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Research Article

The Sleep Position 'Aid and Toll' in Obstructive Sleep Apnea: A Lung-Brain Axis Perspective

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Abstract

Background: Supine sleep position has been shown to induce or aggravate sleep-breathing disorders, namely obstructive sleep apnea. In patients suffering from moderate-to-severe Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS), sleeping supine may significantly rise the Apnea-Hypopnea Index (AHI) scores, often causing profound oxygen desaturation. As such, OSAHS-related complications would emerge secondary to chronic hypoxemia. In a fraction of cases where the condition is even more-strongly dependent to sleeping position, the term positional-OSAHS would apply. In this study, we examined the sleep position and other polysomnographic bioparameters in sleep apnea patients, in relation to their AHI and the incidence of concurrent medical conditions. This report also discussed the emerging evidence regarding the potential effects of sleep position and OSAHS on the brain.

Materials and Methods: A retrospective analysis was done on 78 patients (mean age of 43.6 years), demonstrating increased AHI in supine compared to other positions, which referred to our sleep disorders unit and underwent polysomnography during 2013-2015. They were divided into mild (n=31) and moderate-to-severe (n=47) groups depending on their AHI scores (5≤AHI≤15 and AHI>15, respectively).

Results: There found to be a significantly higher prevalence of hypertension, coronary artery disease and cerebrovascular disease in moderate-to-severe OSAHS patients. Likewise, Epworth Sleepiness Score (ESS) was notably higher and the mean oxygen saturation was lower in moderate-to-severe compared to mild OSAHS patients. Moderate-to-severe OSAHS cases were spending more time in supine rather than non-supine positions during sleep. AHIs and arousal index were found to be higher in supine position.

Conclusion: Sleeping supine seems to worsen OSAHS and increase AHI, subsequently contributing to medical comorbidities. In case of positional-OSAHS, treatments should be individualized to prevent position-dependent airway collapse during sleep. Patients' awareness on the significance of sleep position in preventing OSAHS-related complications needs to be improved, and medically-proven interventions to maintain non-supine position during sleep should be advised in such patients.

Keywords: Obstructive sleep apnea; Sleep position; AHI; Polysomnography; Lung-brain axis

Introduction

Sleep-Disordered Breathing (SDB) and Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) in particular, are among the frequently encountered disorders in the practice of sleep medicine with their prevalence reaching up to 5% in men and 2% in women aging 20 years and beyond [1]. OSAHS encompasses breathing pauses and significant airflow limitations, referred to as apneas and hypopneas, respectively. Both event types follow a common pathophysiology and tend to leave patients with medical and neurocognitive consequences in long run. The severity of OSAHS can be quantified through various approaches such as measuring the number of apnea and hypopnea events per hour of sleep, number and depth of the oxygen desaturation during sleep or the presence and severity of diurnal symptoms, namely Excessive Daytime Sleepiness (EDS) [2].

Some baseline demographic particulars such as advanced age, male gender, high Body-Mass Index (BMI), short neck, use of drugs, genetic factors, upper airway anatomy and hyper-reactivity, and the habitual dominant sleep position are regarded as contributing factors to the development of OSAHS. Regardless of the cause, the outcome is partial or complete upper-airway collapse and consequent O2 desaturation during sleep. Oropharynx is perhaps the most common site where obstruction develops [3,4].

Sleep position and the role of gravity are among mechanical contributors to the development of upper-airway collapse during sleep. Across SDB and healthy subjects, the caliber of upper-airway is subject to decrease in supine sleep position and this phenomenon becomes critical in OSAHS [5]. The supine position leads to the back

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Table 1: Demographic and clinical features of enrolled subjects in mild and moderate-to-severe OSAHS groups. Values represent mean± standard error of mean (M±SEM). *P* values≤0.05 indicate statistically-significant differences between groups.

Demographic and clinical features of enrolled subjects	Mild OSAHS 5≤AHI≤15 (n=31)	Moderate-to-severe OSAHS AHI>15 (n=47)	*P* value
Age (year) (M±SEM)	44.23±7.89*	47.30±10.4	0.2
Body-Mass Index (kg/m^2)	29.18±5	29.4±4.23	0.8
Epworth Sleepiness Scale(M±SEM)	10.12±4.3	17.07±2.5	0.0
O_2 saturation % (M±SEM)	89±3.5	81.26±2.5	0.0
Minimum O_2 saturation % (M±SEM)	72.61±2.2	63.34±3.5	0.01
Sleep Efficiency (%)	80.58±8.6	75.45±11.3	0.0
Arousal Index (M±SEM)	15.94±8.7	24.14±11.1	0.001
Incidence of hypertension (%)	18.6	38.6	0.0
Incidence of Coronary Artery Disease (%)	4.2	12.2	0.0
Incidence of Cerebrovascular Disease (%)	0.4	2.6	0.0
Supine Sleep Position (min, M±SEM)	170.31±31.2	225.32±21.87	0.0
Non Supine Sleep Position (min, M±SEM) ±SEM)±SEM))	210.65±25.3	130.25±36.55	0.0

shift of the tongue and narrowing of the soft palate-pharyngeal air passage [3,4].

The term 'position-dependent-or positional-OSAHS' would apply when the supine AHI scores are twice higher than the AHI in non-supine positions [6,7]. As inferred from the terminology, the severity of the disease is markedly increased in the supine position.

From the neuroscience perspective, a perivascular pathway has been introduced by which interstitial solutes including amyloid β (Aβ) are removed from the brain parenchyma. In this so-called "glymphatic" pathway, a large amount of subarachnoid cerebrospinal fluid (CSF) enters the parenchyma along the perivascular spaces, exchanges with the Interstitial Fluid (ISF), and exits via para-venous pathway [8].

Accumulating evidence have indicated that the function of glymphatic pathway is influenced by the arousal level. In the other words, natural sleep increases exchange of CSF with ISF compared with the awake state [9]. Such novel insights corroborate other study findings indicating the restorative function of sleep [10,11]. On the other hand, with reference to sleep position-related functions, a recent investigation has suggested the brain's waste removal capacity in terms of brain glymphatic transport as well as amyloid beta (Aβ) clearance. Translational research in animal models have employed dynamic contrast-enhanced Magnetic Resonance Imaging (MRI) and kinetic modeling in anesthetized rodents, showing that most glymphatic transport occurs in the lateral-compared to supine position. In addition, optical imaging and radiotracer studies showed that Aβ clearance tend to be lower in supine sleep position compared to lateral [12].

Based on these findings, and considering the fact that most apnea/hypopnea events occur in supine position, it might be speculated that lateral sleep position possibly acts as a protective factor for the brain by providing an optimal condition for removing waste materials from its parenchyma.

OSAHS patients are found to suffer from neurocognitive function decline in long-term [5,13,14]. The question of "in addition to respiratory events, to what extent neurocognitive consequences of OSAHS is related to patients' sleep position and the impaired function of the brain glymphatic system?' deserves systematic research endeavors.

Though a significant number of patients who refer to our sleep facility are found to have SDB, their awareness on the significance of sleeping position is scant. The emerging need to define our OSAHS patients' sleeping position, its clinical impact, and potential remediative strategies prompted us to pursue with the present study.

The current investigation attempted to compare cases with mild and moderate-severe position-dependent OSAHS in relation with their medical comorbidities, and polysomnographic features. Further to this, the present report tried to review current remediative approaches and emphasize the significance of sleep position not only in the severity of OSAHS but also brain health.

Material and Methods

Subjects

We conducted a retrospective study on patients who underwent polysomnographic evaluation at our sleep disorders unit (Namazi Hospital, Shiraz University of Medical Sciences, Shiraz, Iran) during 2013-2015. Demographic data sleep questionnaires and polysomnographic study reports of a total number of 138 patients were reviewed. The AHI cut-off of 5 was used to isolate patient files with OSAHS. AHI score of all patients in non-supine and supine positions were evaluated. Positional-OSAHS was defined when supine-AHI was at least twice higher than non-supine AHI. Accordingly, we extracted the files of 78 patients who fulfilled our inclusion criteria. Based on the most recent American Academy of Sleep Medicine (AASM) task force criteria [15] and depending on the values of 5≤AHI≤15 and AHI>15, patients were categorized into mild (n=31) and moderate-to-severe (n=47) OSAHS, respectively. Patients diagnosed with Upper-Airway Resistance Syndrome (UARS), central- or mixed-type sleep apneas or Periodic Limb Movements during Sleep (PLMS) were excluded from the analysis.

Our polysomnography setup used a built-in position sensor (accelerometer) which enabled determining sleeping positions and positional transitions of the subjects during sleep. The Persian

Table 2: Snore analysis with respect to sleeping position in moderate-to-severe OSAHS patient group. Values represent mean± standard error of mean (M±SEM).

Snore analysis	All	Prone	Supine	Left lateral	Right lateral	Up right
Snore, M±SEM (Mean Index)	2583±535 (329.2)	135±42 (12.6)	2242±135 (302.1)	130±23 (11.3)	70±35 (43.9) (7.4)	25±12 (3.1)
Absolute Snore (min, M±SEM)	64.6±11.5	4.5±2.3	58.3±7	2.6±1.5	1.8±0.7	0.12
Episodic Snore (min, M±SEM)	146.40±12	3.2±1	138.50±16	3±1.6	2.5±0.18	0.16
Episodic Snore (% Sleep time)	66.70±18.5	-	-	-	-	-

Table 3: Descriptive analysis of the respiratory events in relation with sleeping position in moderate-to-severe OSAHS patient group. Values represent mean± standard error of mean (M±SEM).

Respiratory events	Prone position	Supine position	Left lateral	Right lateral	Upright position
Sleep Time Fraction % (M±SEM)	2.1±0.8	85.7±4.5	2.8±1.5	3.9±1.1	5.5±2.4
Total events, M±SEM (Mean Index)	8.5±2.3 (1.25)	286±22 (42.2)	9.1±2.2 (1.34)	5.5±2.1 (0.82)	12±8.5 (1.8)
Obstructive apneas, M±SEM (Mean Index)	2.8±0.8 (0.41)	108±10 (15.8)	3±1.5 (0.45)	2.5±0.9 (0.37)	3.5±1.1 (0.52)
Hypopnea, M±SEM (Mean Index)	5.7± 1.9 (0.84)	178±11 (25.7)	6.1±2.2 (0.91)	3±1.8 (0.44)	8.5±2.3 (1.3)

version of Epworth sleepiness scale (ESS) was used to assess Excessive Daytime Sleepiness (EDS) [16]. Informed consents were obtained from all patients during their polysomnographic assessment.

Polysomnography

Full-night polysomnography evaluations were done at our hospital-based sleep disorders laboratory using the SOMNO screen TM system and DOMINOTM analysis software. A technician was present during the entire recordings. Acquired data were documented to a special sleep recording computer and manually scored by a sleep specialist according to the latest AASM manual for sleep scoring and analysis. Electroencephalogram (F3-A2, F4-A1, C3-A2, C4-A1, O1-A2 and O1-A2), electro-occulogram (right and left), surface electromyogram (sub mental/right and left anterior tibialis), breathing effort (chest and abdomen), air intake (mouth/nose air flow), snoring sounds, oxygen saturation, plethysmogram, electrocardiogram, heart rate and sleeping position were recorded during all polysomnography studies.

Apneas and hypopneas were scored by at least 10-second airflow cessation and the reduction in ventilation by at least 50% causing decreased arterial O_2 saturation of 4% or more due to total or partial airway obstruction, respectively [17]. The sleep position data and other sleep-related biopapameters from all included patients were extracted to undergo comparative analyses.

Statistical analysis

All statistical analyses were done using the SPSS statistical software (version 19.0 for Windows). Statistical differences were considered significant at $p<0.05$ cut-off. Independent t test was used to determine the significance of differences between the groups. When the data lacked normal distribution, Mann-Whitney U test was applied. In addition, Chi-square (X^2) and Fisher Exact Chi-square tests were employed to analyze the categorical data. Arithmetic Means And Standard Errors Of Mean (M±SEM) were calculated for all obtained values.

Results

A total of 78 cases with position-dependent OSAHS (63 male), with a mean age of 43.6±14 years, were enrolled in our analysis. Based on the total AHI scores, patients were classified into two groups, i.e. mild (n=31) and moderate-to-severe (n=47) OSAHS. The demographic and clinical data of patients are outlined in (Table 1).

The comparison of the incidence of comorbidities indicated a significant difference between the groups, favoring less concurrent medical conditions in mild vs. moderate-to-severe OSAHS patients (p=0.04, 0.02 and 0.01 for hypertension ,coronary artery disease and cerebrovascular disease, respectively). The mean±SEM ESS values in mild and moderate-to-severe OSAHS groups were 10.12±4.3 and 17.07±2.5, respectively (p=0.02). In 18 out of the 47 moderate-to-severe OSAHS patients, ESS values were higher than 21, suggesting significant EDS. Likewise, the analysis revealed a significant difference in mean ± SEM O_2 saturation between mild and moderate-to-severe OSAHS groups (89±3.5 and 81.26±2.5, respectively; p=0.05). The nadir oxygen saturation was lower in moderate-to-severe as compared to mild OSAHS patients (63.34±3.5 vs. 72.61±2.2, p=0.001). According to the position analysis in polysomnographic studies, the time passed in supine position was higher in moderate-to-severe group (p=0.03).

Given the significance of findings in moderate-to-severe OSAHS patients in relation to their sleep position, their snore analysis and sleep-related respiratory events with respect to sleeping position are in turn summarized in Tables 2 and 3.

Discussion

Positional-OSAHS

The most significant outcome of this retrospective analysis is to highlight the prevalence of position-dependent OSAHS among our patients who were found to have SDB. Moreover, the relation between moderate-to-severe positional-OSAHS with medical comorbidities (including hypertension, coronary artery disease and cerebrovascular disease) turned to be of note. With a higher frequency of snores and sleep-related respiratory and desaturation events in moderate-to-severe OSAHS, these patients tend to spend more time in supine than non-supine positions during sleep. This perhaps re-emphasizes on the necessity to plan individual approaches to help these patients avoid supine position while asleep. With specific approaches in positional therapy of OSAHS such as tennis ball jacket, positional alarms and special pillows, we may remediate position-dependent symptoms in such patients [18]. Our study group indents to run a prospective study to revalidate the effect of sleep position in OSAHS on sleep parameters as well as functional and neurocognitive outcomes using a novel sleep position trainer and objective assessment measures.

OSAHS is characterized by repetitive episodes of upper-airway collapse during sleep. In supine sleep position, owing to the gravity effect and positional configuration, tongue and soft palate converge to narrow the functional pharyngeal area leading to complete or partial blockade of the air passage [4].

Considering the fact than much of apnea and hypopnea events tend to occur in supine posture in case of positional-OSAHS, maintaining non-supine positions during sleep is recommended in such patients [19]. Supine sleep position is linked with increased frequency of snores, respiratory events and O_2 desaturations in this patient group. The same was found to hold true in our present investigation. Generally, patients are stratified into positional and non-positional OSAHS [6,7,19-22]. Studies have shown that up to 60% of sleep apnea patients are classified as positional [21], which has been in agreement with our findings. Such a high incidence deserves a critical attention and position-related AHIs need to be carefully taken into consideration. The repetitive oxygen desaturations which occur following almost all apneas and many hypopneas, affect autonomic nervous system and cause a physiologic challenge on the brain. Consequently, frequent arousals and awakenings may take place. On the other hand, brady- and tachycardia occur concurrently or right after the apneic events leading to cardiovascular disorders such as dysrhytmias and blood pressure fluctuations during sleep potentially causing various type arrhythmias and hypertension [23].

Apneas and hypopneas which contribute to micro arousals, interferences in sleep, daytime fatigue and sleepiness are found to be more frequently seen in supine sleeping position among positional-OSAHS patients[24]. Our study demonstrated higher ESS scores in moderate-to-severe as compared to mild positional OSAHS patients. In 18 out of the 47 our moderate-to-severe OSAHS patients, ESS values were higher than 21, suggesting significant EDS.

In practice, evaluating the OSAHS patients based on the total AHI alone, without considering sleep-position related AHIs, would result in underestimating the significance of the position-related syndrome. Positional- OSAHS patients need to be carefully assessed for their AHI and oxygen saturation in the supine position.

Palliative measures including sleep position therapy would be an efficient auxiliary tool in addition to Continuous Positive Airway Pressure (CPAP) therapy and the use of oral appliances.

The lung-brain axis perspective

The idea of the interface between the lung and brain (the so-called lung-brain axis perspective) has appealed to the interest of our research group recently. Given the novel evidence supporting the role of sleeping position on the capacity of the brain in detoxifying wastes from the neural tissue, and the consequence of sleep disorders (namely OSAHS) on neurodegenerative diseases [25], research in this area looks intriguing.

Recently, a classical lymphatic drainage system in the central nervous system has been discovered [26]. These lymphatic vessels line the dural sinuses and express the markers of lymphatic endothelial cells. These structures transfer fluid and immune cells from the CSF to the deep cervical lymph nodes. Thus, it is the second step in the drainage of the ISF from the brain parenchyma into the periphery after glymphatic drainage. It is beneficial that we consider the importance of these two main CNS drainage systems for evaluation the relationship among body posture, sleep and neurological diseases [26].

From the translational-neuroscience standpoint, recent studies have investigated the relationship between body posture during sleep and brain fluid and solute homeostasis. Key discussions in this context revolve around the recently described glymphatic pathway. This CSF-ISF exchange pathway removes waste materials such as Aβ from interstitial space. It is noteworthy that astrocytes in the perivascular end feet play a main role for fluid and solute movement. In fact, water channel aquaporin-4 (AQP4) in astrocytes is responsible for creating a bulk fluid flow and consequently interstitial solute clearance [8].

Lulu Xie et al. investigated the function of glymphatic pathway in sleep. By evaluation of tetramethylammonium diffusion and two-photon imaging in live mice, they postulated the key role of interstitial space in the clearance of Aβ which has been pooled up during sleep [9].

In the same vein, Hedok Lee and colleagues evaluated the influence of body posture on brain glymphatic transport in anesthetized rodents. By intrathecal administration of contrast/tracer followed by image analysis using MRI and fluorescent assay, they demonstrated that retention of the tracer was more in supine rather than non-supine postures. Moreover, they reported that Aβ clearance in lateral positions was higher than that in the supine posture [12].

More human studies are absolutely needed to corroborate animal study findings with regard to sleep position, sleep related respiratory events and the brain fluid homeostasis. To date, there is no publication about the effect of sleep position and sleep-disordered breathing on glymphatic transport in humans. Moreover, the relationship between body positions (especially head position) during sleep, the incidence of position-related sleep issues (OSAHS, in particular) and the rate of neurological disorders in human has not been investigated.

Palliative measures in positional OSAHS

A large body of evidence has supported the clinical benefits of commonly used clinical interventions (such as oro-pharyngeal repositioning surgeries, oral appliances and CPAP) to resolve symptoms of obstructive sleep apnea [27]. Ideally, a long-term interdisciplinary approach is required to improve sleep efficiency among OSAHS sufferers. Beside common practice, there exist some alternative auxiliary treatments which are increasingly being modified and field-tested to meet patients' needs and compliance [18]. The tennis-ball jacket and similar tools have been among the first designed positional therapies used in several variations such as positional alarms and special pillows. Though, they demonstrated favorable results, some modifications are needed to turn them into more efficient and compliant tools [28]. More recently, combinations of positional therapies such as sleep position trainer and mandibular advancement devices have been tried to accommodate further efficiency and compliance among positional OSAHS patients [18,27,28]. Patients' convenience and the resultant adherence to positional therapy is a key indicator of success in such an approach. The use of bulky mass in the back of patients while asleep (e.g. tennis-ball jacket, backpack or slumber-bump) arguably causes a significant discomfort. On the other hand, newer tiny devices using

an accelerometer-vibrator setup, may cause repeated arousals and awakenings, and hence negatively affect sleep integrity and efficiency [29]. Our research group has started to sketch up creative ideas to develop position therapy methods to serve patient's compliance, even further.

Conclusion

Patients with positional-OSAHS should be assessed and approached as an almost separate clinical entity and their treatment plans need to be individualized accordingly. Through this approach, OSAHS-related complications including medical, neurological and cognitive consequences, resulting from chronic hypoxemia, could be prevented at earlier stages.

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