ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research

Vol. 16, Issue, 05, pp.28153-28155, May, 2024

DOI: https://doi.org/10.24941/ijcr.47136.05.2024

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

OBSTRUCTIVE SLEEP APNEA

Dr. Ligil A.R., Dr. Rosaline Tina Paul, Dr. Roshna Mandayapurathu, Dr. Joseph K. Thanikunnel and Dr. Parsen Paul

PSM Dental College, India

ARTICLE INFO

Article History:
Received 19th February, 2024
Received in revised form
09th March, 2024
Accepted 25th April, 2024
Published online 20th May, 2024

Key words:
Obstructive Sleep Apnea.

Corresponding author:** ***Dr. Ligil A.R.,

ABSTRACT

Obstructive sleep apnea (OSA) is a potentially serious and life-threatening disorder affecting millions of people around the world. It is a sleep-related respiratory condition, characterized by the complete or partial collapse of breathing because of a narrowing or closure of the upper airway during sleep, resulting in intermittent cessations of breathing (apneas) or reductions in airflow (hypopneas) despite ongoing respiratory effort. The symptoms include excessive daytime sleepiness, Mood changes, Fragmented sleep, as well as the decreased health-related quality of life. Patients often complain of snoring, Gasping or choking, frequent nocturnal awakenings, early morning headaches, poor concentration and coordination, anxiety, irritability, and insomnia, yet many patients are unaware of these symptoms and disease onset is insidious. This article details the obstructive sleep apnoea

Citation: Dr. Ligil A.R., Dr. Rosaline Tina Paul, Dr. Roshna Mandayapurathu, Dr. Joseph K. Thanikunnel and Dr. Parsen Paul. 2024. *"Obstructive sleep apnea". International Journal of Current Research, 16, (05), 28153-28155.*

INTRODUCTION

Obstructive sleep apnea (OSA) is a potentially serious and life-threatening disorder affecting millions of people around the world. It is a sleep-related respiratory condition, characterized by the complete or partial collapse of breathing because of a narrowing or closure of the upper airway during sleep, resulting in intermittent cessations of breathing (apneas) or reductions in airflow (hypopneas) despite ongoing respiratory effort. The symptoms include excessive daytime sleepiness, Mood changes, Fragmented sleep, as well as the decreased health-related quality of life. Patients often complain of snoring, Gasping or choking, frequent nocturnal awakenings, early morning headaches, poor concentration and coordination, anxiety, irritability, and insomnia, yet many patients are unaware of these symptoms and disease onset is insidious. The economic burden of OSA is substantial due to its high prevalence and economic costs in the community globally, the profound clinical effects on an individual's cognitive and general functioning and the increased risk of adverse health complications. Moreover, mounting evidence suggests that OSA can increase the risk of cardiovascular diseases (hypertension, coronary heart failure, stroke etc.), metabolic syndrome, and neurological problems. "Apnea" is the Greek word for "without breath." Obstructive sleep apnea (OSA) was first described by Charles Dickens in 1837 in Charles Dickens' habitus, daytime behaviour, snoring and sleep. The character Joe of Pickwick Papers was fat, loud snorer, who was difficult to arouse from sleep, and had severe daytime sleepiness: all of which are the constellation of the clinical features of the obstructive sleep apnoea (OSA). Periodic breathing was reported by British physician Hunter and by Irish physicians Cheyne and Stokes in heart failure patients in the early to mid-19th Century, and also by the British Physiologists John Scott Haldane, C. G. Douglas, and Mabel Fitzgerald at the turn of the 20th Century in healthy subjects sleeping in the hypoxia of high altitudes.[2] In 1918, Sir William Osler described relationship between obesity and Pickwickian syndrome. It was recognised in the early 1970s, that these symptoms may reflect disturbed breathing during sleep. Gastaut and associates in 1965 showed that cessation of respiration was due to the obstruction of upper airway and Obstructive Sleep Apnoea was recognised.[1]This observation opened an entirely new area of respiratory medicine, namely sleep-related breathing disorders (SRBD). In normal sleep there is an orderly progression of stages of sleep, starting with light sleep (stages I and II), progressing to deeper sleep (stages III and IV), and ending up with rapid eye movement (REM) sleep. During non-REM sleep (stages I- IV) breathing is slow and regular, while during REM sleep the breathing pattern is erratic, with rapid, shallow respirations.[2]This relative hypotonia is particularly pronounced in REM sleep, where most of the disordered breathing events occurs. [3]

Apnea is a complete cessation of airflow for 10 seconds or longer. A hypopnoea is defined as a reduction in airflow (30-50%) that is followed by an arousal from sleep or a decrease in oxyhaemoglobin saturation (3-4%). During the course of a night's sleep a person with OSA will have multiple apnea and/or hypopnea events, as many as 100 per night, which will trigger signals to the brain to arouse the person from their sleep state to regain a normal course of breathing. The person struggles to breathe, the diaphragm and chest muscles work harder to open the obstructed airway and pull air into their lungs. This is then followed by a loud gasp, snort or body jerk. [3.] To restore pharyngeal patency, individuals have recurrent arousals from sleep, which lead to activation of the sympathetic nervous system and fragmentation of sleep. These haemodynamic events have been clearly associated with development of systemic hypertension and may also be the substrate for subsequent myocardial infarction, cerebrovascular events, and congestive heart failure. Fragmentation of sleep can lead to reduced neurocognitive function, decreased quality of life, and increased risk of motor vehicle and occupational accidents. Obtaining a deep level of sleep each night is vitally important for sustaining immune function, memory, psychologic wellbeing, biochemical refreshment and decreasing fatigue. With the decrease in oronasal airflow, arterial blood oxygen levels are also decreased (known as hypoxemia) thereby increasing the risk of organ tissue damage due to oxygen/carbon dioxide imbalances. [4]

Changes in the airway dimensions during respiration have been well characterized. In inspiration upper airway area is relatively constant, inferring a balance between airway dilator muscle activity and negative intraluminal pressure. During early expiration, increased intrathoracic pressure leads to maximal airway enlargement. Finally, in late expiration there is significant reduction in airway caliber to its smallest dimensions at end of expiration. These findings have also been replicated in patients with OSA. Thus, the greatest susceptibility for airway collapse or narrowing is at the end of expiration or during inspiration.[4]

Normally, with each inspiration the pressure in the upper airway falls below atmospheric pressure, thus creating a tendency for the upper airway to collapse. This tendency is exaggerated during sleep because of the posterior and downward displacement of the tongue and the soft palate. This displacement increases pharyngeal resistance and further lowers pharyngeal transmural pressure (difference between the pressure in the airway lumen and the pressure exerted by the tissues surrounding the site of collapse). In patients with OSA, pharynx is narrower and more lax than normal subjects due to fat deposition in the parapharyngeal fat pads, in the tongue and under the mandible in the submental region significantly reducing the upper airway calibre, which predisposes them to development of complete pharyngeal occlusion during sleep.[5]Imaging studies have also demonstrated that the total volume of fat surrounding the airway is greater in apnoeic than in BMI-matched normal individuals, suggesting that fat deposition in neck has an important role to play in the pathogenesis of OSA.[6]A neck circumference >43.2 cm is also a positive risk factor for OSA.[7] Other factors that have important bearing on upper airway narrowing include genetics, male gender, pharyngeal dilators muscle dysfunction, soft tissue oedema (secondary to snoring/ apnoea related trauma), airway tissue properties (surface tension), vascular perfusion , variety of craniofacial defects, obesity, nasal obstruction, enlarged tonsils/adenoids, macroglossia and low-lying soft palate.[8]

There are 3 types of sleep apnea: [9] Obstructive Sleep Apnea (OSA) is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstructionduring sleep. OSA is the most common form of sleepapnea. Central Sleep Apneais not obstructive in natureinstead caused when respiratory control centers in the braintemporarily pause the activation of the muscles involved inbreathing. Mixed is a combination of OSA and Central Sleep Apnea. Sleep apnea is diagnosed using polysomnography (PSG). A PSG, or sleep study, is performed in a sleep laboratory and involves continuous overnight recordings measuring the patient's breathing and respiratory efforts as well as blood oxygen saturation, body position, heart monitoring and brain wave function. A PSG will yield an AHI (apnea-hypopnea index) for the patient. As hypopneas lead to the same clinical consequences as apneas, the apnoea-hypopnoea index (AHI) is widely used for the diagnosis and the assessment of the severity of OSA. The AHI or respiratory disturbance index (RDI) refers to the mean number of apnoeas or hypopneas per hour of sleep. An AHI (average number of apneas and hypopneas per hour of sleep) greater than 5 defines OSA.[9]

OSAHS may be subdivided into three categories degrees of breathing abnormality, on the basis of AHI: [9]

- Mild AHI 5-14/hr
- Moderate AHI 15-30/hr
- Severe AHI >30/hr

American Sleep Disorders Association (ASDA) has classified OSAHS[9] Sleepiness into:

- Mild: unwanted sleepiness or involuntary sleep episodes occur during activities that require little attention.
- Moderate: unwanted sleepiness or involuntary sleep episodes occur during activities that require some attention.
- Severe: unwanted sleepiness or involuntary sleep episodes occur during activities that require active attention.

To have a comprehensive view of the entire patient, a thorough evaluation also should be done, including an accurate medical history, physical examination, airway analysis, and other additional aids to identify the sites of obstruction in the airway. The physical examination includes the weightand body mass index (BMI) of the patient. The clinical examination of the airway starts at the nasal aperture. Any nasal septal deviation, internal or external valve collapse, turbinate hypertrophy, nasal polyps, chronic sinusitis, and masses can contribute to reduced nasal airway, which leads to increased negative inspiratory pressure and bring about collapse in the posterior airway.[10]

The Epworth Sleepiness Scale (ESS) is a validated method used to assess the probability of falling asleep. The score subdivide the patients clinically into 4 categories: [11]

- Normal range ESS <11
- Mild subjective daytime sleepiness ESS =11
- Moderate subjective daytime sleepiness ESS =16
- Severe subjective daytime sleepiness ESS >18

In the last two decades, advances in sleep medicine and the availability of improved diagnostic tools have led to a better recognition and treatment of the disease. The management of patients with OSA requires a multidisciplinary approach and many treatment options are currently available. oximetry is used as the first screening tool for OSAHS. These are spectrophometric devices that are used for the detection and calculation of the differential absorption of light by presence of oxygenated and deoxygenated haemoglobin in blood.[12] Positive airway pressure (PAP), available since the beginning of the 1980s, provides the most effective and commonly used treatment. Alternative options include weight control, mandibular advancement devices and a number of upper airway surgical approaches. Despite the recent advances in diagnostic technology in the field of sleep medicine and increased awareness of OSA in the public, a majority of those affected are still undiagnosed. Therefore, it is important for primary care physicians and specialists to be competent to recognise and identify those affected subjects for early and appropriate treatments.

REFERENCES

1. Gastaut H, Tassinari CA, Duron B. Polygraphic study of the episodic diurnal and nocturnal (hypnic and respiratory) manifestations of the Pickwick syndrome. Brain Res 1: 167–186, 1966
2. Douglas CG, Haldane JS, Henderson Y, Schneider EC. Physiological observations made on pikes peak CO, with special reference to adaptaion to low barometric pressure. Phil Trans R SocLondSer B 203: 185–318, 1913
3. Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnoea. J Clin Invest 1995; 96: 1897–904.
4. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of sleep apnea. Physiol Rev. 2010;90(1):47-112
5. Mezzanotte WS, Tangel DJ, White DP. Influence of sleep onset on upper-airway muscle activity in apnoea patients versus normal control. Am J RespirCrit Care Med 1996; 153: 1880–87.
6. Schwab RJ, Gupta KB, Gefter WB, Metzger LJ, Hoffman EA, Pack AI. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing: significance of the lateral pharyngeal walls. Am J RespirCrit Care Med 1995; 152: 1673–89
7. Shelton KE, Woodson H, Gay S, Suratt PM. Pharyngeal fat in obstructive sleep apnea. Am Rev Respir Dis. 1993;148:462–6.
8. Schlosshan D, Elliott MW. Sleep 3: Clinical presentation and diagnosis of the obstructive sleep apnoea hypopnea syndrome. Thorax 2004;59:347–352
9. American Academy of Sleep Medicine Task Force 1999. Sleeprelatedbreathing disorders in adults: recommendations forsyndrome definition and measurement techniques in clinicalresearch. Sleep 22: 667-689.
10. KokWeng Lye and Joseph R. 2009. DeatherageSurgical Procedures for the Treatment of Obstructive Sleep ApneaSeminars in Orthodontics, Vol 15, No 2 (June), pp 94-98
11. Johns MW 1993. Daytime sleepiness, snoring, and obstructive sleep apnea. The Epworth Sleepiness Scale. Chest 103: 30-36.
12. Vazquez JC, Tsai WH, Flemons WW, Masuda A, Brant R, Hajduk E, et al. (2000) Automated analysis of digital oximetry in the diagnosis of obstructive sleep apneoa. Thorax 55: 302-307

****