

Objective sleep measures after endoscopic sinus surgery in patients with chronic rhinosinusitis

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Background: Poor sleep quality is common in chronic rhinosinusitis (CRS). Prior studies have demonstrated improvements in patient-reported measures of sleep quality following endoscopic sinus surgery (ESS). The purpose of this study was to evaluate the effect of ESS on objective sleep parameters and identify any associations between specific objective measures and patient characteristics, comorbidities, and patient-reported outcome measures (PROMs).

Methods: Adults with CRS undergoing ESS were prospectively enrolled from 4 centers across North America. Any subject previously diagnosed with a known primary sleep disorder was excluded. Objective sleep indices were recorded using a portable sleep diagnostic device preoperatively and postoperatively. Patient-reported outcome instruments were completed including the Pittsburgh Sleep Quality Index (PSQI) and the 22-item Sino-Nasal Outcome Test (SNOT-22).

Results: Thirty-six patients (mean age 47 years, 56% male) completed baseline and postoperative sleep studies with mean $\pm$ standard deviation (SD) follow-up 9.6 ± 7.7 months. Mean PSQI and SNOT-22 before and after ESS was $10.2 \pm$

3.9 vs 7.8 ± 4.4 ($p = 0.001$); and 54.6 ± 14.6 vs 28.5 ± 15.3 ($p < 0.001$), respectively. Total sleep time, sleep latency, and awakenings after sleep onset did not change following ESS (all $p > 0.5$) despite improvements in PSQI and SNOT-22. Changes in PSQI did not correlate with comorbidities or objective sleep indices (all $p > 0.1$).

Conclusion: In this multicenter prospective cohort, objective sleep indices were not improved following ESS for CRS despite significant improvements in patient-reported sleep quality and CRS-specific QOL. © 2020 ARS-AAOA, LLC.

Key Words:

chronic rhinosinusitis; endoscopic sinus surgery; sleep; polysomnography; quality of life; patient reported outcome measure

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Sleep has an important role in maintaining quality of life (QOL) and a healthful and productive lifestyle. Chronic sleep dysfunction is common among patients with chronic rhinosinusitis (CRS),^{1–3} and poor sleep quality is an important contributor to QOL impairments in CRS.^{4,5} Although QOL domains measured by the 22-item Sino-Nasal Outcomes Test (SNOT-22) yield insight on CRS-specific QOL, the psychological and sleep dysfunction domains have demonstrated the strongest correlation with productivity losses.^{6–8} As a result, there is increased interest in understanding the health-related effects of sleep dysfunction associated with CRS so that we may better inform providers

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of sleep outcomes following treatment and align treatment decisions with patient preferences.

Much of the literature examining the impact of endoscopic sinus surgery (ESS) on sleep quality in CRS has focused on subjective, patient-reported outcome measures (PROMs). Prior investigations have demonstrated that ESS has a beneficial impact on patient-reported sleep quality, disease-specific QOL, and productivity.^{9–11} However, it remains unclear whether objective measures of sleep change after surgery. Self-reported sleep quality could reflect different aspects of the sleep experience, such as sleep onset, the duration of sleep, or frequency of awakenings.¹² Importantly, self-reported sleep quality does not always align with polysomnography (PSG) findings.^{12–14} Although patient-centered outcomes are essential to the investigation of sleep dysfunction in CRS, identifying objective indices associated with changes in patient-reported sleep quality following surgery may provide further insight on any potential variance in sleep experience, symptom expression, and the mechanisms of sleep morbidity observed in the CRS population. The aim of this study was to prospectively evaluate the impact of ESS on objective sleep measures in a multicenter cohort of patients with CRS. Our goal was to assess home PSG indices in CRS patients before and after ESS, and identify any associations between these objective measures and patient characteristics, comorbidities and PROMs.

Patients and methods

Study population

Adult (>18 years) patients with CRS were prospectively recruited from rhinology clinics at the Medical University of South Carolina (Charleston, SC), the University of Utah (Salt Lake City, UT), the University of Colorado (Aurora, CO), and Boston University (Boston, MA). This multi-institutional, prospective cohort study was approved in advance by the Institutional Review Boards at each institution and all subjects were provided written, informed consent in English. CRS was diagnosed using criteria from the Clinical Practice Guideline of the American Academy of Otolaryngology–Head and Neck Surgery.¹⁵ Each study participant self-selected ESS for treatment of recalcitrant symptoms associated with CRS. Extent of surgical treatment was left to individual surgeon discretion and tailored to the extent of disease seen clinically and radiographically.

Characteristics related to demographics and medical comorbidities were collected by each patient self-report and when necessary by medical chart review, including presence of asthma, immunodeficiency, aspirin intolerance, gastroesophageal reflux disease, allergies confirmed by testing, obstructive sleep apnea (OSA) by history or testing, migraine, pain disorder, and depression. Body mass index (BMI) was obtained preoperatively for each individual. Tobacco and alcohol intake was recorded as number of cigarettes per day and number of alcoholic drinks per week, respectively. Current medication use was recorded and included antihis-

tamines, antidepressants, anxiolytics, antipsychotics, and other medications known to impact sleep. Any subject previously diagnosed with a primary sleep disorder such as OSA, narcolepsy, or insomnia was excluded from the study.

Sleep quality evaluation

Subjective sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), a self-reported questionnaire measuring sleep quality and disturbance over the preceding 1-month period.¹⁶ A PSQI score ≤ 5 is considered the threshold for “good” sleep quality. A PSQI score > 5 is considered “poor” sleep quality with a sensitivity of 89.6% and specificity of 86.5% ($\kappa = 0.75$; $p < 0.001$). The PSQI contains 7 component scores (range, 0 to 3) including: sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, sleep medication usage, and daytime dysfunction. These component scores have high internal consistency (Cronbach’s $\alpha = 0.83$).

Sleep was objectively assessed with the WatchPAT device (Itamar Medical, Inc, Franklin, MA), a U.S. Food and Drug Administration (FDA)-approved, wrist-worn device with integrated finger and snoring sensors allowing measurements of peripheral arterial tone (PAT), oxygen saturation, pulse rate, actigraphy, and snoring. All patients were given detailed instruction on administration of the home sleep test by a dedicated study coordinator at each study site. Subjects were directed to administer the home sleep test on a night where they follow a typical nightly routine. Respiratory Disturbance Index (RDI) and Apnea-Hypopnea Index (AHI) were measured by simultaneous measurement of PAT, oxygen saturation, and heart rate. Sleep architecture was recorded, and indices included sleep onset latency, total sleep time, number of awakenings, light sleep, deep sleep, and percentage of time in rapid eye movement (REM) and non-REM (NREM) sleep. Direct comparisons of the WatchPAT device and in-office PSG have demonstrated excellent correlation for both RDI and AHI (r , 0.82 to 0.89),^{17–20} as well as excellent epoch-by-epoch agreement ranging from 80% to 90%.^{21–23} Home sleep testing was performed at baseline and at least 4 months postoperatively to allow for complete recovery after surgery.

CRS assessment and other PROMs

Patient-reported sinonasal-specific QOL and CRS severity was measured using the SNOT-22, a 22-item questionnaire (range, 0 to 110) with higher scores indicating worse QOL.²⁴ Daytime sleepiness was measured by the Epworth sleepiness scale (EpSS; range, 0 to 24), with higher scores indicating increased sleepiness.²⁵ The level of fatigue experienced over the preceding 2-week period was measured by the fatigue severity scale (FSS; range, 1 to 7), with score > 4 indicating more fatigue.²⁶ Presence of depressed mood and anhedonia over the preceding 2 weeks were assessed using the Patient Health Questionnaire-2 (PHQ-2; range, 0 to 6), with score ≥ 3 suggestive of depressed mood.²⁷ The experience of pain was measured

by the short-form McGill Pain Questionnaire (SF-MPQ).²⁸ Nasal obstruction was measured by the Nasal Obstruction Symptom Evaluation (NOSE; range, 0 to 100), with higher scores indicating greater severity of symptomatic nasal obstruction.²⁹

Statistical analysis

Statistics were carried out via SPSS, version 25 (IBM Corporation, Armonk, NY). Patient characteristics were described using means and standard deviations (SDs) for continuous variables while frequencies and percentages were used for categorical variables. Assessment of differences between preoperative and postoperative patient-reported and objective sleep testing measures were conducted using paired *t* tests for normally distributed variables and Wilcoxon signed rank tests for those non-normally distributed. The Mann-Whitney *U* test was used to compare differences in the non-normally distributed baseline objective sleep testing measures between nasal polyp status groups. Change in sleep test variables were made by subtracting follow-up scores from baseline scores, so that a positive number indicated improvement. Pearson correlations were used to explore associations between change in variables that met assumptions of linearity, homoscedasticity, and no outliers. If assumptions were not met, Spearman correlations were used. Values of $p \leq 0.05$ were considered statistically significant.

Results

A total of 36 patients, with an average age of 47 years (range, 21 to 67 years) were enrolled in the study with 55.6% being male. Seventy-eight percent of the patients were white. Twenty-one (58.3%) had a history of prior sinonasal surgery. Average BMI was 28.7 ± 6.9 . Among the overall cohort, 56% of patients were diagnosed with CRS with nasal polyposis (CRSwNP) and 44% as CRS without nasal polyposis (CRSSNP). One-third of patients had asthma and 58% had allergies by testing. Table 1 includes baseline demographics, extent of surgery, medical comorbidities, and medication usage that may impact sleep. The average time to postoperative questionnaire and objective home sleep testing follow-up was 9.6 ± 7.7 months. The time from surgery to postoperative QOL and sleep testing was at least 4 months for all patients in the study with a range of 4 to 44 months. Variations in follow-up intervals were due to variations in patient follow-up and availability for postoperative assessment.

Table 2 reports the preoperative and postoperative PROMs. CRS-specific disease severity as measured by SNOT-22 significantly improved following surgery from 54.6 ± 14.6 to 28.5 ± 15.3 ($p < 0.001$). In addition, subjective sleep quality as measured by PSQI improved from 10.2 ± 3.9 to 7.8 ± 4.4 ($p = 0.001$) with significant improvements noted postoperatively for the following PSQI subdomains: sleep disturbance ($p = 0.002$), daytime dys-

TABLE 1. Baseline demographics, comorbidities, and medication usage that may impact sleep

Characteristic	Value
Demographics	
Age (years), mean $\pm$ SD	47 $\pm$ 12.9
Sex, n (%)	
Male	20 (55.6)
Female	16 (44.4)
Race, n (%)	
White	28 (77.8)
African American	7 (19.4)
Other	1 (2.8)
Ethnicity, n (%)	
Hispanic/Latino	0 (0.0)
Non-Hispanic/Latino	36 (100.0)
BMI (kg/m ²), mean $\pm$ SD	28.7 $\pm$ 6.9
Previous sinus surgery, n (%)	21 (58.3)
Polyp status, n (%)	
CRSSNP	16 (44.4)
CRSwNP	20 (55.6)
Bilateral full-house sinus surgery, n (%) ^a	18 (50.0)
Septoplasty, n (%)	15 (41.7)
Comorbidities	
Asthma, n (%)	12 (33.3)
ASA intolerance, n (%)	2 (5.6)
Allergy by testing, n (%)	21 (58.3)
Depression (physician diagnosis), n (%)	5 (13.9)
Obstructive sleep apnea (physician diagnosis), n (%)	0 (0.0)
Pain disorder, n (%)	2 (5.6)
Migraine, n (%)	5 (13.9)
Alcohol (alcoholic drinks/week), mean $\pm$ SD	2.4 $\pm$ 4.5
Smoking (cigarettes/day), mean $\pm$ SD	0 $\pm$ 0.0
Immunodeficiency, n (%)	1 (2.8)
Bed partner of CRS patient, n (%)	
No	11 (91.7)
Yes	1 (8.3)
Medications, n (%)	
Antihistamines	14 (41.2)
Antidepressants	4 (11.4)

(Continued)

TABLE 1. (Continued)

Characteristic	Value
Anxiolytics	1 (2.9)
Antipsychotics	0 (0.0)
Other medications that impact sleep	
Melatonin	2 (5.6)
Trazodone	1 (2.8)
Ritalin	1 (2.8)

^a Full-house sinus surgery (maxillary antrostomy + complete sphenoidotomy + frontal sinusotomy).

ASA = aspirin; BMI = body mass index; CRS = chronic rhinosinusitis; CRSsNP = CRS without nasal polyposis; CRSwNP = CRS with nasal polyposis; SD = standard deviation.

function due to sleepiness ($p = 0.002$), sleep efficiency ($p = 0.018$), and overall sleep quality ($p = 0.002$). Significant improvements following surgery were also observed in NOSE score (15.1 ± 3.8 to 6.8 ± 5.2 , $p < 0.001$), FSS score (3.9 ± 1.3 to 3.0 ± 1.4 , $p = 0.002$), and SF-MPQ (11.3 ± 7.9 to 3.9 ± 4.4 , $p < 0.001$). Improvement in EpSS was observed after surgery, from 8.4 ± 5.1 to 7.1 ± 4.0 ; however, this did not reach statistical significance ($p = 0.064$).

Objective home sleep testing collected from 36 patients before and after surgery is presented in Table 3. Each home sleep test measured 25 individual indices. Total sleep time was nearly identical before and after intervention, and relatively few significant changes were observed following surgery in these indices. AHI increased from 13.2 ± 13.2 to 16.4 ± 18.1 ($p = 0.045$), and RDI increased from 18.8 ± 13.5 to 22.6 ± 16.3 ($p = 0.009$) following surgery. In addition, oxygen desaturation index (ODI) increased after surgery from 7.0 ± 9.7 to 10.0 ± 14.3 ($p = 0.015$), and percent deep sleep decreased from 19.3 ± 5.9 to 16.2 ± 6.5 ($p = 0.003$). There were no correlations observed between any objective indices of sleep and PSQI following surgery; Table 4 shows no correlation between change in PSQI and change in selected indices commonly associated with sleep quality, including total sleep time, sleep latency, number of wakes, and percentage of deep sleep. Change in PSQI did not correlate with medical comorbidities listed in Table 1 (all $p > 0.1$). Decrease in number of wakes was weakly associated with longer length of postoperative follow-up ($r = 0.383$, $p = 0.023$), however, no correlation was observed between follow-up and other objective sleep quality indices. The extent of surgery did not impact outcomes for sleep quality nor objective testing. These findings were similar among patients undergoing bilateral full-house surgery as well as targeted surgery. Among CRSwNP, there were no significant changes in any of the sleep indices postoperatively with the exception of deep sleep percent, which decreased from 19.2 ± 6.0 to 16.0 ± 7.7 ($p = 0.040$). Among CRSsNP patients, RDI, REM RDI, NREM RDI, and REM AHI increased from 14.2 ± 9.9 to 20.7 ± 11.1 ($p = 0.004$); 19.7 ± 11.7 to 29.1 ± 13.8 ($p = 0.002$); 12.3 ± 9.5 to 17.7

± 11.1 ($p = 0.020$); and 13.6 ± 12.7 to 19.4 ± 17.3 ($p = 0.016$), respectively. None of the remaining sleep indices significantly changed following surgery among CRSsNP patients (all $p > 0.05$).

Objective sleep testing parameters were reviewed on the basis of AHI and RDI (Table 5). Preoperative AHI was <5 for 40% of patients and >5 for 60% of patients; with 36% of patients demonstrating an AHI >15 preoperatively and 39% of patients demonstrating an AHI >15 postoperatively. Preoperative RDI was <15 for 55% of patients and >15 for 45% of patients; with 45% of patients demonstrating an RDI >15 preoperatively and 58% patients demonstrating an RDI >15 postoperatively.

Discussion

Prior investigations have demonstrated that patients with CRS report poor sleep quality at baseline, with improvements following treatment including both medical and surgical therapy.²⁻⁴ This study supports those findings with similar baseline impairment in PSQI, as well as significant improvement postoperatively. Interestingly, no objective indices captured by home sleep testing improved following sinus surgery for CRS, despite these subjective improvements in sleep quality as measured by the PSQI. Nor were any significant associations identified between individual sleep parameters and change in PSQI after surgery. These findings question the idea that abnormal sleep quality reported by patients with CRS is a direct reflection of underlying abnormalities in objective measures of sleep. Alternatively, the observed disparity between subjective and objective sleep findings in this study may perhaps be the result of the intrinsic limitations of the sleep device utilized in this study. Although the PAT algorithms have been validated in measuring and screening for OSA, there are limitations in the evaluation of sleep architecture.²⁰⁻²³ Objective evaluation of sleep with alternative testing modalities, such as electroencephalogram (EEG), electromyography (EMG), and electrooculogram (EOG), are more accurate at identifying changes in sleep architecture.¹²⁻¹⁴ Similar observations that objective PSG measures may not differ from a “normal” control population despite poor patient-reported sleep quality have also been reported for patients with insomnia as well as attention deficit hyperactivity disorder.^{14,30}

Our original hypothesis was that patient-reported sleep dysfunction would be a consequence of underlying changes in respiratory indices or sleep architecture, such as sleep onset latency, total sleep time, number of awakenings, or percentage of time in deep sleep based on prior work demonstrating abnormal objective sleep parameters in CRS compared with controls.^{2,31} However, we did not observe notable changes in any of these sleep indices following surgical intervention. There are several possible suggestions for the lack of findings. One possible explanation is that small changes in objective sleep indices exist, but were not captured due to insufficient power. However, given the magnitude of observed change in PSQI, it seems unlikely that we

TABLE 2. CRS disease severity and patient-reported outcome measures

Parameter	Preoperative (mean $\pm$ SD)	Postoperative (mean $\pm$ SD)	<i>p</i>
SNOT-22 (n = 31)	54.6 $\pm$ 14.6	28.5 $\pm$ 15.3	<0.001
PSQI (n = 31)	10.2 $\pm$ 3.9	7.8 $\pm$ 4.4	0.001
PHQ-2 (n = 31)	1.6 $\pm$ 1.4	1.3 $\pm$ 1.4	0.225
FSS (n = 30)	3.9 $\pm$ 1.3	3.0 $\pm$ 1.4	0.002
EpSS (n = 31)	8.4 $\pm$ 5.1	7.1 $\pm$ 4.0	0.064
NOSE (n = 32)	15.1 $\pm$ 3.8	6.8 $\pm$ 5.2	<0.001
SF-MPQ (n = 29)	11.3 $\pm$ 7.9	3.9 $\pm$ 4.4	<0.001
Change in PSQI subdomains following sinus surgery			
Sleep duration	1.3 $\pm$ 1.1	1.0 $\pm$ 1.0	0.346
Sleep disturbance	2.0 $\pm$ 0.7	1.5 $\pm$ 0.6	0.002
Sleep latency	1.6 $\pm$ 1.1	1.4 $\pm$ 1.0	0.260
Daytime dysfunction due to sleepiness	1.6 $\pm$ 0.7	1.1 $\pm$ 0.6	0.002
Sleep efficiency	1.3 $\pm$ 1.2	0.8 $\pm$ 1.1	0.018
Overall sleep quality	1.8 $\pm$ 0.9	1.3 $\pm$ 0.8	0.002
Needs medications to sleep	1.2 $\pm$ 1.4	0.9 $\pm$ 1.3	0.398

CRS = chronic rhinosinusitis; EpSS = Epworth Sleepiness Scale; FSS = fatigue severity scale; NOSE = Nasal Obstruction Symptom Evaluation; PHQ-2 = Patient Health Questionnaire; PSQI = Pittsburgh Sleep Quality Index; SD = standard deviation; SF-MPQ = short-form McGill Pain Questionnaire; SNOT-22 = 22-item Sino-Nasal Outcome Test.

would see no discernible improvements from baseline sleep indices. Another possible explanation for the lack of any observed changes in objective sleep could be the methodology employed to assess the experience of sleep. PSG as performed in this fashion offers an objective set of sleep parameters and measurements on 1 particular night and therefore may not be fully representative of the overall sleep experience as assessed by PROMs such as the PSQI. A number of studies have suggested that traditional PSG may not comprehensively reflect sleep quality.^{12–14} The evaluation of sleep quality in the context of PSG often focuses on identifying sleep indices relevant to patient-reported sleep dysfunction, including sleep onset latency, total sleep time, and time awake after sleep onset but before the final awakening. Similar to the sleep stage scoring that is traditionally used to characterize the sleep experience, these measures may be a crude indicator of sleep quality due to the synthetic segmentation of a continuous process. Although the wrist-worn ambulatory sleep recorder used in this study has previously been validated in several studies and demonstrated reliability in the detection of autonomic arousals and sleep/wake status, as well as sleep architecture,^{20–23} formal in-laboratory investigations may provide more granularity.

It is also possible that the disparity between subjective improvement in sleep quality and the lack of changes in objective sleep indices are mediated through an alternative mechanism. Somnogenic proinflammatory cytokines have

previously been implicated in the pathophysiology of sleep for several inflammatory conditions including CRS.^{4,32–34} Furthermore, it has been hypothesized that changes in local cytokine production following sinus surgery may have a role in the observed improvements in sleep quality.⁹ This may be supported by the observed improvement in FSS and EpSS following surgical intervention. Although significant improvements in PSQI were observed following surgical intervention, it is noted that the mean PSQI score was 7.8 postoperatively, which remains above what is considered a normal range. Alternatively, it is possible that patients conceptualize the disease-specific symptoms of CRS, such as facial pain and pressure, in the context of their overall sleep experience. Perhaps by integrating these CRS symptoms into their experience of sleep and wakefulness, the improvements observed in CRS-specific QOL following surgery may also impart perceived changes in patient-reported sleep quality. Prior studies have demonstrated that improved sleep quality scores have a corresponding association with improved disease-specific QOL (as measured by SNOT-22) as well as pain scores.^{6,9} This finding is also supported by our study's observation that SNOT-22, NOSE, and SF-MPQ pain scores significantly improved following sinus surgery; suggesting that improvements in subjective sleep quality following sinus surgery may be the result of this improvement in CRS-specific disease burden, rather than improvement in objective measures of sleep, such as

TABLE 3. Objective home sleep study results before and after ESS

Parameter	Preoperative (mean $\pm$ SD)	Postoperative (mean $\pm$ SD)	<i>p</i>
Total sleep time (n = 36)	374.7 $\pm$ 73.1	374.5 $\pm$ 73.8	0.990
Sleep latency (n = 36)	22.2 $\pm$ 14.3	23.8 $\pm$ 12.9	0.480
Number of wakes (n = 35)	9.0 $\pm$ 5.0	8.8 $\pm$ 4.4	0.797
AHI (n = 33)	13.2 $\pm$ 13.2	16.4 $\pm$ 18.1	0.045
REM AHI (n = 33)	17.7 $\pm$ 15.4	21.1 $\pm$ 16.7	0.094
Non-REM AHI (n = 33)	11.5 $\pm$ 13.2	14.3 $\pm$ 19.2	0.339
RDI (n = 33)	18.8 $\pm$ 13.5	22.6 $\pm$ 16.3	0.009
REM RDI (n = 33)	24.3 $\pm$ 14.5	28.3 $\pm$ 13.4	0.076
Non-REM RDI (n = 33)	16.8 $\pm$ 14.0	20.2 $\pm$ 18.0	0.112
ODI (n = 33)	7.0 $\pm$ 9.7	10.0 $\pm$ 14.3	0.015
REM ODI (n = 33)	9.4 $\pm$ 10.1	11.9 $\pm$ 12.3	0.156
Non-REM ODI (n = 33)	6.0 $\pm$ 9.9	8.8 $\pm$ 15.5	0.175
Mean oxygen saturation (n = 33)	93.5 $\pm$ 2.6	93.5 $\pm$ 2.6	0.763
Oxygen nadir (n = 33)	86.9 $\pm$ 5.2	86.4 $\pm$ 5.6	0.311
Mean pulse rate (n = 35)	68.2 $\pm$ 12.1	66.5 $\pm$ 11.0	0.190
Light sleep percent (n = 34)	55.6 $\pm$ 10.7	60.3 $\pm$ 11.0	0.045
Deep sleep percent (n = 34)	19.3 $\pm$ 5.9	16.2 $\pm$ 6.5	0.003
REM latency (n = 35)	95.0 $\pm$ 68.8	111.3 $\pm$ 65.4	0.323
REM percent (n = 34)	26.0 $\pm$ 12.4	23.5 $\pm$ 7.4	0.320
Percent supine position (n = 36)	44.6 $\pm$ 30.3	43.4 $\pm$ 27.9	0.863
Percent prone position (n = 36)	9.6 $\pm$ 18.0	12.7 $\pm$ 17.9	0.749
Percent right side position (n = 36)	20.2 $\pm$ 21.1	20.3 $\pm$ 19.2	0.871
Percent left side position (n = 36)	25.5 $\pm$ 23.7	23.5 $\pm$ 17.2	0.980
Snoring (n = 35)	41.5 $\pm$ 3.2	41.4 $\pm$ 3.4	0.916
Percent sleep snoring >40 dB (n = 36)	23.9 $\pm$ 26.3	20.3 $\pm$ 21.6	0.320

AHI = apnea-hypopnea index; ESS = endoscopic sinus surgery; ODI = oxygen desaturation index; RDI = respiratory disturbance index; REM = rapid eye movement; SD = standard deviation.

TABLE 4. Correlation between change in PSQI and change in selected indices commonly associated with sleep quality

Parameter	Change in PSQI		
	Correlation coefficient	<i>p</i>	n
Change in total sleep time	−0.013	0.944	31
Change in sleep latency	0.090	0.631	31
Change in number of wakes	0.048	0.801	30
Change in deep sleep percent	0.113	0.558	29

PSQI = Pittsburgh Sleep Quality Index.

TABLE 5. Baseline and postoperative AHI and RDI status

Parameter	Preoperative n (%)	Postoperative n (%)
AHI		
<5	13 (39.4)	14 (38.9)
5 through <15	8 (24.2)	8 (22.2)
15 through <30	9 (27.3)	6 (16.7)
≥30	3 (9.1)	8 (22.2)
RDI		
<5	2 (6.1)	0 (0)
5 through <15	16 (48.5)	15 (41.7)
15 through <30	10 (30.3)	12 (33.3)
≥30	5 (15.1)	9 (25.0)

AHI = apnea-hypopnea index; RDI = respiratory disturbance index.

sleep onset latency, total sleep time, and awakenings. This mechanism may also contribute to the relatively high prevalence of baseline subjective sleep dysfunction among patients with CRS as observed in our cohort as well as in prior studies.¹⁻⁴ Certainly, this is an area that deserves further investigation given that sleep quality does associate closely with an individual's burden of disease, QOL, and productivity.⁷⁻¹¹

Patients with a history of diagnosed OSA were excluded from enrollment in this study. However, our baseline sleep data showed that 60% of our cohort had an AHI >5; 36% had an AHI >15; and 45% had an RDI >15. Several prior studies have found good correlations between the WatchPAT and the gold standard PSG in the assessment of AHI, RDI, and lowest oxygen saturation.^{18,19,35-37} Prior work investigating the WatchPAT ability to detect a patient with OSA demonstrated that at AHI >5, the sensitivity was 0.95 and specificity was 0.80; and at AHI >15, the sensitivity was 0.96 and specificity was 0.79.³⁸ These baseline characteristics suggest that some of the patients in our CRS cohort may have had undiagnosed and/or untreated OSA. Despite this finding, neither AHI nor RDI correlated with change in PSQI following surgery, suggesting that concurrent OSA was not a major contributing factor for the observed improvements in subjective sleep quality. Additionally, there were small, but statistically significant, increases in AHI, RDI, and ODI, and decreases in percentage deep sleep on postoperative follow-up home sleep testing. Although the clinical relevance of these small changes is unclear, it is perhaps possible that some of these patients were developing progression of undiagnosed OSA and would benefit from formal evaluation and treatment for undiagnosed OSA. Additionally, although none of the patients in this study were utilizing continuous positive airway pressure (CPAP), it should be noted that 15 subjects underwent

ESS with septoplasty, and prior studies have demonstrated improved CPAP compliance rates following septoplasty to correct nasal obstruction.³⁹

Findings from this study were surprising and in many ways generate more questions than answers. Although logistically challenging, future studies examining objective measures of sleep quality will likely need to be large in size, and data will need to be collected over several nights to capture subtleties in objective measures. In the context of our study, the collection of objective sleep data on a single night is a limitation for interpretation of the broader sleep experience. Additionally, although our analysis did not demonstrate an association between follow-up duration to home sleep testing and individual sleep parameters, we acknowledge the variation in postoperative follow-up among this study cohort (range, 4 to 44 months). Follow-up sleep testing performed more than 3 years after surgery may be sub-optimal in considering the impact of surgery on sleep, and future studies may choose to target a shorter duration to follow-up analysis in order to limit potential confounding factors such as changes in BMI or progression of untreated sleep apnea. Future studies should also consider alternative testing strategies such as formal PSG, EEG, EMG, or EOG given the sensitivity of these instruments in assessing sleep architecture as compared to the home PAT device. Performing sleep testing on multiple occasions may also capture the richness of the patient's sleep experience over time. Additionally, although baseline demographics including BMI and somnogenic medications were accounted for at the time of study participant enrollment, the potential for changes in BMI, medication compliance, or the introduction of new sleep-related medications were not uniformly accounted for. Nonetheless, these data are useful for perioperative counseling, as sleep dysfunction is one of the driving factors for patients electing ESS.⁴⁰ Our data indicate that improvements in perceived sleep quality and nasal obstruction can be expected for most patients, but are independent of underlying objective sleep measures such as oxygen saturation or REM sleep time, which may not change significantly.

Conclusion

Objective sleep indices were not improved following ESS for the patients in this prospective, multicenter cohort study despite significant improvements in patient-reported sleep quality and CRS-specific QOL. The QOL burden as it relates to baseline fatigue, excessive sleepiness, and pain scores were similar to prior literature examining CRS, and sinus surgery was associated with improvements in these PROMs. Interestingly, although subjective sleep impairment and daytime fatigue were present in this study population and improved following surgical intervention, no objective improvements were observed on home sleep testing. AHI, RDI, ODI, and deep sleep percent demonstrated a small, but statistically significant worsening following surgery. The objective measurement of the physiological

correlates of sleep quality is complex and the mechanism through which these improvements may occur remains unknown. Further studies characterizing the sleep experience

would be helpful in identifying potential variance, symptom expression, and mechanisms of the sleep morbidity observed in CRS. 

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