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Review Article

Sleep Apnea

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Abstract

Sleep apnea was first described in humans in 1965 [1,2]. Sleep disorder breathing (SDB) is usually multi-factorial in nature without a single solution available. It is comprises a wide spectrum of sleep-related breathing abnormalities; those related to increase upper airway resistance include snoring, upper airway resistance syndrome (UARS) and Obstructive Sleep apnea-Hypopnea Syndrome (OSAHS) [4]. Central Sleep Apnea (CSA) is hyperventilation without upper airway obstruction likes high altitude sickness.

The estimated prevalence of OSA in North America is 20–30% in males and 10-15% in females [5]. Risk factors for OSA include advanced age, male gender and craniofacial or upper airway soft tissue abnormalities [6]. A recent cluster analysis identified OSA asymptomatic status associated with discrete cardiovascular risk profiles and associated with significant cardiovascular, metabolic, and neurocognitive consequences [9].

Polysomnography (PSG) and portable home sleep testing are the main methods of diagnosing OSA. Continuous positive airway pressure (CPAP) therapy is first-line treatment for OSA and it is first line therapy of mild, moderate and severe OSA [10]. Dental Appliances also includes in first line therapy for OSA. Second-line therapy is recommended for the snoring and mild-to-moderate OSA intolerant of CPAP and oral devices. Adjuvant therapies include avoidance of alcohol and narcotics at nights, positioning belts, weight loss and bariatric surgery [11,12].

Keywords: *Sleep Apnea; Sleep Disorder Breathing (SDP); Obstructive Sleep Apnea (OSA) Continuous Positive Airway Pressure (CPAP)*

Introduction

Sleep apnea was first described in humans in 1965 [1,2]. Sleep disorders are usually multi-factorial in nature, very often without a single solution available. Sufficient evidence now exists to indicate that prevention of the onset of the symptoms, which are often referred to as 'the disease', can minimize or even eliminate the need for intervention. It is not possible for a person to have functional breathing during the day and dysfunctional breathing during the night, any more than it is to have the reverse situation. Majority of people spend approximately two thirds of their lives awake and active, there is a significant likelihood that addressing daytime dysfunction is at least as important as, if not more important than, applying short term night time intervention.

This is why there is so much validity in the concept of Breathing Disordered Sleep (BDS) as the etiology of the problem - rather than the other way round. There is no doubt that there are millions of people suffering from Sleep Disorders, and that only a very small percentage is being reported and successfully treated and it is beyond important that we try to understand why this is so.

Sleep disorder breathing is now being a multi-billion dollar industry of less than 10% of the suspected cases of Sleep Disorders. Therefore, looking into disproportionate benefits with such vast amounts of money being spent on so small segment of the respiratory medical market. Greater concern till today in the area of Sleep Apnea is of low compliance rate of these devices and the actual cost per successful patient.

Sleep-disordered breathing is an umbrella term for several chronic conditions in which partial or complete cessation of breathing occurs many times throughout the night during sleep resulting in daytime sleepiness or fatigue that interferes with a person's ability to work and reduces quality of life [3]. While other School of thought defines Sleep Disorder Breathing (SDB) comprises a wide spectrum of sleep-related breathing abnormalities; those related to increase upper airway resistance include snoring, upper airway resistance syndrome (UARS), and obstructive sleep apnea-hypopnea syndrome (OSAHS) [4].

Etiology

Sleep Disorder Breathing (SDB) is multi factorial breathing disorder caused by many know and yet unknown causes. The following picture of the ice berg analogy could explain many known and unknown involved factors responsible for SDB.

The Iceberg Analogy

The 10% of the iceberg that is visible is not what presents the danger. It is the 90% hidden below the surface that sinks ships. This diagram indicates the ratio of symptoms to etiology - and if one takes into consideration the billions of dollars expended on above-the-line intervention, against the almost negligible focus on the below-the-line causes or exacerbatory factors, it is no wonder that the problems remain unresolved to the extent that they are.

Figure 1

Therefore addressing the 'below-the-line' issues such as posture, breathing mechanics, nutrition, stress and the rest of the co-factors would go a long way in providing a more comfortable, acceptable and stable outcome with far greater compliance.

Ignoring the presence of these multiple factors will result in being confined to short-term night-time intervention. Handling such multi-factorial issues requires the involvement of a trained team of therapists, working in conjunction, to get the required result.

Pathophysiology

The Pathophysiology of SDB is a combination of factors which involve the brain, the heart and lungs. A key factor is enhanced chemoreceptor responses, the degree to which someone hyperventilate in response to hypoxia and hypercapnia.

In healthy individuals carbon dioxide levels normally rise during sleep. If we go to sleep with a low CO_2 level below a certain value (the apnea threshold') breathing will be inhibited and reduce or even stop until the CO_2 rises, when breathing resumes.

A characteristic feature of Central Sleep Apnea (CSA) is hyperventilation without upper airway obstruction like high altitude sickness. The difference is that in altitude sickness it's hypoxia that is the primary factor driving hyperventilation and hypocapnia is a consequence of that. The mean cause of Central Sleep Apnea is heart failure where increased ventilation due to a number of factors including

the fact that the lungs are wet and receptors in the lungs are sending nerve signals to the respiratory control centers in the brainstem to cause hyperventilation. Patients with heart failure and healthy individuals at high altitude hyperventilate and the resultant hypocapnia making breath unstable during sleep. This is due to the prolongation of circulation time that the effects of blood gas changes in the periphery take longer to reach the brainstem where control of breathing occurs.

Obstructive sleep apnea (OSA) describes recurrent complete (apnea) or partial (hypopnea) collapse of the upper airway during sleep. The estimated prevalence of OSA in North America is 20–30% in males and 10-15% in females and this figure rapidly on the rise with increase prevalence of obesity [5].

Additional risk factors for OSA include advanced age, male gender, and craniofacial or upper airway soft tissue abnormalities. During sleep, efforts of breathing against an occluded upper airway lead to impaired gas exchange, swings in intrathoracic pressure, and sleep fragmentation. OSA severity is quantified by the apnea hypopnea index (AHI) which measures the frequency of disordered events [6].

Obstructive Sleep Apnea (OSA) is susceptibility of the upper airway collapses during sleep. This susceptibility is governed both by passive anatomical properties and by active control of airway dilator muscles. During sleep, the susceptibility of collapse is quantified by determining the luminal collapsing pressure, called the critical pressure ("Pcrit") [7,8].

In normal subjects, substantial negative pressure may be necessary to induce airway collapse during sleep (e.g. Pcrit = -10 cm H_20) whereas in OSA the Pcrit may be greater than 0, reflecting the need for CPAP to maintain airway patency.

OSA is associated with significant cardiovascular, metabolic, and neurocognitive consequences. Patients with OSA can have different clinical presentations ranging from insomnia to excessive daytime sleepiness [9]. For example, a recent cluster analysis identified OSA patients with sleep-related complaints, daytime sleepiness, or asymptomatic status associated with discrete cardiovascular risk profiles [9].

Diagnosis

History and physical exam alone often are not diagnostic as > 50% of patients doesn't have daytime sleepiness. Witnessed snoring and apneas have a high positive predictive value of 64%, with witnessed apneas being the best historic predictor.

Apnea–Hypopnea Index (AHI)

This Measure of the severity of sleep apnea.

Number of apneas + hypopneas/hour of sleep:

1. AHI 0–5 Normal
2. AHI 5–15 Mild SDB
3. AHI 15–30 Moderate SDB
4. AHI > 30 Severe SDB

Respiratory Disturbance Index (RDI)

Number of apneas + number of hypopneas + respiratory effort-related arousals/hour of sleep:

1. RDI 0–5 Normal
2. RDI 5–15 Mild
3. RDI 15–30 Moderate
4. RDI > 30 Severe

In laboratory, Polysomnography (PSG) and portable home sleep testing are the main methods of diagnosing OSA. PSG is the gold standard for the diagnosis of SDB (Figure 2). Portable sleep studies are used for patients with a high pretest probability for OSA and without co morbid medical conditions such as cardiovascular disease, stroke, chronic obstructive pulmonary disease (COPD), and hypoventilation syndromes.

Polysomnography (PSG)

Records physiologic variables during sleep using electroencephalography/-gram (EEG), electrooculography/-gram (EOG), chin electromyography/-gram (EMG), electrocardiography/-gram (ECG), oxygenation, snoring, respiratory effort, and leg (anterior tibialis) EMG (Figure 2).

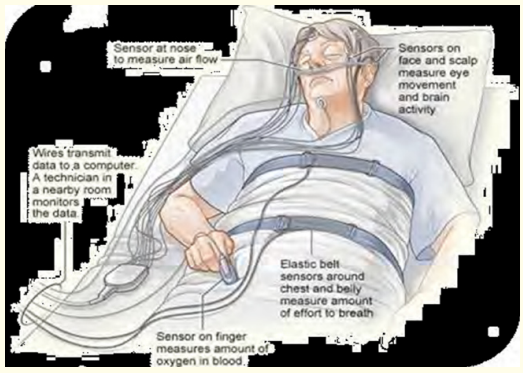

Figure 2: *Polysomnography setup where patient lies in bed with EEG, EOG, Chin EMG, ECG, airflow, oxygenation, snoring, respiratory effort, and leg EMG (Electromyogram).*

Treatment

Continuous positive airway pressure (CPAP) therapy is first-line treatment for OSA (figure 3) and it is first line therapy of Mild, Moderate and severe OSA [10]. CPAP Improves AHI, daytime sleepiness, quality of life.

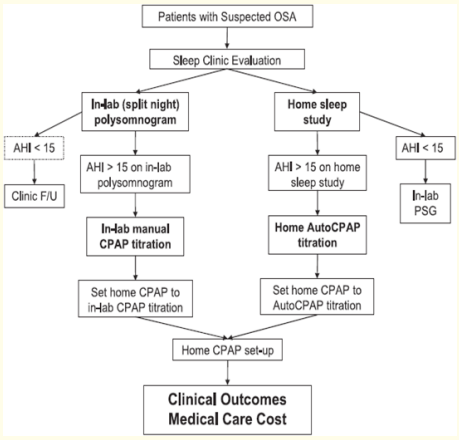

Figure 3: *Alternative clinical management pathways for patients with obstructive sleep apnea (OSA). The pathway on the left represents the standard in-labortory management and the pathway on the right represents a possible approach using home-unattended portable-monitor testing .PSG, polysomnogram; CPAP, continuous positive airway pressure.*

Other first line therapies for sleep apnea include dental appliances such as mandibular advancement devices or tongue retaining devices.

Second-line therapy is recommended for the snoring and mild-to-moderate OSA intolerant of CPAP and oral devices. Uvulopalatopharyngoplasty (U3P) generally is unsuccessful in resolving OSA and is not recommended in most cases.

In addition, Maxillomandibular advancement is successful in resolving OSA while Nasal EPAP is considered to be an alternative therapy for Mild OSA intolerant of CPAP and oral devices.

Adjuvant therapies like avoidance of alcohol and narcotics at nights, positioning belts and pillows helps patients to sleep laterally as OSA is generally is worse in the supine position. Weight loss and bariatric surgery improve but rarely resolve OSA and apnea–hypopnea index (AHI) [11,12].

Conclusion

The rising prevalence of SDB/OSA and increasing evidence of its association with cardio-metabolic diseases demands further research to address the true impact of this disorder. Going forward, we advocate for increasing attention to clinical realism in modeling OSA and other types of SDB.

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