Effect of Sleep Disordered Breathing on the Sleep of Bed Partners in the Sleep Heart Health Study

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Objective: To study the sleep quality of bed partners of persons with sleep disordered breathing in a non-clinical population based sample in a home environment.

Design: Cross-sectional study in a community sample.

Methods: 110 pairs of subjects living in the same household from the Tucson, Minnesota, and Pittsburgh sites of the Sleep Heart Health Study (SHHS) were included if both partners had an in-home, unattended polysomnogram (PSG) performed as a part of SHHS exam cycle 2. Sleep disordered breathing (SDB) was considered present if the respiratory disturbance index (RDI) was ≥10 events/h and no SDB if RDI was <5 events/h. Pairs were classified according to their SDB status and assigned to one of 3 groups: 1) NoSDB-NoSDB (n = 46), 2) NoSDB-SDB (n = 42), and 3) SDB-SDB (n = 22).

Results: There were no differences between the NoSDB-NoSDB and the SDB-SDB partners in their demographic, PSG, or quality of life variables. However, within the NoSDB-SDB group, NoSDB in comparison to their SDB partners weighed less (mean BMI: 26 vs. 29 kg/m^2, P < 0.0003), had decreased stage 2% (55 vs. 64, P < 0.0001), increased stage 3 and 4% (21 vs. 11, P < 0.0005) and a lower arousal index (13.8 vs. 20 events/h, P < 0.0001). When comparing the NoSDB subjects from the NoSDB-SDB group to subjects in the NoSDB-NoSDB group and to subjects in the SDB-SDB group, significant differences were seen for RDI and BMI but not for any other parameter.

Conclusion: In a non-clinical population based sample, the sleep quality of bed partners of SDB subjects without SDB is better than their SDB bed partner. However, their sleep quality was not different in comparison to the sleep of those without SDB who also had a bed partner without SDB.

Keywords: Obstructive sleep apnea, quality of life, adverse effects, sleep architecture

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SLEEP DISORDERED BREATHING (SDB) IS COMMON, REPORTED IN 2% TO 4% OF MIDDLE-AGED PERSONS.[1] THESE INDIVIDUALS FREQUENTLY PRESENT WITH complaints of excessive daytime sleepiness, loud snoring, witnessed apnea and gasping for air.[2,3] Empiric observations indicate that bed partners of SDB patients frequently complain that snoring, breathing pauses, gasping for air and excessive movement disrupt their own sleep. Thus, it would be expected that bed partners of SDB patients experience both poor quality sleep and reduced quality of life.

With respect to sleep quality, there have been relatively few studies addressing this issue in bed partners of persons with SDB. Two studies used cross-sectional questionnaire data obtained from bed partners of SDB patients.[4,5] However, there were no comparisons to a control group of non SDB patients' bed partners. In the only study with such a control group, snoring was used as a surrogate for SDB.[6] Nevertheless, all of these studies generally found that the sleep of bed partners was disturbed. Others have observed that the subjective sleep quality of bed partners improves after treatment of the SDB partner with nasal continuous positive airway pressure (CPAP).[7,8] However, data from studies using polysomnography (PSG) as an objective indicator of sleep quality are conflicting. In a small group of patients with suspected SDB, couples underwent simultaneous split-night polysomnography.[9] Bed partner's sleep efficiency was reduced and their arousal index elevated, but both improved after elimination of SDB and snoring with CPAP.[9] In contrast, another study reported no changes in bed partner's PSG indices before and after their SDB partner was treated with CPAP for 1 month.[8]

Although persons with SDB have a reduction in quality of life when this is assessed using validated instruments,[10,11] which improves after treatment of the SDB,[12] this issue has not been fully examined in bed partners of persons with SDB. In an early retrospective study using an unvalidated instrument, bed partner quality of life increased after 2-12 months of CPAP therapy for the SDB patient.[7] Subsequent studies using validated instruments confirmed these initial findings.[13,14,8] However, in one of these studies, baseline quality of life for bed partners was not found to be significantly different from national norms and not all quality of life domains improved.[14]

From the studies that have been published heretofore,[4-9,13,14] it would appear that both subjective sleep quality and quality of life are impaired in bed partners of SDB patients, but some issues remain to be clarified. First, except for the study by Ulfberg and colleagues that used snoring as a surrogate for SDB,[6] all previous studies have used bed partners of SDB persons identified from a clinic population. It remains uncertain whether bed partners of SDB affected individuals who have not sought treatment have impairment in either their sleep or quality of life. Because most SDB remains undiagnosed and untreated,[15] this

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is potentially an important problem for cohabitating couples. Second, there is conflicting data as to whether objective indices of sleep quality in bed partners of SDB persons are affected by their bed partner with SDB. Third, there have been no comparisons of the sleep of SDB bed partners to the sleep of persons who do not have SDB bed partners. Thus, the purpose of this study was to examine the sleep of bed partners in a subsample of the Sleep Heart Health Study, a large community based cohort. Our primary hypothesis was that the sleep and quality of life of bed partners of those with SDB, but who themselves did not have SDB, would be impaired in comparison to the sleep of similar bed partners of those without SDB.

METHODS

The Sleep Heart Health Study (SHHS) is a multicenter cohort study designed to investigate the relationship between SDB and development of cardiovascular diseases. Details of the study design have been published elsewhere.[16] Briefly, 6,441 men and women over 40 years of age were recruited from the following studies: the Offspring Cohort and the Omni Cohort of the Framingham Heart Study; the Hagerstown, MD, and Minneapolis, MN, sites of the Atherosclerosis Risk in Communities Study; the Hagerstown, MD, Pittsburgh, PA, and Sacramento, CA, sites of the Cardiovascular Health Study; 3 hypertension cohorts (Clinic, Worksite, and Menopause) in New York City; the Tucson Epidemiologic Study of Airways Obstructive Diseases and the Health and Environment Study; and the Strong Heart Study (SHS) of American Indians in Oklahoma, Arizona, North Dakota, and South Dakota. A 5-year SHHS follow-up survey enrolled 3,079 of the original participants. As in the baseline study, subjects were recruited to undergo an overnight home polysomnogram, completion of several questionnaires, and collection of physical examination data. All participants provided informed consent for this study, which was approved by the institutional review board at all of the sites.

Participants and data for this study were identified from the second SHHS evaluation which included administration of the SHHS Sleep Habits Questionnaire (SHQ) and the Morning Survey (MS).[16] All SHHS field centers were queried to determine whether they had a record of participants who were sleeping in the same household. Three field centers, Tucson, Minnesota, and Pittsburgh had this information. Participants were included in this analysis if they answered "1" to the following 2 questions in the MS:

1. What is your usual sleeping arrangement? 1) another person in same bed; 2) another person in same room but different bed; 3) alone in the room.
2. What was your sleeping arrangement last night? 1) another person in the same bed; 2) another person in the same room but different bed; 3) alone in room.

A total of 110 bed partners from the Tucson (53), Minnesota (56), and Pittsburgh (1) sites were thus identified. Twelve of these couples slept in the same bed but were studied on separate nights. All other couples had been studied on the same night. We divided these couples into 3 groups as follows: (1) Neither person had SDB (NoSDB-NoSDB, 46 couples); (2) 1 person with SDB and 1 person without SDB (NoSDB-SDB, 42 couples); and (3) Both persons with SDB (SDB-SDB, 22 couples). SDB was defined as a respiratory disturbance index (RDI) ≥10 events/h with ≥4% associated oxygen desaturation. No SDB was defined as having an RDI 4% of <5 events/h (*vide infra*). An additional 67 couples with at least one bed partner having a RDI ≥5, but less than 10 were excluded to minimize the likelihood of misclassifying a couple's SDB status.

Polysomnography

Overnight PSG was performed using the Compumedics Portable PS-2 system (Abottsville, AU), as previously described.[17] Sensors were placed and equipment was calibrated during an evening visit by a certified technician. Data collection included C4/A1 and C3/A2 electroencephalograms (EEG); right and left electrooculograms (EOGs); a bipolar submental electromyogram (EMG); thoracic and abdominal excursions (inductive plethysmography bands); "airflow" (detected by a nasal-oral thermocouple, Protec, Woodinville WA), oximetry (finger pulse oximetry [Nonin, Minneapolis, MN]), electrocardiogram (ECG) and heart rate (using a bipolar ECG lead); body position (using a mercury gauge sensor); and ambient light (on/off, by a light sensor secured to the recording garment). Following equipment retrieval, the data, stored in real time on PCMCIA cards, were downloaded to the computers at each respective clinical site, locally reviewed, and forwarded to a central reading center (CWRU, Cleveland, OH). Sleep stages were scored according to the guidelines developed by Rechtschaffen and Kales,[18] with the exception that Stages 3 and 4 were combined.

The RDI was calculated as the number of apneas plus hypopneas per hour of total sleep time. An apnea was defined as a complete or almost complete cessation of airflow (at least ≤25% of baseline), as measured by the amplitude of the thermocouple signal, lasting ≥10 sec. Hypopneas were identified if the amplitude of a measure of flow or volume (detected by the thermocouple, thorax, or abdominal inductance band signals) decreased to ≤70% of the amplitude of "baseline" breathing for ≥10 sec, but did not meet the criteria for apnea. Only apneas or hypopneas associated with ≥4% oxygen desaturation were considered in the calculation of the RDI. Arousals were identified according to American Sleep Disorders Association (American Academy of Sleep Medicine) criteria.[19]

Non-Polysomnographic Data

The SHQ contained questions regarding sleep habits. The habitual total sleep time and habitual sleep onset latency during the weekdays and weekends were derived from specific questions on the SHQ. Weekend or weekday sleep scores were used respectively according to whether the PSG was performed on a weekend or weekday. Height and weight were measured directly and body mass index (BMI) determined. Based upon answers provided on the SHQ, subjects were classified as snorers only if they snored ≥3 nights a week and reported that their snoring was louder than talking or extremely loud (could be heard through a closed door). Data for ethnicity, gender, and education were derived from data obtained from the SHHS parent cohorts and information available from the first SHHS examination. Ethnicity included 96% Caucasian, 1% African American, and 3% Hispanic, and therefore ethnicity was di-

Table 1—Median Values for Polysomnographic, ESS, and SF-36 Variables Comparing the No-SDB Partners in Group 1

	NoSDB-NoSDB Group				
	Partner A		Partner B		
	No-SDB Partner	Min-Max	No-SDB partner	Min-Max	P-value*
N	46		46		
Age (years)	61	45–80	62	46–80	0.70
BMI	26	19–39	27	21–43	0.70
RDI 4%	1.3	0–4.9	1.8	0.1–4.9	0.62
Sleep onset latency	12	0–102	17	0–76	0.90
Total sleep time	384	239–495	383	267–460	0.53
Sleep efficiency	84	60–96	86	58–99	0.25
Stage 1 %	5	1–15	4	1–14	0.53
Stage 2 %	54	23–70	57	29–76	0.53
Stage 3 and 4 %	17	1–53	16	0.3–42	0.86
REM%	23	9–30	22.6	5–41	0.40
Arousal index	13	5–34	12	5–29	0.41
Habitual SOL (min)	15	2–60	15	3–150	0.34
Habitual TST (min)	465	195–540	420	315–540	0.34
ESS	8	0–14	6	1–17	0.28
Physical functioning	50	28–50	50	10–50	0.68
Bodily pain	74	32–100	84	22–100	0.30
General health	77	47–100	80	27–100	0.46
Vitality	65	30–100	70	0–100	0.25
Social functioning	100	50–100	100	25–100	0.81
Role-emotional	100	0–100	100	0–100	0.37
Mental health	84	40–100	88	40–100	0.49

*P-value for Wilcoxon matched-pairs signed-ranks test. ESS (Epworth Sleepiness Scale).

chotomized into Caucasians and other ethnic groups. Years of education were categorized into high school (<12 years), college (12–16 years), and graduate school (>16 years). Participants were classified as having chronic lung diseases if they answered yes to having a physician telling them they had chronic bronchitis, emphysema, chronic obstructive pulmonary disease, or asthma. Chronic heart disease was classified as being present if a participant was ever told by a physician that they had any of the following: stroke, angina, heart attack, or heart failure; or ever having had any of the following procedures: coronary angioplasty, coronary bypass surgery, insertion of a pacemaker, or any other heart or cardiac surgery.

Sleepiness and Quality of Life Measures

The Epworth Sleepiness Scale (ESS) is a validated 8-item questionnaire that measures subjective sleepiness.[20] Subjects are asked to rate how likely they are to fall asleep in different situations. Every question is answered on a scale of zero to 3. ESS values range from zero (unlikely to fall a sleep in any situation) to 24 (high chance of falling sleep in all 8 situations). The SF-36 is a validated questionnaire that measures quality of life (QoL).[21] The SF-36 addresses questions related to general health; however, it does not assess the direct effect of sleep problems on health. The SF-36 contains 8 dimensions of health status, including: 1) physical activities; 2) social activities; 3) physical health problems; 4) bodily pain; 5) general mental health; 6) emotional problems; 7) vitality; 8) general health perception. Scores are calculated using an SF-36 specific standardized scoring algorithm.

Statistical Analysis

For analysis purposes, one bed partner in each pair was designated partner "A" and the other partner "B". For the NoSDB-NoSDB and the SDB-SDB groups, assignments as either partner "A" or partner "B" were done randomly. For the NoSDB-SDB group, partner "A" was the member of each pair without SDB and partner "B: was the member with SDB.

Differences in proportion were assessed using the χ^2 test for categorical variables, i.e., gender, ethnicity, education, and chronic lung and heart diseases. As data were not normally distributed, the Wilcoxon matched-pairs sign-ranks sum test was used to compare differences between the NoSDB-NoSDB, NoSDB-SDB, and SDB-SDB partners for continuous variables, i.e., age, BMI, sleep, ESS, and QoL variables. The Kruskal-Wallis test was used to assess differences in group medians among the NoSDB bed partners from the NoSDB-NoSDB and NoSDB-SDB groups, and SDB bed partners in the SDB-SDB group. The Wilcoxon rank-sum (Mann-Whitney) test was used to assess pairwise differences between the A partners with a significance of 0.0167 used to adjust for multiple comparisons.

RESULTS

No significant differences were found comparing respective bed partners in the NoSDB-NoSDB and the SDB-SDB groups for the various sleep, respiratory, and QoL variables analyzed (Tables 1 and 3). Median values for polysomnographic and other variables for the NoSDB-SDB partners are presented in Table 2. Compared to their SDB partners, NoSDB subjects tended to be younger (median age 63 vs 66 y, $P < 0.0001$), had significantly

Table 2—Median Values for Polysomnographic, ESS, and SF-36 Variables Comparing the No-SDB and SDB Partners in Group 2

	NoSDB-SDB Group				
	Partner A		Partner B		
	No-SDB Partner	Min-Max	SDB partner	Min-Max	P-value*
N	42		42		
Age (years)	63	47–74	66	48–77	<0.0001
BMI	26	22–34	29	15–42	0.0003
RDI 4%	1.8	0–4.9	17.9	10–68	<0.0001
Sleep onset latency	14	0–53	16	1–69	0.29
Total sleep time	400	333–456	365	260–456	0.25
Sleep efficiency	82	57–95	82	57–95	0.24
Stage 1 %	4	1–10	5	1–17	0.46
Stage 2 %	55	30–71	64	35–86	0.0001
Stage 3 and 4 %	21	5–47	11	0–42	0.0005
REM%	20.3	8–35	20	5–35	0.22
Arousal index	13.8	5–27	20	5–60	<0.0001
Habitual SOL (min)	15	4–120	15	2–90	0.61
Habitual TST (min)	420	300–600	450	300–540	0.94
ESS	6	0–15	8	2–19	0.09
Physical functioning	50	28–50	50	6–50	0.27
Bodily pain	84	0–100	74	22–100	0.57
General health	82	20–100	77	30–100	0.90
Vitality	73	45–90	75	20–90	0.61
Social functioning	100	25–100	100	50–100	0.50
Role-emotional	100	0–100	100	0–100	0.75
Mental health	84	32–100	84	68–100	0.68

*P-value for Wilcoxon matched-pairs signed-ranks test. ESS (Epworth Sleepiness Scale).

lower BMI (median 26 vs 29 kg/m^2, P = 0.0003), percentage of stage 2 sleep (55% vs 64%, P = 0.0001), and arousal index (13.8 vs 20 events/h, P = 0.0001). The NoSDB partners also had an increased percentage of stage 3 and 4 sleep and higher sleep efficiency (21% vs 11%, P = 0.0005). However, no significant differences were seen between the NoSDB and SDB partners for any of the QoL measures.

Table 4 shows the demographic and health information for the "A" partners in all 3 groups. A higher percent of female subjects (83%) was seen in the NoSDB-SDB group and in the SDB-SDB groups (68%) in comparison to the NoSDB-NoSDB group. There were no significant differences for ethnicity, education, and chronic lung or heart diseases for the "A" partners in all 3 groups. Although it did not quite achieve statistical significance, it is notable that only 17% of "A" partners in the SDB-SDB group endorsed loud snoring. This is similar to the 16% of "A" partners in the NoSDB-NoSDB group and in contrast to the 37% of "A" partners in the NoSDB-SDB group.

Comparisons were made to determine if polysomnographic, sleep, and QoL variables of NoSDB bed partners in the NoSDB-SDB group differed from NoSDB and SDB bed partners in the NoSDB-NoSDB and SDB-SDB groups (Table 5). Age and BMI was higher for the "A" partners in the SDB-SDB group compared to those in the NoSDB-NoSDB and NoSDB-SDB groups. Predictably, the RDI was higher for the SDB partners in the SDB-SDB group compared to the NoSDB partners in the NoSDB-NoSDB and NoSDB-SDB groups. Arousal index was higher for the SDB partners in the SDB-SDB group compared to NoSDB partners in the other groups. However, no significant difference was found for arousal index between the NoSDB partners in the NoSDB-NoSDB group and the NoSDB partners in the NoSDB-SDB group (P = 0.40). Similarly, except for slightly better physical function in NoSDB partners in the NoSDB-SDB group in comparison to the SDB-SDB group, no significant differences were seen among the A partners for any of the other QoL measures. Finally, there were no significant differences for habitually reported total sleep time or sleep onset latency for any of the comparisons. Analyses using all participants in the NoSDB-NoSDB group instead of just "A" partners found similar results to those observed with only "A" partners. Furthermore, analyses using "B" partners in the NoSDB-NoSDB group also were not different than those found with "A" partners.

The influence of one partner's snoring on the other partner's sleep architecture and QoL was also evaluated. In all 3 groups combined, 40% snored. No significant differences were seen within any of the groups for any sleep or QoL measurements when medians were compared between subjects with bed partners who snored and those with bed partners who did not snore.

DISCUSSION

Assessments of bed partner's sleep and quality of life have been largely ignored in the general population. In our analysis of data from three sites of SHHS, we found no differences in the sleep or quality of life between bed partners when the SDB status of both bed partners was the same. However when one bed partner had SDB and the other did not, the bed partner without SDB had better sleep quality although quality of life was not different. In contrast, comparison of sleep architecture between bed partners without SDB sleeping with SDB partners to those

Table 3—Median Values for Polysomnographic, ESS, and SF-36 Variables Comparing the SDB Partners in Group 3

	SDB-SDB Group				
	Partner A		Partner B		
	SDB Partner	Min-Max	SDB partner	Min-Max	P-value*
N	22		22		
Age (years)	65	51–84	70	53–81	0.19
BMI	33	22–50	30	24–53	0.16
RDI 4%	18	11–75	23	11–65	0.43
Sleep onset latency	12	6–80	14	6–152	0.92
Total sleep time	385	221–427	388	291–427	0.51
Sleep efficiency	81	63–92	83	61–94	0.78
Stage 1 %	5	2–18	6	1–16	0.24
Stage 2 %	60	38–80	64	35–86	0.80
Stage 3 and 4 %	14	0.3–47	14	1–51	0.83
REM%	21	7–35	22	11–34	0.94
Arousal index	19	6–54	25.1	8–48	0.43
Habitual SOL (min)	15	2–45	15	0–75	0.81
Habitual TST (min)	480	300–720	480	240–600	0.96
ESS	7	1–19	8	3–15	0.90
Physical functioning	44	17–50	50	28–50	0.53
Bodily pain	72	31–100	79	31–100	0.25
General health	77	32–100	72	20–100	0.90
Vitality	65	25–90	57.5	5–85	0.39
Social functioning	100	50–100	100	38–100	0.26
Role-emotional	100	0–100	100	0–100	0.52
Mental health	84	56–96	88	44–96	0.85

*P-value for Wilcoxon matched-pairs signed-ranks test. ESS (Epworth Sleepiness Scale).

sleeping with NoSDB partners failed to demonstrate that having a SDB partner was associated with worse sleep quality.

In our study, SDB bed partners of NoSDB subjects had worse sleep efficiency, increased arousals, and a greater percentage of lighter sleep. In general, sleep architecture and sleep quality in persons with SDB is characterized by lower sleep efficiency, more arousals, and less stage 3 and 4 sleep.[22] Thus, our finding that the sleep of persons with SDB is worse than their bed partner without SDB is to be expected and provides additional validation of the adverse impact of SDB on sleep architecture. However, consistent with the observations of McArdle et al,[8] we did not find evidence that the sleep of bed partners of those with SDB was impaired in comparison to bed partners of those without SDB. In contrast, Beninati et al, found a reduction in sleep efficiency and a higher arousal index in bed partners of those with SDB after treatment with CPAP.[9] Patients often are referred to sleep clinic because their bed partners complain of sleep disruption due to snoring. Body movements, periodic limb movements, and choking and gasping for air are other factors that could disrupt the sleep of bed partners of persons with SDB. Young et al reported 67% of the males and 52% of the females in their population snored.[1] Repeated noise stimuli in healthy volunteers can cause sleep fragmentation and impair daytime function.[23] Analysis of questionnaire data from a random sample in Sweden found loud snoring caused bed partner's insomnia, daytime sleepiness, and headache.[6] Comparison between subjective and objective measurement of snoring before and after uvulopalatopharyngoplasty showed significant improvement in subjective perception of snoring, bed partner's sleep, and quality of life.[24] Given this previous data, it is unclear why we failed to find that the sleep of bed partners of persons with SDB is worse than the sleep of bed partners of persons without SDB. However, it is likely that this finding in part relates to the recruitment of subjects with symptomatic SDB from a clinical population in previous studies versus our use of a community-based population not actively seeking medical attention and not necessarily symptomatic. The snoring and other nocturnal behavior in persons with SDB recruited from the community may not be sufficiently disturbing to sleep to have a negative impact on their bed partner's sleep architecture and quality.

We found no evidence that NoSDB subjects with SDB bed partners had worse quality of life or more insomnia than those with NoSDB bed partners. Extensive data is available on the effects of nasal CPAP on sleep and quality of life in OSA populations.[12] Only a few studies have addressed these issues in bed partners of an OSA population.[7-9,14] In one of these studies, a questionnaire survey was performed in bed partners of OSA patients after CPAP treatment.[7] Bed partners noted improvement in their sleep quality, daytime symptoms, and personal relationships.[7] However, this was a retrospective study and bed partners were asked to compare their past experience with current experience. In another study, Parish et al. did a prospective study in their sleep clinic population, enrolling 54 bed partners of SDB patients with an average apnea-hypopnea index of 48. Six weeks of CPAP treatment in the OSA patients resulted in improvement in the SF-36 scales of role-physical, social functioning, mental health, and social functioning, and the ESS in their bed partners.[14] However, a comparison of the baseline QoL of bed partners with national norms found no significant differences.[14] The discrepancy between our observations and those of others may be explained again by the recruitment of symptomatic patients and their bed partners in the previous

Table 4—Demographic Distribution and Health Problems for A Partners in All Three Groups

	No-SDB partner of No-SDB % (N)	No-SDB partner of SDB % (N)	SDB partner of SDB % (N)	P-value*
N	46	42	22	
Gender				
Female	48 (22)	83 (35)	68 (15)	
Male	52 (24)	17 (7)	32 (7)	0.002
Ethnicity				
White	100 (46)	95 (40)	95 (21)	
Others	0	5 (2)	5 (1)	0.33
Education				
HS/College	48 (22)	62 (26)	77 (17)	
Graduate	52 (24)	38 (16)	23 (5)	0.06
Chronic Lung Disease				
No	87 (40)	93 (39)	91 (20)	
Yes	13 (6)	7 (3)	9 (2)	0.65
Heart Disease				
No	93 (41)	87 (36)	96 (21)	
Yes	7 (3)	12 (5)	5 (1)	0.51
Snores†				
≤2 nights a week	54 (21)	37 (15)	56 (10)	
≥3 nights a week	46 (18)	63 (26)	44 (8)	0.22
Snores extremely loud‡				
No	84 (32)	63 (24)	83 (15)	
Yes	16 (6)	37 (14)	17 (3)	0.07

*P-value for Chi-square test, †n = 98, ‡n = 94

studies in comparison to relatively asymptomatic couples from the community in our study.

We did not find any differences between groups related to snoring. Snoring is often used as a surrogate for SDB. However, it is an imperfect marker. Endorsement is quite subjective and usually relates to a complaint from the bed partner, and not the person who snores. Nevertheless, we did observe a tendency for SDB bed partners of persons with SDB to snore less than noSDB bed partners of persons with SDB. This suggests that SDB-SDB couples either adapt to snoring better or are self selected by being inherently more tolerant to snoring.

There are limitations to this study. First, a few couples (n = 12) were not studied on the same night. However, repeated analyses excluding these couples did not significantly affect our results. Moreover, we believe that the sleep of the recorded partner potentially would still have been affected by their non-recorded partner. Second, periodic leg movements (PLMs) were not measured. Bed partners' sleep disturbance produced by primary PLMs or secondary to SDB was not assessed. However, we would have still expected that bed partner sleep disruption from leg movements related to SDB to be manifested by differences in sleep architecture or the arousal index, Third, there was a greater percentage of females in the NoSDB-SDB and the SDB-SDB groups. It is possible that women were less disrupted by their bed partners SDB, thus minimizing any possible differences with those in the NoSDB-NoSDB group. This seems unlikely because empiric observations suggest that women more frequently complain of their partners' disruptive sleep. Fourth, it is possible that we had insufficient number of couples to detect relatively small differences in the various parameters of sleep architecture. For example, in the NoSDB-SDB group, a post hoc analysis showed 19% power to detect a 15-min difference in Habitual TST at $\alpha = 0.05$. However, if this difference had been 40 min, we would have had 85% power. Fifth, SHHS used a thermistor and not a nasal pressure signal to detect airflow. Thus, it is possible that some participants who were classified as NoSDB may have been misclassified due to the presence of subtle SDB events. We think that this is unlikely because a threshold RDI ≤ 5 was used to define NoSDB and ≥ 10 to identify SDB. Furthermore, hypopneas were identified by using changes in thoracic and abdominal excursions as well as reduction in flow amplitude. Sixth, although the highest RDI observed in these participants was 68, there were relatively few subjects with severe SDB in the sample. In contrast to previous studies using subjects recruited from sleep clinics who generally had a high RDI, the median RDI of the SDB participants in the NoSDB-SDB (17.9) and the SDB-SDB (23) groups reflected only a mild to moderate severity of SDB. Thus, integration of our results with other studies from clinic-based populations suggests that bed partner sleep disruption may only become apparent when the other member of the couple has severe SDB. Consistent with this hypothesis, it is possible that couples in whom one member's sleep is disrupted by the other's severe SDB have already been evaluated and treated, and thus not eligible for our analysis. Last, and perhaps most importantly, SHHS is a population-based sample and not necessarily representative of a clinic population. Although SHHS is a longitudinal cohort study, participants were not recruited randomly from the general population. Rather, they were volunteers who were active participants in other established cohort studies.[16,25] Thus, while SHHS is a community derived cohort, it is not representative of an adult (>40 years old) general population of the United States. Nevertheless, participants were free-living persons not actively being treated or seeking treatment for SDB and consequently may be distinct from symptomatic persons with SDB recruited from a clinical population.

We believe our finding that quality of life and objectively assessed sleep quality in bed partners of those with SDB were not impaired in this population has important implications for clinicians. It is generally acknowledged that SDB is underrecognized.[15] In part this may be related to the absence of symptoms in many persons with SDB.[26] Thus, our finding that the sleep and quality of life of bed partners of persons with SDB in the community also are not impaired provides another reason why such persons may not seek medical attention. Clinicians need to be cognizant that absence of a bed partner's complaint of sleep disruption should not necessarily be taken as evidence against the presence of SDB in the patient.

In summary, in contrast to previous studies performed in clinical populations, in a community-based population sample, bed partners without SDB of SDB persons do not appear to have impairment in their sleep quality or quality of life. Future studies are needed to identify which factors predict bed partner sleep disruption and impairment of quality of life related to the presence of SDB.

Table 5—Comparison of Median Values for Polysomnographic, ESS, and SF-36 Variables for A Partners in All Three Groups

	Group 1		Group 2		Group 3		
	No-SDB partner of No-SDB	Min-Max	No-SDB partner of SDB	Min-Max	SDB partner of SDB	Min-Max	P-value*
N	46		42		22		
Age (years)	61	45–80	63	47–74	65	51–84	0.14
BMI	26†	19–39	26	22–34	33	22–50	0.0001
RDI 4%	1.3†	0–4.9	1.8	0–4.9	18	11–75	0.0001
Sleep onset lat.	12	0–102	14	0– 53	12	6–80	0.91
Total sleep time	384	239–495	400	333–456	385	221–427	0.14
Sleep efficiency	84	60–96	82	57–95	81	63–92	0.16
Stage 1 %	5	1–15	4	1–10	5	2–18	0.46
Stage 2 %	54	23–70	55	30–71	60	38–80	0.44
Stage 3 and 4 %	17	1–53	21	5–47	14	0.3–47	0.10
REM%	23	9–30	20.3	8–35	21	7–35	0.07
Arousal index	13‡	5–34	13.8§	5–27	19	6–54	0.002
Habitual SOL	15	2–60	15	4–120	15	2–45	0.94
Habitual TST	465	195–540	420	300–600	480	300–720	0.60
ESS	8	0–14	6	0–15	7	1–19	0.52
Physical function	50	28–50	50δ	28–50	44	17–50	0.03
Bodily pain	74	32–100	84	0–100	72	31–100	0.54
General health	77	47–100	82	20–100	77	32–100	0.86
Vitality	65	30–100	73	45–90	65	25–90	0.18
Social function	100	50–100	100	25–100	100	50–100	0.78
Role-emotional	100	0–100	100	0–100	100	0–100	0.49
Mental health	84	40–100	84	32–100	84	56–96	0.69

*P-value for Kruskal-Wallis test. ESS (Epworth Sleepiness Scale).
†P-value < 0.0001 for Wilcoxon rank-sum (Mann-Whitney) test between groups 1 and 3.
‡ P-value < 0.0167 for Wilcoxon rank-sum (Mann-Whitney) test between groups 1 and 3.
§ P-value < 0.0001 for Wilcoxon rank-sum (Mann-Whitney) test between groups 2 and 3.
δ P-value < 0.01 for Wilcoxon rank-sum (Mann-Whitney) test between groups 2 and 3.

ABBREVIATIONS

BMI	body mass index
CPAP	continuous positive airway pressure
ECG	electrocardiogram
ESS	Epworth Sleepiness Scale
MS	Morning Survey
PCMCIA	personal computer memory card international association
PLM	periodic leg movements
QoL	Quality of Life
RDI	respiratory disturbance index
RDI 4%	respiratory disturbance index with events requiring a minimum 4% O_2 desaturation
SDB	sleep disordered breathing
SF-36	The Medical Outcomes Study 36 Item Short-Form Survey
SHHS	Sleep Heart Health Study
SHQ	Sleep Habits Questionnaire
SOL	sleep onset latency
TST	total sleep time

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