Egyptian Journal of Bronchology

Vol 3, No 2, December, 2009

ORIGINAL ARTICLE

A STUDY OF SLEEP BREATHING DISORDERS IN THE ELDERLY

By
Wafaa El-Shimy, Tarek El-Behidy, Amgad Farhat, Gehan Abo El-Magd
Chest Department, Faculty of Medicine, Tanta University.

Correspondence to: Amgad Farhat, Email: amgad_farhat@yahoo.com

Background: *Sleep complaints are common in the geriatric age group with more than half of the elderly endorsing a complaint about their sleep quality. The most common complaint is difficulty with maintenance of sleep.*
Methods: *This study was conducted on 100 apparently healthy elderly subjects (above 50 years). Sleep study was conducted in the sleep laboratory, Chest Department, Tanta University Hospital at night during sleep for at least 6 hours. Patients with apnea hypopnea index (AHI) >5 events/hour (Group II) underwent another diagnostic study in the sleep laboratory in the second night after insertion of Swan Ganz catheter. Therapeutic study was done on the 3rd night for these patients for CPAP titration.*
Results: *The mean values of age in Group I (subjects with AHI <5 events/hour was 57.86±5.83 years, while in Group II it was 63.23±8.04 years. Age was significantly higher in group II compared to group I. The prevalence of mild AHI (5-15) event/ hour was 36% and 25% in men and women respectively. The prevalence of moderate AHI (16-30) event/ hour was 18% and 7% in men and women respectively. The prevalence of severe AHI (> 30) was 12.5% and 2% in men and women respectively. The mean value of cardiac output (CO) L/min, mean arterial blood pressure (MAP), pulmonary artery pressure (PAP) and pulmonary capillary wedge pressure (PCWP) mmHg. were 4.51±0.08, 86.12±1.53, 21.59±3.51 and 13.03±0.6 in group II before CPAP respectively and 5.01±0.09, 85.23±1, 17.76±1.28 and 12.33±0.53 after CPAP respectively.*
Conclusions: *A significant positive correlation was found between apnea hypopnea index (AHI) and age indicating that age is a risk factor for sleep-related breathing disorders. The incidence of sleep-breathing disorders was higher in men than in women. CPAP caused significant improvement in sleep breathing disorders and hemodynamic parameters in the studied elderly subjects.*

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a part of a collection of sleep-related breathing disorders, that include snoring, upper airway resistance syndrome.[1] In this syndrome there is not only repetitive upper airway obstruction during sleep, during which the subject continues to make efforts against a closed upper airway during sleep but also, is accompanied by an array of daytime symptoms dominated by excessive daytime sleepiness. The clinical sequalae of untreated OSAS are often severe and include daytime hypersomnolence, cognitive impairment, systemic hypertension, pulmonary hypertension, myocardial infarction, cardiac arrhythmias, and increased risk of motor vehicle crashes.[2] Sleep-disordered breathing was assessed using the apnea-hypoapnea index (AHI), defined as the average number of apneic plus hypoapneic episodes per hour of sleep. The apnea hypopnea index, also referred to as the respiratory disturbance index (RDI), is the most common index used to describe and quantify sleep disordered breathing (SDB).[3]

Sleep complaints are common in the geriatric age group.[4]

with more than half of the elderly endorsing a complaint about their sleep quality. The most common complaint is difficulty with maintenance of sleep. Reports of poor sleep correlate strongly with health complaints and depressive symptoms. There are many reasons that the ability to sleep decreases with age. Two main reasons are changes in circadian rhythms and the presence of sleep disorders.[5] In a meta-analytic review of polysomnographic data, several studies[6] showed that older women may objectively sleep better than older men, although older women complain more about their sleep than older men.[7]

Sleep-disordered breathing (SDB) has been assumed to be a condition associated primarily with men. In clinical samples, the ratio between men and women of the prevalence of SDB has been considered to be about 10:1.[8] Most estimates of the male/female ratio in the general public range between 2:1 and 4:1.

Treatment with CPAP breathing could normalize hemodynamic disorders in patients with sleep apnea syndrome (SAS) and may perhaps prevent development of cor pulmonale. CPAP increases pulmonary intravascular but not transmural, true pressure. Treatment of obstructive sleep apnea syndrome (OSAS) caused a reduction in daytime mean arterial pressure and diastolic blood pressure, associated with a reduction of vascular resistance.(9) Some investigators have reported an immediate decrease in nocturnal and morning awake intra-arterial blood pressure after CPAP therapy.[10]

PATIENTS AND METHOD

Patient characteristics: This study was conducted on 100 apparently healthy elderly persons (50 years or more), they were admitted in sleep laboratory, Chest Department, Tanta University Hospitals. Subjects were divided into 2 groups after sleep study was done as follow:

Group I: included 72 subjects who had AHI < 5 events/hour.

Group II: included 28 subjects who had AHI ≥ 5 events/hour, they underwent CPAP auto titration.

Methods

Sleep study: It was conducted in the sleep laboratory, Chest Department, Tanta University Hospital at night during sleep for at least 6 hours. Patients with AHI > 5 events/hour underwent another diagnostic study in the sleep laboratory in the second night after insertion of Swan Ganz catheter. Therapeutic study was done on the 3rd night for these patients for CPAP auto-titration. The study was done using Auto Set portable II plus system (ResMed, Australia).

Statistical analysis: All results in this study are presented in mean ± standard deviation. Comparison between 2 groups was done using t-test between group I & group II, and paired t-test between group II before & after CPAP. The correlation between different parameters was done using Pearson test. The difference was considered significant when $p < 0.05$.

RESULTS

Group I: included 72 subjects, 34 males and 38 females. Their ages ranged between 50 and 73 years with a mean of 57.86±5.83 years (45 subjects aged < 60 years and 29 subjects aged > 60 years). They had apnea index < 5 events/hour.

Group II: included 28 subjects, 20 males and 8 females. Their ages ranged between 51 and 77 years with a mean of 63.23±8.04 years (8 subjects aged < 60 years and 18 subjects aged > 60 years). They had AHI > 5 events/hour, they underwent CPAP auto titration.

The mean values of age were 57.86±5.83 years and 63.23±8.04 years, group I and II respectively. Age was significantly higher in group II compared to group I (Fig. 1) while body mass index was 27.8 in group I and 29.1 in group II. There was no statistical significant difference in the two studied groups.

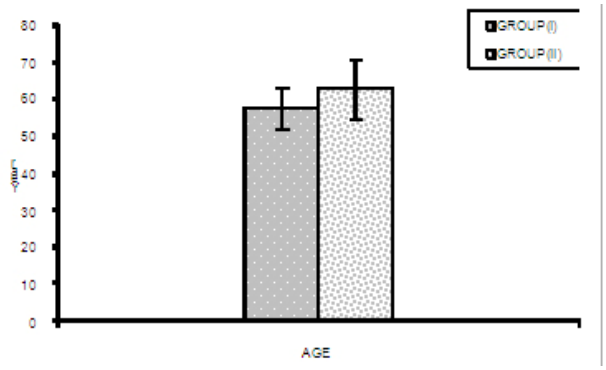

Fig 1. Age in years in the two studied groups.

The prevalence of sleep apnea syndrome was classified according to severity into:

- Mild (AHI 5-15 event/h), it was found in 15 subjects out of 28 (53.6%).
- Moderate (AHI 16-30 event/h), it was found in 7 subjects out of 28 (25%).
- Severe (AHI > 30 event/h), it was found 6 subjects out of 28 (21.4%).

In the present study, the prevalence of mild AHI (5-15) event/hour was 36% and 25% in men and women respectively and the prevalence of moderate AHI (16-30) event/hour was 18% and 7% in men and women respectively while the prevalence of severe AHI (> 30) was 12.5% and 2% in men and women respectively.

The mean values of AHI, AI and respiratory irregularity index (account of the number of sudden increases in ventilation) were 3.35±1.29, 2.16±2.67 and 22.86±10.69 event/hour in group I respectively and 28.54±13.41, 17.65±12.09 and 35.08±14.2 in group II respectively. These parameters were found to be significantly higher in group II compared to group I ($p<0.05$) (Fig. 2).

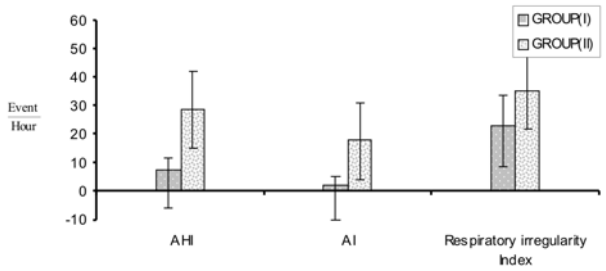

Fig 2. Statistical comparison of sleep breathing disorders in the two studied groups.

Significant positive correlation was found between AHI and: age, BMI and respiratory irregularity index (r = 0.34, 0.33 and 0.39 respectively and p <0.05) (Fig. 3). Insignificant positive correlation was found between age and: BMI and respiratory irregularity index (r =-0.13, 0.09 respectively and p > 0.05).

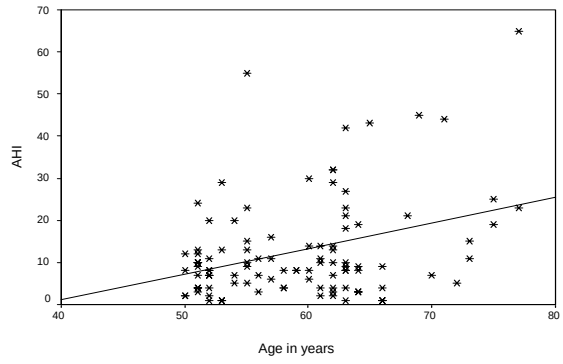

Fig 3. Correlation between AHI events/hour and age in years.

Cardiac output (CO) L/min was found to be significantly increased after CPAP. Mean arterial blood pressure (MAP), pulmonary artery pressure (PAP) and pulmonary capillary wedge pressure (PCWP) mmHg. were found to be significantly decreased after CPAP (Fig. 4).

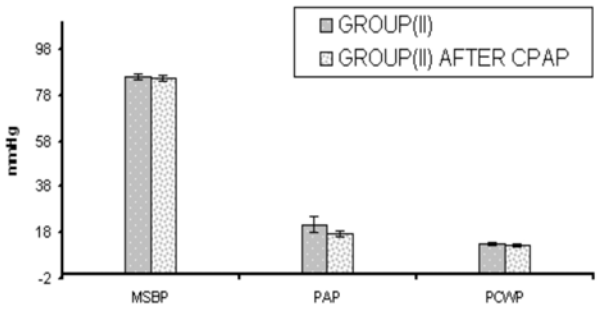

Fig 4. Hemodynamic parameters before & after CPAP in group II.

Significant positive correlation was found between AHI and pulmonary artery pressure (PAP) (r = 0.86 and p < 0.05) (Fig. 5).

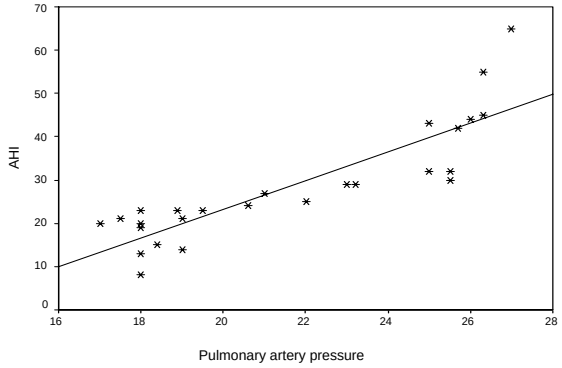

Fig 5. Correlation between pulmonary artery pressure mmHg. and AHI events/hour in group II.

DISCUSSION

Aging is per se associated with a decrease in the quality of sleep; SDB may further disrupt the sleep architecture in older subjects. The prevalence of obstructive sleep apnea (OSA) increases with aging; available study report prevalence rate of 11 – 62 %.[11] In the present study, the number of subjects

having SAS (AHI ≥5) was 28 subjects out of 100. In one of the largest studies, 427 community dwelling elderly were randomly selected and studied.[12,13] The prevalence rate of sleep-disordered breathing was 24% with AI ≥5 per hour of sleep (with a mean of apnea index of 13) and 81% AHI ≥5 per hour of sleep (mean AHI of 38). In another large study,[14] it was found that 55% of 229 residents from two retirement villages in Australia had five or more respiratory disturbances in sleep. Both studies, however, indicate that respiratory disturbances in sleep are very common in the elderly.

On the basis of sleep laboratory criteria alone (AHI ≥ 5) it has been observed that older people have a greater prevalence of apneas during sleep than the young, and more than five respiratory pauses per hour have been found among at least one-third of the elderly.[13,15]

Some studies have found OSA to be highly prevalent in people older than 65 years of age. In the first large population-based study of older people, some authors[12] conducted home polygraphy on 427 men and women aged 65 to 95 years. OSA (AHI ≥ 10 event/hour) occurred in 70% of the men and 56% of the women, approximately 3-fold higher than the prevalence estimates for OSA in middle age.

The higher male risk of OSA was explained by clear sex differences in upper airway shape and genioglossal muscle activity during the awake state, in craniofacial morphology, and pattern of fat deposition.[16]

Pulmonary arterial hypertension was defined as pulmonary artery pressure of >20mm Hg. Precapillary pulmonary hypertension was defined as pulmonary artery pressure >20 mm Hg associated with a pulmonary capillary wedge pressure of <15 mm Hg.[17] Acute increases in pulmonary artery pressure (PAP) secondary to repeated episodes of upper airway obstruction and alveolar hypoxia during sleep have been documented in patients with obstructive sleep apnea (OSA) syndrome.[18]

In agreement with the present work, some authors[19] examined thirty-seven patients (35 men and two women) with obstructive sleep apnea syndrome (OSAS) without any known cardiovascular and lung disease by Doppler echocardiography. Eight of the 37 (21.6%) patients experienced daytime pulmonary hypertension, and all of them had severe (OSAS) with an AHI of > 30. After CPAP, the reduction in mean PAP was observed without any change in BMI of the patients.

In contrast, there are other studies[20] with negative results in terms of the effect of CPAP treatment on pulmonary hemodynamics; however, these studies have included OSA patients with COPD as well and it is possible that these patients have fixed pulmonary hypertension due to structural changes and remodeling of pulmonary vasculature. One might speculate that CPAP treatment may prevent further increases in PAP in such a cohort of patients with COPD and OSA.

In Conclusion a significant positive correlation was found between apnea hypopnea index (AHI) and age indicating that age is a risk factor for sleep-related breathing disorders. The incidence of sleep-breathing disorders was higher in men than in women. Pulmonary arterial hypertension is frequently observed in patients with OSA. CPAP treatment reduces raised pulmonary artery pressure due to obstructive sleep apnea in the elderly.

Sleep study is recommended for elderly persons who have manifestations of sleep apnea especially daytime hyper somnolence to improve their quality of life.

REFERENCES

1. Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistro P. A cause of excessive daytime sleepiness: The upper airway resistance syndrome. Chest. 1993;104:781-7.

2. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. New Engl J Med. 2000;342:1378-84.

3. Redline S, Kapur V, Sanders M, Quan SF, Gottlieb DJ, Rapoport DM. Effects of varying approaches for identifying respiratory disturbances on sleep apnea assessment. Am J Respir Crit Care Med. 2000;161:369-74.

4. Foly DJ, Monjan AA, Brown SL, Simonsick EM, Wallace RB, Blazer DG. Sleep complaints among elderly persons: an epidemologic study of three communities. Sleep. 1995;18,425-32.

5. Ancoli Israel S. Sleep problems in older adults: putting myths to bed. Geriatrics. 1997;52:20-30.

6. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and Psychiatric Disorders: A Meta-analysis. Archive of General Psychiatry. 1992;49:651-68.

7. Ohayon MM, Cault M. Insomnia and psychotropic drug consumption. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 1995;19:421-31.

8. Vgontzas AN, Tan TL, Bixler EO, Martin LF, Shubert D, Kales A. Sleep apnea and sleep distribution in obese patients. Arch Intern Med. 1994;154:1705-11.

9. Voogel AJ, Van Steenwijk RP, Karemaker JM, Van Montfrans GA. Effects of treatment of obstructive sleep apnea on circadian hemodynamics. J Auton Nerv Syst. 1999;24;77:177-83.

10. Meyer TJ, Hill NS. Noninvasive positive pressure ventilation to treat respiratory failure. Ann Intern Med. 1994;120:760-70.

11. Janssens JP, Pautex S, Hilleret H, Michel JP. sleep disordered breathing in the elderly. Aging (Milano). 2000;12:417-29.

12. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ. Sleep-disordered breathing in community dwelling elderly. Sleep. 1991;14:486-95.

13. Ancoli-Israel, S. Epidemiology of sleep disorders. Clin Geriatr Med. 1989;5:347-62.

14. Mant A, Saunders NA, Eyland EA, Pond CD, Sawyer B, Saltman DC. Sleep habits and sleep related respiratory disturbances in the elderly population. Sleep, ed. Horne, J. Gustav Fischer Verlag, Stuttgart. 1989;260-1.

15. Bixler EO, Vgontzas AN, Ten Have T, Tyson K, Kales A. Effects of age on sleep apnea in men: I. Prevalence and severity. Am J Respir Crit Care Med. 1998;157:144-8.

16. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. Population Health Perspective. Am J Respir Crit Care Med. 2002;165:1217-39.

17. Bady E, Achkar, Pascale S, Orvonen E, Laabun JP. Pulmonary arterial hypertension in patients with sleep apnea syndrome. Thorax. 2000;55:934-9.

18. Pozdus T. Pulmonary hemodynamics in sleep-related breathing disorders; In Smirne, S.; Franceschi, M.; Ferini Strambi, L. eds: Sleep and Ageing. Milano, Masson. 1991;122-34.

19. Yamakawa H, Shiomi T, Sasaaanabe R, Hasegawa R, Ootake L, Wakayama H, katada M, Kobayashi T. Pulmonary hypertension in patients with obstructive sleep apnea. Psychatry Clin Neurosci. 2002;56:311-12.

20. Marrone O, Bomsignore MR. Acute and chronic influences of obstructive sleep apnea on the pulmonary circulation. Monaldi Arch Chest Dis. 1997;52:263-7.