Changes in sleep quality of athletes under normobaric hypoxia equivalent to 2,000-m altitude: a polysomnographic study

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Submitted 20 March 2007; accepted in final form 7 August 2007

Hoshikawa M, Uchida S, Sugo T, Kumai Y, Hanai Y, Kawahara T. Changes in sleep quality of athletes under normobaric hypoxia equivalent to 2,000-m altitude: a polysomnographic study. J Appl Physiol 103: 2005–2011, 2007. First published August 9, 2007; doi:10.1152/japplphysiol.00315.2007.—This study evaluated the sleep quality of athletes in normobaric hypoxia at a simulated altitude of 2,000 m. Eight male athletes slept in normoxic condition (NC) and hypoxic conditions equivalent to those at 2,000-m altitude (HC). Polysomnographic recordings of sleep included the electroencephalogram (EEG), electrooculogram, chin surface electromyogram, and electrocardiogram. Thoracic and abdominal motion, nasal and oral airflow, and arterial blood oxygen saturation ($SaO_2$) were also recorded. Standard visual sleep stage scoring and fast Fourier transformation analyses of the EEG were performed on 30-s epochs. Subjective sleepiness and urinary catecholamines were also monitored. Mean $SaO_2$ decreased and respiratory disturbances increased with HC. The increase in respiratory disturbances was significant, but the increase was small and subclinical. The duration of slow-wave sleep (stage 3 and 4) and total delta power (<3 Hz) of the all-night non-rapid eye movement sleep EEG decreased for HC compared with NC. Subjective sleepiness and amounts of urinary catecholamines did not differ between the conditions. These results indicate that acute exposure to normobaric hypoxia equivalent to that at 2,000-m altitude decreased slow-wave sleep in athletes, but it did not change subjective sleepiness or amounts of urinary catecholamines.

normobaric hypoxia; slow-wave sleep; polysomnography; respiratory disturbances

“LIVING-HIGH, TRAINING-HIGH” altitude training conditions have been widely used to enhance athletic performance (6). In recent years, athletes have also used devices such as altitude tents or nitrogen houses to simulate the reduced oxygen concentrations at high altitude for the same purpose (41). Recent studies on the effects of both actual and simulated altitudes by athletes suggest that performances improve when subjects live at mildly to moderately high (~3,000 m) altitude and spend training hours at sea level (20, 21, 33). Even in such “living-high, training-low” conditions, athletes also spend several hours sleeping in a high-altitude condition. In both living-high, training-high and “living-high, training-low” conditions, some athletes complain of headache and difficulty breathing during sleep (32). In their review, Fulco et al. (10) found that reduced appetite, restless sleep, and altitude sickness commonly occur during acute altitude residence, which could impair exercise training and performance.

Here, we focused on the physiological and subjective changes in sleep under acute hypoxia. Although the primary purpose for sleep remains unknown, sleep is known to play important roles in recovering from waking activities. Sleep disturbance has been shown to decrease subsequent physical work capacity and to increase the subjective perception of fatigue (2). At altitudes above 3,500 m, several studies have demonstrated a range of sleep disturbances such as increases in the number of arousals, increases in stage 1 and 2 duration, and decreases in the durations of slow-wave and rapid eye movement (REM) sleep (11, 23, 24, 28, 30). Slow-wave sleep is particularly believed to represent a homeostatic process (8) or restorative processes (25, 31). Furthermore, computer analysis of sleep EEG revealed that delta frequency electroencephalogram EEG increased in the recovery sleep from sleep loss (9).

Athletes training in living-high conditions sleep at altitudes below 3,000 m in most cases. The number of studies that have examined sleep quality at altitudes below 3,000 m is limited. Although some coaching staff claim that athletes experience some degree of sleep disturbance in practical living-high conditions (32, 40), no study to date has demonstrated any decrease in slow-wave sleep below 3,200 m (16, 24, 26, 42). Kinsman et al. (18) recently reported that some athletes showed respiratory events and periodic changes in breathing at a simulated altitude of 2,650 m (18). However, they did not report any statistically significant decreases in slow-wave sleep (stage 3 and 4) time (16). Pedlar et al. (26) reported that the subjective “behavior following waking” score of the Leeds Sleep Evaluation Questionnaire was significantly lower after sleep at a simulated altitude of 2,500 m. Again, however, they did not find any decreases in slow-wave sleep.

Thus, although subjective problems have been reported after sleep at altitudes below 3,000 m, decreases in slow-wave sleep have not been reported. However, in those studies, sleep disturbances and sleep quality, as reflected by well-consolidated expressions of slow-wave sleep, were only estimated by the standard methods for visual sleep stage scoring. If the coaching staffs’ observations of increased sleep disturbance were correct, then reductions in slow-wave sleep at altitudes below 3,000 m may be very subtle or otherwise difficult to detect from visual scoring. Visual sleep stage scoring was developed as a method based on amplitude threshold criteria for the purpose of reducing the complexities of EEG epochs into categorical stages. Thus standard visual scoring provides

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no method to capture changes in the mix of ongoing waveform amplitudes, allowing the possibility that substantial changes in slow-wave expression would produce no change in the number of epochs counted as sleep stage 3 and 4.

In the present study, we investigated the effects of acute exposure to normobaric hypoxia, which simulated a relatively low altitude (2,000 m), on sleep quality. We evaluated sleep quality using both visual sleep stage scoring and fast Fourier transformation (FFT) analyses of the slow EEG. Slow-wave activity was quantified using the total delta (< 3 Hz) power from whole-night non-REM (NREM) sleep EEG recordings, which we expect to be more effective for assessing subtle changes in slow-wave sleep. The purpose of this study was to evaluate the changes in sleep quality of athletes at a simulated altitude of 2,000 m.

METHODS

Subjects. Eight male intercollegiate-level athletes (age, 21.9 ± 1.6 yr; height, 174.0 ± 3.7 cm; weight, 57.4 ± 4.1 kg; maximal oxygen consumption, 62.8 ± 5.5 ml kg⁻¹min⁻¹) participated in this study. All specialized in middle- or long-distance running. Each reported regular sleeping habits, and no subject was medicated with psychoactive or other agents. All subjects had been kept away from high-altitude and hypoxic environments for more than 5 mo before the study. The subjects were not studied during the training season, but they maintained their normal off-season training programs, such as few kilometers of light jogging, throughout the study. Written informed consent was obtained after explaining the experimental procedure and possible risks. This study was approved by the Japan Institute of Sports Sciences Ethics Committee.

Data collection and altitude simulation. Subjects slept for 3 consecutive nights monitored by polysomnography (PSG). The first and second nights were under normoxic conditions (NC, natural altitude of 22 m, 20.9% oxygen). On the third night a normobaric hypoxic condition simulated the oxygen concentration at a moderate altitude of 2,000 m (HC; 16.4% oxygen). The first night allowed adaptation to the PSG procedures and equipment; only data from the second and third nights were used for analyses. In this study, all subjects experienced the two conditions in the same order. Although counterbalancing would have been better, we were concerned that sleep under HC could affect next-day sleep quality under NC. The experiments were performed in a specially designed room at the Japan Institute of Sports Sciences (Tokyo, Japan), where NH can be configured by oxygen filtration. The room temperature was set at 22 ± 1°C.

Nocturnal PSG. For each day of the study, subjects were instructed to abstain entirely from alcohol and from caffeinated beverages after noon. Subjects did not nap during the day. On arrival at their dormitory rooms around 2000, electrodes were attached and the subjects were encouraged to relax before going to bed. They were free to attempt natural sleep. Lights were turned off when they requested. They requested to turn off the lights between 2230 and 2400. The subjects were allowed to remain in their beds for 8 h. PSG recordings included EEG(C3/A2 and C4/A1), electrocrocogram (EOG), a chin-surface electromyogram (EMG), and an electrocardiogram (ECG), which were monitored and recorded on a personal computer using sleep analysis software (Neurofax EEG-1524, Nihon Kohden, Tokyo, Japan) at a sampling rate of 500 Hz. The EEGs were recorded with a high-cutoff filter set at 120 Hz and a low-cutoff filter set at 0.5 Hz (a time constant of 0.3 s). The EOGs were recorded with a high-cutoff filter set at 30 Hz and a low-cutoff filter set at 0.08 Hz (a time constant of 2.0 s). The EMG was recorded with a high-cutoff filter set at 120 Hz and a low-cutoff filter set at 5.3 Hz (a time constant of 0.03 s). The ECG was recorded with a high-cutoff filter set at 60 Hz and a low-cutoff filter set at 0.16 Hz (a time constant of 1 s). The same monitoring system recorded thoracic and abdominal motion from piezoelectric bands, nasal and oral airflow from thermistor temperature changes, and arterial blood oxygen saturation (SaO₂) determined by pulse oximetry (model OLY-3100, Nihon Kohden). Thoracic and abdominal motion, nasal and oral airflow were recorded with a high-cutoff filter set at 30 Hz and a low-cutoff filter set at 0.08 Hz (a time constant of 2.0 s).

Data analysis: sleep, respiratory variables, and SaO₂. The PSG data were segmented into 30-s epochs. Visual sleep stage scoring was performed on the 30-s epochs according to standard Rechtschaffen and Kales criteria (27). Microarousals were further defined as increases in EEG frequency for 3 s or longer, coincident with any duration of increased EMG activity (34).

Delta (< 3 Hz) EEG power was obtained by FFT analyses of the C3/A2 EEG (500-Hz sampling; eight 4.096-s data sequences) epochs identified by the visual scorer to be free from artifacts. Because of the low-cutoff filter, the delta EEG power mainly reflects a power of 0.5–3 Hz. Because the eight 4.096-s sequences are longer than the visual epoch definitions, both sides of each sequence overlapped (Fig. 1). Epochs for visual scoring and those for FFT analysis were thus identical. Each 2,048 data points (4.096 s) was tapered by Hamming window. The eight FFT values for each of eight sequences were summed to represent power value for the epoch. Details of this operation were described by Uchida et al. (36). Epochs containing artifacts were visually identified and excluded from the analysis. For statistical analysis, the total delta power of all NREM sleep EEG was calculated for each night.

Standard clinical criteria for scoring respiratory events (3) were applied to quantify the respiratory disturbance rate and episodes of periodic breathing. Apnea was defined as a pause in airflow of > 10 s accompanied by SaO₂ desaturations of > 4%. Hypopnea was defined as a 50% reduction in the amplitude of respiratory movements and airflow, if accompanied by SaO₂ desaturations of > 4%. An apnea-hypopnea index (AHI) was calculated as the rate of respiratory events from the combined number of apnea and hypopnea episodes per hour.

For SaO₂ data, mean and minimum values of whole night, time spent in SaO₂ below 90% (< 90% SaO₂), and oxygen desaturation index were calculated. The oxygen desaturation index was expressed as the rate of SaO₂ desaturation events (> 4%) per hour.

Subjective sleepiness. Subjective sleepiness was monitored using the Stanford Sleepiness Scale (14) and a visual analog scale. The visual analog scale was presented with a 100-mm straight line without any tic marks. The word “very sleepy” (in Japanese; representing 0) was presented on the left edge of the scale, and the word “very clear” (in Japanese; representing 100) was presented on the other edge of the scale. The subjects were instructed to write a tick mark on the scale to show their feelings. For the Stanford Sleepiness Scale and the visual analog scale, each subject filled in the forms immediately after waking.

Catecholamine measurements. The nocturnal urinary catecholamine excretion was measured. Urine samples were collected during total sleep time (from 2300 to 0700, kept acidic; pH < 3) and stored at −80° centigrade. After hydrolysis and column extraction (PBA and PSA columns, VAC ELUTE, Varian, Palo Alto, CA), the total catecholamine concentrations were measured by high-performance liquid chromatography with an electrochemical detector (model 5600A, CouArray System, ESA, Biosciences, Chelmsford, MA).

Statistical analysis. Data are expressed as means ± SD. Statistical comparisons between normal and hypoxic conditions were conducted using paired t-tests.
Table 1. HR and SaO2, for NC and HC

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<th>NC</th>
<th>HC</th>
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<tbody>
<tr>
<td><strong>Mean SaO2, %</strong></td>
<td>95.4±1.68</td>
<td>89.6±1.85*</td>
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<tr>
<td><strong>Minimum SaO2, %</strong></td>
<td>90.7±3.64</td>
<td>80.8±3.65*</td>
</tr>
<tr>
<td>&lt;90% SaO2, min</td>
<td>0.23±0.49</td>
<td>270.7±209.52*</td>
</tr>
<tr>
<td>% total sleep time</td>
<td>0.04±0.10</td>
<td>58.40±45.20*</td>
</tr>
<tr>
<td>O2 desaturation index</td>
<td>1.09±0.63</td>
<td>7.02±2.65*</td>
</tr>
<tr>
<td>Mean HR beats/min</td>
<td>51.3±4.47</td>
<td>55.6±4.96*</td>
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Values are means ± SD. NC, normoxic conditions; HC, hypoxic condition; HR, heart rate; SaO2, arterial blood oxygen saturation. *Significantly different from NC, P < 0.01.

RESULTS

SaO2 and heart rate. Data from SaO2 and heart rate (HR) measures for both the NC and HC conditions are shown in Table 1. Mean and minimum SaO2 showed a substantial significant decrease for HC compared with NC. Time spent in SaO2 below 90% (<90% SaO2) and oxygen desaturation index increased for HC compared with NC. Mean HR increased modestly but significantly for HC compared with NC.

Visual sleep stage scoring. Visually scored sleep stage parameters for both conditions are shown in Table 2. The duration of sleep stage 3 and 4 (slow wave) across the night was significantly lower for HC (69.7 ± 26.8 min) compared with NC (86.1 ± 22.3 min). Other sleep parameters did not show any statistical differences.

Changes in delta power of NREM sleep EEG. Figure 2 shows an example of delta power changes in NREM sleep EEG for one representative subject. A comparison of Fig. 2, A and B, demonstrates the decline in delta power of NREM sleep EEG for HC. Figure 3 presents the HC-related significant decrease in whole night delta power of all NREM sleep EEG for eight subjects (1,282,424 ± 705,289 V2/Hz for NC and 1,099,809 ± 605,816 V2/Hz for HC; P < 0.01).

Apnea, hypopnea, and periodic breathing. The changes in AHI across conditions are shown in Fig. 4. Respiratory disturbances reflected by the AHI were slightly but significantly greater for HC (2.7 ± 1.9 events/h) than for NC (1.1 ± 0.6 events/h). Severity of the AHI in HC varied widely among subjects, although subjects who showed higher AHIs for NC also tended to show higher AHIs for HC. Respiratory disturbances for HC were apparently central in origin, with reductions in effort indicated by cessations of both abdominal and rib cage movements.

Figure 5 shows an example of sleep stage, respiratory disturbances, and SaO2, under each condition for one representative subject. Mean SaO2 values for the subject were 94.7 and 87.5% for NC and HC, respectively. Times spent in SaO2 below 90% for this subject were 0.3 and 92.2% total sleep time for NC and HC, respectively. Respiratory disturbances increased for HC (AHI: 6.0) compared with NC (AHI: 1.9). No central apnea or hypopnea was observed for NC, whereas many respiratory disturbances were central in origin for HC.

![Fig. 2](image2.png)

![Fig. 3](image3.png)
Table 3 shows the numbers of episodes of respiratory disturbances for NC and HC. Statistical analysis revealed that HC increased respiratory disturbances, which were central in origin.

Subjective sleepiness. Neither the Stanford Sleepiness Scale nor the visual analog scale completed by subjects showed significant differences between NC and HC conditions (Stanford Sleepiness Scale 4.8 ± 1.07 for NC and 4.3 ± 0.95 for HC; visual analog scale 37.4 ± 16.4 for NC and 44.2 ± 16.8 for HC).

Catecholamines. Excretion of urinary norepinephrine was 56.3 ± 29.6 µg/8h for NC and 57.8 ± 22.4 µg/8h for HC. Excretion of urinary epinephrine was 2.29 ± 2.42 µg/8h for NC and 4.00 ± 3.74 µg/8h for HC. The differences in each parameter were not significant between conditions.

DISCUSSION

The present study investigated the effects of acute exposure to normobaric hypoxia equivalent to that at 2,000-m altitude on the sleep quality of athletes. There are two major findings in this study: 1) HC decreased slow-wave sleep (Table 2) and delta power, and 2) one of our subjects manifested typical periodic breathing under HC (Fig. 6). To the best of our knowledge, this is the first study to show that a hypoxic environment equivalent to that at 2,000-m altitude decreased slow-wave sleep and induced periodic breathing. This simulated altitude was well below the minimum at which these phenomena have been previously reported (18).

As described in the introduction, coaching staff have believed that athletes experience some degree of sleep disturbance in living-high conditions (32, 40). These feelings are consistent with and supported by the present findings of reduced total delta power in the all-night NREM sleep EEG and reduced minutes of visually scored stage 3 and 4 sleep. However, our results did not demonstrate any changes in arousal frequency, sleep efficiency, or stage W between conditions. Subjective levels of sleepiness did not change between the conditions either. While sleep disturbances were associated
with respiratory events, the mean AHI for HC remained below the common clinical criterion of 5 events/h (Fig. 4). In addition, although HR showed a modest but significant increase under HC, implying alteration in autonomic nervous control (Table 1), urinary catecholamines did not increase for HC. We consider these results to indicate that whatever stress was induced by HC remained subtle.

One of the factors underlying the decrease in slow-wave sleep may be related primarily to the degree of reduction in $\mathrm{SaO}_2$ of subjects under hypoxic conditions. As shown in Table 1, time spent in $\mathrm{SaO}_2$ below 90% ($<90\% \mathrm{SaO}_2$) increased to 58.4% and mean $\mathrm{SaO}_2$ decreased to 89.6% for HC. Although the 2,000-m altitude we simulated was one of the lowest among studies to date, the mean $\mathrm{SaO}_2$ in the present subjects was not much higher than that found at substantially higher altitudes. For an example, Pedlar et al. (26) reported that mean $\mathrm{SaO}_2$ was 89.9% at an altitude of 2,500 m. Hernandez-Zenteno et al. (12) reported that mean $\mathrm{SaO}_2$ was 93% at an altitude of 2,240 m. And Hugelin et al. (15) reported that low arterial oxygen pressure, induced by high-altitude environments, elicited cortical arousal via afferent chemoceptor activation of the ascending reticular activating system. Such mechanism could explain decreased slow-wave sleep in our subject under HC.

The lower than expected $\mathrm{SaO}_2$ in our subjects relative to prior studies could have a number of explanations. The first could be the lack of recent exposure to altitudes or hypoxic environments. Our subjects had lived in normoxic conditions for more than 5 mo before the study. Subjects in the studies of Kinsman et al. (16) and Pedlar et al. (26) had no such restrictions on previous exposure; if their subjects were recently exposed to hypoxic conditions before the studies, residual acclimatization could have affected their results. A second explanation may lie with the subject populations. Subjects in the studies of Zelinski et al. (42) and Hernandez-Zenteno et al. (12) were nonathletes, whereas ours were athletes. It is known that athletes have lower hypoxic and hypercapnic ventilatory responses, which are associated with exercise training (4). We did not test these responses because of technical limitations, nor did we examine nonathletes for comparison with athletes. However, these issues are definitely of interest and further study will be needed to clarify these points.

A difference in experimental designs may have also contributed to the differences in results between our and previous studies. While we recorded an adaptation night before sleep monitoring, Zelinski et al. (42) had not. If subjects are unaccustomed to the monitoring process, their sleep quality is likely to be impaired (1). Thus, in the earlier study, subjects were likely to have shown poor baseline sleep quality at low altitude. The poor quality of sleep at low altitude might have concealed changes in sleep quality attributable to the effects of altitude.

The intensity of daytime physical training could also have contributed to the different results between studies. Kinsman et al. (16) stated that a greater demand for slow-wave sleep, as demonstrated in athletes after daytime training, might overwhelm any fragmenting effects of moderate altitude (2,650 m). If a subject’s body temperature is elevated just before sleep, the warmer status might itself increase slow-wave sleep. Horne and Staff (13) demonstrated that high-intensity aerobic exercise or passive heating increased slow-wave sleep, but low-intensity aerobic exercise did not influence slow-wave sleep. Our subjects were not training or did only light jogging during the daytime. Therefore, we believe that subjects’ training did not affect sleep quality in this study.

In this study, the decrease in slow-wave sleep under HC was accompanied by an increase in subjects’ AHI (Fig. 4). Because many arousals coincided with hyperpnea under HC (Fig. 6) (19, 28), it could be hypothesized that stability of the breathing pattern was associated with a decreased frequency of arousal, which allowed better maintenance of deeper sleep (29). Weil’s review (38) stated: “observations at low altitude suggest that apnea-associated arousals correlate poorly with chemical variables but have a consistent relationship to mechanical stimuli, Table 3. Number of episodes of respiratory disturbances for NC and HC

<table>
<thead>
<tr>
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<th>NC</th>
<th>HC</th>
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<tr>
<td>Obstructive</td>
<td>5.1±4.1</td>
<td>4.0±3.2</td>
</tr>
<tr>
<td>Mixed</td>
<td>2.6±2.0</td>
<td>5.6±4.3</td>
</tr>
<tr>
<td>Central</td>
<td>0.3±0.5</td>
<td>12.0±9.1*</td>
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Values are means ± SD expressed as the number of episodes per 8 h. *Significantly different from NC, $P < 0.01$. 

Fig. 6. Respiratory pattern consistent with periodic breathing under HC. EEG1, C3/A2; EEG2, C4/A1.
suggesting linkage to the resumption of ventilatory effort rather than blood chemistry.” Not only hypothermia but also mechanical stimulation induced by hypopneas may have activated the arousal neural mechanism to induce the decrease in slow-wave sleep (28, 38).

The subject who had the highest AHI showed a respiratory pattern consistent with periodic breathing under HC (Fig. 6). Because AHI in HC varied widely between individuals (Fig. 4), individual adjustments of oxygen concentration could be a suitable and practical solution for the first night of sleep under hypoxia. Subjects who showed high AHIs under NC also tended to show high AHIs under HC (Fig. 4). Although the increased respiratory disturbances under NC tended to be obstructive or mixed, events under HC tended to be central in appearance (Table 3, Fig. 5). Although the causes of central and obstructive apneas/hypopneas are distinct, some researchers insist there is a common link to the system controlling ventilation (7, 22). However, the relationship between respiratory disturbances under hypoxia and normoxia has not yet been elucidated and further studies will be needed.

Although previous studies failed to show any decrease in slow-wave sleep at altitudes below 3,000 m, some practical means are frequently adopted to avoid accumulation of fatigue in living-high conditions, which might not stimulate slow-wave sleep changes above baseline levels. Some coaches and athletes control training intensity and volume during the daytime (32,40). Some coaches and athletes modify their living-high strategy to include recovery nights at low altitude (16) or with oxygen enrichment (17) between blocks of altitude exposure. These means may be effective at avoiding accumulation of fatigue when athletes sleep even at altitudes of 2,000 m.

The report of an American Academy of Sleep Medicine Task Force (3) said that use of thermostors was not thought to be suitable and practical. Although we detected hypopnea by a reduction in respiratory movements from piezoelectric bands and SaO2 desaturations in conjunction with a reduction in airflow from a thermostor, it may be more tolerant, subjective tolerate, subjective fatigue, and sleepiness. Appl Psychophysiol Biofeedback 23: 207–217, 1998.


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