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Sleep–wake misperception in sleep apnea patients undergoing diagnostic versus titration polysomnography

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ABSTRACT

Objective: Insomnia is commonly co-morbid with obstructive sleep apnea. Among patients reporting insomnia symptoms, sleep misperception occurs when self-reported sleep duration under-estimates objective measures. Misperception represents a clinical challenge since insomnia management is based entirely on patient self-report. We tested the hypothesis that misperception occurring in sleep apnea patients would improve with subsequent treatment.
Methods: We compared subjective sleep–wake reports with objective sleep in adults with obstructive sleep apnea (n = 405) in two nights of polysomnography (diagnostic and treatment) within a median interval of 92 days.
Results: Sleep latency was generally over-estimated, while wake after sleep onset and number of awakenings were under-estimated. None of these estimations differed between diagnostic and treatment polysomnograms. We observed a large spectrum of total sleep time misperception values during the diagnostic polysomnogram, with one third of the cohort under-estimating their total sleep time by at least 60 min. Of those with >60 minute misperception, we observed improved total sleep time perception during treatment polysomnography. Improved perception correlated with improvements in self-reported sleep quality and response confidence. We found no polysomnogram or demographic predictors of total sleep time misperception for the diagnostic polysomnogram, nor did we find objective correlates of improved perception during titration.
Conclusion: Our results suggest that misperception may improve with treatment of obstructive sleep apnea in patients who also exhibit misperception. Within subject changes in misperception are consistent with misperception being, at least to some extent, a state characteristic, which has implications for management of patients with comorbid insomnia and sleep apnea.

Introduction

Sleep misperception, the mismatch between objective laboratory polysomnogram (PSG) data and subjective self-reported patient accounts, has been reported most commonly in patients with insomnia, but it can also occur in other sleep disorders such as obstructive sleep apnea (OSA) [1,2]. The diagnostic category of "paradoxical" insomnia refers to an extreme form of misperception, in which the sleep is objectively normal yet the patient reports little or no sleep [3]. However, there is no accepted clinical framework for phenotyping patients with misperception with less severe manifestations, or in whom misperception occurs concurrently with another sleep disorder such as OSA. In our prior retrospective study of misperception, the range of sleep misperception among patients with OSA (with or without insomnia symptoms) was quite large during diagnostic PSG nights [4]. Vanable and colleagues showed no difference in total sleep time (TST) misperception among patients with OSA, and they too observed a large variation in that group [5].

The mechanisms underlying sleep misperception remain to be conclusively elucidated, in part because misperception is likely influenced by various psychological, cognitive, and physiological factors. For example, physiological arousal [6], alpha–delta sleep [7] (but not all studies have found a relation with alpha–delta sleep [8]), cyclic alternating pattern [9], rapid eye movement (REM) sleep or slow wave sleep content [10–12], high frequency EEG content [13,14] and personality traits [15] have been associated with sleep misperception. Misperception can occur in healthy adults without sleep problems during routine laboratory and home conditions [16,17], and with extended time in bed [18]. One compelling hypothesis regarding sleep misperception is that it relates to fragmentation of sleep architecture, in which light stages or high frequency awakenings might predict decreased perception of sleep. However, we have not been able to definitively link the degree of misperception to the amount of stage N1, the degree of fragmentation, or other sleep–wake stage composition metrics [4,18].

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Understanding misperception is important for several reasons: 1) objective short sleep may carry the preponderance of risk associated with insomnia [19]; 2) feedback techniques may improve the sleep of those with insomnia and misperception [20,21]; and 3) patients who under-estimate their sleep may develop increased arousal related to anxiety about insomnia, which could then result in objective sleep disturbance as a perpetuating factor [2,22].

The approach to insomnia symptoms, and in particular sleep misperception, may be of particular interest for patients with comorbid OSA. Several cohort studies have suggested that those with insomnia may be at higher risk for OSA [23,24]. Specifically, the prevalence of OSA (using various respiratory event rate cutoffs of 5, 10 or 15 per hour) ranged from 15 to 75% [25–31]. Patients with diagnosed OSA are more likely to report insomnia symptoms, and concomitant treatment of both disorders may be mutually beneficial [24,32]. In particular, treatment of OSA may improve insomnia symptoms, but it may also be the case that positive airway pressure (PAP) treatment itself represents disturbance disruptive stimulus in the susceptible insomnia patient.

Methods

This retrospective database study was approved by the Partners Institutional Review Board without requiring additional consent for use of clinically acquired data. Inclusion criteria were: age ≥16 who underwent clinical polysomnography in our sleep center between January 2009 and June 2013, exhibited a respiratory disturbance index (RDI) or apnea–hypopnea index (AHI) of greater than 5, had a subsequent titration PSG within 12 months of the diagnostic PSG. We excluded those with missing questionnaires (although partially completed subjective data was allowed) or who had <2 h of sleep in either diagnostic or titration study. The final cohort included 405 patients. The reason for referral was not an inclusion criterion, but most were referred for OSA, and most were referred by non-sleep specialists. The median time between diagnostic and titration PSGs was 92 days. Our lab employs criteria for converting a diagnostic PSG into a titration study (split-night), based on severity, and thus the cohort studied here is enriched for mild to moderate severity OSA, although some severe cases were present. CPAP titrations were performed in accordance with the recommendations of the American Academy of Sleep Medicine. We separated those with AHI >10 during the titration study (n = 74) as a pre-specified marker for incomplete titrations, meaning that substantial apnea persisted. Since our goal was to investigate potential impact of treatment in the sense that apneas and hypopneas were reduced on the titration night, it was important to avoid inclusion of patients with ongoing apneas and hypopneas (even if their titration was eventually successful in the later hours of the study). Therefore, we pre-specified an AHI value of 10 during titration as a cutoff in this regard; this subset represented approximately the top 20% of AHI values during titrations. The remainder of PSG metrics were specified according to routine clinical reporting. The spontaneous arousal index was used (instead of total arousal index) to distinguish arousals not related to breathing pauses, which are the dominant form of arousal in OSA patients.

Self-report data was obtained through pre-sleep and post-sleep questionnaires that are routinely administered for all clinical PSGs in our lab. Pre-sleep questionnaire responses were taken from the diagnostic PSG nights, while post-sleep questionnaires were used from both diagnostic and titration PSGs. Pre-sleep questionnaires were used to assess self reported insomnia symptoms. Formal insomnia diagnostic categorization was not possible because the majority of patients did not undergo evaluation by a sleep specialist. We used insomnia symptom data to pre-specify division of the OSA patients into four categories: onset insomnia, maintenance insomnia, both, or neither. Onset insomnia was defined as reporting taking 30–60 or more minutes to fall asleep and/or choosing "I have trouble falling sleep" as a complaint from a list of check-boxes. Maintenance insomnia was defined as selecting "When I wake up at night, it takes me a long time to fall back asleep", and/or "I have trouble staying asleep", and/or reporting waking up 3 or more times at night.

Table 1
Demographics and PSG metrics for diagnostic and treatment PSGs

n		331
Sex		54% male
Age		54 (16, 89)
BMI		31 (27, 35)
ESS		8 (5, 11)
Time between studies (days)		93 (47, 161)
	Dx	PAP
Subj Lat (min)	30 (15, 60)	20 (15, 45)
Subj TST (min)	360 (300, 420)	360 (300, 420)
Subj # wakes	3 (2, 5)	3 (2, 5)
Subj WASO (min)	20 (10, 60)	20 (10, 45)
S–O Lat (min)	15 (3, 34.4)	10 (−3, 23.3)
S–O TST (min)	−34 (−91, 14)	−19 (−69, 36)*
S–O # wakes	−17 (−26, −10)	−16 (−23, −10)
S–O WASO (min)	−16 (−46, 5)	−22 (−49, −0)
TST (min)	383 (345, 417)	374 (326, 402)
Lat (min)	5 (2, 10)	4 (1, 10)
LPS (min)	12 (4, 27)	13 (5, 29)
REM Lat	124 (84, 205)	104 (73, 182)
N1%	14 (10, 23)	12 (8, 19)
N2%	55 (48, 62)	52 (45, 62)
N3%	12 (5, 19)	16 (8, 24)
R%	14 (9, 19)	16 (11, 22)
Eff (%)	88 (80, 94)	87 (78, 93)
#30sW	22 (14, 30)	20 (14, 27)
#60sW	11 (6, 16)	11 (7, 16)
Sp AI (h^{-1})	3 (2, 5)	5 (3, 7)
LMAI (h^{-1})	1 (0, 4)	2 (0, 6)
PLMI (h^{-1})	4 (1, 17)	7 (1, 24)
AHI (h^{-1})	13 (8, 20)	2 (1, 4)*
AHI supine	17 (10, 30)	2 (1, 5)*
AHI REM	26 (12, 44)	2 (0, 5)*
RDI (h^{-1})	27 (17, 37)	4 (2, 7)*
O_2 nadir REM	85 (80, 89)	92 (89, 94)
O_2 nadir NR	85 (82, 88)	90 (88, 92)

Median values, with 25–75% range shown in parentheses, separated by commas. Significant differences using Kruskal–Wallis with Dunn's correction are given by *. AHI, apnea–hypopnea index; BMI, body mass index; Dx, diagnostic PSG; Eff, efficiency; ESS; Epworth Sleepiness Scale; Lat, latency; LMAI, limb movement arousal index; LPS, latency to persistent sleep; min, minutes; N1–N3, NREM stages of sleep; O_2, oxygen; REM, rapid eye movement; PAP, positive airway pressure treatment PSG; PLMI, periodic limb movement index; S–O, subjective–objective difference; Sp AI, spontaneous arousals; Subj, subjective; TST, total sleep time; WASO, wake after sleep onset; #30sW, number of wakes ≥30 s long; #60sW, number of wakes ≥60 s long.

Post-sleep questionnaires were obtained on the morning after both the diagnostic and titration studies. Patients made subjective assessments of their sleep onset latency (Lat), total sleep time (TST), wake after sleep onset (WASO), number of awakenings, and overall quality of sleep. Each of these subjective queries was accompanied by a 7-point Likert scale rating the confidence of their response.

Statistics

Statistics were performed using Prism (GraphPad software, La Jolla, CA). Since most of the sleep measures being considered (TST, Latency, WASO, number of awakenings) were distributed non-normally, we used the non-parametric Kruskal–Wallis ANOVA (with Dunn's multiple comparison post hoc testing) for group comparisons, or Friedman test for Likert scale values. For the tables, the pre-specified groupings within which to use these ANOVA tests were diagnostic versus titration groups (two groups), and misperception groups (three groups). When the paired ANOVA (Friedman test) was used, we removed subjects with incomplete data from the questionnaires (which amounted to <5% of the cohort). Correlation analysis was done using non-parametric methods. We pre-