PREVALENCE OF SLEEP RELATED BREATHING DISORDERS (SRBD) AND THE ASSESSMENT OF QUALITY OF SLEEP IN PATIENTS WITH CHRONIC HYPERCAPNIC RESPIRATORY FAILURE

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CERTIFICATE

This is to certify that the dissertation on **"PREVALENCE OF SLEEP RELATED BREATHING DISORDERS (SRBD) AND THE ASSESSMENT OF QUALITY OF SLEEP IN PATIENTS WITH CHRONIC HYPERCAPNIC RESPIRATORY FAILURE"** is a record of research work done by DR.K.RAJARAJAN in partial fulfilment for M.D. (PULMONARY MEDICINE) Examination of the Tamil Nadu, Dr.M. G .R. Medical University to be held in April 2015.The period of study is from December 2013 to July 2014.

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DECLARATION

I hereby declare that the dissertation entitled **"PREVALENCE OF SLEEP RELATED BREATHING DISORDERS (SRBD) AND THE ASSESSMENT OF QUALITY OF SLEEP IN PATIENTS WITH CHRONIC HYPERCAPNIC RESPIRATORY FAILURE"** submitted for the Degree of Doctor of Medicine in M.D., Degree Examination, Branch XVII, PULMONARY MEDICINE is my original work and the dissertation has not formed the basis for the award of any degree, diploma, associate ship, fellowship or similar other titles. It had not been submitted to any other university or Institution for the award of any degree or diploma.

Place: Chennai Signature of the Scholar

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CONTENTS

PREVALENCE OF SLEEP RELATED BREATHING DISORDERS AND THE ASSESSMENT OF QUALITY OF SLEEP IN PATIENTS WITH CHRONIC HYPERCAPNIC RESPIRATORY FAILURE

ABSTRACT

Background:

COPD will be the third leading cause of death by year 2020. In India, COPD and Post TuberculousSequelae are very common chronic respiratory diseases that have significant morbidity and mortality.Sleep related symptoms occur in about 40% of cases in patients with COPD. Sleep related breathing disorders constitute the greatest number of disorders of sleep in patients treated by sleep medicine, pulmonary, and general practitioners in the outpatient setting.

Aim :

1.To know the prevalence of sleep related breathing disorders (SRBD) in patients with Chronic Hypercapnic Respiratory Failure.

2.To assess the quality of sleep in patients with Chronic Hypercapnic Respiratory Failure.

Material and Methods:

Patients enrolled in the COPD registry at Government Hospital Of Thoracic Medicine Tambaram,with severe stable COPD or COPD with Pulmonary Tuberculosis Sequelaeare evaluated. Spirometry is done for those patients. Patients with FEV1< 40% by spirometry are included , Arterial Blood Gas analysis is done. Those with Chronic Hypercapnic Respiratory Failure are included in the study. Patients with similar degree of obstruction without Respiratory Failure are used as comparison group. Overnight Polysomnography was performed in those patients. Epworths Sleepiness Score and Pittsburg sleep quality index scoring is done. Data is analyzed by standard statistical methods.

Results :

FortyFive patients are enrolled into the study in total. Thirty two patients are Patients with ChronicHypercapnic Respiratory Failure (Group A). Thirteen patients had COPD or COPD with Pulmonary Tuberculosis sequelae and with similar degrees of airflow obstruction without Respiratory failure (Group B).The mean Age group is 57.2 vs 57.8 years. Significant Nocturnal Desaturation is seen in 68.8% of patients with Group A and 38.5% of patients in Group B. Snoring is present in 41% of Group A and 23.07% of Group B. Obstructive Sleep apnea is seen in 2 patients in Group A (6.25%). Sleep latency (in minutes) is 62.7 vs 42.4. arousal index is 31.1/hour vs 20.4/ hour,NREM1,2 (in Minutes) 208.1 vs 180.1, NREM3(Min) is 20.7 vs 33.9 ,REM (min) 34.7 vs 48.6. In Group A, 68.8 % of patients have significant nocturnal desaturation vs 38.5% in Group B . Mean Epworth Sleepiness score is 11.5vs 9.7, MeanPSQI score is 13.2vs 7.3

Conclusion :

Nocturnal Desaturation is seen in significant proportion of patients with Chronic Hypercapnic Respiratory Failure (68.8%).

There is good correlation between the Quality of sleep measurement by Pittsburgh Sleep Quality Index scoring and the sleep variables determined by polysomnography.

Patients with Chronic Hypercapnic Respiratory Failure have decreased Total Sleep Time, Increased Sleep Latency, Decreased Sleep Efficiency, Decreased NREM Stage 3 Sleep, Decreased REM Sleep, Increased Arousal, Increase in duration of Wake after Sleep Onset when compared to normal values of that age . Based on these variables it is concluded that Sleep Quality in patients with Chronic Hypercapnic Respiratory Failure is poor.

The prevalence of sleep related breathing disorders (SRBD) in patients with Chronic Hypercapnic Respiratory Failure is 6.25% which is similar to that general population.

KEY WORDS : Sleep related breathing disorders (SRBD), Quality of sleep Chronic hypercapnic respiratory failure, Polysomnography.

INTRODUCTION

Human Beings spend almost 30% of their lives in sleeping. But still much attention has not been paid to sleep disorders. Only from the 1970s, consequences of sleep disturbances produced by the abnormal breathing patterns, or Sleep Related Breathing Disorder (SRBD) are being recognized by the physicians.

Sleep related symptoms occur in about 40% of cases in patients with COPD[1]. SRBD is associated with considerable morbidity. Obstructive Sleep Apnea (OSA) should be considered in patients reporting daytime hyper somnolence irrespective of BMI or snoring history.

The control of respiration in patients with chronic respiratory disease follows similar basic principles as that of the normal subjects, both during awake state and sleep, but with an expected lower feedback response during sleep[2].

In patients with chronic respiratory disease this lower feedback response affects the nocturnal gas exchange and sleep quality especially in those patients with hypercapnia. The primary mechanisms of this include decreased ventilatory responsiveness to hypercapnia, decreased respiratory muscle output, and marked increases in the upper airway resistance[3].

Sleep hypoventilation (SH) may be a important factor in the development of hypercapnic respiratory failure in patients with chronic respiratory disease, particularly during rapid-eye-movement sleep, where marked respiratory muscle atonia occurs. This leads to increase in sleep disruption, arousal, pulmonary hypertension, and is associated with higher mortality[3].

In India, COPD and Pulmonary Tuberculous Sequelae are very common chronic respiratory diseases that have significant morbidity and mortality.

Many patients develop chronic respiratory failure and the quality of life is very much affected. Intervention at various levels are needed to improve the quality of life of those patients.

Sleep in those patients need to be paid more attention as the improvement in sleep quality will lead to improvement in the patients quality of life, as well as decreasing the morbidity.

Sleep Related Breathing Disorders and the structural diseases of Lung can progress to cause Pulmonary Hypertension and Corpulmonale. Both Disorders can coexist as well.

This study aims at evaluating the sleep quality and the prevalence of sleep related breathing disorders in those patients so that interventions could be done to improve the quality of life in those patients.

REVIEW OF LITERATURE

Definition of Sleep

Sleep is defined as period of bodily rest characterized by reduced awareness of the environment, a species-specific posture, and for most species, a particular sleep place[4].

Behavioural characteristics of the human sleep includes

Absence or marked reduction in movement

Decreased responsiveness to external stimuli (easy reversibility)

Recumbent body position

Closing of eyes

Slow and regular breathing pattern

The function of the sleep remains to be a subject of debate. Some authors have suggested that energy is being saved during sleep when an animal has nothing better to do.

According to some authors, sleep helps in the consolidation of memory and improved learning. As we know, one feels better or being restored after a good night's sleep; but how and what have been restored is a subject of debate.

It is postulated that sleep is not just as a behaviour (similar to feeding, which happens only during wakefulness), but as a state that could also sub serve multiple functions (just as the waking state does). The dramatic difference in physiological differences between NREM and REM sleep suggest this hypothesis[4].

Proposed Functions

Regulation of somatic growth (growth hormone release during NREM stages 3 and 4 sleep)

Neural growth and processing

Memory consolidation (REM sleep)

Thermoregulation and Energy conservation

Normal Sleep Requirements

The total duration of sleep required every day varies from individual to individual and age-related differences are present . Most adults sleep for about 6 to 9 hours (average of 8 hours) during a night.

Sleep duration for a period of less than 5 to 6 hours per day is usually associated with the symptoms of sleep deprivation[5].

Percentages of Sleep Stages in Healthy Adults[6]

Sleep stage	Percentage of Total sleep time
Stage 1 NREM	2–5%
Stage 2NREM	45–55%
Stage 3/4 NREM	5-20%
Stage REM	20–25%

A sleep cycle is the period from NREM (Non Rapid Eye Movement) stages 1 to 4 to REM (Rapid Eye Movement) sleep. There are usually three to five NREM-REM sleep cycles, each occurring about every 90 to 120 minutes in adults (every 60 minutes in infants and young children) during the night. Each sleep stage is not necessarily seen in every sleep cycle. The duration of NREM stages 3 and 4 sleep is greater during the initial part of sleep, whereas REM sleep is relatively more common during the latter part of sleep.

REM density (frequency of rapid eye movements during REM sleep) also increases during the latter portion of the night. Whereas NREM stages 3 and 4 sleep is related to the length of prior wakefulness

and to sleep onset, REM sleep is related to circadian rhythms of body temperature.

Arousal threshold is the lowest during NREM stage 1 sleep (easiest to awaken) and is highest during NREM stages 3 and 4 sleep (most difficult to awaken)[7].

STAGE : WAKE

Electroencephalography

Lower frequency activity when a person is relaxed

Prominent alpha (8–13 Hz) activity, when the person is drowsy and eyes are closed

Low-voltage, high-frequency activity when the person is alert and as eyes are open

Electro-oculography

Slow rolling eye movements occurs when a person is drowsy and as eyes are closed

Blinking or rapid eye movements occurs when the person is awake and alert.

Electromyography

There will be high chin muscle activity (ie, high chin EMG amplitude)

Associated features

Alpha activity is generally more prominent in the occipital leads compared to the central leads

Eye opening suppresses the alpha activity

EEG and EOG tracings will be demonstrate muscle artefacts when the person is tense.

Recording of occipital leads can help in the recognition of alpha waves as well as the timing of onset of sleep.

STAGE 1 SLEEP

Electroencephalography

Low-voltage, mixed-frequency activity.

Alpha activity occupies < 50% of the epoch.

Prominent theta activity; beta rhythms may be present

Absence of the sleep spindles or K-complexes.

Vertex sharp waves (high-amplitude, brief, negative deflections) will be present (more prominent in central leads).

Electro-oculography

No rapid eye movements.

Occasional slow rolling eye movements.

Electromyography

High chin muscle activity is seen (ie, high chin EMG amplitude) that is less than or equal to that during wakefulness

Associated features

Occurs at sleep onset or following arousals from sleep.

Person will be unresponsive but easily aroused.

Accounts to about 2% to 5% of total sleep time in an adult

Transitions into stage 2 sleep within a few minutes.

STAGE 2 SLEEP

Electroencephalography

Low-voltage, mixed-frequency activity.

Delta activity will occupy < 20% of the epoch

Presence of sleep spindles and K complexes (generally maximal over the vertex)

Electromyography

Low chin muscle activity.

Electro-oculography

No movements

Associated features

Accounts for the greatest proportion (45% to 55%) of total sleep time in adults. Three-minute stage 2 sleep scoring rule: Sleep spindles and K complexes are episodic and may not be present in every epoch. An epoch is scored as stage 2 sleep if the intervening period between sleep spindles or K complexes is less than 3 minutes, would otherwise meet criteria for stage 1 sleep (low-amplitude, mixed-frequency EEG), and is not associated with an arousal. An epoch is scored as stage 1 sleep if the intervening period is equal to or greater than 3 minutes.

STAGE 3 SLEEP

Electroencephalography

Sleep spindles may be present.

Delta activity will occupy between 20% and 50% of the epoch.

Electromyography

Low chin muscle activity (usually less than that in stages 1 and 2 sleep).

Electro-oculography

No movements

Associated features

Along with NREM stage 4 sleep, has the highest arousal threshold by external stimuli among the different sleep stages.

Accounts for about 10% of total sleep time in an adult.

Certain parasomnias (disorders of arousal such as sleep terrors or sleepwalking) usually occur during NREM stages 3 and 4 sleep. EEG amplitude of delta waves are increased .

STAGE 4 SLEEP

Electroencephalography

Sleep spindles may be present.

Delta activity occupies > 50% of the epoch.

Electro-oculography

No movements

Electromyography

Low chin muscle activity (generally less than that in stages 1 and 2 sleep)

Associated features

Accounts for about 10% of total sleep time in an adult.

Along with NREM stage 3 sleep, has the highest arousal threshold by external stimuli among the different sleep stages.

Certain parasomnias (disorders of arousal such as sleep terrors or sleepwalking) occur during stages 3 and 4 sleep.

Amount of NREM stages 3 and 4, and EEG amplitude of delta waves are increased among adolescents and reduced in older adults.

REM SLEEP

REM sleep is composed of two components, namely, **Tonic** (without rapid eye movements) and **Phasic** (with rapid eye movements) sleep.

Electroencephalography

Saw-tooth waves will be present (more prominent over vertex and the frontal leads).

Low-voltage, mixed-frequency activity (theta and beta rhythms)

Vertex sharp waves are not prominent.

Alpha waves are 1 to 2 Hz slower than those occurring during wakefulness and NREM stage 1 sleep.

Electro-oculography

Bursts of the conjugate and rapid eye movements.

No movements (tonic REM sleep)

Electromyography

Amplitude of chin EMG are reduced or absent (ie, at least equal to or, more commonly, lower than the lowest amplitude during NREM sleep).Loss of postural muscle tone due to postsynaptic hyperpolarization of the spinal motor neurons[7].

THE INTERNATIONAL CLASSIFICATION OF SLEEP DISORDERS-2 (2005) (ICSD-2) classified the sleep disorders into six major categories[8] :

I. **Sleep related breathing disorders**

II. Insomnia

III. Parasomnia

IV. Hypersomnias

V. Circadian rhythm sleep disorder

VI. Sleep related movement disorders

Types of sleep-related breathing disorders

Sleep-related breathing disorders are classified into four major types[9]

1.Central apnea syndromes

1. Primary Central Sleep apnoea
2. Periodic respiration of high altitude
3. Central sleep apneas without Cheyne-Stokes respiration secondary to other disorders (cardiac/renal disorders, malignant, vascular, degenerative or traumatic disorders of the central nervous system)
4. Central apneas due to medicine or other substances
5. Cheyne - Stokes respiration
6. Primary sleep apnea of the newborn

2. **Obstructive Sleep apnea syndromes**

1. Obstructive Sleep apnea in adults
2. Obstructive Sleep apnea in children

3. **Sleep associated Hypoventilation syndromes**

Hypoventilation/hypoxemia secondary to disorders of:

Lung (e.g. COPD), or

Vascular (e.g. Pulmonary Hypertension);

Neuromuscular ; Thoracic wall abnormalities;

Obesity- Hypoventilation Syndrome,

Idiopathic Non-obstructive Alveolar Hypoventilation

Congenital Central Hypoventilation.

4. Undefined/non-specific sleep disorders

(Disorders without specific characteristics and where further investigation is required to allow their classification into any of the previous categories)[9].

Obstructive sleep apnea[10]

The obstructive sleep apnea syndrome (OSAS) is present when the Apnea Hypopnea Index is greater than 5 to 10 events per hour and the patient have symptoms of excessive daytime somnolence, unrefreshing sleep, or chronic fatigue.

Central Sleep Apnea

Central sleep apnea (CSA) is less common than obstructive sleep apnea.

In Central sleep apnea there is temporary cessation of respiration. The Central command for the respiratory muscles are not present. It is characterised by repeated episodes of apnea without respiratory muscle effort .

Polysomnographic recording shows absence of thoraco abdominal excursion and nasal-oral airflow.

Criteria:

The individual must fulfill A, B, and C to be diagnosed with the central sleep apnea- hypopnea syndrome.

A. At least one of the following symptoms that is not explained by other factors:

Excessive daytime sleepiness

Frequent nocturnal arousals/awakenings

B. Overnight monitoring that demonstrates 5 to 10 or more central apneic events plus hypopneic events per hour of sleep.

C. Normocarbia while awake ($PaCO_2$ less than 45 mm Hg)[10].

Upper airway resistance syndrome (UARS) :

UARS represent a milder form of the OSA spectrum, although there is debate whether or not UARS patients demonstrate different clinical and upper airway characteristics compared with OSA patients.

UARS is not associated with apneas or significant oxyhemoglobin desaturations. The arousals result in sleep fragmentation and daytime sleepiness.

Nonetheless, many patients with the upper airway resistance syndrome also have evidence for concomitant obstructive sleep apnea

Risk Factors for Obstructive Sleep Apnea[10]

Gender (male/female 2:1)

Upper airway anatomy

Obesity (>120% ideal body weight)

Neck size (collar size >17 inches in males, >15 inches in females)

Lateral peritonsillar narrowing

Macroglossia

Elongation/enlargement of the soft palate

Narrowing of the hard palate

Tonsillar hypertrophy

Nasal septal deviation

Retrognathia, micrognathia

Class III/IV modified Mallampati airway

Specific genetic diseases, e.g., Treacher Collins, Downs syndrome, Apert's syndrome, Achrodorophsia, etc.

Genetic factors

Endocrine disorders—hypothyroidism, acromegaly

Alcohol, sedative or hypnotic use

Conditions in which Sleep Apnea Should be Suspected :

Systemic hypertension

Obesity

Myocardial infarction

Cerebrovascular accident

Pulmonary hypertension

Type II diabetes mellitus

Nocturnal cardiac arrhythmias

Driver involved in a sleep-related automobile crash

Preoperative anaesthesia evaluation

Consequenses of OSA

1. Day Time Sleepiness leading to automobile accidents , decreased quality of life.
2. Cardiovascular Diseases – Systemic Hypertension, Pulmonary Hypertension, Corpulmonale, Coronary Artery Disease, Congestive Cardiac Failure, Arrhythmia, Cerebrovascular Accidents
3. Diabetes Mellitus

Respiratory failure

Respiratory failure is defined by failure of the respiratory system in one or both of its gas-exchanging functions, oxygenation and carbon dioxide elimination from, pulmonary arterial blood[11].

Respiratory failure is divided into acute or chronic.

Distinctions between Acute and Chronic Respiratory Failure

Category	**Characteristic**
Hypercapnic respiratory Failure	$PaCO_2$>45 mmHg
Acute	Develops in minutes to hours
Chronic	Develops over several days or longer
Hypoxemic respiratory failure	PaO_2<55 mmHg when FIO2≥0.60
Acute	Develops in minutes to hours
Chronic	Develops over several days or longer

Respiratory failure is classified as hypercapnic or hypoxemic.

Hypercapnic respiratory failure is defined as an arterial $PaCO_2$ ($PaCO_2$) of greater than 45 mmHg.

Hypoxemic respiratory failure is defined as an arterial PaO_2 of less than 55 mmHg when the fraction of oxygen present in inspired air (FiO_2) is 0.60 or greater.

Hypercapnic and hypoxemic respiratory failure coexist in many patients . Disorders initially causing hypoxemia may be later complicated

by respiratory pump failure and hypercapnia. Similarly, diseases that produce respiratory pump failure can be complicated by hypoxemia due to secondary pulmonary parenchymal involvement.

Acute hypercapnic respiratory failure is defined by a $PaCO_2$ greater than 45 mmHg with accompanying academia (i.e. pH less than 7.30). The physiological effect of a sudden increase in $PaCO_2$ depends on the prevailing level of serum bicarbonate anion.

In patients with chronic hypercapnic respiratory failure there is a long-standing increase in $PaCO_2$ resulting in renal "compensation" causing increased serum bicarbonate concentration. A superimposed acute increase of $PaCO_2$ has a less clinical effect than does a comparable increase in patients with a normal bicarbonate level.

Respiratory failure can result due to an abnormality in any of the following "effector" components of the respiratory system— the central nervous system, peripheral nervous system, respiratory muscles, the chest wall, airways, or alveoli .

A defect in any of central nervous system, peripheral nervous system, respiratory muscles, the chest wall and airways, which constitute the "respiratory pump," may cause coexistent hypercapnia and

hypoxemia. The disorders of the alveoli are more apt to result in hypoxemia initially[11].

Ventilatory Supply vs Demand

The pathophysiology of hypercapnic respiratory failure is based on the relationship between ventilatory supply and ventilatory demand .

Ventilatory supply

The maximal spontaneous ventilation that can be maintained without development of respiratory muscle fatigue, which is also known as maximal sustainable ventilation (MSV).

Ventilatory demand is the spontaneous minute ventilation, when maintained results in a stable $PaCO_2$ (assuming a fixed rate of CO2 production).

Ventilatory supply normally greatly exceeds ventilatory demand. So, major changes in the minute ventilatory requirements (e.g., during exercise) usually occur without hypercapnia.

In patients with lung disease, significant abnormalities will be present before ventilatory demand encroaches upon MSV. As result, hypercapnia is a late finding.

As ventilatory demand exceeds MSV, $PaCO_2$ increases. The general rule is MSV is approximated as one-half the maximal voluntary ventilation. Therefore, normally there is a 10- to 15-fold difference between resting ventilatory demand and MSV.

Ventilatory pump accomplishes the bulk transfer of air to and from the alveoli. Hence, diseases that interfere with the mechanical properties of any component of the ventilator pump cause disturbance with CO_2 elimination and O_2 uptake. If disturbances in the function of the ventilatory pump are very severe, alveolar hypoventilation and respiratory acidosis may ensue.

Diseases causing Hypercapnic Respiratory Failure do so by derangement in respiratory mechanics and lung dead-space volume (e.g., Chronic Obstructive Pulmonary Disease [COPD], asthma, or kyphoscoliosis) or by impairment in the contractile properties of the respiratory muscles (e.g., neuromuscular disease).

Chemoreceptor-induced increases in inspiratory and expiratory muscle activity are directly proportional to the severity of abnormalities in arterial blood gas tensions and represent a feedback control loop that restores arterial blood gas tensions toward normal by enhancing alveolar ventilation.

The magnitude of the changes in intrathoracic pressure and resistance and compliance of the airways are determined by these changes in respiratory motor activity.

The maintenance of arterial blood gas tensions within a relatively narrow, normal range from new-born to senescence attests to the power of this homeostatic mechanism.

Chemosensitivity induced increases in the respiratory activity also affects the timing of respiratory motor activity and this is reflected in the duration of inspiration (Ti) and expiration (Te).

Hypoxia and hypercapnia lead to decrease in Ti and Te, resulting in the frequency of breathing to increase. Reductions in the Te are generally out of proportion to the reduction in Ti, thereby resulting in increase in the fraction of the respiratory cycle spent in inspiration.

The partitioning of the respiratory cycle is represented by the Ti/Tt ratio, where Tt is the total duration of respiratory cycle.(i.e., the sum of Ti and Te).

Postinspiratory inspiratory activity (PIIA)

The effects of Hypoxia and Hypercapnia on the activity of the inspiratory muscles after cessation of the inspiratory airflow ,so-called Postinspiratory inspiratory activity (PIIA) is different.

Hypoxia increases PIIA in both the chest wall inspiratory muscles and the muscles that constrict laryngeal aperture. Hence, hypoxia has a braking effect on the rate of expiratory airflow. The Te decreases with increasing hypoxic drive, resulting in increase in end-expiratory lung volume.

PIIA causes increase in lung volume, increase in the calibre of intrathoracic airways and increase in the O_2 content of the lung. PIIA due to hypoxia affects the load on the respiratory muscles in a complex fashion that is, PIIA due to hypoxia reduces inspiratory resistive work of breathing but causes increase in the inspiratory elastic and expiratory resistive work of breathing. The overall effect of hypoxia-induced PIIA is a reduction in the overall energy expenditure during breathing. In contrast to hypoxia, hypercapnia diminishes the duration of PIIA[12].

Ventilatory response to Hypercapnia

The ventilatory response to hypercapnia is determined by the prevailing level of PaO_2 and is increased as PaO_2 decreases. In fact, there is multiple interaction between the hypoxemic and hypercapnic stimuli to enhance the inspiratory and expiratory motor activity.

Worsening of hypoxemia enhances the hypercapnic ventilatory response in accordance with the O_2–CO_2 interaction. The strength of a patient's chemosensitivity to O_2 and CO_2 , particularly, to the O_2–CO_2 interaction is a powerful feedback mechanism opposing the retention of CO_2 in patients with ventilatory pump dysfunction. Hence , treatment of the hypercapnic, hypoxemic patient with supplemental O_2 may cause decrease in Vt/Ti and Ti/Tt,resulting in worsening of hypercapnia .

The rise of PaO_2 in hypoxic, hypercapnic subjects shifts the O_2 response to the right (less stimulus) predisposing to hypercapnic respiratory failure and shifts the ventilatory response to hypercapnia to the right .

A higher CO_2 stimulus is required to maintain ventilation at the baseline level in those patients. Accordingly, ventilation falls and the $PaCO_2$ rises.

The magnitude of the hypercapnia in patients with COPD in acute respiratory failure induced by supplemental O_2 varies widely among subjects as it is determined by their chemosensitivity.

Hypercapnia induced by supplemental O_2 in the patients with COPD is multifactorial and reflects increases in the lung dead-space volume as well as reductions in alveolar ventilation.

Hypoxia causes bronchoconstriction by increase in parasympathetic outflow to airway smooth muscle. Relief in hypoxemic stimulus causes bronchodilation and increased dead-space volume.

Role of Blunted Chemosensitivity in Development of Respiratory Failure

Hypoxemic and hypercapnic chemosensitivities are inherited and there are ethnic traits that vary widely. Sensitivity of respiratory chemoreceptors to both hypoxemia and hypercapnia declines with age.

This decline in chemosensitivity with aging may explain why elderly patients with lung disease (e.g., COPD) or chest wall disease (e.g.,

kyphoscoliosis) more frequently develop hypercapnic respiratory failure than the young adults.

When the chemosensitivity is low, patients with diseases of the ventilatory pump are predisposed to development of hypercapnic respiratory failure

The severity of airway obstruction required to cause CO2 retention in the patients with advanced COPD varies widely from subject to subject. Patients with a greatest respiratory effort response to the changes in $PaCO_2$— as measured by the diaphragm EMG, the respiratory work of breathing, or the occlusion pressure—will have $PaCO_2$ values closer to normal than the patients with blunted responses to CO_2 but with the same severity of lung dysfunction.

Hence, patients with diseases of the ventilatory pump with low chemosensitivity, are predisposed to the development of hypercapnic respiratory failure.

However, since hypercapnia per se may blunt the response to acute rise in $PaCO_2$, studies in patients with respiratory failure could not determine whether the blunted CO_2 responses are a cause or a consequence of respiratory failure.

The tendency for chemosensitivity to be inherited has been used in a number of subsequent studies to assess the role of hypoxic and hypercapnic responses in the pathogenesis of CO2 retention in the setting of obstructive lung disease.

Study of relatives with normal lung function and blood gases has been employed to circumvent the effects of CO2 retention on respiratory chemosensitivity in patients with COPD.

In general, normal relatives of hypercapnic patients with COPD have lower ventilatory responses to hypoxia and hypercapnia than relatives of eucapnic patients with COPD.

In hypercapnic patients with COPD, it appears that the blunted chemosensitivities to hypoxia and hypercapnia are premorbid ventilatory pump and, thereby, stimulate mechanoreceptors in the ventilatory pump.

The diseases of the airways (COPD and Asthma) or chest wall (Kyphoscoliosis) changes the resistance and compliance properties of the mechanoreceptor afferent inputs increase inspiratory neuromuscular output as reflected by the airway occlusion pressure in response to bronchoconstriction or external resistances or elastance.

The changes in ventilation during acute increase in airway resistance are inversely proportional to changes in occlusion pressure. Hence, the magnitude of motor response to increases in the respiratory load determines the ventilatory response.

These ventilator responses are eliminated by general anesthesia and reduced in stages III and IV and REM sleep.

The phasic and tonic inspiratory activity in the dilator muscles of the upper airway (e.g., posterior arytenoid, alaenasae, genioglossus), are increased in patients with hypercapnia and hypoxia.

These increase in the activity of dilator muscles of the upper airway causes decrease in the load on the chest wall pumping muscles by decreasing the resistance to inspiratory airflow .

The increased activity of these muscles further diminishes the susceptibility of upper airway to collapse as the inspiratory efforts become greater and sub pharyngeal pressure becomes more sub atmospheric.

Patients with COPD who are hyper inflated and dyspnoeic often assume a body posture that improves mechanical advantage of diaphragm, neck accessory, and pectoral girdle muscle.

The posture is forward flexion of the trunk, with extension of the head and neck and bracing of the pectoral girdle by rounding of the shoulders, holding of the thighs with the arms.

The net effect of this posture is increase in abdominal pressure (thereby increasing diaphragm precontraction length and the radius of curvature) providing a more favourable alignment of the scalene and sternomastoid muscles with the upper rib cage; and also anchor the pectoral girdle muscles, thereby allowing them to apply an inspiratory action on the rib cage.

With this posture, transdiaphragmatic pressure is increased and diaphragm and sternomastoid muscle EMG activity is decreased[12].

Sleep Related Ventilatory Changes in patients with COPD:

COPD patients with day time hypoxia have the oxygen saturation level in the steep portion of oxyhemoglobin dissociation curve. These patients have much greater fall in the oxygen saturation during night

Sleep Hypoventilation

During all sleep stages, particularly during REM sleep the response of the respiratory centre are reduced and the response of respiratory

muscle to respiratory centre outputs are also decreased, especially to those involving accessory muscles of respiration[13,14] .

In healthy individuals during REM sleep, ventilation may be decreased by 40% than during wakefulness. This is predominantly due to a reduction in tidal volume, and as a result of increase in the upper airway resistance and reduced inspiratory drive, that causes a slight decrease in arterial oxygen saturation (SaO_2) that is clinically not significant in normal subjects[15].

Even though the breathing pattern is same as the normal subjects, the hypoventilation during sleep is exaggerated in patients with COPD[16] .

Exaggerated physiological dead space in COPD, which results in greater alveolar hypoventilation during sleep contributes to the profound nocturnal hypoxemia than in normal subjects[17].

The decrease in the basal metabolic rate and ventilatory requirements during NREM sleep causes decrease in central respiratory drive.

During REM sleep, there is decrease in response of the respiratory centre to chemical and mechanical inputs[18].

Previous studies in patients with COPD have documented that a response of minute ventilation to rise in CO2 is lower even during wakefulness.

Increased Resistance of Airways

The loss of muscle tone in the upper airways causes increased resistance for breathing during sleep[19,20]. Due to the increase in the resistance there is altered response in ventilation to hypoxia which results in hypoventilation.

The Upper airway resistance in patients with COPD and normal subjects are similar but the airway dilation in response to hypercapnia is not seen in COPD patients. This contributes to the nocturnal desaturation in these patients[20].

During the normal circadian change in airway calibre there is mild bronchoconstriction during sleep. This bronchoconstriction may be increased during sleep in COPD. Therefore there is also an increased lower airway resistance[21].

Skeletal muscle tone is decreased during sleep. So the tone of intercostal muscles, tongue, pharyngeal muscles are reduced[22,23].

There is presynaptic inhibition of afferent terminals from muscle spindles and supraspinal inhibition of gamma- motor neurons and the during the REM sleep.

This inhibition results in decreased tone of intercostal muscles. The diaphragm is not affected by this because it is supplied by alpha motor neurons.

Also there is decreased number of muscle spindles in diaphragm. As COPD patients are much dependent on the accessory muscles they are more affected during the sleep due to these changes.

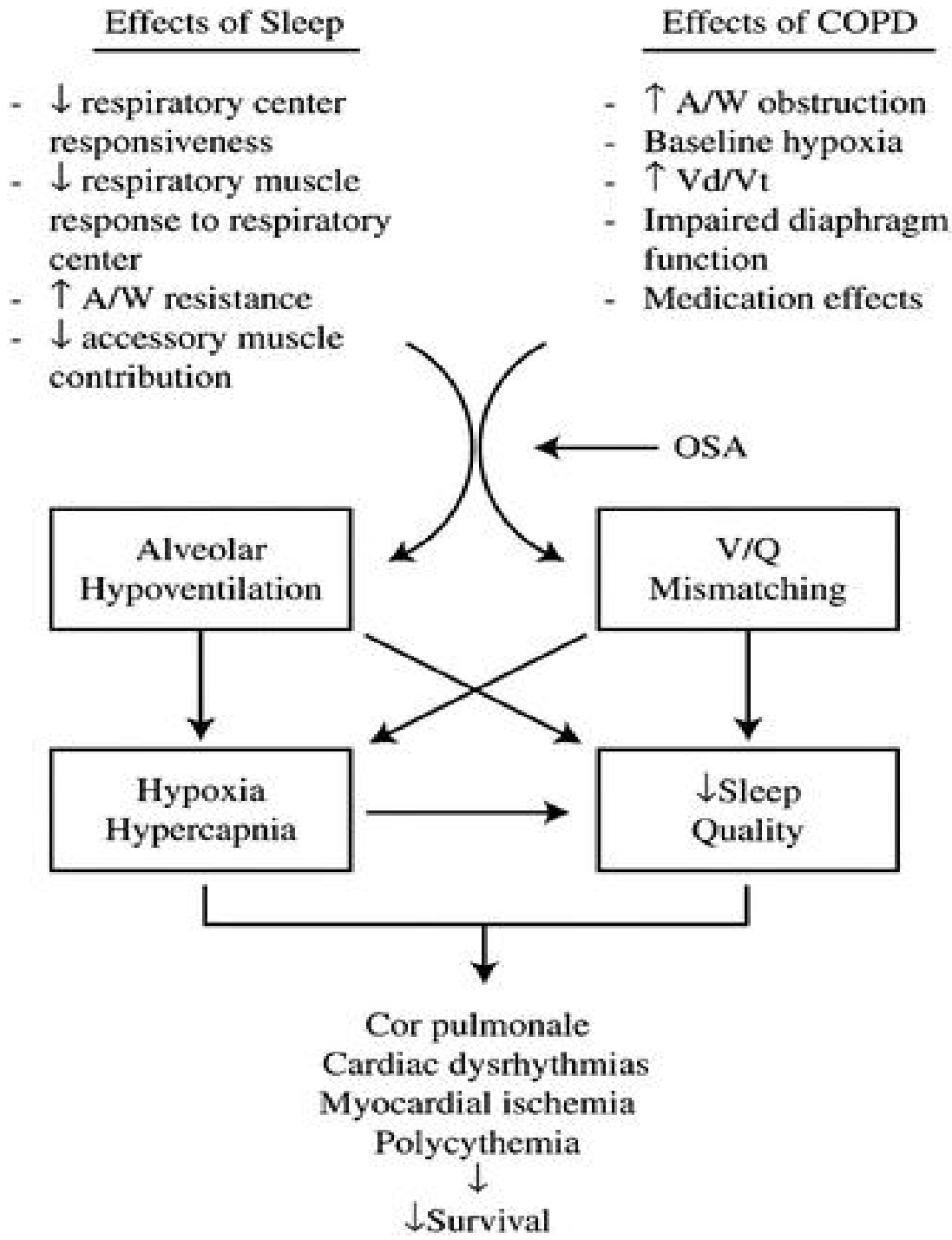
Hyperinflation of the Lung, one of the pathological consequences of COPD causes stretching of diaphragm that results in poor diaphragmatic contraction[24].

Patients with severe COPD have atrophy and skeletal muscle dysfunction, which may further result in reduction the contribution made by accessory muscles[25].

During sleep, supine position compromise the diaphragmatic efficiency as result of pressure by abdominal contents.

In REM sleep diaphragm is the only muscle that is active in patients with COPD.

This probably explains why there are significant correlation observed between the factors related to respiratory muscle strength and the mean nocturnal oxygen saturation in COPD .



Emphysematous destruction of the pulmonary capillary bed and progressive airflow limitation results in ventilation perfusion mismatch in COPD.

There is a reduction in Functional Residual Capacity, that occurs during sleep, as a result of decreased tone of the muscles and the increased resistance to the airways, which causes increase in the ventilation perfusion mismatch[26].

In normal subjects, the supine position causes about 10% decrease in Functional Residual Capacity.

The Small airways situated in the dependent part of the lungs gets closed due to the alterations mentioned above, which also causes the ventilation perfusion mismatch[27].

The degree of hypoventilation during sleep is similar in patients with significant nocturnal desaturation and those with minor nocturnal desaturation. This is proved by the fact that the increase in $PaCO_2$ levels are identical in both groups[28].

Effect of COPD on sleep quality

Reduction in quality of life is seen by patients with COPD due to Impairment of sleep quality[29,30]. These patients have complaints of fatigue, sleepiness, impaired concentration.

When compared with the general population, there is increased prevalence of insomnia, hypnotic medications and increased daytime sleepiness are seen in COPD [31].

Patients with symptoms of nocturnal cough or wheezing have difficulty initiating or maintaining sleep. If both symptoms are present 53% of COPD patients reported to have difficulty initiating or maintaining sleep, and 23%of COPD patients have excessive daytime sleepiness[31].

There is very little impact on sleep quality in patients with mild obstructive airways disease. But, as the COPD becomes more severe, there are increase in the number of complaints pertaining to sleep[32] .

Increased fragmention of sleep is seen in COPD, with increased arousals and decreased quantity of deep sleep and REM sleep[33].

Correlation between hyperinflation and decreased sleep efficiency in overlap syndrome patients, was reported but this effect is independent of OSA after correction was made for the apnoea/ hypopnoea index (AHI).

Hypercapnia seems to be the main factor behind the poor sleep in patients with COPD. This is reinforced by the fact that the sleep quality is not much improved by adding nocturnal oxygen[34] .

The work of breathing is increased in COPD, due to the hyperinflation .The stimulation of mechanoreceptors present in the chest wall and lower airways are stimulated due to this increased work of breathing that results in increased arousals.

The arousal response to increased inspiratory load is relatively preserved but the arousal response is lowest for hypercapnia and hypoxia in REM sleep.

Symptom of nocturnal cough may be the cause for sleep disturbance in some patients. The use of drugs like Theophylline, used as a bronchodilator may also affect sleep quality.

Cigarette smokers have disturbances in the sleep due to effect of nicotine in cigarette smoke and the withdrawal may also cause the subjective experience of non-restorative sleep[35].

Sleep impairment is an important aspect in assessment of the impact of COPD on the quality of life, but this is frequently neglected by many physicians.

Also, it has been shown that in COPD patients with sleep deprivation there is a reduction in forced vital capacity (FVC) (about 5%) and forced expiratory volume in 1 s (FEV1) (about 6%)[36].

OMACHI et al[37] have demonstrated that sleep disturbances can be predictive of COPD exacerbations and mortality of COPD patient. However, ITO et al[38] have proved that depression is the independent factor predicting exacerbations and hospitalisation in COPD but not sleep disturbances.

Overlap syndrome of COPD and OSA

David Flenley was first to describe the ‘‘Overlap Syndrome’’ . He defined it as the coexistence of COPD and obstructive sleep apnoea[39] .

Administration of nocturnal oxygen to these patients is questioned by him .The patients with Overlap Syndrome have worse clinical course and prognosis than those patients suffering from COPD or OSA alone.

Epidemiology

The diagnosis of COPD should always be considered in those patients who have symptoms of, chronic cough with sputum production and exertional dyspnoea, with history of exposure to risk factors for the disease (cigarette smoking).

Post-bronchodilator FEV1/FVC < 0.70 by spirometry confirms the presence of persistent airflow limitation and the diagnosis of COPD. OSA is considered as one of the comorbidities of COPD. Patients with OSA develops recurrent upper airway collapse during sleep and there is cessation of respiration (apnoea).

These events causes repetitive hypoxia and carbon dioxide retention, causing nocturnal awakenings (i.e. arousals) that restores the airflow. But as the sleep cycle starts again there is recurrence of obstruction and subsequent apnoea.

Daytime sleepiness or nocturnal complaints are the usual presenting symptoms, but in many patients the first to push for medical

evaluation comes from close companions due to the concerns regarding snoring and/or witnessed apnoea.

Conformation of OSA is done by polysomnography.

In Chest clinics, COPD and asthma and OSA are the commonly reported disorders.

Studies have shown prevalence of nearly 4% in males and 2% in females in general population within the age group of 30 to 60 years[40,41].

COPD prevalence is dependent on the prevalence of tobacco smoking, but in general about 10% of the population in the world have moderate-to-severe COPD.

Main risk factor for OSA is obesity and the rates of obesity have been on the rise since these studies were done. Hence, prevalence of OSA may be higher now. OSA occurred in 3% of mild COPD patients in a European study [43]

COPD and OSA

In a recent COPD study about nocturnal symptoms states that about 78.1% reported to have some degree of night-time symptoms[44].

This study also reported to have increase in nocturnal symptoms with increase in the severity of airflow limitation .

Patients having nocturnal symptoms have more exacerbations than patients without nocturnal symptoms. The OSA symptoms or sleep studies are not recorded in this study. This is the main limitation of this study.

Increased arousals and difficulty in maintenance of sleep is seen in COPD patients with symptoms of nocturnal wheezing, cough and sputum.

Reduction in total sleep time, sleep efficiency in COPD patients is documented in sleep studies , this causes the daytime hypersomnolence seen in patients[45].

Significant nocturnal oxygen desaturation is seen patients with COPD and presence gas exchange abnormalities in daytime, particularly a lower PaO_2 predictive of nocturnal oxygen desaturation.

Previous studies documented that about 50% of COPD patients who have daytime SaO_2<90% without co-existing sleep apnoea have significant desaturation during sleep[46].

There is an increased mortality in the patients who have significant nocturnal desaturation compared with those who do not desaturate .

The prevalence of OSA is found to be similar in COPD patients compared with the non-COPD population from the previous studies . But presence of some predisposing factors for OSA such as age, active smoking, oral corticosteroids, presence of peripheral oedema increases the risk of obstructive apnoea events.

Obesity is the main risk factor for sleep disordered breathing, obesity hypoventilation syndrome, pulmonary hypertension and irrespective of the airflow obstruction severity in COPD patients .

Management of Sleep Disturbances in COPD

Nocturnal supplemental Oxygen therapy

The most important complication that occurs during sleep hypoventilation is hypoxemia.

Hypoxemia when corrected alone in patients with COPD with hypercapnia will result in worsening of ventilation because the chemoreceptor stimulation due to hypoxia gets abolished. Hence, when nocturnal oxygen is administered,

Thus, the supplementation should be targeted to bring the oxygen saturation level above 90%[47].

Mode of oxygen delivery during sleep is usually via nasal cannula because face masks are dislodged during sleep .

The amount of time spent during sleep with less than 90% oxygen saturation is the main indicator for calculating the magnitude of hypoxemia.

Medications

There is increased cholinergic tone in night and it results in increased airflow obstruction and the deterioration in nocturnal gas exchange.

Anti cholinergic agents like ipratropium has shown to improve sleep quality and nocturnal gas exchange in patients with COPD

Improvement in nocturnal oxygen saturation with the once-daily anticholinergic agent, Tiotropium, is documented without much change in the sleep quality in one study[48].

These agents cause significant changes in oxygen saturation, particularly in REM sleep. This finding is significant because it is during the REM sleep there is more significant desaturation.

Beta agonist salmeterol has shown improvement in oxygen saturation to same extent as that of Tiotropium[49].

Theophylline causes bronchodilation, increased contractility of diaphragm. It causes central respiratory stimulation. These effects are useful in COPD patients having respiratory disturbance during sleep[50].

Theophylline has shown evidence of beneficial effects in OSA. But the disadvantage is the effect of this drug on sleep quality.

Benzodiazepine and non-benzodiazepine hypnotics are shown to decrease sleep latency, decrease arousal frequency, improve sleep efficiency but due to the effects of these drugs on ventilation causing hypoxaemia and hypercapnia. So it is recommended that these drugs should be avoided, in patients having severe COPD.

Use of hypnotics, such as zolpidem, has been documented in less severe COPD patients without having much impact on gas exchange [51]

Melatonin receptor antagonists, like ramelteon, is shown to improve sleep efficiency and shorten sleep latency. This drug have no

adverse effects on apnoea frequency or nocturnal oxygen saturation levels in patients with COPD[52].

Role of Non Invasive ventilation

Patients with COPD not responding to pharmacological therapy must be considered for non-invasive ventilation. Patients with COPD patients with hypercapnic respiratory failure should be considered for NIV.

Improvements in respiratory muscle strength and endurance are reported in patients treated with NIV .

NIV along with supplemental oxygen has shown improvement in quality of sleep and diurnal PaO_2 and $PaCO_2$ levels when compared to patients treated with supplemental oxygen alone .

Mechanism of Action

1. By reversing micro atelectasis and also preventing collapse of the airways causing reduction in the work of breathing and increases lung compliance.

2. By giving rest to chronically fatigued respiratory muscles results in improvement of daytime respiratory muscle function[54].

Patients with diagnosis of overlap syndrome should be treated by nocturnal pressure support . Decision between CPAP or Bi-level positive airway pressure can be determined on the basis of pattern of sleep disordered breathing.

In patients where OSA predominates, Continuous Positive Airway Pressure (CPAP) may be the most appropriate, but in patients where there is significant nocturnal hypoventilation with associated periods of sustained hypoxaemia, Bilevel positive airway pressure may be more appropriate.

Pittsburgh Sleep Quality Index (PSQI)

Quality of sleep measurement can be done by the Pittsburgh Sleep Quality Index (PSQI).

There are seven different domains like sleep latency, subjective sleep quality, habitual sleep efficiency ,sleep duration, use of sleep medication, daytime dysfunction sleep and disturbances over the last month. The client himself rates each of these seven domains of sleep. Based on the scores the Quality of sleep is measured.

The questionnaire has 19-items for evaluation of subjective sleep quality over the past 1 month. Scoring of the answers is based on a scale of 0 to 3, whereby 3 reflects the negative extreme .

The scores of these 7 components are added to obtain a global score that ranges from 0–21, with increasing scores indicating worse sleep quality.

A global score of 5 or greater indicates a “poor” sleeper. Several research groups evaluated the clinical and psychometric properties of the PSQI.

The PSQI has a specificity of 86.5% and sensitivity of 89.6% for identifying cases with sleep disorder, with a cut-off score of 5.

Epworth Sleepiness Scale (ESS)

Epworth Sleepiness Scale (ESS) consists of 8 self-rated items, each item is scored from 0–3, that measures a patients habitual “likelihood of falling asleep” in situations of daily living. There is no specific time frame .

The ESS score ranges from 0–24, it represents the sum of individual items. Values >10 are considered to indicate excessive sleepiness depending on the situation. Values >15 are considered as excessive daytime sleepiness and the patient definitely needs medical evaluation.

AIM OF THE STUDY

Aim

1. To know the prevalence of sleep related breathing disorders (SRBD) in patients with Chronic Hypercapnic Respiratory Failure.
2. To assess the quality of sleep in patients with Chronic Hypercapnic Respiratory Failure.

Primary Objectives

To estimate the prevalence of Sleep Related Breathing Disorders (SRBD) in patients with Chronic Hypercapnic Respiratory Failure.

To know the sleep quality in patients with Chronic Hypercapnic Respiratory Failure.

Secondary Objectives

To identify the risk factors for Sleep related breathing disorders in patients with chronic hypercapnic respiratory failure

To identify correlation between the Quality of Sleep measurement by Pittsburgh Sleep Quality Index scoring and the sleep variables determined by polysomnography

To estimate the excessive daytime sleepiness by Epworth Sleepiness scale in patients with Chronic Hypercapnic Respiratory Failure.

MATERIALS AND METHODS

SITE OF INVESTIGATION

Government Hospital of Thoracic Medicine, Tambaram Sanatorium, Chennai

STUDY PERIOD

December 2013 to July 2014

STUDY DESIGN

Prospective Case control study

SAMPLE SIZE

45 patients

Statistical analysis

By using SPSS version 7 software – Independent t test, Chisquare analysis.

Inclusion criteria

1) A clinical history consistent with severe stable COPD without an exacerbation of airways disease for at least 4 weeks at the time of evaluation

2) Patients with diagnosis of COPD or COPD with Pulmonary Tuberculosis Sequelae with airflow obstruction evidenced by Post bronchodilator Forced expiratory volume in one second (FEV1) of less than 40% predicted, FEV1/forced vital capacity ratio of <0.70

3) Patients with Chronic Hypercapnic Respiratory Failure is defined by daytime awake $PaCO_2$> 45 mmHg while in a stable condition with PaO_2>60 mm Hg and pH >7.350 are included in study group and those with awake $PaCO_2$< 45 mmHg with PaO_2>60 mm Hg and pH >7.350 are included in control group.

4) Patients with Systemic Hypertension, Diabetes Mellitus under control are included in the study.

Exclusion criteria

1) Patient with diagnosed OSAS are excluded.

2) Age >80 yrs.

3) Patients with Cardiac, Hepatic and Renal Diseases.

4) Patients with Uncontrolled Diabetes Mellitus

5) Patients with Exacerbation < 4 weeks.

6) Patients with Respiratory Acidosis.

7) Patients with history of Smoking and Alcoholism including those who left within a period of less than 6 months.

Methods

Patients diagnosed to have COPD or COPD with Pulmonary Tuberculosis sequelae on regular follow-up at Government Hospital of Thoracic Medicine, Tambaram are enlisted. Patients without history of exacerbation in the last 4 weeks are evaluated.

1) Patients underwent Spirometric analysis and those with post bronchodilator FEV1 < 40% are asked for willingness to participate in the study. Those who are willing to participate are screened for inclusion into the study.

2) Informed Consent was obtained from all the patients

3) Arterial Blood Gas analysis was done for those patients and Patients fulfilling the inculsion criteria are enrolled for study. Blood for Arterial Blood Gas analysis was drawn from radial artery, Analysis done in calibrated Blood Gas Analyser.

4) Detailed History, symptoms of nocturnal cough, wheeze,history of lifetime alcohol, smoking and clinical examination of patients done.

5) Height and weight were measured and BMI calculated, Neck circumference, Waist circumference measured.

6) Respiratory Rate, Pulse rate, Blood pressure, Day time Oxygen saturation measured

7) Blood was drawn for analysis of Fasting and Post Prandial Blood Sugar, Blood Urea, Serum Creatinine, Liver Function Tests were done.

8) Epworth Sleepiness Scale scoring, Pittsburgh Sleep Quality Index Scoring using Standard questionnaire is done.

9) Patient was advised to avoid intake of caffeine on the day of study. He/She is refrained from having nap at daytime on the day of study.

10) Patient was asked to go to bed one hour before the usual sleep time, hooking up of the polysomnogram instrument was completed and the lights are off at the usual sleep time and the recording was started.Full attended polysomnography was performed with Medicaid systems, Sleep care SC 32 Poysomnogram.

11) Recording was done for minimum period of 6 hours. Lights were put on once patient wakes up from sleep.

12) Measured parameters are electroencephalography (EEG), left and right electro-oculography, Thoracoabdominal movement by inductance bands, airflow (by nasal pressure cannula), body position ,leg movements and arterial oxygen saturation.

13) Polysomnography was done and the following sleep variables according to American Academy of Sleep Medicine (AASM) criteria are recorded.

- Total Bed Time(TBT),
- Total Sleep Time (TST),
- Sleep efficiency,
- Sleep latency,
- Sleep stages in minutes and as percentage of TST,
- Arousal index,
- Respiratory event (apnoea and hypopneas) were measured in seconds.

- Apnoea-hypopnea index,
- Minimal nocturnal oxygen saturation,
- Mean nocturnal oxygen saturation and
- Patients with nocturnal desaturation are recorded.

Criteria for scoring based on American Academy of Sleep Medicine (AASM)[55]

Apnea

Apnea is scored when there is a drop in the peak signal excursion by ≥ 90% of pre-event baseline for ≥ 10 seconds

The Apnea is scored as Obstructive if the above criteria with continued or increased inspiratory effort throughout the entire period of absent airflow.

The Apnea is scored as Central if the above criteria is met with absence of inspiratory effort

The Apnea is mixed apnea if it begins as a central apnea, but towards the end there is effort to breathe without airflow.

Hypopnea is diagnosed if all the following are present:

The peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure sensor.

The duration of the ≥30% drop in signal excursion is ≥10 seconds.

There is a ≥ 4% oxygen desaturation from pre-event baseline.

AHI (**Apnea Hypopnea Index**) is the average number of apneas and hypopneas per hour of sleep.

Cheyne Stokes Breathing

Cheyne Stokes Breathing is diagnosed if there are atleast 3 consecutive cycles of cresendo decrescendo change in breathing amplitude and atleast 1 of the following

Five or more Central apneas or hypopneas per hour

The cyclical cresendo decrescendo breathing has duration of atleast10 consecutive minutes.

Total Bed Time (TBT) is the time from Lights out to Lights on

Total Sleep Time (TST) is Total Stages N1, N2, N3, REM (in minutes)

Total Sleep Time =Total Bed Time (TBT) – Total Wake Time

Wake After Sleep Onset (WASO) is the total amount of wake time after the first epoch of Sleep

Sleep Efficiency (%) is the percentage of time asleep compared to the time spent in bed

Sleep Efficiency (%) = Total Sleep Time (TST) ÷ Total Bed Time (TBT) x 100%

% of Sleep Stages

% of Sleep Stages is the Total Time of a particular sleep stage divided by Total Sleep Time (TST) This is calculated for Stages N1, N2, N3 & REM

% Stage N1= Total Stage N1 (in minutes) ÷ TST x 100%

% Stage N2= Total Stage N2 (in minutes) ÷ TST x 100%

% Stage N3= Total Stage N3 (in minutes) ÷ TST x 100%

% REM= Total REM (in minutes) ÷ TST x 100

Sleep latency is lights out to first epoch of any sleep stage in minutes

Arousal is the total number of awakenings associated with transient desaturation compared to the preceding two minute period per hour of sleep

Arousal index is the average number of arousal per hour of sleep.

Nocturnal Desaturation is defined by Patients with Oxygen saturation below 90 for > 30% of total sleep time[9].

Criteria for Obstructive sleep apnea

Individuals must fulfill criterion A or B, plus criterion C to be diagnosed with OSA:

A. Excessive daytime sleepiness that is not explained by other factors

B. Two or more of the following that are not explained by other factors:

- Choking or gasping during sleep
- Recurrent awakenings from sleep
- Unrefreshing sleep
- Daytime fatigue
- Impaired concentration

C. Overnight monitoring demonstrates 5 to 10 or more obstructed breathing events per hour during sleep or greater than 30 events per 6 hours of sleep. These events may include any combination of obstructive apnea, hypopnea.

OBSERVATION AND RESULTS

Forty Five patients are enrolled into the study in total. Thirty two patients are Patients with Chronic Hypercapnic Respiratory Failure (Group A). Thirteen patients had COPD or COPD with Pulmonary Tuberculosis sequelae and with similar degrees of airflow obstruction without Respiratory failure (Group B). In Group A -Twenty seven patients were male, and five patients were female. In Group A - 22 patients had COPD and 10 patients had COPD with Pulmonary Tuberculosis sequelae . In Group B - Ten Male and three patients were Female. Ten patients had COPD and three had COPD with Pulmonary Tuberculosis sequelae.

Table 1

Baseline Characteristics

Variables	**Group A (Mean ± Standard Deviation)** (n=32)	**Group B (Mean ± Standard Deviation)** (n-13)
Age	57.19 ± 8.56	57.85 ± 5.81
Gender (No. of Patients)	Male - 27, Female - 5	Male - 10, Female – 3
Smoking Index (Pack Years)	29.09	26.92
Life time Alcohol (No. Of Patients)	8	2

Variables	**Group A (Mean ± Standard Deviation)** (n=32)	**Group B (Mean ± Standard Deviation)** (n-13)
Systemic Hypertension (No. Of Patients)	8	3
Diabetes (No. Of Patients)	7	2
Waist circumference (cm)	80.47 ± 2.95	79.54 ± 6.70
Neck Circumference (cm)	34.09 ± 3.25	32.92 ± 1.93
BMI	22.1 ± 3.4	21.3 ± 2.6
FEV1/FVC	0.53 ± 0.08	0.57 ± 0.08
FEV1%	26.78 ± 4.48	25.92 ± 4.05
FEV1(L)	0.68 ± 0.28	0.72 ± 0.19
FVC (L)	1.44 ± 0.35	1.25 ± 0.26
PaO_2 (mm)	65.03 ± 2.48	74.01 ± 3.46
$PaCO_2$(mm)	50.9 ± 3.15	38.55 ± 1.68
pH	7.37 ± 0.05	7.39 ± 0.02
HCO_3(mm)	30.03 ± 1.21	25.66 ± 0.68
Snoring(%)	41%	23.07%
Nocturnal Awakenings(%)	78.1%	69.0%
Morning Headache(%)	41.0%	15.4%
Chocking Episodes(%)	6.25%	0

(BMI- Body Mass Index, FEV1 – Forced Expiratory Volume - 1 Second, FVC- Forced Vital capacity)

Table 1 shows the baseline parameters of the Group A and B

The mean Age group is 57.2 vs 57.8 years, BMI is 22.1 ± 3.4 vs 21.3 ± 2.6. Systemic Hypertension is seen in 8 patients in group A and 3 patients in Group B. Diabetes is seen in 7 patients in Group A and 2 patients in Group B. Mean $PaCO_2$ is 50.9 mm in Group A and 38.55 mm in Group B. Significant Nocturnal Desaturation is seen in 68.8% of patients with Group A and 38.5% of patients in Group B. Snoring is present in 41% of Group A and 23.07% of Group B

Table 2

Independent t test (Tests of Between-Subjects Effects)

Comparison of Polysomnographic variables between Group A and B

Polysomnography variable	**Group A** (n-32)	**Group B** (n-13)	**P Value**
Total bed (TBT) (in minutes)	399.9 ± 35.3	419 ± 46.5	0.272
Total sleep Time(TST) (in minutes)	234.7 ± 33.6	290.9 ± 50.9	**0.035**
Sleep latency (in minutes)	62.7 ± 18.5	42.4 ± 9.7	**0.003**
Sleep efficiency %	58.5 ± 6.3	69.1 ± 8.4	**0.042**
Arousal index (per hour)	31.1 ± 5.6	20.4 ± 5.6	**0.003**
WASO (Wake after sleep onset) (in minutes)	100 ± 23.9	84 ± 28.6	0.599
WASO (% of TBT)	24.9 ± 5.3	20.1 ± 6.6	0.295
AHI(Apnea hypopnea Index)	3.19 ± 2.13	2.4± 1.7	0.987
ESS score (Epworth Sleepiness Score)	11.5± 5.5	9.7 ± 4.2	0.219
PSQI score(Pittsburgh Sleep Quality Index)	13.2± 1.9	7.3±2.1	**0.018**

Mean Values of Group A and Group B are presented in the Table 1 and 2. Group A (Patients with Chronic Hypercapnic Respiratory Failure) Vs Group B (Patients without Respiratory failure)

The mean Total Bed Time (TBT) (in minutes) is 399 $\pm$ 35.3 vs 419 ± 46.5, Total Sleep Time (TST) (minutes) is 234.7$\pm$ 33.6 vs 290.9 ± 50.9. Sleep latency (in minutes) is 62.7 ± 18.5 Vs 42.4 ± 9.7, Sleep efficiency 58.5 ± 6.3 vs 69.1 ± 8.4, Arousal index is 31.1 ± 5.6 vs 20.4 ± 5.6, WASO (Wake after sleep onset) (in minutes) 100 ± 23.9 vs 84 ± 28.6 . Epworth Sleepiness score is 11.5 $\pm$ 5.5 vs 9.7 ± 4.2, PSQI is 13.2+2.3 vs 7.3+2.1 .There is a statistically significant difference in the Total Sleep Time, Sleep latency, Sleep efficiency, Arousal index and PSQI score between the two groups.

Table 3

Independent t test (Tests of Between-Subjects Effects)

Comparison of Sleep Stages Between the Group A and B

Sleep Stages	**Group A** (n-32)	**Group B** (n-13)	**P value**
NREM1,2 (in Minutes)	180.1 ± 29	208.1 ± 34.2	0.322
NREM 1,2 (%of TST)	76.8 ± 5.7	71 ± 6	**0.019**
NREM3(Min)	20.7 ± 8.5	33.9 ± 15.4	**0.002**
NREM3 (% of TST)	8.8 ± 3.4	11.4 ± 3.9	**0.002**
REM (min)	34.7 ± 7.8	48.6 ± 13.7	**0.030**
REM (% of TST)	14.7 ± 3.2	16.7 ± 3.3	0.167

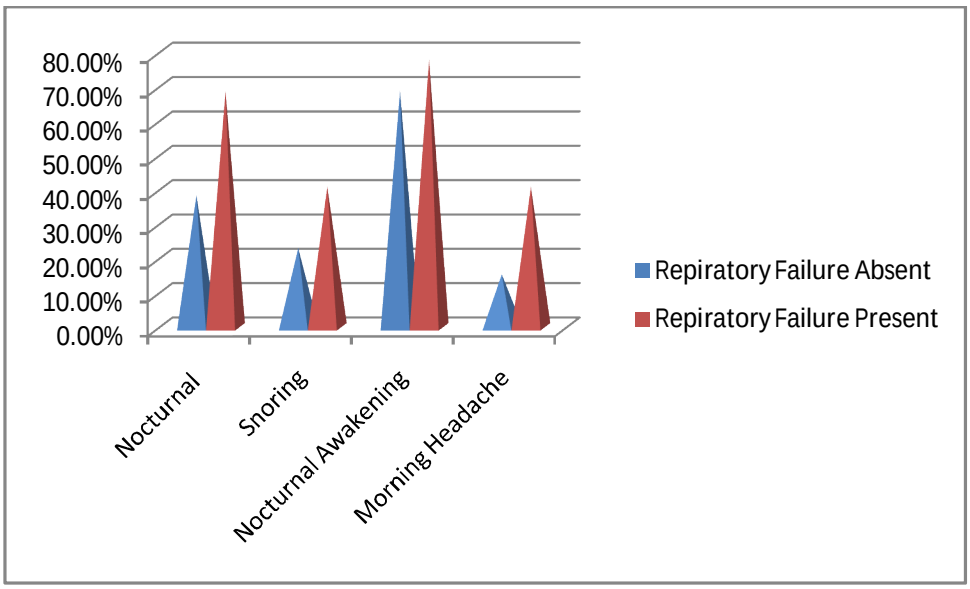
Table 3 showing various sleep stages among the two groups - NREM1,2 (in Minutes) 208.1 ± 34.2 vs 180.1 ± 29 . NREM3(Min) is 20.7 ± 8.5 vs 33.9 ± 15.4. REM (min) 34.7 ± 7.8 vs 48.6 ± 13.7, There is a statistically significant difference in the NREM1,2 (%), NREM3(Min), NREM 3(%), REM(min) between the two groups.

Table 4

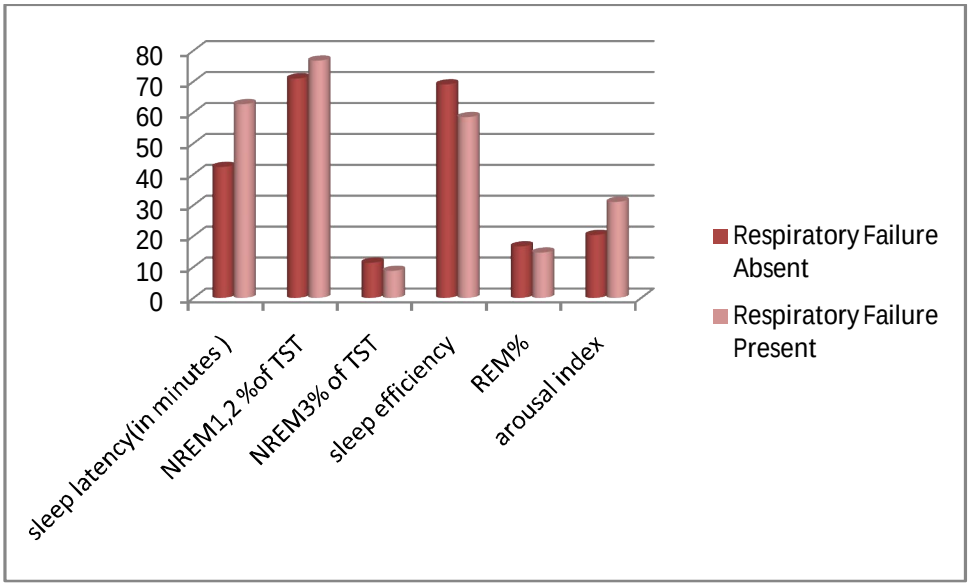
Comparison of Oxygen Saturation between the Group A and B

Oxygen Saturation	**Group A** (n-32)	**Group B** (n-13)	**P Value**
Daytime SpO_2 %	92.6 ± 1.6	93.8 ± 1.3	**0.03**
Nocturnal SpO_2 %	85.3 ±2.8	88.9 ± 2.9	**0.001**
Minimal nocturnal SpO_2 %	75.2± 5.4	79.4 ± 6.2	**0.001**
Significant Nocturnal Desaturation(%)	68.80%	38.50%	-

Table 4 showing the Mean Day time Oxygen saturation during daytime, nocturnal and the minimal nocturnal Oxygen saturation with statistically significant difference in the two groups . Significant Nocturnal desaturation is defined by patients spending > 30% of Total sleep Time with Oxygen Saturation of < 90% . 68.8 % of patients in Group A have significant nocturnal desaturation, vs 38.5% in Group B .



Picture Depicting the Difference between the symptoms in Patients with and without Respiratory Failure



Picture Depicting the Difference between the Sleep Variables in Patients with and without Respiratory Failure

Table 5

Prevalence of Sleep Related Breathing Disorders in Group A and Group B

Sleep Related Breathing Disorders	Group A No.of Patients (%)	Group B No.of Patients (%)
Obstructive Sleep Apnea	2 (6.25%)	1 (6.66%)
Central Sleep Apnea	0	0
Mixed Sleep Apnea	0	0

Table5 Showing the prevalence of Obstructive Sleep Apnea in Group A is 6.25% vs 6.66 % in Group B .All the 3 Patients diagnosed with OSA in the cohort had OSA of Moderate Severity (AHI 15 to 30). No patient was diagnosed with Central sleep apnea or Mixed apnea.

Table 6

Comparison of sleep variables in Patients with Chronic Hypercapnic Respiratory Failure based on Symptoms of Nocturnal Cough ± Wheeze

Independent t test (Tests of Between-Subjects Effects)

Sleep variable	Chronic Hypercapnia with Breathlessness (n=19)	Chronic Hypercapnia with Breathlessness+ Nocturnal Cough ± Wheeze (n-13)	P value
Total bed Time (TBT) (in minutes)	414.3 ± 56.6	400.1± 31.2	0.427
Total sleep time (TST) (in minutes)	285.6 ±62.4	225.8 ±26.1	0.003
Sleep latency (in minutes)	48.3 ±12.7	63.7 ±20.0	**0.021**
Sleep efficiency (%)	68.3 ±8.0	56.6 ±6.4	**0.001**
Arousal index (Per hour)	23.9 ±7.1	32.0 ±5.8	**0.003**
WASO (in minutes)	79.9 ±25.7	107.2 ±22.0	**0.006**
WASO (% of TBT)	19.4 ±6.5	26.7 ±4.7	**0.003**
AHI (Apnea hypopnea Index)	2.9 ±1.6	2.5 ±1.8	0.569
PSQI score (Pittsburgh Sleep Quality Index)	9.3 ±3.0	12.2 ±2.8	**0.012**
ESS score	11.0 ±5.4	13.8 ±5.2	0.180

Patients with Chronic Hypercapnic Respiratory Failure with symptoms of Nocturnal Cough or Wheeze had decreased Total sleep time(TST) 225.8 minutes, sleep efficiency 56.6%. They had increased sleep latency 63.7 minutes, arousal index 32.0/hr, WASO 26.7%, PSQI score 12.2, ESS score of 13.8. Compared to patients without nocturnal cough or wheeze these patients have poor sleep variables.

Table 7

Comparison of sleep stages in Patients with Chronic Hypercapnic Respiratory Failure based on Symptoms of Nocturnal Cough ± Wheeze

Independent t test

Sleep Stages	**Chronic Hypercapnia with Breathlessness** (n-19)	**Chronic Hypercapnia with Breathlessness+ Nocturnal Cough ± Wheeze** (n-13)	**P Value**
NREM1,2 (in Minutes)	209.5 ±40.5	174.9 ±28.2	**0.016**
NREM 1,2(%of TST)	74.1 ±6.8	75.5 ±6.9	0.603
NREM3(Minutes)	29.8 ±16.8	19.2 ±8.2	**0.048**
NREM3(% of TST)	10.0 ±4.0	8.7 ±3.8	0.377
REM(min)	46.2 ±16.1	33.1 ±7.2	**0.012**
REM(% of TST)	15.8 ±3.9	14.8 ±3.2	0.461

Comparison of sleep stages in Patients with Chronic Hypercapnic Respiratory Failure based on Symptoms of Nocturnal Cough ± Wheeze shows statistically significant difference in NREM 3 duration and REM sleep duration between the groups.

Table 8

Comparison of Oxygen Saturation variables in Patients with Chronic Hypercapnic Respiratory Failure based on Symptoms of Nocturnal Cough ± Wheeze

Independent t test

Oxygen Saturation	**Chronic Hypercapnia with Breathlessness** (n-19)	**Chronic Hypercapnia with Breathlessness+ Nocturnal Cough ± Wheeze** (n-13)	**P value**
Daytime SpO_2 %	93.2 ±1.4	92.2 ±1.2	0.057
Nocturnal SpO_2%	87.5 ±3.4	84.8 ±2.8	**0.038**
Minimal Nocturnal SpO_2%	76.3 ±4.4	72.9 ±5.2	0.073

There is statistically significant reduction in the mean nocturnal oxygen saturation (84.8%) of patients with Symptoms of nocturnal cough or wheeze.

Table 9

Comparison in Patients with Chronic Hypercapnic Respiratory Failure with Sleep efficiency of < 60 and >60 based on Body Mass Index

Sleep efficiency	Body Mass Index (No. Of patients)				Total
	< 18.5	18.5 - 24.9	25 - 29.9	> 30	
< 60	4	10	3	0	17
> 60	1	11	2	1	15
Total	5	21	5	1	32
P -value	0.402	Chisquare			

There is no statistically significant correlation between the Body mass index and sleep efficiency based on the Chisquare analysis

Table 10

Comparison in Patients with Chronic Hypercapnic Respiratory Failure with Sleep efficiency of < 60 and>60 based on Systolic blood Pressure(mm Hg)

Sleep Efficiency	Systemic Hypertension		Total
	Absent	Present	
< 60	12	5	17
> 60	12	3	15
Total	24	8	32
P -value	0.539	Chisquare	

There is no statistically significant correlation between Systolic blood pressure and sleep efficiency of patients with Chronic Hypercapnic Respiratory Failure

Table 11

Comparison in Patients with Chronic Hypercapnic Respiratory Failure with Sleep efficiency of < 60 and >60 based on Gender

Sleep Efficiency	Gender (No. of Patients)		Total
	Male	Female	
< 60	14	3	17
> 60	13	2	15
Total	27	5	32
P -value	0.737	Chisquare	

There is no statistically significant correlation between the Gender and Sleep Efficiency based on the Chisquare analysis.

Table 12

Comparison of sleep variables in Patients with Chronic Hypercapnic Respiratory Failure with and without Significant Nocturnal Desaturation

Independent t test

Sleep variables	Nocturnal Desaturation		P value
	Present	Absent	
	Mean ±Std.Deviation (n-22)	Mean ± Std. Deviation (n-10)	
Total bed (TBT) in minutes	413.9 ±64.8	401.9 ±32.9	0.490
Total sleep time (TST) in minutes	293.8 ±69.2	236.5 ±35.5	**0.004**
Sleep latency (in minutes)	47.2 ±13.7	61.0 ±18.1	**0.014**
Sleep efficiency(%)	70.7 ±8.3	58.6 ±6.4	**0.018**
Arousal index(per hour)	22.9 ±7.9	29.6 ±6.4	0.170
WASO (in minutes)	72.2 ±24.4	102.3 ±21.2	**0.001**
WASO (% of TBT)	17.6 ±6.1	25.5 ±5.0	**0.001**
PSQI score	8.3 ±3.4	12.0 ±2.5	**0.001**
ESS score	9.4±3.7	14.0±5.8	**0.029**

This table shows that Patients with nocturnal desaturation have decreased TST, Sleep efficiency and increased Sleep latency, WASO (% of TBT), AHI, PSQIscore and ESS score

Table 13

Comparison of sleep stages in Patients with Chronic Hypercapnic Respiratory Failure with and without Significant Nocturnal Desaturation

Independent t test

Sleep Stages	Significant Nocturnal Desaturation		P Value
	No (n-10)	**Yes** (n-22)	
NREM 1,2(in Minutes)	215.7 ±47.9	180.9 ±27.5	**0.014**
NREM 1,2 (%of TST)	71.3 ±7.5	76.8 ±5.8	**0.031**
NREM 3(Min)	33.0 ±17.7	19.6 ±9.4	**0.009**
NREM 3 (% of TST)	10.9 ±4.5	8.3 ±3.3	0.065
REM(min)	47.7 ±16.3	35.6 ±10.8	**0.018**
REM (% of TST)	16.4 ±4.4	14.8 ±3.3	0.259

Table 13 shows that Patients with nocturnal desaturation have statistically significant Increase in duration of NREM 1,2 (minutes) and % of NREM 1,2, decreased NREM 3 duration & decreased REM duration.

Table 14

Comparison of sleep variables in Patients with Chronic Hypercapnic Respiratory Failure with Epworth Sleepiness score of ≥ 15 and < 15

Independent t Test

Sleep Variables	Epworth Sleepiness score		P value
	≤15	**> 15**	
	Mean ±Std. Deviation (n-19)	**Mean ± Std. Deviation** (n-13)	
Total bed (TBT) (in minutes)	403.2 ±50.7	409.0 ±35.3	0.726
Total sleep time(TST) (in minutes)	266.9±59.9	236.1 ±41.0	0.119
Sleep latency (in minutes)	51.0 ±14.6	64.8±19.5	**0.029**
Sleep efficiency%	65.6 ±8.7	57.6±7.0	**0.01**
WASO (in minutes)	84.8 ±28.2	104.5±17.4	**0.033**
WASO (% of TBT)	21.2 ±7.1	25.6 ±4.1	0.054
Arousal index (per hour)	25.4 ±7.2	30.4±7.0	0.065
PSQI score (Pittsburgh Sleep Quality Index)	10.2±3.6	11.7 ±2.4	0.188

This table shows that Patients with >15 have statistically significant reduction in Sleep efficiency and increased Sleep latency, WASO (% of TBT), AHI, PSQI score .

Table 15

Comparison of Sleep Stages in Patients with Chronic Hypercapnic Respiratory Failure with Epworth Sleepiness score of > 15 and < 15

Independent t Test

Sleep Stages	**Epworth Sleepiness score**		**P Value**
	≤15	**> 15**	
	Mean ± Standard Deviation (n-19)	**Mean ± Standard Deviation** (n-13)	
NREM1,2 (in Minutes)	197.1 ±42.6	183.8 ±29.9	0.34
NREM1,2 (% of TST)	72.8 ±6.3	78.4 ±6.1	0.017
NREM3 (Min)	27.7 ±14.5	17.9±10.6	**0.045**
NREM3 (% of TST)	10.1 ± 3.8	7.4±3.2	**0.047**
REM (minutes)	42.7 ± 13.43	34.46 ± 13.05	0.092
REM (% of TST)	16.07 ± 3.46	14.17 ± 3.75	0.153

Table 15 shows that Patients with ESS > 15 have decreased NREM 3 duration and NREM % of TST, which is statistically significant.

Table 16

Comparison of Oxygen Saturation in Patients with Chronic Hypercapnic Respiratory Failure with Epworth Sleepiness score of > 15 and ≤ 15

Independent t Test

Oxygen Saturation	Epworth Sleepiness score		P value
	≤15 (n=19)	>15 (n=13)	
DaytimeSpO_2%	92.9 ±1.3	92.3 ±1.3	0.244
Mean Nocturnal SpO_2 %	86.8 ±3.6	85.2 ±2.3	0.163
Minimal Nocturnal SpO_2%	75.1 ±4.5	74.0 ±5.4	0.546

Patients with ESS > 15 have decreased Mean nocturnal SpO_2 85.2 % but the difference is not statistically significant.

Table 17

Comparison of sleep variables in Patients with Chronic Hypercapnic Respiratory Failure

with Pittsburgh Sleep Quality Index Score of >10 and ≤10.

Sleep Variables	PSQI Score		P value
	≤ 10	**>10**	
	Mean ±SD (n=14)	**Mean ± SD** (n=18)	
Total bed (TBT) In minutes	415.9 ±54.8	398.6 ±35.9	0.288
Total sleep time(TST) in minutes	291.5 ±62.5	229.1 ±29.1	**0.001**
Sleep latency(in minutes)	44.9 ±9.1	64.7 ±18.1	**0.001**
Sleep efficiency	69.4 ±8.3	57.6 ±5.8	0.001
Arousal index(per hour)	22.0 ±6.7	31.3 ±5.4	**0.02**
WASO (in minutes)	79.4 ±23.3	102.0 ±24.3	**0.013**
WASO (% of TBT)	19.4 ±6.4	25.5 ±5.3	**0.007**
DaytimeSpO_2 %	93.3 ±1.2	92.3 ±1.3	**0.035**
Mean NocturnalSpO_2%	88.3 ±3.5	84.8 ±2.3	**0.002**
Minimal Nocturnal SpO_2	75.4 ±5.4	74.3 ±4.6	0.531

This table shows that Patients with PSQI >10 have decreased TST, Sleep efficiency and increased Sleep latency, WASO (% of TBT), Arousal index, mean nocturnal SpO_2 %

Table 18

Comparison of sleep stages in Patients with Chronic Hypercapnic Respiratory Failure with Pittsburgh Sleep Quality Index Score of >10 and ≤10.

Sleep Stages	Pittsburgh Sleep Quality Index (PSQI) Score		P Value
	≤10	**>10**	
	Mean ±SD (n=14)	**Mean ± SD** (n=18)	
NREM1,2 (in Minutes)	214.0 ±40.9	176.6 ±27.9	**0.004**
NREM 1,2 % of TST	72.3 ±7.1	77.1 ±5.9	**0.046**
NREM 3(Min)	30.1 ±18.1	19.5 ±7.7	**0.029**
NREM 3 (% of TST)	9.8 ±4.4	8.6 ±3.4	0.368
REM (min)	47.3 ±16.8	34.0 ±7.7	**0.005**
REM (%)	16.2 ±4.0	14.7 ±3.4	0.271

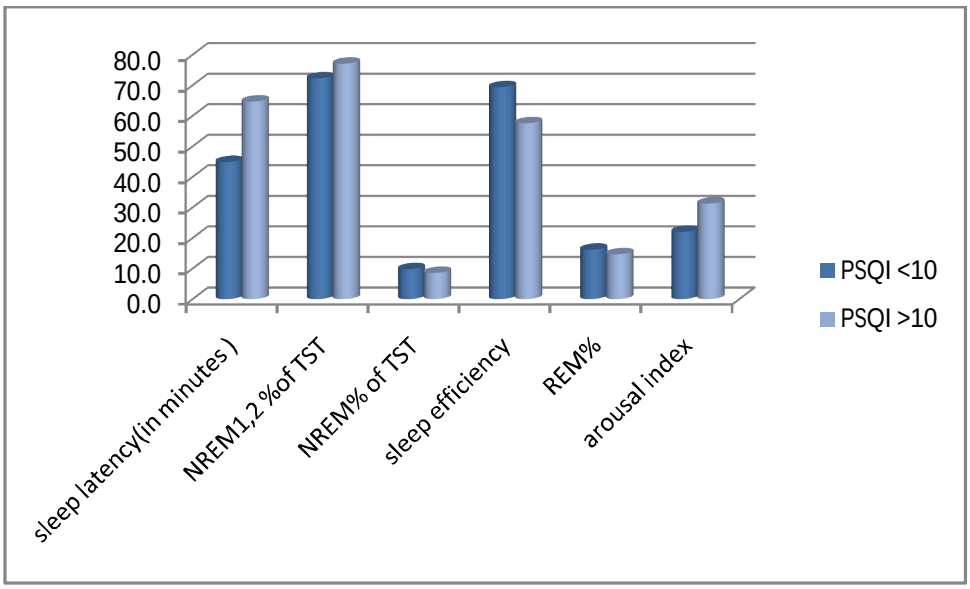
This table shows that Patients with PSQI >10 have decreased NREM 3 and REM sleep duration, increased duration of NREM 1,2

Table 19

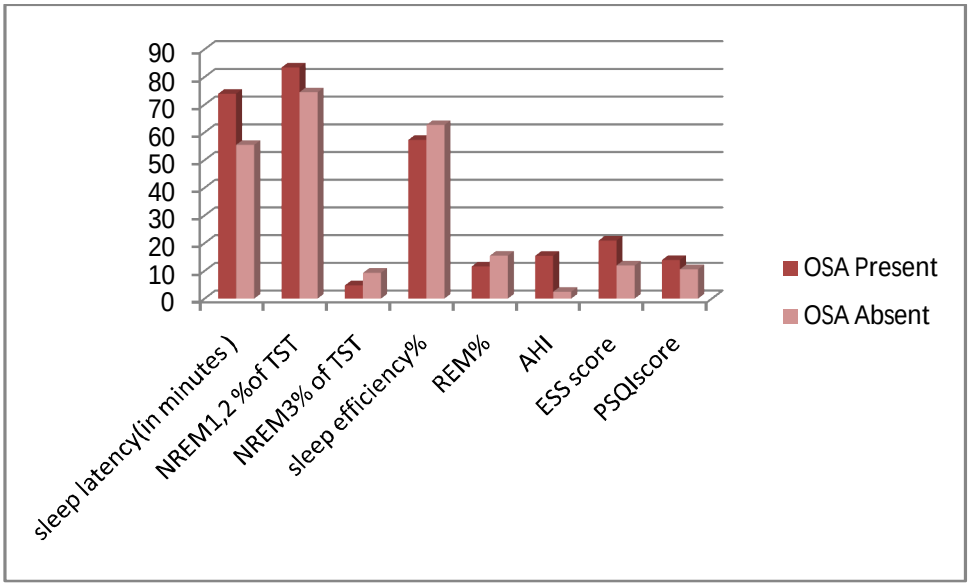
Comparison of baseline characteristics in Patients with Chronic Hypercapnic Respiratory Failure with and without OSA.

Sleep Variable	Chronic hypercapnic Respiratory Failure				
	With OSA (n=2)		**With out OSA** (n=30)		**P value**
	Mean	**Std. Deviation**	**Mean**	**Std. Deviation**	
Age(Years)	61.0	4.24	56.9	8.8	0.527
BMI	27.8	0.74	21.6	3.1	**<0.001**
Neck circeumference (cm)	41	1.41	33.63	2.77	**<0.001**
Waist circumference (cm)	102.5	2.12	79	6.87	**<0.001**

Table showing the baseline characteristics of Chronic Hypercapnic Respiratory Failure patients with and without OSA. There is a statistically significant difference in the two groups in BMI, Neck Circumference and Waist circumference.



Picture Depicting the Difference between the Sleep Variables in Patients with Pittsburgh Sleep Quality Index of<10 and >10



Picture Depicting the Difference between the Sleep Variables in Patients with Respiratory Failure with and without OSA

Table 20

Comparison of Sleep Variables in Patients with Chronic Hypercapnic Respiratory Failure with and without OSA.

Sleep Variable	Chronic hypercapnic Respiratory Failure				
	With OSA (n=2)		With out OSA (n=30)		P value
	Mean	Std. Deviation	Mean	Std. Deviation	
Total bed (TBT) (in minutes)	415.5	10.61	404.9	45.94	0.751
Total sleep time (TST) (in minutes)	238.5	41.72	255.5	55.67	0.677
Sleep latency(in minutes)	74	14.85	55.53	17.65	0.160
NREM1,2 (in Minutes)	199.75	42.07	191.25	38.5	0.766
NREM 1,2 (%of TST)	83.5	2.97	74.56	6.61	0.069
NREM3(Min)	11.5	4.95	24.61	13.84	0.196
NREM 3(% of TST)	4.85	1.06	9.37	3.77	0.105
REM(min)	27	4.95	40.23	13.74	0.190
REM (% of TST)	11.65	4.03	15.55	3.58	0.149
WOSO (in minutes)	103.25	15.91	92.16	26.63	0.566
WASO (% of TBT)	24.91	4.46	22.87	6.56	0.670
Sleep efficiency	57.35	8.41	62.74	9.04	0.418
Arousal index	32.55	2.76	27.15	7.59	0.330

Table showing the sleep variables of Chronic Hypercapnic Respiratory Failure patients with and without OSA. There is decreased sleep time, sleep efficiency, Slow wave sleep, REM sleep in patients with OSA. There is increased Sleep Latency, WASO, Arousal Index. But difference is not statistically significant in the two groups based on sleep variables.

Table 21

Comparison of Oxygen saturation in Patients with Chronic Hypercapnic Respiratory Failure with and without OSA.

Oxygen Saturation	Chronic hypercapnic Respiratory Failure				
	With OSA (n=2)		With out OSA (n=30)		P value
	Mean	Std. Deviation	Mean	Std. Deviation	
Daytime SpO_2%	91.5	0.71	92.8	1.32	0.184
Nocturnal SpO_2 %	84.5	2.12	86.33	3.35	0.453
Minimal Nocturnal SpO_2%	74	4.24	74.77	4.97	0.835
AHI(Apnea hypopnea Index)	15.5	4.1	2.51	1.77	**<0.001**
ESS score	21	1.41	12	5.32	**0.025**
PSQIscore**(**Pittsburgh Sleep Quality Index)	14	1.41	10.63	3.24	0.160

Table showing sleep parameters in Chronic Hypercapnic Respiratory Failure patients with and without OSA. Statistically significant difference is seen in AHI , ESS values.

DISCUSSION

The average age of patients with Chronic Hypercapnic Respiratory Failure (Group A) is 57.2 years . Patients in Group A have slightly higher BMI 22.1 when compared to Group B (21.3) but this difference is not statistically significant.

The Apnoea Hypopnea Index in Group A is higher than the AHI of Group B. But the difference is not statistically significant. The Prevalence of Sleep related Breathing Disorders inpatients with Chronic Hypercapnic Respiratory Failure is 6.25% . For the whole cohort the prevalence is 6.66% .

In General population various studies reported prevalence of OSA around 5%[56]. In population of 50 – 70 year one study reported the prevalence as around 7 %[57]. Prevous Studies in COPD patients reported prevalence OSA of around 10%[58,59]. The prevalence of OSA in patients with Chronic Hypercapnic Respiratory Failure in our study is similar to the previous studies . Our Study results emphasizes the need for evaluation for OSA in COPD patients presenting with Excessive daytime sleepiness.

Sleep Hypoventilation Syndrome is estimated to be around 43% in patients with Chronic Hypercapnia in previous studies[60]. In our study we

could not estimate the sleep Hypoventilation as Et co2 measurement was required. This is a limitation in our study.

Obstructive Sleep Apnoea is diagnosed in 2 Patients in Group A and one in Group B. No case of Central or Mixed Sleep Apnoea is seen.

Patients with OSA when compared to the other patients have higher Body Mass Index. All the 3 patients diagnosed to have OSA are Male patients with history of snoring. The Mean Neck Circumference and Abdominal Circumference is higher than the patients not having OSA. This suggest that Patients with risk factors of OSA are similar to those among the general population[3].

Results of our study suggest that COPD patients with Hypercapnia does not have increased prevalence of SRBD, the prevalence is similar to general population. Even though the BMI of these patients are on the lower side, the risk for OSA remains the same.

The average ESS score of patients with SRBD is much higher than the other patients with Chronic Hypercapnic Respiratory Failure. Excessive Day time sleepiness evidenced by ESS > 15 is seen in all the patients diagnosed with OSA.

The mean sleep time in Group A is 234.7 minutes which is lower than the patients in Group B. The sleep Latency is much higher in Group A 62.7, as against 42.4 minutes in Group B which is statistically significant. The sleep latency in patients with Chronic Hypercapnic Respiratory Failure in higher (62.7 min) than in other studies which showed Sleep latency of around 32 minutes[61]. There is statistically significant difference in Sleep Efficiency of 58.5% in Group A vs 69.1% in Group B . The sleep efficiency of 75% is acceptable in study conditions according to previous studies but our study showed much decreased sleep Efficiency in patients with Chronic Hypercapnic Respiratory Failure[61]. Statistical analysis showed there is no significant difference in sleep efficiency of patients while comparing the patients Chronic Hypercapnic Respiratory Failure based on BMI, Sex, Systemic Hypertension.

Arousal Index in Patients Chronic Hypercapnic Respiratory Failure is 31.1/ hour which is higher than Group B (Patients without Respiratory Failure) is 20.4/hr, which is statistically significant.

Wake After Sleep Onset (WASO) Is 24.9% of total bed time in Group A vs 20.1% in Group B. Even though there is increased WASO in

Group A There is no Statistically significant difference between the groups .

Comparison of Sleep Stages shows that there is statistically significant difference in Mean NREM 1,2 in two groups . NREM 1,2 as % of TST is higher in Group A than Group B. This shows increase in percentage of Stage 1,2 sleep in Patients with Chronic Hypercapnic Respiratory Failure.

There is statistically significant difference in Mean NREM 3 % in two groups . NREM 3 as % of TST is lower in Group A than Group B. This finding is similar to other studies in patients with COPD which suggest decrease in Slow Wave sleep in Patients with Chronic respiratory Failure[61]. Similarly REM sleep is decreased in Group A but there is no statistically significant difference between two groups.

Patients with Chronic Hypercapnic Respiratory Failure have decreased Total Sleep Time, Increased Sleep Latency, Decreased Sleep Efficiency, Decreased NREM Stage 3 Sleep, Decreased REM Sleep, Increased Arousal, Increase in duration of Wake after Sleep Onset when compared to normal values of that age[62].

Daytime SpO_2% in Group A is 92.6 %,Mean Nocturnal SpO_2% is 87.9% which is lower than the comparison group which is statistically significant. Nocturnal Oxygen Desaturation is defined by reduction in Oxygen saturation to less than 90% for more than 30% of Total Sleep Time. Various studies on COPD shown that prevalence of Nocturnal Desaturation varies from 25 to 65 %[63,64]. In our study 68.8% of patients in Group A have nocturnal desaturation. Comparison between patients with and without nocturnal desaturation showed that the decreased quality of sleep in patients with nocturnal desaturation by measurement of PSQI and sleep variables in polysomnography. There is statistically significant decreased TST, Sleep efficiency, increased sleep latency , increased number of arousal, increased wake after sleep onset in patients with Chronic Hypercapnic respiratory failure . This study shows higher prevalence of Significant Nocturnal Desaturation in patients with Chronic hypercapnic respiratory Failure.

The ESS score of > 10 is considered as having day time sleepiness[65]. Score of >15 is considered as excessive day time sleepiness that needs medical evaluation. The Average ESS score of Patients with Chronic Hypercapnic Respiratory Failure 11.5. Excessive Day time sleepiness by ESS score of >15 is seen in 34.37% of patients with

Chronic Hypercapnic Respiratory Failure after excluding patients with OSA. High prevalence of Excessive day time sleepiness is expected in Patients with Chronic Hypercapnic Respiratory Failure but in our study shows that it is seen in only 34% and not all patients with hypercapnia have Excessive Day time sleepiness.

Patients with ESS score of > 15 in Group A have decreased Total Sleep Time, decreased sleep efficiency, increased sleep latency, increased Wake after sleep onset, increased number of arousal.. There is a statistically significant decrease in NREM stage 3 Sleep. Patients with ESS score of >15 have lower mean nocturnal Oxygen saturation.

Pittsburgh Sleep Quality Index of less than 5 is considered as poor sleep quality[8]. Our Study shows correlation between PSQI and the sleep variables by polysomnogaphy. 93.75% of patients in Group A have PSQI of > 5. This indicates poor sleep quality in high number of patients with Chronic Hypercapnic Respiratory Failure. Comparing the patients with PSQI > 10 and < 10 patients with PSQI > 10 have statistically significant reduction in Total Sleep Time, decreased sleep efficiency, increased sleep latency, increased wake after sleep onset, increased number of arousal.

There is a statistically significant reduction in mean nocturnal Oxygen saturation.

Patients with COPD and Pulmonary Tuberculous Sequelae have breathlessness as predominant symptom. Sleep quality is expected to be decreased in the presence of nocturnal cough with or without wheeze . This pattern is reflected in the results of our study which showed a statistically significant difference in Total sleep time, Sleep efficiency, sleep latency Wake After Sleep onset, Arousal index. The mean Pittsburgh Sleep Quality Index is higher than the patients without nocturnal symptoms. Our study emphasises the importance of considering the symptoms of nocturnal cough or wheeze while determining the quality of sleep in patients with Chronic Hypercapnic Respiratory Failure.

Compared to previous studies done in Pulmonary Tuberculosis Sequelae and COPD patients with respiratory failure by Schonhofer et al[66] and S.K.Sharma et al[67] , our patients had poor sleep quality with Sleep Latency of 62.7, and Sleep Efficiency of 58.5. The mean Apnea Hypopnea Index is higher 3.19 when compared to the previous study which is 1.8.

CONCLUSION

Conclusion of our study are

1) Obstructive sleep apnoea is the Sleep Related Breathing Disorder diagnosed in patients with Chronic Hypercapnic Respiratory Failure.

2) Prevalence of OSA in patients with Chronic hypercapnic respiratory Failure is 6.25%. The Prevalence of OSA is similar to the prevalence in general population. Hence our study concludes that there is no increased risk for Obstuctive Sleep Apnea in patients with Chronic Hypercapnic Respiratory Failure.

3) Male gender, Increased BMI, Increased neck circumference and abdominal circumference are risk factors for OSA in patients with Chronic Hypercapnic Respiratory Failure which are similar to the general population.

4) Patients with Chronic Hypercapnic Respiratory Failure have decreased Total Sleep Time, Increased Sleep Latency, Decreased Sleep Efficiency, Decreased NREM Stage 3 Sleep, Decreased REM Sleep, Increased Arousal, Increase in duration of Wake

after Sleep Onset when compared to normal values of that age . Based on these variables it is concluded that Sleep Quality in patients with Chronic Hypercapnic Respiratory Failure is poor.

5) Sleep stages in patients with Chronic Hypercapnic Respiratory failure show increase in NREM 1,2 and decrease in NREM stage 3 and REM Sleep.

6) Presence of symptoms of nocturnal cough or wheeze has significant impact in quality of sleep of the patients.

7) Even though Excessive Daytime sleepiness is expected in majority of patients with Hypercapnia, Epworth Sleepiness score in our study shows it is seen only in 34.37% of patients with Chronic hypercapnic respiratory Failure.

8) Patients with Excessive Daytime sleepiness by Epworth Sleepiness scale scoring have more sleep impairment than those without Excessive Daytime sleepiness.

9) Nocturnal Desaturation is seen in significant proportion of patients with Chronic Hypercapnic Respiratory Failure (68.8%).

10) There is good correlation between the Quality of sleep measurement by Pittsburgh Sleep Quality Index scoring and the sleep variables determined by polysomnography.

In summary the Sleep Quality of patients with Chronic Hypercapnic Respiratory Failure is poor, OSA is the Sleep Related Breathing Disorder diagnosed in those patients. The OSA prevalence rates are similar to those seen in the general population. There is good correlation between Pittsburgh Sleep Quality Index scoring and polysomnography in the diagnosis of Sleep Quality in patients with Chronic Hypercapnic Respiratory Failure. Our study suggests that poor sleep quality in patients with Chronic Hypercapnic respiratory failure. This may recommend the use of NIV even in stable severe COPD as recommended in some of the earlier studies, so that the sleep quality and the Quality of life could be improved.

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ANNEXURE

INSTITUTIONAL ETHICAL COMMITTEE,

STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prevalence of Sleep Related Breathing Disorders (SRBD) and the Assesment of Quality of Sleep in Patients with Chronic Hypercapnic Respiratory Failure

Principal Investigator :Dr. K. Rajarajan.

Designation : Post Graduate in M.D (TB&Chest)

Department : Department of TB & Chest,
Stanley Medical College,
Chennai – 01

The request for an approval from the Institutional Ethical committee was considered on the IEC meeting held on 11.02.2014 at the council Hall,Stanley Medical College,Chennai at 2pm

The Members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the Principal Investigator.

The Principal Investigator and their team are directed to adhere to the guidelines given below:

1. Youshould inform the IEC in case of changes in study procedure, site, investigator,investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide by the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY
IEC,SMC,CHENNAI

Prevalence of Sleep Related Breathing

BY 201327051M.D (TB &RD DR..K.RAJARAJAN

Human Beings spend almost 30% of their lives in sleeping. But still much attention has not been paid to sleep disorders. Only from the 1970s, consequences of sleep disturbances produced by the abnormal breathing patterns, or Sleep Related Breathing Disorder (SRBD) are being recognized by the physicians.

Sleep related symptoms occur in about 40% of cases in patients with COPD[1]. SRBD is associated with considerable morbidity. OSA should be considered in patients reporting daytime hyper somnolence irrespective of BMI or snoring history.

The control of respiration in patients with chronic respiratory disease follows similar basic principles as that of the normal subjects, both during awake state and sleep, but with an expected lower feedback response during sleep[2]

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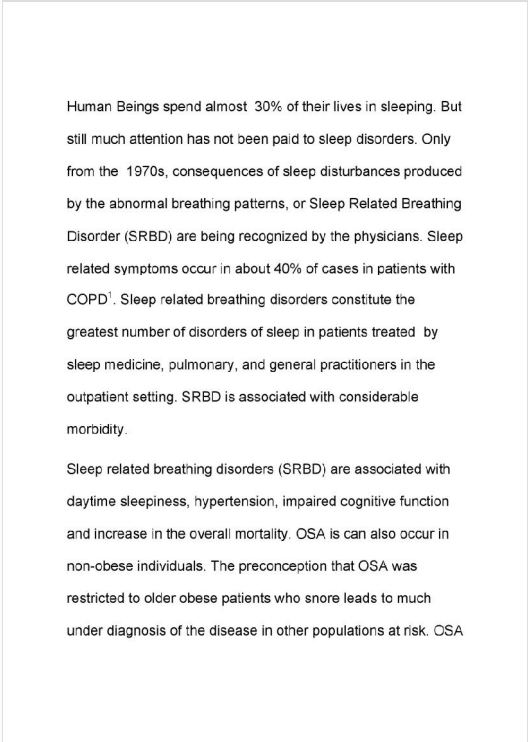

Human Beings spend almost 30% of their lives in sleeping. But still much attention has not been paid to sleep disorders. Only from the 1970s, consequences of sleep disturbances produced by the abnormal breathing patterns, or Sleep Related Breathing Disorder (SRBD) are being recognized by the physicians. Sleep related symptoms occur in about 40% of cases in patients with COPD[1]. Sleep related breathing disorders constitute the greatest number of disorders of sleep in patients treated by sleep medicine, pulmonary, and general practitioners in the outpatient setting. SRBD is associated with considerable morbidity.

Sleep related breathing disorders (SRBD) are associated with daytime sleepiness, hypertension, impaired cognitive function and increase in the overall mortality. OSA is can also occur in non-obese individuals. The preconception that OSA was restricted to older obese patients who snore leads to much under diagnosis of the disease in other populations at risk. OSA

PROFORMA

Name:

Age:

Sex:

OPno:

IP no:

Address:

Duration of symptoms:

Smoking;
Alcohol:

Last Hospitalisation:

Medications:

BP:

Pulse:

Spo2:

ABG : pH- pCO2 PaO2- Hco3- Na- k- Cl-

Neck circumference-

Chest circumference

Abdominal circumference-

Ht-

Wt-

Bmi-

comorbidities -

HB:

CXR :

PFT:

Clinical Diagnosis:

Polysomnography:

TST:

NREM 1 ;

NREM 2:

NREM 3:

NREM 4:

REM :

Respiratory effort related - Arousal Count:

Movement :

Position:

Sleep Duration :

Sleep Latency:

Sleep Efficiency:

SPO2 – DAYTIME:

Spo2- NOCTURNAL:

Minimum Nocturnal Saturation:

Wakefulness after sleep onset :

ObstuctiveApnoea:

Central Apnoea ;

Hypapnoea:

AHI :

ESS:

PSQI Score :

FINAL DIAGNOSIS:

Sl.no	Name	age(years)	sex		Lifetime alc	symptoms	smoking(pa	bp	pulse	height(cm)	weight(kg)	BMI	totalbed (T	totalsleep	woso (in m	wasop (% c	sleep laten	NREM1,2(i	NREM %of	NREM3(M	NREM% of	sleep effici	REM(min)	REM%	arousal ind
1	Ponnamal	55	1	0	1	0	150/90	82	161	47	18.13	439	310	78.5	17.9	50.5	225	72.8	37	12	70.61	47	15.2	22.5
2	ladasamy	39	0	0	2	20	120/70	94	173	51	17.04	391.5	232.5	84.5	21.6	74.5	189.5	81.5	13.2	5.7	59.4	29.5	12.8	26.4
3	mookan	68	0	1	1	55	130/70	90	171	59	20.2	369	228	97	26.3	44	168	73.5	19	8.5	61.8	41	18	18.4
4	ponnu	41	0	1	2	35	120/80	88	161	55	21.21	362.5	217.5	96.5	26.6	48.5	171.5	78.9	17	7.8	60.4	29	13.3	28.6
5	kuppusamy	60	0	1	1	64	110/80	103	161	55	21.21	364	218	76.5	21	69.5	153	70.1	22.5	10.4	59.8	42.5	19.5	21.8
6	ethiraj	55	0	0	1	50	100/60	94	162	64	24.3	310	201	46.5	15	54.5	160	76.7	25	12	67.41	42.5	20.3	32.8
7	gopal	61	0	0	2	30	110/80	89	171	61	20.8	384	279	74.5	19.4	30.5	232.5	60.5	14.5	5.1	72.6	32	11.5	22.6
8	balu	64	0	0	1	60	150/90	88	158	68	27.3	423	268	92	21.7	63.5	229.5	85.6	15	5.6	63.3	23.5	8.8	30.6
9	gandhi	65	0	0	1	35	100/80	91	158	55	22.03	369	243	67.5	18.3	58.5	185	76.13	10.5	4.3	62.33	47.5	19.5	30.1
10	kupana	55	0	1	1	33	130/70	78	159	55	21.75	411.5	273	98.5	23.9	39.5	185	67.8	37	13.6	66.37	50.5	18.6	15.9
11	saroja	55	1	0	1	0	130/70	78	160	54	20.09	401.5	277.5	88.5	22	34.5	190	68.4	37	13.4	66.37	50.5	18.2	15.9
12	kanagaraj	69	0	0	1	50	150/90	94	164	48	17.8	528	380.5	93.5	17.7	54.5	281.5	74	62.5	16.5	72.06	36.5	9.5	26.4
13	kittu	52	0	1	2	38	130/70	94	167	58	20.7	417	241.5	124	29.7	51.5	196	81.2	16.5	6.8	58	29	12	38.2
14	muthusamy	61	0	1	2	41	110/80	85	161	60	23.14	408	224.5	119.5	29.3	64	168	74.8	36	16	55.02	36	16	29.6
15	muniyan	63	0	0	1	31	100/60	80	161	58	22.37	411	273	94	22.9	44	212.5	82.5	17	6.2	66.4	41	11.3	28.3
16	kabali	65	0	0	2	28	100/70	81	155	60	24.97	371	213	104	28	54	164	77	15	7	57.5	38	18	33.7
17	perumal	58	0	0	1	30	140/80	85	164	76	28.35	408	209	114.5	28.1	84.5	170	81.4	8	4.1	51.4	30.5	14.5	34.5
18	muthaye	58	1	0	1	0	110/80	94	140	61	30.68	445	297	92	20.7	56	224	75.5	28	9.5	66.2	44.5	15	31.1
19	puusotham	61	0	0	1	45	150/90	91	173	63	20.38	401	257.5	105	26.2	38.5	164	63.8	40.5	15.7	64.28	52.5	20.5	17.1
20	malliga	56	1	0	2	0	120/80	91	148	58	26.4	382	215.5	110	28.8	56	149.5	69.4	24	11.2	56.4	42	19.4	32.4
21	kasi	63	0	0	2	51	110/70	88	169	63	22.05	403.5	236	107	26.5	60.5	171	72.3	28.5	12.1	58.48	36.5	15.6	14.8
22	aasaithamb	52	0	0	1	30	130/80	85	168	59	20.9	388	314	31.5	8.1	42.5	254.5	81	18	5.8	80.92	41.5	13.24	15.5
23	nandan	48	0	1	2	23	130/80	88	165	48	17.63	391.5	203	132	33.7	56.5	164.5	81	17	8.4	51.9	21.5	10.6	31.5
24	kuppan	58	0	0	2	0	140/90	93	168	59	20.9	419	233	137	32.7	49	184	79	21	9	55.6	28	12	38.1
25	velu	58	0	0	1	30	110/80	90	172	59	19.9	512	409	57	11.1	46	278.5	68.12	51	12.4	79.8	79.5	19.5	15.1
26	kamala	57	1	0	2	0	120/80	80	164	48	17.84	440	270.5	125.5	28.5	44	184.5	68.2	36.5	13.6	61.48	47	17.5	20.8
27	karupan	52	0	0	2	30	110/60	95	170	61	21.1	407.5	218.5	120.5	29.6	68.5	171.5	78.5	17.5	8	53.6	29.5	13.5	22.6
28	ranganatha	68	0	0	1	58	140/70	96	164	51	18.7	428	327.5	62.5	14.6	38	206.5	63	52.5	16.5	76.5	68.5	21	16.4
29	chinasamy	50	0	0	1	31	130/60	94	158	45	18.02	388	228.5	88.5	22.8	71	145	63.45	25	11	58.5	33.5	14.6	31.4
30	arokyam	48	0	0	2	15	110/80	90	163	63	23.71	391	229.5	84.5	21.6	77	146.5	64	34.5	15	58.69	48	21	36.4
31	ponnima	52	1	0	2	0	120/60	90	145	54	25.6	388	170.5	76.5	19.7	91	125.5	73.6	17	10	43.9	28	16.4	39.4
32	durai	58	0	1	1	21	140/90	89	160	50	19.53	407.5	244	110	27	53.5	201	82.5	13.5	5.5	59.87	29	12	29.3
33	elumalai	50	0	0	1	39	130/80	88	168	48	17.01	369.5	209.5	91.4	24.7	68.5	165.5	79	20.5	9.4	56.63	24.5	11.6	21.4
34	kandan	64	0	0	1	34	110/60	90	171	65	22.22	485	302.5	98	20.2	45.5	226	74.8	33	11	62.57	43	14.2	16.8
35	rathinam	61	0	1	2	30	110/80	87	155	58	24.14	488	294	152.5	31.3	41.5	222.5	75.6	23.5	8	60.24	48	16.4	25.9
36	selvan	70	0	0	1	44	100/70	91	163	70	26.3	385.5	266.5	83.5	21.7	35.5	202.5	75.9	28	10.5	63.19	36	13.6	36.4
37	soundaran	47	0	0	1	22	130/80	91	159	55	21.7	469.5	333.5	96.5	20.6	39.5	212	63.7	50	14.9	71.5	71.5	21.4	13.4
38	mohan	49	0	0	1	49	130/80	79	169	55	19.25	401	327	45	11.2	29	210.5	64.4	49	15	81.5	67.5	20.6	19.5
39	kandasamy	55	0	0	2	29	120/80	67	163	49	18.42	389	309	54.5	14	25.5	221	71.5	43	14	79.4	44.5	14.5	21.9
40	muniyappa	57	0	0	1	0	110/70	79	159	56	22.15	445	367	49	11	29	238	65	62	17	82.4	66	18	20.8
41	raghu	50	0	0	1	35	140/70	83	143	49	23.9	384	279.5	59	15.4	45.5	201	71.91	33	11.8	72.78	45	16.1	14.8
42	rakaye	54	1	0	1	0	110/80	93	141	52	26.1	421	325.5	64.5	15.3	31	228	70	39	12	77.31	58.5	18	16.1
43	kannaka	60	1	0	1	0	140/90	96	151	49	21.49	382.5	225.5	131	34.2	26	175	77.6	14.5	6.4	58.98	36	16	29.67
44	ponraj	68	0	0	1	38	139/70	94	158	59	23.63	386	272	71.5	18.5	41.5	213.5	78.6	20	7.4	70.46	38.5	14	21.4
45	kutty	72	0	1	2	55	150/80	88	170	62	21.4	488	257.5	130	26.6	106.5	211	82	6.5	2.5	52.7	40	15.5	36.4

SEX 0- Male , 1- Female
Lifetime Alcohol 0- Absent, 1- Present
Symptoms 1- Breathlessness, 2- Breathlessness + Wheeze
Diagnosis 1- COPD with Respiratory Failure ,2- COPD with PT Sequelae Respiratory Failure, 3-COPD with respiratory Failure With OSA,4- COPD with out Respiratory Failure ,5-COPD with PT Sequelaewithout Respiratory Failure,6-COPD without Respiratory Failure with OSA,

Diabetes 0- absent, 1- present
Snoring 0- absent, 1- present
Chocking, 0- absent,1- present,
nocturnal awakenings, 0- absent, 1- present.

dignosis	daytime Sp	nocturnal s	minimal no	Nocturnal I	AHI	ESS score	PSQIscore	pH	Paco2	PaO2	HCO3	FEV1%	FEV1(L)	FVC(L)	FEV1/FVC	Neck Circu	Waist Circ	Diabetes	Snoring	Nocturnal A	Morning H	Chocking Episodes
4	96	91	88	1	2.5	11	11	7.426	35.59	80.65	25.67	20	0.7	0.88	0.62	33	73	0	1	0	0	0
2	92	86	80	1	0	21	12	7.362	50.86	61.38	30.47	23	0.87	1.46	0.6	32	74	0	0	0	0	0
1	93	90	71	0	0	8	7	7.368	47.4	66.89	29.67	28	0.8	1.77	0.45	35	75	0	0	1	0	0
2	94	85	81	1	3.1	5	7	7.619	48.9	67.54	28.72	29	0.93	1.9	0.48	34	78	0	0	1	0	0
1	94	85	79	1	0	8	14	7.368	48.6	68.43	30.43	23	0.64	0.91	0.7	32	70	1	1	1	0	0
1	92	90	78	0	4.4	12	12	7.361	48.84	62.87	29.4	31	0.9	1.66	0.54	34	85	1	1	1	1	0
1	94	91	68	0	0	9	9	7.354	50.4	64.86	32.41	35	1.08	2.07	0.52	35	77	0	0	1	1	0
3	92	86	71	1	12.6	22	13	7.356	55.62	64.78	30.21	25	0.61	1.102	0.55	42	104	0	1	1	1	1
1	93	80	71	1	3.8	11	9	7.361	49.84	70.84	28.67	21	0.5	1.16	0.43	33	71	0	1	1	0	0
4	93	91	83	0	2.1	8	5	7.377	37.76	74.41	25.53	33	0.83	1.25	0.66	30	72	0	0	1	1	0
5	93	87	83	1	2.1	8	5	7.377	37.76	74.41	25.53	25	0.52	0.86	0.6	35	80	0	0	1	0	0
1	93	91	73	0	4.1	6	5	7.364	49.4	64.88	31.2	34	0.87	1.93	0.45	33	73	0	0	0	0	0
1	91	84	69	1	3.1	16	15	7.354	56.54	65.43	30.85	24	0.76	1.35	0.56	31	77	0	1	1	1	0
1	92	81	70	1	0	11	14	7.365	54.67	64.56	32.21	22	0.58	1.18	0.48	34	84	0	1	1	1	0
1	94	85	74	1	2.8	18	14	7.355	50.54	63.86	29.68	24	0.62	1.06	0.58	31	75	0	0	1	1	0
1	91	83	71	1	3.8	17	12	7.364	48.67	68.42	28.19	30	0.68	1.45	0.6	36	85	0	0	1	0	0
3	91	83	77	1	18.4	20	15	7.366	56.5	62.45	31.44	30	0.86	1.432	0.6	40	101	0	1	1	1	1
1	91	84	75	1	4.6	8	13	7.351	50.87	63.45	30.69	25	0.47	1.02	0.46	43	98	1	1	1	0	0
4	94	89	83	1	3.7	7	9	7.385	38.56	70.48	26.78	22	0.69	1.07	0.64	31	81	0	0	1	0	0
1	93	84	71	1	4.1	19	10	7.356	56.67	64.09	31.45	24	0.5	0.93	0.64	38	83	1	0	1	1	0
4	95	87	81	1	0	14	7	7.368	36.48	73.88	24.5	22	0.69	1.5	0.46	32	78	0	0	1	0	0
1	93	90	80	0	0	4	5	7.361	47.89	64.47	28.9	30	0.96	1.93	0.5	34	81	1	0	0	0	0
2	92	85	65	1	0	19	11	7.358	50.59	63.86	29.48	21	0.67	1.28	0.52	30	77	0	0	1	1	0
2	91	83	78	1	4.4	7	11	7.354	51.54	63.45	30.16	19	0.59	0.86	0.68	35	74	0	0	1	1	0
1	94	91	78	0	3.2	9	6	7.365	48.54	64.98	28.78	33	1.06	2.16	0.49	30	77	0	0	0	0	1
4	96	90	88	0	3.4	10	10	7.408	40.78	73.09	24.87	29	0.55	1.52	0.55	32	77	0	0	1	0	0
2	94	83	74	1	3.9	8	17	7.358	54.58	63.43	30.48	19	0.62	1.63	0.4	35	76	0	1	1	1	0
1	94	90	71	0	4.1	7	8	7.368	49.52	68.42	29.87	28	0.77	1.28	0.6	31	72	0	0	0	0	0
2	95	84	71	1	3.6	11	11	7.361	47.78	68.89	28.76	30	0.92	1.53	0.6	30	70	0	0	1	0	0
2	92	90	69	0	4.8	13	16	7.362	49.48	62.61	31.48	31	0.91	1.42	0.68	37	80	0	1	1	0	0
1	92	85	80	1	2.8	17	13	7.359	56.67	65.9	29.61	28	0.63	1.14	0.55	34	88	0	1	1	1	0
2	93	91	71	0	3.3	17	8	7.359	48.58	64.48	29.37	30	0.92	1.73	0.53	31	73	0	0	1	0	0
2	94	84	77	1	2.7	21	11	7.361	49.58	62.57	28.34	23	0.75	1.86	0.42	31	71	1	1	1	0	0
4	95	91	81	0	2.6	5	4	7.374	40.43	70.3	25.57	24	0.72	1.1	0.65	33	80	1	1	1	0	0
4	93	87	81	1	3.9	14	9	7.386	40.98	70.45	25.1	26	0.62	1.1	0.68	35	84	0	0	1	1	0
6	95	90	80	0	15.8	22	17	7.389	38.54	72.38	26.51	30	0.74	1.54	0.48	34	98	0	1	1	0	0
2	95	89	85	1	0	19	7	7.351	47.55	62.67	29.65	21	0.62	1.37	0.45	34	81	1	0	0	0	0
4	95	92	90	0	0	12	7	7.377	37.76	74.41	25.53	21	0.55	1.18	0.59	31	73	0	0	0	0	0
4	95	90	87	0	0	7	9	7.385	38.56	70.48	26.78	29	0.78	1.67	0.47	31	76	0	0	0	0	0
5	96	91	85	0	3.8	10	4	7.374	40.43	77.54	25.57	26	0.8	1.32	0.52	35	79	0	0	0	0	0
5	94	91	83	0	3.6	7	9	7.416	37.59	79.65	25.67	30	0.61	1.35	0.45	36	83	0	0	1	0	0
1	94	90	80	0	2.8	9	7	7.362	49.54	70.24	28.67	28	0.56	1.07	0.52	32	87	0	0	0	0	0
1	93	87	81	1	0	5	13	7.354	50.4	65.89	28.67	31	0.67	1.52	0.44	35	79	0	1	1	0	0
1	90	86	80	1	3.6	8	10	7.359	46.5	62.12	32.42	25	0.58	1.52	0.4	33	89	0	0	1	0	0
1	91	83	72	1	2.4	17	12	7.361	55.8	62.43	30.65	32	0.87	1.4	0.62	36	90	0	0	1	1	0

The Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the
most accurate reply for the majority of days and nights in the past month. Please answer all questions. During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times week (3)
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):				

6. During the past month, how often have you taken medicine (prescribed or “over the counter”) to help you sleep?

7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?

Very good (0) Fairly good (1) Fairly bad (2) Very bad (3)

9. During the past month, how would you rate your sleep quality overall?

Component 1 #9 Score.....

C1_____

Component 2 #2 Score (≤15min=0; 16-30 min=1; 31-60 min=2, >60 min=3) + #5a Score
(if sum is equal 0=0; 1-2=1; 3-4=2; 5-6=3)..... C2_____

Component 3 #4 Score (>7=0; 6-7=1; 5-6=2; <5=3).....
C3_____

Component 4 (total # of hours asleep)/(total # of hours in bed) x 100
>85%=0, 75%-84%=1, 65%-74%=2, <65%=3..... C4_____

Component 5 Sum of Scores #5b to #5j (0=0; 1-9=1; 10-18=2; 19-27=3).....
C5_____

Component 6 #6 Score .....

C6_____

Component 7 #7 Score + #8 Score (0=0; 1-2=1; 3-4=2; 5-6=3).....
C7_____

GLOBAL PSQI SCORE _____

EPWORTH SLEEPINESS SCALE FORM

Instructions: Be as truthful as possible. Print the form. Read the situation in the first column; select your response from the second column; enter that number in the third column. Total all of the entries in the third column and enter the total in the last box.

Situation	Responses	Score
Sitting and Reading	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
Watching Television	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
Sitting inactive in a public place, for example, a theater or a meeting	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
As a passenger in a car for an hour without a break	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
Lying down to rest in the afternoon	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
Sitting and talking to someone	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
Sitting quietly after lunch when you've had no alcohol	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
In a car while stopped in traffic	0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing	
TOTAL SCORE		

CONSENT FORM

I Mr / Mrs / Miss / _____ have understood the procedure read by the Doctors. I in my whole conscious awareness give consent for the procedure. I understand that the procedure is done in good faith for the best therapeutic results possible. I fully understand the consequences of the procedure. I can resign from the study at any point of time.

Signature :

Name :

Date and Time :

Signature of Researcher :

சுயஒப்புதல்படிவம்

ஆய்வுசெய்யப்படும்தலைப்பு:

நீண்டகாலநுரையீரல்செயலிழப்புஉள்ளோர்ரில்தூக்கத்தின்தன்மைமற்றும்தூக்கத்தின்போதுஎர்படும்சுவாசபிரச்சனைகள் .

ஆராய்ச்சிநிலையம்:அரசுநெஞ்சகநோய் மருத்துவமனை ,

தாம்பரம்சானடோரியம், சென்னை.

பங்குபெறுபவரின்பெயர்:

பங்குபெறுபவரின்எண் :

பங்குபெறுபவர் ( ) இதனைக்குறிக்கவும் :

மேலேகுறிப்பிடப்பட்டுள்ள ஆய்வின்விவரங்கள்எனக்குவிளக்கப்பட்டது.என்னுடையசந்தேகங்களைக்கேட்கவும்,அதற்க்கானதகுந்தவிளக்கங்களைப்பெறவும்வாய்ப்பளிக்கப்பட்டது.நான்இவ்வாய்வில்தன்னிச்சையாகத்தான்பங்கேற்கிறேன்.எந்தகாரணத்தினாலோஎந்தகட்டத்திலும் எந்தசட்டச்சிக்கலுக்கும்உட்படாமல்நான்இவ்வாய்வில்இருந்துவிலகிக்கொள்ளலாம்என்றும்அறிந்துகொண்டேன்.

இந்தஆய்வுசம்பந்தமாகவோ,இதைச்சார்ந்தமேலும்ஆய்வுமேற்கொள்ளும்போதும் இந்தஆய்வில்பங்குபெறும்மருத்துவர்என்னுடையமருத்துவஅறிக்கையைபார்ப்பதற்குஎண்அனுமதிதேவையில்லைஎனஅறிந்துகொள்கிறேன்.நான் ஆய்வில் இருந்துவிலகிக்கொண்டாலும்இதுபொருந்தும்எனஅறிகிறேன்.

இந்தஆய்வுமூலம்கிடைக்கும்தகவல்களையும்பரிசோதனைமுடி
வுகளையும்மற்றும்சிகிச்சைதொடர்பானதகவல்களையும்மருத்துவர்மே
ற்க்கொள்ளும்ஆய்வில்பயன்படுத்திக்கொள்ளவும்அதைப்பிரசுரிக்கவும்
எண்முழுமனதுடன்சம்மதிக்கிறேன .

இந்தஆய்வில்பங்குகொள்ளஒப்புக்கொள்கிறேன்.எனக்குக்கொடுக்
கப்பட்டஅறிவுரைப்படிநடந்துகொள்வதுடன்இந்தஆய்வைமேற்கொள்ளு
ம்மருத்துவஅணிக்குஉன்னமையுடன்இருப்பேன்என்றுஉறுதிஅளிக்கின்
றேன்.என்உடல்நலம்பாதிக்கப்பட்டாலோஅல்லதுஎதிர்பாராதவழக்கத்தி
ற்குமாறானநோய்க்குறிதென்பட்டாலோஉடனேஅதைமருத்துவஅணிக்
குத்தெரிவிப்பேன்எனஉறுதிஅளிக்கிறேன் .

பங்குபெறுபவரின்கையொப்பம் ----------

இடம் ----------

தேதி ----------

கட்டைவிரல்ரேகை

பங்குபெறுபவரின் பெயர்மற்றும்விலாசம் ----------

ஆய்வாளரின்கையொப்பம் ----------

இடம் ----------

தேதி ----------

ஆய்வாளரின்பெயர் ----------

நோயாளிக்கானதகவல்படிவம்

மதிப்பிற்குரியஐயா / அம்மையீர்,

உங்கள்விருப்பத்தின்பேரில்

நீண்டகாலநுரையீரல்செயலிழப்புஉள்ளோர்ரில்தூக்கத்தின்தன்மைமற்றும்தூக்கத்தின்போதுஎர்படும்சுவாசபிரச்சனைகள் .

பங்கேற்கும்படிஅன்புடன்கேட்டுக்கொள்கிறோம்.
இந்தஆய்வில்ஆரய்ச்சிநோக்கத்துக்காகதாங்கள்பரிசோதனைக்குஉட்படுத்தப்படுவீர்கள்.தகுந்தசிகிச்சைதங்களுக்குதொடங்கப்படும்.தங்களுக்குஇந்தஆய்வில்பங்கேற்கவிருப்பம்இருந்தால்தாங்கள்அருள்கூர்ந்துஒப்புதல்படிவத்தைப்படித்துப்பார்த்துக்கையொப்பம்இடும்படிக்கேட்டுக்கொள்கிறேன்.