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Obstructive sleep apnoea and oral breathing in patients free of nasal obstruction

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Short title: Sleep apnoea and oral breathing

Abstract

Although there is an association between nasal obstruction, oral breathing and obstructive sleep apnoea syndrome (OSAS), it remains unknown whether increased oral breathing occurs in patients with OSAS free of nasal obstruction. This study evaluated the relationship between breathing route and OSAS in patients without nasal obstruction.

We examined during an overnight polysomnography the breathing route of 41 snorers (25 men; aged 26-77 yrs) with normal nasal resistance by using a nasal cannula/pressure transducer and an oral thermistor.

Twenty-eight patients had OSAS (apnoeics) and 13 patients were simple snorers. Apnoeics had higher percentage of oral ($p=0.004$) and oro-nasal ($p<0.001$) breathing epochs. Oral and oro-nasal breathing epochs were positively related with apnoea-hypopnoea index (AHI), duration of apnoeas/hypopnoeas and inversely related to oxygen saturation. Additionally, oro-nasal breathing epochs correlated with body mass index. In multiple linear regression analysis, oral breathing epochs were independently related only to AHI ($R^2=0.443$), and oro-nasal breathing epochs were independently related to AHI ($R^2 = 0.736$) and body mass index ($R^2 = 0.036$).

In conclusion, apnoeics spend more time breathing orally/oro-nasally than simple snorers, and AHI is a major determinant of the time that they spend breathing orally and oro-nasally.

Key Words: apnoeics, obstructive sleep apnoea syndrome, oral and oro-nasal breathing epochs, snorers.

Introduction

Humans preferentially breathe *via* the nasal route, the purpose being to filter, warm and humidify the inspired air [1]. During sleep healthy subjects free of nasal disease are estimated to inhale *via* the oral route only ~4% of the total ventilation, irrespective of sleep stage or body position [2]. It is only ageing that influences breathing route and thus, older individuals have decreased nasal breathing during sleep [3].

In healthy subjects, it is not surprising that nasal obstruction results in an increase of the proportion of mouth breathing [4]. However, mouth breathing during sleep has been associated with breathing disorders [5]. Indeed, experimental nasal occlusion disturbed sleep and triggered the induction of obstructive apnoeas [6]. Similarly, allergic rhinitis provoked both sleep fragmentation and obstructive sleep apnoea syndrome (OSAS) [7]. The OSAS appeared to be reversible during remissions of the allergic symptoms [7].

The association between nasal obstruction and OSAS has been thoroughly evaluated in several studies [8, 9, 10]. For example, LOFASO *et al* [9] demonstrated that patients with OSAS tended to have higher nasal resistance than snorers without OSAS, and that nasal resistance was an independent risk factor for OSAS. McLean *et al* [10] demonstrated a marked reduction in mouth breathing when nasal resistance was reduced with a decongestant. However, in the latter study the impact of nasal resistance on sleep apnea severity was very modest indeed. Thus, it remains uncertain whether increased oral breathing occurs in patients with OSAS in the absence of increased nasal resistance. Data linking mouth breathing and sleep apnea in the absence of increased nasal resistance is sparse and indirect [3]. An evaluation of the breathing route during sleep in OSAS could be an important step in understanding the upper airway physiology in these patients.

Therefore, in the present study, we aimed to document the breathing route in patients with OSAS without nasal obstruction. We hypothesized that these patients have increased oral or oro-nasal breathing, which may be associated with the severity of the disease. That is, because nocturnal hypoxemia along with sleep fragmentation may lead to further instability of the ventilatory control mechanisms [11] affecting the breathing route that patients use during sleep.

Material and methods

Study subjects

The study comprised 62 subjects who referred to the Center of Sleep Disorders of "Evangelismos" General Hospital of Athens for suspected sleep disordered breathing during a three month period. The enrolment criteria were: 1) snoring and either excessive daytime sleepiness or observed apnoeas, 2) no upper or lower respiratory tract disease, including a history of nasal allergy, 3) no recent surgery involving upper airway, 4) no history of evaluation or treatment for sleep apnoea, 5) no use of medications known to influence nasal resistance (antihistamine, decongestants, etc), 6) no history of any neuromuscular disorder, and 7) no history of cardiovascular disease. Enrolment criteria were chosen to provide a spectrum of disease ranging from clinically significant OSAS to non-apnoeic snoring. Exclusion criteria were: 1) nasal resistance values exceeding the normal values (see below), 2) central apnoeas more than three per hour or five percent of total apnoeas, and 3) total sleep time less than three hours. Our hospital ethics committee approved the study and all subjects gave their written informed consent prior to enrolment to the study.

Study design

Each subject reported to the sleep laboratory between 9 and 10pm. Nasal resistance was measured in upright seated and supine positions. A full-night diagnostic polysomnography with concomitant monitoring of the breathing route during sleep was then performed, usually from midnight to 7am.

Rhinomanometry

For each subject, nasal resistance to airflow was measured during wakefulness without decongestion, first in the upright seated position and then in supine position after lying down for 10 minutes. Active anterior rhinomanometry (PDD-301/r, Piston, Budapest, Hungary) was performed and international recommendations [12] were followed. In brief, patients wore a closely fitting face mask which didn't distort the nostrils or the nasal valve and breathed through one only nostril (first the left and afterwards the right) with the mouth closed. The pressure probe was placed at the opening of the contralateral occluded nostril not being tested. Total resistance was then automatically calculated from the 2 unilateral measurements. Nasal resistance was given

at a pressure difference of 150 Pascal across the nasal passage. Active posterior rhinomanometry (the pressure sensor was placed transorally into the posterior pharynx) was also performed in all subjects who had normal nasal resistance in the active anterior rhinomanometry. However, these measurements were not reliable or reproducible in 6 subjects (lack of cooperation despite extensive coaching) and they were omitted from further analysis. The nasal resistance values of the remaining subjects were similar to those of the active anterior rhinomanometry. Nasal resistance values below 3.0 $cmH_20 \cdot L^{-1} \cdot s$ were considered within normal limits [13].

Polysomnography

A full-night diagnostic polysomnography (EMBLA S7000, Medcare Flaga, Iceland) was performed in each subject. To determine the stages of sleep an electroencephalogram (with four channels, C4-A1, C3-A2, O2-A1, O1-A2), electro-oculogram (with two channels) and electromyogram of the submentalis muscle (with one channel) were obtained. Arterial blood oxyhemoglobin was recorded with the use of a finger pulse oximeter. Thoracoabdominal excursions were measured qualitatively by respiratory effort sensors [XactTrace belts featuring Respiratory Inductive Plethysmography (RIP), Medcare Flaga, Iceland] placed over the rib cage and abdomen (two channels). Snoring was detected with a vibration snore sensor (one channel) and body posture with a body position sensor (one channel). Airflow was monitored using an oral thermistor (oral flow sensor, Medcare Flaga, Iceland) placed in front of the mouth and a nasal cannula/pressure transducer (21in/53cm, Medcare Flaga, Iceland) inserted in the opening of the nostrils and linked to independent channels. Both of them were supported by hooking their leads behind the ears and taping them to the sides of the face.

Thermally sensitive devices such as thermistors cause a minimum amount of disturbance to the patient during sleep. However, the flow signal they provide is not a direct measure of actual flow [14] and thus, the detection of airflow provide only qualitative information [15]. On the contrary, the signal obtained by nasal cannula/pressure transducer has been shown to be comparable in both shape and amplitude to that of a conventional pneumotachograph [16]. In consequence, this non-obtrusive device is ideal not only for qualitative measurements but also for quantitative monitoring of respiration during sleep [17]. Cross-contamination between the oral and

nasal channel was meticulously excluded by regular testing during polysomnographic calibration. Thus, we asked subjects to breathe normally and exclusively through the nose for 30 s and subsequently through the mouth for another 30 s in both supine and right lateral postures so that we could verify that each sensor was activated exclusively. We continuously checked sensors during the recording to avoid dislodgement.

All variables were recorded with a digital acquisition system (Somnologica 3.3, Medcare Flaga, Iceland).

Analysis

Sleep stage was scored manually in 30-s epochs [18]. Obstructive respiratory events were scored using standard criteria [15, 19] by an experienced technician. Thus, apnoea was defined as the absence of airflow for more than 10 s in the presence of continued respiratory efforts [15]. Hypopnoea was defined as the reduction in chest wall movement to an amplitude that was smaller than approximately 70% of the baseline level, lasting more than 10 s, and leading to a decrease in hemoglobin saturation of at least 4% [19]. The number of episodes of apnoeas and hypopnoeas per hour of sleep is referred to as the apnoea-hypopnoea index (AHI). Obstructive sleep apnoea was diagnosed if AHI was >5 [15].

Route of breathing was evaluated by using the oral and nasal sensors signals to classify each 30-s epoch as nasal, oral or oro-nasal breathing epoch [20]. Nasal breathing epoch was defined as an epoch containing ≥ 3 consecutive phasic signals on the nasal sensor only. Oral breathing epoch was defined as an epoch containing ≥ 3 consecutive phasic signals on the oral sensor only. Oro-nasal breathing epoch contained ≥ 3 consecutive phasic linked signals on both the nasal and oral sensors. Epochs containing apnoeas, hypopnoeas, arousals, movements and signal artifacts were excluded from analysis. The occurrence of nasal, oral and oro-nasal breathing epochs was expressed as a percentage of the total sleep epochs (TSE) analyzed [20]. Body posture was expressed as a percentage of total sleep time.

Quantitive data are reported as mean ± SD. The normality of data distribution was assessed by the Kolmogorov-Smirnov test. Comparison of data between apnoeics and snorers was carried out using the unpaired t-test. Relationships between oral or oro-nasal breathing epochs and various variables were investigated by performing simple linear

regression analysis for each variable separately. Multiple linear regression analysis was performed to identify the variables independently related to oral or oro-nasal breathing epochs. All variables that significantly correlated with oral or oro-nasal breathing epochs in simple linear regression analysis were the independent variables included in the model. The stepwise procedure was used to select the best model. A p value of less than 0.05 was considered to indicate statistical significance.

Results

Of 62 snorers (38 men) initially enrolled in the study, eligible for further analysis were considered 41. Twenty-one snorers were excluded (9 had nasal resistance values exceeding normal limits in seated and/or in supine position, 3 had more than three central apnoeas per hour, 2 had central apnoeas more than five percent of total apnoeas detected, and 7 slept less than 3 hours).

The remaining 41 subjects (25 men, mean age 51.3±14.5, mean body mass index 31.5±5.7 $kg \cdot m^{-2}$) had nasal resistance within the normal range in erect seated (mean 1.86±0.45 $cmH_2O \cdot L^{-1} \cdot s$, range 1.10-2.60 $cmH_2O \cdot L^{-1} \cdot s$) and in supine position (mean 2.3±0.45 $cmH_2O \cdot L^{-1} \cdot s$, range 1.30-2.90 $cmH_2O \cdot L^{-1} \cdot s$). The mean total sleep time was 301.0±55.5 min and the mean sleep efficiency was 89.3±10.9 %. The mean total sleep epochs analyzed were 434.5±173.0 (range 102-731 epochs). Total sleep time was divided in the following sleep stages: stage 1, 4.3±4.0 %; stage 2, 76.9±10.4 %; stage 3, 2.8±4.6 %; stage 4, 1.5±3.6 %; and REM 14.4±7.5 %. The subjects spent 50.5±29.3 % of total sleep time in supine position and the rest in lateral positions. Prone position was seldom detected.

Obstructive sleep apnoea syndrome was diagnosed in 28 patients (apnoeics). Their mean AHI was 27.6±5.2 $events \cdot h^{-1}$ (range 8.3-103.1 $events \cdot h^{-1}$). The remaining 13 subjects (snorers) had mean AHI 1.3±0.3 $events \cdot h^{-1}$ (range 0.5-4.7 $events \cdot h^{-1}$). Table 1 illustrates anthropometric data, sleep parameters and nasal resistance in snorers and apnoeics.

Nasal breathing epochs were more frequent in snorers than in apnoeics ($p<0.001$; fig.1a), whereas the inverse was true for oral ($p=0.004$; fig.1b) and oro-nasal breathing

($p<0.001$; fig.1c) epochs. Oral breathing epochs were rare in snorers (0.02 ± 0.08 % of TSE) and apnoeics (2.3±3.9 % of TSE).

In apnoeics, 41.4 % of the oral or oro-nasal breathing epochs occurred in the first quarter of time encompassed between two events (i.e., after apnoeas/hypopnoeas), and 32.2 % in the last quarter of time (i.e., immediately before apnoeas/hypopnoeas); 12.8 % and 13.6 % of the oral or oro-nasal breathing epochs occurred in the second and third quarter of time.

Relationships between occurrence of oral or oro-nasal breathing epochs and polysomnographic or anthropometric variables are shown in tables 2 and 3, respectively. As can be seen, oral breathing epochs were positively related with AHI (fig.2a), mean and longest duration of apnoea-hypopnoea, and inversely related to average and lowest oxygen saturation. In addition, the relationship between oro-nasal breathing epochs and AHI (fig.2b), mean and longest duration of apnoea-hypopnoea and body mass index was positive, whereas average and lowest oxygen saturation were inversely related to oro-nasal breathing.

Results of the forward-stepwise multiple linear regression analysis are summarized in tables 4 and 5. All variables significantly related to oral (table 2) and oro-nasal breathing epochs (table 3) in simple linear regression analysis, were the independent variables included in the models. Oral breathing epochs were independently related only to AHI (higher in patients with increased AHI). AHI explained 44.3 % of the variance of oral breathing epochs (R^2=0.443; table 4). Oro-nasal breathing epochs were independently related to AHI (higher in patients with increased AHI) and body mass index (higher in patients with increased body mass index). AHI accounted for almost all of the variance (R^2 = 0.736) whereas body mass index for only a small amount of the variance (R^2 = 0.036) explained by the model (R^2 = 0.772; table 5).

Discussion

The main findings of the present study are: 1) patients with OSAS demonstrate an increased proportion of sleep time breathing orally or oro-nasally in comparison with snorers, 2) OSAS severity expressed by the apnoea-hypopnoea index is a major determinant of time spent breathing orally and oro-nasally, and 3) body mass index is

also an independent contributor to the time spent breathing oro-nasally but its contribution is small.

In this study, it is impressive that AHI determines a significant part of oral breathing epochs (R^2=0.443; table 4) and simultaneously constitutes such a powerful predictor of oro-nasal breathing epochs accounting for more than two thirds of their variance (R^2 = 0.736; table 5). A pathophysiological mechanism is inevitably implied. It has been noted that apnoeas tend to be followed by large mouth breaths [3], as if the suffocated patients try to compensate for the lack of air. These breaths were excluded from analysis in the majority of cases as they were part of epochs containing apnoeas-hypopnoeas. During intervals of undisturbed sleep between apnoeas/hypopnoeas, episodes of oral and oro-nasal breathing epochs reappeared. Interestingly, these episodes were more frequent after and before an apnoea/hypopnoea event, as if there were an affinity between oral or oro-nasal breathing epochs and apnoeas/hypopnoeas. Accordingly, it is plausible to consider that the appearance of oral or oro-nasal breathing triggers a vicious cycle in which a number of other factors contribute to further increase of apnoeas/hypopnoeas and thus, to more frequent mouth breathing. These factors are associated either with mouth opening/breathing or absence of nasal breathing. Indeed, it is well documented that mouth opening increases the propensity to airway collapse [21-23]. However, mouth opening does not necessarily imply mouth breathing because it has been shown that the tongue and soft palate can be in close apposition with concomitant preservation of nasal breathing [21]. On the contrary, the reverse condition is intuitively obvious [24]. When the oral thermistor detects the presence of oral airflow, opening the mouth is an evident prerequisite given that the possibility of cross-contamination by nasal airflow is minimal. Therefore, in this study, mouth breathing was considered to coincide with different degrees of mouth opening. In addition, there is evidence suggesting that irritation of nasal airflow-sensitive receptors during nasal breathing may be important in maintaining upper airway patency by increasing oro-pharyngeal muscle activity. In fact, WHITE *et al* blocking these receptors using 4% lidocaine local anesthesia provoked a four fold increase in the sleep disordered breathing events [25], whereas BASNER *et al* measured increased genioglossal and alae nasi electromyographic activity in awake humans breathing through the nose [26]. The latter finding was not corroborated by authors who,

subsequently, confirmed only the increase in alae nasi activity [27, 28]. Therefore, it is possible that the above mentioned vicious cycle, along with nocturnal hypoxemia and sleep fragmentation may progressively influence the ventilatory control mechanisms of patients with OSAS [11], finally affecting the breathing route that patients use during sleep and establishing a frequent oral or oro-nasal breathing.

Interestingly, body mass index, albeit weak, was a determinant of oro-nasal breathing. We suppose that as body mass index increases, adipose tissue deposition under the jaw rises rendering jaw opening easier and favoring oro-nasal breathing [28].

Three other studies have examined the correlation between apnoeas and mouth breathing or mouth opening. GLEESON *et al* [3] investigated breathing route during sleep in 14 healthy individuals. Although, nasal resistance was not measured in all the participants (8 out of 14 subjects) and the measurements took place several months after the time sleep studies were conducted, the authors concluded that the proportion of mouth breathing was significantly higher in subjects with apnoeic episodes (4 men who had 3 to 9 apnoeas per hour). In another study, HOLLOWEL and SURATT [24] demonstrated that during sleep the jaws of patients with OSAS were open more than those of normal subjects and opened further at the termination of apnoeas. The authors pointed out that the greater jaw opening in patients with OSAS could destabilize and compromise upper airway. Moreover, FITZPATRICK *et al* [29] studied 12 healthy subjects who underwent overnight polysomnography with a face mask with independent oral and nasal compartments and forced them alternatively to either oral or nasal breathing. The occurrence of upper airway obstruction episodes was significantly higher in oral ventilation (apnoea-hypopnoea index 43±6) than in nasal ventilation (apnoea-hypopnoea index 1.5±0.5).

Some possible weaknesses of the current study must be acknowledged and deserve consideration. First, the instrumentation of nasal cannula/pressure transducer and oral thermistor to detect airflow presents some drawbacks that have been thoroughly discussed previously [20]. Although these devices are non-obtrusive and easily tolerated, they cannot quantify ventilation, partly because their signal-flow relationship is non-linear. Indeed, the nasal cannula/pressure transducer has a quadratic pressure-flow relationship that may result in underestimation or absence of ventilation at low flows

[15]. In addition, the signal-flow relationship of the oral thermistor is logarithmic that may result in overestimation of ventilation at low flows [15]. Accordingly, it would be possible that oral only breathing may still have a nasal component, and any detection of oral only breathing might actually be scarce. Therefore, the frequency of oral only breathing epochs could be overestimated in the present study, although it was already rarely encountered. Moreover, with the instrumentation used, assessment of the heterogeneity of oro-nasal breathing pattern is unfeasible since it is impossible to distinguish different percentages of oral and nasal components. Second, sensor dislodgement from the nares or from the mouth could be a potential problem. The incidence of signal loss has been reported to reach approximately 5% of cases [17]. In our study, only three subjects had a brief loss of signal in some part of the recording that never exceeded 5 min. The loss of signal was easily recognized and corrected by the technician on duty. However, it is likely slight deviations in thermistor position may not have been avoided and this may have then resulted in nasal airflow contamination of the oral signal. Finally, reliable posterior rhinomanometry measurements were not available in all patients. In the present study, nasal resistance was measured in seated and supine positions using both posterior and anterior rhinomanometry. Key point in posterior rhinomanometry is to keep the soft palate elevated and the upper surface of the tongue away from the palate so as to allow free communication between the oropharynx and the oral cavity. Insufficient palatal control and cooperation were the reasons of poor reproducibility that was encountered in 6 subjects of the present study. On the contrary, anterior rhinomanometry gave reliable and reproducible findings in all patients. Indeed, anterior rhinomanometry requires minimal cooperation, thus increasing reproducibility and minimizing failure rate, although posterior nasal malformations cannot be determined [12].

Mouth breathing, as it is indicated with an oral thermistor, appears to be one of the signs of OSAS which probably must be taken into account when we assess the clinical presentation of a subject referring to a sleep disorders center. However, interpretation of this sign warrants attention, because self-reports of mouth breathing during sleep [10] and even during wakefulness [4] are not frequently reliable as they may diverge from sleep study data [4, 10].

In conclusion, this study illustrates that apnoeics and snorers without nasal obstruction differ in the breathing route used during sleep. Apnoeics tend to spend more time than snorers breathing either orally or oro-nasally. Additionally, as the severity of OSAS increases the proportion of oral and oro-nasal breathing epochs appears to rise.

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Table 1. Anthropometric data, nasal resistance values and sleep parameters in snorers and apnoeics.

	Snorers (n=13)	Apnoeics (n=28)
Age yrs	45.6±19.4	54±10.9
BMI $kg \cdot m^{-2}$	28.9±6.6	32.7±4.9 *
Nasal Resistance seated $cmH_2O \cdot L^{-1} \cdot s$	1.76±0.57	1.90±0.39
Nasal Resistance supine $cmH_2O \cdot L^{-1} \cdot s$	2.21±0.49	2.34±0.43
Average oxygen saturation %	94.9±2.3	91.8±4.5 *
Lowest oxygen saturation %	89.3±2.9	77.8±10.1**
Mean duration of apnoea-hypopnoea s	18.8±5.8	23.5±5.8 *
Longest duration of apnoea-hypopnoea s	27.5±7.9	58.2±19.6 **
Total sleep time min	304.5±68.3	301.3±48.2
Sleep efficiency %	88.4±13.3	89.3±9.7
Non-REM min	248.9±57.8	263.4±43.8
REM min	55.6±21.8	36.9±24.1 *
Sleep time in supine posture min	155.5±104.0	150.0±90.8

Data are presented as mean±SD. *: $p < 0.05$ *versus* snorers; **: $p < 0.001$ *versus* snorers.

Table 2. Simple linear regression analysis models for oral breathing.

Independent variable	B	SE	r^2	p
Apnoea-hypopnoea index	0.078	0.014	0.443	<0.0001
Lowest oxygen saturation %	-0.204	0.044	0.351	<0.0001
Average oxygen saturation %	-0.477	0.107	0.338	<0.0001
Longest duration of apnoea-hypopnoea s	0.058	0.023	0.137	0.017
Mean duration of apnoea-hypopnoea s	0.196	0.084	0.123	0.025
Body mass index $kg \cdot m^{-2}$	0.169	0.093	0.079	0.075
Age yrs	0.003	0.038	0.000	0.937
Gender (1 = male, 2 = female)	-0.776	1.115	0.012	0.491

B: parameter estimate; SE: standard error.

Table 3. Simple linear regression analysis models for oro-nasal breathing.

Independent variable	B	SE	r^2	p
Apnoea-hypopnoea index	0.577	0.055	0.736	<0.0001
Lowest oxygen saturation %	-1.474	0.209	0.560	<0.0001
Average oxygen saturation %	-2.768	0.607	0.331	<0.0001
Longest duration of apnoea-hypopnoea s	0.489	0.120	0.298	<0.0001
Mean duration of apnoea-hypopnoea s	1.407	0.460	0.194	0.004
Body mass index $kg \cdot m^{-2}$	1.488	0.498	0.186	0.005
Age yrs	0.121	0.217	0.008	0.581
Gender (1 = male, 2 = female)	-10.227	6.204	0.065	0.107
Nasal resistance in supine position $cmH_2O \cdot L^{-1} \cdot s$	7.892	6.920	0.032	0.261

B: parameter estimate; SE: standard error.

Table 4. Multiple linear regression analysis for oral breathing.

Model 1 ($R^2 = 0.443$)

	B	SE	p	PC
Constant	-0.662	0.579	0.261	_
Apnoea-hypopnoea index	0.078	0.014	<0.001	0.666
Average oxygen saturation %				-0.254
Lowest oxygen saturation %				-0.019
Mean duration of apnoea-hypopnoea s				0.006
Longest duration of apnoea-hypopnoea s				-0.069

Selection of variables was made by the forward stepwise procedure. Only apnoea-hypopnoea index fulfilled the criterion ($p<0.05$) for variable entry in the model. R^2: total variance explained by the model; B: parameter estimate; SE: standard error; PC: partial correlation

Table 5. Multiple linear regression analysis for oro-nasal breathing.

	Model 1 (R^2 = 0.736)				Model 2 (R^2 = 0.772)			
	B	SE	p	PC	B	SE	P	PC
Constant	12.037	2.280	<0.001	_	-8.142	8.621	0.351	_
Apnoea-hypopnoea index	0.577	0.055	<0.001	0.858	0.538	0.055	<0.001	0.848
Body mass index $kg \cdot m^{-2}$				0.365[#]	0.676	0.280	0.021	0.365
Average oxygen saturation %				-0.063				0.108
Lowest oxygen saturation %				0.028				0.205
Mean duration of apnoea-hypopnoea s				-0.016				0.048
Longest duration of apnoea-hypopnoea s				0.041				0.035

Selection of variables was made by the forward stepwise procedure. Only apnoea-hypopnoea index and body mass index fulfilled the criterion ($p<0.05$) for variable entry in the model. R^2: total variance explained by the model; B: parameter estimate; SE: standard error; PC: partial correlation. [#]: $p<0.05$.

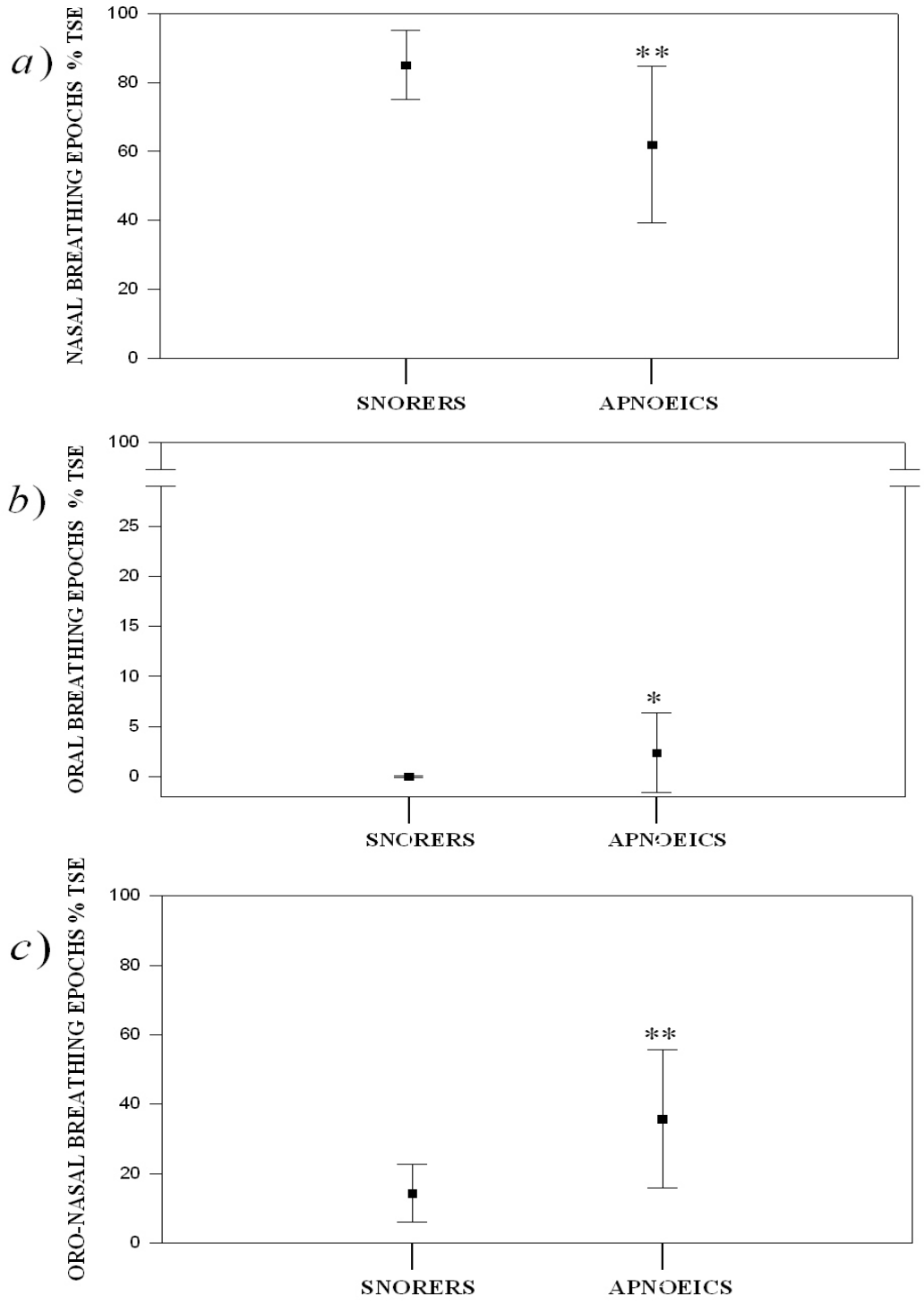
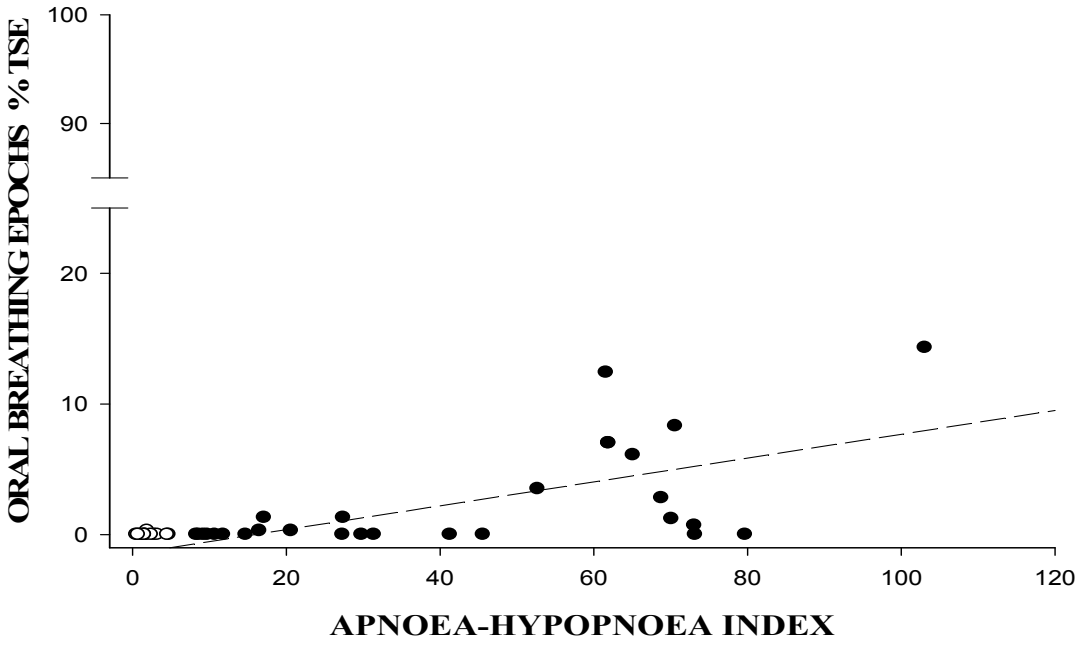

a)
NASAL BREATHING EPOCHS % TSE
100
80
60
40
20
0
**
SNORERS
APNOEICS
b)
ORAL BREATHING EPOCHS % TSE
100
25
20
15
10
5
0
*
SNORERS
APNOEICS
c)
ORO-NASAL BREATHING EPOCHS % TSE
100
80
60
40
20
0
**
SNORERS
APNOEICS

Figure 1. Occurrence (mean ± SD) of a) nasal b) oral and c) oro-nasal breathing epochs in snorers and apnoeics during sleep. TSE: total sleep epochs. *: p=0.004 *versus* snorers; **: p<0.001 *versus* snorers

a)



b)

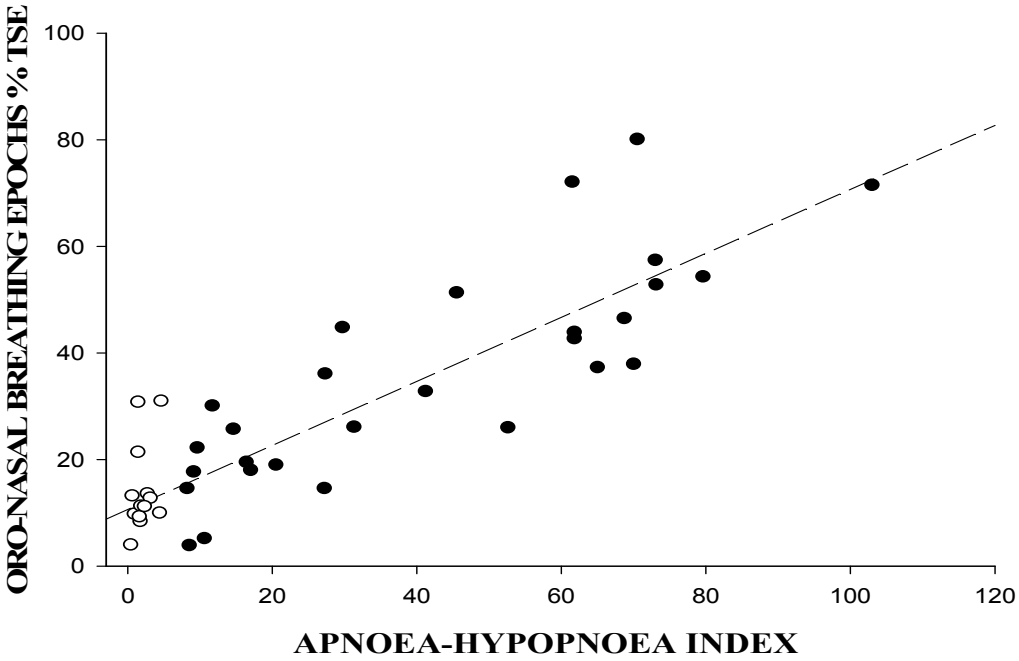


Figure 2. Relationship between apnoea-hypopnoea index and a) oral (R^2 = 0.443; $p<0.0001$) and b) oro-nasal (R^2 = 0.736; $p<0.0001$) breathing epochs in snorers (°) and apnoeics (•). TSE: total sleep epochs.