms of illness. Similar data were found in critical care patients after severe head injury using the same oxymeter. It seems that the brain can manage very low values, it there is time enough to adapt.

CONCLUSIONS
At an altitude of 5050 m, when inspired partial pressure is about one-half of the value compared to sea level, rSO<sub>2</sub> values can decrease to absolute values of about < 49 %
without any clinical symptoms of AMS. These data are reproducible within two trekking groups using two different routes of ascent with the same starting point and rate of ascent at an altitude of 5050 m. All in all, 27 persons were investigated. It seems, that the brain can manage lower levels of oxygenation, if there is enough time to adapt, but the actual mechanism is still unknown. This study documents reproducible data of cerebral oxygen status at an altitude of 5050 m after acclimatisation. Further measurements are necessary to obtain data from the acute phase of hypobaric hypoxia and from persons with AMS.

ACKNOWLEDGEMENTS

This study was supported by the Styrian Government, Department of Science and Research (“Neuromonitoring Silberpyramide 2002”; GZ: FA6A-12S57-02/1). The project has been developed within the framework of the Ev-K²-CNR/RONAST Joint Scientific and Technological Research Project and further by the “Österreichische Gesellschaft für Alpin- und Höhenmedizin” (Project leader: “Silberpyramide 2002” Dr. Robb Waanders, Feldkirch, Austria)

The authors would also like to express their thanks to all the members of the scientific team, especially for the excellent support of the expedition doctor Ascher Reinhard M.D., Tirol, Austria and finally to all the volunteers for their precious cooperation.

References

Author Information

Irmgard Hadolt, MD
Department of Biomedical Engineering and Research in Anesthesia and Critical Care, University of Graz

Gerhard Litscher, PhD
Department of Biomedical Engineering and Research in Anesthesia and Critical Care, University of Graz

Vera Sattelmeyer, MD
Department of Neurosurgery, Horst-Schmidt-Klinik