




Does Nasal Obstruction Induce Obstructive Sleep Apnea in Healthy Women?

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Purpose: Obstructive sleep apnea (OSA) is less prevalent among women and is associated with different symptoms and consequences to OSA in men. The reasons for these differences are unknown and difficult to tease apart in clinical populations. If OSA could be temporarily induced in healthy men and women, the causes of some of these differences could be investigated. Nasal blocking has been used to induce OSA in healthy men but its effect in women has not been reported.

Patients and Methods: A total of 14 healthy individuals (10 women) underwent in-laboratory diagnostic sleep studies on two occasions separated by a week. On one occasion, the nasal passages were blocked, whereas on the other occasion, participants slept naturally. In both conditions, a full-face mask was used to monitor respiratory events. Participants' self-reported sleepiness, mood and performance on a motor learning task were assessed in the evening and morning of both sleep studies. Furthermore, endothelial function and self-reported sleep quality were assessed in the morning following each study.

Results: Nasal blockage induced OSA in healthy young (age=22±3 years) and slim (BMI=22.2±3.2 kg/m²) women (control AHI=2.0±2.6, blocked AHI=33.1±36.7 events/hr, p=0.02). One night of OSA was associated with poorer self-reported sleep quality (p<0.001) and increased self-reported snoring (p<0.04), choking and gasping during sleep (p<0.001) but was not associated with alterations in mood, neurocognitive or endothelial function on the following morning.

Conclusion: Nasal blockage induces OSA in healthy, young, and normal weight women. However, whether the induced OSA is representative of naturally occurring OSA and the technique useful for future studies is unclear.

Keywords: pathophysiology, upper airway collapse, sex, breathing route, obstructive sleep apnea, nasal blockage, female

Plain Language Summary

Obstructive sleep apnea (OSA) is more common and associated with different symptoms and consequences in men as compared to women. The reasons for these differences are unknown and difficult to tease apart in clinic populations. Blocking the nose during sleep has been shown to induce OSA in otherwise healthy men, but no studies of nasal occlusion have been conducted in women to date. This study demonstrates that nasal occlusion induces OSA in otherwise healthy young women raising the possibility that nasal blockage may be useful technique for investigating sex differences in OSA.

Introduction

Obstructive sleep apnea (OSA) is approximately half as prevalent in women as it is in men.¹ The reasons for the lower prevalence of OSA in women are not entirely clear. The pathogenesis of OSA is considered to be multifactorial, with elements of airway anatomy, dilator muscle responsiveness, respiratory system instability and

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the threshold for arousal from sleep all implicated.² Women have been shown to have a less collapsible airway for any given body mass index,^{3,4} which may at least in part explain the lower prevalence of OSA. However, other aspects of the pathogenesis including respiratory control stability and airway dilator muscle responsiveness do not appear to differ between the sexes.^{3,5,6} Further, it is unclear whether the respiratory arousal threshold differs between sexes,⁷ although it should be noted that there is substantial within-subject variability in respiratory arousal threshold across the night.⁸ Thus, the causes of the sex difference in OSA prevalence are incompletely understood.

In addition to the sex difference in the prevalence of OSA, the disease phenotype also differs between men and women.⁹ Women are more likely to demonstrate REM sleep predominant OSA,¹⁰ have shorter apneas^{11,12} with less blood oxygen desaturation,¹³ and have a smaller change in severity from supine to lateral body position¹⁴ than men. It is presently unclear whether these differences are related to women having lower severity of OSA than men in clinical samples,^{11,14} or if they occur irrespective of the severity of OSA. Alternatively, the differences may be influenced by sex differences in demographic factors, such as women with OSA being more likely to be older and have higher body mass indices than men with equally severe OSA.^{9,10}

Finally, the symptoms and consequences of OSA also appear to differ between sexes,^{9,15} with women more likely to report insomnia, fatigue, headaches and depression, and to experience greater mood disturbance, impaired quality of life and altered metabolic consequences of OSA than men.^{9,16-18} There are also reports of improved mortality in women with OSA compared to men with OSA.^{19,20} Whether the different consequences of OSA relate to the altered characteristics of OSA mentioned above (shorter events with more sleep fragmentation and higher airway resistance but less desaturation) or reflect an underlying sex difference in susceptibility to OSA consequences is not known. It is difficult to tease apart these possibilities in clinical patients as either potentially biased samples of men and women who are matched on certain characteristics, or large numbers of subjects and statistical accounting for potentially confounding sex differences, are required.

An alternative way of investigating sex differences in OSA would be by temporarily inducing OSA in otherwise healthy individuals with and without the underlying confounding differences that exist in clinical samples. Numerous studies have demonstrated that blocking the

nose of healthy men during sleep can induce OSA.²¹⁻²⁵ However, none of these studies included any female participants. Thus, the primary aim of this study was to determine whether a single night of nasal blockage induces OSA in healthy young women. Although the primary aim of the study was to investigate the effect of nasal blockage in women, we also recruited approximately 1/3 men to ensure that the prior reported effects of nasal blockage were observed in our laboratory population (typically young, slim university students) using current OSA scoring rules.²⁶ The secondary aim was to determine whether one night of nasal blocking was associated with negative consequences (sleepiness, memory impairment, impaired mood and/or cardiovascular function) in otherwise healthy young men and women.

Patients and Methods

Subjects

Healthy individuals aged between 18 and 65 years were recruited using flyers placed around the University of Melbourne Parkville campus. Individuals were excluded from participation if they were self-reported poor sleepers or were currently experiencing sleep disturbances, had recently travelled through more than 1 time-zone, were smokers, had been diagnosed with any sleep disorder, cardiorespiratory disease or diabetes, or were allergic to Amethocaine. The study was approved by the University of Melbourne Human Research Ethics Committee (1545303) and was performed in accordance with the Declaration of Helsinki. All participants gave informed written consent to participate.

Design

The present study utilised a crossover design, with each participant completing an experimental (nose blocked) and control night in the sleep laboratory, with the two nights separated by approximately 1 week. As the experimental intervention was self-evident, it was not possible to blind participants to experimental conditions. To prevent order effects, the experimental conditions were counterbalanced for every 10 participants, using a computer-generated pseudo-random sequence.

Procedures

Participants were asked to maintain their normal sleep schedule for the week leading up to the study and to refrain from ingesting caffeine or alcohol for 12 hours before attending the sleep laboratory. Participants arrived

at the laboratory approximately three hours prior to their regular bedtime. Upon arrival, individuals had their age, sex, height, and weight recorded. They were then led into a quiet room where they completed the multivariable apnea risk index (MAPI, first night only),²⁷ positive and negative affect schedule (PANAS),²⁸ Karolinska Sleepiness Scale (KSS),²⁹ and a motor learning task (motor sequence task: MST, details below) on a computer. Participants then prepared for bed, and were prepared for polysomnography. Specifically, participants were fitted with six EEG electrodes (F3, F4, C3, C4, O3, O4 referenced to the contralateral A1 or A2 electrode), left and right EOG, masseter EMG, oximeter, respiratory bands and leg sensors. On the experimental night participants then had their nose blocked (details below). On both nights a full-face mask (FreeMotion RT041, Fisher and Paykel, Auckland, NZ) was worn and was connected to a pitot flow sensor (Seleon GmbH, Heilbronn, Germany) to assess airflow. All signals were amplified and filtered using a Compumedics Graef (Abbotsford, VIC, Australia) unit and recorded using Profusion PSG 3 software (Compumedics, Abbotsford, VIC, Australia) in accordance with the AASM guidelines.²⁶ Once the quality of the signals was confirmed, the lights were turned off and participants were allowed to sleep for at least 8 hours. If the nasal blockage became too bothersome during the night, participants could remove it for approximately 20 minutes, before it was re-inserted.

Following awakening, participants moved to a comfortable chair and 10 minutes elapsed before two blood pressure measurements (HEM 7130, Omron Healthcare, Singapore) were taken and endothelial function assessed (Endopat, Itamar Medical, Caesarea, Israel). Endopat measures of arterial stiffness (Augmentation index) and reactive hyperemia index (RHI) were automatically calculated. Participants then rated the quality of their sleep on a Likert scale ranging from 1 (very poor) to 10 (excellent), and reported how strongly they agreed with experiencing “Vivid dreams”, “Snoring”, “Gasping for air/feeling like I was choking” and “Feeling sweaty” (1 strongly disagree, 10 strongly agree). They were then provided with breakfast that did not include any caffeinated products after which they repeated the PANAS, KSS and MST. Subsequently, the electrodes and physiological sensors were removed and the participants were discharged from the laboratory.

Nasal Blockage: Nasal packing was employed to occlude the nares in accordance with prior studies.²⁴ This involved

inserting the ends of a petroleum jelly coated roll of gauze approximately 1.5 cm into each nostril until the participant was unable to breathe through their nose. The gauze was secured in place overnight using Slick tape placed underneath the roll onto the nostrils and over the bridge of the nose.

Motor Sequence Task: The MST followed the protocol of Tucker et al.³⁰ Specifically, the participant typed a 5-number sequence as many times and as accurately as they could for 30s using their non-dominant hand. Twelve trials were performed with 30s rest between each trial. On the first experimental session, participants used the sequence 41324, whereas on the second session participants used the sequence 23142.

Data Analysis

PSG data were visually scored for sleep stages and respiratory events in accordance with the recommended AASM (2012) guidelines²⁶ by an expert technician who was blinded to the experimental night. Consistent with prior studies,³⁰ the first 3 and last 3 trials of each MST session were averaged to monitor task performance. The positive affect questions were summed, as were the negative affect questions in order to generate a single value of positive and negative affect to compare between evening and morning, as well as between conditions.

Statistical Analysis

The primary aim was assessed with paired samples Student's *t*-tests comparing the Apnea Hypopnea Index (AHI) between conditions in women only. This was repeated for the male subjects, although it should be noted this comparison was to ensure the previously reported effect in males was observed with our laboratory procedures. Subjective sleepiness and sleep quality as well as the other polysomnographic characteristics and secondary outcome variables were also compared between the experimental and control conditions using Student's *t*-tests for all participants (men and women combined). Exploratory Pearson's correlations were conducted to ascertain whether the overnight change in AHI was associated with baseline AHI or demographic characteristics (age, BMI, MAPI). In addition, preliminary insight into whether sex differences in OSA characteristics exist was determined by comparing sleep study variables between sexes. Due to only 4 men being studied, we have not reported *p* values but rather effect sizes, which were manually calculated (using the following equation: $r = z \div \sqrt{n}$). Effect sizes were interpreted according to Cohen's (1988) criteria, which defined small, medium and large

effect sizes as being equivalent to 0.1, 0.3, and 0.5, respectively.³¹ All analyses were conducted using IBM SPSS Statistics 25 software (Armonk, NY). Mean \pm SD are presented and $p < 0.05$ considered significant.

Results

Ten women and four men completed the protocol. The women were aged 22 ± 3 years and had a mean BMI of 22.2 ± 3.2 kg/m². The men were aged 26 ± 7 years and had a mean BMI of 22.6 ± 3.7 kg/m². All participants had low probability of apnea based on the MAPI (range 0.01–0.19).

Figure 1 shows the AHI on the two experimental nights in all participants. The AHI was significantly higher on the night when the nasal passages were blocked compared to the control night in women (AHI = 33.1 ± 36.7 versus 2.0 ± 2.6 events/hr, respectively, $p = 0.02$). Although the results appeared similar in the four men they were non-significant given the small sample size (AHI = 19.3 ± 36.7 versus 1.1 ± 2.6 events/hr, respectively, $p = 0.12$). Among all participants (men and women combined), the only significant predictor of the change in AHI between nights was the baseline AHI ($p = 0.006$, $r^2 = 0.49$). Other sleep characteristics on each night for both sexes are shown in Table 1.

Four women developed severe OSA whereas the other 6 women studied developed no-mild OSA. The mean age (severe = 21.0 ± 1.4 and no-mild = 22.7 ± 4.8 years) and BMI (severe = 21.6 ± 2.5 and no-mild = 22.7 ± 3.8 kg/m²) were very similar between these sub-groups of women. Further, the AHI difference could be observed in both supine (severe = 71.4 ± 27.3 and no-mild = 6.4 ± 2.8 events/hr) and non-supine (severe = 55.6 ± 8.6 and no-mild = 8.6 ± 7.1 events/hr) positions.

However, the four women who developed severe OSA appeared to have shorter sleep time (Total sleep time = 367 ± 18.9 min and 438 ± 24.2 min) and more disturbed sleep (Sleep efficiency = $76.2 \pm 5.2\%$ and $87.7 \pm 4.5\%$) on the control night when compared to the women who went on to develop no-mild OSA. This was accompanied by a tendency for more REM-related respiratory events on the control night in the 4 women who went on to develop severe OSA (REM AHI severe 15.1 ± 17.2 and no mild = 2.4 ± 1.7 events/hr). The subjective reports of feeling like choking/gasping also tended to be higher in the 4 women who developed severe OSA as compared to those who developed no-mild OSA (7.3 ± 1.5 and 5.2 ± 3.2 , respectively) whereas sleep quality tended to be lower in with severe OSA (2.3 ± 1.3 and 3.8 ± 2.4 , respectively).

Table 2 summarises the effect of nasal blockage on subjective sleepiness, self-reported sleep quality and cardiovascular function. KSS ratings in the evening of the sleep study did not differ between conditions (5.0 ± 1.9 prior to control night, 4.6 ± 1.5 prior to blocked night, $p = 0.58$). It did however differ between conditions on the morning following testing (Table 2). The subjective quality of sleep was poorer, and self-reported snoring and choking/gasping were increased, following the blocked night. Vivid dreams and self-reported sweatiness were not altered (Table 2). One male subject did not complete the cardiovascular testing due to missing equipment; hence, the cardiovascular data were obtained from 13 participants. Although there were trends towards differences in the arterial stiffness measure (AI) and heart rate, these were not significantly different between conditions (Table 2).

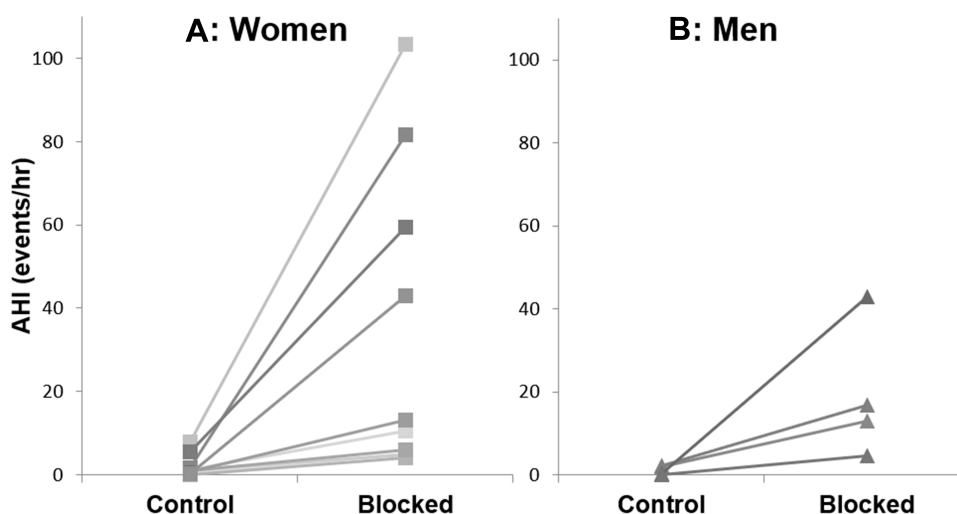


Figure 1 Individual Apnea-Hypopnea Indices (AHI) on the control and experimental (Blocked) nights in women ((A), squares, $n = 10$) and men ((B), triangles, $n = 4$)

Table 1 Sleep Characteristics on the Control and Experimental (Blocked) Nights in All Participants (n=14)

	Control	Blocked	P value
Total sleep time (min)	404.4±47.1	334.4±85.2	=0.001
Sleep onset latency (min)	22.5±21.5	29.5±27.0	0.27
Wake after sleep onset (min)	60.4±32.5	125.2±73.9	=0.001
Sleep efficiency (%)	82.5±7.7	67.8±16.4	<0.001
Total Arousal index (no/hr)	11.1±4.6	41.7±28.2	<0.001
Respiratory arousal index (no/hr)	2.3±2.5	27.1±27.0	<0.001
Spontaneous arousal index (no/hr)	8.8±2.7	14.5±5.7	<0.001
Nadir SaO ₂ (%)	93.1±1.7	90.6±2.8	<0.001
4% ODI (no/hr)	2.1±3.8	14.9±19.7	0.02
N2 (%TST)	46.8±7.2	44.5±10.8	0.5
N3 (%TST)	24.2±4.7	16.1±10.7	0.01
REM (%TST)	24.4±5.6	21.1±10.9	0.2

Abbreviations: SaO₂, finger oxygen saturation; ODI, oxygen desaturation index; N2, time spent in stage 2 sleep; N3, time spent in slow-wave sleep; REM, times spent in rapid eye movement sleep; TST, total sleep time.

Two participants were inadvertently given different test sequences (41324 vs 23142) from morning to evening on the first week of testing and therefore all MST data were excluded for both nights for these participants. The performance on the MST was slightly improved from the end of evening learning to the initial performance in the morning on both the control (23.4 ±4.5 and 25.5±6.5 correct trials, respectively, p=0.03) and blocked nose nights (22.1±6.2 and 25.9± 6.6 correct trials, respectively, p=0.004). However, the magnitude of the overnight improvement in MST performance did not differ between conditions (Figure 2, M1-M3 minus E10-E12, p=0.32).

Neither positive nor negative affect differed in the evening between study nights (Table 3). Likewise, neither positive nor negative affect differed between control and blocked nose nights in the morning (Table 3).

The characteristics of OSA on the blocked nose night are shown separately for men and women in Table 4. This analysis should be considered in light of the fact that there are only 4 male participants. Given the low subject numbers, the effect sizes (a quantitative measure of the magnitude of the difference between the two groups³¹) are reported, and were quite small (in accordance with Cohen's (1988) criteria, page 79–80³¹) for all comparisons except the obstructive apnea index which was moderately

Table 2 Subjective Sleep and Cardiovascular Function on the Control and Experimental (Blocked Nose) Nights in All Participants

	Control	Blocked	P value
Subjective Sleep (n=14)			
KSS (morning measure)	4.3±1.3	5.4±1.7	0.03
Quality of sleep (10 best)	6.7±2.1	3.1±1.9	<0.001
Vivid dreams (10 strongly agree)	5.3±2.9	4.8±3.2	0.45
Snoring (10 strongly agree)	2.2±1.3	3.8±2.7	0.039
Gasping/Choking (10 strongly agree)	2.4±1.7	6.2±2.2	<0.001
Sweaty (10 strongly agree)	3.5±3.1	3.8±2.2	0.77
Cardiovascular Function (n=13)			
BPsys	108±12	108±13	0.98
BPdia	70±8	70±9	0.96
HR	65.8±10.5	70.9±13.8	0.060
RHI	1.79±0.36	2.03±0.47	0.11
AI (%)	-6.23±11.6	-11.6±9.7	0.057

Abbreviations: KSS, Karolinska Sleepiness Scale; BPsys, systolic blood pressure; BPdia, diastolic blood pressure; HR, heart rate; RHI, reactive hyperemia index; AI, augmentation index.

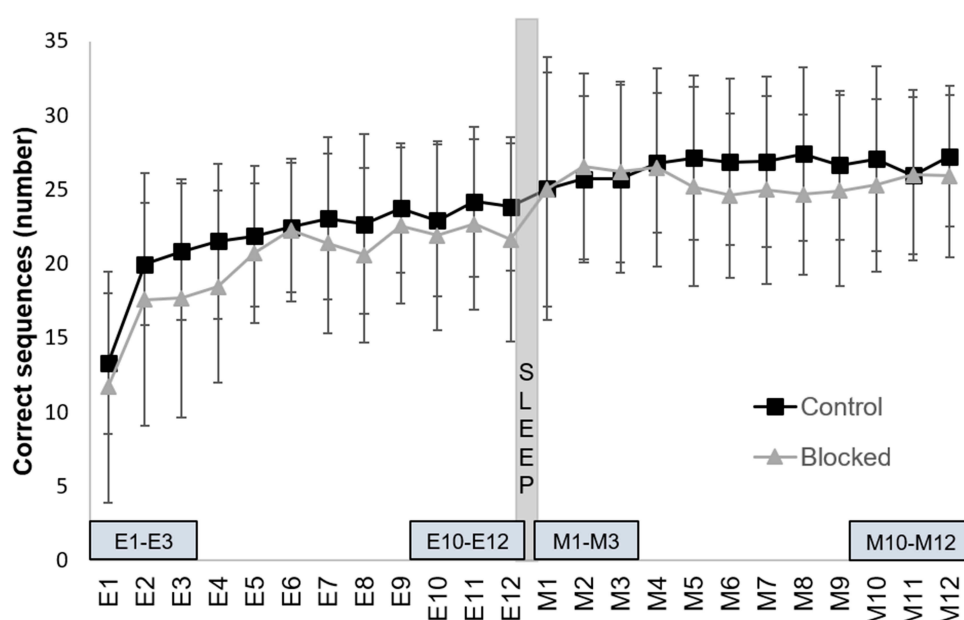


Figure 2 Motor sequence task performance in men and women with adequate data ($n=12$). The number of correct sequences typed during the motor sequence task over the twelve 30s trials conducted in the evening (E1–E12) and morning (M1–M12) on control (black square) and blocked nose (grey triangle) nights. The time points that were averaged for statistical comparisons are indicated in the horizontal bars at the bottom (E1–E3, E10–E12, M1–M3, M10–M12) and the time of the sleep period indicated by the grey vertical bar.

different between sexes (although still non-significant with $p=0.21$).

Discussion

The present study has demonstrated that blocking the nose of healthy, young women with normal body weight induces OSA in a similar fashion to what has been demonstrated in men.^{21–25} As per previous studies in men, there was a wide variability in the severity of OSA that was induced in women. The severity of OSA induced in these healthy individuals failed to correlate with the MAPI, BMI and age of participants. Only the AHI on the baseline night (which ranged from 0 to 7.9) was correlated with the magnitude of increase in AHI between conditions. Likewise, the four women who developed severe OSA on the blocked nose night appeared to have worse sleep

quality and more REM-related events even on their control night perhaps indicating these individuals were lighter sleepers with a predisposition to airway collapse at least in REM. While blocking the nose clearly increased the AHI, it may have also independently disturbed sleep because the spontaneous arousal index and wake after sleep onset time were also increased on the blocked nose night while total sleep time was reduced in contrast to what is typically observed in at least chronic OSA.³² In addition, the OSA that was induced was associated with

Table 4 Obstructive Sleep Apnea Characteristics on the Blocked Nose Night in Men and Women. Due to the Small Number of Men Included the Effect Sizes are Reported (a Quantitative Measure of the Magnitude of the Difference Between the Two Groups) with Small, Medium and Large Effect Sizes Being Equivalent to 0.1, 0.3, and 0.5 (Pages 79–80).³¹

	Women ($n=10$)	Men ($n=4$)	Effect Size
NREM AHI	33.5±37.7	29.5±17.1	0.14
REM AHI	26.5±33.5	24.8±19.5	0.06
Supine AHI	32.4±37.2	28.9±23.1	0.11
OA index	11.8±23.7	1.7±2.1	0.60
Hypopnea index	21.2±22.4	17.5±16.6	0.19
OA duration (s)	15.6±6.4	14.3±9.7	0.15
Hyp duration (s)	22.5±4.6	22.5±6.6	0

Abbreviations: AHI, Apnea Hypopnea Index; OA, obstructive apnea; Hyp, hypopnea.

Table 3 Positive and Negative Affect Scores in the Morning and Evening of Control and Experimental (Blocked Nose) Nights in All Participants ($n=14$). No Significant Differences Between Evening-Morning or Between Conditions Were Observed

	Control		Blocked Nose	
	Evening	Morning	Evening	Morning
Positive affect	27.3±7.7	26.9±7.4	31.0±7.2	24.7±7.2
Negative affect	15.9±4.7	16.3±5.9	16.1±4.5	16.3±6.4

relatively mild blood oxygen desaturation (90% average, ODI = 15 events/hr) and was similar between body positions and NREM versus REM sleep. Finally, snoring was not commonly noted by the overnight staff. Thus, the OSA that was induced by blocking the nose appears to differ somewhat from spontaneously occurring OSA.

The mechanisms by which enforcing oral breathing induce OSA are uncertain. Upper airway resistance is similar when breathing through the oral as compared to nasal route while awake.³³ However, even in the absence of respiratory events, airway resistance during sleep is actually higher during oral than nasal breathing.³³ Elevated resistance during oral breathing while asleep may occur because of a relative reduction in upper airway dilator muscle activity due to reduced stimulation of upper airway negative pressure receptors during oral breathing,³⁴ although not all studies demonstrate reduced dilator muscle activity during oral breathing.³⁵ In addition, it is likely that participants adopted a more open jaw position on the blocked nose night which would predispose them to upper airway collapse^{36,37} and possibly shorten the transverse genioglossal fibers, thereby reducing the muscles mechanical efficacy.³⁸ Another possibility is that during oral breathing the tongue is no longer restricted in its movement by the hard and soft palate and therefore is able to flop further back into the retroglossal space and therefore predispose to collapse. Finally, it is likely that the humidification of inspired air is reduced during oral breathing which may lead to a relative drying of the airway and increased surface tension forces as a result.³⁹ Which, if any, of these mechanisms contributed to the increase in AHI observed in the current study during nasal occlusion is unknown.

Although there were very few men studied, the characteristics of the induced OSA appear similar between sexes. This contrasts with OSA in the sleep clinic and general populations in which men are typically reported to have longer respiratory events¹¹ than women and a more even distribution of respiratory events between REM versus NREM as well as supine versus non-supine sleep positions.¹⁴ This may indicate that the cause of sex differences in OSA characteristics in clinical or general population samples are a result of comorbid conditions or anthropometric differences between sexes, such as higher body mass indices or older age in women with OSA. Alternatively, the similarity in OSA characteristics observed between sexes in this study may be related to the unique and un-natural cause of airway collapse

(obligatory oral breathing) whereas the pathophysiology of naturally occurring airway collapse may cause the differences in OSA characteristic between sexes that are typically observed. For example, if the upper airway dilator muscles of women were more effective at holding open/re-opening the collapsible segment of the upper airway than the dilator muscles in men, it could explain why men get more OSA, have longer events (they are more reliant on arousal to re-open the airway) and why women would be relatively protected from events in NREM sleep compared to men but equally susceptible in REM (when the benefit of improved dilator muscle effectiveness would be reduced). Likewise, if sex differences in sleep-related breathing route preference exist then this advantage would have been removed in the current study. Further research with suitable numbers of men and women are required to assess such possibilities.

Despite quite notable group changes in the AHI and subjective reports of increased sleepiness on the morning following the blocked nose condition, the self-reported mood and performance on the motor sequence task were not altered between conditions. This may relate to the fact that sleep was relatively well preserved (N2 and REM were unchanged between conditions and N3 was reduced from 24% to 16% of total sleep time). Alternatively, these analyses may have been underpowered. However, it is also possible that one night of OSA is insufficient to elicit these consequences of OSA, particularly in this young cohort. Similarly, cardiovascular function may only be altered by events associated with more severe hypoxemia or exposure to respiratory events over more than one night. Future research studies employing multiple nights sleeping with the nasal passages blocked in a larger sample may be required to separate these possibilities.

The study is limited by several factors, including the different cause of OSA compared to the natural situation, possible additional sleep disturbance from the intervention itself, and small number of men included in this study. A further limitation was the fact that it was not possible to blind participants as to the experimental condition. This was particularly problematic as, for safety reasons, participants were warned during the initial screening session that they may experience poorer sleep during the experimental condition and therefore should not schedule it before important activities the next day such as exams or long drives. It is therefore plausible that morning performance following the experimental intervention may have been influenced by participant expectation.

Conclusion

This study has shown that OSA can be induced in otherwise healthy, young normal weight women by blocking the nasal passages for a single night. However, the induced OSA appeared to differ somewhat from naturally occurring OSA. Therefore, whether blocking the nose to induce OSA will be useful for future research studies will depend on the question being asked. It is possible that more than one night is required to more adequately represent OSA and its consequences. We would advise anyone attempting such research to try to recruit individuals who report snoring or who have baseline AHIs in the high normal range (AHI 3–5) in order to maximise the chances of inducing significant OSA during nasal blockage.

Ethics Approval and Informed Consent

All procedures performed in this study were in accordance with the ethical standards of the University of Melbourne and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

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Disclosure

All authors declare they have no conflicts of interest in this work.

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