NEW RESEARCH

JCSM
Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Do Weather-Related Ambient Atmospheric-Pressure Changes Influence Sleep Disordered Breathing?

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Objective: High-altitude studies of sleep disordered breathing (SDB) show increases in apnea hypopnea indices with elevation gains. Hypoxic changes, rather than reductions in atmospheric pressure (AP), are thought to be the driving factor. Ambient pressure-related changes in SDB have not been extensively studied at low altitude. We performed a cross-sectional study of weather-related AP effects on measures of SDB at the University of Washington Medicine Sleep Institute, a Seattle, Washington-based polysomnography lab located 200 feet above sea level.

Method: Obstructive, central, and apnea-hypopnea indices from 537 patients were retrospectively correlated to mean 8-hour date-matched overnight AP data. Linear regression analysis and interquartile comparison of AP-related respiratory indices were performed and adjusted for age, sex, and body mass index.

Results: The obstructive apnea index increased with lower weather-related APs (p = 0.01 for linear trend), interquartile analysis showed significant worsening with lowered mean, minimum, and maximum nightly APs. Similar changes were not seen with central or apnea-hypopnea indices.

Conclusions: The obstructive apnea index is altered by changes in weather-related AP during diagnostic polysomnography performed at 200 feet above sea level. Small changes in ambient atmospheric pressure due to weather systems may be important in the pathophysiology and diagnosis of obstructive sleep apnea.

Keywords: Barometric, apnea, sleep

Citation: Doherty MJ; Youn CE; Haltiner AM; Watson NF. Do weather-related ambient atmospheric-pressure changes influence sleep disordered breathing? *J Clin Sleep Med* 2010;6(2):152-156.

Recently, we showed a small increase in the odds of seizure occurring in patients with epilepsy undergoing diagnostic video-electroencephalographic telemetry when moderate changes in atmospheric pressure (AP) occurred in the 24 hours preceding the event.[1] Speculative mechanisms include possible epileptogenic changes in respiratory patterns due to AP shifts. One of those mechanisms may be through a low-level hyperventilatory effect, the study of which would require prospective data.[2] A second mechanism, a lowering of seizure threshold resultant from atmosphere-influenced sleep disordered breathing (SDB), was proposed.[3]

AP changes are studied in limited case series of altitude-based models of SDB.[4-6] In these models, apnea hypopnea indices (AHI) rise in concert with elevation gains, either real or simulated by decreases in barometric pressure. These changes are likely due to hypoxia rather than lower AP and are primarily driven by central apneas and hypopneas rather than obstructive events.[6,7] The impact of low-altitude, ambient, weather-related changes in AP on respiration during sleep, however, has been largely unstudied. We performed a cross-sectional, retrospective study of the association between near sea-level, weather-related AP changes and indicators of SDB in patients who underwent diagnostic evaluations for sleep disorders at the University of Washington (UW) Medicine Sleep Institute, a Seattle, Washington, polysomnography laboratory. The goal was to investigate a possible association between weather-related changes in AP and the obstructive apnea index (OAI), central apnea index (CAI), and overall AHI.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Prior studies of atmospheric pressure and sleep disordered breathing have concentrated on altitude effects. Our study concentrated on ambient effects of atmospheric pressure on measurements of sleep disordered breathing.

Study Impact: Our study of 537 patients undergoing diagnostic polysomnography showed that with lower ambient atmospheric pressure close to sea level, measurements of obstructive sleep apnea such as the obstructive apnea index significantly increased, while the apnea hypopnea and the central apnea indices did not. Ambient atmospheric pressure changes from passing weather systems may influence the diagnosis of obstructive sleep apnea.

METHODS

Consecutive patients evaluated for sleep disorders at the UW Medicine Sleep Institute between January 1, 2004, and June 2, 2006, were eligible for inclusion in the study (n = 629). Those absent diagnostic polysomnography data or missing data were excluded from the study (excluded n = 92). Common reasons for exclusion included unavailable hypopnea counts, apnea counts, or total sleep times. Split-night studies were not examined.

AP data were obtained from a publicly available National Weather Service database containing automated, land-based, hourly measurements corresponding to downtown Seattle.[8] To control for differences in ward and National Weather Service pressures, 48 consecutive hourly AP measurements taken at the UW Medicine Sleep Institute with a digital barometer (Starpath,

Seattle, WA) were correlated with National Weather Service data ($p < 0.01$, Pearson correlate 0.991). The altitude difference between the automated National Weather Service weather station and the polysomnography laboratory is 200 feet. Correction in AP was not made for this altitude difference because it was assumed to be a constant.

Eight hours of overnight AP data collected from 21:00 to 07:00 was matched with concurrent diagnostic polysomnography data in consecutive patients undergoing polysomnography at the UW Medicine Sleep Institute. Individual patient AP data included nightly mean AP, as well as the maximum and minimum AP value registered during the night. Patients fell asleep and awoke at inconsistent times during polysomnography; a standard 8-hour range was chosen to ensure collection of atmospheric data for the hours when the majority of patients would typically be asleep during their polysomnogram.

Electroencephalographic electrodes were positioned at 2 frontal (F7, F8), 2 central (C3, C4), and 2 occipital (O1, O2) locations (International 10-20 system of measurement) and were referenced to the contralateral mastoids. Chin electromyogram and right and left electrooculogram electrodes were also applied. Airflow was measured using a nasal pressure cannula placed in the nose and a thermistor placed in the nose and over the mouth, allowing differentiation between nasal and oral breathing. Chest and abdominal respiratory effort were assessed by piezo respiratory-effort bands placed around the chest and abdomen, and snoring with a small microphone sensor placed on the throat just lateral to the trachea (Pro-Tech Services, Inc., Mukilteo, WA). Oxygen saturation was measured from the index finger via pulse oximetry (Nellcor, Pleasanton, CA). Bilateral electromyogram electrodes were placed on the anterior tibialis muscle to monitor leg movements. All polysomnograms were single-night diagnostic studies. Sleep and wake stages were scored by a blinded technician in 30-second epochs according to standard criteria.[9,10] Key sleep-architecture variables reported here include sleep latency to Stage 1, rapid eye movement (REM) latency (sleep onset to first epoch of REM sleep), total sleep time (TST, the total amount of time in non-REM (NREM) and REM sleep), time in bed (lights out to final arising), sleep efficiency (total sleep time/time in bed), OAI (total obstructive apneas per hour of sleep), CAI (total central apneas per hour of sleep), AHI (total apneas [obstructive, central, and mixed] and hypopneas per hour of sleep), minimum oxygen saturation, and the percentage of NREM (stages 1-4) and REM sleep stages, expressed as a percentage of TST. Obstructive apneas were defined by at least a 90% reduction in the pressure-flow signal with corresponding respiratory effort; central apneas were defined by at least a 90% reduction in pressure-flow signal without respiratory effort; mixed apneas were events that represented a combination of obstructive and central characteristics with a corresponding 90% or greater reduction in the pressure flow-signal; and hypopneas were defined as a great than 50% reduction in amplitude in pressure-flow signal lasting at least 10 seconds, desaturations or arousals were not necessary. Heights and weights were used to calculate body mass index.

For all analyses, mean, nightly minimum, and nightly maximum AP represented the independent variable and polysomnography measures of SDB the dependent variable, specifically, OAI, CAI, and overall AHI. Linearity of relationships was established 2 fold. First, scatterplots and regression lines were evaluated graphically. Next, dependent variables were divided into quartiles and regressed against the independent variable using an analysis of covariance approach, with the lowest barometric pressure quartile as the reference. A monotonic trend in coefficients demonstrated linearity. In the event of nonlinearity, robust standard error estimates allowed unequal variances. All models used a multiple linear regression approach with continuous independent and dependent variables with p values representing the trend of interquartile comparisons. The analysis of covariance approach interquartile comparison p values are also presented. Age, sex, and BMI were modeled as precision variables. Results are presented as quartile means with 95% confidence intervals. Stata 9.0 statistical software package was used for all analyses (StataCorp LP, College Station, TX). This research was approved by the University of Washington Institutional Review Board.

Table 1—Sample descriptive statistics.

	N	Mean	SD
Men, %	537	58.9	
Age, y	530	46.9	13.9
Weight, kg	534	100.5	31.7
Height, cm	519	172.4	11.1
BMI, kg/m²	519	34.0	9.8

RESULTS

Complete, sequential, diagnostic overnight polysomnography data from 537 patients were studied. From **Table 1**, a slight majority of patients were men, with a mean age of 47 years, and a mean body mass index of 34, indicating class I obesity as a whole. In general, patients had severe SDB, with a mean AHI of 39.6 and a mean SaO_2 nadir of 83.1%. Further descriptive statistics based on polysomnography results are included in **Table 2**, while **Table 3** demonstrates the minimum, median, and maximum values of the nightly AP range, minimum, and maximum.

Multivariate linear regression analysis revealed a significant relationship between mean, nightly minimum, and nightly maximum AP and OAI (all $p = 0.01$, **Table 4**). For mean AP, the fourth AP quartile OAI was significantly different from the first (16.6 vs 21.7 events/h; $p = 0.05$). For minimum AP, the second (18.9 events/h, $p = 0.05$), third (17.5 events/h, $p = 0.01$), and fourth (17.1 events/h, $p < 0.01$) AP quartile OAIs were significantly different than the first (23.9 events/h). For maximum AP, the third (17.4 events/h, $p = 0.05$), and fourth (16.9 events/h, $p = 0.03$) AP quartile OAIs were significantly different from the first (22.4 events/h). For every 1-millibar decrease in mean, minimum, or maximum nightly AP, OAI was increased by 0.3 events per hour. We found no significant overall trends or interquartile differences between mean, minimum, or maximum AP and CAI or AHI (**Tables 5** and **6**). Furthermore, AP was not associated with hypopneas or mixed apneas (data not shown).

Table 2—Sample diagnostic polysomnography statistics .

	No.	Mean	SD
TST, h	537	5.6	1.3
Sleep efficiency, %	537	76.8	14.8
Sleep latency, min	537	20.1	24.6
REM sleep latency, min	501	159.4	91.2
OAI	536	19.2	21.7
CAI	536	1.7	6.4
AHI	536	39.6	28.9
Min SaO_2, %	534	83.1	9.8
Sleep stage, %			
1	508	25.6	17.8
2	508	46.6	14.4
3-4	508	12.6	11.3
REM	508	15.3	8.5

REM refers to rapid eye movement; OAI, obstructive apnea index; CAI, central apnea index; AHI, apnea-hypopnea index. The sleep efficiency is calculated by dividing the total sleep time (TST) by the time in bed (TIB).

Table 3—The minimum, median, and maximum values of the nightly AP range, minimum, and maximum.

AP value. mB	Minimum	Median	Maximum
Range	0.3	2.3	11.3
Minimum	983.9	1015.3	1034
Maximum	987.9	1018.1	1035.6

AP refers to atmospheric pressure.

Table 4—Age-, sex-, and BMI-adjusted OAI by quartiles of mean, minimum, and maximum barometric pressure.

Mean barometric pressure quartiles	OAI, events/h	95% CI	Interquartile p value[a]	p value[b] (trend)
1	21.7	18.1, 25.2	NA	0.01
2	21.3	17.8, 24.8	0.89	
3	17.8	14.3, 21.2	0.12	
4	16.6	13.0, 20.1	0.05	
Minimum barometric pressure quartiles				
1	23.9	20.4, 27.4	NA	0.01
2	18.9	15.4, 22.4	0.05	
3	17.5	13.9, 21.0	0.01	
4	17.1	13.6, 20.6	< 0.01	
Maximum barometric pressure quartiles				
1	22.4	18.9, 25.9	N/A	0.01
2	20.6	17.1, 24.1	0.47	
3	17.4	13.9, 20.9	0.05	
4	16.9	13.4, 20.5	0.03	

OAI refers to obstructive apnea index; CI, confidence interval.
[a]Interquartile p values represent comparison of mean, minimum, and maximum barometric pressure quartiles 2, 3, and 4 to quartile 1 (the lowest quartile).
[b]p value for trend represents the adjusted regression coefficient using continuous independent (barometric pressure) and dependent (OAI) variables.

DISCUSSION

This large retrospective study of 537 patients undergoing diagnostic polysomnography documents that low nighttime weather-related AP can be associated with increased obstructive events as measured in the OAI, but not CAI or AHI. The OAI changes surprisingly do not match those seen in altitude studies, which have suggested that obstructive event numbers are either static or fall with altitude or decreased AP, whereas central apnea counts increase.[4,6] In our study, CAI did not change with decreased AP.[4,6] These differences could be due to a number of factors external to air pressures. In the altitude studies reviewed, altitude-related decreases in partial pressures of oxygen were either simulated or real; in this study, partial pressures of oxygen were not altered or investigated and are likely minimally altered.

Central apneas may be more responsive to changes in hypoxia, which are unlikely to occur with the small weather-related AP changes seen close to sea level. As in many sleep centers, the proportion of those diagnosed at UW Medicine Sleep Institute with idiopathic central sleep apnea is low, less than 5%, and most of patients with central apneas concomitantly have obstructive events as well. The number of central apneas in the study population was small, which may have affected our ability to discern associations due to type II errors. Other aspects to oxygenation might include humidity and, hence, air viscosity. We did not measure humidity in the ward.

Importantly, AHI failed to show a significant association with weather-related AP. Perhaps this is not surprising considering that the calculation for AHI includes a mixture of events (hypopneas and central, mixed, and obstructive apneas) with various physiologic etiologies. Hypopneas can be either obstructive or, to a lesser extent, central in nature. Differentiating these 2 types is impossible without an esophageal pressure manometer, which is not routinely employed in our sleep laboratory.

An alternate explanation relates to our hypopnea definition and polysomnography scoring. We scored hypopneas in both NREM and REM sleep. However, hypopnea-like respiratory variability in REM sleep is often physiologic, particularly when not associated with a cortical arousal or oxygen desaturation. These events may represent normal dream-related respiratory variability. Our hypopnea index is probably inflated by physiologic events in REM, possibly preventing us from associating hypopnea index, or overall AHI, with weather-related AP changes.

By definition, AHI measures are not pure assessments of central or obstructive events, and, by concentrating on findings of pure obstructive or central events, we can ascertain which is more commonly corrupted by weather-related AP changes. These findings suggest that the regulation of obstructive events

Table 5—Age-, sex-, and BMI-adjusted CAI by quartiles of mean, minimum, and maximum barometric pressure.

Mean barometric pressure quartiles	CAI, events/h	95% CI	Interquartile p value[a]	p value[b] (trend)
1	1.0	0.3, 1.6	N/A	0.13
2	1.9	0.7, 3.0	0.19	
3	1.8	0.8, 2.9	0.17	
4	2.3	0.7, 3.8	0.14	
Minimum barometric pressure quartiles				
1	1.1	0.4, 1.7	N/A	0.14
2	1.7	0.6, 2.8	0.34	
3	1.9	0.8, 3.0	0.21	
4	2.3	0.8, 3.8	0.15	
Maximum barometric pressure quartiles				
1	1.0	0.3, 1.6	N/A	0.10
2	1.3	0.8, 1.7	0.45	
3	2.5	1.0, 4.0	0.06	
4	2.2	0.7, 3.7	0.15	

CAI refers to central apnea index; CI, confidence interval.
[a]Interquartile p values represent comparison of mean, minimum, and maximum barometric pressure quartiles 2, 3, and 4 to quartile 1 (the lowest quartile).
[b]p value for trend represents the adjusted regression coefficient using continuous independent (barometric pressure) and dependent (CAI) variables.

Table 6—Age-, sex-, and BMI-adjusted AHI by quartiles of mean, minimum, and maximum barometric pressure.

Mean barometric pressure quartiles	AHI, events/h	95% CI	Interquartile p value[a]	p value[b] (trend)
1	40.1	35.5, 44.7	N/A	0.61
2	41.5	36.9, 46.0	0.69	
3	38.8	34.4, 43.3	0.69	
4	39.3	34.6, 44.0	0.81	
Minimum barometric pressure quartiles				
1	41.9	37.1, 46.8	N/A	0.73
2	38.5	34.3, 42.7	0.30	
3	39.1	34.6, 43.6	0.42	
4	40.1	35.4, 44.7	0.58	
Maximum barometric pressure quartiles				
1	40.3	35.7, 44.8	N/A	0.67
2	40.5	35.9, 45.0	0.95	
3	40.0	35.4, 44.5	0.92	
4	38.9	34.3, 43.5	0.69	

AHI refers to apnea-hypopnea index; CI, confidence interval. Robust standard-error estimates were used for the minimum barometric pressure analyses.
[a]Interquartile p values represent comparison of mean, minimum, and maximum barometric pressure quartiles 2, 3, and 4 to quartile 1 (the lowest quartile).
[b]p value for trend represents the adjusted regression coefficient using continuous independent (barometric pressure) and dependent (AHI) variables.

might be susceptible to small changes in ambient AP, whereas central events are not. Future prospective and experimental studies are necessary to determine causality.

Possible Mechanisms and Implications

Perhaps there is a critical threshold during which air pressure assists, albeit minimally, in holding pharyngeal airways open and therefore preventing an obstructive event. With decreases in ambient air pressures, that threshold is achieved. If that were the case, might continuous positive airway pressure delivery systems help compensate for those changes?

Most current continuous positive airway pressure machines correct pressure delivery through altering fan speeds based on nondynamic, preset altitude settings. Typically these adjustments focus on large AP changes associated with travel to significant altitude, such as Denver, Colorado (5280 feet above sea level), and not smaller, weather-related AP changes. Whether or not altering pressure delivery according to weather-related AP changes improves the control of SDB remains of interest. Such a responsive delivery may have the potential to improve care, functional outcomes, and device compliance.

Pitfalls

Problems with this study include the patient population, a selected group seen in a referral sleep center. In comparison with other published reports, our AHI mean is high. This may represent referral bias and methodologic differences in sleep scoring, particularly regarding hypopneas. We do not know if patients had concomitant pulmonary disease or if they were taking medications (opiates, acetazolamide) that might alter pH balances and subtly alter respiratory drive. Because this was an exploratory pilot study, no effort was made to control for multiple comparisons, and, as such, there are potential type I errors. Additionally we chose a standard time period, 8 hours from 21:00 to 07:00, which was longer than the mean sleep duration of 5.5 hours. Given that sleep is fractured and bed and wake times are variable, choosing a standard interval for all patients during which AP means could be calculated was deemed most appropriate. Our sleep dataset does not document the times during which sleep occurred; they are, instead, recorded as sums. With 537 patients studied, these time-based compromises should randomize out in a large group. Future prospective studies could correlate real-time sleep and weather-related AP with respiratory events. Lastly, we were unable to definitively determine if AP shifts influence oxygenation levels. Follow-up studies should assess the influence of AP on desaturation indices and nightly hypoxemic burden.

CONCLUSION

We found that OAI increased in a linear fashion with decreased AP. This relationship is modest and may have no clinical implications. A possible future study might be to test CPAP delivery systems responsive to dynamic weather-related AP

changes—particularly if those devices improve clinical outcome and tolerability. Additionally, AP changes due to weather systems may need to be considered in polysomnographic evaluation because mild OSA may be occasionally overdiagnosed or underdiagnosed and treated based on passing weather systems at the time of study.

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Submitted for publication June, 2009
Submitted in final revised form July, 2009
Accepted for publication September, 2009
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DISCLOSURE STATEMENT

This was not an industry supported study. Dr. Doherty has received consulting fees from Neurovista and speaker fees from UCB Pharma and GlaxoSmithKline. The other authros have indicated no financial conflicts of interest.