

Prevalence of Positional Obstructive Sleep Apnea in Patients Undergoing Polysomnography and the Effect of Sleep Stage

Mahesh Shetty and M. Jeffery Mador

Introduction

Determining whether sleep apnea is positional or not has important therapeutic consequences as discussed elsewhere in this book. In this chapter, we will discuss the prevalence of positional sleep apnea in patients with sleep apnea, factors that make positional sleep apnea more likely in a patient with sleep apnea, and the effect of sleep stage on positional sleep apnea.

Prevalence

Obstructive sleep apnea (OSA) is a common disorder, estimated to occur in approximately 2 % and 4 % of middle-aged women and men, respectively (based on the presence of an abnormal sleep study plus self-reported sleepiness), in a landmark study published in 1993 [1]. The prevalence of sleep apnea in 2013 is likely greater than this based on the increase in obesity levels since then. A recent study examining changes in OSA prevalence using the same Wisconsin state cohort that was used in the original investigation show increases in prevalence rates from 14 % to 55 %

M. Shetty, M.D.

Division of Pulmonary, Critical Care and Sleep Medicine, University at Buffalo,
Buffalo, NY, USA

M.J. Mador, M.D. (✉)

Division of Pulmonary, Critical Care and Sleep Medicine, University at Buffalo,
Buffalo, NY, USA

Western New York Veteran Affairs Healthcare System,

3495 Bailey Ave, Buffalo, NY 14223, USA

e-mail: mador@buffalo.edu

Table 1 Prevalence of positional sleep apnea

Definition	Author	Total sample size	Number of pos SA	Number of non-pos SA	Prevalence of pos SA (%)
Lenient definition	Richard [8]	120	67	53	55.8
	Sunwoo [9]	91	65	26	71.4
	Teerapraipruk [10]	144	96	48	66.7
	Gillman [4]	100	63	37	63
	Oksenberg [11]	574	321	253	55.9
Strict definition	Gillman [4]	100	23	77	23
	Mador [5]	258	69	189	26.7

Pos SA positional sleep apnea, *non-pos SA* non-positional sleep apnea

in 2007–2010 compared to those obtained in 1988–1994 [2]. Positional sleep apnea defined as a worsening of sleep apnea in the supine posture compared to the non-supine posture is extremely common. The exact prevalence depends on the definition used to diagnose positional sleep apnea.

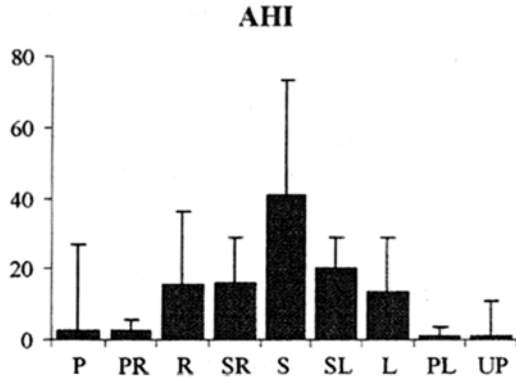
The usual definition of positional sleep apnea is a supine apnea-hypopnea index (AHI) that is greater than twice the non-supine AHI as initially described by Cartwright in 1984 [3]. However, another perhaps more clinically useful definition is to define positional sleep apnea as the presence of sleep apnea only in the supine posture. With this definition, the AHI in the non-supine posture is less than 5 per hour *and* less than 50 % of the supine AHI, while the overall AHI and supine AHI both exceed 5 per hour [4, 5]. This definition is useful because in such patients elimination of supine sleep should result in resolution of sleep apnea. With the original definition while demonstrating a clear positional tendency, the clinical importance is less clear. A reduction in AHI from 60 per hour in the supine posture to 29 per hour in the non-supine posture meets the definition for positional sleep apnea. However, complete elimination of supine sleep would not successfully treat sleep apnea. Thus, positional therapy in this type of patient could only be used as an adjunct measure. Identification of this positional tendency would still be clinically useful if it predicted the response to therapy to different modalities. There have been recent studies that suggest that mandibular advancement devices are more effective in positional sleep apnea patients compared to non-positional sleep apnea patients [6, 7]. Thus, identification of a positional component is potentially a simple way of phenotyping patients with sleep apnea identifying patients more likely to respond to some sleep apnea treatment modalities. Using the original more lenient definition, studies have found that the prevalence varies from 56 % to 71.4 % [4, 8–11], mean 62.6 % \pm 6.8 (SD) (Table 1). One of the studies examined the prevalence in a strictly Asian sample and found a prevalence of 67 % indicating that positional sleep apnea is common in non-Caucasian populations as well [10]. There have been some studies that have measured the prevalence data with the much stricter definition discussed above (ratio of the supine AHI and non-supine AHI greater than 2 along with a non-supine AHI less than 5 events per hour) [4, 5]. With this strict definition, the prevalence for positional sleep apnea was substantially less than with the original definition but still ranged from 23 % to 27 % (Table 1) [4, 5].

One of the abovementioned studies was a prospective cohort trial performed in Melbourne, Australia, on 100 consecutive OSA patients, and the prevalence was measured by both definitions [4]. When positional sleep apnea was defined using the lenient definition, the prevalence was 63 %, but when they used the stricter definition for positional sleep apnea, the prevalence decreased to 23 % [4]. In our study, the prevalence using the lenient definition was 58 % and decreased to 27 % with the stricter definition [5]. It is still noteworthy that even with the strict definition, positional sleep apnea is common (Table 1). Thus, methods that can successfully eliminate supine sleep and are tolerable have the potential to be an important treatment modality.

One methodologic point that needs to be considered is the effect that the polysomnographic procedure has on promoting supine sleep in the laboratory. During a sleep study, the patient is connected to a multitude of wires. The patient may try to sleep on his/her back in the sleep laboratory because they are concerned that other postures may promote lead dislodgement. In a prospective study in 12 positional sleep apnea patients, the patients were studied for 3 nights, once with standard PSG leads attached (PSG night) and the other 2 nights without any leads attached, but position was monitored with a position sensor [12]. The time spent supine was 56 % greater during the PSG night compared to the non-PSG nights [12]. This can artifactually magnify the severity of the patients' disease (when they have a positional component) compared to what would occur at home when supine sleep would be less.

There have also been significant differences in the AHI when tested in the right lateral compared to the left lateral position. A retrospective review performed in Turkey noted that the left lateral position had a higher AHI at 30.2 ± 32.6 per hour compared to the right lateral position at 23.6 ± 30.1 per hour that was statistically significant ($p < 0.001$) [13]. Another interesting study evaluated 105 sleep apnea patients in the sleep laboratory using a 9-position thoracic sensor. Positions identified were the left lateral (L), prone (P), prone left (PL), prone right (PR), right (R), supine (S), supine left (SL), supine right (SR), and upward position (UP) [14]. It was noted that the AHI gradually improved when the body position changed from the supine to prone position [14]. Thus, in this study, the AHI was worse in the supine posture, intermediate in the lateral posture, and best in the prone posture (Fig. 1). The AHI in the upright posture was similar to that observed in the prone position. The supine left or right was a position intermediate between the supine and lateral position and had an AHI that was similar to that observed in the lateral position. Similarly, the prone right and left was a position intermediate between the prone and lateral position and had an AHI similar to that observed in the prone position [14].

Fig. 1 AHI in different body positions. *L* left lateral, *P* prone, *PL* prone left, *PR* prone right, *R* right, *S* supine, *SL* supine left, *SR* supine right, and *UP* upward position. Figure adapted from *Pneumologia*, 2011. **60**(4): p. 216–21 [14] with permission



Factors That Predict Occurrence of Positional Sleep Apnea

Multiple factors have been evaluated in the last few decades that are strongly associated or directly affect the prevalence of positional sleep apnea.

Sleep Apnea Severity

As recommended by the American Academy of Sleep Medicine, sleep apnea severity is classified into three categories based on the AHI [15]. Mild sleep apnea has an AHI between 5 and 15 events per hour; moderate sleep apnea, an AHI between 15 and 30 events per hour; and severe sleep apnea, an AHI more than 30 events per hour [15].

There have been several studies performed to evaluate the prevalence of positional sleep apnea among the different severities of sleep-disordered breathing.

A study by Mador et al. evaluated patients at two different settings: at a veterans affairs medical center with a predominantly male population and a free-standing ambulatory sleep center with equal gender proportions. They noted that positional sleep apnea prevalence (defined using the strict definition for positional sleep apnea) decreased as the sleep apnea severity increased [5]. It was 49.5 % in mild sleep apnea, 19.4 % in moderate sleep apnea, and 6.5 % in severe sleep apnea [5]. Another study by the same group evaluated 80 sequential patients who were referred to the Veterans Affairs Medical Center in Western New York and enrolled 20 patients in each of the three sleep apnea severity categories and an additional 20 patients who did not meet the definition for sleep apnea [16]. The hazard ratio for events (apneas and hypopneas) was significant in patients during supine compared to non-supine sleep in the mild and moderate sleep apnea groups at 1.25 (95 % CI 1.02–1.52) and 1.24 (95 % CI 1.04–1.47), respectively [16], meaning that events were

more likely to occur in the supine posture compared to the non-supine posture in patients with mild and moderate sleep apnea but not in the normal and severe sleep apnea groups [16].

A retrospective analysis by Oksenberg et al. evaluated 574 sleep apnea patients diagnosed by a respiratory distress index (RDI) greater than 10 per hour. They were further categorized into four different RDI categories (10–19.9, 20–29.9, 30–39.9, greater than 40 per hour). It was noted that the prevalence of positional sleep apnea (defined using the more lenient definition) remained high between 65.1 and 69.0 % in the mild, moderate, severe sleep apnea categories (RDI 10–19.9, 20–29.9, and 30–39.9 per hour), but showed a statistically significant reduction to 32.4 % in the most severe category (RDI greater than 40 per hour) [11]. Chung et al. evaluated 218 positional sleep apnea patients and randomly selected 109 of these patients to be matched with non-positional sleep apnea patients by age, gender, and body mass index (BMI) [17]. It was noted that both the matched and unmatched positional sleep apnea patients had less severe AHI values compared to the non-positional sleep apnea patients [17].

Although “positionality” gets weaker as the severity of sleep apnea worsens, it should be noted that in severe sleep apnea patients, the severity of each apneic event (apnea duration, the lowest oxygen desaturation, and duration of arousals) is still worse in the supine position compared to the non-supine position [18]. A retrospective review performed by Oksenberg et al. evaluated 638 patients undergoing two diagnostic polysomnograms (PSG)s at least 6 months apart [19]. Obstructive sleep apnea was seen in 566 patients [19]. They were then divided into four groups according to whether a positional component occurred during the two recorded PSGs: “non-positional sleep apnea (NPP) who remained NPP,” “positional sleep apnea (PP) who remained PP,” “PP who became NPP,” and “NPP who became PP” [19]. The “NPP who remained NPP” group were found to have a higher AHI in their initial PSG compared to the other three groups. The group of patients that had “PP who became NPP” had a significant increase in the AHI compared to the other three groups [19]. Thus, worsening of the AHI reduces the prevalence of positional sleep apnea.

Ozeke et al. evaluated the AHI on the left side and compared it to the right side in patients with different severities of obstructive sleep apnea [13]. It was noted that on the left side, AHI was significantly higher compared to the right side in patients with moderate and severe OSA, but was not significantly different in patients with mild OSA [13].

Why is positional sleep apnea more common in patients with mild to moderate sleep apnea? Patients with positional sleep apnea might have a less collapsible upper airway that only collapses in the supine posture when gravity works against the patient. Patients with severe sleep apnea likely have a more collapsible airway that collapses in all postures, and since the AHI is already severely increased in the non-supine posture, it is difficult for that metric to substantially worsen in the supine posture. The prior study by Oksenberg et al. shows that as sleep apnea worsens, patients often will switch from a positional component to a non-positional component [19].

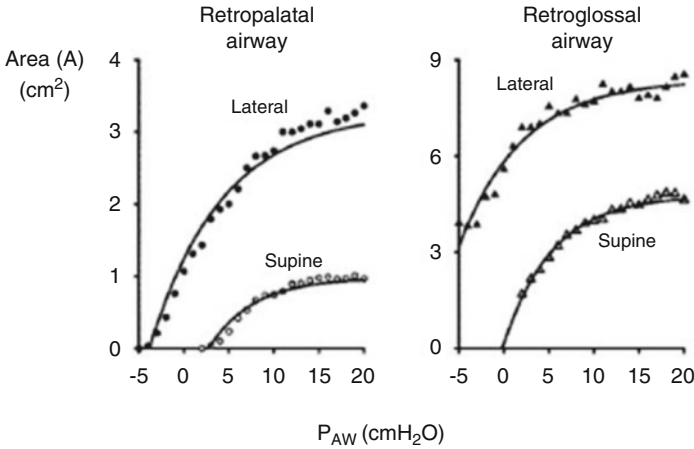


Fig. 2 Static pressure area plot in supine and lateral positions. P_{AW} airway pressure. Figure adapted from *Anesthesiology*, 2002. **97**(4): p. 780–5 [21] with permission

Anatomic/Morphological Factors

There are multiple studies performed to evaluate the anatomic factors that would determine the propensity of having positional sleep apnea.

A Belgium study evaluated the effect of changes in body position from prone to right side to supine by fast-computed tomography scanning in six awake position-dependent OSA patients and five position-independent OSA patients [20]. It was noted that the former (positional patients) had a larger minimum cross-sectional area than the latter and changes in body position affected the lateral but not the anteroposterior dimensions of the upper airway [20]. A study on 91 Korean patients with OSAS evaluated the level of obstruction based on video fluoroscopy [9]. The patients with soft palate obstruction had a higher chance of having positional sleep apnea compared to the patients with tongue base obstruction [9]. A Japanese study tried to evaluate the pathophysiology of positional sleep apnea [21]. They induced total muscle paralysis with general anesthesia in eight OSA patients in order to eliminate neuromuscular factors that could contribute to pharyngeal wall patency [21]. The cross-sectional area of the pharynx was measured endoscopically at different static airway pressures [21]. Then, a static pressure area plot between the positions was assessed to evaluate the influence of body position on the mechanical properties of the pharyngeal wall [21]. Static pressure area curves were higher in the lateral position compared to the supine position indicating a larger upper airway area at any given pressure in the lateral position (Fig. 2) [21]. In another small Japanese study, ten BMI-, age-, and AHI-matched positional and non-positional sleep apnea patients, respectively, underwent pharyngeal magnetic resonance imaging and cephalometric radiography during wakefulness [22]. They noted that positional sleep apnea patients have smaller volumes of the pharyngeal lateral wall soft

tissues compared to non-positional sleep apnea patients [22]. They also have a larger maxilla-nasion-mandible angle and a smaller lower facial height than the latter [22]. They concluded that positional sleep apnea patients could have a wider airway laterally and a more backward position of the lower jaw compared to non-positional sleep apnea patients [22] which would lead to obstruction primarily in the supine posture. A Chinese study on 103 OSA patients revealed that the modified Mallampati (MMP) grade and the neck circumference were important morphological features that helped to predict which patients were likely to have positional sleep apnea [23]. Those with a thick neck or small airway by Mallampati score were less likely to have positional sleep apnea [23]. A Japanese study published in 2008 evaluated the upper airway morphology in positional sleep apnea compared to non-positional sleep apnea patients [24]. They noted that the horizontal distance between the tonsillar fauces was significantly greater in positional sleep apnea patients [24].

An Australian study that evaluated the critical pressure at which the pharynx collapses in 23 OSA patients noted a significant decrease in the passive pharyngeal collapsibility associated with change from supine to lateral position [25].

There is also a change of shape of the upper airway from the supine to non-supine posture. When anatomical optical coherence tomography was used to scan the upper airway in OSA patients, it was noted that the upper airway changed from a more transversely oriented elliptical shape when supine to a more circular shape in the non-supine position [26]. It was suggested that the increase in circularity has a lesser propensity for tube collapse and this may be responsible for the reduction in obstructive episodes in the non-supine position [26]. Another Chinese study noted that the distance from the velum tip to the pharyngeal wall was narrower in non-positional sleep apnea patients compared to positional sleep apnea patients [27]. Thus, changes in pharyngeal shape, size, and collapsibility all occur when patients shift from the lateral to supine posture promoting a worsening of sleep apnea in the supine posture. The less pronounced these changes are in the non-supine posture, the more likely the patient is to display a clinically significant worsening in the AHI when he/she moves to the supine posture.

Obesity

Obesity has always been strongly associated with sleep apnea. A longitudinal analysis of the Wisconsin cohort concluded that a 10 % increase in body weight could be associated with a six times risk of developing OSA in subjects who did not have sleep apnea on their original study [28]. With regard to positional sleep apnea, there have been many studies evaluating the effect of body weight on positional sleep apnea. The studies date back as early as 1985, when Cartwright et al. evaluated 24 male patients with sleep apnea [3]. Fourteen out of the 24 were classified as having positional sleep apnea, 12 of whom were nonobese (defined as less than 25 % above their ideal weight), while 7 out of the 10 non-positional sleep apnea patients met the obesity criterion (more than 25 % above their ideal weight) [3]. In a retrospective

study performed on 574 sleep apnea patients subjects were divided into five different categories depending on the BMI (20–24.9; 25–29.9; 30–34.9; 35–39.9; and greater than 40) [11]. There was a significant decrease in the prevalence of positional sleep apnea as the BMI increased [11]. The authors also found that when the total group was divided into obese (BMI greater than 30) and nonobese (BMI less than or equal to 30) groups, the prevalence of positional sleep apnea was 42.2 % and 68 %, respectively. Three of the four severely obese patients, who had non-positional sleep apnea, successfully lost weight and converted to positional sleep apnea [11]. In another study of 218 positional sleep apnea patients and 123 non-positional sleep apnea patients, the BMI correlated with the AHI severity only in non-positional sleep apnea patients [17]. A study that evaluated 120 sleep apnea patients observed a higher BMI in non-positional sleep apnea patients compared to the positional sleep apnea patients, although the difference did not reach statistical significance [8]. A Japanese study classified 257 sleep apnea patients into a normal weight group (BMI under 24.0), mild obese group (BMI 24.0–26.4), and obese group (BMI 26.4 and heavier), which is different from the classification of obesity in the United States [29]. They noted that the prevalence of positional sleep apnea proportionally decreased as the BMI increased from 90.9 % in the normal weight group to 74.0 % in the mild obese group and to 57.4 % in the obese group [29]. Another Japanese study that evaluated the effect of BMI on positional sleep apnea concluded that improvements due to changes in posture became increasingly smaller with increases in the BMI [30].

A retrospective review of 2,077 OSA patients concluded that non-positional sleep apnea patients had a higher BMI compared to positional sleep apnea patients [31].

These authors also noted in another study that weight gain could convert patients with positional sleep apnea to non-positional sleep apnea, while weight loss could convert patients with non-positional sleep apnea to positional sleep apnea [19].

Thus, BMI has an inverse relationship with positional sleep apnea [11]. An increase in weight makes positional sleep apnea less likely. One mechanism that could explain this phenomenon is the changes noted in upper airway size and shape with weight gain due to deposition of adipose tissue in the upper airway that could convert positional sleep apnea to non-positional sleep apnea.

Age

In one study, the prevalence of positional sleep apnea decreased to 48.6 % in patients 60 years and older compared to 59.2 % in patients who were younger than 60 years [11]. A subsequent larger study performed by the same author revealed no significant age difference between the positional sleep apnea and the non-positional sleep apnea patients [31]. Another study noted a statistically significant difference in the ages between positional sleep apnea and non-positional sleep apnea patients, 47.1 years and 53.9 years, respectively [8].

Age may play a small role in identifying positional sleep apnea patients although results are conflicting. The positional effect seems to decrease with increase in age, and thus younger patients have a higher probability of having positional sleep apnea.

Effect of Sleep Stage on Positional Sleep Apnea

The effect of sleep stages on OSA has been well studied and documented in the past with worsening of respiratory events during rapid eye movement (REM) sleep compared to non-rapid eye movement (NREM) sleep. Muscle atonia is more prominent during REM sleep, and this is felt to be the major reason why sleep apnea worsens during this sleep stage. There is a significant amount of literature that has evaluated the effects of various sleep stages on positional sleep apnea, especially the effect of REM sleep compared to NREM sleep. A study of 263 subjects noted that sleep architecture was better preserved in positional sleep apnea patients than in non-positional sleep apnea patients with a lower arousal index of 26.0 ± 17.4 per hour and 45.4 ± 30.5 per hour, respectively, and higher slow wave sleep 15.2 ± 10.1 % and 10.2 ± 9.5 %, respectively [10].

A study published in Germany on 16 male OSA patients evaluated the pharyngeal critical pressure and the resistance of the upstream segment during light sleep, slow wave sleep, and REM sleep in the supine and non-supine position [32]. It was noted that during light sleep, the pharyngeal critical pressure decreased from 0.6 ± 0.8 cm H₂O (supine) to -2.2 ± 3.6 cm H₂O (non-supine) ($p < 0.01$) [32]. A more negative critical closing pressure means that the airway is less collapsible in that state. During slow wave sleep stage, the pharyngeal critical pressure decreased from 0.3 ± 1.4 cm H₂O (supine) to -1.7 ± 2.6 cm H₂O (non-supine) ($p < 0.05$), and during REM sleep the pharyngeal critical pressure decreased from 1.2 ± 1.5 cm H₂O (supine) to -2.0 ± 2.2 cm H₂O (non-supine) ($p < 0.05$) [32]. The resistance of the upstream segment had no dependence on body position or sleep stages except in the non-supine position during REM sleep [32].

A carefully performed study evaluated 253 PSGs performed over 3 months. Since the study was from Australia, subjects were classified as having OSA by an $AHI \geq 15$ events per hour [33]. In this study, the AHI and to a lesser extent the arousal index progressively decreased from stage 1 NREM sleep to slow wave sleep (Fig. 3). The AHI during REM sleep was intermediate between that observed in stage 1 and stage 2 NREM sleep. The AHI in the lateral position was less than that observed in the supine posture during all NREM sleep stages and also during REM sleep. In both patients with sleep apnea and those who would be characterized in the United States or Europe as having either mild sleep apnea (AHI 5–15 per hour) or no sleep apnea (AHI less than 5 per hour), respiratory events in the lateral position were 50–60 % of that observed in the supine position and arousal events 60–80 % of the supine values.

Studies in the past have examined the phenomena of REM-related OSA that has been defined as an AHI during REM sleep that is more than twofold higher than that

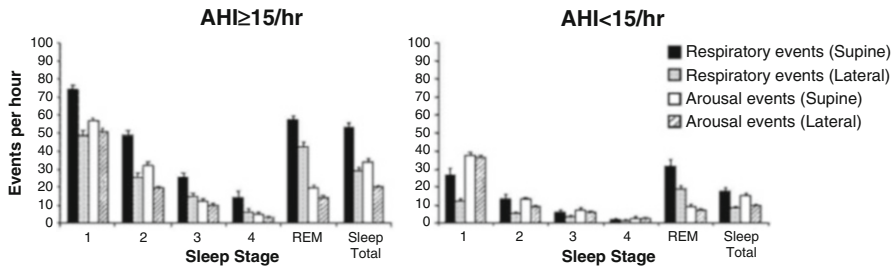


Fig. 3 Respiratory and arousal event frequencies in various sleep stages in supine and lateral positions. Figure adapted from *J Clin Sleep Med*, 2009. **5**(6): p. 519–24 [33] with permission

observed during NREM sleep with a total AHI greater than 5 per hour [34]. In a study by Eiseman et al., REM-related OSA was observed in about 50 % of patients, and positional sleep apnea (lenient definition) was noted in about 60 % of the patients [34]. The median amount of REM sleep was 16 % of total sleep time, while the median amount of supine sleep was 65 % of total sleep time [34]. It was thus noted that body position had a greater impact on the overall AHI than did the REM sleep AHI [34] simply because the subjects spent more time supine than they spent in REM sleep. Since normally patients spend 15–25 % of total sleep time in REM sleep, there are greater limits to how much REM sleep apnea can influence the overall severity of sleep apnea compared with positional sleep apnea [35].

A prospective analysis by Mador et al. evaluated 80 sequential PSGs performed at the Veterans Affairs Hospital in Buffalo and classified them into four groups: no OSA, mild OSA, moderate OSA, and severe OSA [16]. The hazard ratio for respiratory events in REM sleep compared with NREM sleep was significantly increased for the no OSA, mild, and moderate OSA groups but not the severe group. The hazard ratio for respiratory events in the supine compared with the non-supine position was significant for the mild and moderate OSA groups but not the normal or severe groups [16]. There was no statistically significant interaction effect between the various sleep stages and the supine and non-supine positions [16]. Thus, in this particular study, patients were not additively worse when in the supine posture during REM sleep compared to when they were in the supine posture during NREM sleep. The odds ratio of sleeping in the supine position for REM sleep versus NREM sleep was 0.47 (95 % CI 0.27–0.82) for moderate OSA and 0.54 (95 % CI 0.3–0.95) for severe OSA [16]. Thus, in this study, patients with moderate or severe sleep apnea avoided or at least had less REM sleep in the supine posture which may have in part explained the lack of additive effect of REM and supine sleep.

In an older smaller study [36], it was noted that 9 out of 22 patients who had “positional dependency” during NREM sleep lacked the feature during REM sleep [36]. Another small study [37] noted that the apnea duration was longer in REM sleep compared to NREM sleep even after adjusting for body position [37]. Although the AHI was greater in the supine position compared to the non-supine position, this difference was only noted in NREM sleep [37]. A subsequent study noted that the

sleep apnea severity was worse in REM sleep in supine position, followed by NREM sleep in supine position, followed by REM sleep in non-supine position, and lastly by NREM sleep in non-supine position [38].

Some studies have shown that the AHI is worse during REM stage in supine sleep compared to supine NREM sleep or REM non-supine sleep while others have not. However, when one study carefully looked for an interaction between sleep stage and body position, it was not found. Some studies have also shown that positional sleep apnea is more prominent during NREM compared to REM sleep.

However, the largest studies have found a positional effect during both NREM and REM sleep. The finding that patients with moderate and severe OSA had a lesser tendency to sleep in the supine position during REM sleep compared to NREM sleep stage could be the bodies' way of responding to worsening of respiratory events and hypoxemia [16]. In patients with severe neurologic disease who demonstrate significant nocturnal hypoventilation, the amount of REM sleep decreases, and in the most severe cases, no REM sleep is observed at all.

References

1. Young T, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med.* 1993;328(17):1230–5.
2. Peppard PE, et al. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol.* 2013;177(9):1006–14.
3. Cartwright RD. Effect of sleep position on sleep apnea severity. *Sleep.* 1984;7(2):110–4.
4. Gillman A, et al. Comparison of supine-only and REM-only obstructive sleep apnoea. *Sleep Med.* 2012;13(7):875–8.
5. Mador MJ, et al. Prevalence of positional sleep apnea in patients undergoing polysomnography. *Chest.* 2005;128(4):2130–7.
6. Lee CH, et al. The effect of positional dependency on outcomes of treatment with a mandibular advancement device. *Arch Otolaryngol Head Neck Surg.* 2012;138(5):479–83.
7. Chung JW, et al. Treatment outcomes of mandibular advancement devices in positional and nonpositional OSA patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010;109(5):724–31.
8. Richard W, et al. The role of sleep position in obstructive sleep apnea syndrome. *Eur Arch Otorhinolaryngol.* 2006;263(10):946–50.
9. Sunwoo WS, et al. Association between positional dependency and obstruction site in obstructive sleep apnea syndrome. *Clin Exp Otorhinolaryngol.* 2012;5(4):218–21.
10. Teeraprairuk B, et al. Clinical and polysomnographic data of positional sleep apnea and its predictors. *Sleep Breath.* 2012;16(4):1167–72.
11. Oksenberg A, et al. Positional vs nonpositional obstructive sleep apnea patients: anthropomorphic, nocturnal polysomnographic, and multiple sleep latency test data. *Chest.* 1997;112(3):629–39.
12. Metersky ML, Castriotta RJ. The effect of polysomnography on sleep position: possible implications on the diagnosis of positional obstructive sleep apnea. *Respiration.* 1996;63(5):283–7.
13. Ozeke O, et al. Influence of the right- versus left-sided sleeping position on the apnea-hypopnea index in patients with sleep apnea. *Sleep Breath.* 2012;16(3):617–20.
14. Tiotiu A, et al. Body position and breathing abnormalities during sleep: a systematic study. *Pneumologia.* 2011;60(4):216–21.

15. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22(5):667–89.
16. Mador MJ, et al. Are the adverse effects of body position in patients with obstructive sleep apnea dependent on sleep stage? *Sleep Breath*. 2010;14(1):13–7.
17. Chung JW, et al. Patients with positional versus nonpositional obstructive sleep apnea: a retrospective study of risk factors associated with apnea-hypopnea severity. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2010;110(5):605–10.
18. Oksenberg A, et al. Association of body position with severity of apneic events in patients with severe nonpositional obstructive sleep apnea. *Chest*. 2000;118(4):1018–24.
19. Oksenberg A, et al. Obstructive sleep apnoea in adults: body postures and weight changes interactions. *J Sleep Res*. 2012;21(4):402–9.
20. Pevernagie DA, et al. Effects of body position on the upper airway of patients with obstructive sleep apnea. *Am J Respir Crit Care Med*. 1995;152(1):179–85.
21. Isono S, Tanaka A, Nishino T. Lateral position decreases collapsibility of the passive pharynx in patients with obstructive sleep apnea. *Anesthesiology*. 2002;97(4):780–5.
22. Saigusa H, et al. Three-dimensional morphological analyses of positional dependence in patients with obstructive sleep apnea syndrome. *Anesthesiology*. 2009;110(4):885–90.
23. Weihs C, et al. Relationship of body position, upper airway morphology, and severity of obstructive sleep apnea/hypopnea syndrome among Chinese patients. *Acta Otolaryngol*. 2011;131(2):173–80.
24. Soga T, et al. Upper airway morphology in patients with obstructive sleep apnea syndrome: effects of lateral positioning. *Auris Nasus Larynx*. 2009;36(3):305–9.
25. Ong JS, et al. Variability of human upper airway collapsibility during sleep and the influence of body posture and sleep stage. *J Sleep Res*. 2011;20(4):533–7.
26. Walsh JH, et al. Effect of body posture on pharyngeal shape and size in adults with and without obstructive sleep apnea. *Sleep*. 2008;31(11):1543–9.
27. Chang ET, Shiao GM. Craniofacial abnormalities in Chinese patients with obstructive and positional sleep apnea. *Sleep Med*. 2008;9(4):403–10.
28. Peppard PE, et al. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA*. 2000;284(23):3015–21.
29. Itasaka Y, et al. The influence of sleep position and obesity on sleep apnea. *Psychiatry Clin Neurosci*. 2000;54(3):340–1.
30. Akita Y, et al. Posture of patients with sleep apnea during sleep. *Acta Otolaryngol Suppl*. 2003;550:41–5.
31. Oksenberg A, et al. The significance of body posture on breathing abnormalities during sleep: data analysis of 2077 obstructive sleep apnea patients. *Harefuah*. 2009;148(5):304–9. 350, 351.
32. Penzel T, et al. Effect of sleep position and sleep stage on the collapsibility of the upper airways in patients with sleep apnea. *Sleep*. 2001;24(1):90–5.
33. Ratnavadivel R, et al. Marked reduction in obstructive sleep apnea severity in slow wave sleep. *J Clin Sleep Med*. 2009;5(6):519–24.
34. Eiseman NA, et al. The impact of body posture and sleep stages on sleep apnea severity in adults. *J Clin Sleep Med*. 2012;8(6):655–6A.
35. Kryger MH, Roth T, Dement WC. Principles and practice of sleep medicine. 5th ed. Philadelphia, PA: Saunders/Elsevier; 2011.
36. Pevernagie DA, Shepard Jr JW. Relations between sleep stage, posture and effective nasal CPAP levels in OSA. *Sleep*. 1992;15(2):162–7.
37. George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. *Sleep*. 1988;11(1):90–9.
38. Oksenberg A, et al. REM-related obstructive sleep apnea: the effect of body position. *J Clin Sleep Med*. 2010;6(4):343–8.