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A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]



The Effect of Altitude Descent on Obstructive Sleep Apnea*

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Background: The present requirement for “at facility” polysomnograms requires many residents in mountain communities to descend in elevation for sleep testing, which may cause misleading results regarding the severity of obstructive sleep apnea (OSA).

Design: Eleven patients with previously undiagnosed sleep apnea living at an altitude > 2,400 m (7,900 feet) in Colorado underwent diagnostic sleep studies at their home elevation and at 1,370 m (4,500 feet), and 5 of the 11 patients were also studied at sea level.

Results: The mean (SE) apnea-hypopnea index (AHI) fell from 49.1 (10.5)/h to 37.0 (11.2)/h on descent to 1,370 m ($p = 0.022$). In the five patients who traveled to sea level, the AHI dropped from 53.8 (13.2)/h at home elevation to 47.1 (14.8)/h at 1,370 m, and to 33.1 (12.6)/h at sea level ($p = 0.018$). The reduction in AHI was predominantly a reduction in hypopneas and central apneas, with little change in the frequency of obstructive apneas. Duration of the obstructive apneas lengthened with descent. Of eight patients with an AHI < 50/h at their home elevation, two patients had their AHI fall to < 5/h at 1,370 m, and a third patient dropped to < 5/h at sea level, *ie*, below many physicians’ threshold for providing therapy. Patients with the most severe OSA had the least improvement with descent.

Conclusions: Because AHI decreases significantly with descent in altitude, polysomnography is most accurately done at the home elevation of the patient. Descent to a sleep laboratory at a lower elevation may yield false-negative results in patients with mild or moderate sleep apnea. (CHEST 2006; 130:1744–1750)

Key words: altitude; evaluation; obstructive sleep apnea

Abbreviations: AHI = apnea-hypopnea index; non-REM = non-rapid eye movement; OSA = obstructive sleep apnea; RDI = respiratory disturbance index; RERA = respiratory effort-related arousal; SaO₂ = arterial oxygen saturation

In mountainous areas such as Colorado, the nearest sleep laboratory for a resident may often be in an urban center at a substantially lower elevation. Presently, patients who live 1,000 to 1,500 m in elevation above sleep laboratories in Grand Junction, CO, at 1,370 m (4,500 feet), or Denver, CO, at 1,585 m (5,200 feet) may need to utilize services in these cities because Medicare and most insurance companies will not authorize “at-home” polysomnography. Three atmospheric variables change with descent: (1) air density and thus viscosity through a critically narrowed upper airway, (2) oxygen content, and (3) barometric pressure or external compressive effect. Changes in these variables with descent raise the concern

that we may be misestimating the severity of sleep apnea and depriving some patients of necessary therapy if they descend to a sleep laboratory for evaluation.

In this study, we evaluated patients with symptoms of obstructive sleep apnea (OSA) residing at high elevation and assessed the change in sleep apnea severity during polysomnography, comparing home elevation findings with sleep study results at one or two lower elevations. Our goal was to understand the consequences of bringing these patients down from their home elevation to metropolitan centers in Colorado, or even further down to tertiary referral centers near sea level for sleep evaluation.

MATERIALS AND METHODS

Study Design and Enrollment

Eleven individuals with suspected sleep apnea living at an altitude > 2,400 m in western Colorado underwent attended full-night diagnostic polysomnography at two elevations, one in the home and one in a clinical sleep laboratory in Grand Junction at 1,370 m. Five of the 11 patients also underwent a third polysomnogram in a clinical sleep laboratory at sea level. Each patient was referred from a primary practitioner who was aware of the study protocol and was concerned about the possibility of OSA. All patients provided informed consent for the protocol, which was approved by the St. Mary's Hospital Investigational Review Board. The Grand Junction studies and the sea level studies were performed in clinical sleep laboratories. Alice IV sleep recording equipment (Respironics; Murrysville, PA) was used in 25 of 27 studies; the two other studies utilized Sandman (Pleasanton, CA) and Grass-Telefactor (West Warwick, RI) equipment. The median time between the two Colorado studies was 3 days (range, 2 to 34 days). Six of the 11 patients underwent the home study first, with the other 5 patients being first studied in Grand Junction. In all five of the sea level travelers, the sea level study was the third study. It followed the last Colorado study by a median of 7 days (range, 4 to 61 days).

Polysomnography

Sleep was recorded with four EEG leads, chin electromyogram, and bilateral electrooculogram. Bilateral leg electromyogram position and ECG were also recorded. Respiration was monitored with chest and abdominal belts with piezo strain gauges, pulse oximetry, snoring, and nasal and oral airflow monitors in each patient. In 22 of the 27 studies, the patients had nasal/oral thermistors; but in 5 of the Colorado studies, the thermistor was only an oral thermistor, recording in parallel with the nasal pressure airflow transducer. In all the Colorado studies, a nasal pressure airflow transducer (Pro-Tech; Mukilteo, WA) was used. This was available in only two of the five sea level studies.

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Dr. D. Patz directs and owns a private sleep laboratory in Grand Junction, Western Colorado Sleep Institute, which financed the research study. Dr. White receives income from Respironics (Murrysville, PA) for being Chief Medical Officer, but this had nothing to do with the choice of sleep recording systems in this study. Mobile Sleep Diagnostics owned Alice IV (Respironics) recording equipment for years prior to the conception of this research study. The study was also completed before Dr. White began his employment with Respironics. Dr. White is also a consultant for AspireMedical, WideMed, PAVAD, and Itamar Medical. The authors have no conflicts of interest to report. Manuscript received May 25, 2006; revision accepted June 27, 2006.

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Sleep was staged according to Rechtschaffen and Kales.¹ Apnea required 10-s duration, a flat nasal pressure signal (when available), and at least 80% reduction in the thermistor signal. Hypopneas required a detectable reduction in either a flow signal or respiratory belt for 10 s, associated with 4% oxygen desaturation. The 4% desaturation criteria for hypopneas was utilized because Medicare and the largest private insurance provider in western Colorado require this definition to decide who qualifies for insurance payment for OSA therapy. Respiratory effort-related arousals (RERAs) were defined as snoring periods or hypopneas lasting ≥ 10 s, with < 4% oxygen desaturation but leading to EEG arousal. The apnea-hypopnea index (AHI) was defined as the number of apneas plus hypopneas per hour of sleep. The respiratory disturbance index (RDI) was defined as the number of apneas plus hypopneas plus RERAs per hour of sleep.

The 25 studies using Alice IV equipment were scored by a research sleep technician in Boston who was blinded to the study design. They were edited by an American Board of Sleep Medicine diplomat who also was blinded to their identity. The studies using Grass-Telefactor and Sandman equipment were not accessible to blinding.

Statistical Analysis

Each variable was analyzed using a repeated-measure analysis of variance (SAS PROC MIXED; SAS Institute; Cary, NC), with CONTRAST statements facilitating pairwise comparisons in the *post hoc* analysis. The data were analyzed in two ways. All 11 patients were analyzed at two elevations, and the 5 patients who traveled to sea level were analyzed at three elevations. Data are expressed as mean value (SE). Demographics are expressed as mean ± 1 SD.

RESULTS

Table 1 summarizes the individual patient data, and Table 2 summarizes how the respiratory indexes (mean [SE]) changed with altitude descent. The patients were 46 to 70 years old (mean 55 ± 7.3 years). Three of the 11 patients were women. Body mass index was 20 to 58 kg/m² (mean, 33.9 ± 11.3 kg/m²). The home elevations ranged from 2,417 to 3,139 m (7,930 to 10,300 feet) [mean, $2,701 \pm 214$ m]. Home elevation severity of sleep apnea in these patients ranged from mild to severe, with AHIs from 13.5 to 125.6/h. In these 11 patients, there was a trend of reduction in AHI with descent, with AHI of 49.1 (10.5)/h at home elevations > 2,400 m, and an AHI of 37.0 (11.2)/h in Grand Junction at 1,370 m ($p = 0.022$). In the 5 of 11 patients who also traveled to sea level, AHI decreased from 53.8 (13.2)/h above 2,400 m to 47.1 (14.8)/h at 1,370 m, and 33.1 (12.6)/h at sea level ($p = 0.018$) [Fig 1, *left*].

Of the eight patients with a home elevation AHI from 13.5 to 50/h, two patients had their AHI drop to < 5/h coming to Grand Junction (1,370 m), and a third patient dropped his AHI to < 5/h at sea level (*ie*, below many physicians' threshold for providing therapy). Adding the RERAs, the RDI followed a similar trend of reduction with descent (Fig 1, *right*).

Table 1—Respiratory Indices vs Altitude in 11 Individuals*

| Patient No. | Age, yr/ Gender/Body Mass Index, kg/m ² | Elevation, m | Mean AHI | RDI | OA-MA Index | Duration of OA-MA, s | OA, No./MA, No. | CA Index | Hypopnea Index | RERA Index | Lowest SaO ₂ , % |
|-------------|--|-----------------|-------------|-------|----------------|-------------------------|--------------------|-------------|-------------------|---------------|-----------------------------------|
| 1 | 62/M/35 | 2,423 | 90.7 | 90.7 | 81.3 | 22 | 407/185 | 5.7 | 3.6 | 0 | 61 |
| | | 1,370 | 76.1 | 76.1 | 70.2 | 27.4 | 498/38 | 1.2 | 4.7 | 0 | 60 |
| | | Sea level | 55.4 | 55.4 | 51.7 | 35.2 | 366/0 | 0 | 3.7 | 0 | 62 |
| 2 | 46/M/33 | 2,643 | 76.6 | 76.6 | 59.6 | 24.5 | 453/1 | 0.3 | 16.6 | 0 | 60 |
| | | 1,370 | 83.8 | 83.8 | 72.4 | 28.8 | 483/19 | 1.3 | 10.1 | 0 | 58 |
| | | Sea level | 69.6 | 69.6 | 67.2 | 34.4 | 412/41 | 0.3 | 2.1 | 0 | 53 |
| 3 | 54/M/24 | 2,707 | 17.5 | 20.6 | 3.4 | 22.7 | 22/2 | 0.4 | 13.6 | 3.1 | 81 |
| | | 1,370 | 13.9 | 19.5 | 8.6 | 23.4 | 62/3 | 0.1 | 5.2 | 5.6 | 78 |
| | | Sea level | 12.8 | 18.9 | 5 | 28.6 | 24/5 | 0.2 | 7.5 | 6.1 | 80 |
| 4 | 70/M/26 | 2,713 | 39.7 | 58.5 | 1.8 | 15 | 9/1 | 17.7 | 20.2 | 17.2 | 85 |
| | | 1,370 | 47.8 | 67.5 | 19.3 | 20.2 | 95/22 | 19.5 | 8.9 | 19.4 | 87 |
| | | Sea level | 23.6 | 40.9 | 16.8 | 21.9 | 57/1 | 4.4 | 2.3 | 17.1 | 88 |
| 5 | 48/M/41 | 3,139 | 44.4 | 45.5 | 4.5 | | 33/0 | 1.1 | 38.8 | 1.2 | 69 |
| | | 1,370 | 13.9 | 18.3 | 0 | | 0/0 | 0 | 13.9 | 3.9 | 83 |
| | | Sea level | 4.1 | 8.2 | 0.5 | | 3/0 | 0.3 | 3.3 | 4.1 | 89 |
| 6 | 55/F/34 | 2,621 | 31.6 | 34 | 5.1 | 12.6 | 37/2 | 4.5 | 22.1 | 2.3 | 61 |
| | | 1,370 | 24.1 | 28.1 | 8.5 | 18 | 60/2 | 1.1 | 14.5 | 4.1 | 64 |
| | | Sea level | | | | | | | | | |
| 7 | 46/F/46 | 2,815 | 125.6 | 125.6 | 95.7 | 18.8 | 117/587 | 24.7 | 5.2 | 0 | 63 |
| | | 1,370 | 110.9 | 111.2 | 95.3 | 21.1 | 358/11 | 1.3 | 14.3 | 0.3 | 69 |
| | | Sea level | | | | | | | | | |
| 8 | 50/F/58 | 2,417 | 24.6 | 31.6 | 0.6 | | 1/0 | 1.2 | 22.8 | 7 | 64 |
| | | 1,370 | 18.8 | 25.8 | 4.4 | | 25/0 | 0.9 | 13.6 | 7 | 62 |
| | | Sea level | | | | | | | | | |
| 9 | 58/M/22 | 2,957 | 49.5 | 58.4 | 0 | | 0/0 | 35.6 | 13.9 | 9.3 | 86 |
| | | 1,370 | 4.2 | 30.2 | 0 | | 0/0 | 1.7 | 2.4 | 26 | 90 |
| | | Sea level | | | | | | | | | |
| 10 | 59/M/35 | 2,560 | 26 | 33.4 | 1.1 | | 7/0 | 14.7 | 10.2 | 7.4 | 86 |
| | | 1,370 | 9.7 | 16.6 | 6.7 | | 37/3 | 1.3 | 1.8 | 6.7 | 89 |
| | | Sea level | | | | | | | | | |
| 11 | 55/F/20 | 2,713 | 13.8 | 18.9 | 0.2 | | 1/0 | 0 | 13.5 | 5.2 | 81 |
| | | 1,370 | 3.8 | 12.4 | 0.4 | | 2/0 | 0 | 3.4 | 8.7 | 87 |
| | | Sea level | | | | | | | | | |

*M = male; F = female; OA = obstructive apnea; MA = mixed apnea; CA = central apnea.

The values for the indexes for individual respiratory event types as altitude changed from home elevation to Grand Junction in all 11 patients are shown in Figure 2, *left*. The obstructive apneas and mixed apneas are grouped together and did not decrease. The central apneas decreased by 70% ($p = 0.06$), and the hypopneas decreased by 49% ($p = 0.008$). Focusing exclusively on the five patients who also traveled to sea level (Fig 2, *right*), we see similar trends. In both groups, there was no remarkable change in RERAs.

The duration of obstructive and mixed apneas was

longer at lower elevation. Six of the 11 patients had at least 10 obstructive or mixed apneas at at least two elevations. The average duration of these events was longer, at lower elevation, in each of these six patients ($p < 0.0001$) [Fig 3]. Thus, the percentage of total sleep time that these patients were apneic with obstructive apnea or mixed apnea was approximately the same or greater at lower elevation.

The average non-rapid eye movement (non-REM) arterial oxygen saturation (SaO₂) consistently rose with decreasing elevation, as expected ($p = 0.002$) [Fig 4, *left*]. However, the trend of the single lowest

Table 2—Summary of Results: Respiratory Parameters vs Altitude*

| Parameters | Subset | Altitude | | | p Value |
|-------------------------------------|------------|-------------|-------------|-------------|----------|
| | | > 2,400 m | 1,370 m | Sea Level | |
| AHI | All 11 pts | 49.1 (10.5) | 37.0 (11.2) | | 0.022 |
| AHI | 5 of 11 | 53.8 (13.2) | 47.1 (14.8) | 33.1 (12.6) | 0.018 |
| RDI | All 11 pts | 54.0 (9.9) | 44.5 (10.2) | | 0.023 |
| RDI | 5 of 11 | 58.4 (12.2) | 53.0 (14.4) | 38.4 (11.4) | 0.02 |
| OA-MA index | All 11 pts | 23.0 (11.1) | 26.0 (10.6) | | 0.21 |
| OA-MA index | 5 of 11 | 30.1 (16.8) | 34.1 (15.5) | 28.2 (13.2) | 0.53 |
| Obstructive apnea index | All 11 pts | 13.3 (6.7) | 24.6 (10.2) | | 0.11 |
| Obstructive apnea index | 5 of 11 | 24.8 (13.3) | 31.9 (14.8) | 26.8 (12.5) | 0.09 |
| Mixed apnea index | All 11 pts | 9.8 (7.5) | 1.5 (0.5) | | 0.26 |
| Mixed apnea index | 5 of 11 | 5.3 (5.2) | 2.3 (1.0) | 1.4 (1.2) | 0.59 |
| Central apnea index | All 11 pts | 9.1 (2.7) | 2.7 (1.0) | | 0.061 |
| Central apnea index | 5 of 11 | 5.0 (3.2) | 4.4 (3.8) | 1.0 (0.84) | 0.18 |
| Hypopnea index | All 11 pts | 16.4 (2.9) | 8.4 (1.5) | | 0.008 |
| Hypopnea index | 5 of 11 | 18.6 (5.8) | 8.5 (1.7) | 3.8 (3.1) | 0.017 |
| RERA index | All 11 pts | 4.9 (1.7) | 7.5 (2.5) | | 0.095 |
| RERA index | 5 of 11 | 4.6 (3.6) | 5.9 (3.6) | 5.3 (3.2) | 0.15 |
| OA-MA duration, s | 6 of 11 | 19.3 (1.9) | 23.2 (1.7) | 30.0 (3.1) | < 0.0001 |
| SaO ₂ non-REM average, % | All 11 pts | 86.9 (2.6) | 90.1 (1.3) | | 0.0002 |
| SaO ₂ non-REM average, % | 5 of 11 | 86.6 (2.6) | 89.6 (2.2) | 91.5 (2.4) | 0.0022 |
| Lowest SaO ₂ nadir | All 11 pts | 72.5 (5.1) | 75.2 (3.8) | | 0.083 |
| Lowest SaO ₂ nadir | 5 of 11 | 71.2 (5.1) | 73.2 (6.0) | 74.4 (7.2) | 0.58 |

*Measures are expressed as mean (SE). pts = patients; see Table 1 for expansion of abbreviations.

SaO₂ nadir of the night for the patients did not invariably increase with descent ($p = 0.099$) [Fig 4, right], perhaps due to some of the longer events at lower elevation.

Our three patients with the most severe OSA, patients 1, 2, and 7, who had predominantly obstructive/mixed apneas at all elevations studied, had the least improvement with descent from > 2,400 to 1,370 m. Their average AHI dropped only from 97.6 to 90.4/h. Their average time apneic with obstructive/mixed apneas increased from 47 to 56%, and only one of these three patients had an increase in SaO₂ nadir with descent.

DISCUSSION

The AHI decreased with descent to lower elevations, with the reduction being primarily secondary to fewer hypopneas and central apneas. This finding suggests that the increased oxygen content in the air at lower elevation is the more important of the three

atmospheric factors affecting respiratory events with elevation change, within the altitude range studied.

Our findings parallel the findings of other investigators who noted AHI decreased with supplemental oxygen therapy. Smith et al² found that nocturnal oxygen reduced the number of central apneas more than obstructive apneas in patients with primarily obstructive sleep apnea, but obstructive apneas did decrease. Gold et al³ found that in patients with a greater mixture of central and obstructive apneas, the AHI decreased with oxygen but the number of obstructive apneas actually increased.

Five of our patients (patients 4, 6, 7, 8, and 10) had a mixture of obstructive apneas and central apneas, with > 25% of the home elevation apneas being central. In this group, each had a reduction in central apneas ($p = 0.07$) and an increase in obstructive apneas with descent ($p = 0.007$). The pattern followed the changes described by Gold et al,³ when his patients with a mixture of central and obstructive apneas were administered oxygen.

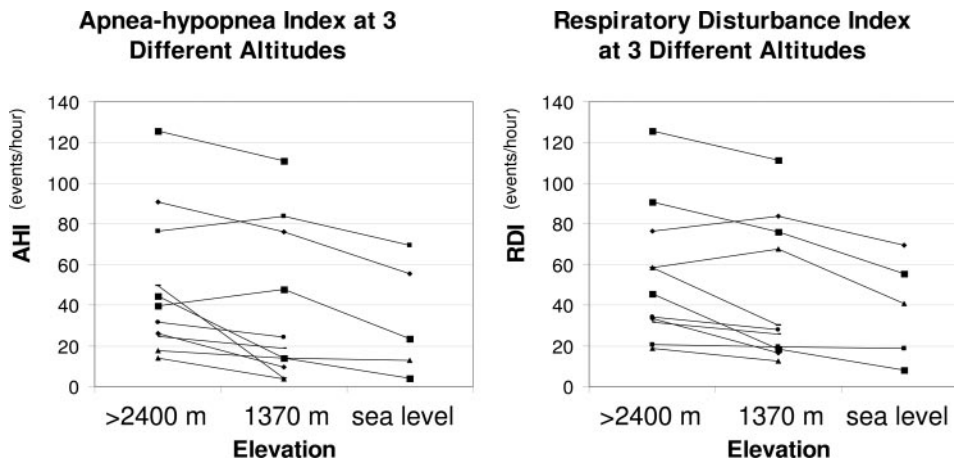


FIGURE 1. *Left*: Each line connects the two or three data points of one individual. The decrease in AHI with descent for all 11 patients descending from home elevation to 1,370 m in Grand Junction is significant ($p = 0.022$). For the five patients who also traveled to sea level, the decrease in AHI is also significant ($p = 0.018$). *Right*: RDI (including apneas, hypopneas, and RERAs) vs altitude in 11 patients residing at $> 2,400$ m similarly decreases with descent ($p = 0.023$ for all 11 patients descending to 1,370 m; $p = 0.020$ for 5 of the 11 patients who also traveled to sea level).

The reduction in hypopneas with descent in our patients may be in part due to another phenomenon described by Gold et al.³ With oxygen administration, they found not only an increase in the baseline SaO_2 but also a reduction in the magnitude of the SaO_2 drop with each respiratory event.³ This effect becomes quite significant when a 4% SaO_2 drop is part of the definition of hypopnea. Patient 5, who had primarily snoring and hypopneas, had his AHI fall from 44.4/h in Breckenridge at 3,139 m, to 13.9/h

in Grand Junction at 1,370 m, and to 4.1/h near sea level. However his RERA index only increased from 1.2/h at 3,139 m to 4.1/h traveling to sea level.

Central apneas may be a manifestation of instability in ventilatory control.⁴ Wellman et al⁵ found that the OSA patients with the most instability in their ventilatory feedback loop, measured with the proportional assist ventilation technique, had the greatest decrease in their AHI in response to oxygen. Our patient with the greatest reduction in his AHI with

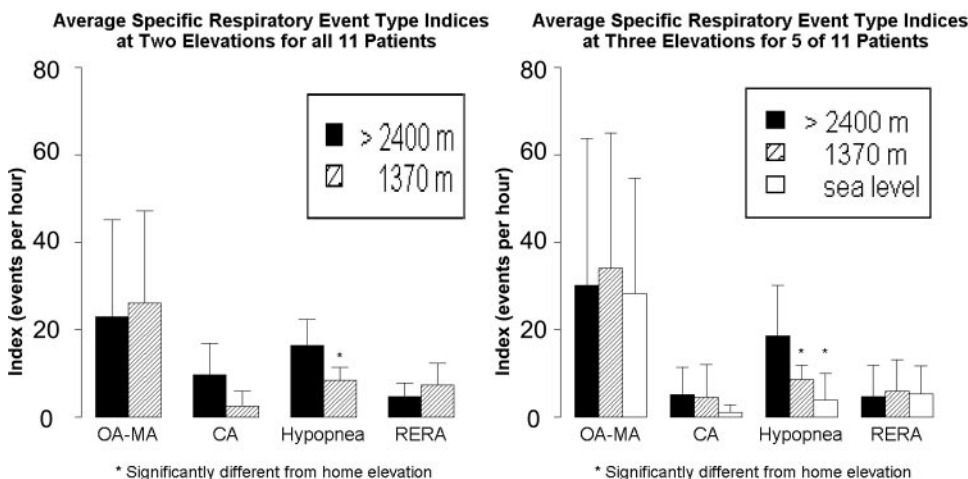


FIGURE 2. *Left*: Average specific respiratory event-type indexes are displayed for all 11 patients at two elevations, 2,400 m and 1,370 m. Altitude descent did not effect the obstructive/mixed apnea (OA-MA) index or the RERA index. The central apnea (CA) index decreased but was not statistically significant ($p = 0.061$). The hypopnea index decreased significantly ($p = 0.008$). *Right*: Average specific respiratory event-type indexes are displayed for the five patients who also traveled to sea level. Altitude descent did not change the obstructive/mixed apnea index or the RERA index. There appeared to be reduction in the central apneas at sea level, but this was not statistically significant ($p = 0.18$). There was significant reduction in hypopneas ($p = 0.017$).

Average Duration of Obstructive & Mixed Apneas vs Elevation

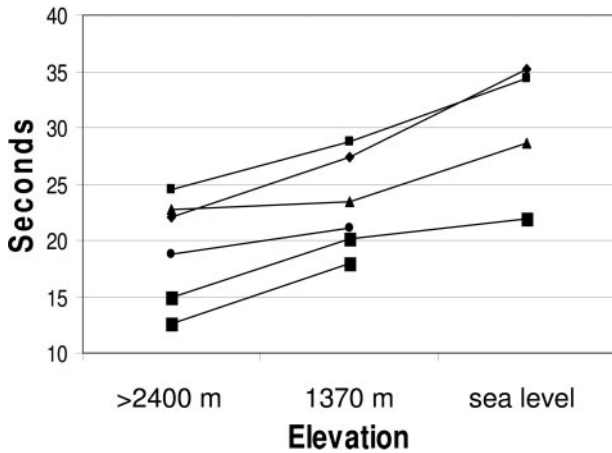


FIGURE 3. Duration of obstructive and mixed apneas with altitude descent increased in all 6 of the 11 patients who had ≥ 10 such apneas in two or more elevations ($p < 0.0001$).

descent (patient 9) had exclusively central apneas at his home elevation, perhaps a reflection of his increased loop gain. The AHI decreased from 49.5 to 4.2/h descending from 2,957 to 1,370 m. At 1,370 m, he had predominantly snoring, hypopneas, and RERAs.

The increased duration of the obstructive apneas and mixed apneas at lower elevation may also be an effect of oxygen. The falling PO_2 , as well as the rising PCO_2 , provides stimulus for arousal during a respiratory event.⁶ At lower altitude, the threshold SaO_2 to trigger arousal may take longer to be reached,

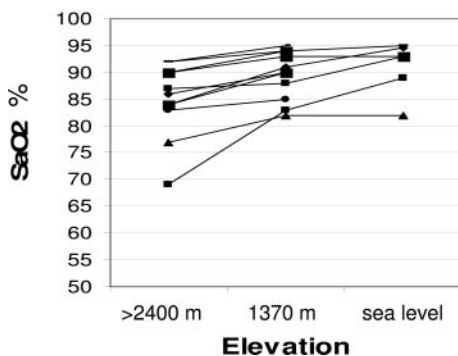
leading to a longer apnea. Motta and Guilleminault⁷ noted longer duration of sleep-induced apneas in response to oxygen.

Several studies have addressed the effect of increasing altitude on OSA. Netzer et al⁸ and Zielinski et al⁹ noted an increase in obstructive events during sleep, as a snoring mountain climber⁸ and as nine healthy volunteers⁹ ascended $> 3,000$ m. Burgess et al,¹⁰ however, observed the elimination of all obstructive apneas as an Australian climber, who had moderate OSA at sea level, reached 3,446 m and had prominent periodic breathing with exclusively central apneas.

Burgess et al¹¹ then studied five patients with known moderate OSA at sea level, at 610 m, and at “2,750 m” simulated with isobaric hypoxia. At the simulated 2,750 m, all five OSA patients had exclusively central apnea with periodic breathing. The obstructive RDI fell from $25.5 \pm 14.4/h$ at sea level to $0.5 \pm 0.7/h$ at simulated 2,750 m, with the central RDI increasing from 0.4 ± 0.5 to $78.8 \pm 29.7/h$.

Our findings are not incompatible with those of Burgess et al.¹¹ Our patient 4 had largely central apneas at altitude and obstructive apneas at sea level. Four other patients with a mixture of central and obstructive events at their home elevation had primarily obstructive events with descent. However, in our patients, obstructive events did not totally disappear at their residences $> 2,400$ m. This may be due to our patients being acclimated to their home elevation, while the patients of Burgess et al¹¹ spent just 1 night at the simulated 2,750 m. With acclimatization, periodic breathing and central apneas might decrease allowing obstructive events to appear. Several authors^{12,13} have found, below 4,400 m, periodic breathing decreases in some subjects with acclima-

Average non-REM SaO2 vs Elevation



SaO2 Nadir vs Elevation

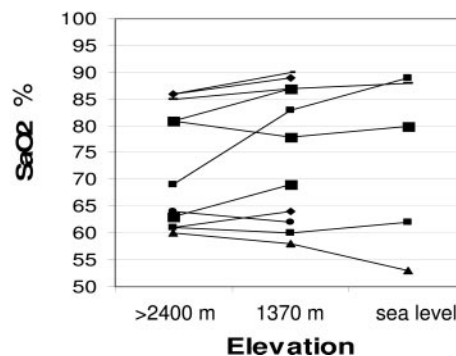


FIGURE 4. *Left:* Average non-REM SaO_2 in each of the 11 patients rises with altitude descent ($p < 0.002$). *Right:* The single lowest SaO_2 nadir of the night less consistently rises with descent in the 11 patients ($p =$ not significant).

tization. However, Salvaggio et al¹⁴ noted an increase in periodic breathing during 1 month at 5,050 m.

Warner et al¹⁵ also exposed snorers and OSA patients to severe hypoxia during non-REM sleep. Most patients had exclusively central apneas with severe hypoxia. However one patient with high baseline upper airway resistance and OSA in a normoxic setting had a pattern of mixed apneas with hypoxia. This may be analogous to our patient 7, whose obstructive/mixed apnea ratios were 358:11 at 1,370 m and 117:587 at 2,815 m. He had predominantly obstructive apneas at 1,370 m, but at his home elevation he had predominantly mixed apneas. The findings of Warner et al¹⁵ also parallel our findings that our three patients who maintained a predominance of obstructive and mixed apneas at their home elevation were the three patients who required ≥ 17 cm of continuous positive airway pressure, perhaps a reflection of very high upper airway resistance; all of our other patients required ≤ 13 cm of continuous positive airway pressure.

Limitations

In considering our findings several limitations must be considered. First, this was a small population of patients but is likely representative of patients living at altitude. Second, there was some inconsistency in monitoring equipment. In five patients, the thermistor was exclusively oral in one of their Colorado studies, compared to nasal/oral thermistors in all of the other studies. However, in all of these five studies a nasal pressure cannula was also used. Thus, this likely had minimal impact. The oral thermistor was used in two patients at home elevation and three patients at 1,370 m, thus not inducing a bias. Third, there were also three studies done at sea level without the nasal pressure cannula. This may have led to an underscoring of RERAs, thus underestimating the RDI at sea level. However, this would not have affected the AHI since there were few 4% oxygen desaturations that occurred at sea level without some recognizable reduction in thermistor signal or belts. Fourth, 2 of the 27 studies could not be reviewed blinded as to the site of study. However, if we delete those two studies, the decrease of AHI, RDI, and hypopnea index with descent remains significant ($p < 0.05$ for each). A fifth limitation is the lack of physiologic studies to help characterize the individual patients regarding their hypoxic ventilatory responses and critical closing pressures. These measurements, in retrospect, may have helped explain why different patients responded differently to altitude change.

CONCLUSION

For patients residing between 2,400 m and 3,139 m, the AHI and RDI decrease with descent. All of the changes we observed regarding the effect of altitude descent paralleled previous observations on the effect of oxygen on OSA patients at sea level. Central apneas and hypopneas decreased in number, and obstructive apneas lengthened. It is important to appreciate that some patients with mild or moderate sleep apnea traveling from their home elevation in the mountains to lower urban sleep centers for sleep studies may fail to qualify for sleep apnea therapy. It therefore appears important for patients to undergo polysomnography at their home elevation if accurate diagnostic and therapeutic decisions are to be made.

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