IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 3 Ver.15 March. (2018), PP 59-63
www.iosrjournals.org

Effect of Severity of OSA on Oxygen Saturation: An Observational Study Comparing Lowest Oxygen Saturation Attained By Patients of Moderate and Severe Obstructive Sleep Apnea.

Dr. Shubham Agarwal[1], Dr. Praveen K Singh[2]
[1](*Department of Tuberculosis and Respiratory Diseases/Institute of Medical Sciences, Banaras Hindu University, India.)*
[2(]*Department of Tuberculosis and Respiratory Diseases/Institute of Medical Sciences, Banaras Hindu University, India.)*
Corresponding author: Dr. Shubham Agarwal1

Abstract: *Obstructive sleep apnoea(OSA) is a common but under reported disease, especially of the obese population. The resulting apneic and hypopneic events during sleep result in sympathetic activation[1-4] which even persists during the day[5] and lead to various complications, especially in the cardiovascular system. A lot of emphasis is laid on the AHI values demonstrating linearity between elevated AHI and likelihood of adverse effects. However AHI does not capture significant aspects such as degree of oxygen desaturation, nocturnal hypoventilation or associated sleep disturbance. In our study we look at the degree of oxygen desaturation with rising AHI values. This observational study was conducted on patients presenting to the OPD of Sir Sunder Lal Hospital with symptoms of sleep disordered breathing. 50 patients were included in the study between june 2015 and june 2016.*

Date of Submission: 15-03-2018 Date of acceptance: 30-03-2018

I. Introduction

There are important changes in ventilation during sleep. During NREM sleep, ventilation declines. Ventilation in REM sleep is also consistently less than that in wakefulness.[6]

The decrease in ventilation in NREM is largely due to increase in upper airway resistance[7], which increases progressively from stage N1 TO N3. During REM sleep, pharyngeal muscle activity is shut down via sleep specific motor inhibition [8] which further predisposes at risk individuals to OSA. Moreover during there is loss of the central wakefulness ventilatory drive. Consequent to the decrease in ventilation there is a decrease in PaO_2 and increase in $PaCO_2$ in the blood. In healthy individuals the PaO_2 does not fall to a level where significant desaturations occur. However in patients with low functional residual capacity as a result of obesity or intrinsic lung diseases, the drops in PaO_2 will be greater.

II. Objective

To evaluate the effect of severity of obstructive sleep apnea (and increasing AHI) on the degree of oxygen desaturation during episodes of apnea and hypopnea.

III. Material And Methods

This study was conducted in the chest ward of department of Tuberculosis and Respiratory Diseases, Sir Sunderlal Hospital, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India from June 2015 to June 2016. 50 obese subjects (BMI≥30) were selected for the purpose of study and categorized into having moderate or severe OSA on the basis of their Apnea Hypopnea Indices (AHI) after a full night type 1 polysomnography. Baseline oxygen saturation was measured by means of a portable pulse oximeter on the night of study before commencing the study and the falls in oxygen saturation was observed during the polysomnography from the epochs of the polysomnography recordings.

Inclusion criteria

1. Age 20 and over, with symptoms suggestive of sleep disordered breathing.
2. Patients as well as attendants willing to give informed consent.
3. Patients ready to undergo sleep study (PSG).

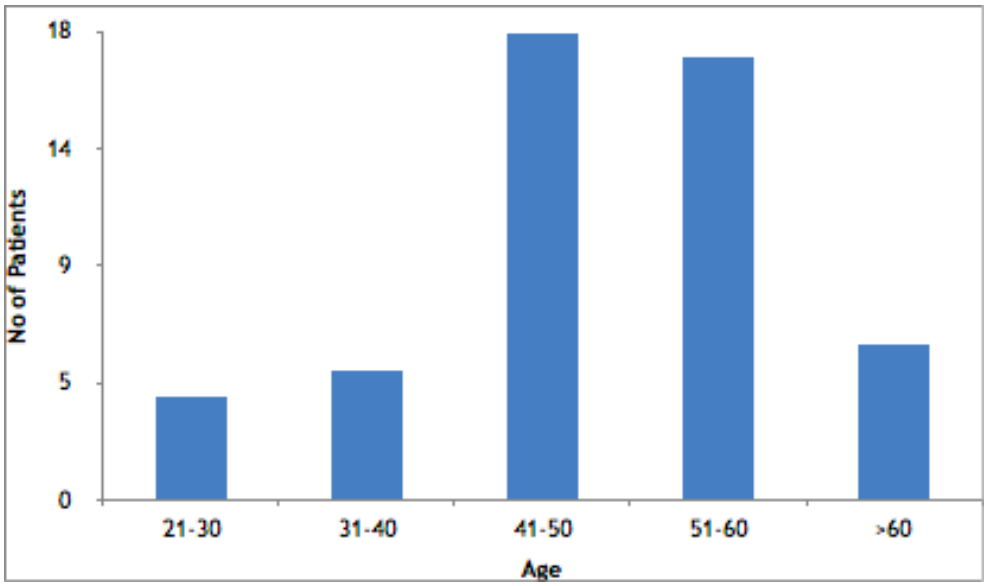
Exclusion criteria:

1. Unstable medical or psychiatric conditions that would interfere with the demands of the study. Example include unstable congestive heart failure, neuromuscular diseases, cancer, and renal failure
2. Chronic respiratory failure or insufficiency with suspected or known neuromuscular diseases, moderate or severe COPD or other pulmonary disorders, or participants qualifying for oxygen therapy (arterial saturation 88% for more than five minutes.)
3. Consumption of ethanol more than 4 nights per week (CAGE criteria)
4. Use of recreational drug within the past 12 months.
5. Woman who are pregnant or currently lactating.
6. Patients with mild form of OSA and other sleep disordered breathings.

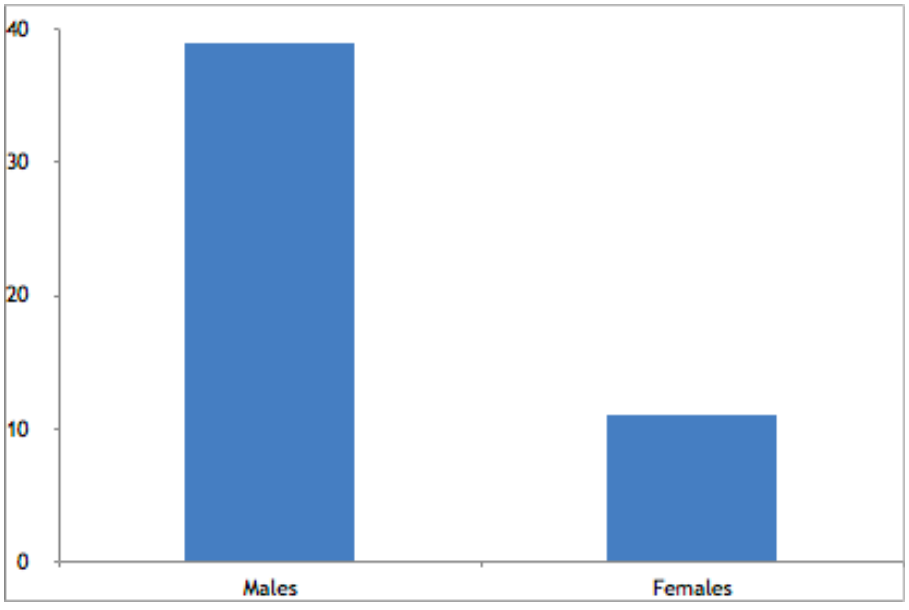
IV. Observations

50 subjects of moderate to severe sleep apnea participated in this study. Mean age was 52.6years.

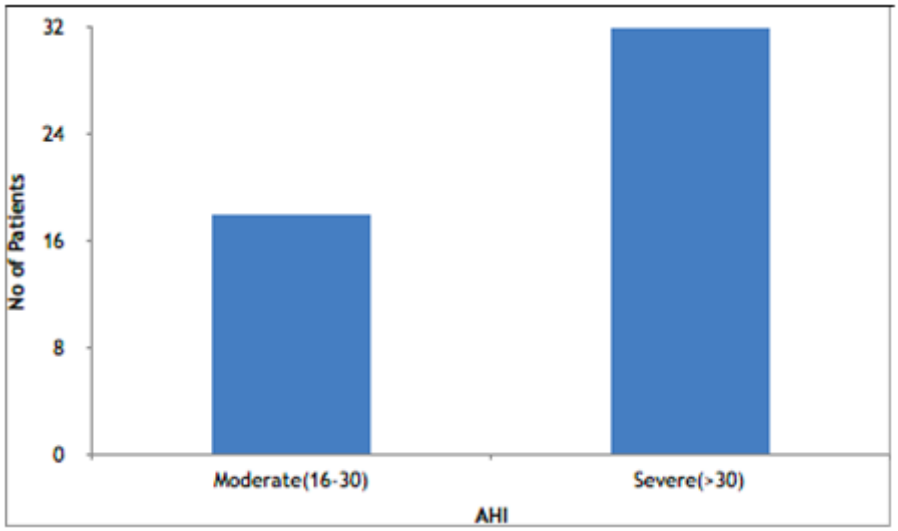
The age distribution amongst the subjects is depicted in the following bar diagram.



Sex distribution among subjects in the study is depicted below:



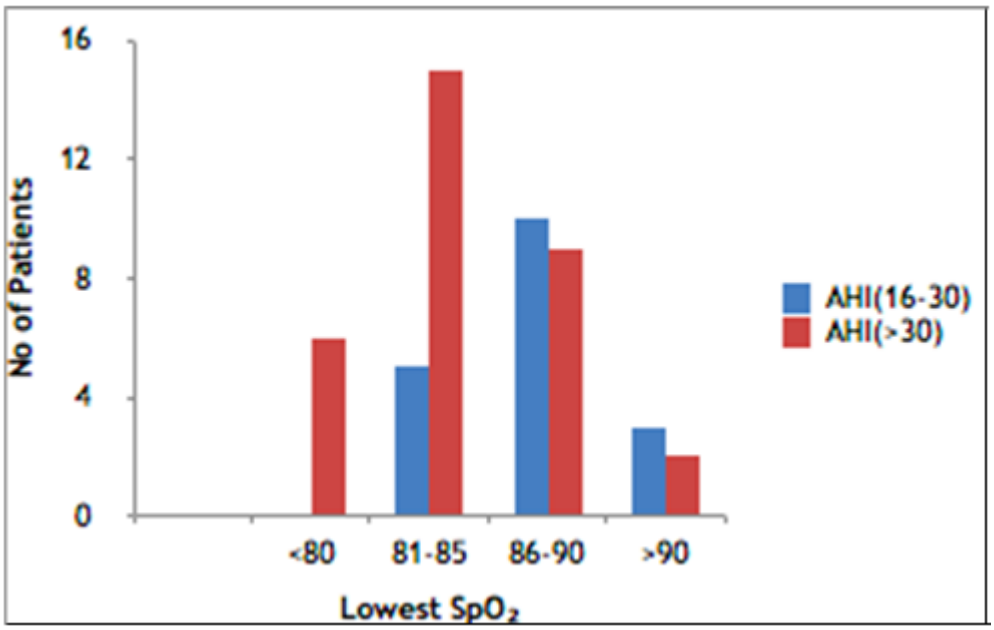
Distribution of AHI amongst the patients was as flows: (moderate and severe)



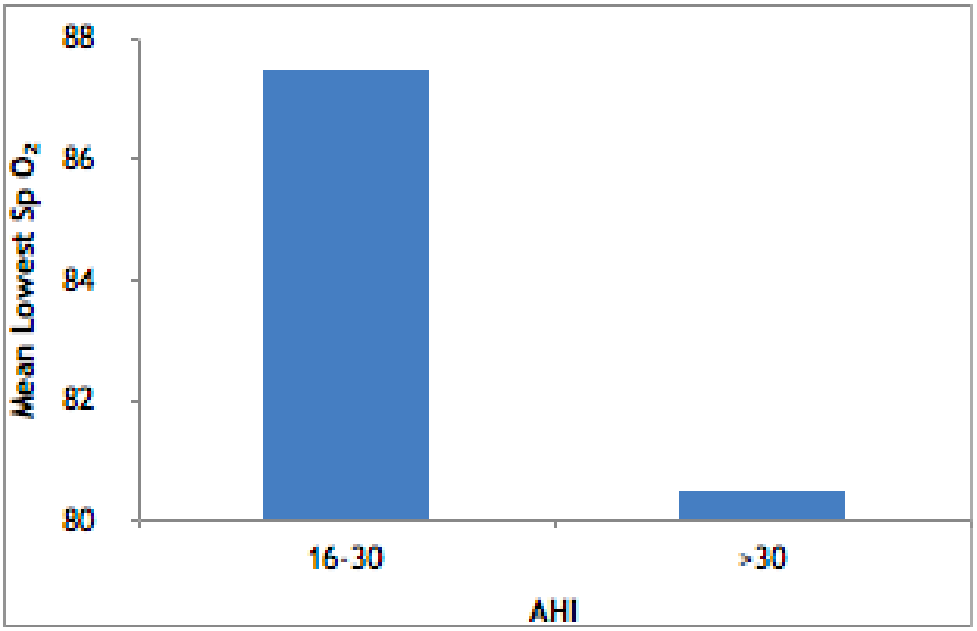
Of the total 50 subjects included in the study,18 fell in the moderate criteria of OSA with AHI in the range of 16-30, while 32 subjects were of severe OSA with AHI>30.

After polysomnography the distribution of lowest SpO_2 among the two AHI groups was as follows:

AHI	SPO_2			
	<80	81-85	86-90	>90
16-30	0	5	10	3
>30	6	15	9	2

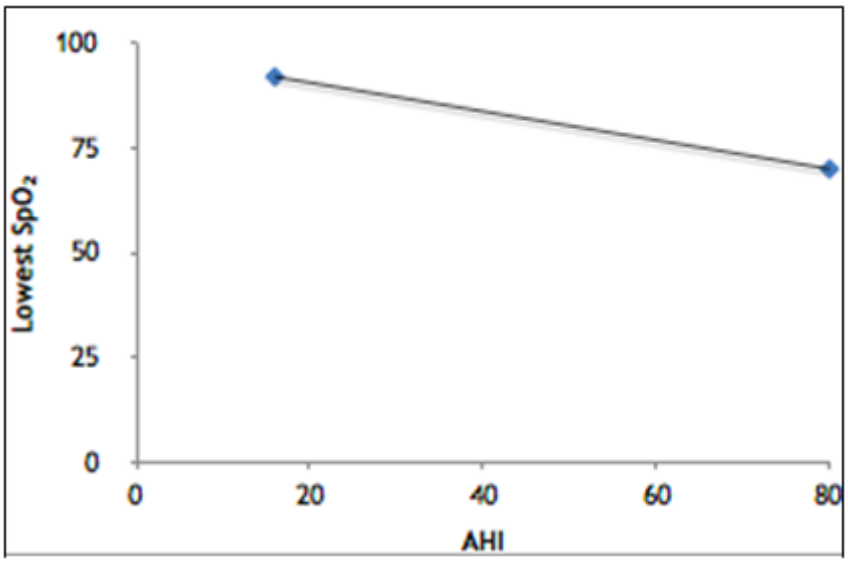


Mean (lowest) SpO_2 attained in the two groups:



The mean lowest SpO_2 in the moderate OSA group was 87.5% while that in the severe OSA group was 80.5%. Statistical analysis was done using SPSS Software. The two groups were compared using student t test which showed a statistically significant difference in the lowest SpO_2 attained between the two groups with a p value of 0.023.

A negative correlation was seen between increasing AHI values and the lowest nocturnal SpO_2 attained among the subjects as shown below.



V. Discussion

Obstructive sleep apnea is a common yet under-recognized condition especially of the obese population. The persence of apneas and hypoponeas with associated hypoxemia and oxygen desaturation is a known fact in the disorder with various mechanisms operating in conjunction for the same *viz* the collapse of upper airway with reduction of its intraluminal diameter and increase of upper airway resistance,[9,10] neural modulation of upper airway patency[11-18], reduction of wakefulness ventilatory drive and altered ventilatory response during sleep[19-22]. The hypoxemia may lead to excess sumpathetic activation and the myriad of consequent adverse effects.

In our study there was a progressive drop in lowest oxygen saturation during sleep as the AHI increased from moderate to severe levels with lowest SpO_2 in the moderate AHI group being 87.5% and in the severe AHI group being 80.5% with a significant difference (p value of 0.023)

The findings were in conjunction with previous studies of Wael Alkhiary et al in 2015 and Renata Trimer et al 2016 who found similar findings of lower oxygen saturations and higher desaturation indices in patients with severe obstructive sleep apnea.

VI. Conclusion

This study highlights the effects of increasing AHI, not only on the sleep architecture and quality but also on the level of hypoxemia that the patient may attain which may lead to a cascade of adverse effects due to the sympathyhetic system stimulation in response to it.

References

[1] Floras JS. Sympathetic nervous system activation in human heart failure: clinical implications of an updated model. *J Am Coll Cardiol*. 2009; 54(5):375–385.

[2] Horner RL, Brooks D, Kozar LF, Tse S, Phillipson EA. Immediate effects of arousal from sleep on cardiac autonomic outflow in the absence of breathing in dogs. *J Appl Physiol*.

[3] 1995; 79(7559214):151–162.

[4] Nalivaiko E, Catcheside PG, Adams A, Jordan AS, Eckert DJ, McEvoy RD. Cardiac changes during arousals from non-REM sleep in healthy volunteers. *Am J Physiol Regul Integr Comp Physiol*. 2007; 292(17110530):1320–1327.

[5] Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest*. 1995; 96(4):1897–1904.

[6] Carlson JT, Hedner J, Elam M, Ejnell H, Sellgren J, Wallin BG. Augmented resting sympathetic activity in awake patients with obstructive sleep apnea. *Chest*. 1993; 103(6):1763–1768.

[7] Krieger J, Maglasiu N, Sforza E, Kurtz D. Breathing during sleep in normal middle-aged subjects. Sleep. 1990;13:143–154.

[8] Tangel DJ, Mezzanotte WS, White DP. Influence of sleep on tensor palatini EMG and upper airway resistance in normal men. *J Appl Physiol (1985)*. 1991;70:2574–2581.

[9] Grace KP, Hughes SW, Horner RL. Identification of the mechanism mediating genioglossus muscle suppression in REM sleep. *Am J Respir Crit Care Med*. 2013;187:311–319.

[10] Hudgel DW. Variable site of airway narrowing among obstructive sleep apnea patients. *J ApplPhysiol (1985)*. 1986; 61(4):1403–1409.

[11] Suratt PM, McTier RF, Findley LJ, Pohl SL, Wilhoit SC. Changes in breathing and the pharynx after weight loss in obstructive sleep apnea. *Chest*. 1987;92(4):631–637.

[12] Malhotra A, Pillar G, Fogel RB, et al. Genioglossal but not palatal muscle activity relates closely to pharyngeal pressure. *Am J Respir Crit Care Med*. 2000;162(3 Pt 1):1058–1062.

[13] Horner RL, Innes JA, Morrell MJ, Shea SA, Guz A. The effect of sleep on reflex genioglossus muscle activation by stimuli of negative airway pressure in humans. *J Physiol*. 1994; 476(8046629):141–151.

[14] Wheatley JR, White DP. The influence of sleep on pharyngeal reflexes. *Sleep*. 1993;16(8 Suppl):S87–S89.

[15] Wheatley JR, Tangel DJ, Mezzanotte WS, White DP. Influence of sleep on response to negative airway pressure of tensor palatini muscle and retropalatal airway. *J Appl Physiol*. 1993; 75(8307868):2117–2124.

[16] Hudgel DW, Chapman KR, Faulks C, Hendricks C. Changes in inspiratory muscle electrical activity and upper airway resistance during periodic breathing induced by hypoxia during sleep. *Am Rev Respir Dis*. 1987;135(4):899–906.

[17] Badr MS, Toiber F, Skatrud JB, Dempsey J. Pharyngeal narrowing/occlusion during central sleep apnea. *J Appl Physiol (1985)*. 1995;78(5):1806–1815.

[18] Horner RL. Impact of brainstem sleep mechanisms on pharyngeal motor control. *Respir Physiol*. 2000;119(2–3):113–121.

[19] Fogel RB, Trinder J, Malhotra A, et al. Within-breath control of genioglossal muscle activation in humans: effect of sleep-wake state. *J Physiol*. 2003;550(Pt 3):899–910.

[20] Berthon-Jones M, Sullivan CE. Ventilatory and arousal responses to hypoxia in sleeping humans. *Am Rev Respir Dis*. 1982;125:632–639.

[21] Douglas NJ, White DP, Weil JV, et al. Hypoxic ventilatory response decreases during sleep in normal men. *Am Rev Respir Dis*. 1982;125:286–289.

[22] White DP, Douglas NJ, Pickett CK, Weil JV, Zwillich CW. Hypoxic ventilatory response during sleep in normal premenopausal women. *Am Rev Respir Dis*. 1982;126:530–533.

[23] Douglas NJ, White DP, Weil JV, Pickett CK, Zwillich CW. Hypercapnic ventilatory response in sleeping adults. *Am Rev Respir Dis*. 1982;126:758–762.

Dr. Shubham Agarwal1" Effect Of Severity of OSA on Oxygen Saturation: An Observational Study Comparing Lowest Oxygen Saturation Attained By Patients of Moderate And Severe Obstructive Sleep Apnea."IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 3, 2018, pp 59-63.