"EVALUATION OF SLEEP DISORDERED BREATHING BY LEVEL I POLYSOMNOGRAPHY IN A TERTIARY CARE HOSPITAL: A HOSPITAL BASED OBSERVATIONAL STUDY."

By

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Dissertation

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Date: *Dr Anusha.C.M*

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LIST OF ABBREVIATIONS USED

AASM	American Academy of Sleep Medicine
AHI	Apnea-Hypopnea Index
BIPAP	Bi-level positive airway pressure
BMI	Body Mass Index
COPD	Chronic obstructive airway disease
CPAP	Continuous Positive Airway Pressure
CSR	Cheyne-stokes respiration
EEG	Electroencephalogram
EDS	Excessive daytime sleepiness
EMG	Electromyogram
EOG	Electrooculogram
EPAP	Expiratory pressure airway therapy
HST	Herbst appliance
KZY	Karwetzky activator
LAGB	Laparoscopic adjustable gastric banding
GERD	Gastroesophageal reflux disease
HLP	Hyperlipoproteinemia
INOX trial	Internal nocturnal oxygen trial
IHD	Ischemic heart disease
MAP	Multivariable Apnea Prediction
MAS	Mandibular advancement splint
NIV	Non-invasive ventilation
NREM.	Non rapid eye movement
OSA	Obstructive Sleep Apnea

OSAS	Obstructive Sleep Apnea Syndrome
OA	Oral appliances
PAP	Positive airway pressure
PLM	Periodic limb movement
PSG	Polysomnography
RDI	Respiratory Desaturation Index
REM Sleep	Rapid Eye Movement Sleep
SDB	Sleep Disorder Breathing
SWS.	Slow wave sleep
TIB	Time in bed
TST	Total sleep time
WASO	Wake up after sleep onset

ABSTRACT

"EVALUATION OF SLEEP DISORDERED BREATHING BY LEVEL I POLYSONOGRAPHY IN A TERTIARY CARE HOSPITAL : A HOSPITAL BASED OBSERVATONAL STUDY"

Authors: Dr. Anusha CM, Dr. Gajanan S Gaude

Background: Sleep Disorder Breathing (SDB) is described as group of disorders with characters of abnormal respiratory patterns like apnea or hypopneas, or inadequate oxygen while asleep. With increasing urbanization and changes in life style modification, obstructive sleep apnea with hypersomnolence may have bigger social impact in a developing country like India. Hence our study was conducted to evaluate sleep discorded breathing patterns which was diagnosed using Level 1 polysomnography study and its correlation of severity of sleep apnea syndrome with BMI

Aims and Objective: To study various sleep disordered breathing patterns diagnosed by Level 1 polysomnography study and to correlate severity of sleep apnea syndrome with BMI.

Materials and methods : Following institutional ethical committee approval, the study was conducted at KLE's Dr Prabhakar Kore Charitable Hospital, Belagavi. 60 patients coming to outpatient clinic with Epworth sleepiness scale between 8 to 24 representing increasing levels of Excessive day time sleepiness along with with nocturnal awakening, choking episodes, day time tiredness, weight gain were included after informed written consent.Patients with symptoms of SDB were subjected to detailed history taking and clinical examination. Level I

Polysomnography study in a quiet, dark, temperature controlled room with constant monitoring. The ECG, Central-Occipital EEG, Submental EMG, Nasal-oral air flow, Arterial Oxygen saturation, breathing pattern by Thermister, Cannula, Thoracic wall movements, Abdominal Movements, Anterior Tibialis EMG, Snoring, Body Position were variables measured by sleep technician from 10pm to 6am. Sleep scoring was done by sleep technician.

Results: There were 60 (34 males and 26 females) subjects with mean age of 56.45±12.51 years and body mass index (BMI) of 28.7±3.1. 16, 9, 22 subjects had Mild, moderate, severe OSA respectively. 73 % had excessive daytime sleepiness (EDS), 10 % had history of previous accidents with mean BMI 28.7±3.1. Mean ESS was 15.1±6.3. PSG parameters showed poor sleep efficiency (mean ; 71±22.2%) and lowest TST (219.3±137.9 minutes) in severe OSA . Mean AHI was 28.49±30.98. We found that age >55 years, BMI >25 kg/m^2, witnessed apneas, EDS, reduced slow wave sleep duration, RDI correlated well with OSA severity while the BMI, Mallampatti score, ESS showed no association.

Conclusion: OSA predominantly affects middle-aged men who are overweight with many having high neck circumference.. Severe OSA patients had longer apneas and more severe nocturnal desaturation, compared to mild to moderate disease despite having similar sleep architectures and fragmentations.

Keywords: Sleep Disorder Breathing, Polysomnography, Obstructive Sleep Disorder Breathing, Apnea Hypoapnea Index.

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INTRODUCTION

Sleep Disorder Breathing (SDB) is described as group of disorders with characters of abnormal respiratory patterns like apnea or hypopneas, or inadequate oxygen while asleep. In history more than 100yrs ago (1836) Charles Dickens had given a lively description of a 'fat boy' and his frequent naps in Pickwick Papers.In 1960s, OSA was found to be associated not only with Obesity with other comorbidities. Later in 1965 the first polysomnography was used to record apnea's during sleep.[1,3]

First sleep clinic was started by William Dement at Stanford University, USA in 1970. In 1972, Christian Guilleminault was associated with William Dement and did extensive research on sleep disordered breathing.In 1970, Eliot Phillipson started investigation on dogs for respiratory control. In 1976, Colin Sullivan experimented on dogs and in 1979, he created a mask to deliver air.[2.]

Same experiments of which was done on dogs by Sullivan was carried out on patients with a plaster cast fitting their nose. There were entry and exit opening for air and fixed to the patients face with silicone adhesive, from that the tubes were attached to devices and continuous positive airway pressure given and in 1981 research paper he said that patients of 5 number with long snoring history and excessive daytime sleepiness with CPAP completely prevented upper airway collapsibility in each of 5 patients.The acceptance of treatment for Sleep Disorder with CPAP was not accepted immediately.[4]

Nineteen years later, in 2000, four paper were published which showed correlation between OSA and hypertension ,turned to be a turning point in sleep apnea

studies since it had a large database . In 2001 , there was a rise in ischemic heart disease , cardiac failure and stroke which was associated with AHI ≥ 5 per hour shown by many reports.[5]

The prevalence of sleep disorder breathing based on the Wisconsin cohort study in USA between age group of 30-60years in 2% in females and 4% in males according to Young et al.[6] and according to survey conducted on semi-urban Indian population it was found that high risk OSAS in 6.2% and obese population were at 33.5% were at high risk of OSAS.[7] The impact of sleep breathing disorder is increasing because of urbanization as well as life style modifications and it turn leads to increase rates of cardiovascular disease and obesity.

In a community based study of Chinese middle-aged office-based male workers in Hong Kong [131], 1542 men were included in the study. The estimated prevalence of SDB and obstructive sleep apnea syndrome (OSAS) (defined as SDB in the presence of EDS) at various AHI cutoff threshold values was 8.8% and 4.1% (AHI > 5), 6.3% and 3.2% (AHI > 10), and 5.3% and 3.1% (AHI > 15).

A study by Young et al [130], estimated the prevalence of SDB to be at least 6% for US adults,though treatment is available, at least 75% of cases of severe SDB remain undiagnosed.

The estimated prevalence of SDB has been observed to be 19.5% and that of Obstructive sleep apnea with hypersomnolence was 7.5% by Udwadia et.al[9]

A Swedish study [155] done in 1988, found the prevalence of Sleep Apnea syndrome to be 1.5% who were mostly males in their 5th decade. The patients mostly presented with habitual snoring and excessive daytime sleepiness

A prospective study on urban middle aged Indian population by Reddy et.al[170], OSA prevalence was estimated to be 9.3% and that of OSAS was 2.8%. A positive correlation was found between prevalence and social economic status of population.

In 2006, a study done in North India,[8] found the overall prevalence of OSA to be 13.7% (M: F - 19.7% : 7.4 %) and OSAS (M: F – 4.9% : 2.1 %) was 3.57%.

In 2017, an Indian study by Singh et.al[7] included 1512 patients and found the prevalence to be 6.2 % (M – 4.6%, F- 1.5 %). They also found that 33% of the obese population were at high risk for OSAS.

Continuous positive airway pressure in the form of BiPAP, CPAP or AutoPAP has been implemented as the effective treatment of OSA. Other treatment options includes life style modification,weight reduction,positional therapy. Upper airway surgery is advisable in patients who are non-complainant to CPAP or those who cannot tolerate it

With increasing urbanization and changes in life style modification, obstructive sleep apnea with hypersomnolence may have bigger social impact in a developing country like India. Hence our study was conducted to evaluate sleep discorded breathing patterns which was diagnosed using level 1 polysomnography study and its correlation of severity of sleep apnea syndrome with BMI

AIMS AND OBJECTIVES OF THE STUDY

AIM:

To Evaluate Sleep Disordered Breathing by Level 1 Polysomnography in a Tertiary Care Hospital.

OBJECTIVES:

- To Study various sleep disordered breathing patterns diagnosed by Level 1 Polysomnography Study.
- To Correlate severity of sleep apnea syndrome with Body Mass Index.

REVIEW OF LITERATURE

Sleep disordered breathing is an extremely common medical disorder, in India around 80% remain undiagnosed and untreated which is associated with important morbidity. The struggle still persists with simple awareness, diagnosis and management.

HISTORICAL REVIEW:

The SDB first reported in 19th Century, which was likely influenced by observations which was description of ''Obese boy'' in Charles Dickens series, " Posthumous Papers of the Pickwick Club" , where an extremely fat boy who snores loudly and sleeps excessively has been described - the classic description of Pickwickian Syndrome.[10,11]

Later in 1918, Sir William Osler made the first ever association of Pickwickian Syndrome and Obesity. [12.] In 1965 Gastautet al.[20] demonstrated, that obstructive sleep apnea causes upper airwayclosure and thereby ceases the breathing.

In 1970 Coccagna and co-workers[13] postulated the existence of a hypersomnia-hyperventilation syndrome which was based on their own case report and had findings which was daytime hypersomnolence, respiratory abnormalities during sleep and hypotonia of the nasopharyngeal muscles during sleep. Then, later in 1976 Guilleminault et al. [14] concluded that sleep apnea syndrome can be found in non obese people too.

In 1969, treatment option began to emerge where W. Kuhlo et.al [15] saw that the tracheostomy was the treatment of OSA, in which permanent tracheal cannula was

inserted and then breathing was completely normalized and sleep was without periodic arousal. Later on around 10yrs after, first reports by R John Kimoff[17] was published describing on the reversal of OSA with positive airway pressure, where he used nasal CPAP which was a relative simple, safe and inexpensive means of treatment. Then later in 1981 by Colin Sullivan et.al [18] in Sydney, Australia improved and modified CPAP devices. The designs of CPAP machine were later designed and modified and improved in later 1980s rapidly.[19] The Baxter centre was established by Dr. Peter Farrell in 1986, this centre did further research and development on CPAP devices by Dr. Farrell and Dr. Sullivan and later devised the Sullivan Nasal CPAP System (R2) in 1988.[19] In last 50 years, a lot of research has been done on understanding the pathophysiology, clinical features, work up and management of sleepapnea disorder. So, after all research and study we can say that Nasal CPAP is the front line therapy for sleep apnea disorders.

PHYSIOLOGY OF SLEEP

Sleep is a transient state of detachment from the external environment.

Sleep has been divided into:

1. Slow wave sleep
2. Rapid eye movement sleep- REM sleep, where eyes have rapid movement in-spite of person being asleep.

Most of the sleep is Slow wave sleep which is deep and person gets most of the rest in this sleep which happens in the initial one hour of the sleep after being awake. REM sleep occupies 25% of the sleep which recurs every 90 minutes and is associated with vivid dreams.

Slow Wave Sleep: This is a state of resting,where peripheral vascular tone is lost. Also known as “no dream sleep ″ but occasionally nightmares can be seen.

REM Sleep:

It is characterized by the presence of low-voltage, mixed-frequency brain waveforms, loss of muscle tone, and rapid eye movements. In a normal sleep, REM sleep lasts for about 5-30 minutes, and if a person is very sleepy, it may not be present.

Characteristics of REM Sleep:

- It is form of sleep where active dreaming occurs and body movements are present
- Difficult to arouse.
- Muscle tone is suppressed because spinal cord reflexes are abolished.
- Breathing pattern are erratic , tachycardia is seen
- Although all peripheral muscles are depressed , irregular muscle movements do occur.[54, 55]

Neuronal Centre, Neurohumoral Substances, and Mechanisms That Can Cause Sleep—A Possible Specific Role for Serotonin

There are many specific areas in the brain which when stimulated can produce natural sleep. They are the following:

1. Raphe nuclei in the lower half of pons and medulla. Few nerve endings secrete serotonin.When serotonin inhibitor is given , a person’s sleep is altered. Hence assumed that serotonin is the neurotransmitter involved in sleep

2. Stimulation of Nucleus of tractussolitarius which is located at termination of medulla and pons can also cause sleep. [175, 176, 177]

Sleep cycle is divided as:

1. NREM (Non rapid eye movement)
2. REM (Rapid eye movement) which alternate cyclically in an entire sleep episode

   A sleep episode begins with a short period of NREM stage 1 progressing through stage 2, followed by stages 3 and 4 and finally to REM. NREM and REM alternates every 90 min during sleep cycle.[56]

Stages of NREM

1. NREM 1: Duration is about 1 to 7 minutes constituting 2-5 % of entire sleep. It is characterised by wakeful relaxation state. It contains Alpha waves of frequency 8-13 cpm. Easy to arouse.
2. NREM 2: Duration is about 10 to 25 minutes accounting to 45 to 55% of entire sleep time. A person who is in this stage requires a stronger stimuli to arouse than in stage 1. Low-voltage, mixed-frequency waves are seen along with sleep spindles and K-complexes.
3. NREM 3 : Constitutes about 8 % of total sleep. High-voltage, slow-wave activity seen.
4. NREM 4 : Duration is about 20 to 40 minutes and constitutes 15% of total sleep time . Very difficult to wake up an individual in this stage and has high-voltage, slow-wave activity on the EEG

Both NREM 3 and 4 are togetherly known as slow wave sleep which accounts to one third of the sleep[55,56,57]

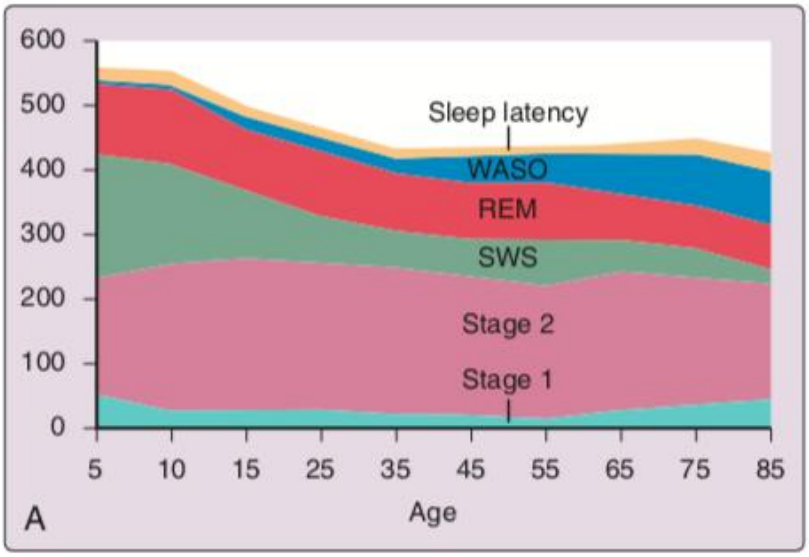


Figure 1: Sleep progression in a sleep cycle

SLEEP DISORDERED BREATHING:

Sleep disorder breathing consists of various conditions, all of which are associated with partial or complete cessation of breathing leading to functional disability and poor life quality

TYPES:

There are three main types of sleep-disorder breathing which are manifested in sleep apnoea. They are as follows:

1. Obstructive Sleep Apnea (OSA)
2. Central Sleep Apnea (CSA)
3. Complex Sleep Apnea

1. OBSTRUCTIVE SLEEP APNEA (OSA)

OSA Is defined as presence of repetitive episodes of upper airway obstruction during sleep.

Apnea-hypopnea index of equal to or greater that 5 events/hr is commonly used, with obstruction or mixed events having 50% of the total.[24]

OSA is usually classified according to AHI as:

- Mild is 5-15events/hr
- Moderate is 15-30events/hr
- Severe is >30 events/hr
- There are few suggestions that the severity should be defined by associations with adverse clinical outcome rather than the event index. But the recent changes in clinical hypopnea definitions by American Academy of Sleep Medicine have also provoked debate over parameters of severity of disease.[24,25,26]

EPIDEMIOLOGY OF OSA:

The characteristics of population factors like obesity, ethnicity depends on prevalence of OSA. There are different methods to analyse the SDB with help of hypopnea and AHI index to define OSA.

Karl et.al[52] did a review on epidemiology of sleep apnea from 2008 to 2013 and they saw the prevalence of OSA, AHI ≥5 were a mean of 22% in males and 17% in females. They even saw that the AHI and EDS occurred in 6% of males and in 4% of females. The current prevalence obtained from different studies have increased and was reported as 37% of males and in 50% of females .

Snigdha et.al[53] also did a stop-bang questionnaire study in Indian population, of total 1012 patients where they saw prevalence of 13.7%. the OSA prevalence was highest in age group of 50-59 (21.7%) and least in the age group of 18-29 (12.0%), and according to gender they saw in male was 14.8% and females was 12.9%.

PATHOGENESIS OF OBSTRUCTIVE SLEEP APNEA:

The anatomical and neurological components both are involved in the OSA. The upper airway begins from the posterior margin of nasal septum to the larynx and is devoid of bony support. It is a complicated structure the performs multiple physiological functions which includes respiration, deglutition, vocalization.

Upper airway is divided in four regions:

- Nasopharynx: between nares and hard palate
- Retropalatal oropharynx: between the hard palate and soft palate
- Retroglossal oropharynx: between soft palate and the base of the epiglottis
- Hypopharynx: from tongue base to the larynx.

The most of the airway boundaries are of soft palate and anteriorly tongue, the pharyngeal constrictor muscles, lymphoid tissue, parapharyngeal fat pad and mandibular rim laterally; and the pharyngeal constrictor muscles posteriorly. So the anatomical factors plays a major role in airway collapse during sleep.[27]

The upper airway lacks supportive framework of cartilaginous rings and therefore it is at risk for collapse due to extraluminal tissue pressure exerted by soft tissue structures and circumferential craniofacial and negative pressure associated with inspiration. The gradient between the airway lumen pressure and the pressure

exerted by contiguous tissue is known as pharyngeal transmural pressure. This in turn modulates upper airway size. The upper airway is kept unobstructed by the pressure of extraluminal tissue which is sets by pharyngeal dilator muscles.[27,28]

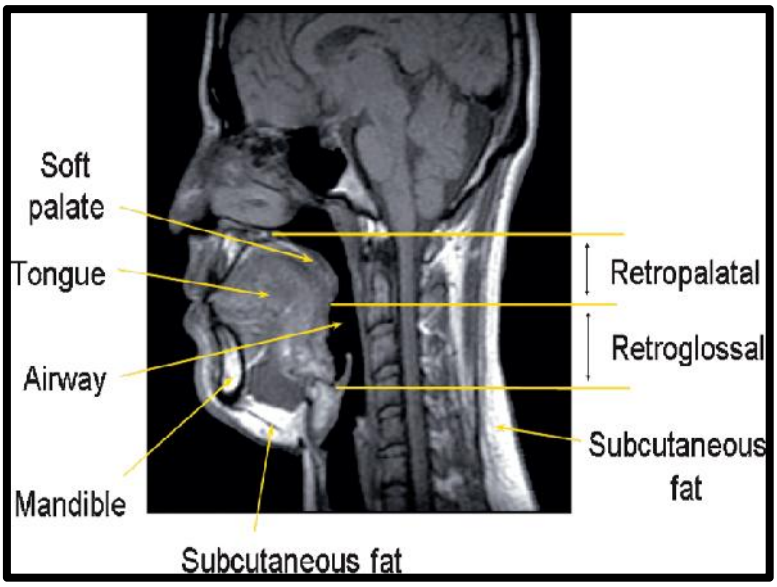


Figure 2 : Midsagittal MRI demonstrating the anatomic regions of the upper airway

The upper airway collapse occurs most commonly in retropalatal and retroglossal regions which is seen in individuals with sleep apnea leading to reduce airway intraluminal diameter and increasing airway resistance, which in turn leads to apneas and hypopneas. The calibre of upper airway doesn't entirely explains OSA risk.[29]

In recent observations in patients with OSA, they have seen that men have longer airway length, and thus they have more chances of collapsible, pharyngeal airways compared to women.[30]

There are studies where they have seen that neck circumference has a very strong correlation with OSA. A study by Hoffstein et.al [31] found that neck circumference was higher in apneic patients than in non apneic patients (41.2 +/- 3.5 cm vs 39.1 +/- 3.7 cm). It was deduced that sleep apnea patients had fatter neck than in non apneic obese patients.

Patients with OSA are more likely to have longer soft palate and longer tongue, and increased upper airway structures which contributes to narrowing of the apnoeic airway.The image studies have revealed that the fat tissue surrounding the airway is more inapneic patients when compared to normal patients. Due to increased soft tissue mass there may be increase tissue pressure which in turn result in airway collapse and decreased airway volume.[28,30,33,34]

There are examples of increased hyoid-mandibular plane distance and retroposed mandible which have higher risk of apnea. This may be due to craniofacial morphology which can influence upper airway configuration.[35]

Studies by Schwab et.al[33] and Verbraeckenet.al[36] have seen that respiratory cycle can also influence the chances of airway collapse during both expiration and inspiration. They have seen that in expiration the airway dilator muscles decreases, intraluminal pressure rises, and the airway expands maximum, at the end of the expiration upper airway dimension is reduced, whereas in inspiration the upper airway is almost constant maintaining its balance. So therefore the patients with OSA have more of narrowing during end expiration when compared to normal controls.

There are studies by Shiota et.al [37] and Redolfi et.al [38] where they have seen redistribution of extracellular fluid which occurs during supine or sleep which may lead to increase in circumference of neck and which in turn lead to reduction in upper airway area and airway collapsibility. This mechanism is associated with the degree of SDB.

Neurological influence on upper airway patency:

The neural control over the upper airways involves as complex system which has several neurotransmitters like norepinephrine, serotonin, orexin-acetylcholine and gamma-aminobutyric acid which are influenced by sleep.The genioglossus muscle is the one which is importantly studied muscle in upper airway, there are three neurological mechanisms with regard to this muscle which has vulnerability to obstructive apnoeic event.[39]

Firstly, the mechanoreceptors which activates the genioglossus muscle through the increased hypoglossal nerve discharge, which will be detected by these receptors due to negative airway pressure. This reflexes are reduced during NREM sleep, it further reduces during REM sleep which leads to risk of collapsing airway. There is furthermore reduction of airway reflexes during sleep It may be due to repetitive injury from oxidative stress or snoring which leads to neural or muscular damage, this is still controversial debate.[41,40,42]

Secondly, the respiratory control centres in the medulla controls the upper airway muscles which increase or decrease in activity with relation to respiratory drive. When the upper airway muscle activity is diminished which may lead to

collapse, that leads to obstructive apnea, such situations are seen in ventilatory control instability, where respiratory drive waxes and wanes.[43]

Thirdly, the serotonergic and noradrenergic neuros mechanism have a tonic excitatory influence on genioglossus activity. Whereas in sleep there is reduction in arousal-modulated excitatory output musculature of upper airway. So when there is upper airway collapse during sleep, arousal from sleep will restore the airway patency in response to respiratory activation. In OSA there is diminished ability to bring back the ventilation without cortical arousal when compared to non-snorers.[44,45]

Apneic Event:

The physiological disturbances is caused by the occlusion of airway. In OSA there is reduction in intrathoracic pressure which is created by breathing efforts, which in turn increases atrial natriuretic peptide release and left ventricular transmural pressure, which compromises the Left ventricular filling and increases afterload and preload. Due to apnea-related hypoxia, myocardial oxygen demand increases despite decreased oxygen delivery and decreased coronary blood flow delivery.[46,47,48]

There is tachycardia and increased blood pressure post apnea due to the surges in sympathetic nerves system which occurs due to hypoxia, apnea, hypercapnia which arouses results in increased peripheral resistance and cardiac stimulation. This response is seen in day time also in patients with OSA.The blood pressure is elevated due to intermittent hypoxemia is associated with increased reactive oxygen species production and oxidative stress, which impair endothelially mediated vasodilatation.[49,50] The endothelial damage and atherosclerosis are caused due to intermittent hypoxia.

The termination of obstructive apneas may be by awakening from sleep or transient arousal from sleep. These arousals will involve both chemical and mechanical stimuli.This mechanism will adversely affect and alters in ingestion of alcohol, chemosensitive system or use of sedatives and hypnotics, leading to prolongation of apneic events.[51]

RISK FACTORS OF SLEEP APNEA:

Risk Factors for Obstructive Sleep Apnea.

- Obesity (BMI >30kg/m^2)
- Gender- Male and Female 2-3:1
- Neck Size (Collar Size >17inches in males, >16inches in females)
- Genetic Factor/Family history
- Upper airway and Craniofacial anatomy: Macroglossia, Tosillar Hypertrophy, Enlargement of soft palate, Lateral peritonsillar narrowing, Nasal septal deviation, Narrowing of hard palate, Mallampati Airway of Class III/IV.
- Endocrine disorders like hypothyroidism, polycystic ovarian syndrome, acromegaly
- Specific genetic disorders like Down syndrome, Treacher Collins, Apert Syndrome, etc.
- Alcohol, Smoking , Sedative or Hypnotic use. [54]

OSA has been found to be associated with varied spectrum of co morbidities. It has been found to increase the risk of cardiovascular events like stroke, Coronary artery disease,pulmonary hypertension,diabetes mellitus, COPD, asthma,obesity ,GERD, hyperlipidemia, DVT, depression etc.

Pinto et.al [135]did aretrospective study of 100 patients in Sau Paulo, Brazil evaluating over a span of 3 years. The prevalence of comorbidities were hypertension (39%), obesity (34%), depression (19%), gastroesophageal reflux disease (GERD) (18%), diabetes mellitus (15%), hypercholesterolemia (10%), asthma (4%), and no comorbidities (33%). Comorbidities occurred in 56.2% patients diagnosed with mild OSA, 67.6% with moderate OSA, and 70% of patients with severe OSA.

Yaggi et.al [136] found that patients with either incident stroke or death had 2-fold increased adjusted risk for those diagnosed with OSA (mean AHI = 35) versus not diagnosed (mean AHI = 2)

In a study by Bielicki et.al[137] studied 5,353 patients and found that the most frequent comorbidity was hypertension, followed by obesity, diabetes mellitus type 2 and coronary artery disease, with a statistically higher incidence of hypertension in non-smokers (59.2 vs 64.7 %, p = 0.005).

Bajpals et.al[138] retrospectively evaluated 617 patients and found that the evidence of depression was almost same in the OSA and the non apnea groups (40.9% vs 40.3%, respectively). They observed that when patients were referred for polysomnography, irrespective of the diagnosis of OSA , higher rates of depression were observed in such individuals.

Another South Indian report by Hasan et.al[133], among the patients with OSA, 31% were having hyperlipidemia, 59% were diabetics, and 86% were hypertensive

Obesity is a significant risk factor for progression and development of OSA. Obesity is defined as a BMI≥ 30, whereas a BMI ≥ 25.0 indicates the person is overweight. However obesity has not been limited to adults, obese children have 46% of OSA when compared with children seen in a general paediatric clinic (33%). There are studies which suggest that paediatric population who are suffering from OSA are 6 times more prone to have metabolic syndrome than normal children.[139,140]

Dixon et.al[141] conducted a prospective study on 25 severely obese patients, effect of weight loss following laparoscopic adjustable gastric banding (LAGB) on the PSG changes in patients with severe OSA was studied. The mean percentage weight loss were 50.17% (range 24–80%) and 44.9 kg (range 18–103 kg), respectively with significant reduction in AHI from 61.6734 to 13.4713, improved sleep architecture with increased REM and stage III and IV sleep, daytime sleepiness, as measured by Epworth Sleepiness Scale, of 13±7.0 to 3.8±73.0, and lesser patients requiring nasal continuous positive airways pressure (CPAP). Thus, proved that Obesity is a major determinant of OSA and therby weight reduction does tremendable improvement in improvement of OSA and reduction of CPAP need.

Young et.al[142] estimated that about 17% of adults have mild or worse SDB. BMI was higher in subjects with AHI of ≥5 compared with AHI of <5. They estimated that most (58%) of more severe SDB is due to overweight and obesity among US adults and hence concluded that obesity is responsible for incidence and progression of SDB

Namysłowski G et.al[143] studied 106 patients and observed statistically significant correlation between BMI and RDI in Obese class 1 and 2. Mean BMI of class 1 was 31.7 ± 1.47 kg/m2 and mean RDI was 30 and that of class 2 was 38.8 ±3.2 and 49 respectively. It was also demonstrated mean RDI increased with increasing BMI

A study done on rural population by Choudharyet.al[134], 200 community residents were evaluated using the Berlin questionnaire (BQ). Among them, 25% had a high likelihood of OSA. The associated risk factors were age >35 years,BMI≥25kg/m2 , alcoholism , and hypertension. They concluded the high prevalence of OSA even in rural community with Obesity (BMI ≥ 25 kg/m2), alcoholism, and hypertension were independent risk factors associated with OSA.

In a Chinese study by Zhu et.al[145] included 138 cases of non-OSA and 581 cases of OSA, among which smoking rate of OSA group was significantly higher than that of non-OSA group (41.5% vs. 27.5%,P<0.01).The logistic regression analysis showed that compared to non-smokers, the odd ratios for OSA in moderate smokers were 1.72 (95%CI 1.08-2.7) and in severe smokers were 2.68 （95%CI 1.61-4.46） concluding the severity of smoking increased with OSA severity

Lui et.al [146], in Hongkong , enrolled 114 men, and found that The PAT (Peripheral arterial tonometry) ratio decreased with increasing pack-year group (p = 0.018).

CLINICAL PRESENTATION AND SCREENING FOR OSA:

The diagnosis of OSA is usually done bydetailed history and physical examination with screening of patients. Patients have both daytime and night-time symptoms. The history is best given by the bed partners who will give detailed history in suspected cases of OSA.

OSA clinical presentations are witnessed apneas, loud, habitual snoring, nocturnal awakening, gasping of choking episodes during sleep, nocturnal sweating, nocturia , excessive daytime sleepiness, unrefreshing sleep, morning headaches, irritability, memory loss, personality change, automobile or work related accidents, decreased libido. Individuals with OSA may also report of choking, nocturnal diaphoresis, gasping and restlessness related to airway obstruction.[55]

Excessive daytime prevalence is major health problem with high prevalence as much as 18 %. It is associated with wide spectrum of cardiac,pulmonary, neurologic and psychiatric conditions. [147]

Chen et.al[148] studied 1035 patients and reported that higher ESS scores was found in the moderate OSAHS patients (10.4±4.6, $p < 0.01$ vs. primary snoring and $p < 0.05$ vs. mild OSAHS), and was highest in the severe OSAHS patients (13.0±5.0, $p < 0.01$ vs. moderate OSAHS)

A report byZeki et.al[149] concluded that patients who had EDS had short REM latency (99 ± 65 vs 125 ± 81) when compared with patients without EDS .awake SaO2, AHI, minimum SaO2, oxygen desaturation index and arousal index between EDS group were statistically significant

SCREENING OF OSA

Patients are screened by a questionnaire for assessing the degree of subjective sleepiness in sleep apnea patients called as The Epworth sleepiness scale . It is a self administered questionnaire with 8 questions. Patients are usually asked to rate, on a 4-point scale (0-3), their usual chances of dozing off or falling asleep while they are doing various daily routine activities. The sum of the scores of 8 questions ranges from 0-24

The Interpretation is as follows:

0-5 : Lower Normal Daytime Sleepiness

6-10 : Higher Normal Daytime Sleepiness

11-12 : Mild Excessive Daytime Sleepiness

13-15 : Moderate Excessive Daytime Sleepiness

16-24 : Severe Excessive Daytime Sleepiness

It is usually seen elevated in sleep apnea patients where scores are 0 to 24 and if the score are more then 10 its considered abnormal, and can be used to accessing sleepiness in clinical settings.[56,57]

Inexpensive tools have been developed to assess the likelihood of apnea. The ability of a number of standardized instruments such as Multivariable Apnea Prediction(MAP) Index and the Berlin questionnaire, to recognise the probability for sleep apnea have been evaluated.[59]

The sensitivity and specificity of questionnaires vary depending on the population studied, the population cut offs chosen and the severity of OSA to be identified.[60]

In order to identify severe OSA, the surgical preoperative questionnaire called STOP-BANG is shown to have a good predictive value. The clinical situation in which sleep apnea evaluation to be considered are obesity, myocardial infarction, systemic hypertension, type 2 DM, cerebrovascular accidents, pulmonary hypertension, polycystic ovarian syndrome, atrial fibrillation[123].

In the past, overnight pulse oximetry was been used to screen OSA. The overnight oximetrydo not apneas or hypopneas or arousals in the absence of oxygen desaturation. [61]

PHYSICAL EXAMINATION:

The head and neck should be inspected carefully in patients who are suspecting OSAS. The presence of BMI >30kg/m^2 determines the presence of obesity. The neck circumference more than 43.2cm in males and 40.6cm in females suggests an increased risk of OSA.[58]

DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA:

The OSA risk stratification can be accessed by Epworth Sleepiness Scale, MAP Models which will lead to the diagnosis and determine disease severity.[62]

POLYSOMNOGRAPHY

Polysomnography (PSG) it is the gold standard sleep study in which it records many parameters when patient is a sleep. Electrooculogram to monitor eye movements, Electromyogram for muscle activity and Electroencephalogram to indicate sleep state with chest and waist bands to measure respiratory efforts, arterial oxygen saturation, oronasal thermistor and nasal pressure sensor for respiratory

airflow. All these provide the sleep staging and it also indicates apneas, hypopneas and arousals. AHI remains the primary determinant of OSA for diagnosis and severity. But the AHI in OSA has limitations, like it does not capture other significant aspects of disorder like oxyhemoglobin desaturation, nocturnal hypoxemia, associated sleep disruption, hypoventilation. So it usually does not correlate with sleep quality measures and clinical outcome.[64]

INDICATIONS

Polysomnography is used to diagnose various sleep disordered breathing like:

1. Obstructive sleep apnea
2. Periodic limb movement disorder,
3. Central sleep apnea,
4. REM behaviourial disorder
5. Parasomnias,
6. Hypoventilation syndrome
7. Cheynes-stroke breathing
8. Sleep related seizure disorder

   This study is indicated for confirming OSA if it is suspected clinically and when of the following are present:

   - EDS
   - Disruptive snoring
   - Epworth sleepiness scale >11
   - Engagement in safety critical occupation
   - Any witnessed apneic , choking or respiratory events during sleep

PROCEDURE

The electrodes in Polysomnography transfer biopotential from patient to circuit. Recorded variables usually include electroencephalogram (EEG), electromyogram (EMG) of the submentalis muscle, electrooculogram (EOG), a measure of airflow (usually sensors by the nose and mouth), respiratory efforts (chest wall and abdominal movement, EMG of parasternal muscles, or changes in esophageal pressure), oxygen saturation (pulse oximetry), pulse rate, electrocardiogram (ECG), body position, EMG of legs (anterior tibialis muscle, for leg movements), and snoring (usually by microphone). Various laboratories perform video recording of the entire study to observe parasomnias and it would also serve the purpose of medico legal purposes.

PARAMETERS TO BE MONITORED DURING OVERNIGHT POLYSOMNOGRAPHY

1. Electro encephalogram (EEG)

It is the recordings of the brain measured along the scalp. The EEG measures the voltage fluctuations which results from ionic current flows within the neurons of the brain. The placing of electrodes aredecided by the International 10–20 System of Electrode Placement. These electrodes gives the information of sleep activity through various sleep stages (NREM 1,2, 3, 4 which are referred as N1, N2, N3. N4, REM sleep as Stage R and wakefulness)

2. Electro oculogram (EOG)

To monitor eye movements using corneo-retinal potential difference inside the eye as electro oculography is used. An electrode is applied at the outer canthus of the right eye (ROC) and is offset 1cm above the horizontal. Another electrode is applied

to the outer canthus of the left eye (LOC) and is offset by 1cm below the horizontal. The infraorbital and supra- orbital electrodes are useful in the MSLT. These electrodes are applied on the surface of the skin using glue tape.

3. Electromyography (EMG)

It summates the activities of the the individual motor end plates. Atleast 3 EMG electrodes are applied from mentalis to sub mental In case of bruxism, extra electrodes can be applied to masseter muscle to detect the outburst of EMG activity . To detect PLM in sleep , additional electrodes are placed over anterior tibialis and extensor digitorum.

4. Electrocardiography (ECG)

Among the 10 ECG electrodes, only 2-3 are used. They are positioned under the collar bone on each side and on the left side at the level of 7^{th} rib. These measure electrical activity of heart and records P wave, QRS complex and T waves.

5. Respiratory movements

. It is necessary to record at least 3 parameters: Nasal / oral airflow, thoracic effort, and abdominal effort. A thermal sensor (thermistor, thermocouple or polyvinylidenechloride) to detect apnea. Pressure transducers easily detect the flow of air and can be used for both Hypo-apnea and RERA detection. When used together, reach the maximum level of efficacy.

6. Blood Oxygenation (Oxygen Saturation - SpO2)

Pulse oximeter is used for assessment of Oxygen saturation. It is mostly placed over ear lobe, fingertip or toe. Two different frequencies of light through a pulsatile vascular bed transmitted by Pulse oximeter to measure arterial oxygen saturation. It gives important information regarding the severity of sleep disordered breathing.

7. Capnography

Patient's carbon dioxide levels is assessed using capnography.End tidal capnography is measured using nasal /oral cannula or a tight fitting mask. End tidal measurement depicts the amount of CO_2 in the lungs and in the blood at the end of expiration. The normal range is 35-45 mmHg.

8. Body position

Apneas can be intensified by body position during sleep. Body position needs to be monitored throughout the sleep for disgnosis of various sleep disordered breathing [86,87, 88]

Polysomnography and Apnea Hypopnea Index are calculated as the number of respiratory events per hour of sleep. The PSG are studied in various types like Type I study and Type II and III are differentiated by number of variables measured. Still there are chances of underdiagnosed hypopneas in the absence of EEG. Therefore the portable monitors may have reduced sensitivity to detect OSA in patients with mild disease.[65]

Diagnostic Testing Options for Sleep Disordered Breathing:

Diagnostic Testing Options for Sleep-Disordered Breathing

	Type	Parameters Measured
	I	EEG, EOG, EMG, ECG, Airflow, Respiratory effort, O_2 Saturation, Usually Video (all conducted in a sleep laboratory with a sleep professional present)
Out of Centre Testing	**II**	Seven Minimum channels including EEG, EOG, chin EMG, ECG/HR, Airflow, Respiratory Efforts, and O_2 Saturation
	III	Minimum of four channels including ECG/HR, O_2 saturation and at least two channels of respiratory movement or respiratory movement and airflow.
	IV	Airflow and/or O_2 Saturation.[66]

Based on AHI values, Obstructive sleep apnea can be categorised as:

- Normal: AHI<5
- Mild sleep apnea: 5-15
- Moderate sleep apnea: 15-30
- Severe sleep apnea: >30 [21]

The 2007 update of portable sleep testing was recommend that out-of-centre sleep testing with a portable monitor be performed only in patients who have high possibility of having moderate-to-severe OSA without significant comorbidities including CHF, Neuromuscular disease, Severe COPD and other sleep disorders.[67]

INTERPRETATION OF POLYSOMNOGRAPHY

When the sleep study is complete, the sleep lab technician analyses the data in 30 second epochs.

The sleep report has the following details

- Sleep Onset: Calculated from the time lights are turned off. It is normally less than 20 minutes.
- Sleep Efficiency: The number of minutes of sleep divided by total number of minutes in bed. Normally it is more than 85%.
- Sleep Staging: This is using 3 out of 7 channels ie EEG, EOG, chin EMG. The each 30 second Epoch is scored as “awake” or one of the 4 sleep stages, NREM 1,2, 3, 4 and REM sleep. Epoch is a short period of arbitrarily defined length, usually 20-60 seconds. The sleep stage from each epoch is established by polysomnography recording.
- Breathing abnormalities like Apnea, Hypoapnea are detected.
- Sudden interruptions in the brain activity are described as “arousals”. Any extraneous sounds, body movements, breathing irregularities can cause arousals. These arousals, when in increased number can be an indication of fragmented sleep which is responsible for daytime fatigue or sleepiness in patients.
- Cardiac abnormalities are detected using ECG
- Leg and body movements
- Oxygen saturation during sleep.

After the scoring is done, the data is interpreted by sleep medicine physician along with the history, anthropometric measurements and other relevant information regarding the patient. The final report is written with the diagnosis and titration of the pressures required for CPAP therapy

Overall, the PSA in laboratory is best proven investigation for diagnosis of sleep apnea. The access of diagnostic testing has technologically advanced with portable monitors and acceptance of out-of-centre sleep testing by third party payers.

The sequelae of OSA can be broadly categorized by the neurocognitive, cardiovascular and metabolic effects. OSA should be considered as systemic disorder with effects on multiple organ systems. [21, 68,178]

Treatment of OSA:

As per latest AASM guidelines, Positive airway pressure is the mainstay treatment. Moderate to severe OSA (AHI>15) with PAP devices, reduces cardiovascular risk, improves neurological behaviour and enhances quality of life [89,90,91,92]

Treatment options include

1. Non Pharmacological treatment: Life style modification changes like Weight reduction , avoidance of alcohol, sedatives and hypnotics
2. Specific treatment : PAP therapy, CPAP, Bilevel system , AutoPAP, Mandibular reposition devices, , Positional therapy , Pharyngeal muscle stimulation
3. Surgical treatment : Uvulopalatopharyngoplasty(UPPP), Nasal surgery, Tonsillectomy and/or Adenoidectomy, Laser assisted uvuloplasty, Radiofrequency volumetric tissue reduction, Lingular tonsillectomy, Sliding genioplasty,

Maxillomandibular advancement osteotomy, Genioglossus and hyoid advancement, Tracheostomy.

4.

General measures:

Alcohol, sedative hypnotics and opiods should be avoided by patients with OSA. They are proved to be reduce the upper airway muscle tone. Alcohol consumption increases the duration apneic episodes and reduces arterialoxyhaemoglobin saturation.

One has to adhere to good sleep hygiene practices to maintain adequate amount of sleep. Sleep fragmentation can be avoided by avoiding the ingestion of stimulents (eg- caffeine), alcohol, sedatives, opiods[93,94,95]

Weight reduction:

Obesity is an important determinant for development of OSA. It causes upper airway collapse due to increased extraluminal pressures associated with excess soft tissue/fat and the encroachment of the tongue and soft palate. Abdominal fat or what has been termed as Truncal Obesity has pivotal role in reducing the compliance of respiratory system and chest wall. Weight reduction via dietary modification helps in reducing AHI.

Over the last decade, Bariatric surgeries have been implicated in extremely obese indiviuduals and therefore has been in effective in treatment of OSA. Observational studies of major weight loss following bariatric surgery, data suggests that there has been improvement in symptoms in about 60% to 80% of patients. Within 2 years of Laparoscopic adjustable gastric banding (LAGB) approximately

20% of initial body weight was achieved but significant reduction in SDB was not seen .[93,96,97,98]

Specific Medical Therapy

1. Intraoral devices

Patients who have milder OSA, oral appliances (OAs) are a very good substitute. By pushing the lower jaw forwards, it increases the upper airway volume and reduce collapsibility of pharynx. Few intra oral devices are Karwetzky activator (KZY), mandibular advancement splint (MAS), tongue retaining device (TRD) and Herbst appliance (HST).Currently , intra oral devices are can be used for patients who have snoring as their predominant symptom , and who are not fit for CPAP therapy or surgery. Generally given in mild to moderate OSA. [99,100,101]

2. Nasal expiratory pressure airway therapy :

Nasal EPAP device comprises of reusable one-way valves, placed in front of nostrils using an glue tape. It provides high expiratory resistance in the setting of very low inspiratory resistance by utilizing patients own breathe. Due to tracheal pull, upper airway dilates and thereby lungs expand during expiration. Nasal EPAP shows to reduce the AHI and improve symptoms in mild to moderate OSA.However, the AHI reduction was less when compared to CPAP. Krygeret al.[105] did a 12-month followup which showed that after using this device, AHI was reduced,alongn with snoring, and subjective daytime sleepiness. It is as efficacious as intra oral devices or less invasive surgical procedures which are inferior to CPAP. Many studies have shown contradicting results. This is because, it does not act directly on on the upper respiratory tract like PAP devices. Nasal EPAP could used by those who cannot

tolerate PAP devices or without electricity or during travelling, and for those who have mild or position dependent OSA without other co morbidities.

Figure 3 : Nasal EPAP

3. Position therapy :

Sleeping in supine position makes it more prone for airway obstruction due to falling back of tongue. Positional OSA typically is defined as “that associated with an overall AHI less than 5, with a supine AHI that is at least twice the nonsupine AHI”. In 1984, Cartwright[107],defined positional-dependent sleep apnea as SDB in which the AHI while asleep in the supine position was at least twice as high as in the lateral position. In patients with position dependent sleep apnea , symptoms seems to be relieved by givinglateral decubitus position.. Several studies have suggested that positional therapy is equivalent to CPAP in the treatment of positional dependent sleep apnea. When combined with other therapies has been found to reduce AHI. It is a relatively simple and inexpensive technique [106,108,109,110]

4. Pharyngeal muscle stimulation

Genioglossus muscle is the largest upper airway dilator muscle. When electricallystimulated causes protrusion of tongue and contraction of anterior pharyngeal wall, thus serves as potential means of treating OSA. Base of tongue, when contracted provides support and stabilization of posterior pharynx through palatal and oropharyngeal constrictors and thereby maintains patent airway. Early studies using sub mental or other intramuscular stimulating electrodes have appeared to increase theupper airway width , pharyngeal collapsibility and maximum inspiratory flow , also reduction in AHI as witnessed by polysomnography. However, major limitation remains inducing arousals due to sensory stimulation. Recently, implantable upper airway stimulation device has been formulated to increase the hypoglossal nerve activity during sleep. Kezirian and collegues[112]studiedsafety, efficacy and feasibililty, which showed significant improvement in AHI severity and functional outcomes of sleep. A randomized open label ongoing trial [113] may hopefully provide more information regarding these devices for treatment of OSA [105,111,112,113]

5. Positive airway pressure therapy

Collin Sullivianet al.[16] in 1981 was the first person to use nasal CPAP to treat OSA. Currently, CPAP remains to be mainstay treatment. CPAP is delivered via nasal mask at a fixed pressure which remains same throughout the respiratory cycle. It maintains the patent upper airway in a controlled manner. It increases airway caliber in the retro palatal and retro glossal areas and hence increase the lateral dimension of the airway and thins the lateral pharyngeal wall. It is to be noted that, CPAP is the treatment and not cure for the disease [93,114,115]

Indications for Continous positive airway pressure in OSA

1. Moderate to severe obstructive sleep apnea (≥15 events per hour of sleep) with or without associated symptoms or comorbid diseases
2. Mild obstructive sleep apnea (≥5 to ≤14 events per hour of sleep) with symptoms or associated comorbid diseases:

• Symptoms: Daytime hypersomnolence, impaired cognition, mood disorders, or insomnia

• Comorbid diseases: Hypertension, Coronary artery disease, or history of stroke"

3. AHI <5 : To reduce the primary snoring. To consider weight reduction and behavioural intervention.[93]

Effects of CPAP

- **On Day time sleepiness**

It reduces the number of apneic episodes during sleep and also improves daytime Sleepiness in OSA patients. In a huge meta-analysis of RCT , CPAP therapy was proved to be statistically significant when it was compared with placebo or conservative management. Another study[93] showed that about 20% to 30% of CPAP-compliant patients who used it for almost 7 hours per night , yet complained of subjective sleepiness (Epworth Sleepiness Scale score of >10) after 3 months of usage. The mechanism could be due to oxidative injury as the result of long term intermittent hypoxemia on sleep wake cycle areas of brain.

- **On NeuroBehavorial function**

Most data demonstrate inconsistent improvement in neurobehavorial performance. There is no confirmed evidence that suggest CPAP use in improving

mood and quality of life. This may be due to the fact that OSA affects less severely on neurocognitive and life quality. Further studies needs to be done to in this direction.

- **Cardiovascular disease**

Untreated OSA is one of the major risk factors for hypertension and other cardiovascular diseases, but research on usefulness of CPAP on cardiovascular disease is still doubtful. [115]

Complications / Side effects of CPAP

1. Nocturnal arousals
2. Rhinitis, nasal irritation, and dryness
3. Aerophagia
4. Dry mouth due to mask leaks
5. Facial rash or irritation
6. Difficulty with exhalation
7. Claustrophobia
8. CPAP can be delivered via various interfaces. These mainly include nasal masks, nasal inserts or full face masks (covering nose and mouth) [93]

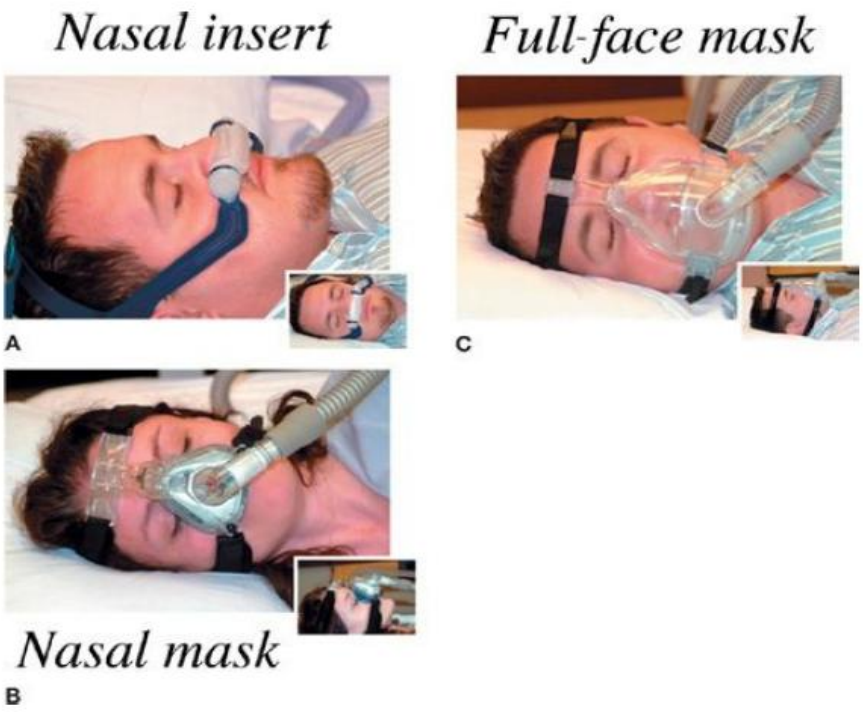


Figure4 :Types of CPAP

Other newer modalities of positive airway ventilation

It is very troublesome for some patients to tolerate high positive pressure, particularly to breathe against high jet of air. It becomes inevitable for few patients, in order to control the disease and use high pressure therapy. Therefore, different manner of pressure therapy have been used to treat OSA.[114]

Bilevel PAP

It two different levels of pressure is delivered, which is higher during inspiration and lower during expiration. The pressures tend to be much lesser than CPAP and also better accepted than CPAP. It is costlier than CPAP and very useful in patients with associated hypoventilation or chronic obstructive pulmonary disease [93, 115,114,116].

AutoPAP

It is a complex device which is a better substitute for conventional CPAP. The latter delivers single fixed pressure through the entire respiratory cycle, while the former adjusts pressure accordingly in order to maintain upper airway patency as per the changes in airflow resistance. According to Smith et al, 2009,[117] compliance with AutoPAP was better than fixed CPAP [93,115,114]Hirshkowitz and colleagues [118] devised criterion based on AASM evidence based standards of practice recommendations

CRITERIA FOR A FULL NIGHT EVALUATION

1. Any patient with AI > 20 events per hour of sleep

2. Any patient with AHI > 30 events per hour of sleep

3. A sleepy patient with an AHI > 10 events per hour of sleep

4.A sleepy patient with RAI (Respiratory arousal index) > 10 events per hour of sleep

5. On an individual basis, a sleepy patient without other obvious cause and an AHI 5 but < 10 may be considered for CPAP as one of several options, including weight loss, dental appliance, and positional therapy.

CRITERIA FOR SPLIT-NIGHT DIAGNOSTIC-TITRATION EVALUATION

1. Any patient with AHI > 40 events per hour of sleep during a 2- or more-hour baseline portion of the sleep evaluation

2. Any patient with AHI ranging from 20 to 40 events per hour during the first 2 hours of sleep, as warranted by clinical judgment (e.g., cases where the patient is sleepy or has chronic heart disease, lung disease, neuromuscular disease, or hypertension).

A patient survey by Eagleman et.al[150] was done in 204 patients average CPAP use was 5.2 ± 2 hours/ night. Mean AHI 47 ±38 per hour, average minimum oxygen saturation of 74±18%, and mean duration of CPAP treatment of 632 days. ESS score and road traffic accidents were effectively reduced after CPAP therapy ($p < 0.0001$). Most of the patients reported significant improvement in symptoms

Brill et.al[151] studied 564 participants of stroke or Transient Ischemic Attacks with SDB. Mean CPAP use was 4.53 hours per night. The reduction in AHI to < 10/hour was reported in 3 studies. Also, improvements were seen in neuro cognitive functions.

Battan et.al[152] recruited 73 patients (Moderate OSA : 24 , Severe OSA : 23) and observed that ESS score in both subgroups improved to 11.63±3.79, $p=0.022$ and 14.13 ±3.74, $p < 0.001$ respectively after one month of CPAP with Average use of 6-7 hours / day.

6. Surgical therapy

Upper airway surgery is the treatment choice for certain group of patients who have failed PAP therapy or who could not tolerate it. Nevertheless, it's a well-established fact that its less effective than PAP devices. It improves the upper airway obstruction in Nose, oropharynx and nasopharynx. These surgeries have been divided as Phase 1 surgeries which include nasal, palate reduction, and tongue advancement or reduction surgeries and Phase 2 is maxillomandibular advancement [MMA] . Other surgical treatment involve distraction osteogenesis of the maxilla and mandible, and finally, tracheotomy. Commonly, these surgeries are performed sequentially, but at times, Phase 2 is performed first, or less commonly combine both phase 1 and 2 surgeries. The success of the surgeries depends on the patient selection , surgeon

skills and also the type of procedure performed .Phase I surgeries have been successful in about 50–60% of the cases and has appreciably improved OSA by >50% in selected patients, whereas phase II surgeries have been almost as successful as 90%. These surgeries cannot be a replacement for CPAP therapy, rather just provides an alternative solution who do not tolerate CPAP or are unfit for it. [119]

CENTRAL SLEEP APNEA SYNDROMES: (CSA)

Central Sleep apnea is where there isrepetitive episodes of breathing cessation which occurs in absence of respiratory effort, is characterized by an altered ventilatory motor output.CSA is less common then OSA, there is estimated prevalence in general population <1%. CSA is considered as a physiological process in normal individuals in response to an arousal in children and elderly as an indication of breathing instability in number of medical conditions like Cheyne stroke respiration in Congestive Heart Failure and High Attitude or in some neurological diseases like stroke, myasthenia gravis, Shy-Drager syndrome, brainstem Infarction, encephalitis.

Central Sleep Apnea is divided as Hypercapnia and Hypocapnia types. In hypocapnia CSA syndromes are associated with increased chemoresponsiveness of the ventilator control system and most commonly with heart failure and altitude are commonly seen in individuals.

High altitude periodic breathing with hypocapnic central apnea occurs at high altitude with many healthy individuals. At that altitude the peripheral chemoreceptors in the carotid body which sense hypoxia, it increases ventilatory drive. theapnea threshold is reached as there is fall in P_{CO2}, there is delay between changes in P_{O2} and

P_{CO2} levels, the cycle time for periodic breathing at altitude tents to be 12-34seconds which is short.[69,70]

Poor Sleep quality is frequently seen as a complain at attitude which is likely due to hypoxemia and periodic breathing with frequent arousals. It can be treated with using acetazolamide which increases the magnitude of the change in P_{CO2} which requires to produce central sleep apneas.[71,72]

The Cheyne-stokes respiration (CSR) is mostly seen in people with systolic heart failure and during wakefulness, it has higher mortality in this population. Its seen in population with encephalopathies, stroke and other conditions.[73,74]

CSR clinical presentation of patients vary. The patients with CSR may have sleep fragmentation and day time sleepiness.[75]

The CSR can be diagnosed using PSG where we can see repetitive apneas in the absence of thoracic-abdominal excursion. Central SDB must compromise >50% of total event to make diagnosis of CSA. CSA can be treated in several ways like aggressive pharmacological management. CPAP has seen to not have much use for the patients with heart failure. [76]

IDIOPATHIC CENTRAL SLEEP APNEA:

Analogous to CSR which is described in individuals with normal cardiac function. They usually present with snoring, insomnia, witnessed apnea and excessive sleepiness. in such individuals during sleep and wakefulness there is increased ventilatory response to CO_2. There is no as such proven treatment for such patients

but the CPAP has proven effective in some patients and stimulants like acetazolamide and use of benzodiazepines and hypnotics improves sleep.[77]

COMPLEX SLEEP APNEA:

It is termed in recent years as complex sleep apnea during titration of CPAP. The total prevalence of complex sleep apnea is between 5-15% in patients undergoing CPAP titration. It is more likely to be seen in for patients who have old cardiac disease compared to individuals without complex sleep apnea. It is seen in male patients and seen more commonly in obese once.

There are studies by Morgenthaler et.al[78] , Javaheri et.al [79], Cassel et.al [80] where it has been observed that where complex sleep apnea was diagnosed 20-25% of patients who returned for PSG after using CPAP for 1 to 3months had persistent central apneas>5hr.

The results of several studies with the use of PAP devices for treatment of sleep apnea suggests reducing AHI. However with the available evidences the complex apnea resolves in majority of patient, it seems reasonable to initiate a trial of CPAP therapy and to reserve ASV for few individuals who exhibit persistent apnea. [81]

COPD OSA (OVERLAPSYNDROME)

COPD and OSA are two highly prevailing pulmonary diseases whose association has been studied based on similar characteristics. COPD exists in around one tenth of the population, similar figures are seen in OSA. Thus based the previous data, this Overlap syndrome should be present in around 1% of the population.

Direct effects of sleep on COPD

Poor sleep quality is seen COPD with reduced REM and slow wave sleep whichcan be responsible for day time fatigue and reduced survival rates. It has been suggested that lung hyperinflation is linked with poor sleep quality in COPD patients. [120]

Epidemiology

OSA can predispose COPD and vice versa which has been been explored in last two decades but comparison of epidemiology has been demanding owing to different of sleep study, study population and varied definition of sleep apnea and sleep disordered breathing.[121]

Bednarek et al [122] found severe nocturnal desaturation during sleep in overlap patients. According to Marin et.al [121] patients with the overlap syndrome have higher chances of COPDexacerbations. Both diseases have wide range of collective functional consequences like hypoxia and inflammation and thus the probability of being associated with comorbidities is also high. Nocturnal desaturation, which may be seen more evident in overlap syndrome is responsible for development of pulmonary hypertension.

Clinical Assessment

Morning headaches,cyanosis, and peripheral oedema are commonly found symptoms.

Screening

Questionnaires like Stop BANG and NOSAS can be used. The gold standard would be polysomnography.

Management

Inhaled long acting beta agonist and anticholinergics have found be to benefitted in COPD patients with nocturnal hypoxemia. Theophylline has shown to reduce AHI in certain overlap patients but side effects limits the use of it. The INOX trial is yet to give its report on this.In COPD-OSA overlap, non-invasive pressure support is a proved treatment option. In patients with predominant OSA, standard continuous positive airway pressure therapy is the preferred [121,126,127,128,129]

Patients having COPD as the dominant component, non-invasive ventilation (NIV) in the form of bi-level positive airway pressure (BIPAP) is a better option. [129]Marin et al [121]showed that when patients with COPD-OSA overlap were given long-term CPAP, the survival rate matched to patients with COPD only, but when overlap patients did not use CPAP, it was associated with hospital admission for an exacerbation with greater mortality risk. Thus the overlap between COPD and OSA is potent area for future research since many questions still remains unanswered.

MATERIAL AND METHODS

SOURCE OF DATA:

All the patients undergoing Level I polysomnography study at KLE's Dr. Prabhakar Kore Hospital, Belgaum over a period of January 2018 to December 2018.

Methodology:

Patients whose symptoms are suggestive of sleep disordered breathing attending the outpatient clinic of Dr KLE's Prabhakar Kore Charitable Hospital and MRC will be evaluated by

1. Detailed history taking

2. Clinical examination

3. Level I polysomnography study

INCLUSION CRITERIA

1. Patients with EPWORTH sleepiness scale between 8 to 24.

2. Representing increasing levels of Excessive day time sleepiness along with nocturnal awakening, choking episodes, day time tiredness, weight gain, morning headaches, irritability, memory loss, personality change, Automobile or work related accidents, Decreased libido etc.

EXCLUSION CRITERIA

1. Patients who already on CPAP therapy
2. Patients on LTOT
3. Chronic debilitated patients
4. Uncooperative patients.

DIAGNOSTIC TEST:

Level I polysomnography:

Every patient underwent overnight polysomnography. Informed consent was taken and subjects were explained about the procedure by a sleep technician. A bystander was allowed to stay with the patient. During the procedure, the sleep activity was recorded throughout the night. The PSG recording and scoring of sleep stages and respiratory events were done by Sleep scoring technologist as per AASM guidelines. [21]

Polysomnography study takes place in a quiet, dark, temperature controlled room with constant monitoring. The various variables recorded during the study will include ECG, Central and occipital EEG, EOG, Submentalis EMG, Nasal and oral air flow, Arterial Oxygen saturation , breathing pattern by Thermister, Cannula, Thoracic wall movements, abdominal movements, Anterior Tibialis EMG, snoring and body position. Sleep study will be conducted by sleep technician from 10pm to to 6am next day. Sleep scoring is done by sleep technician.

The prerequisites for sleep study include

• Patient to be in comfortable night wear

• Patient is advised to avoid taking any sleep tranquilisers or sedatives

• Patient is instructed to remove all ornaments and avoid applying oil and lotions

• Patient to asked to avoid drinking coffee or tea 3 hours

PARAMETERS INCLUDED

1. Sleep Onset : Usually used for first 3 consecutive epoch of stage 1 or first epoch of any stage of sleep.
2. TST (mins) : Total Sleep Time. The amount of time spent sleeping in minutes.
3. Latency to sleep onset : Time from lights out to the first of 3 continuous epochs of stage 1 or any other stage of sleep in minutes.
4. Sleep Efficiency: The amount of time spent sleeping divided by the total time in bed
5. Wake After Sleep Onset. The amount of time spent awake after sleep onset in minutes
6. Sleep stages : S1, S2, S3, S4 and REM sleep

RDI can also be used in grading OSA severity. It includes apneas, hypoapneas along with other respiratory disturbances such as snoring, arousals, desaturation index. RDI may be higher than AHI.

SCORING OF SLEEP STUDY

Apnea : An event lasting ≥ 10 sec characterized by ≥ 90% reduction from pre-event baseline in oronasal thermistor airflow. An apnea is scored as:

1. Obstructive : if there is continued or increasing respiratory effort throughout the event Central, if effort is absent throughout the entire event

2. Mixed : if effort is initially absent, then resumes in the latter part of the event

Hypoapnea: An event lasting ≥ 10 sec characterized by a ≥ 30% reduction from pre-event baseline in peak nasal pressure inspiratory airflow that is associated with:

Either ≥ 3% reduction in arterial oxygen saturation (SO2) pre-event baseline or a microarousalOR≥ 4% reduction in arterial SO2 from pre-event baseline value

Respiratory effort–related arousal (RERA): A sequence of breaths lasting ≥ 10 sec that does not meet criteria for apnea or hypopnea, which is characterized by increasing respiratory effort or inspiratory flattening of the nasal pressure flow signal leading to arousal.

Hypoventilation: An increase in PCO2 to > 55 mm Hg for ≥ 10 min or PCO2 increase ≥ 10 mm Hg above awake supine values to >50 mm Hg for ≥ 10 min.

Apnea-hypopnea index (AHI): Number of apneas and hypopneas during sleep divided by total sleep time.

The severity of Obstructive Sleep Apnea is assessed by Apnea Hypoapnea Index.

It is calculated by dividing the number of apneic and hypoapneic events by the number of hours of sleep multiplied by 60 .Generally expressed as events per hour. Based on the AHI values , OSA can be categorized as:

- Normal: AHI<5 per hour
- Mild sleep apnea: 5-15 per hour

- Moderate sleep apnea: 15-30 per hour
- Severe sleep apnea: >30 per hour

  Adding arousal to the hypoapnea definition increases the percentage diagnosed with OSA.

RULES FOR SCORING

- A 30 second epoch scoring with each assigned to each epoch.
- If more than sleep stages occurs in 1 epoch, stage the one with majority of the epoch.
- N1 indicated sleep onset, and is scored when alpha are replaced by and more than 50% of epoch has LAMF (low amplitude mixed frequency) activity.
- N2 scoring is done if both or one of the following occur in either first half of that epoch or last half of previous epoch

  1.One or more K complexes without arousals

  2.One or more trains of sleep spindles.
- Major body movement is scored when muscle or movement artefact obscure more than half of epoch.
- Score REM sleep on epoch when :

  1.Low amplitude mixed frequency EEG activity.

  2.Low chin EMG tone.

  3.Rapid eye movement present.[182]

Titration methods:

Generally, the diagnostic study in the polysomnography is followed by "Full night" titration study. But in certain situations where the sleep disordered breathing is very severe, then first two hours will be used to diagnose the sleep apnea and the rest of the night, titration study continues. Such a study has been termed as "Split night study"

General guidelines:

- Appropriate mask size to be used that fits properly.
- Nasal decongestant can be used before the start of titration.
- The water coloumn manometer should be at 0 cm.
- When BIPAP system is used, inspiratory pressure should be kept equal to expiratory pressure.
- Titration to be started with CPAP=2cm water.
- 2 cm water pressure has to be increased until the AHI is less than 5.
- If associated snoring, desaturation or hypoapnea events are present, pressure to be increased by 1cm water.
- When optimal pressure is achieved, patient is allowed to go into REM sleep.[118]

Figure 1 : Sleep technician inserting various leads

Figure 2 : Patient undergoing polysomnography

STATISTICAL METHODS:

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance.

The following assumptions on data is made,

Assumptions:

1. Dependent variables should be normally distributed.

2. Samples drawn from the population should be random, Cases of the samples should be independent.

Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients , Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) on metric parameters. Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups, Non-parametric setting for Qualitative data analysis. Fisher Exact test used when cell samples are very small.

Pearson correlation between study variables is performed to find the degree of relationship, Pearson correlation co-efficient ranging between -1 to 1, -1 being the perfect negative correlation, 0 is the no correlation and 1 means perfect Positive correlation.

Significant figures

+ Suggestive significance (P value: $0.05<P<0.10$)

* Moderately significant (P value: $0.01<P \leq 0.05$)

** Strongly significant (P value: $P \leq 0.01$)

Statistical software:

The Statistical software namely SPSS 18.0, and R environment ver.3.2.2 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc. [82, 83, 84, 85]

RESULTS

Table 1: Age distribution of patients

Age in years	No. of patients	%
21-30	3	5.0
31-40	5	8.3
41-50	7	11.7
51-60	21	35.0
61-70	18	30.0
>70	6	10.0
Total	60	100.0

The mean age was 56.45±12.51 in our study

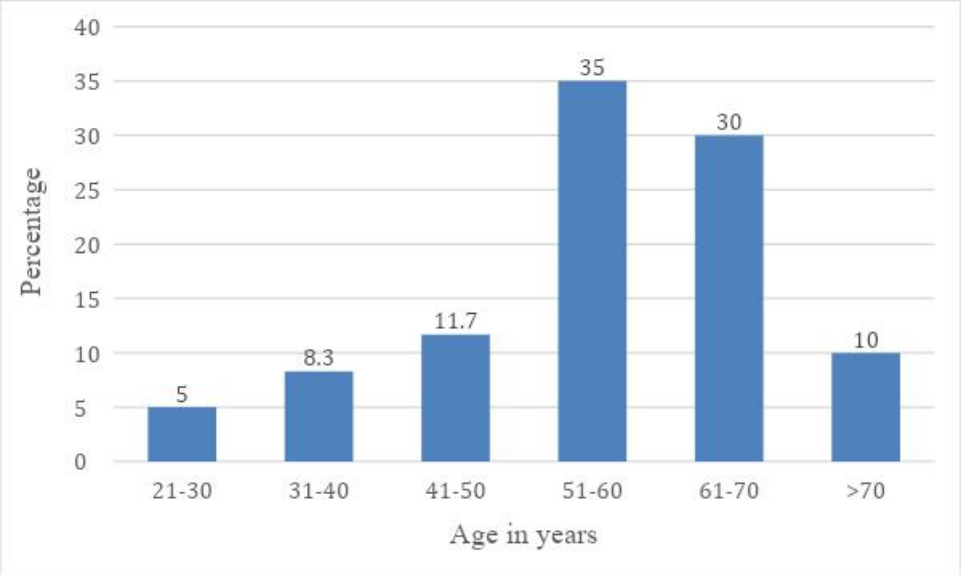


Figure 1 : Bar graph of age distribution of patients

Table 2: Gender distribution of patients

Gender	No. of patients	%
Male	34	56.7
Female	26	43.3
Total	60	100.0

Sixty patients of either sex had participated in the study, of which 56.7% were males and 43.3% were females patients

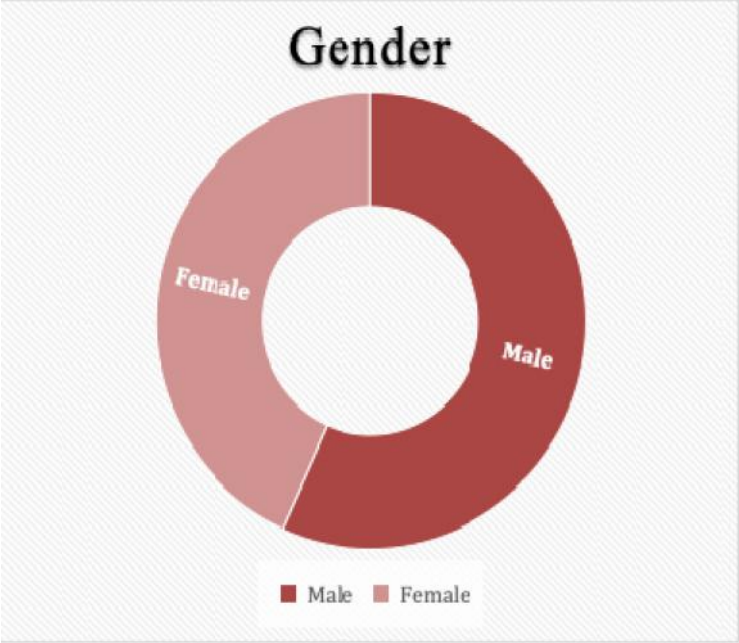


Figure 2 : Pie chart of gender distribution

Table 3: Smoking status of the patients

Smoking (Pack Years)	**Total**
Non-Smoker	34(56.7%)
1-10	1(1.7%)
11-20	22(36.7%)
>20	3(5%)
Total	60(100%)

A total of 56.7% were non-smokers and 11-20 pack years smoking history was present in 36.7% patients

Table 4: BMI (kg/m^2) distribution of patients

BMI (kg/m^2)	**Gender**		**Total**
	Male	**Female**	
18.5-25	6(17.6%)	6(23.1%)	12(20%)
25-30	19(55.9%)	13(50%)	32(53.3%)
>30	9(26.5%)	7(26.9%)	16(26.7%)
Total	34(100%)	26(100%)	60(100%)

In BMI distribution 53.3% were between 25-29.9, 26.7% were >30 and 20% were between 18.5-24.9 out of 60 patients in the study.

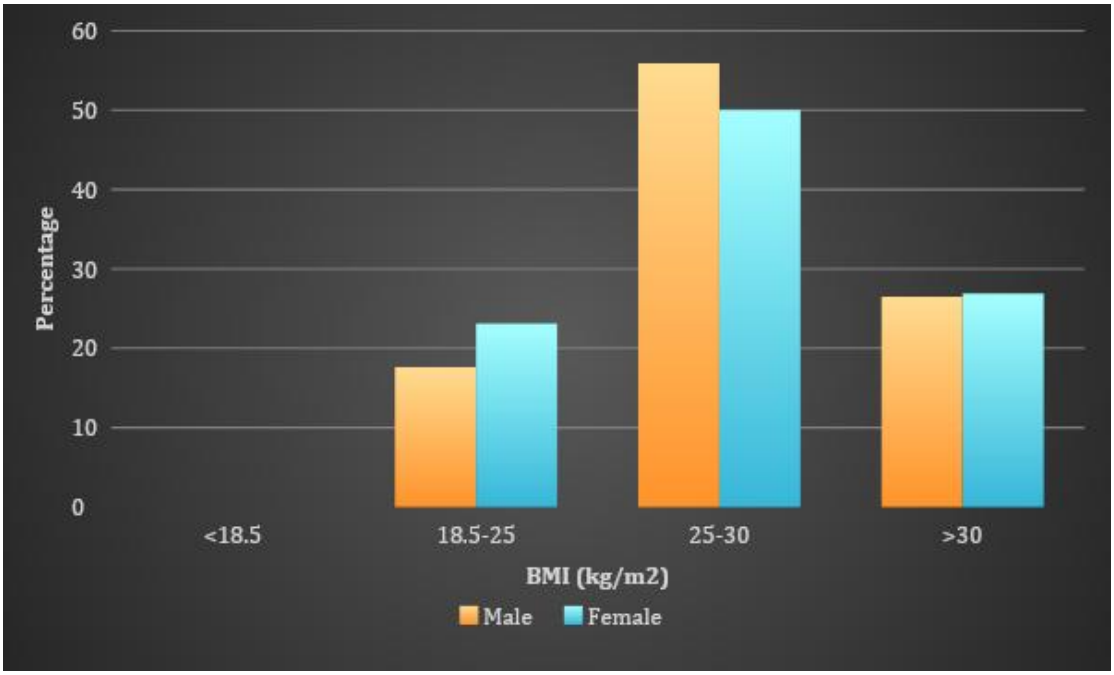


Figure 3: Bar graph showing BMI distribution among patients

Table 5: Habits of the patients

Habits	Total No of Patients (60)
Alcohol	28
Smoking	26
Tobacco chewer	20

Among 60 patients, 28 were alcoholics, 26 were smokers and 20 were tobacco chewer.

Table 6: History of previous accident of patients

History of accident	Gender		Total (n=60)
	Male (n=34)	Female (n=26)	
No	31(91.2%)	23(92%)	54(90%)
Yes	3(8.8%)	3(12%)	6(10%)

There were only 10% of patients who had history of accidents due to OSA in the study and remaining 90% did not have any history of accidents.

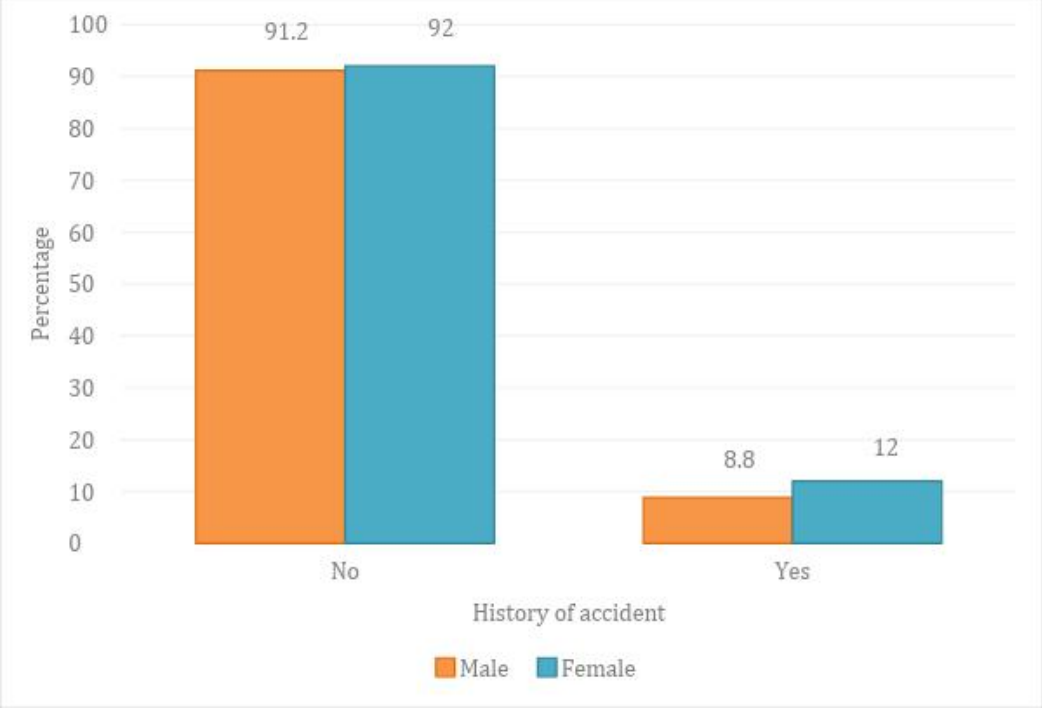


Figure 4 : Bar graph showing the accident history

Table 7: Comorbidities distribution of patients

Comorbidities	Total (n=60)
HTN	43(71.7%)
DM	42(70%)
IHD	18(30%)
HYPOTHYROIDISM	17(28.3%)
COPD	24(40%)
CVA	5 (8.3%)
STROKE	7 (11.6%)

In this, study, Hypertension was found in 71.7% and Diabetes Mellitus in 70% of patients. There were 40% of patients who had COPD. CVA and Stroke accounted to 8.3 % and 11.6 % respectively

Table 8: Epworth scale distribution of patients

Epworth	Gender		Total
	Male	Female	
<10	6(17.6%)	4(15.4%)	10(16.7%)
10-20	22(64.7%)	17(65.4%)	39(65%)
>20	6(17.6%)	5(19.2%)	11(18.3%)
Total	34(100%)	26(100%)	60(100%)

In this study , the Epworth Scale were between 10-20 in 65% of patients , score of more than 20 was seen in 18.3% of patients and less than 10 was found to be in 16.7% of patients.

Table 9 : Mallampati score of patients

Mallampati score	Total
1	7(11.7%)
2	21(35%)
3	15(25%)
4	17(28.3%)
Total	60(100%)

A total of 35% of patients had Mallampatti score of 2, 28.3% of patients had a Mallampatti score of 4

Table 10: AHI distribution of patients

AHI	No of patients
• <5	16 (26%)
• 5-15	13(21.6%)
• 15-30	11(18.3%)
• >30	20 (66.6%)

In the present study , AHI score of <5 was observed in 26% of patients. In 21.6%, it was between 5-15, another 18.3% of patients had score between 15-30 , while 66.6% of patients had score of >30

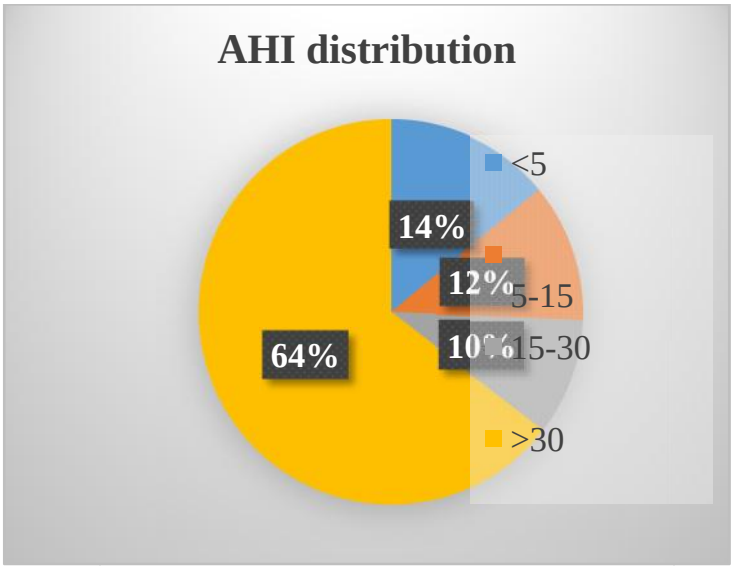


Figure 5:Pie chart depicting AHI distribution

Table 11: AHI- diagnostic and titration study

AHI	Gender		Total	P value
	Male	Female		
Diagnostic	29.34±28.88	27.37±34.08	28.49±30.98	0.810
Titration	3.12±2.69	2.53±2.47	2.87±2.59	0.473
Difference	26.22	24.84	25.62	-
P value	<0.001**	<0.001**	<0.001**	-

AHI assessment in diagnostic and titration study was done in diagnostic study MeanAHI was 28.49±30.98. After titration study, with appropriate titration done with CPAP/ BIPAP pressures,it was 2.87±2.59 and the P value <0.001, which means its strongly significant.

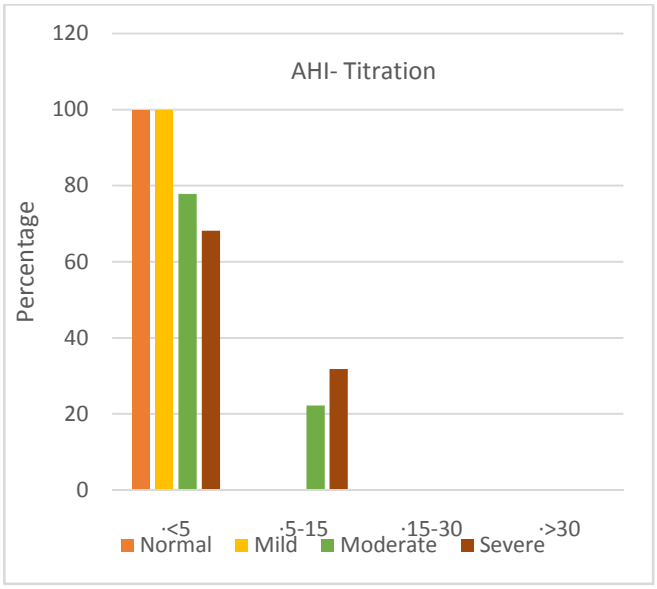


Figure 12, 13: Bar graph showing diagnostic and titration study

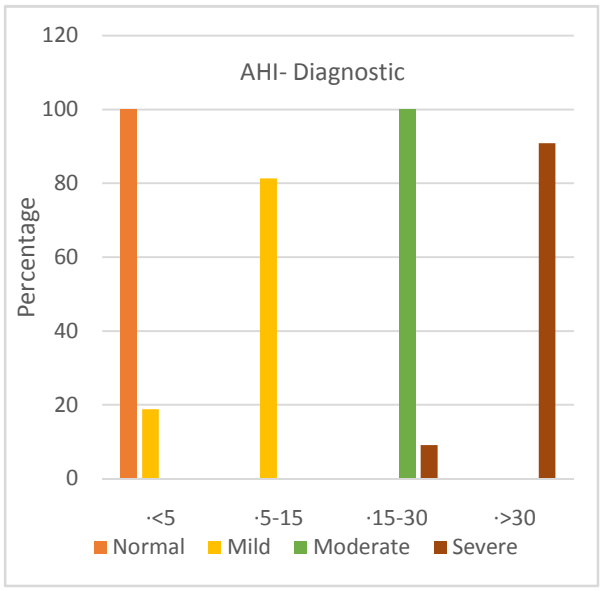


Table 12: Respiratory events distribution of patients

Respiratory events (Apnea+Hypopnea)	Total
<5	5(8.3%)
5-15	6(10%)
15-30	9(15%)
>30	40(66.7%)
Total	60(100%)

Respiratory events like Apnea and Hypopnea were studied in 60 of patients and >30 events were 66.7% of patients and 15-30 were 15% of patients in the study.

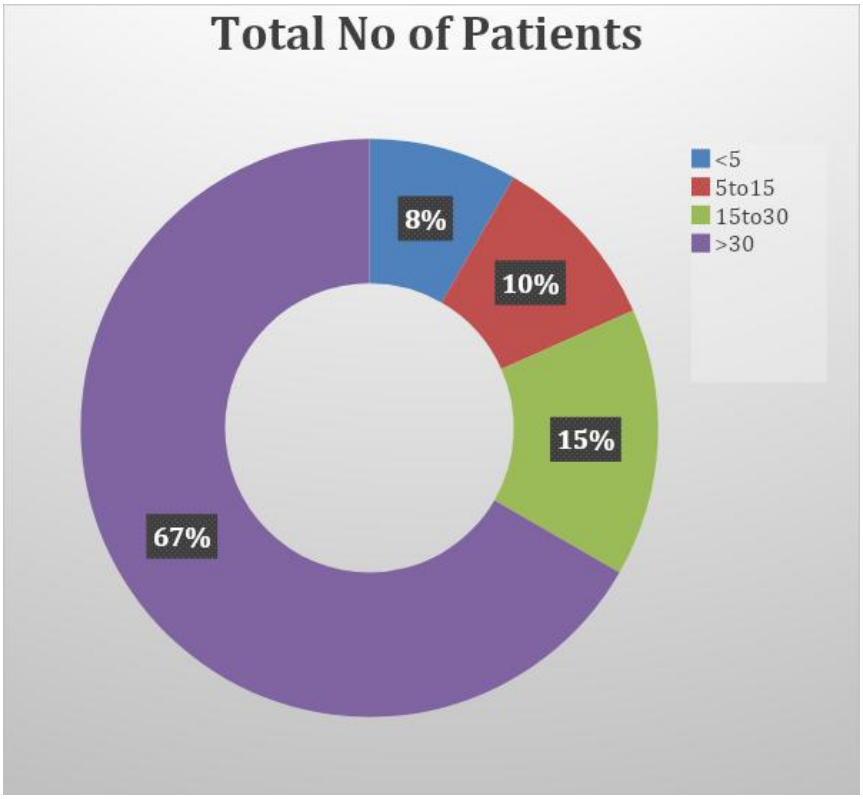


Figure SEQ Figure * ARABIC 22: Respiratory events studied in the patients

Table 13: Snoring distribution of patients studied

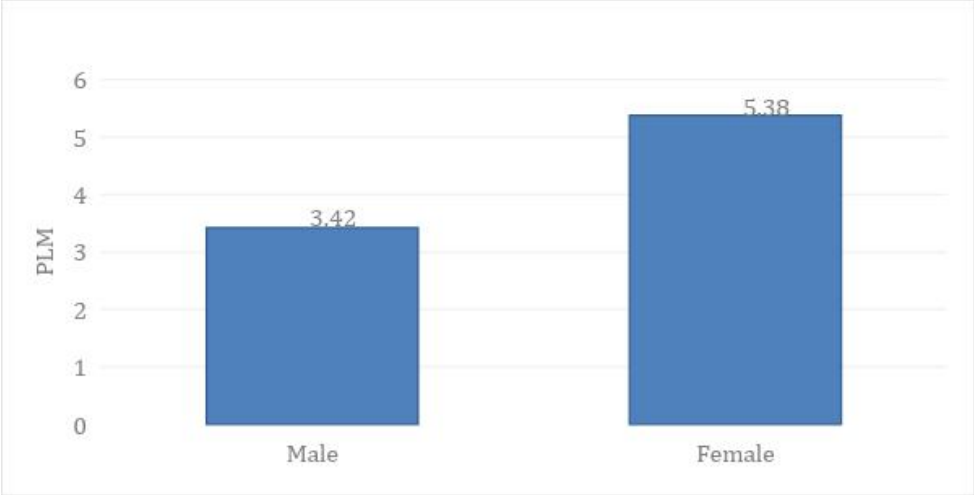
Snoring score	Total
<3	17(28.3%)
3-8	33(55%)
>8	10(16.7%)
Total	60(100%)

Snoring score distribution of patients studied were divided into <3, was observed in 28.3% of patients , and another 55% had score between 3-8 >8 and score of >8 was found in 16.7% of patients

Table 14: Polysomnography results of patients

Diagnosis	Gender		Total
	Male	Female	
Normal	6(17.6%)	8(30.8%)	14(23.3%)
OSA	28(82.4%)	18(69.2%)	46(76.7%)
PLM	1 (2.9%)	0	1

Diagnosis in the study where we saw normal patient and OSA patient in which patients with OSA was observed in 46 patients (76.7%) while polysomnography was observed to be normal in 14 patients. PLM seen was seen in 1 patient .



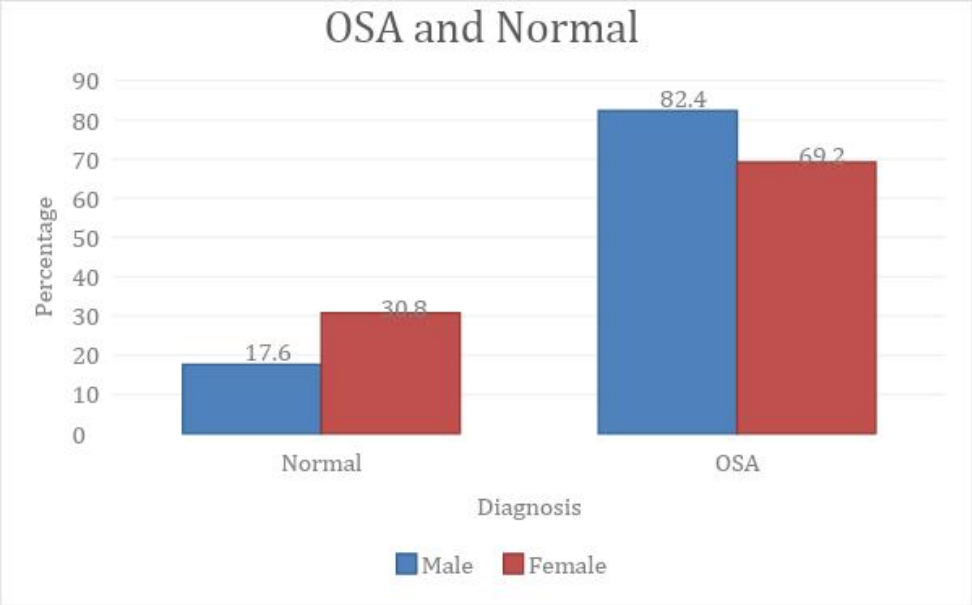


Figure 6 : Bar graph showing polysomnography results of patients

Table 15 : Severity of OSA

Severity	Gender		Total
	Male	Female	
Normal	5(14.7%)	8(30.8%)	13(21.7%)
Mild	12(35.3%)	4(15.4%)	16(26.7%)
Moderate	3(8.8%)	6(23.1%)	9(15%)
Severe	14(41.2%)	8(30.8%)	22(36.7%)
Total	34(100%)	26(100%)	60(100%)

Mild OSA was observed in 16 (21.7%) patients, 9 (15%)) patients had moderate OSA while 22 (36.7%) had severe OSA.

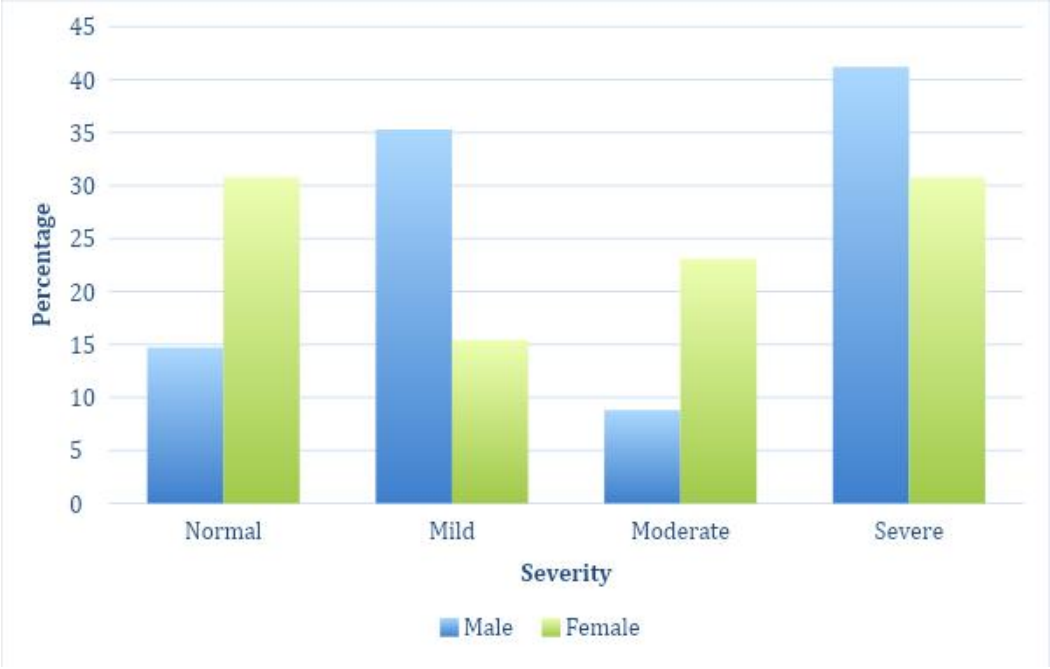


Figure 7 : Bar graph showing severity of OSA in patients

Table 16: Arousals distribution of patients

AROUSALS	Total
<40	5(8.3%)
40-50	5(8.3%)
>50	50(83.3%)
Total	60(100%)

In this study , 50(83.3%) patients had >50 arousals during sleep, while 5 (8.3%) patients had 40-50 arousals and the rest 5(8.3%) had <40 arousals during sleep

Table 17: Symptoms distribution of patients

SYMPTOMS	Total (n=59)
Night time awakening	12(20%)
Snoring	43(71.7%)
Tiredness	1(1.7%)
Increased sleep time latency	3(5%)
Day time sleepiness	44(73.3%)
Weight gain	28(46.7%)
Choking	42(70%)

In our study, the symptomatology was as follows, 73.3% had day time sleepiness, 71.7% had snoring, 46.7% had weight gain.

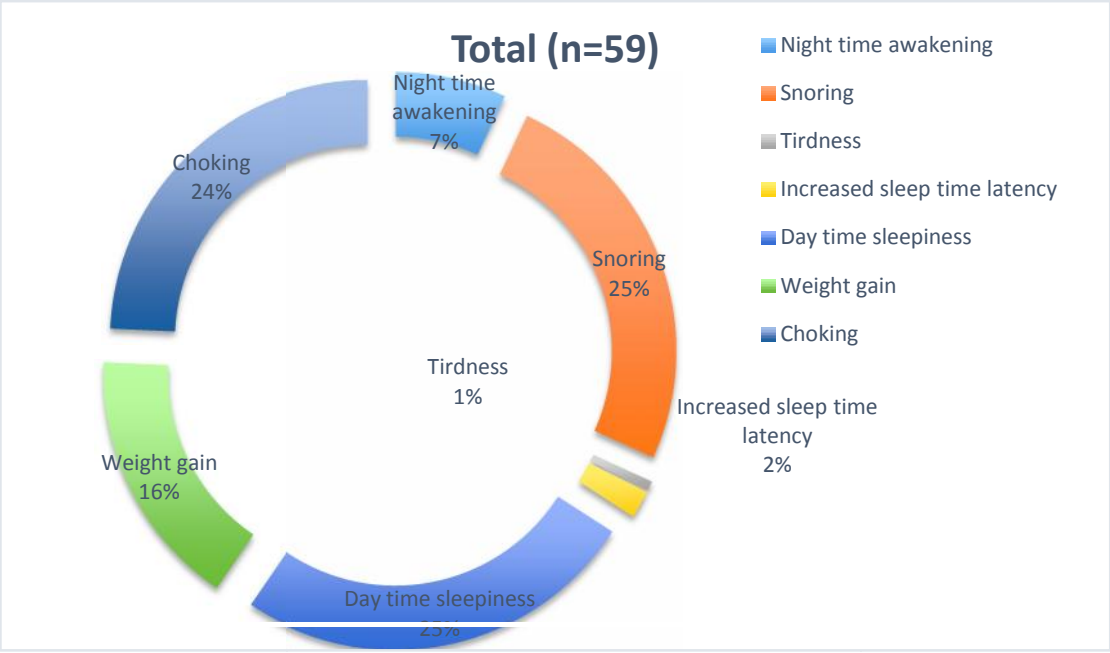


Figure 8 : Pie chart showing symptoms of patients

Neck circumference (cm)	Total
<30	2(3.3%)
30-40	50(83.3%)
>40	8(13.3%)
Total	60(100%)
Mean ± SD	37.63±3.05

Neck Circumferences of patients were measured and were divided to three groups. 2(3.3%) of patients had neck circumference of <30 cm, 50(83.3%) of patients had 30-40, while the rest 8(13.3%) of patients had >40 cm

Mean ± SD was 37.63±3.05.

Table 21: Symptoms distribution according to severity of OSA of patients studied

SYMPTOMS	OSA Severity				Total (n=60)
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)	
Night time awakening	1(7.7%)	6(37.5%)	3(33.3%)	2(9.1%)	12(20%)
Snoring	8(61.5%)	12(75%)	8(88.9%)	15(68.2%)	43(71.7%)
Tiredness	0(0%)	1(6.3%)	0(0%)	0(0%)	1(1.7%)
Increased sleep time latency	0(0%)	0(0%)	0(0%)	3(13.6%)	3(5%)
Day time sleepiness	11(84.6%)	8(50%)	5(55.6%)	20(90.9%)	44(73.3%)
Weight gain	6(46.2%)	5(31.3%)	7(77.8%)	10(45.5%)	28(46.7%)
Choking	7(53.8%)	10(62.5%)	8(88.9%)	17(77.3%)	42(70%)

Most common symptom was Exccessive day time sleepiness in 44 patients (73.3%) followed by Snoring in 43 patients (71.7%) and Choking in 42 patients (70%) . About 90.9% of patients with Severe OSA has EDS (90.9%) as the main complaint.

Table 22: AHI distribution according to severity of OSA

AHI	OSA Severity				Total (n=60)	P value
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)		
Diagnostic study	13	16	9	22	60	
• <5	13(100%)	3(18.8%)	0(0%)	0(0%)	16(26.7%)	<0.001**
• 5-15	0(0%)	13(81.3%)	0(0%)	0(0%)	13(21.7%)	
• 15-30	0(0%)	0(0%)	9(100%)	2(9.1%)	11(18.3%)	
• >30	0(0%)	0(0%)	0(0%)	20(90.9%)	20(33.3%)	
Titration study	N=1	N=11	N=9	N=22	N=43	
• <5	1(100%)	11(100%)	7(77.8%)	15(68.2%)	34(79.1%)	0.136
• 5-15	0(0%)	0(0%)	2(22.2%)	7(31.8%)	9(20.9%)	
• 15-30	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	
• >30	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	

Among 60 patients, 22 (33.3%) of patients had severe OSA with AHI > 30 (in 90% of patients). It was significantly reduced after the titration study. AHI shows linear correlation with OSA

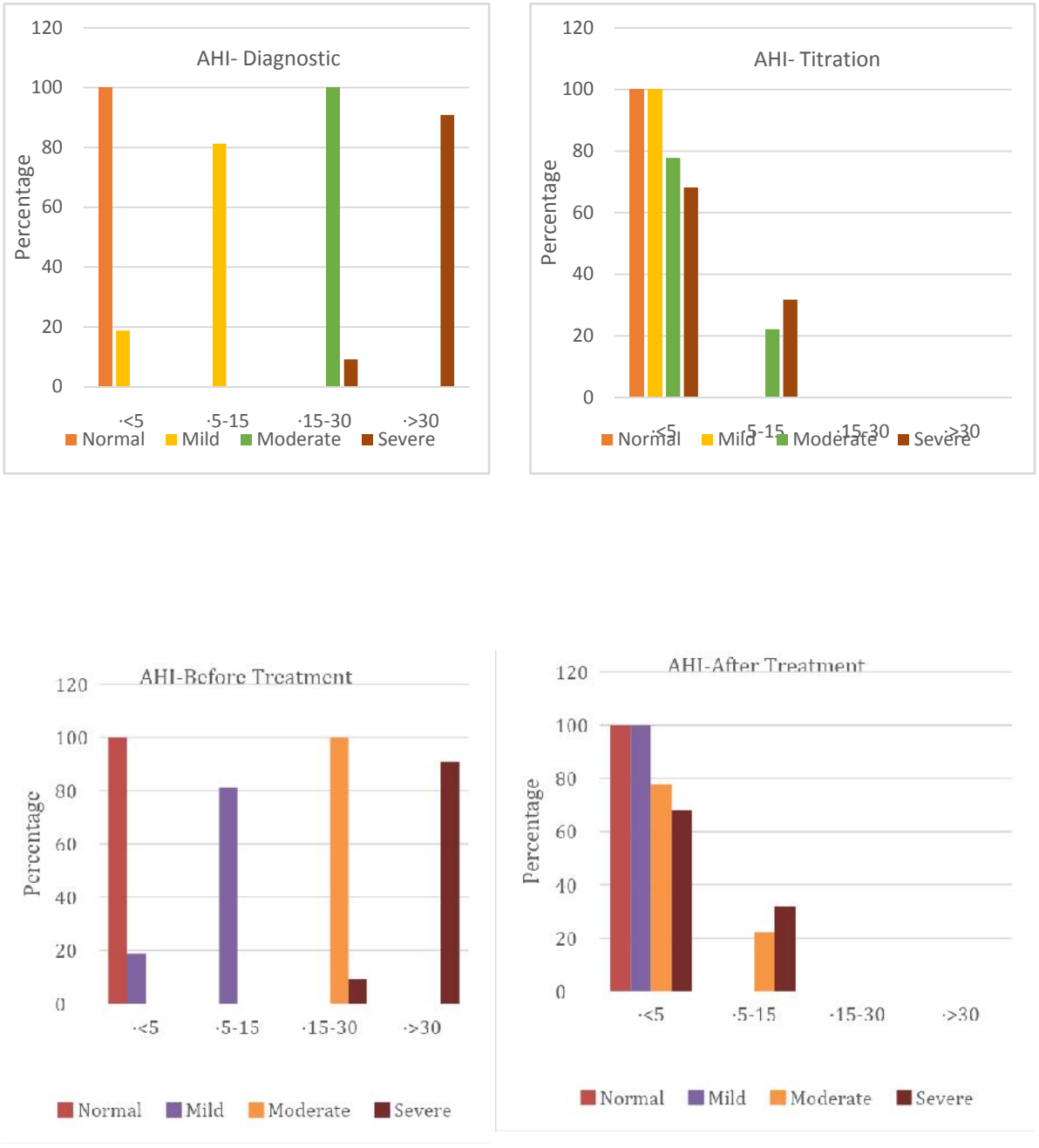


Figure 9 : Bar graph showing severity of OSA in Diagnostic and titration study

Table 23: AHI- Comparison according to severity of OSA of patients

AHI	OSA Severity				Total (n=60)	P value
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)		
Diagnostic study	1.70±1.67	8.49±3.67	20.73±3.35	62.02±26.98	28.49±30.98	<0.001**
Titration study	0.00±0.00	0.03±0.09	3.24±2.64	4.27±2.00	2.87±2.59	<0.001**

The mean AHI of all patients was 28.49±30.98 which was reduced to 2.87±2.59 after titration study

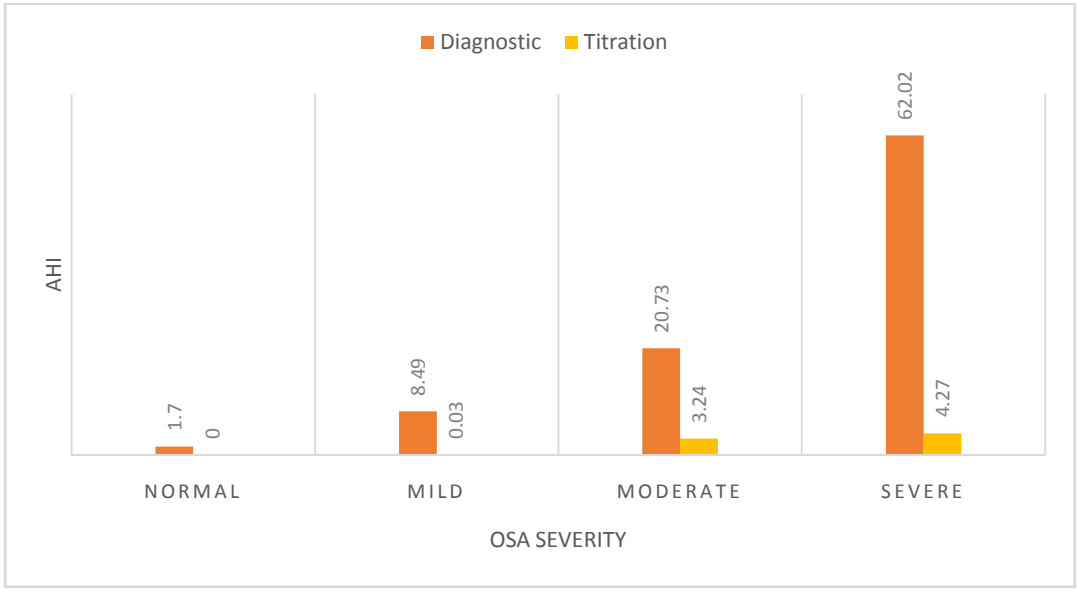


Figure 10 : Bar graph showing the comparison of AHI in diagnostic and titration study

Table 24: Epworth score distribution in relation to Mallampati score of patients

Epworth score	Mallampati Score				Total
	1	2	3	4	
Mean ± SD	11.71±4.82	15.48±5.75	12.20±7.23	18.71±4.95	15.13±6.32

P=0.010**, Significant, ANOVA TEST

In our study , patients with Mallampatti score of 2 had mean Epworth score of 15.48±5.75 while those with the Mallampatti score of 4 had mean Epworth score of 18.71±4.95

Table 25: Neck Circumference distribution in relation to Malampatti score of patients

Neck Circumference	Malampatti Score				Total
	0	1	2	3	
Mean ± SD	38.14±2.12	37.38±2.06	36.27±4.32	38.94±2.68	37.63±3.05

In this study , patients with mean neck circumference of 38.94±2.68 correlated with Mallampatti score of 3.

Table 26: Various sleep variables in comparison with OSA severity

Study variables	OSA Severity				Total (n=60)	P value
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)		
PLM INDEX	7.9±8.9	2.1±3.7	4.1±5.9	3.7±4.9	4.3±6.1	0.082+
TOTAL SLEEP TIME (MINUTES)	430.2±132.1	357.6±112.2	398.2±94.7	296±144.3	356.8±134.9	0.023*
TIME IN BED (MINUTES)	316.7±120	254.9±120.3	324.8±115.1	219.3±137.9	265.7±130.8	0.079+
NREM SLEEP TIME	258.2±93.3	204.6±78.8	262.1±92	184.2±99.8	217.4±95.8	0.060+
REM SLEEP TIME	55±40.6	50.3±49.7	62.7±39.2	35.1±46.1	47.6±45.1	0.381
TIME IN BED WHEN SATURATION <90% (IN %)	7±16.4	8±14.4	17.6±20.9	19.6±24.5	13.5±20.3	0.182
REM AHI	2.8±4.9	10.4±13.9	36±16.2	32±34.9	20.5±26.6	0.001**
NREM AHI	1.1±2.2	7.4±9.1	24.5±25.1	59.4±31.9	27.7±33.4	<0.001**
SLEEP EFFICIENCY (%)	67.7±25.5	69.7±21.7	81±18.7	69.9±22.2	71±22.2	0.536

In this study,

- The least amount of sleep time was seen in severe OSA patients ie about 296 minutes in which total sleep time was around 219 minutes.
- Mean sleep efficiency was 71±22.2 %, severe OSA patients had 69.9±22.2 %.
- The time in bed with O2 saturation less than 90% was least seen in moderate OSA subjects , ie around 19.6±24.5 %
- REM AHI was maximum in moderate OSA subjects ie 36 ±16.2, NREM AHI maximum in Severe OSA subjects ie 59.4±31.9

Table 27: Study variables- Comparison severity of OSA with various parameters

Study variables	OSA Severity				Total (n=60)	P value
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)		
Age in years	57.1±14.8	58.6±7.2	58.4±12.5	53.7±14.3	56.5±12.5	0.621
Weight in kg	83.7±14.6	78.8±12	75.2±7.3	80.5±11.6	79.9±11.9	0.419
BMI (kg/m2)	29.5±2.9	28.4±3.8	27.3±3.2	29±2.5	28.7±3.1	0.389
EPWORTH	13.5±6.6	14.1±7.5	17.1±5.8	16±5.4	15.1±6.3	0.479
Mallampatti score	2.4±1.3	2.7±0.9	3.1±0.9	2.7±1	2.7±1	0.439
RDI	3.1±3.5	9.9±3.8	27.6±10.5	63.9±26.6	30.9±31.2	<0.001**

In our study,

- Mean BMI 29±2.5 was Maximum in Severe OSA patients
- Mean Epworth score 17.1±5.8 was Maximum in Moderate OSA patients
- Maximum RDI 63.9±26.6 was seen in Severe OSA patients
- In severe OSA patients, Mean Mallampatti score was 2.7±1, whereas maximum score of 3.1±0.9 was seen in moderate OSA subjects

Table 28: Mallampati score distribution according to severity of OSA of patients

Mallampatti score	OSA Severity				Total (n=60)
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)	
1	4(30.8%)	1(6.3%)	0(0%)	2(9.1%)	7(11.7%)
2	4(30.8%)	6(37.5%)	3(33.3%)	8(36.4%)	21(35%)
3	1(7.7%)	6(37.5%)	2(22.2%)	6(27.3%)	15(25%)
4	4(30.8%)	3(18.8%)	4(44.4%)	6(27.3%)	17(28.3%)

In the current study, 6 (27.3%) patients with severe OSA patients had mean Mallampatti score of 4.

Table 29: Snoring score distribution according to severity of OSA of patients

SNORING SCORE	OSA Severity				Total (n=60)
	Normal (n=13)	Mild (n=16)	Moderate (n=9)	Severe (n=22)	
0	8(61.5%)	3(18.8%)	0(0%)	4(18.2%)	15(25%)
1-3	2(15.4%)	1(6.3%)	0(0%)	0(0%)	3(5%)
4-6	3(23.1%)	11(68.8%)	2(22.2%)	1(4.5%)	17(28.3%)
7-10	0(0%)	1(6.3%)	7(77.8%)	17(77.3%)	25(41.7%)

P<0.001**, significant, Fisher Exact test

Maximum snoring score from 7-10 was observed in 7 (77.8%) patients of Moderate OSA group and 17 patients (77.3%)in severe OSA group

Table 30: Comparison of study variables according to Epworth scale

Variables	EPWORTH			Total	P value
	1-10	10-20	>20		
AHI	13.18±17.87	33.52±33.41	24.55±27.98	28.49±30.98	0.162
PLM	5.70±6.72	4.15±6.26	3.45±5.47	4.28±6.13	0.694
AROUSALS	182.40±132.33	125.39±92.49	137.73±90.49	137.36±100.15	0.282
Neck circumference	34.30±4.42	38.26±2.41	38.45±1.29	37.63±3.05	<0.001**

- Maximum AHI 33.52±33.41 was found in subjects with Epworth score between 10 and 20
- Patients with AHI between 1-10 had maximum arousals 182.40±132.33
- Neck circumference of 38.45±1.2cm was highest in subjects with AHI > 20

Table 31: Comparison of various Pressure devices

CPAP	BIPAP	AUTOPAP	NO CPAP/ BIPAP/AUTOPAP
10	7	30	14

Among the 59 subjects, 10 patients required CPAP, 7 patients were advised BiPAP and 30 were given AUTOPAP, after titration study study depending upon the pressure required to achieve AHI<5

DISCUSSION

Sleep disordered breathing (SDB) is correctable but often an underdiagnosed condition. SDB has been associated with various cardiovascular disease,diabetes melltius, hypertension , COPD, stroke and other morbidity. Obesity is an important precipitating factor. Overnight polysomnography has been the gold standard in diagnosis of sleep disordered breathing. Studies have demonstrated that CPAP is the most effective medical treatment, used for about 12-15 hours / day. This along with lifestyle modifications has been proven to show tremendous improvement In mortality and morbidity[153,92]

The present cross sectional study included 60 patients after satisfying the inclusion criteria attending the outpatient clinic of tertiary care hospital over a period of 12 months underwent detailed history taking , clinical examination followed by Level 1 polysomnography. Sleep study reports were analysed and subjects were categorized into Mild,Moderate, Severe OSA based on AHI values. Various parameters were correlated with OSA severity.

Most of the patients belonged age group of 51- 60 years with mean age being 56.45±12.51 years. Hasan et al [132] and Sreedharan et al.[133] were few Indian studies and few international studies like Young et.al[154] and Gislason et al[155] have found that SDB occurs in middle aged male population which was similar to our cohort According to Sharma et al[8], age was an independent risk factor

Obesity is one of the foremost risk factor in development of SDB. Our study cohort showed that more than 50% of the overweight individuals had OSA. In a hospital based study[157], higher BMI was found in OSA but it did not correlate with the

severity of OSA. While another study in Indian urban men, a linear correlation was found with increasing BMI and obesity[9.]

Neck circumference, a measure of central obesity, a mean of 37.63±3.05 was noted in our study. It was similar as reported by Udwadia [9] and Ahabab et.al [157]. Higher Neck Circumference and BMI in OSAS patients was associated with aerobic capacity. physical inactivity. and excess body fluid. [133,132]

Mallampatti score has also been one of the predictors of OSA severity. In our cohort, 27.3 % of subjects with a score of 4 had severe OSA. A global comparison by Sutherand et.al [158] could not prove any correlation between Mallampati score and OSA severity across ethnic groups. But an Indian study by Shyamala and colleagues[159] showed that increase in OSA severity was associated with increased mallampatti score.

OSA patients are frequently observed with various comorbid conditions like diabetes mellitus, hypertension, Stroke, CVA, depression, Obesity etc. It is noticed that obesity and OSA , each are potently linked to Hypertension. Our results showed 71.7% of OSA patients were hypertensive.[133] Smith et.al[160] had similar prevalence of hypertension in OSA patients. It is shown that, after every apneic or hypoapneic event, the sympathetic drive increases, which leads to greater cardiac output, that in turn raises the peripheral vascular resistance and sodium reabsorption in kidney with resultant rise of blood pressure.[133]The Wisconsin Sleep Cohort Study concluded that, four years after the diagnosis of sleep disordered breathing, the development of new hypertension was seen [161] .

In our study, it was observed that 70% of OSA patients were having diabetes mellitus. OSA is believed to be more prevalent inalcoholics, because it is known to increase airway collapsibility and also contributes to higher BMI[162] . In our cohort, 46% of patients had history of alcohol consumption. It causes the reduction in tone of genioglossus muscle and thereby increases upper airway resistance [162, 163]. Simou et .al [162] showed that people who consumed alcohol are approximately having 25% more chances of having OSA. Another study [163, 164] suggested that, frequency of alcohol consumption do affects the occurrence of OSA .

In our cohort, excessive daytime sleepiness was the one the frequent complaint that accounted to 73.3% of the study population followed by snoring and choking episodes. Patients experiencing OSA have been classified in various clusters as the "disturbed sleep group" (cluster 1), "minimally symptomatic group" (cluster 2) and "excessive daytime sleepiness group" (cluster 3). Our cohort has majority of patients in cluster 3 who also had a somewhat higher likelihood of presenting with classic OSA symptoms, such as night-time breathing pauses and loud snoring disturbing their spouse's sleep. The findings were similar to a cluster analysis study by Ye et.al [165] Johns proposed the Epworth Sleepiness Scale (ESS), an objective measure of daytime sleepiness as a instrument to differentiate a primary snorer from OSAS, dating back to 1993. [57]. The AHI – EDS association has conflicting results in literature with some revealing strong co relation[166,167] while others show weak correlation[168,169]. Our Cohort had a positive correlation, indicating that AHI can be a diagnostic tool in predicting OSA severity, similar to other Indian studies [9,157]. This may not be completely true because a study done at Delhi by Reddy et.al[170],could not prove any association between OSA severity and AHI.

The sleep characteristics like sleep efficiency , REM and NREM AHI , nocturnal desaturation , total sleep time and time in bed were comparable with OSA severity . In our cohort , it was found that overall sleep efficiency of the patients was about 71%. Lowest sleep efficiency (69.9%) was found in moderate OSA subjects. Patients with severe OSA had mean AHI of 32. Our study also showed that maximum nocturnal desaturation was found in severe OSA group. Total sleep time in severe OSA patients was 356 minutes where as in normal subjects it was 430 minutes. There were around 137 arousals in severe OSA patients. REM sleep duration was analogous , greater reduction of slow wave sleep in patients with severe OSA . In our study, REM AHI was found to be highest in moderate OSA group. Bianche et al[171] concluded that OSA accelerated the decay rate of REM and NREM sleep manifesting as shorter sleep bouts with increased number of sleep transition. Another study, [172] which compared sleepy with non-sleepy patients showed that shorter sleep latency and slower deep wave sleep was seen in EDS subjects. From the above study it can be concluded that sleepy patients have worse sleep related breathing parameters with lighter and fragmented pattern than non-sleep patients . Since the number of subjects without EDS was less in our cohort, its association with slow wave sleep shortening could not be assessed.

Our study showed nocturnal desaturation varied with OSA severity. Few studies support this evidence. Sasai et.al [173]reported that late night desaturations were more commonly seen in severe OSA patients. Another study by Huamani and colleagues [174], found independent association between sleepiness and nocturnal hypoxemia.

Periodic limb movements during sleep are repetitive limb contractions that happens during sleep which are associated with arousal or awakening [180.] They are a very common finding in OSAS subjects. In our study, only 1 patient had PLM. Patients with PLM usually have excessive daytime sleepiness and and insomnia. In a study by Scofiled et.al [181] the prevalence of PLM was found to be 7.6 % when 562 patients in USA were studied. Low ferritin and iron levels are found to be linked with PLMs. The study by Careli et.al [180] found no correlation between PLM and OSAS. However, more research is needed in this perspective.

STRENGTH OF STUDY

Our Study involves using Level 1 polysomnography in a rural population. Very fewer studies are available targeting the rural population of South India. Furthermore, our study correlates the anthropologic and polysomnographic measurements with OSA severity, and based on titration study positive pressure therapy devices have been prescribed and henceforth , this study can further contribute to literature on this topic.

LIMITATIONS OF THE STUDY:

- This a small study with lesser sample size at a tertiary care centre and therefore might not be a representative of the overall OSA population.
- Unequal sex distribution is a limiting factor.
- Data was collected only on the presence or absence of risk factors and did not inspect their duration or control status.
- The effect of CPAP in improvement of AHI,EDS,BMI and other parameters are not studied
- The complaints, compliance and complications of CPAP use has not been studied.
- Prevalance of OSA - overlap syndromes has not been investigated in detail.
- The study population involved adults > 18 years of age, thus the paediatric group is neglected.
- All the PSGs were manually scored by the sleep experts; there might have beeen interobserver differences in the sleep staging and marking the duration of apneas but these are only negligible voids, which is unlikely to have an impact on the final results.

FUTURE PERSPECTIVES

Future research may point towards studying the prevalence of sleep disordered breathing in children. CPAP, which remains the mainstay treatment of OSA, its effects on various clinical, anthropologic and polysomnographic parameters needs to be evaluated.

CONCLUSION

This study throws light over the prevalence of Sleep disordered breathing in northern part of Karnataka. Our study revealed that OSA is more prevalent in middle aged men with risk factors like alcohol consumption, diabetes mellitus, hypertension, coronary artery diseases, CVA, Stroke etc. They tend to be overweight with increased neck circumference and most commonly present with EDS, indicating the each of the above mentioned factor can be a determinant of OSA severity and sometimes can be fatal. CPAP aids in lessening the morbidity, mortality and thereby improve the quality of life. Awareness about this disease, identifying the red flag signs with prompt usage of PAP devices, may reduce the disease burden.

SUMMARY

- Our cohort included a total of 60 patients with 34 (56.7%) males and 26 (43.3%) females with mean age of 56.4 years.
- About 46% of the subjects were alcoholics and 43% were current and exsmokers
- Most common co morbidity were hypertension followed by diabetes mellites, COPD and CAD
- 16 patients had COPD-OSA overlap syndrome.
- Mean AHI was 28.49 and mean BMI was 28.7kg/m^2.
- Excessive day time sleepiness (73.3 % of patients) andsnoring (71.1% of patients) were the most common symptoms at presentation.
- About 36.7% of the study population had severe OSA and 1 patient had PLM.
- Mallampatti score,BMI, Snoring score varied with OSA severity.
- Patients with mean neck circumference of 38.94±2.68 correlated with mallampatti score of 3.
- Maximum AHI of 33.52±33.41 was found in subjects with Epworth score between 10 and 20.
- 22 (33.3%) of patients had severe OSA with AHI > 30 (in 90% of patients).
- Excessive daytime sleepiness was found in patients with higher AHI
- Obese patients has higher AHI
- In severe OSA patients, mean mallampatti score was 2.7±1
- Epworth sleepiness scale, which quantifies EDS, showed positive correlation with OSA severity.
- About 50(83.3%) patients had >50 arousals during sleep.

- Maximum snoring score from 7-10 was in found in 25 (41.7%) of patients , and among those , it was found to be in moderate and severe OSA
- About 10 patients were recommended CPAP, 7 were advised BiPAP and rest 30 were recommended AutoPAP devices depending upon the pressures required after titration study.

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ANNEXURE 1- INFORMED CONSENT

EVALUATION OF SLEEP DISORDERED BREATHING BY LEVEL 1 POLYSONOGRAPHY IN A TERTIARY CARE HOSPITAL: A HOSPITAL BASED OBSERVATONAL STUDY

Purpose of the study:

You are being asked to enroll in the study as you are eligible for participation in this study. All patients who are supected to have sleep disordered breathing will be included in this study. During this study, patients will be asked questions regarding their presenting complaints and they are supposed to answer to the best of their knowledge. The principal investigator of the study is DR._____under the guidance of DR. _____(guide).

The purpose of this study is to study various Sleep disordered breathing patterns in patients who come to OPD with signs and symptoms of the sleep apnea and to correlate the severity of sleep apnea syndrome with BMI. Obstructive sleep apnoea syndrome (OSAS) has been associated with obesity related co-morbid diseases like type 2 diabetes mellitus, coronary artery disease, congestive heart failure, hypertension, chronic kidney disease, dyslipidaemias and metabolic syndrome as applicable to the Indian population.In a resource limited setting like India, there isa paucity of health care facilities with multi-channel polysomnography equipment, thereby further limiting the evaluation of OSAS patients. In spite of the constraints, researchers in India and its neighbouring countries in the last decade have made a preliminary attempt to study OSAS and co-morbid conditions.

Procedure:

Patients whose symptoms are suggestive of sleep disordered breathing attending the outpatient clinic of Dr KLE's Prabhakar Kore Charitable Hospital and MRC will be subjected to Detailed history taking, Clinical examination followed by Level 1 polysomnography study

Polysomnography study takes place in a quiet, dark,temperature controlled room with constant monitoring. The various variables recorded during the study will include ECG, Central and occipital EEG, EOG, Submentalis EMG, Nasal and oral air flow, Arterial Oxygen saturation , breathing pattern by Thermister, Cannula, Thoracic wall movements abdominal movements, Anterior Tibialis EMG, snoring and body position. Sleep study will be conducted by sleep technician from 10pm to to 6am next day. Sleep scoring is done by sleep technician.

Risks and benefits:

There are no risks or benefits involved.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part now,you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may terminate your participation in this study anytime.

Privacy and confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study will be published but your identity will be confidential in any publication.No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Institution/sponsor's policy:

Does not apply to this research.

Financial incentivesfor participation:

You will not be paid /offered any gift/incentives for participating in this study.

Authorization to publish results:

The results of this study would be forwarded to the KLE University, Belgaum as a part of requirement towards the completion of MD degree, review and publishing..

Questions:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact:

- Dr. _____, Department of Respiratory Medicine, KLES Hospital and MRC,
- Dr. _____ , Professor and Head, Department of Pulmonary Medicine, KLES Hospital and MRC,Belagavi.
- If you have any queries about your rights as a study subject, you may call Dr. Roopa Bellad, Professor, Department of Pediatrics, J.N. Medical College Institutional Ethical Committee for Human Subjects Research, or extension 4052 at J.N. Medical College, Belagavi.

CONSENT STATEMENT

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

In case of the queries during the study or in future you may contact following person.

Principle investigator :

Guide :

Name of the participant: (signature/thumb print)

Name of the witness: (signature)

Name of the investigator: (signature)

Date: Place:

Address:

एकवाहकदेखभालअस्पतालमे 1

पॉलीसोनोग्रैफेद्वारालेटेहुएसोजानेकीघटनाकामूल्याकन:

एकांचोंकेत्सीयआधारितबाहरीअध्ययन

अध्ययनकाउद्देश्य:

आपकोइसअध्ययनमेभागलेनेकोलेएकहाजारहाहैक्योंकेआपइसअध्ययनमेभागलेनेकोलेएपात्रहैं।जिनरोंगेयोकोनींदमेखललपडected ेकीआशकाहै,

उन्हेइसअध्ययनमेशांमेलांकेयाजाएगा।इसअध्ययनकेदौरान,

मरीजोसेउनकीवतेमानांशेकायतोकेबारेमेसवालपूछेजाएगेऔरउन्हेअपनेज्ञानकासबसेअच्छाजवाबदेनाचांहिए।अध्ययनकेमुख्यअन्वेषकहैं

(गाइड) केमागेदशेनमे।

इसअध्ययनकाउद्देश्यरोंगेयोमेनींदांवेकारकोंवोंभिन्नप्रकारोकाअध्ययनकरनाहै,

जोओपीडीमेस्लीपएपांनेयाकेलक्षणोऔरलक्षणोकेसाथआतेहैंऔरबीएमआइकेसाथस्लीपएपांनेयांसेड्रोमकीगभीरताकोसहतेहैं।ऑब्सट्रांक्टिवस्लीपएपांनेयांसेड्रोम (OSAS) मोटापेसेसबांधितसह-रुग्णरोगोजैसोंकेटाइप 2 मधुमेह, कोरोनरीधमनीरोग, ंदिलकोंवेफलता, उच्चरक्तचाप, क्रोंनेकांकेडनीरोग, ंडोंस्लिप्लोंमेयाऔरमेटाबोंलेकांसेड्रोमसेसबांधितहैजोभारतीयजनसख्यापरलागूहोताहै।भारतजैसेससाधनसींमेतसोंटेग, मल्टी-चैनलपॉलीसोम्नोग्राफीउपकरणकेसाथस्वास्थ्यदेखभालसुंविधाओकीएकसमताहै, जिससे OSAS रोंगेयोकेमूल्याकनकोसींमेतांकेयाजासकताहै।बाधाओकेबावजूद, ंपिछलेएकदशकमेभारतऔरइसकेपड़ोसीदेशोकेशोधकतोओनेओएसएएसऔरसह-रुग्णपांरोंस्थांतेयोकाअध्ययनकरनेकोलेएप्रांरांभिकप्रयासांकेयाहै।

प्रक्रिया:

जिनमरीजोकेलक्षणनीदकीगड़बड़ीकेसकेतहोतेहैं,

वेडॉ।केएलइकेप्रभाकरकोरेचौरेटेबलअस्पतालऔरएमआरसीकेबाह्यरोगीक्लिनेकमेजातेहैं,

उन्हेंवेस्तृतइतिहासजाचकेअधीनकियाजाएगा, क्लिनेकलपरीक्षाकेबादलेवल 1

पॉलीसोमोग्राफीअध्ययन

पॉलीसोम्नोग्राफीअध्ययनरातेरातनेगरानीकेसाथएकशात, अंधेरे,

तापमानियंत्रितकमरेमेहोताहै।अध्ययनकेदौरानदजॉकेएगएवेभिन्नचरमेइसीजी,

सेट्रलऔरओसीसीपटलइइजी, ईओजी, सबमेंटलेसइएमजी, नाकऔरमौंखेकवायुप्रवाह,

धमनीऑक्सीजनसतृप्ति, थर्मिस्टर, कैनुला, श्वाससबधीपेटकेआदोलनोद्वारापेटकेआदोलनो,

पूवेकालटिबिआलेसइएमजी,

खरोटेऔरशामिलहोगे।शरीरकीस्थिति।स्लीपटेक्नीशियनद्वारास्लीपटेक्नीशियनद्वाराअगले

दिनरात 10 बजेसेसुबह 6

बजेतकआयोजितकियाजाएगा।स्लीपस्कोरिंगस्लीपटेक्नीशियनद्वारकियाजाताहै।

जोखिमऔरलाभ:

इसमेकोइजोखिमयालाभशामिलनहीहै।

विकल्प:

इसअध्ययनमेभागलेनास्वैच्छिकहै।आपइसअध्ययनमेभागनहीलेनेकाचयनकरसकतेहैं,

यादिआपअभीभागलेनेकानिणेयलेतेहैं,

तोआपबादमेअपनाविचारबदलसकतेहैऔरअध्ययनसेहटसकतेहैं।आपकानिणेयवतेमानयाभावे

ष्यकोस्वास्थ्यदेखभालयाआपकेद्वाराप्राप्तअन्यसेवाओकोनहोबदलेगा।अध्ययनकेचोकेत्सक याप्रायोजककभीभीइसअध्ययनमेआपकोभागीदारीकोसमाप्तकरसकतेहै।

गोपनीयताऔरगोपनीयता:

इसअध्ययनकेदौरानआपकेबारेमेएकत्रकोगइेसभीजानकारीकोकानूनद्वाराअनुमतसीमातकगो पनीयरखाजाएगा।कोडनबरइसशोधरेकॉडेमेआपकोपहचानकरेगे।इसअध्ययनसेसूचनाप्रकाशे तकोजाएगी,

लेकेनआपकोपहचानकेसीभीप्रकाशनमेगोपनीयहोगी।अनुसधानकेदौरानआपकेद्वारादोगइे जानकारीयाआपकेबारेमेजानकारीकेअलावाआपकोलेखेतअनुमतिकेबिनाअन्यकेलेएखुलासा कियाजाएगा:

1. अपनेअधिकारोऔरकल्याणकोरक्षाकेलेएआपातकालमे।

2. यदिकानूनद्वाराआवश्यकहो।

सस्थान / प्रायोजककोनीति:

इसशोधपरलागूनहोहोताहै।

वित्तीयप्रोत्साहनभागीदारी:

इसअध्ययनमेभागलेनेकेलेएआपकोकोइउपहार / प्रोत्साहननहीदियाजाएगा।

परिणामप्रकाशितकरनेकेलेएप्राधिकरण:

इसअध्ययनकेपरिणामएमडीडिग्री,

समीक्षाऔरप्रकाशनकेपूराहोनेकीआवश्यकताकेएकभागकेरूपमेकेएलइीवेश्वविद्यालय,

बेलगामकोभेजेजाएंगे।

प्रश्न:

यदिआपकेपासअध्ययनसेसबंधितकोइप्रश्नहै,

तोभविष्यमेयाअध्ययनसेसबंधितचोटयाबीमारीकेमामलेमे, आपसपकेकरसकतेहै:

• रेस्पिरेटरीमेडिसिनविभाग, केएलइएसअस्पतालऔरएमआरसी, पीएच।

• अंजानजी.एस.गुड, प्रोफेसरऔरप्रमुख, पल्मोनरीमेडिसिनविभाग, KLES अस्पतालऔर MRC, बेलगाम

• यदिआपकेपासअध्ययनविषयकेरूपमेआपकेअधिकारोकेबारेमेकोइप्रश्नहै, तोआपजेएनमेडिकलकॉलेजमेप्रोफेसर, पैथोलॉजीविभाग, प्रोफेसर, जेएनमेडिकलकॉलेजसस्थागतनैतिकअनुसधानसमिति, मानवसंसाधनअनुसंधान, फोननंबर 9448863676 परकॉलकरसकतेहै। ,बेलगाम।

वतेमानस्थिति

मैस्वेच्छासेनीचेहस्ताक्षरकरकेइसअध्ययनमेभागलेनेकेलिएसहमतहू।मैंकेसीभीसमयवापसले सकताहू।मैइसफॉमेपरहस्ताक्षरकरकेकोइकानूनीअधिकारनहीछोड़रहाहू।नीचेदियागयामेराह स्ताक्षरदशातहैकिमैनेपढ़ाहै, यायहमुझे, इसपूरीसहमतिकेरूपमेपढ़ागयाहै, औरमेरेसभीसवालोकेजवाबदिएहै।

अध्ययनकेदौरानयाभविष्यमेप्रश्नोकेमामलेमेआपनेम्नलिखितव्यक्तिसेसपकेकरसकतेहै।

सिद्धातअन्वेषक: मुख्यमत्री

गाइड:

प्रतिभागीकानाम: (हस्ताक्षर / अगूठेकानिशान)

साक्षीकानाम:

ತೃತೀಯಆರೈಕಆಸ್ಪತ್ರಯಲ್ಲಲವಲ್ 1 ಪಾಲಸೂನೂಗ್ರಫಯಿಂದಸ್ಲೀಪ್ಡಸಾರ್ಡಡ್ರ್ಮೌಲ್ಯಮಾಪನ:

ಒಂದುಹಾಸ್ಪಟಲ್ಆಧಾರತಆಬ್ಸರ್ವಾಟೋನಲ್ಅಧ್ಯಯನ

ಅಧ್ಯಯನದಉದ್ದೀಶ:

ಈಅಧ್ಯಯನದಲ್ಲಭಾಗವಹಸಲುನೀವುಅರ್ಹರಾಗರುವುದರಂದನಮ್ಮನ್ನುಅಧ್ಯಯನಕ್ಕಸೀರಸಲುಕೀಳಲಾ

ಗುತ್ತದ.ನದ್ರಾಹೀನತಯಉಸರಾಟವನ್ನುಹೂಂದರುವಎಲ್ಲಾರೂೀಗಗಳನ್ನುಈಅಧ್ಯಯನದಲ್ಲಸೀರಸಲಾ

ಗುತ್ತದ.ಈಅಧ್ಯಯನದಸಮಯದಲ್ಲ,

ರೂೀಗಗಳಗಅವರಪ್ರಸ್ತುತದೂರುಗಳಬಗ್ಗಪ್ರಶ್ನಗಳನ್ನುಕೀಳಲಾಗುತ್ತದಮತ್ತುಅವರುತಮ್ಮಜ್ಞಾನದಅತ್ಯುತ್ತ

ಮಉತ್ತರಗಳಗಉತ್ತರಸಬೀಕಾಗುತ್ತದ.ಅಧ್ಯಯನದಪ್ರಧಾನತನಖಾಧಕಾರಡ.ಆರ್.

(ಮಾರ್ಗದರ್ಶ) ಮಾರ್ಗದರ್ಶನದಲ್ಲ.

ಸ್ಲೀಪ್ಅಪ್ನಯಾದಚಹ್ನಗಳುಮತ್ತುರೂೀಗಲಕ್ಷಣಗಳೂಂದಗಬಪಡಗಬರುವರೂೀಗಗಳಲ್ಲವವಧಸ್ಲೀಪ್ಡಸಾ

ರ್ಡರ್ಡ್ಉಸರಾಟದಮಾದರಗಳನ್ನುಅಧ್ಯಯನಮಾಡುವುದುಮತ್ತುಸ್ಲೀಪ್ಅಪ್

ನತೀವ್ರತಯನ್ನುಬಎಂಐನೂಂದಗಪರಸ್ಪರಸಂಬಂಧಸುವುದುಈಅಧ್ಯಯನದಉದ್ದೀಶವಾಗದ.

ಅಬ್ಸ್ಟ್ರಕ್ಟವ್ಸ್ಲೀಪ್ಅಪ್ನಯಾಸಂಡ್ರೂೀಮ್ (ಒಎಸ್ಎಎ) ಬೂಜ್ಜುಸಂಬಂಧತಸಹ-

ಅಸ್ವಸ್ಥಕಾಯಿಲಗಳಾದಟೈಪ್ 2 ಡಯಾಬಟಸ್ಮಲ್ಲಟಸ್, ಪರಧಮನಯಕಾಯಿಲ, ರಕ್ತಕಟ್ಟಹೃದಯಸ್ಥಂಭನ,

ಅಧಕರಕ್ತದೂತ್ತ , ದೀರ್ಘಕಾಲದಮೂತ್ರಪಂಡಕಾಯಿಲ,

ಡಸ್ಲಪಡಮಯಾಸ್ಮತ್ತುಮಟಾಬಾಲಕ್ಸಂಡ್ರೂೀಮ್ಅನ್ನುಭಾರತೀಯಜನಸಂಖ್ಯಗಅನ್ವಯಿಸ .

ಭಾರತದಂತಹಸಂಪನ್ಮೂಲಸೀಮತಸಟ್ಟಂಗ್, ಮಲ್ಟ-

ಚಾನಲ್ಬಾಲಸೂಮ್ನೋಗ್ರಫಿಉಪಕರಣಗಳೂಂದಿಗಆರೋಗ್ಯಸೌಲಭ್ಯಗಳಕೊರತೆಯಿದ,

ಇದರಿಂದಾಗಿಒಎಸ್ಎಎಸ್ಸೂೀಗಗಳಮೌಲ್ಯಮಾಪನವನ್ನುಮತ್ತಷ್ಟುಸೀಮಿತಗೂಳಿಸುತ್ತದ.

ನಿರ್ಬಂಧಗಳನಡುವಯೂ,

ಕಳದದಶಕದಲ್ಲಭಾರತಮತ್ತುಅದರನರಯರಾಷ್ಟ್ರಗಳಸಂಶೂೀಧಕರುಒಎಸ್ಎಎಸ್ಮತ್ತುಸಹ-

ಅಸ್ವಸ್ಥತೆಗಳನ್ನುಅಧ್ಯಯನಮಾಡಲುಪ್ರಾಥಮಿಕಪ್ರಯತ್ನವನ್ನುಮಾಡದ್ದಾರ.

ವಿಧಾನ :

ಡಾ.ಕವಲ್ಇಯಪ್ರಭಾಕರ್ೋರಚಾರಟೀಬಲ್ಆಸ್ಪತ್ರಮತ್ತುಎಂಆರ್ಸಿಯಹೂರರೂೀಗಚಕತ್ಸಾಲಯಕ್ಕ

ಹಾಜರಾಗುವನಿದ್ರಾಹೀನತಯಉಸಿರಾಟದಲಕ್ಷಣಗಳುಕಂಡುಬರುವರೂೀಗಗಳಗವಿವರವಾದಇತಿಹಾಸತ

ಗದುಕೂಳ್ಳುವಿಕ, ಕ್ಲಿನಿಕಲ್ಪರೀಕ್ಷಯನಂತರಹಂತ 1 ಪಾಲಸೂಮ್ನೋಗ್ರಫಿಅಧ್ಯಯನ

ಪಾಲಸೂಮ್ನೋಗ್ರಫಿಅಧ್ಯಯನವುಶಾಂತವಾದ, ಗಾ dark ವಾದ,

ತಾಪಮಾನನಿಯಂತ್ರತಕೂೀಣಯಲ್ಲನಿರಂತರಮೀಲ್ವಚಾರಣಯೂಂದಿಗನಡಯುತ್ತದ.ಅಧ್ಯಯನದಸಮ

ಯದಲ್ಲದಾಖಲಾದವಿವಿಧಅಸ್ಥಿರಗಳಲ್ಲಇಸಿಜಿ, ಸಂಪ್ರಲ್ಮತ್ತುಆಕ್ಸಿಪಿಟಲ್ಇಇಜಿ, ಇಒಜಿ,

ಸಬ್ಮೆಂಟಲಸೌಇಎಂಜಿ, ಮೂಗನಮತ್ತುಮೌಖಿಕಗಾಳಯಹರವು, ಅಪಧಮನಿಯಆಮ್ಲಜನಕಶುದ್ಧತ್ವ,

ಥರ್ಮಿಸ್ಟರ್, ಕ್ಯಾನುಲಾ, ಥೂರಾಸಿಕ್ಗೂೀಡಯಚಲನಗಳುಕಬ್ಬೂಟ್ಟಯಚಲನಗಳು,

ಮುಂಭಾಗದಟಬಯಾಲಿಸೌಇಎಂ , ಗೂರಕಮತ್ತುದೇಹದಸ್ಥಾನ.

ನಿದ್ರಯಅಧ್ಯಯನವನ್ನುನಿದ್ರಯತಂತ್ರಜ್ಞರುರಾತ್ರ 10 ರಿಂದಮರುದನಬಳಗ್ಗ 6 ರವರಗನಡಸಲದ್ದಾರ.

ಸ್ಲೀಪ್ಸ್ಟೀರಿಂಗ್ಅನ್ನುಸ್ಲೀಪ್ತಂತ್ರಜ್ಞರುಮಾಡುತ್ತಾರ.

ಅಪಾಯಗಳುಮತ್ತುಪ್ರಯೋಜನಗಳು:

ಇದರಲ್ಲಿ ವುದೇಅಪಾಯಗಳುಅಥವಾಪ್ರಯೋಜನಗಳಿಲ್ಲ.

ಪರ್ಯಾಯಗಳು:

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವುದುಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದ.ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸದಿರಲುನೀವುಆ

ಯ್ಕೆಮಾಡಬಹುದು, ಅಥವಾನೀವುಈಗಭಾಗವಹಿಸಲುನಿರ್ಧರಿಸಿದರ,

ನೀವುನಂತರನಿಮ್ಮಮನಸ್ಸನ್ನುಬದಲಾಯಿಸಬಹುದುಮತ್ತುಅಧ್ಯಯನದಿಂದಹಿಂದಸರಯಬಹುದು.ನಿಮ್ಮ

ನಿರ್ಧಾರವುಪ್ರಸ್ತುತಅಥವಾಭವಿಷ್ಯದಆರೋಗ್ಯರಕ್ಷಣಅಥವಾನೀವುಸ್ವೀಕರಿಸುವಇತರಸೇವಗಳನ್ನುಬದ

ಲಾಯಿಸುವುದಿಲ್ಲ.

ಅಧ್ಯಯನದವೈದ್ಯರುಅಥವಾಪ್ರಾಯೋಜಕರುಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಭಾಗವಹಿಸುವಿಕಯನ್ನುಯಾವುದೇ

ಸಮಯದಲ್ಲಿಕೂನಗೂಳಿಸಬಹುದು.

ಗೌಪ್ಯತಮತ್ತುಗೌಪ್ಯತ:

ಈಅಧ್ಯಯನದಸಮಯದಲ್ಲಿನಿಮ್ಮಬಗ್ಗಸಂಗ್ರಹಿಸಲಾದಎಲ್ಲಾಮಾಹಿತಿಯನ್ನುಕಾನೂನಿನಿಂದಅನುಮತಿಸು

ವಮಟ್ಟಿಗಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದ.ಈಸಂಶೋಧನಾದಾಖಲಿಯಲ್ಲಿಕೋಡ್ಸಂಖ್ಯೆಗಳುನಿಮ್ಮನ್ನುಗುರುತಿಸುತ್ತ

ವ.ಈಅಧ್ಯಯನದಮಾಹಿತಿಯನ್ನುಪ್ರಕಟಿಸಲಾಗುವುದುಆದರಯಾವುದೇಪ್ರಕಟಣಿಯಲ್ಲಿನಿಮ್ಮಗುರುತುಗೌ

ಪ್ಯವಾಗಿರುತ್ತದ.ನಿಮ್ಮಬಗ್ಗಯಾವುದೇಮಾಹಿತಿಅಥವಾಸಂಶೋಧನಿಯಸಮಯದಲ್ಲಿನೀವುಒದಗಿಸಿದಮಾ

ಹಿತಿಯನ್ನುನಿಮ್ಮಲಿಖಿತಅನುಮತಿಯಿಲ್ಲದಇತರರಿಗಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ:

1. ನಿಮ್ಮಹಕ್ಕುಗಳುಮತ್ತುಕಲ್ಯಾಣವನ್ನುರಕ್ಷಿಸಲುತುರ್ತುಪರಿಸ್ಥಿತಿಯಲ್ಲಿ.

2. ಕಾನೂನಿನಪ್ರಕಾರಅಗತ್ಯವಿದ್ದರ.

ಸಂಸ್ಥ / ಪ್ರಾಯೋಜಕರನೀತ:

ಈಸಂಶೋಧನಗಿಅನ್ವಯಿಸುವುದಿಲ್ಲ.

ಭಾಗವಹಿಸುವಿಕಗಾಗಿಆರ್ಥಿಕಪ್ರೋತ್ಸಾಹ:

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಿಮಗಯಾವುದೇಉಡುಗೂರ / ಪ್ರೋತ್ಸಾಹಧನನೀಡಲಾಗುವುದಿಲ್ಲ.

ಫಲಿತಾಂಶಗಳನ್ನುಪ್ರಕಟಿಸಲುಅಧಿಕಾರ:

ಈಅಧ್ಯಯನದಫಲಿತಾಂಶಗಳನ್ನುಎಂಡಿಪದವಿ,

ವಿಮರ್ಶಮತ್ತುಪ್ರಕಟಣಪೂರ್ಣಗೊಳಿಸುವಅಗತ್ಯತೆಯಭಾಗವಾಗಿಬಲ್ಗಾಂನಕಲೆಳಿಇಶ್ವವಿದ್ಯಾಲಯಕ್ಕೆರ

ವಾನಿಸಲಾಗುವುದು ..

ಪ್ರಶ್ನೆಗಳು:

ನೀವುಅಧ್ಯಯನಕ್ಕೆಸಂಬಂಧಿಸಿದಯಾವುದೇಪ್ರಶ್ನೆಗಳನ್ನುಹೊಂದಿದ್ದರೆ,

ಭವಿಷ್ಯದಲ್ಲಿಅಥವಾಅಧ್ಯಯನಕ್ಕೆಸಂಬಂಧಿಸಿದಗಾಯಅಥವಾಅನಾರೋಗ್ಯದಸಂದರ್ಭದಲ್ಲಿ,

ನೀವುಸಂಪರ್ಕಿಸಬಹುದು:

ಉಸಿರಾಟದ ine ಷಧವಿಭಾಗ, ಕವಿಲೆಳಿಇವಿಸೌಆಸ್ಪತ್ರೆಮತ್ತುಎಂಆಿರ್ಸಿ,

- ಡಾ.ಗಜಾನನೌಎ: .ಗೌಡ, ಪ್ರಾಧ್ಯಾಪಕಮತ್ತುಮುಖ್ಯಸ್ಥ, ಪಲ್ಮನರಮಡಿಸಿನ್ವಿಭಾಗ, ಕವಿಲೆಳಿಇವಿಸೌಆಸ್ಪತ್ರೆಮತ್ತುಎಂಆಿರ್ಸಿ, ಬಲ್ಗಾಂಪಿಎಚ್

Rights ಅಧ್ಯಯನದವಿಷಯವಾಗಿನಮ್ಮಹಕ್ಕುಗಳಬಗ್ಗೆನೀವುಯಾವುದೇಪ್ರಶ್ನೆಗಳನ್ನುಹೊಂದಿದ್ದರೆ,

ನೀವುಜವಿನ್ಯೆದ್ಯಕೀಯಕಾಲೇಜಿನಲ್ಲಿರೋಗಶಾಸ್ತ್ರವಿಭಾಗದಪ್ರಾಧ್ಯಾಪಕ,

ಜವಿನ್ಯೆದ್ಯಕೀಯಕಾಲೇಜುಸಾಂಸ್ಥಿಕನೈತಿಕಸಮಿತಿಯಅಧ್ಯಕ್ಷರು, ಫೋನ್ಸಂಖ್ಯೆಗಳಿಗೆರಮಾಡಬಹುದು. ,

ಬಲ್ಗಾಂ.

ಕನ್ಸೆಂಟ್ಸ್ಟೇಟ್ಮೆಂಟ್

ಕಳಗಿಸಹಿಮಾಡುವಮೂಲಕಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಾನುಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದಒಪ್ಪುತ್ತೇನೆ.

ನಾನುಯಾವುದೇಸಮಯದಲ್ಲಿಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.ಈಫಾರ್ಮ್ಗೆಸಹಿಮಾಡುವಮೂಲಕನಾನುಯಾವ

ದೇಕಾನೂನುಹಕ್ಕುಗಳನ್ನುಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ.

ಕಳಗಿನನನ್ನಸಹಿನಾನುಒದಿದ್ದೇನೆಅಥವಾಈಸಂಪೂರ್ಣಒಪ್ಪಿಗೆಯರೂಪವನ್ನುನನಗೆಒದಿದ್ದೇನೆಮತ್ತುನನ್ನ

ಎಲ್ಲಾಪ್ರಶ್ನೆಗಳಿಗೆಉತ್ತರಿಸಿದಎಂದುಸೂಚಿಸಿ .

ಅಧ್ಯಯನದಸಮಯದಲ್ಲಿಅಥವಾಭವಿಷ್ಯದಲ್ಲಿನೀವುಈಕಳಗಿನವ್ಯಕ್ತಿಯನ್ನುಸಂಪರ್ಕಿಸಬಹುದು.

ತತ್ವತನಿಖಾಧಿಕಾರ:

ಮಾರ್ಗದರ್ಶಿ:

ಭಾಗವಹಿಸುವವರಹಸರು: (ಸಹಿ / ಹಬ್ಬರಳುಮುದ್ರಣ)

ಸಾಕ್ಷಿಯಹಸರು:

ANNEXURE 1- INFORMED CONSENT

EVALUATION OF SLEEP DISORDERED BREATHING BY LEVEL 1 POLYSONOGRAPHY IN A TERTIARY CARE HOSPITAL: A HOSPITAL BASED OBSERVATONAL STUDY

Purpose of the study:

You are being asked to enroll in the study as you are eligible for participation in this study. All patients who are supected to have sleep disordered breathing will be included in this study. During this study, patients will be asked questions regarding their presenting complaints and they are supposed to answer to the best of their knowledge. The principal investigator of the study is DR.ANUSHA CM
under the guidance of DR.GAJANAN.S.GAUDE (guide).

The purpose of this study is to study various Sleep disordered breathing patterns in patients who come to OPD with signs and symptoms of the sleep apnea and to correlate the severity of sleep apnea syndrome with BMI . Obstructive sleep apnoea syndrome (OSAS) has been associated with obesity related co-morbid diseases like type 2 diabetes mellitus, coronary artery disease, congestive heart failure, hypertension, chronic kidney disease, dyslipidaemias and metabolic syndrome as applicable to the Indian population.In a resource limited setting like India, there isa paucity of health care facilities with multi-channel polysomnography equipment, thereby further limiting the evaluation of OSAS patients. In spite of the constraints, researchers in India and its neighbouring countries in the last decade have made a preliminary attempt to study OSAS and co-morbid conditions.

Procedure :

Patients whose symptoms are suggestive of sleep disordered breathing attending the outpatient clinic of Dr KLE's Prabhakar Kore Charitable Hospital and MRC will be subjected to Detailed history taking, Clinical examination followed by Level 1 polysomnography study

Polysomnography study takes place in a quiet, dark ,temperature controlled room with constant monitoring. The various variables recorded during the study will include ECG, Central and occipital EEG, EOG, Submentalis EMG, Nasal and oral air flow, Arterial Oxygen saturation , breathing pattern by Thermister, Cannula, Thoracic wall movements abdominal movements, Anterior Tibialis EMG, snoring and body position. Sleep study will be conducted by sleep technician from 10pm to to 6am next day. Sleep scoring is done by sleep technician.

Risks and benefits:

There are no risks or benefits involved.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part now,you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may terminate your participation in this study anytime.

Privacy and confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study will be published but your identity will be confidential in any publication.No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Institution/sponsor's policy:

Does not apply to this research.

Financial incentivesfor participation:

You will not be paid /offered any gift/incentives for participating in this study.

Authorization to publish results:

The results of this study would be forwarded to the KLE University, Belgaum as a part of requirement towards the completion of MD degree, review and publishing..

Questions:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact:

- Dr.Anusha CM , Department of Respiratory Medicine, KLES Hospital and MRC, Ph. No. 0831-2551376 or phone number: 8722810383
- Dr.Gajanan.S.Gaude, Professor and Head, Department of Pulmonary Medicine, KLES Hospital and MRC,Belagavi. Ph.: 0831-2551376
- If you have any queries about your rights as a study subject, you may call Dr. Ganga Pilli, Professor, Department of Pathology, Chairman of J. N. Medical College Institutional Ethical Committee of Human Subjects Research, Phone No. 9448863866, at J. N. Medical College, Belagavi.

CONSENT STATEMENT

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

In case of the queries during the study or in future you may contact following person.

Principle investigator: DR.ANUSHA CM

Guide **:** DR.GAJANAN.S.GAUDE

Name of the participant: (signature/thumb print)

Name of the witness: (signature)

Name of the investigator: (signature)

Date: Place:

Address:

Phone no:

एकवाहकदेखभालअस्पतालमे 1

पॉलीसोनोग्रैफेद्वारालेटेहुएसोजानेकोघटनाकामूल्याकन:

एकांचोंकेत्सीयआधारितबाहरीअध्ययन

अध्ययनकाउद्देश्य:

आपकोइसअध्ययनमेभागलेनेकोंलेएकहाजारहाहैक्योंकेआपइसअध्ययनमेभागलेनेकोंलेएपात्रहैं।जिनरोंगेयोकोनींदमेखललपडected ेकोआशकाहै,

उन्हेइसअध्ययनमेशांमेलांकेयाजाएगा।इसअध्ययनकेदौरान,

मरीजोसेउनकोवतेमानांशेकायतोकेबारेमेसवालपूछेजाएगेऔरउन्हेअपनेज्ञानकासबसेअच्छाजवाबदेनाचांहिए।अध्ययनकेमुख्यअन्वेषक DR.ANUSHA CM हैं

DR.GAJANAN.S.GAUDE (गाइड) केमागेदशेनमे।

इसअध्ययनकाउद्देश्यरोंगेयोमेनींदांवेकारकोंवोंभिन्नप्रकारोकाअध्ययनकरनाहै,

जोओपीडीमेस्लीपएपांनेयाकेलक्षणोऔरलक्षणोकेसाथआतेहैऔरबीएमआइकेसाथस्लीपएपांनेयांसेड्रोमकोगभीरताकोसहतेहैं।ऑब्सट्रांक्टिवस्लीपएपांनेयांसेड्रोम (OSAS) मोटापेसेसबांधितसह-रुग्णरोगोजैसोंकेटाइप 2 मधुमेह, कोरोनरीधमनीरोग, ंदिलकोंवेफलता, उच्चरक्तचाप, क्रोंनेकांकेडनीरोग, ंडिस्लिप्लोंमेयाऔरमेटाबोंलेकांसेड्रोमसेसबांधितहैजोभारतीयजनसख्यापरलागूहोताहै।भारतजैसेससाधनसींमेतसोंटेग, मल्टी-चैनलपॉलीसोम्नोग्राफीउपकरणकेसाथस्वास्थ्यदेखभालसुंवेधाओकोंएकसमताहै, जिससे OSAS रोंगेयोकेमूल्याकनकोसींमेतांकेयाजासकताहै।बाधाओकेबावजूद, ंपिछलेएकदशकमेभारतऔरइसकेपड़ोसीदेशोकेशोधकतोओनेओएसएएसऔरसह-रुग्णपांरोंस्थांतेयोकाअध्ययनकरनेकोंलेएप्रांरांभिकप्रयासांकेयाहै।

प्रक्रिया:

जिनमरीजोकेलक्षणनींदकीगड़बड़ीकेसकेतहोतेहैं,

वेडॉ।केएलइकेप्रभाकरकोरेचौरेटेबलअस्पतालऔरएमआरसीकेबाह्यरोगीक्लिनेकमेजातेहैं,

उन्हेंवेस्तृतइतिहासजाचकेअधीनकियाजाएगा, क्लिनेकलपरीक्षाकेबादलेवल 1

पॉलीसोमोग्राफीअध्ययन

पॉलीसोम्नोग्राफीअध्ययनरातरानेगरानीकेसाथएकशात, अंधेरे,

तापमानरेयात्रेतकमरेमेहोताहै।अध्ययनकेदौरानदजॉकेएगएवॉभेन्नचरमेइसीजी,

सेट्रलऔरओसीसीपटलइइजी, ईओजी, सबमेटालेसइएमजी, नाकऔरमौंखेकवायुप्रवाह,

धमनीऑक्सीजनसतृप्ति, थॉमेस्टर, कैनुला, श्वाससबधीपेटकेआदोलनोद्वारापेटकेआदोलनो,

पूवेकालाटेंबेआलेसइएमजी,

खरोटेऔरशामिलहोगे।शरीरकीस्थिति।स्लीपटेक्नीशियनद्वारास्लीपटेक्नीशियनद्वाराअगले

दिनरात 10 बजेसेसुबह 6

बजेतकआयोजितकियाजाएगा।स्लीपस्कोरिंगस्लीपटेक्नीशियनद्वाराकियाजाताहै।

जोखिमऔरलाभ:

इसमेकोइजोखिमयालाभशामिलनहोहै।

विकल्प:

इसअध्ययनमेभागलेनास्वैच्छिकहै।आपइसअध्ययनमेभागनहोलेनेकाचयनकरसकतेहैं,

यादिआपअभीभागलेनेकानिणेयलेतेहैं,

तोआपबादमेअपनाविचारबदलसकतेहैऔरअध्ययनसेहटसकतेहैं।आपकानिणेयवतेमानयाभावे

ष्यकोस्वास्थ्यदेखभालयाआपकेद्वाराप्राप्तअन्यसेवाओकोनहोबदलेगा।अध्ययनकेचोकेत्सक याप्रायोजककभीभीइसअध्ययनमेआपकोभागीदारीकोसमाप्तकरसकतेहैं।

गोपनीयताऔरगोपनीयता:

इसअध्ययनकेदौरानआपकेबारेमेएकत्रकोगइेसभीजानकारीकोकानूनद्वाराअनुमतसीमातकगो पनीयरखाजाएगा।कोडनबरइसशोधरेकॉडेमेआपकोपहचानकरेगे।इसअध्ययनसेसूचनाप्रकाशे तकोजाएगी,

लेकेनआपकोपहचानकेसीभीप्रकाशनमेगोपनीयहोगी।अनुसधानकेदौरानआपकेद्वारादोगइे जानकारीयाआपकेबारेमेजानकारीकेअलावाआपकोलेखेतअनुमतिकेबिनाअन्यकेलेएखुलासा कियाजाएगा:

1. अपनेअधिकारोऔरकल्याणकोरक्षाकेलेएआपातकालमे।

2. यदिकानूनद्वाराआवश्यकहो।

सस्थान / प्रायोजककोनीति:

इसशोधपरलागूनहोहोताहै।

वित्तीयप्रोत्साहनभागीदारी:

इसअध्ययनमेभागलेनेकेलेएआपकोकोइउपहार / प्रोत्साहननहीदियाजाएगा।

परिणामप्रकाशितकरनेकेलेएप्राधिकरण:

इसअध्ययनकेपरिणामएमडीडिग्री,

समीक्षाऔरप्रकाशनकेपूराहोनेकीआवश्यकताकेएकभागकेरूपमेकेएलइीवेश्वविद्यालय,

बेलगामकोभेजेजाएंगे।

प्रश्न:

यदिआपकेपासअध्ययनसेसबंधितकोइप्रश्नहै,

तोभविष्यमेयाअध्ययनसेसबंधितचोटयाबीमारीकेमामलेमे, आपसपकेकरसकतेहैं:

• अनुशासीएम, रेंस्पिरेटरीमेडिसिनविभाग, केएलइएसअस्पतालऔरएमआरसी, पीएच।नं। 0831-2551376 याफोननंबर: 8722810383

• अंजानजी.एस.गुड, प्रोफेसरऔरप्रमुख, पल्मोनरीमेडिसिनविभाग, KLES अस्पतालऔर MRC, बेलगाम Ph।: 0831-2551376

• यदिआपकेपासअध्ययनविषयकेरूपमेआपकेअधिकारोकेबारेमेकोइप्रश्नहैं, तोआपजेएनमेडिकलकॉलेजमेप्रोफेसर, पैथोलॉजीविभाग, प्रोफेसर, जेएनमेडिकलकॉलेजसस्थागतनैतिकअनुसधानसमिति, मानवसंसाधनअनुसंधान, फोननंबर 9448863676 परकॉलकरसकतेहैं। , बेलगाम।

वतेमानास्थिति

मैंस्वेच्छासेनीचेहस्ताक्षरकरकेइसअध्ययनमेभागलेनेकेलिएसहमतहू।मैंकेसीभीसमयवापसले सकताहू।मैंइसफॉमेपरहस्ताक्षरकरकेकोइकानूनीअधिकारनहीछोड़रहाहू।नीचेदियागयामेराह स्ताक्षरदशाताहैंकेमैनेपढ़ाहै, यायहमुझे, इसपूरीसहमतिकेरूपमेपढ़ागयाहै, औरमेरेसभीसवालोकेजवाबदिएहै।

अध्ययनकेदौरानयाभविष्यमेप्रश्नोकेमामलेमेआपनेम्नलिखितव्यक्तिसेसपकेकरसकतेहैं।

सिद्धातअन्वेषक: DR.ANUSHA मुख्यमत्री

गाइड: DR.GAJANAN.S.GAUDE

प्रतिभागीकानाम: (हस्ताक्षर / अगूठेकानिशान)

साक्षीकानाम:

ತೃತೀಯಆರೃಕಆಸ್ಪತ್ರಯಲ್ಲಲವಲ್ 1 ಪಾಲಸೂನೂಗ್ರಫಿಯಿಂದಸ್ಲೀಪ್ಡಸಾರ್ಡರ್ಡ್ಯಲ್ಯಮಾಪನ:

ಒಂದುಹಾಸ್ಪಟಲ್ಆಧಾರತಆಬ್ಸರ್ವಾಟೂನಲ್ಅಧ್ಯಯನ

ಅಧ್ಯಯನದಉದ್ದೇಶ:

ಈಅಧ್ಯಯನದಲ್ಲಭಾಗವಹಿಸಲುನೀವುಅರ್ಹರಾಗಿರುವುದರಿಂದನಮ್ಮನ್ನುಅಧ್ಯಯನಕ್ಕಸೇರಿಸಲುಕೇಳಲಾ

ಗುತ್ತದ.

ನದ್ರಾಹೀನತಯಿಉಸರಾಟವನ್ನುಹೂಂದಿರುವಎಲ್ಲಾರೋಗಿಗಳನ್ನುಈಅಧ್ಯಯನದಲ್ಲಸೇರಿಸಲಾಗುತ್ತದ.

ಈಅಧ್ಯಯನದಸಮಯದಲ್ಲ,

ರೋಗಿಗಳಿಗಅವರಪ್ರಸ್ತುತದೂರುಗಳಬಗ್ಗಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳಲಾಗುತ್ತದಮತ್ತುಅವರುತಮ್ಮಜ್ಞಾನದಅತ್ಯುತ್ತ

ಮಉತ್ತರಗಳಿಗಉತ್ತರಿಸಬೇಕಾಗುತ್ತದ. ಅಧ್ಯಯನದಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿಡಿ.ಆರ್.ಅನುಶಾಸ.ಎಂ.

DR.GAJANAN.S.GAUDE (ಮಾರ್ಗದರ್ಶಿ) ಮಾರ್ಗದರ್ಶನದಲ್ಲ.

ಸ್ಲೀಪ್ಅಪ್ನಿಯಾದಚಹ್ನಗಳುಮತ್ತುರೋಗಲಕ್ಷಣಗಳೂಂದಿಗಒಪಿಡಿಗಬರುವರೋಗಿಗಳಲ್ಲಿಎಷ್ಟಸ್ಲೀಪ್ಡಿಸಾ

ರ್ಡರ್ಡ್ಉಸಿರಾಟದಮಾದರಿಗಳನ್ನುಅಧ್ಯಯನಮಾಡುವುದುಮತ್ತುಸ್ಲೀಪ್ಅಪ್ನಿಯಾಸಿಂಡ್ರೋ

ನತೀವ್ರತಯನ್ನುಬಿಎಂಐನೂಂದಿಗಪರಸ್ಪರಸಂಬಂಧಿಸುವುದುಈಅಧ್ಯಯನದಉದ್ದೇಶವಾಗಿದ.

ಅಬ್ಸ್ಟ್ರಕ್ಟಿವ್ಸ್ಲೀಪ್ಅಪ್ನಿಯಾಸಿಂಡ್ರೋಮ್ (ಒಎಸ್ಎಎಸ್) ಬೂಜ್ಜುಸಂಬಂಧತಸಹ-

ಅಸ್ವಸ್ಥಕಾಯಿಲಗಳಾದಟೈಪ್ 2 ಡಯಾಬಿಟಸ್ಮೆಲ್ಲಿಟಸ್, ಪರಿಧಮನಿಯಕಾಯಿಲ, ರಕ್ತಕಟ್ಟಹೃದಯಸ್ತಂಭನ,

ಅಧಿಕರಕ್ತದೂತ್ತ , ದೀರ್ಘಕಾಲದಮೂತ್ರಪಿಂಡಕಾಯಿಲ,

ಡಸ್ಲಿಪಡಮಿಯಾಸ್ಮತ್ತುಮಟಾಬಾಲಕ್ಸಿಂಡ್ರೋಮ್ಅನ್ನುಭಾರತೀಯಜನಸಂಖ್ಯೆಗಅನ್ವಯಿಸುತ್ತದ.

ಭಾರತದಂತಹಸಂಪನ್ಮೂಲಸೀಮಿತಸಟ್ಟಿಂಗ್, ಮಲ್ಟಿ-

ಚಾನಲ್ಪಾಲಿಸೂಮ್ನೋಗ್ರಫಿಉಪಕರಣಗಳೂಂದಿಗಆರೋಗ್ಯಸೌಲಭ್ಯಗಳಕೂರತೆಯಿದ,

ಇದರಿಂದಾಗಿಒಎಸ್ಎಎಸ್ಗೂೀಗಗಳಮೌಲ್ಯಮಾಪನವನ್ನುಮತ್ತಷ್ಟುಸೀಮಿತಗೂಳಿಸುತ್ತದ.

ನಿರ್ಬಂಧಗಳನಡುವಯೂ,

ಕಳದದಶಕದಲ್ಲಿಭಾರತಮತ್ತುಅದರನರಯರಾಷ್ಟ್ರಗಳಸಂಶೂೀಧಕರುಒಎಸ್ಎಎಸ್ಮತ್ತುಸಹ-

ಅಸ್ವಸ್ಥತೆಗಳನ್ನುಅಧ್ಯಯನಮಾಡಲುಪ್ರಾಥಮಿಕಪ್ರಯತ್ನವನ್ನುಮಾಡಿದ್ದಾರ.

ವಿಧಾನ :

ಡಾ.

ಕವಿಲಾಇಯಿಪ್ರಭಾಕರ್ೋರಚಾರಟೀಬಲಾಆಸ್ಪತ್ರಿಮತ್ತುಎಂಆರ್ಸಿಯಿಹೂರರೂೀಗಚಕತ್ಸಾಲಯಿಕ್ಕಹಾಜ

ರಾಗುವನಿದ್ರಾಹೀನತಯಉಸಿರಾಟದಲಕ್ಷಣಗಳುಕಂಡುಬರುವರೂೀಗಗಳಗವಿವರವಾದಇತಿಹಾಸತಗದು

ಕೂಳ್ಳುವಿಕ, ಕ್ಲಿನಿಕಲ್ಪರೀಕ್ಷಯನಂತರಹಂತ 1 ಪಾಲಿಸೂಮ್ನೋಗ್ರಫಿಅಧ್ಯಯನ

ಪಾಲಿಸೂಮ್ನೋಗ್ರಫಿಅಧ್ಯಯನವುಶಾಂತವಾದ, ಗಾ dark ವಾದ,

ತಾಪಮಾನನಿಯಂತ್ರಿತಕೂೀಣಯಲ್ಲಿನಿರಂತರಮೇಲ್ವಿಚಾರಣಯೂಂದಿಗನಡಯುತ್ತದ.

ಅಧ್ಯಯನದಸಮಯದಲ್ಲಿದಾಖಲಾದವಿವಿಧಅಸ್ಥಿರಗಳಲ್ಲಿಇಸ , ಸಂಪ್ರಲ್ಮತ್ತುಆಕ್ಸಿಪಟಲಾಇಇಜ, ಇಒಜ,

ಸಬ್ಮಂಟಲಸೌಇಎಂಜ, ಮೂಗನಮತ್ತುಮೌಖಿಕಗಾಳಯಹರವು, ಅಪಧಮನಿಯಿಮ್ಲಜನಕಶುದ್ಧತ್ವ,

ಥರ್ಮಿಸ್ಟರ್, ಕ್ಯಾನುಲಾ, ಥೂರಾಸಿಕ್ಗೂೀಡಯಚಲನಗಳುಕಿಬ್ಬೂಟ್ಟಯಚಲನಗಳು,

ಮುಂಭಾಗದಟಬಯಾಲಸೌಇಎಂಜ, ಗೂರಕಮತ್ತುದೇಹದಸ್ಥಾನ.

ನಿದ್ರಯಅಧ್ಯಯನವನ್ನುನಿದ್ರಯತಂತ್ರಜ್ಞರುರಾತ್ರ 10 ರಿಂದಮರುದಿನಬಳಿಗ್ಗ 6 ರವರಗನಡಸಲದ್ದಾರ.

ಸ್ಲೀಪ್ಸ್ಟೀರಿಂಗ್ಅನ್ನುಸ್ಲೀಪ್ತಂತ್ರಜ್ಞರುಮಾಡುತ್ತಾರ.

ಅಪಾಯಗಳುಮತ್ತುಪ್ರಯೋಜನಗಳು:

ಇದರಲ್ಲಯಾವುದೇಅಪಾಯಗಳುಅಥವಾಪ್ರಯೋಜನಗಳಿಲ್ಲ.

ಪರ್ಯಾಯಗಳು:

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವುದುಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ.

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸದಿರಲುನೀವುಆಯ್ಕೆಮಾಡಬಹುದು,

ಅಥವಾನೀವುಈಗಭಾಗವಹಿಸಲುನಿರ್ಧರಿಸಿದ ,

ನೀವುನಂತರನಿಮ್ಮಮನಸ್ಸನ್ನುಬದಲಾಯಿಸಬಹುದುಮತ್ತುಅಧ್ಯಯನದಿಂದಹಿಂದಸರಿಯಬಹುದು.

ನಿಮ್ಮನಿರ್ಧಾರವುಪ್ರಸ್ತುತಅಥವಾಭವಿಷ್ಯದಆರೋಗ್ಯರಕ್ಷಣಅಥವಾನೀವುಸ್ವೀಕರಿಸುವಇತರಸೇವೆಗಳನ್ನು

ಬದಲಾಯಿಸುವುದಿಲ್ಲ.

ಅಧ್ಯಯನದವೈದ್ಯರುಅಥವಾಪ್ರಾಯೋಜಕರುಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಭಾಗವಹಿಸುವಿಕೆಯನ್ನುಯಾವುದೇ

ಸಮಯದಲ್ಲಿಕೂಡಗೊಳಿಸಬಹುದು.

ಗೌಪ್ಯತೆಮತ್ತುಗೌಪ್ಯತೆ:

ಈಅಧ್ಯಯನದಸಮಯದಲ್ಲಿನಿಮ್ಮಬಗ್ಗೆಸಂಗ್ರಹಿಸಲಾದಎಲ್ಲಾಮಾಹಿತಿಯನ್ನುಕಾನೂನಿನಿಂದಅನುಮತಿಸು

ವಮಟ್ಟಿಗೆಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ.

ಈಸಂಶೋಧನಾದಾಖಲೆಯಲ್ಲಿಕೋಡ್ಸಂಖ್ಯೆಗಳುನಿಮ್ಮನ್ನುಗುರುತಿಸುತ್ತವೆ.

ಈಅಧ್ಯಯನದಮಾಹಿತಿಯನ್ನುಪ್ರಕಟಿಸಲಾಗುವುದುಆದರೆಯಾವುದೇಪ್ರಕಟಣೆಯಲ್ಲಿನಿಮ್ಮಗುರುತುಗೌಪ್ಯ

ವಾಗಿರುತ್ತದೆ.

ನಿಮ್ಮಬಗ್ಗೆಯಾವುದೇಮಾಹಿತಿಅಥವಾಸಂಶೋಧನೆಯಸಮಯದಲ್ಲಿನೀವುಒದಗಿಸಿದಮಾಹಿತಿಯನ್ನುನಿ

ಮ್ಮಲಿಖಿತಅನುಮತಿಯಿಲ್ಲದಇತರರಿಗೆಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ:

1. ನಿಮ್ಮಹಕ್ಕುಗಳುಮತ್ತುಕಲ್ಯಾಣವನ್ನುರಕ್ಷಿಸಲುತುರ್ತುಪರಿಸ್ಥಿತಿಯಲ್ಲಿ.

2. ಕಾನೂನಿನಪ್ರಕಾರಅಗತ್ಯವಿದ್ದರೆ.

ಸಂಸ್ಥೆ / ಪ್ರಾಯೋಜಕರನೀತಿ:

ಈಸಂಶೋಧನೆಗೆಅನ್ವಯಿಸುವುದಿಲ್ಲ.

ಭಾಗವಹಿಸುವಿಕೆಗಾಗಿಆರ್ಥಿಕಪ್ರೋತ್ಸಾಹ:

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಿಮಗೆಯಾವುದೇಉಡುಗೊರ / ಪ್ರೋತ್ಸಾಹಧನನೀಡಲಾಗುವುದಿಲ್ಲ.

ಫಲಿತಾಂಶಗಳನ್ನುಪ್ರಕಟಿಸಲುಅಧಿಕಾರ:

ಈಅಧ್ಯಯನದಫಲಿತಾಂಶಗಳನ್ನುಎಂಡಿಪದವಿ,

ವಿಮರ್ಶೆಮತ್ತುಪ್ರಕಟಣೆಪೂರ್ಣಗೊಳಿಸುವಅಗತ್ಯತೆಯಿಭಾಗವಾಗಿಬಲ್ಗಾಂನಕಲೆಇವಿಶ್ವವಿದ್ಯಾಲಯಿಕ್ಕೆ

ವಾಪಸಿಲಾಗುವುದು ..

ಪ್ರಶ್ನೆಗಳು :

ನೀವುಅಧ್ಯಯನಕ್ಕೆಸಂಬಂಧಿಸಿದಯಾವುದೇಪ್ರಶ್ನೆಗಳನ್ನುಹೊಂದಿದ್ದರ,

ಭವಿಷ್ಯದಲ್ಲಿಅಥವಾಅಧ್ಯಯನಕ್ಕೆಸಂಬಂಧಿಸಿದಗಾಯಿಅಥವಾಅನಾರೋಗ್ಯದಸಂದರ್ಭದಲ್ಲಿ,

ನೀವುಸಂಪರ್ಕಿಸಬಹುದು:

• ಡಾ.ಅನುಷಾಸಿಎಂ, ಉಸಿರಾಟದ ine ಷಧವಿಭಾ , ಕಲೆಇಎಸ್ಆಸ್ಪತ್ರೆಮತ್ತುಎಂಆರ್ಸಿ, ಪಿಎಚ್ಸಿಂಖ್ಯೆ

0831-2551376 ಅಥವಾದೂರವಾಣಿಸಂಖ್ಯೆ: 8722810383

• ಡಾ.ಗಜಾನನ .ಗೌಡ, ಪ್ರಾಧ್ಯಾಪಕಮತ್ತುಮುಖ್ಯಸ್ಥ, ಪಲ್ಮನರಿಮೆಡಿಸಿನ್ವಿಭಾಗ,

ಕಲೆಇಎಸ್ಆಸ್ಪತ್ರೆಮತ್ತುಎಂಆರ್ಸಿ, ಬೆಲ್ಗಾಂಪಿಎಚ್: 0831-2551376

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ನೀವುಜಎನ್ವೈದ್ಯಕೀಯಕಾಲೇಜಿನಲ್ಲಿರೋಗಶಾಸ್ತ್ರವಿಭಾಗದಪ್ರಾಧ್ಯಾಪಕ,

ಜಎನ್ವೈದ್ಯಕೀಯಕಾಲೇಜುಸಾಂಸ್ಥಿಕನೈತಿಕಸಮಿತಿಯಅಧ್ಯಕ್ಷರು, ಫೋನ್ಸಂಖ್ಯೆ 9448863866

ಗೆಕರೆಮಾಡಬಹುದು. , ಬೆಲ್ಗಾಂ.

ಕನ್ಸೆಂಟ್ಸ್ಟೇಟ್ಮೆಂಟ್

ಕೆಳಗಸಹಿಮಾಡುವಮೂಲಕಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಾನುಸ್ವಯಿಂಪ್ರೇರಣೆಯಿಂದಒಪ್ಪುತ್ತೇನೆ.

ನಾನುಯಾವುದೇಸಮಯದಲ್ಲಿಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಈಫಾರ್ಮ್‌ಸಹಿಮಾಡುವಮೂಲಕನಾನುಯಾವುದೇಕಾನೂನುಹಕ್ಕುಗಳನ್ನುಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ.

ಕಳಗಿನನನ್ನಸಹಿನಾನುಒದಿದ್ದೇನಅಥವಾಈಸಂಪೂರ್ಣಒಪ್ಪಿಗಯರೂಪವನ್ನುನನಗಒದಿದ್ದೇನಮತ್ತುನನ್ನ

ಎಲ್ಲಾಪ್ರಶ್ನಗಳಿಗಉತ್ತರಿಸದಎಂದುಸೂಚಿಸುತ್ತದ.

ಅಧ್ಯಯನದಸಮಯದಲ್ಲಅಥವಾಭವಿಷ್ಯದಲ್ಲನೀವುಈಕಳಗಿನವ್ಯಕ್ತಿಯನ್ನುಸಂಪರ್ಕಿಸಬಹುದು.

ತತ್ವತನಿಖಾಧಿಕಾರಿ: ಡಿ.ಆರ್.ಅನುಶಾಸ.ಎಂ.

ಮಾರ್ಗದರ್ಶಿ: DR.GAJANAN.S.GAUDE

ಭಾಗವಹಿಸುವವರಹಸರು: (ಸಹಿ / ಹಬ್ಬರಳುಮುದ್ರಣ)

ಸಾಕ್ಷಿಯಹಸರು:

ANNEXURE II.ETHICAL CLEARANCE.

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Ref: MDC/DOME/ 57

Date: 22/11/2017

To,

PG student in Respiratory Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"EVALUATION OF SLEEP DISORDERED BREATHING BY LEVEL I POLYSONOGRAPHY IN A TERTIARY CARE HOSPITAL: A HOSPITAL BASED OBSERVATIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II- PROFORMA

NAME:

AGE: SEX:

OCCUPATION:

COMPLETE ADDRESS:

PHONE NO:

SOCIOECONOMIC STATUS:

HEIGHT: WEIGHT: BMI:

D0A: DOD:

PRESENTING COMPLAINTS AND DURATION:

HABITS IF ANY :

H/O OF ANY OTHER CO-MORBID CONDITIONS:

H/O OF ANY PAST ADMISSIONS:

TREATMENT HISTORY:

DIAGNOSIS:

EPWORTH SLEEPINESS SCALE

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just
tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected
you.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would **never** doze

1 = **slight chance** of dozing

2 = **moderate chance** of dozing

3 = **high chance** of dozing

It is important that you answer each question as best you can.

Situation	**Chance of Dozing (0-3)**
Sitting and reading _____	___
Watching TV _____	__
Sitting, inactive in a public place (e.g. a theatre or a meeting) _____	___
As a passenger in a car for an hour without a break _____	___
Lying down to rest in the afternoon when circumstances permit _____	___
Sitting and talking to someone _____	___
Sitting quietly after a lunch without alcohol _____	___
In a car, while stopped for a few minutes in the traffic _____	___

MALLAMPATTI SCORE :

- Class I: Soft palate, uvula, fauces, pillars visible.
- Class II: Soft palate, uvula, fauces visible.
- Class III: Soft palate, base of uvula visible.
- Class IV: Only hard palate visible

SLEEP STUDY REPORT :

APNEA HYPOAPNEA INDEX :

PERIODIC LIMB MOVEMENTS:

DIAGNOSIS : OSA- Obstructive/ Central/ Mixed

RLS- Present/absent

Severity of OSA

TREATMENT INITIATED :

CPAP
BIPAP

SIGNATURE OF INVESTIGATOR **SIGNATURE OF GUIDE**

ANNEXURE -III PHOTOGRAPHS

Figure 1 : Sleep technician inserting various leads

Figure 2 : Patient undergoing polysomnography

ANNEXURE V- KEY TO MASTER CHART

BMI- Body weight index

AHI- ApneaApnea Index

PLM – Periodic limb movement

REM – Rapid eye movement

NREM- Non Rapid eye movement

RDI- Respiratory desaturation index

CPAP- Continuous positive airway pressure

BiPAP- Bilevel positive airway pressure

SpO2- Peripheral capillary oxygen saturation

Chapter 1

Introduction

Chapter 2

Objectives

Chapter 3

Review of Literature

Chapter 4

Methodology

Chapter 5

Results

Chapter 6

Discussion

Chapter 7

Conclusion

Chapter 8

Summary

Chapter 9

Bibliography

Chapter 10

Annexures

MASTER CHART