Arterial oxygen saturation during ascending to altitude under various conditions: Lessons from the field

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Summary When hypoxia increases during ascending in the mountains, ventilatory control and the related oxygenation may be challenged. The pre-treatment by intermittent hypoxia will elevate ventilation and offset hypoxemia and acetazolamide may inhibit peripheral chemosensitivity and act through central mechanisms. To study these effects in the field, one well-trained male mountaineer performed four ascents from low (1300 m) to higher altitude (2600 m): (1) under control conditions, (2) after intermittent hypoxia, (3) after pre-treatment with acetazolamide, and (4) after intermittent hypoxia + acetazolamide. When ascending under control conditions a cascading decrease of arterial oxygen saturation (SaO₂) has been observed probably because of the alternating dominance of peripheral and central mechanisms of ventilatory control. While the pre-treatment with intermittent hypoxia prolonged the constant SaO₂ periods, the intake of acetazolamide eliminated this respiratory periodicity. Oxygen desaturation was best prevented by acetazolamide which was also associated with faster ascent times compared to control conditions.

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Introduction

Typically, mountaineers ascend from low to higher altitudes. In contrast to exercise at low altitude they are exposed to continuously increasing hypoxia. Sufficient ventilation is important to maintain arterial oxygen saturation (SaO₂) and aerobic exercise performance. Ventilatory control, however, is particularly complex during exercise in hypoxia where the controller depends on the interaction among several different mechanisms. Ventilatory control and the related SaO₂ values might be modified, e.g. by pre-exposure to intermittent hypoxia or the intake of acetazolamide. Short-term intermittent hypoxia has been reported to increase ventilation and SaO₂ during subsequent exposure to hypoxia at rest and during
submaximal exercise in hypoxia probably caused by enhanced hypoxic chemosensitivity of the peripheral chemoreceptors.\textsuperscript{3,4} In contrast, the intake of acetazolamide provokes metabolic acidosis and increases the ventilatory response to the exercise mainly via central chemoreceptors and improves pulmonary gas exchange.\textsuperscript{5,6} Although often used by mountaineers, it is unknown how such interventions would impinge on the SaO\textsubscript{2} course during ascending to altitude. Thus, the main objective of this pilot study was the monitoring of the SaO\textsubscript{2} course in one mountaineer during a prolonged ascent to altitude: (1) under control conditions, (2) after intermittent hypoxia, (3) after the intake of acetazolamide, and (4) after combined intermittent hypoxia and acetazolamide.

### Methods

One healthy, well-trained and regularly exercising male mountaineer (52 years, 68 kg, 176 cm, VO\textsubscript{2}\text{max} \sim 65 ml/(min kg)) performed four ascents from low to higher altitude. He had a normal isocapnic hypoxic ventilatory response (0.5 l/(min %saturation)) and was not uncommonly susceptible to acute mountain sickness. He normally lived at low altitude (700 m) and was transported by car to an altitude of 1300 m and from there he ascended to about 2600 m. Ascents were performed in the winter season on skis carrying a light backpack (3 kg) with time intervals of 3 weeks between each ascent. The routes and the track conditions of the four ascents were very similar (solid tracks, temperature: +4 to −4°C, no wind). One month before and during the test period no altitude sojourn above 1800 m was allowed. The first ascent represents control conditions. The second was carried out after intermittent hypoxic exposures consisting of five times of 1 h normobaric hypoxia (14% FiO\textsubscript{2}, equivalent to about 3700 m) on five consecutive days with the intent to enhance the sensitivity of peripheral chemoreceptors.\textsuperscript{4} The third was performed after two times 125 mg intake of acetazolamide within a time interval of 9 h with the intent to enhance the sensitivity mainly of the central chemoreceptor drive.\textsuperscript{5} The effectiveness of such low dosages have been demonstrated and is also indicated because diuresis increased. The fourth ascent was performed after intermittent hypoxia + acetazolamide pre-treatment.

SaO\textsubscript{2}, barometric pressure, and heart rates were monitored continuously using the Physilog device (Driesen + Kern GmbH, Germany). End-tidal CO\textsubscript{2} (PetCO\textsubscript{2}; Tidal Wave Sp, Novametrix, Austria) was measured at about 30-min intervals.

### Results

The altitude-dependent SaO\textsubscript{2} courses of the four ascents are schematically shown in Fig. 1. Original data including PetCO\textsubscript{2} values are shown in the electronic version.

SaO\textsubscript{2} values vary rhythmically. The cycle length after intermittent hypoxia seems to be prolonged and after acetazolamide and intermittent hypoxia + acetazolamide cycles seems even to disappear. In general, each cycle consists of a more steep decrease of SaO\textsubscript{2} values with increasing altitude, followed by a rather horizontal SaO\textsubscript{2} period despite increasing altitude. After intermittent hypoxia the constant SaO\textsubscript{2} period is prolonged and starts at a lower threshold. After acetazolamide, the initial SaO\textsubscript{2} decrease is followed by a slowed continuous decrease of SaO\textsubscript{2}. After intermittent hypoxia + acetazolamide, this slowed decrease starts at somewhat a lower SaO\textsubscript{2} threshold compared to acetazolamide alone. SaO\textsubscript{2} values decreased during the ascent under control conditions from 98% (700 m, rest) to 83% (2600 m, exercise); delta SaO\textsubscript{2}: 15%. Delta SaO\textsubscript{2} after intermittent hypoxia + acetazolamide was 12%, after acetazolamide 9%, and after intermittent hypoxia + acetazolamide also 9%. The average values of heart rates during the four ascents were very similar (136 \pm 2 b/min). The time needed for the ascent under control conditions was 132 min (590 m in altitude per hour), after intermittent hypoxia this time was 128 min (609 m/h), after acetazolamide 122 min (639 m/h), after intermittent hypoxia + acetazolamide 121 min (639 m/h).

![Figure 1](image-url) Schematically presented SaO\textsubscript{2} courses during the ascents from about 1300 m (arrow) to about 2600 m under various conditions. IH = intermittent hypoxia; AZ = acetazolamide; mb = millibar.
Arterial oxygen saturation under various conditions and after intermittent hypoxia + acetazolamide 125 min (624 m/h).

Discussion

The most interesting observations of the presented pilot study are (1) that the SaO2 course during ascending to altitude follows a biphasic pattern and (2) that intermittent hypoxia, acetazolamide, and intermittent hypoxia + acetazolamide clearly changed the rhythmic variation of the SaO2 course seen during the control ascent to altitude.

(1) Under control conditions, constant SaO2 periods with rather decreasing PetCO2 may reflect increasing ventilatory equivalents for CO2 (VE/VCO2) when the inspiratory oxygen pressure decreased during the ascent. In contrast, the periods of decreasing SaO2 with relatively constant PetCO2 may indicate steady state VE/VCO2 despite decreasing inspiratory oxygen pressure during the ascent.

(2) The pre-treatment with intermittent hypoxia and/or acetazolamide clearly modified the SaO2 pattern observed under control conditions. Intermittent hypoxia is known to enhance the hypoxic ventilatory response3,4 associated with an augmented sensory response of peripheral chemoreceptors to acute hypoxia.7 Therefore, this likely explains the prolonged constant SaO2 periods after pre-treatment with intermittent hypoxia. On the other hand, the metabolic acidosis produced by acetazolamide administration stimulates breathing at least partly by increasing the tonic output of the central chemoreceptors.5 This again may explain the flattening of the decreasing SaO2 periods.

But it cannot entirely be excluded that small changes in the ascent rate may have influenced these findings.

In conclusion, intermittent hypoxia and acetazolamide modify the cascading decrease of SaO2 when ascending to altitude under control conditions probably because of their selective effects on peripheral and central mechanisms of ventilatory control. These interventions tend to improve exercise performance.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jsams.2007.08.006.

References