Sleep, Breathing, and Cephalometrics in Older Children and Young Adults*

Part II—Response to Nasal Occlusion

Richard P. Millman, MD, FCCP; Christine Acebo, PhD; Cynthia Rosenberg, DDS; and Mary A. Carskadon, PhD

Study objectives: **We postulated that nasal occlusion would provide a challange enabling us to assess factors predisposing development of sleep apnea in older children/adolescents and young adults. Factors of interest included sex, age, body mass index (BMI), tonsillar hypertrophy, and cephalometric measurements.**

Design: **Sleep and breathing variables were examined and compared for four groups of subjects between one baseline night and one night of nasal occlusion in a sleep research laboratory.**

Subjects: **Healthy, normal boys (n=23, mean age= 13.3±2.1 years), girls (n=22, mean age=13.8±1.8 years), men (n=23, mean age=22.2±1.5 years), and women (n=24, mean age=22.4±1.8 years) were studied.**

Measurements and results: **The following sleep and sleep-related breathing measures showed significant increases in all four groups from baseline to occlusion: percentage of stage 1, number of transient arousals, transient arousal index, apnea index, respiratory disturbance index (RDI), and mean apnea length. No significant relationships were found between occlusion-night RDI and tonsillar size, cephalometric variables, or BMI, either singly or in combination.**

Conclusions: **Subjects' responses to nasal occlusion varied: most demonstrated a minimal and clinically insignificant increase in RDI; few showed a marked increase in RDI. Significant increases of sleep fragmentation—even in the absence of frankly disturbed breathing—indicate that nasal occlusion may secondarily affect waking function if prolonged over a series of nights.** ***(Chest 1996; 109:673-79)***

A=A point; ANB=A point to Nasion to B point angle; ANS=anterior nasal spine; ANS-Gn=anterior nasal spine to Gn; BMI=body mass index; H=hyoid bone; MP-H=vertical position of the hyoid; N=nasion; N-ANS=nasion to ANS; NREM=nonrapid eye movement sleep; OSA=obstructive sleep apnea; PAS=posterior airway space; linear distance (mm) between a point on the base of the tongue and another point on the posterior pharyngeal wall both determined by an extension of a line from point B through Go); PNS=posterior nasal spine; PNS-P=soft palate length; RDI=respiratory disturbance index=apneas+hypopneas per hour of sleep; REM=rapid eye movement sleep; S=sella; SaO_2=arterial oxygen saturation from finger oximetry; SNA=sella to nasion to A point angle; SNB=sella to nasion to B point angle; SN-MP=sella to nasion to Go-Gn angle; SPT=sleep period time (sleep onset to sleep offset); TST=total sleep time

Key words: adolescent; apnea; body mass index; cephalometrics; nasal obstruction; sleep; young adult

Obstructive sleep apnea (OSA) syndrome is more common in men than in women.[1] In part, this may be due to greater prevalence of upper body obesity in men than women, differences in the mechanical properties of the pharynx predisposing to collapse, or differences in ventilatory control.[2] Even though most of the research effort on OSA has concentrated on working adult and elderly populations, it is not clear at what age the gender disposition for pharyngeal collapse actually starts. In another report examining sleep and breathing in normal boys, girls, young men, and young women, we found minimal sex- or age-related changes in breathing variables during sleep.[3] Nonrapid eye movement (NREM) respiratory disturbance index (RDI) was higher in male subjects than female subjects, and the arterial oxygen saturation (SaO_2) nadir was lower in male subjects than female subjects, although these changes were small and not clinically significant. The primary aim of the present study was to provide a challenge to the respiratory system with a nasal occlusion procedure in an effort to unmask latent sex- and age-group differences.

Lavie and Rubin[4] demonstrated that sons of parents with OSA developed a greater number of obstructive apneas during sleep with nasal occlusion than age-matched controls. Nasal occlusion led to increased apnea experimentally in normal subjects[5,6] and with nasal packing in patients.[7,8] It has also been shown to induce OSA during sleep, although the severity of sleep apnea is quite variable across individuals.[9] One

From the Sleep Research Laboratory (Drs. Acebo and Carskadon), Department of Psychiatry and Human Behavior, E.P. Bradley Hospital/Brown University, East Providence, RI; and the Sleep Disorders Center (Dr. Millman) and Department of Plastic Surgery (Dr. Rosenberg), Rhode Island Hospital/Brown University, Providence.

This work was funded by NIH grant HL44138.
Manuscript received April 7, 1995; revision accepted September 25.
Reprint requests: Dr. Carskadon, Sleep Research Laboratory, E.P. Bradley Hospital, 1011 Veterans Memorial Parkway, East Providence, RI 02915

Table 1—*Summary Measures for Sleep Variables: Mean (SD) for Each Group**

	Boys		Girls		Men		Women	
	Baseline	Occlusion	Baseline	Occlusion	Baseline	Occlusion	Baseline	Occlusion
SPT, min	553 (37)	549 (26)	531 (41)	524 (34)	463 (29)	466 (28)	460 (20)	459 (19)
TST, min	515 (55)	499 (42)	510 (36)	486 (35)†	437 (40)	424 (45)	430 (42)	417 (46)
Sleep efficiency, TST % of SPT	89.4 (7.5)	91.0 (6.2)	91.6 (5.2)	93.0 (6.9)	90.3 (7.4)	91.1 (8.6)	89.9 (8.4)	90.7 (8.0)
Sleep onset latency, min	20 (12)	19 (16)	21 (12)	20 (19)	20 (25)	18 (21)	19 (18)	19 (18)
Latency to REM, min	159 (50)	181 (78)	161 (77)	174 (71)	117 (42)	137 (72)	132 (65)	155 (91)
Stage 1, % of TST	8.4 (3.7)	11.6 (5.5)†	6.9 (3.4)	12.4 (7.8)§	8.0 (3.6)	13.5 (8.2)†	8.2 (4.6)	13.9 (9.0)†
Stage 2, % of TST	43.7 (10.9)	45.5 (7.6)	50.0 (7.8)	46.9 (7.2)†	53.8 (8.8)	51.2 (9.9)	50.4 (8.9)	48.8 (8.2)
Stage 3, % of TST	8.8 (4.2)	7.6 (3.5)	7.9 (4.0)	6.1 (3.6)	7.8 (2.3)	7.6 (2.6)	7.6 (3.3)	6.3 (3.3)†
Stage 4, % of TST	20.7 (8.5)	17.9 (6.4)†	17.9 (5.0)	18.0 (5.4)	11.7 (5.3)	10.2 (6.2)	15.7 (6.2)	14.9 (5.3)
REM, % of TST	17.7 (3.7)	16.3 (4.6)	16.8 (3.7)	15.8 (3.8)	17.5 (5.4)	16.6 (5.1)	17.3 (5.5)	15.3 (4.5)
NREM, % of TST	81.6 (3.7)	82.7 (4.7)	82.7 (3.9)	83.5 (3.9)	81.4 (5.6)	82.6 (5.0)	81.9 (5.5)	83.8 (4.5)
WASO, % of SPT	6.8 (6.0)	9.1 (6.2)	4.6 (4.9)	7.1 (6.9)	5.8 (4.9)	9.0 (8.6)	6.6 (6.3)	9.4 (8.0)
Transient arousals, No.	27 (17)	63 (53)‡	39 (26)	79 (43)§	42 (29)	60 (28)†	38 (27)	82 (68)†
Transient arousal index, (No./ TST) × 60	3.2 (2.0)	7.6 (6.1)‡	4.6 (3.1)	9.9 (6.0)§	5.8 (3.7)	8.5 (4.1)†	5.3 (4.1)	12.2 (10.7)‡

*p values show significance of night effect (baseline-occlusion) for each group. SPT=sleep period time; WASO=wake time after sleep onset.
†$p<0.05$.
‡$p<0.01$.
§$p<0.001$.

possible mechanism to explain pharyngeal collapse during sleep with nasal obstruction is that nasal occlusion produces a change in the pressure gradient across the pharynx during sleep. Respiratory intrapleural pressures during sleep have been demonstrated to be consistently greater when the nose is occluded than when it is open.[5] This finding would implicate an increase in oropharyngeal resistance with nasal occlusion favoring collapse of the airway during sleep.

We postulated that nasal occlusion would be an adequate challenge to determine whether there were sex and age factors predisposing for the development of sleep apnea in a group of 92 boys and girls and young adult men and women. Since baseline anatomic abnormalities may also be important, we examined the contributing role of body mass index (BMI), tonsillar hypertrophy, and cephalometric measurements.

Materials and Methods

Subjects

Volunteers were 23 boys (mean age=13.3±2.1 years), 22 girls (mean age=13.8±1.8 years), 23 young men (mean age=22.2±1.5 years), and 24 young women (mean age=22.4±1.8 years) from 84 families recruited through flyers and advertisements in local collegiate and city newspapers. The study was approved by the E. P. Bradley Hospital and Rhode Island Hospital Institutional Review Boards and informed written consent was obtained from each subject and/or parents in accordance with review board procedures. Subject characteristics are described fully in Acebo et al.[3] Briefly, volunteers were excluded for chronic illness, current use of decongestant or antihistamine preparations (medication free for >48 h), upper respiratory tract infection, acute otitis media, ongoing orthodontic treatment, dysmorphic features or syndromes (*eg*, Turner, Noonan, Pierre-Robin, or fetal alcohol syndromes), substance abuse, family history of narcolepsy, mental retardation, or obesity (BMI>27; BMI=kilograms/meter^2). By self or parental report, volunteers were in good health (free of chronic or recurrent illness), had regular sleep habits, were not habitual nappers, and were free of sleep disorders in themselves and in first-degree relatives (except for two girls whose father was diagnosed as having sleep apnea requiring no intervention).

Physical Assessment

Physical examination confirmed self-reported health status. Independent visual inspection of the airway was obtained by an expert in sleep disorders and pulmonary medicine (R.P.M.) and an orthodontist (C.R.) who rated tonsillar size using the Brodsky[10] scale. Height and weight were obtained and BMI was calculated as kilogram per meter squared.

Sleep and Breathing Evaluation

Sleep data were collected on a single first laboratory night within sleeping hours set according to the subject's usual schedule, providing at least 8 h of sleep in young adults and 9 to 11 h (depending on usual sleep requirement) in children. Details of recording procedures are provided in Acebo et al.[3] In brief, standard variables were recorded continuously to measure sleep (EEG, electro-oculogram, electromyogram), breathing (nasal-oral thermistors or thermocouples, abdominal and thoracic mercury-filled capillary strain gauges or piezo-crystal belts, finger oximetry [SaO_2]), and ECG (modified lead II).

Sleep was visually scored in 30-s epochs according to standard criteria of Rechtschaffen and Kales.[11] Sleep onset latency was calculated as elapsed time from lights out to the first of three continuous epochs of sleep. Latency to rapid eye movement (REM) sleep was calculated as elapsed time from sleep onset to the first epoch of REM sleep, including intervening wakefulness. Transient (≤15

Table 2—*Summary Measures for Respiration Variables: Mean (SD) for Each Group

	Boys		Girls		Men		Women	
	Baseline	Occlusion	Baseline	Occlusion	Baseline	Occlusion	Baseline	Occlusion
Apnea index, total	1.0 (1.0)	6.0 (8.6)†	0.9 (0.7)	5.4 (5.2)§	1.1 (1.3)	5.0 (3.8)§	0.7 (0.4)	6.0 (9.6)‡
Apnea index, NREM	1.0 (1.0)	6.9 (9.7)†	0.9 (0.7)	6.3 (6.2)§	1.0 (1.2)	5.1 (3.8)§	0.5 (0.5)	6.7 (11.3)†
Apnea index, REM	1.3 (1.5)	1.2 (1.3)	1.4 (1.2)	1.3 (1.5)	1.4 (3.2)	5.3 (7.2)†	1.5 (1.5)	2.0 (2.0)
RDI, total	1.3 (1.3)	7.3 (9.7)†	1.1 (0.7)	7.1 (5.6)§	1.3 (1.3)	6.1 (4.5)§	0.7 (0.5)	6.8 (10.8)‡
RDI, NREM	1.2 (1.3)	8.4 (11.0)‡	1.0 (0.7)	8.2 (6.7)§	1.2 (1.3)	6.4 (4.7)§	0.5 (0.5)	7.6 (12.3)‡
RDI, REM	1.5 (2.0)	1.6 (1.4)	1.6 (1.3)	1.8 (1.9)	1.6 (3.2)	5.9 (7.5)†	1.6 (1.5)	2.5 (2.4)
Mean length apnea, total	14.2 (4.3)	17.3 (4.0)†	14.1 (2.5)	17.6 (3.2)§	15.3 (3.8)	20.6 (7.6)‡	14.8 (5.9)	18.6 (3.5)†
Mean length apnea in NREM	14.2 (4.4)	17.4 (4.0)†	14.7 (4.3)	17.9 (3.3)‡	16.8 (3.1)	21.1 (7.9)	14.8 (7.4)	18.3 (3.9)
Mean length apnea in REM	12.7 (2.3)	16.8 (10.2)	13.0 (2.5)	14.7 (4.0)	12.3 (3.3)	17.2 (3.6)†	13.8 (4.2)	16.5 (6.0)
Hypopnea mean length, total	20.5 (9.6)	23.1 (6.6)	19.6 (9.3)	24.8 (7.6)	20.3 (9.8)	32.5 (14.0)	26.3 (13.4)	27.9 (15.0)
SaO_2 nadir	93.5 (2.7)	92.1 (1.9)†	94.3 (1.7)	92.4 (2.0)§	92.1 (4.8)	92.3 (2.0)	94.5 (1.7)	92.5 (5.6)

*p values show significance of night effect (baseline-occlusion) for each group.
†$p<0.05$.
‡$p<0.01$.
§$p<0.001$.

s) arousals were evaluated according to the criteria of the American Sleep Disorders Association;[12] transient arousal index was the number of transient arousals per hour of sleep.

Apneas were scored when airflow stopped for at least 10 s. Hypopneas were scored when airflow and effort dropped to less than 50% of basal for at least 10 s *and* when the return to basal excursion was accompanied by any signs of arousal in the recording *or* a fall of 4% in SaO_2 in young adults. The SaO_2 criterion was not used to identify hypopneas in children. Mean length of apneas and hypopneas were calculated for REM and NREM sleep. Apnea index (apneas per hour) and RDI (RDI=apneas+ hypopneas per hour) were also derived separately for REM and NREM sleep. SaO_2 nadir was the lowest measured value of SaO_2 that occurred during the night.

Nasal Occlusion Procedure

On the night following baseline recording, subjects came to the laboratory for a second overnight recording. Procedures were identical to the baseline night except for the addition of nasal occlusion; the nose was closed approximately 2 h before bedtime by placing a small amount of petrolatum-impregnated gauze inside the nasal vestibule bilaterally and covering the nares with tape. The occlusion remained in place until the subject woke up the next morning. Five subjects (one boy, one man, and three women) have not been included in the analyses because they were unable to sleep through the night with the occlusion and asked to have it removed. (These subjects were also excluded from the normal control data presented in Acebo et al.[3]).

Cephalometric Measurements

Clinical evaluation of the upper airway and lateral skull radiography were performed within 1 week of the sleep recordings by an expert in such assessments (C.R.). Details of the technique for cephalograms are provided in Acebo et al.[3] The lateral cephalograms were taken using a film focus distance of 5 feet with the subject's head secured in a cephalostat, in natural head posture, standing, and using a mirror eye reference position.[13] Soft tissue and bony structure points from the radiographs were digitized using cephalometric software (Quick-Ceph; Orthodontic Processing; Chula Vista, Calif), and tracings were constructed. Cephalometric measures obtained from these procedures included soft palate length (PNS-P, mm), vertical position of the hyoid (MP-H, mm), posterior airway space (PAS, mm) measured along the B-Go line, anteroposterior discrepancy between the mandible and maxilla (ANB, degrees), anteroposterior position of maxilla (SNA, degrees), anteroposterior position of mandible (SNB, degrees), mandibular plane angle (SN-MP, degrees), upper facial height (N-ANS, mm), lower facial height (ANS-Gn, mm), maxillary incisor inclination (degrees), mandibular incisor inclination (degrees), anterior overbite (mm), anterior overjet (mm), superior airway space (mm), and soft palate width (mm).

Data Analysis

Sex and age effects were found for many of the sleep and a few of the breathing measures on the baseline night.[3] Therefore, each variable was assessed for change across night (baseline vs occlusion) using repeated-measures analysis of variance for each age and sex group separately. A probability value less than 0.05 was required for significant effects. Relationships among sleep, breathing, cephalometric, and demographic variables were assessed using Pearson product moment or Spearman correlations, as appropriate. Finally, cluster analysis was performed in an attempt to identify homogeneous groups of subjects based on cephalometric measures. Sleep and breathing variables included in all analyses were limited to the principal outcome measures of interest, as indicated by variables listed in Tables 1 and 2. Analyses were performed using software (Systat version 5.2; Systat Inc) for the personal computer (Macintosh).

Results

Sleep Variables

Table 1 presents summary values for each of the sleep measures for each subject group on baseline and occlusion nights. Several consistent changes in sleep architecture were evident on the occlusion night. Percent of stage 1 sleep, number of transient arousals, and

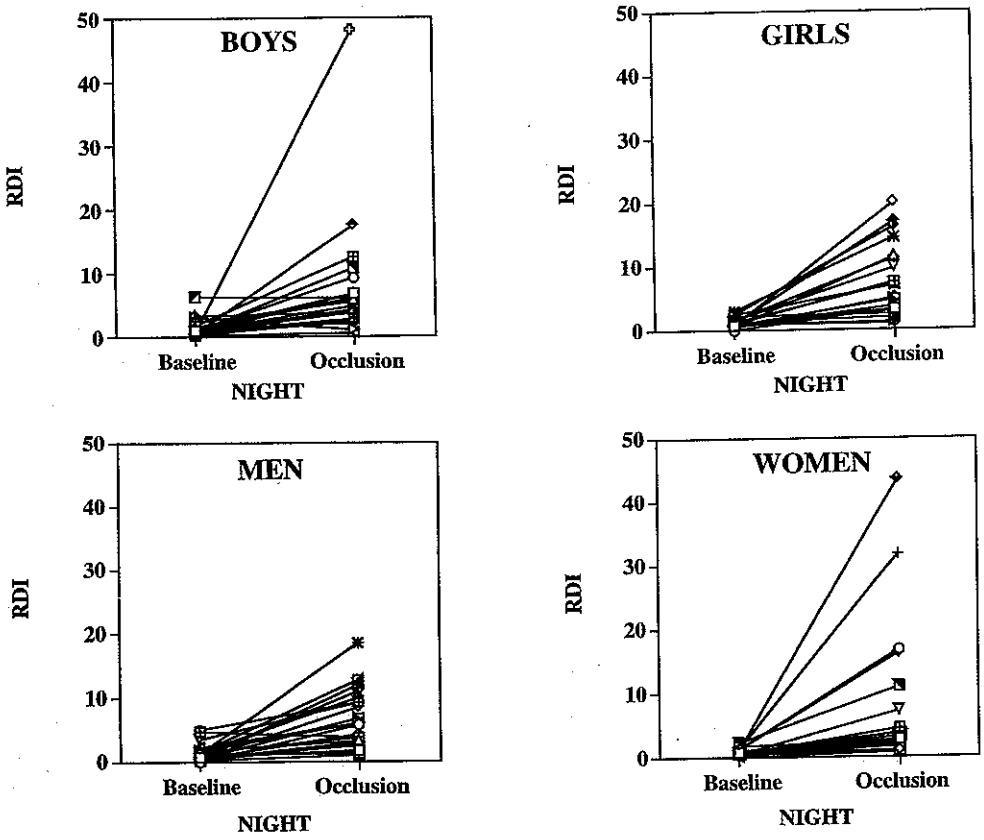

FIGURE 1. Response to nasal occlusion for each subject. Changes in RDI from baseline night to occlusion night are depicted for each subject within groups. RDI increased from baseline to occlusion night in 87 of 92 subjects.

transient arousal index increased significantly in every group. Other significant changes in sleep variables as a function of night were found inconsistently. Thus, for girls, significant decreases were seen for total sleep time (TST) and percent of stage 2 sleep; for boys, percent of stage 4 sleep decreased; and for women, percent of Stage 3 sleep decreased significantly with occlusion.

Breathing Variables

Table 2 presents summary values for each of the respiration measures for the four groups of subjects on both nights. Significant changes were found in all groups for most measures. Apnea index and RDI—measured during NREM or over the total night—increased in all groups. Increases in apnea index and RDI during REM were seen only in the men. Mean length of all apneas increased for each group, although mean length during NREM increased only for boys and girls, and mean length during REM increased only for men. Mean length of hypopneas did not change with occlusion. Finally, SaO_2 nadir decreased across nights for the children but not for the adults.

Figure 1 illustrates the chief finding with regard to sleep-related breathing during occlusion vs baseline. RDI increased from baseline to occlusion night in 87 of 92 subjects.

What Factors Predict Response to Occlusion?

RDI was chosen as the primary outcome variable for attempts to elucidate individual differences in response to nocturnal nasal occlusion, because RDI is a commonly used and reliably measured outcome variable, and it showed the most robust change with occlusion for these subjects.

Our first attempt to identify predictors was to examine simple correlations between RDI and tonsillar size, as well as the cephalometric measures. These analyses failed to identify any significant relationships, either within groups or over all subjects. Likewise, BMI was unrelated to RDI during the occlusion night.

Given the possibility that a combination of factors might predispose to breathing instability during nocturnal nasal occlusion, we performed cluster analysis using all cephalometric measures and BMI. Cluster membership was not related to RDI on occlusion night.

As Figure 1 illustrates, although RDI with nasal occlusion increased vs baseline in most (87 of 92) subjects, this response was minimal for most subjects

Table 3—*Summary of Potential Factors Associated With the Effects of Nasal Occlusion on Nocturnal Breathing: Normal Subjects With RDI of 1 SD or More Over the Subgroup Mean Occlusion Value*

Variable	Boy	Boy	Girl	Girl	Girl	Girl	Man	Man	Man	Man	Woman	Woman
Baseline RDI	0	1	1	2	3	1	1	1	1	0	1	1
Occlusion RDI	18	48	20	16	14	17	12	18	11	12	32	43
BMI*	1	−1		−1	1		1	−1	1	1	1	−1
Cephalometric variables*												
SNA	1			1	−1				−1	1		1
SNB	1				−1				−1	1		
ANB	−1	1	−1	1		−1				−1		
SN-MP						−1		−1	1		−1	−1
N-ANS		−1		−1	2				1	−1	1	
ANS-Gn			−1	−1	1						−1	−1
Maxillary incisor inclination						1	−1					
Mandibular incisor inclination							−1		−1		1	
Overbite		1							−1	−1	1	
Overjet		1				−1				−2		
PAS		−1			2		2	−1				
MP-H		−1	−1			−2		−1	1			
Superior airway space					2			−1				
Soft palate width PNS-P		1			1		1	1	−1	−1	1	

*Values indicate number of SD units above or below the subgroup mean.

and RDI remained within a normal range. To determine if "high responders" might differ from "low responders," we selected from each group the subjects whose occlusion night RDI exceeded the subgroup mean by 1 SD and compared them with a matched number of subjects who showed the least RDI response to occlusion. Table 3 (high responders) and Table 4 (low responders) characterize each subject in these groups by their place in the subgroup distribution of the structural variables (*ie*, BMI and cephalometrics). No patterns emerge from these tables to indicate either protection or susceptibility; extreme values on many structural variables were found in both high responders and low responders. When these data were summarized in a 2×2 frequency distribution table of hypothetical protective vs susceptibility factors (*eg*, high vs low PAS) in high and low responders, again, no significant differences emerged. In these normal subjects, cephalometric measures do not appear to be predictive of the response to occlusion. BMI is similarly nonpredictive.

Discussion

Nasal occlusion in older children and young adults led to an increase in apnea index and RDI as previously described in adult populations,[5,6,9] and an increase in apnea length. Hypopnea length did not change. These changes were significant for both male and female subjects in both age groups, although the degree of response varied among subjects. Though most subjects demonstrated an increase in the RDI on the occlusion night, this response was generally minimal and not clinically significant. RDI remained below 5 in 58% of the subjects despite nasal occlusion. In several subjects, however, there was a marked increase in RDI as high as 48 episodes per hour. This type of variable response to experimental nasal occlusion has also been described in adults.[5,6,9] We expected that this differential response would be related to obvious anatomic narrowing from tonsillar hypertrophy or cranial skeletal abnormalities. We cannot, however, demonstrate any relationship between tonsillar hypertrophy or cephalometric measurements and the response to nasal occlusion. One should not assume from these findings that there is a total lack of anatomic predisposition to pharyngeal collapse with nasal occlusion. Awake cephalometric measurements in the standing position may not accurately reflect airway dimensions and the potential for airway collapse of the pharynx during sleep in the supine position. It is possible that more technically advanced techniques such as fast cine CT[14] or MRIs of the pharynx[15] might have been able to demonstrate an anatomic predisposition to the development of significant sleep apnea with nasal occlusion. It is also possible that anatomy is not the key factor. Every pharyngeal airway has its own inherent collapsibility and this property may be more important than anatomic structure.

A genetic predisposition for airway collapse may have contributed to the variable response. Lavie and Rubin[4] demonstrated that the offspring of sleep apnea patients showed significant obstructive apneas during

Table 4—*Summary of Potential Factors Associated With the Effects of Nasal Occlusion on Nocturnal Breathing: Matched Normal Subjects With RDI Unchanged During Occlusion*

Variable	Boy	Boy	Girl	Girl	Girl	Girl	Man	Man	Man	Man	Woman	Woman
Baseline RDI	3	0	1	1	1	2	0	1	1	0	0	0
Occlusion RDI	1	1	2	1	1	2	2	1	1	1	1	1
BMI*							−1		−1			1
Cephalometric variables*												
SNA	1	−1							−1			
SNB		−1		2					−1	−1		
ANB	2			−1			−1				1	
SN-MP	1	1							1		1	
N-ANS		1	−1			1			1	1	−1	
ANS-Gn		1					1				1	
Maxillary incisor inclination				−1		1	1			−1		
Mandibular incisor inclination												
Overbite	1			−1	1	1	−1					
Overjet	1			−1			−2			1	−1	1
PAS						2		1				
MP-H				1		1	−1				−1	
Superior airway space			−1	−2					−2			
Soft palate width				1					1			−1
PNS-P		1	−1		2					−1		

*Values indicate number of SD units above or below the subgroup mean.

sleep with nasal occlusion. Though none of our subjects historically had parents or siblings with clinically significant OSA, it is possible that an occult history of OSA existed in more distant relatives. The hereditary patterns of sleep apnea have not been well worked out, but there is growing support for family predisposition that stretches beyond first-degree relatives.[16-18]

The sleep stage variables reflect expected findings indicating that sleep fragmentation was associated with nasal occlusion.[9] Arousals were generally quite brief and resulted in increased stage 1 sleep without a significant change in wakefulness *per se*. The number of transient arousals exceeded the number of apneas and hypopneas on the occlusion night. This probably represents a response to the increase in upper airway resistance. Nasal obstruction from allergic rhinitis has been shown to cause an increase in arousals during sleep independent of an increase in obvious OSA.[19,20]

We cannot state with absolute certainty that the increase in RDI seen in this study is a function solely of nasal packing, since we did not use a crossover design. The results for the young adults in our study, however, are in agreement with those of the study of Lavie and colleagues[9] of nasal occlusion that used a five-night design with partial and complete nasal occlusion nights balanced over subjects.

Our findings are clinically significant and indicate that some older children and young adults will develop significant sleep fragmentation from frank obstructive apneas-hypopnea and from transient arousals probably associated with increased upper airway resistance when the nose is plugged during sleep. Though we induced this experimentally, one would expect similar findings during periods of allergic rhinitis or in association with upper respiratory tract infections. Nasal obstruction over several days in these settings could potentially affect daytime performance if sleep obstruction continued without habituation.

ACKNOWLEDGMENTS: The authors thank Katherine Sharkey, Carol Carlisle, Clayton Bennett, Jr, Jon DiIorio, Pamela Bigler, James K. Wyatt, Cecilia Vieira, John Spencer, and Maximillian Stone for their assistance with this project.

References

1 Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993; 328:1230-35
2 Millman RP, Carlisle CC, McGarvey ST, et al. Body fat distribution and sleep apnea severity in women. Chest 1995; 107:362-66
3 Acebo C, Millman RP, Rosenberg C, et al. Sleep, breathing, and cephalometrics in older children and young adults: Part I—Normative values. Chest 1996; 109:664-72
4 Lavie P, Rubin AE. Effects of nasal occlusion on respiration in sleep: evidence of inheritability of sleep apnea proneness. Acta Otolaryngol (Stockh) 1984; 97:127-30
5 Olsen KD, Kern EB, Westbrook PR. Sleep and breathing disturbance secondary to nasal obstruction. Otolaryngol Head Neck Surg 1981; 89:804-10
6 Suratt PM, Turner BL, Wilhoit SC. Effect of intranasal obstruction on breathing during sleep. Chest 1986; 90:324-29
7 Taasan V, Wynne J, Cassisi N, et al. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. Laryngoscope 1981; 91:1163-72
8 Wetmore SJ, Scrima L, Hiller FC. Sleep apnea in epistaxis

patients treated with nasal packs. Otolaryngol Head Neck Surg 1989; 948:596-99
9 Lavie P, Fischel N, Zomer J, et al. The effects of partial and complete mechanical occlusion of the nasal passages on sleep structure and breathing in sleep. Acta Otolaryngol 1983; 95:161-66
10 Brodsky L. Modern assessment of tonsils and adenoids. Pediatr Clin North Am 1989; 36:1551-69
11 Rechtschaffen A, Kales A, eds. A manual of standardized terminology, techniques and scoring system for sleep states of human subjects. Washington, DC: US Government Printing Office, Public Health Service, 1968
12 Bonnet M, Carley D, Carskadon M, et al. EEG arousals: scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep 1992; 15:173-84
13 Cooke MS, We JHY. The reproducibility of nocturnal head posture: a methodologic study. Am J Orthod 1988; 93:280-88
14 Schwab RJ, Gefter WB, Hoffman EA, et al. Dynamic upper airway imaging during awake respiration in normal subjects and patients with sleep disordered breathing. Am Rev Respir Dis 1993; 148:1385-1400
15 Abbey NC, Block AJ, Green D, et al. Measurement of pharyngeal volume by digitized magnetic resonance imaging. Am Rev Respir Dis 1989; 140:717-23
16 El Bayadi S, Millman RP, Tishler RV, et al. A family study of sleep apnea: anatomic and physiologic interactions. Chest 1990; 948: 554-59
17 Pillar G, Lavie P. Assessment of the role of inheritance in sleep apnea syndrome. Am J Respir Crit Care Med 1995; 151:688-91
18 Redline S, Tishler PV, Tosteson TD, et al. The familial aggregation of obstructive sleep apnea. Am J Respir Crit Care Med 1995; 151:682-87
19 Lavie P, Gertner R, Zomer J, et al. Breathing disorders in sleep associated with 'microarousals' in patients with allergic rhinitis. Acta Otolaryngol 1981; 92:529-33
20 McNicholas WT, Tarlo S, Cole P, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. Am Rev Respir Dis 1982; 625-28