Polysomnographic Spectrum in Obstructive Sleep Apnoea

Shyamala K.K., Bidhata Khatri.

1Dr. Shyamala K.K., Assistant Professor, Department of Pulmonology, Dr. BR Ambedkar Medical College, Bengaluru, Karnataka, India, 2Bidhata Khatri, PharmD Intern, Department of Pharmacy Practice, Acharya & BM Reddy College of Pharmacy, Bengaluru, Karnataka, India.

Address for Correspondence: Dr. Shyamala K.K., Assistant Professor, Department of Pulmonology, Dr. BR Ambedkar Medical College, Bengaluru, Karnataka, India. Email- shyamalakk@gmail.com

Abstract

Introduction: Obstructive sleep apnea (OSA) is a common sleep disordered breathing which is often undiagnosed, unrecognized and under treated represents a major public health problem. It is diagnosed by using overnight Polysomnography. The aim of the study was to analyse the Polysomnographic spectrum in Obstructive sleep apnea.

Methods: The study was prospective and observational type which included all patients diagnosed with OSA by overnight polysomnography (AHI ≥ 5 events per hour) who had visited department of pulmonary medicine from September 2015 to August 2016. Their polysomnographic data with relevant clinical data were collected and analysed.

Results: The mean age of the study group was 49.74 ± 11.59. Our study showed males (41/50 82%) were predominant with male to female ratio of 4:1. Among 50 patients 41(82%) had severe OSA, 8 (16%) moderate and 1 (2%) with mild OSA. The architecture of sleep in OSA was disturbed; the amount of sleep spent in stage N1 had increased to 15.34±9.18, stage N2 and N3 varied and the amount of sleep spent in REM was decreased (9.06 ± 7.93) with varied sleep efficiency. Respiratory events were more in N3 and REM sleep. With CPAP there was definite increase in the stage N3 (P=0.010) and REM sleep (P=0.0000). There was reduction in RDI (P=0.0000). Conclusion: Overnight polysomnography is the standard test recommended for diagnosis of OSA. OSA significantly affected the sleep architecture, sleep efficiency. Early diagnosis by regular sleep evaluation in routine clinical practice would result in better treatment outcome.

Keywords: Polysomnography, Obstructive sleep apnea, Sleep architecture, Continuous positive airway pressure.

Introduction

Obstructive sleep apnoea (OSA) is the major sleep disorder during breathing. This disorder is characterized by repetitive episodes of upper airway obstruction that occur during sleep. OSA usually is associated with reduction of blood oxygen saturation and terminated by EEG arousal [1, 2]. It has been reported affecting 4% in male and 2% in female [3]. Common clinical presentations include loud snoring, excessive daytime sleepiness nocturnal breathing pauses, unrefreshed sleep, tiredness, and mood changes [4]. Increased morbidity and mortality is seen from consequences resulting in neurocognitive dysfunction, cardiovascular disease and automobile accidents which affects quality of life [5-9].

Polysomnography (PSG) is considered as the gold standard in diagnosis. [10]. Different types of PSG studies are:

- Type 1: Fully attended polysomnography (≥ 7 channels) in a laboratory setting
- Type 2: Unattended polysomnography (≥7 channels)
- Type 3: Limited channel study (using 4-7 channels)
- Type 4: One or two channels usually using oximetry [11].

Polysomnography assesses the sleep stages by using EEG, EOG, EMG (neurochannels), cardio-respiratory events by respiratory channels (airflow sensor, snoring, oximetry, ECG) [12]. Diagnostic criteria of OSAHS as per published by the American Academy of Sleep Medicine (AASM) diagnosis can be made if [13].
The respiratory disturbance index (RDI) is ≥ 15, independent of occurrence of symptoms or Whenever an RDI >5 are associated with any of the following:
1. Sleep attacks, excessive daytime sleepiness (EDS), unrefreshing sleep, fatigue or insomnia;
2. Awakenings with a choking sensation; or
3. Witnessed heavy snoring and/or breathing pauses referred by the partner.

Severity is classified depending on AHI [12]
- Mild - 5-15 episodes per hour
- Moderate - 15-30 episodes per hour
- Severe - More than 30 episodes per hour

The pathogenesis of OSA is unclear. Various postulates proved are multifactorial. Ryan et al described OSA as a heterogeneous disorder. Small pharyngeal airway, poor muscle responsiveness during sleep, high loop gain, low sleeping lung volume and low respiratory arousal threshold are involved in the pathogenesis of sleep apnea [14].

Sleep architecture: It is the progression of sleep stages across the night through a repetitive cycle. It is displayed as sleep hypnogram. Sleep cycle: Subjects typically pass through stages of NREM sleep, with REM period every 60-120 minutes. Cycle repeats four to six times through the sleep period. Sleep latency is the time from sleep onset to the first occurrence of each stage. REM latency is the time to the first REM period, normally is greater than 90 minutes. In general, in a healthy young adult, NREM sleep accounts for 75-90% of sleep time (3-5% stage N1, 50-60% stage N2, and 10-20% stages N3). REM sleep accounts for 10-25% of sleep time [15].

Diagnostic criteria for apnea and hypopnea are recommended by AASM task force [12].

Sleep efficiency- It is the calculation of the total sleep time divided by the time in bed expressed as percentage.

Sleep stages:
- Awaken stage_ Designated as “W” or “O”.
- Greater than 50% of each epoch contains alpha activity
- Slow-rolling eye movements or eye blinks seen in EOG channels
- Relatively high EMG muscle tone

Stage N1
- Greater than 50% of the epoch contains theta activity (3-7 cps)
- Alpha activity less than 50% of the epoch
- Slow-rolling eye movements in EOG channels at sleep onset
- Relatively high submental EMG tone less than in wake stage
- Later part of stage vertex sharp waves appear
- Stage 1 is brief lasting for about 1-7 minutes

Stage N2
- Theta activity (3-7 cps)
- High tonic submental EMG
- K-complexes and sleep spindles occur episodically for the first time
- Delta is permitted less than 20% of the epoch
- No specific criteria exist for EOG and EMG

Stage N3
- Referred to as slow wave sleep, deepest stage of sleep
- Accounts for 20-50 % of the epoch containing delta waves/ slow waves 2cps/s in frequency and amplitude of 75 v
- Submental muscle tone may be slightly reduced
- Both K complexes and sleep spindles may be in seen
- No specific criteria exist for EOG and EMG

REM sleep:
Known as paradoxical state, active sleep or D state
- Rapid eye movements
- Mixed frequency EEG (similar to stage 1 pattern but absence of vertex sharp wave and slow eye rolling)
- Low tonic submental

OSA results in loss of sleep architecture, decrease efficiency and latency.
CPAP (Continuous positive airway pressure) is the first line of treatment of OSA. Other modalities of treatment include surgery, oral devices etc.

**Materials and Methods**

The study was carried out in the department of pulmonology at Dr. BR Ambedkar Medical College. It was a prospective and observational study. The study was approved by Institutional Ethical Committee (IEC). Polysomnography data of patients diagnosed as OSA based on overnight polysomnography (AHI ≥ 5 events per hour) and patients who visited outpatient department of pulmonary medicine from September 2015 to August 2016 were included in the study.

Patients below 18 years of study and not willing to participate were excluded from the study. Various polysomnographic variables such as apnea, hypopnea, AHI, RDI, OSA, CSA, mixed apnea, snoring, sleep stages (NREM and REM), sleep efficiency, sleep latency etc along with relevant clinical data were studied.

Respiratory events scoring: [16, 17]

I. Apnoea is defined as cessation of airflow for 10 seconds or longer in adults and classified into three categories.

1. Obstructive apnea
   - Complete or near complete cessation of nasal/oral airflow
   - Increasing respiratory effort; usually seen as paradoxical movement of chest and abdomen
   - Arousal, snoring, oxygen desaturation or arrhythmias accompany the events.

2. Central apnea.
   - Complete or near complete cessation of nasal/oral airflow.
   - Complete absence of respiratory effort.
   - There may not be significant oxygen desaturation accompanying the events.

3. Mixed apnea.
   - Combination of central and obstructive apnoea
   - Begins as a central apnoea, followed by obstructive event.

   - Scored when 25 % or more of the event should be without respiratory effort followed by several obstructive breathing attempts.

II. Hypopnoea: Consequences of partial airway occlusion and increased flow limitation. Defined by variety of criteria most definition include event lasting at least 10 seconds.

   - 50 % or greater reduction in a validated measure of breathing or 50 % or less reduction if associated with an oxygen desaturation of at least 3% or an arousal(Chicago criteria)

   - at least 30% reduction in thoraco-abdominal movement or airflow compared with baseline and with 4% or greater oxygen desaturation (centre for Medicare and Medicaid services)

   - 50% reduction in airflow accompanied by any decrease in oxygen saturation, or

   - Any reduction in airflow with or without oxygen desaturation or arousal

Apnoea hypopnea index (AHI) is the sum of apnoeas and hypopnoeas divided by the total sleep time.

Respiratory disturbance index (RDI) is the sum of apnoeas, hypopnoeas and RERAs (Respiratory effort related arousals) divided by the total sleep time.

**Statistics**

The results were averaged (mean + standard deviation) for each parameter for continuous data and numbers and percentage for categorical data were presented in table/figure.

1) Student ‘t’ test.

The student ‘t’ test was used for determining whether there was a statistical difference between the two groups in the parameters measured.

2) Proportions were compared using Chi-square ($\chi^2$) test of significance for (r x c tables)

   $DF=(r-1)*(c-1)$, where r=rows and c=columns

   $DF= Degrees of Freedom (Number of observation that are free to vary after certain restriction have been placed on the data)

3) One way analysis of variance was used to test the difference between groups. When comparing more than two means, an ANOVA F-test was used.
Results

Majority of the patients were found to be males (41/50, 82%) whereas females were found to be less in number (9/50, 18%). The age group ranged from 30-75 years and the mean age of study group was found to be 49.74 (table1). The associated co-morbid observed was predominantly hypertension showing 55% followed by COPD and diabetes.(Table 2)

Table-1: Age and Gender distribution of the Study population.

| Age     | Male | Female | Total |
|---------|------|--------|-------|
| 30-39 yrs | 11   | 11     | 100.0% |
| 40-49 yrs | 10   | 3      | 76.9%  | 100.0% |
| 50-59 yrs | 15   | 2      | 88.2%  | 100.0% |
| 60-69 yrs | 4    | 2      | 66.7%  | 100.0% |
| 70-79 yrs | 1    | 2      | 33.3%  | 100.0% |
| Total    | 41   | 9      | 50    | 82.0%  | 18.0%  |
| 30-60 yrs | present | 36 | 5 | 41 | (87.8%) | (12.2%) | 100.0% |
| Young§    | 24%  | 9%     |       |

Figure-1: Sleep architecture and OSA severity (Fig correction done below).
Table-2: Co-morbid conditions.

| Associated disease | Frequency | Percent |
|--------------------|-----------|---------|
| Hypertension       | 27        | 55      |
| COPD               | 18        | 34      |
| Diabetes           | 11        | 23      |
| Renal Disease      | 8         | 16      |
| IHD                | 7         | 14      |
| Hypothyroidism     | 5         | 8       |
| Epilepsy           | 1         | 3       |
| Respiratory failure| 1         | 3       |

Polysomnography features: OSA Severity: Among 50 patients 41(82%) had severe OSA, 8(16%) moderate and 1 (2%) with mild OSA. Snoring was present in all the patients.

Sleep architecture and sleep efficiency: The architecture of sleep in OSA was disturbed, the amount of sleep spent in stage N1 had increased to 15.34±9.18, stage N2 and stage N3 varied and the amount of sleep spent in REM was decreased (9.06±7.93) with varied sleep efficiency (table 3).

It was found from the above table with increase in the OSA severity, there was significant reduction in REM sleep (P=0.0000) (fig 1).

Table-3: Sleep architecture and sleep efficiency.

| Polysomnography     | N  | Mean   | Std. Deviation | Minimum | Maximum | ANOVA ‘F’ | ‘p’ value |
|---------------------|----|--------|----------------|---------|---------|-----------|-----------|
| RDI                 | 50 | 65.16  | 31.70          | 11      | 124     | 1.961     | .152      |
| % of sleep time     |    |        |                |         |         |           |           |
| Stage N1            | 50 | 15.34  | 9.18           | 2       | 43      | .113      | .893      |
| % of sleep time     |    |        |                |         |         |           |           |
| Stage N2            | 50 | 46.56  | 17.86          | 10      | 81      | .393      | .677      |
| % of sleep time     |    |        |                |         |         |           |           |
| Stage N3            | 50 | 21.34  | 17.17          | 0       | 59      | 11.271    | .000      |
| REM                 | 50 | 9.06   | 7.93           | 0       | 30      | .507      | .606      |
| Sleep Efficiency    | 50 | 82.36  | 15.52          | 33      | 100     |           |           |

Effect of CPAP on Sleep architecture- With CPAP there was definite increase in the stage N3 (P=0.010) and REM sleep (P=0.0000).

Polysomnography results before and with CPAP titration showed significant changes in RDI (P=0.000) and sleep architecture among 30 patients who underwent titration study, without any significant changes noted in sleep efficiency (fig 2).

It was found that OSA severity decreased with increase in the age beyond 60 years, but the result was not statistically significant (table 4).
Figure-2: Effect of CPAP Titration.

Table 4: Age and OSA Severity.

| Age       | OSA Severity | Total |
|-----------|--------------|-------|
|           | Mild | Moderate | Severe |       |
| 30-39 yrs | 2    | 9        | 11     |       |
|           | 25.0%| 22.0%    | 22.0%  |       |
| 40-49 yrs | 4    | 9        | 13     |       |
|           | 50.0%| 22.0%    | 26.0%  |       |
| 50-59 yrs | 1    | 1        | 15     | 17    |
|           | 100.0%| 12.5%    | 36.6%  | 34.0% |
| 60-69 yrs | 1    | 5        | 6      |       |
|           | 12.5%| 12.2%    | 12.0%  |       |
| 70-79 yrs | 3    | 3        |        |       |
|           | 7.3% | 6.0%     |        |       |
| Total     | 1    | 8        | 41     | 50    |
|           | 100.0%| 100.0%  | 100.0% | 100.0%|

Chi-Square Value df 'p' value
5.775 8 .672

Out of the 9 female patients, 7 were found to have severe OSA which is less number compared to males. As the sample size was less, severity between males and females was not statistically significant (table 5).

Table 5: Gender and OSA Severity.

| Sex   | OSA Severity | Total |
|-------|--------------|-------|
|       | Mild | Moderate | Severe |       |
| Male  | 1    | 6        | 34     | 41    |
|       | 2.4% | 14.6%    | 82.9%  | 100.0%|
| Female| 2    | 7        | 9      |       |
|       | 22.2%| 77.8%    | 100.0% |       |
| Total | 1    | 8        | 41     | 50    |
|       | 2.0% | 16.0%    | 82.0%  | 100.0%|

Chi-Square Value df 'p' value
.509 2 .775
Discussion

In our study, age group of the patients ranged from 30-75 years similar to the observation of Sharma and Udwadia [18,19]. OSA was predominantly observed in males with male to female ratio of 3:1 as reported by T. Young [1,3]. In the present study, 82% of the subjects were males. Majority of the patients (55%) of patients had hypertension as other comorbidity similar to the observation reported by McNicholas and Sharma [8,18]. Other associated co morbid conditions noted were 34% of COPD/ RAD, higher than that reported by Chaouat [20] and 3% had hypercapnic respiratory failure in association with COPD/RAD. It was also found that 3% had reported to have RTA/ Near misses in the study group, lesser incidence compared to that reported by George in his study [9].

Polysomnography is considered as a gold standard in the diagnosis of OSA. The severity of OSA is defined on the basis of AHI. Our study group had 41 cases with severe OSA, 8 were moderate, and 1 had mild OSA showing against the epidemiological study by T. Young with majority of patients with mild OSA and less number of severe OSA [1,3]. Polysomnography features showed altered sleep architecture, with significant REM sleep deprivation. RDI of >5-15 events/hour was found in 1 subject, 8 patients had moderate OSA (RDI of 15-30) and remaining 41 subjects had severe OSA (RDI of >30). OSA affected sleep architecture resulting in deprivation of stage N3 and REM sleep though there was not much change in sleep efficiency.

In our study changes were found in sleep architecture with definite decrease in REM sleep with the increase in severity of OSA were improved by the use of CPAP. Though there has been increasing awareness and testing centers are available at major hospitals and medical colleges, the cost of testing and treatment is the major barrier for detecting OSA. Decreasing the cost of the test and wider range of availability of testing at different centers would yield increased diagnosis and better treatment outcome.

Funding: Nil, Conflict of interest: None initiated, Permission from IRB: Yes

References

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. New Engl J Med. 1993; 328 (17):1230–5.

2. Young T, Peppard P, Gottlieb D. Epidemiology of obstructive sleep apnea. Am J Respir Crit Care Med. 2002;165(9):1217-39.

3. Gibson GJ. Obstructive sleep apnoea syndrome: underestimated and undertreated. Br Med Bull. 2005 Mar 29;72:49-65. Print 2004.

4. Duran J, Esnaola S, Rubio R, Iztueta A. Obstructive sleep apnea-hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 year. Am J Respir Crit Care Med. 2001; 163:685-9.

5. Schlosshan D, Elliott MW. Sleep. 3: Clinical presentation and diagnosis of the obstructive sleep apnoea hypopnoea syndrome. Thorax. 2004 Apr;59 (4): 347-52.

6. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. Chest. 1988 Jul; 94 (1):9-14.

7. Engleman HM, Douglas NJ. Sleep. 4: Sleepiness, cognitive function, and quality of life in obstructive sleep apnoea/ hypopnoea syndrome. Thorax. 2004 Jul; 59 (7):618-22.

8. George C. Sleep (middle dot) 5: Driving and automobile crashes in patients with obstructive sleep apnoea/hypopnoea syndrome. Thorax.2004;59(9):804-7.
9. Sleep apnoea as an independent risk factor for cardiovascular disease: current evidence, basic mechanisms and research priorities. W.T. McNicholas, M.R. Bonsignore and the management committee of EU cost action B26. Eur Respir J. 2007;29:156-78.

10. Carmal A. [Internet]. Emedicine.com. 2007 [cited 10 October 2016]. Available from: http://carmal a polysomnography: overview and clinical application. e-medicine.com 2007.

11. SK S. Policy document consensus & evidence-based INOSA guidelines 2014 (First Edition). Journal of Clinical and Scientific Research.2015;:70-91.

12. American Academy of Sleep Medicine. International classification of sleep disorders. diagnostic and coding manual. Westchester, Illinois; AASM: 2005.

13. 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 Aug 1;22(5):667-89.

14. Ryan CM, Bradley TD. Pathogenesis of obstructive sleep apnea. J Appl Physiol (1985). 2005 Dec;99 (6): 2440-50.

15. Harris CD. Neurophysiology of sleep and wakefulness. Respir Care Clin N Am. 2005 Dec;11 (4):567-86.

16. Rechtschaffen A, Kales A: A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Washington, DC: US Government Printing Office, US Public Health Service; 1968.

17. Russo M. [Internet]. e-medicine.com 2005 [cited 10 October 2016]. Available from: http://russo m sleep stage scoring emedicine.com 2005.

18. Udwadia ZF, Doshi AV, Lonkar SG, Singh CI. Prevalence of sleep-disordered breathing and sleep apnea in middle-aged urban Indian men. Am J Respir Crit Care Med. 2004 Jan 15;169(2):168-73. Epub 2003 Nov 6.

19. Sharma SK, Kumpawat S, Banga A, Goel A. Prevalence and risk factors of obstructive sleep apnea syndrome in a population of Delhi, India. Chest. 2006 Jul;130(1):149-56.

20. Chaouat A, Weitzenblum E, Krieger J, Ifoundza T, Oswald M, Kessler R. Association of chronic obstructive pulmonary disease and sleep apnea syndrome. Am J Respir Crit Care Med.1995;151:82-86.

21. Sreedharan SE, Agrawal P, Rajith RS, Nair S, Sarma SP, Radhakrishnan A. Clinical and polysomnographic predictors of severe obstructive sleep apnea in the south indian population. Ann Indian Acad Neurol. 2016;19:216-20.

22. Garg R, Singh A, Prasad R, Saheer S, Jabeed P, Verma R. A comparative study on the clinical and polysomnographic pattern of obstructive sleep apnea among obese and non-obese subjects. Ann Thorac Med. 2012;7(1):26-30.

How to cite this article?

Shyamala K.K, Bidhata Khatri. Polysomnographic Spectrum in Obstructive Sleep Apnoea. Int J Med Res Rev 2016;4(12):2100-2107.doi:10.17511 /ijmrr. 2016.i12.04.