Prospective Randomized Study of Patients with Insomnia and Mild Sleep Disordered Breathing

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Background: Patients with insomnia may present with mild and often unrecognized obstructive sleep apnea (OSA).

Objective: To evaluate both subjective and objective outcomes of patients with complaints of insomnia and mild OSA who receive surgical treatment for OSA versus behavioral treatment with cognitive behavioral therapy for insomnia (CBT-I).

Methods: Prospective study with crossover design of 30 patients with complaints of insomnia and mild OSA. Thirty subjects, matched for age and gender, were randomized with stratification to receive either CBT-I or surgical treatment of OSA as primary treatment. Patients were reassessed after completing the initial intervention and reassigned if agreeable to the alternative treatment option and assessed again on completion of both treatment arms. Outcome measures included clinical impression, Epworth Sleepiness Scale (ESS) score, Fatigue Severity Scale (FSS) score, and polysomnography (PSG) results.

Results: Surgery resulted in greater improvements in total sleep time (TST), slow wave sleep and REM sleep duration, respiratory disturbance index, apnea-hypopnea index, minimum oxygen saturation, FSS, and ESS. CBT-I also improved TST and resulted in shorter sleep latency.

Conclusion: Surgical intervention for the management of patients with complaints of insomnia and mild OSA demonstrated greater improvement in both subjective and objective outcome measures. Initial treatment of underlying OSA in patients with insomnia was more successful in improving insomnia than CBT-I alone. However CBT-I as initial treatment improved TST compared to baseline; following surgical intervention, it had the additional benefit of further increasing TST and helped to control sleep onset difficulties that may be related to conditioning due to unrecognized symptoms of mild OSA.

Keywords: Insomnia, mild obstructive sleep apnea, cognitive-behavioral-therapy, surgery, fatigue, treatment

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COMORBID INSOMNIA IS COMMON AND-ASSOCIATED SYNDROMES CAN OFTEN BE UNRECOGNIZED AND UNTREATED. THE ASSOCIATION BETWEEN INSOMNIA and obstructive sleep apnea (OSA) has been well recognized[1] with approximately 50% to 55% of patients with OSA presenting with complaints of insomnia.[2-4] Insomnia complaints have also been seen more commonly in patients with mild sleep disordered breathing (SDB).[4] Women with SDB may present more often with insomnia features compared to men. Patients with SDB and insomnia appear to suffer less from hypoxia and daytime sleepiness than patients with classic sleep apnea commonly seen in overweight individuals.[3,5] As mild SDB may not be associated with chronic snoring, particularly in premenopausal women, the SDB may be ignored while the chronic insomnia is treated pharmacologically with variable success and incomplete resolution of the daytime complaints. Even when polysomnography is performed (a rare event in the presence of insomnia complaint), arbitrary decisions are taken to treat the SDB when the selected index of severity (apnea-hypopnea index [AHI]) is above a value of 10 or 15 events/hour. This has lead to inappropriate patient care and secondary complications associated with chronic nocturnal sleep disturbances—mainly depression.[5]

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The most common treatment options for OSA consist of surgery, continuous positive airway pressure (CPAP), or surgery and oral appliance use. Cognitive behavioral therapy (CBT-I) has been shown to have the best long-term outcome in patients with chronic insomnia.[6-8] The best management strategy for patients with combined insomnia and OSA remains unclear.[5] In this prospective crossover study, we evaluated the impact of primary management of OSA with surgery followed by cognitive-behavior therapy for insomnia (CBT-I) versus primary management of insomnia with CBT-I followed by treatment of OSA on subjective and objective measures of sleep quality.

CBT-I treatment is not always necessarily available at the time of diagnosis, and patients may have to deal with a long waiting list. Furthermore, nasal CPAP is not necessarily the treatment of choice for patients with primary insomnia complaints who also have mild OSA. As an alternative, many patients may request trials with dental devices, surgical approach, or both treatments.

At the Stanford Sleep Disorders clinic, we recognize that the first-line treatment for insomnia when it occurs comorbidly with SDB, has not been standardized. Because of the extended wait for any form of treatment in our setting, we suggested that these patients consider either a non-drug treatment approach (CBT-I), or treatment for OSA of their choice as the initial treatment, followed by another treatment with after a recovery period. Many patients refused this approach and requested treatment of either insomnia or OSA but not both in sequence, or requested pharmacologic treatment for insomnia. A minority of patients expressed interest in undergoing both treatments sequentially. As we had no clear indication which treatment should be applied

first, these patients were randomized. The main objective of this study was to investigate the impact of the sequence of applied treatments on qualitative and quantitative sleep variables, by treating insomnia or OSA first.

METHODS

Consecutive subjects complaining of insomnia were identified from the population of patients referred to the Stanford Sleep Disorders Clinic (an academic tertiary care referral center for sleep disorders) between January 1, 2002, and December 30, 2003. All 201 patients complaining of insomnia had undergone overnight polysomnography (PSG) that documented the presence of OSA.

All patients underwent the same clinical evaluation. For the past 5 years, we have systematically and prospectively collected the same information on all patients evaluated at the Stanford University Sleep Disorders Clinic. Patients gave informed consent for anonymous usage of collected clinical data. Investigations on specific sleep complaints were performed.

Independent of the initial patient complaints, a validated sleep questionnaire (SDQ),[9] the Epworth Sleepiness Scale (ESS),[10] the Fatigue Severity Scale (FSS),[11] and a set of systematic questions were completed. The sleep specialist performed a standardized physical examination using clinical scales such as the Friedman tonsils and tongue scales[12] and the Mallampati scale.[13] The results of standard intra- and extra-oral examinations were noted.

Otolaryngologic evaluation and cephalometric radiogram were performed and data collected on all patients with abnormal breathing during sleep.. If surgery was considered, additional presurgical evaluation was performed by a surgeon specialized in the treatment of OSA.

Recommendations for the type of surgery were made based on the patient anatomy, disease severity, comorbid conditions, and patient preference. Surgeries included a combination of pharyngoplasty, tonsillectomy and adenoidectomy, genioglossal advancement, septoplasty, and radiofrequency reduction of inferior nasal turbinates. Recommendations for the use of a dental appliance with or without prior surgical treatment were made after evaluation by an otolaryngologist and an orthodontist specialized in the treatment of OSA. The dental device may have been part of the original treatment plan or were added as an adjunctive treatment after demonstration of residual OSA post-surgery. In our clinic, treatment outcome (surgical or nonsurgical) is considered successful if there is a disappearance of clinical symptoms and if follow-up PSG indicates an AHI ≤5/hour with oxygen saturation by pulse oximetry above 92%.

Inclusion/Exclusion Criteria

The criteria for inclusion were complaint of insomnia for ≥3 months, with sleep problems ≥ 3 times per week, and report of sleep disturbance for ≥30 minutes on the insomnia night; associated daytime consequences of daytime fatigue or sleepiness, presence of difficulty concentrating, and impairment of daytime social or professional activities; no hypnotic or psychiatric medications at the time of visit; willingness to undergo a complete sleep clinic evaluation including PSG testing; agreement with the proposed treatment; and signed consent form.

The exclusion criteria were presence of an associated psychiatric diagnosis and ongoing psychiatric treatment; obesity (BMI >28 kg/m^2), untreated medical health problems; daytime sleepiness with impaired driving; oxygen desaturation below 80%; and discovery of another sleep disorder such as periodic limb movement syndrome not related to abnormal breathing during an overnight PSG. Patients taking a scheduled and unchanged dosage of medication for longer than 3 months (e.g., oral contraceptives, antihypertensive medications) were included in the study.

CBT-I was administered in a group setting with an experienced psychologist with board certification in behavioral sleep medicine. Treatment consisted of seven 90-min sessions, over 9 weeks, including standard CBT-I components such as sleep hygiene, stimulus control, relaxation techniques, and sleep restriction.

Polysomnography: The following variables were systematically monitored: electroencephalogram (C3/A2, C4/A1, Fpz/A1-A2, O1/A1), 2 electro-oculograms (right and left eye), chin and leg electromyograms, electrocardiogram (modified V2 lead), and a body position sensor. Respiration was monitored with a nasal cannula pressure transducer, an oral thermistor, a neck microphone, thoracic and abdominal piezoelectric belts, and finger pulse oximetry. The results of the PSG determined the severity of OSA.

A total of 30 subjects matched for age, body mass index, and gender were randomized (stratified randomization) to either CBT-I or surgical treatment of OSA as the initial treatment strategy. CBT-I followed by OSA surgery was defined as treatment sequence A. OSA surgery followed by CBT-I was defined as treatment sequence B. See Table 1 for baseline patient characteristics.

Three months after completing the primary intervention, patients were reassessed with the same instruments used in initial evaluation, including ESS, FSS, and overnight PSG. Except for five patients who refused further evaluation, each patient was then reassigned to the alternative treatment option. Repeat assessments were then performed 3 months after the second intervention (Figure 1).

ANALYSIS

Scoring

Each subject was identified by a number unrelated other identifying information. Anonymous and undated PSGs were rescored by an individual who was unaware of the date and status of the subject to assess scoring consistency following preestablished criteria. Sleep/wake was scored based on 2 international classification systems, including the scoring of short EEG arousals.[14,15] Apnea and hypopnea were scored based on nasal cannula recordings. A drop of ≥30% of the amplitude of the signal ≥3 breaths was required to score a hypopnea, and a drop of at least 80% of the amplitude of the signal to score an apnea. An oxygen desaturation ≥3% or the presence of a visible EEG arousal for ≥3 sec was necessary to score an event. If there was a change in flow at the nasal cannula that did not meet the criteria to score a hypo-

Table 1—Baseline Patient Characteristics (n = 30) [mean (SD) median]

	Sequence A Initial CBT-I n = 15	Sequence B Initial Surgery n = 15	P value
Age (y)	30.9 (5) 31	31.8 (5.4) 30	0.64
Gender	5 men, 10 women	6 men, 9 women	
BMI (kg/m2)	24.1 (2) 23.7	23.9 (2) 23.2	0.81
Neck Circumference (cm)	34.1 (4.4) 33.5	33.7 (4.2) 34	0.83
ESS	6.5 (1.2) 6	6.6 (1.2) 7	0.91
FSS	4.8 (0.4) 5	4.7 (0.8) 5	0.70
Baseline PSG			
TST (min)	373.4 (16.1) 379	374.1 (16) 377	0.91
REM (%)	12.5 (1.6) 12	13.1 (1.8) 12	0.29
SWS (%)	10.9 (1.2) 11	11.5 (1.5) 11	0.24
St 1 (%)	13.9 (1.9) 14	13.2 (2.7) 14	0.40
RDI index (events/h)	21.7 (7.3) 19	21.7 (9) 18	0.98
AHI index (events/h)	7.9 (4.8) 6	9.7 (5.7) 9	0.35
Min. oxygen saturation (%)	91.6 (1.6) 92	91.3 (1.9) 92	0.61
SL (min)	27.3 (8.7) 28	25.8 (10.4) 26	0.67
WASO (min)	39.6 (22.3) 45	45.4 (17.9) 48	0.44

P-value calculated from 2-sample t-test for baseline variables and grouped by sequence.

pnea but was indicative of abnormal breathing as previously reported, it was scored as "flow limitation." At least 4 consecutive breaths were required to score flow limitation (presence/absence of snoring was noted in association with flow limitation). One event of flow limitation was scored until the abnormal pattern at the nasal cannula resolved. Two indices of abnormal breathing during sleep were calculated: apnea-hypopnea index (AHI): number of apneas and hypopneas per hour of sleep, and respiratory disturbance index (RDI): apnea, hypopnea, and number of flow limitation events per hour of sleep.

Statistical Analysis

Data were presented as mean (± SD) and median. After verification of skewness, kurtosis, and determination of distribution, a 2-sample *t*-test was used to compare the 2 groups (CBT-I and OSA treatment) at entry (baseline [BSL]).

Repeated measures ANOVA with treatment as a within-subject factor (BSL-CBT-Surgery) and sequence as a between-subject factor (CBT-Surgery or Surgery-CBT) was used to analyze if there was an effect of treatment sequence on the changes in ESS, FSS, total sleep time (TST), time spent in slow wave sleep (SWS) and REM sleep, RDI, AHI, minimum oxygen saturation (Min O2), sleep latency (SL), and wake time after sleep onset (WASO). When a significant interaction between treatment and sequence was observed, another repeated measures ANOVA was used to analyze the effect of treatment sequence within sequence A and B individually.

Following this, post hoc analysis was done to compare BSL, CBT-I, and surgery. For each variable and treatment, a 2 sample *t*-test was also done to compare sequence A and B (with Bonferroni correction).

The FSS was analyzed as a nonparametric variable because of a lack of variability in FSS data after CBT-I in sequence B. In this case, a Friedman 2-way analysis of variance for each sequence was done, a Wilcoxon signed rank test to compare each treatment (BSL, CBT-I, and surgery) 2 by 2 within each sequence, and a Kruskal-Wallis one-way analysis of variance to analyze the difference of both sequences within each treatment. P values ≤0.05 were considered statistically significant.

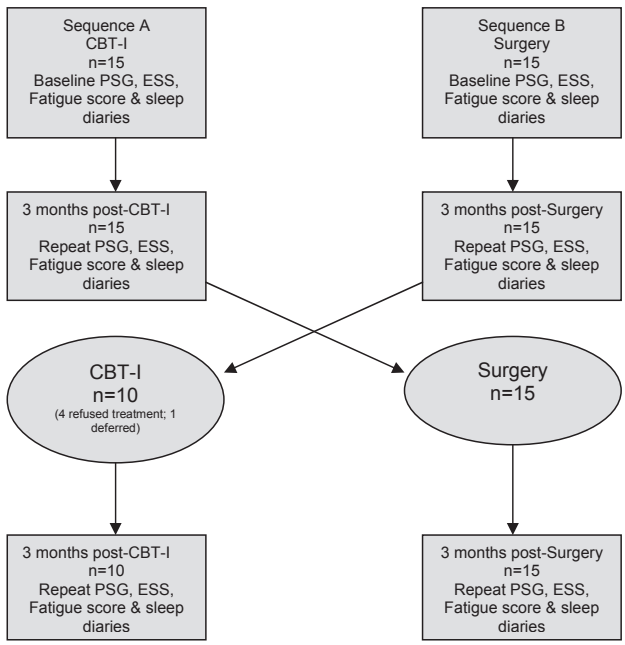

Figure 1—Study Design

RESULTS

The demographics and characteristics of the 30 subjects involved in the study are presented in Table 1. Patients did not differ significantly in any of the considered measures. There were 10 women in the CBT-I group (group A) and 9 women in the OSA treatment (group B) at entry.

Table 2A—Outcomes of Patients Treated With Initial CBT-I Followed by Surgery: Sequence A [mean (SD) median]

	Baseline	After CBT-I	After Surgery	P value
ESS	6.5 (1.2) 6	5.9 (1.6) 6	4.8 (0.8) 5	0.002
FSS	4.8 (0.4) 5	4.5 (1) 4	1.2 (0.4) 1	0.0001
TST (min)	373.4 (16.1) 379	389.8 (7.4) 389	408.7 (7.3) 408	0.0001
REM %	12.5 (1.6) 12	13.1 (1.7) 13	16.2 (1.1) 16	0.0001
SWS %	10.9 (1.2) 11	11.9 (1.4) 12	15.4 (1.1) 15	0.0001
ST 1%	13.9 (1.9) 14	10.8 (2.6) 10	6.2 (1.1) 6	0.0001
RDI (events/h)	21.7 (7.3) 19	21.3 (8.6) 18	6.8 (2.1) 6	0.0001
AHI (events/h)	7.9 (4.8) 6	8.9 (5.7) 7	2.7 (1.2) 2	0.0001
Min. oxygen saturation (%)	91.6 (1.6) 92	91.9 (1.8) 93	96.3 (1.6) 97	0.0001
SL (min)	27.3 (8.7) 28	11.5 (2.1) 12	10.4 (2.6) 10	0.0001
WASO (min)	39.6 (22.3) 45	19.9 (7.9) 20	7.9 (1.9) 8	0.0001

P-value calculated from repeated measures ANOVA for all treatments as a within subject factor (sequence A: BSL-CBT-Surgery).

Table 2B—Effect of Treatment Sequence A on Sleep and Respiratory Variables

Repeated measure ANOVA	P value	Post hoc analysis	P value
TST	0.0001	TST increased after each intervention	0.02
REM sleep	0.0001	REM increased post surgery	0.0001
SWS	0.0001	SWS increased after each intervention	0.02
Stage 1 NREM	0.0001	Stage 1 decreased after each intervention	0.0001
SL	0.0001	Latency decreased after CBT-I	0.0001
WASO	0.0001	WASO decreased after each intervention	0.003
AHI	0.0001	AHI decreased after surgery	0.0001
RDI	0.0001	RDI decreased after surgery	0.0001
Lowest SpO_2	0.0001	Lowest SpO_2 increased after surgery	0.0001

Treatment as a within subject factor (BSL-CBT-Surgery) and sequence as a between subject factor (CBT-Surgery or Surgery-CBT).
SL: sleep latency, WASO: time awake after sleep onset

The post-treatment data presented for these cases are those obtained after surgical treatment, with or without a dental appliance. Only 25 patients underwent CBT-I treatment. The 5 patients that refused CBT-I treatment were all in the initial OSA treatment group. Three months after completion of the first phase of treatment, these 5 subjects (who did not significantly differ in age, BMI, or presenting complaints from the total group at entry) reported disappearance of all insomnia symptoms and declined further treatment.

At entry:

All 30 subjects had similar complaint profiles (see Table 1). There were few complaints of daytime sleepiness, and overall ESS scores were within the accepted normal range. Subjects however did report the presence of daytime fatigue, which was well illustrated by abnormal FSS and inability to take daytime naps despite fatigue. Difficulty on the job due to poor concentration and irritability were mentioned by 29 patients.

When considering nocturnal complaints, 6 subjects complained only of sleep maintenance insomnia, 24 subjects complained of sleep-onset insomnia and difficulty falling asleep from an awakening after sleep onset. Four of the 5 subjects who did not undergo CBT-I treatment after surgery had sleep maintenance insomnia. Four of 6 subjects who did not complain of sleep-onset insomnia were randomized to surgery as a first-line treatment subgroup.

Overall AHIs were low, but RDIs were elevated and correlated with frequent sleep disruptions. The lowest oxygen saturation (SpO_2) was >90% in all cases, except for 2 outliers. Twenty-eight patients had normal BMI and normal abdominal weight distribution, eliminating chest wall restrictive problems during REM sleep as a cause for oxygen desaturation.

Follow-up:

All subjects were included in follow-up evaluation, after the first treatment phase.

a) CBT-I treatment first (A) in treatment sequence

None of the patients who underwent CBT-I treatment felt that their problems were adequately controlled. Data analysis of the effect of treatment sequence A on all variables is presented in Table 2B. In this group (A), there was a disappearance of long sleep latencies at sleep onset or after awakenings after sleep onset, but the complaint of nocturnal sleep disruption and daytime fatigue persisted.

b) OSA treatment first (B) in treatment sequence

Data analysis of the effect of treatment sequence B on all variables is shown in Table 3(A). As mentioned above, 5 pa-

Table 3A—Outcomes of Patients Treated with Initial Surgery Followed by CBT-I: Sequence B [mean (SD) median]

	Baseline	After Surgery	After CBT-I	P value
ESS	6.6 (1.2) 7	4.7 (0.8) 5	4.1 (0.9) 4	0.0001
FSS	4.7 (0.8) 5	1.1 (0.3) 1	1.0 (0) 1	0.0001
TST (min)	374.1 (16) 377	404.9 (7.3) 404	411.4 (9.1) 410	0.0001
REM %	13.1 (1.8) 12	15.9 (0.9) 16	16.6 (16.6) 16.3	0.0001
SWS %	11.5 (1.5) 11	14.5 (1.1) 15	15.4 (2.1) 15	0.0001
ST 1 %	13.2 (2.7) 14	8.1 (1.6) 9	6.2 (1.8) 6	0.0001
RDI (events/h)	21.7 (9) 18	6.4 (1.7) 6	8.1 (5.2) 6.8	0.0001
AHI (events/h)	9.7 (5.7) 9	3.1 (1.3) 3	3.8 (2.5) 3.1	0.0001
Min. oxygen saturation (%)	91.3 (1.9) 92	96.3 (1.4) 97	95.9 (2.2) 97	0.0001
SL (min)	25.8 (10.4) 26	21.6 (9.6) 24	11.4 (2.6) 12	0.003
WASO (min)	45.4 (17.9) 48	8.4 (2.1) 8	6.6 (1.2) 6.5	0.0001

P-value calculated from repeated measures ANOVA for all treatments as a within subject factor (sequence B: BSL-Surgery-CBT). For the FSS, P-value was calculated from nonparametric Friedman 2-way analysis of variance within sequence B.

Table 3B—Analysis of Treatment Sequence B on Sleep and Respiratory Variables

Repeated measure ANOVA	P value	Post hoc analysis	P value
TST	0.000	TST increased after each intervention	0.000
REM sleep	0.000	REM increased each intervention	0.03
SWS	0.000	SWS increased after each intervention	0.003
Stage 1NREM	0.000	Stage1 decreased after each intervention	0.0001
SL	0.003	Latency decreased after CBT-I	0.02
WASO	0.000	WASO decreased after each intervention	0.05
AHI	0.000	AHI decreased after each intervention	0.01
RDI	0.000	RDI decreased after each intervention	0.005
Lowest SpO_2	0.000	Lowest SpO_2 increased after each intervention	0.0001

Treatment as a within subject factor (BSL - Surgery - CBT) and sequence as a between subject factor (Surgery-CBT or CBT-Surgery).
SL: sleep latency, WASO: time awake after sleep onset

tients in the initial OSA treatment group felt that their sleep problems had improved sufficiently to terminate treatment and opted not to undergo the planned CBT-I treatment. The remaining 10 subjects felt that they were not sufficiently improved and underwent CBT-I treatment.

Crossover (Figure 2):

A total of 25 of 30 subjects crossed over and results for sequence A (patients whom underwent CBT-I followed by OSA treatment [n = 15]), and sequence B (patients whom underwent surgical treatment for OSA then underwent CBT-I [n = 10]) are presented in Tables 2 and 3, respectively. The results of the analysis of variance for repeated measures, considering the 3 conditions for both groups, are presented in Tables 2A and 3A.

There were no significant differences for baseline variables when comparing sequences A and B in each variable. There was a significant effect of treatment sequence on the treatments observed for all variables in repeated measures ANOVA for both sequences combined (tx*sequence, $P \leq 0.02$).

At Exit:

In both A and B, an effect of sequence was noted as shown in Tables 2(B) and 3(B). This was also observed in both sequences for ESS scores (repeated measures ANOVA, $P = 0.002$). A lower ESS score was noted after OSA surgery in sequence A (post hoc, $P \leq 0.05$), while it was noted after both OSA surgery and CBT-I in sequence B (post hoc, $P \leq 0.005$). Nonparametric Friedman 2-way analysis of variance also showed an effect of sequence on fatigue ($P < 0.001$) in both sequences A and B. Nonparametric Wilcoxon signed ranks showed that fatigue was significantly decreased after surgery in sequence A ($P = 0.001$) and after both surgery and CBT-I in sequence B ($P \leq 0.005$).

Table 4—Surgical Treatment and Dental Appliances

	n
Pharyngoplasty	14
Pharyngoplasty and nasal surgery	12
Pharyngoplasty and dental appliance	2
Pharyngoplasty, nasal surgery, and dental appliance	1
Pharyngoplasty, nasal surgery, and geniotubercle advancement	1

Pharyngoplasty comprises of tonsillectomy with palatal lateral wall surgery, uvulectomy is not included.
Nasal surgery includes septoplasty with or without radiofrequency turbinate reduction

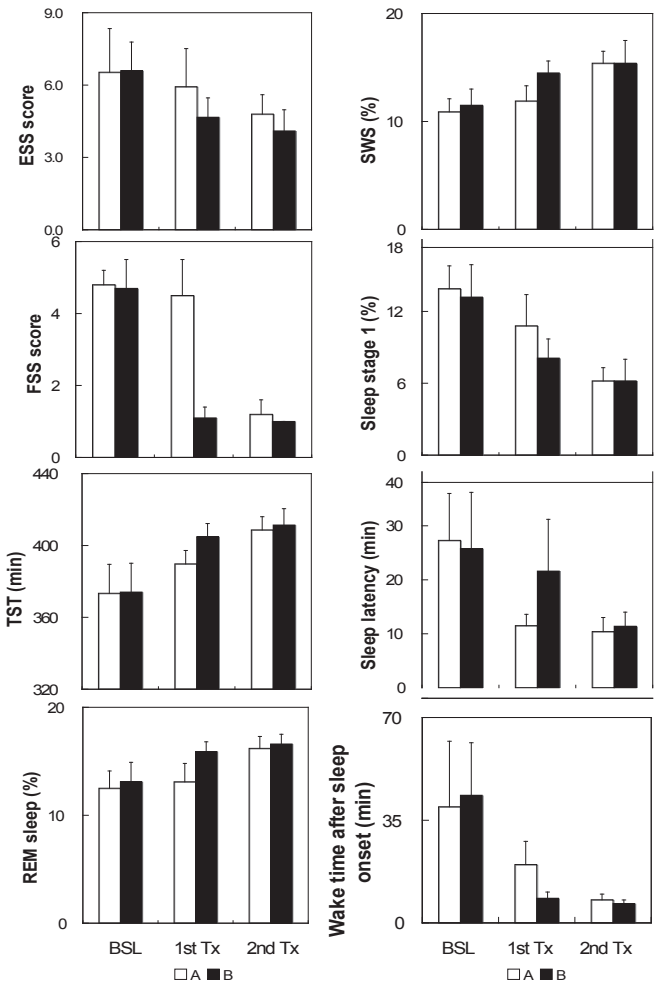

Figure 2—Qualitative and quantitative sleep variables for Baseline and both sequences A and B (mean [SD]). Sequence A (white bar) is CBT-I as first treatment and OSA surgery as second treatment. Sequence B (black bar) is OSA surgery as first treatment and CBT-I as second treatment. When comparing sequences A to B for each variable, TST, REM, SWS, RDI, MinO2, and WASO are mainly improved by surgery independently of sequence. CBT-I done after surgery (sequence B, P=0.01) is the only improved sequence. The sleep stage 1 distribution after CBT-I is lower in sequence B (P = 0.001), while higher after surgery in sequence B (P = 0.03), meaning effects are independent of sequence. There is no significant difference for the AHI index after either treatments in both sequences. SL is mainly improved by CBT-I independently of sequence. This is further supported by being most improved after surgery done after CBT-I (sequence A, P = 0.02).

Additional post hoc analysis for sleep and respiratory variables is presented in Tables 2B and 3B.

ESS and FSS scores improved after surgery, regardless of sequence. Furthermore, ESS and FSS scores were significantly improved when CBT-I was performed after surgery (sequence B). When comparing sequence A to sequence B for each variable, ESS after CBT-I was lower in sequence B (P = 0.04), while no difference was noted after surgery. Nonparametric Kruskal-Wallis one-way analysis of variance showed significantly lower FSS scores after CBT-I in sequence B (P = 0.000), while there was no significant difference after surgery between sequences.

Significantly shorter sleep latency was noted only after CBT-I, independent of treatment sequence (P < 0.02). TST improved independently of the treatment sequence (Figure 2). All other variables were significantly lowered after surgery. When comparing CBT-I variables between sequence A and B with 2-sample *t*-tests, REM, SWS, and WASO were improved significantly after surgery but not after CBT-I. However, there were initial changes in stage 1 NREM sleep after CBT-I, which showed additional significant improvement after surgery (P = 0.0001) (Figure 2).

DISCUSSION

Our patients were a diagnostic challenge for primary care physicians.

All of our patients had previously been evaluated by private practitioners, and all subjects have been classified as "insomniacs." All of them experienced either sleep onset insomnia or had a component of sleep maintenance insomnia and prolonged periods of wake after sleep onset. At one time or another, these patients were prescribed hypnotic medications without discussion of the possibility of an associated sleep disorder. The presentation of these subjects did not fit the typical profile for patients with SDB, in that they were not obese, they did not experience significant snoring, and complaints of fatigue and difficulty falling asleep were not those of the "prototypical" sleep apnea patient with excessive daytime sleepiness. But attention should be given to all findings when considering SDB, as the sleep and breathing disturbances visually noted on the polysomnogram may be more important than the AHI number and/or the strict scoring criteria for OSA.[16,17] When OSA is diagnosed, especially when it is mild, it is often not treated. However, from our study the complaint of "daytime fatigue" seemed to be more related to the breathing problem during sleep than to the report of insomnia as it persisted even after CBT-I treatment but was eliminated with OSA surgical treatment.

Treatment of the breathing problem alone was successful in eliminating the complaint of insomnia in only 5 of 15 subjects in our study, but many of the sleep disturbances noted at polysomnography were normalized by SDB treatment (Table 4). CBT-I treatment did not improve sleep variables but was significantly helpful in addressing the behavioral component of conditioning and other residual sleep disturbances. Both treatments were needed to normalize sleep and associated complaints in the majority of subjects

Complaints of insomnia are often treated symptomatically; one may question if improvement in complaints of insomnia with CBT-I could also have been obtained with pharmacological agents, along with treatment of SDB. We believe that pharmacologic treatment would probably achieve short-term improvements. However studies indicate that CBT-I results in a longer beneficial role for the non-drug approach. Recurrence of insomnia may not have been prevented with withdrawal from the pharmacological agent.

Our study raises the question of the consequences of "comorbid insomnia."[18] The association of poor quality sleep with other sleep disorders is receiving increasing recognition;[19] mood changes are also seen with poor sleep. Our study clearly shows that poor sleep is multifactorial, and a one-step treatment

approach, such as use of hypnotics or antidepressants, will not completely address the problem.

Chronic insomnia represents a significant cost to the patient, families, and payers of the health system organization. Insomnia has been linked to adverse medical and psychological conditions, as well as socioeconomic consequences of lost productivity and risk of accidents. Recognition of all factors involved in this nonspecific complaint, including naso-maxillo-mandibular anatomical findings on physical exam that may suggest mild SDB, is a crucial step.

Treating insomnia and OSA together is clearly the best solution in this co-morbid insomnia, and as shown in our study successful outcome can be achieved. Results can be maximized when mild OSA is treated first with surgery or with another targeted treatment approach for OSA prior to initiation of CBT-I.

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DISCLOSURE STATEMENT

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