

**DISSERTATION ON “A STUDY ABOUT PRE-AND POST
OPERATIVE EVALUATION OF APNEA HYPOPNEA INDEX
IN POLYSOMNOGRAPHY IN ADULT OBSTRUCTIVE SLEEP
APNEA SYNDROME PATIENTS”**

*Dissertation submitted in partial fulfillment of the
regulations for the award of the degree of*

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DEPARTMENT OF OTORHINOLARYNGOLOGY

MADURAI MEDICAL COLLEGE

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THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

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MAY 2022

CERTIFICATE BY GUIDE

This is to certify that this dissertation titled
**“A STUDY ABOUT PRE AND POST OPERATIVE EVALUATION
OF APNEA HYPOPNEA INDEX IN
POLYSOMNOGRAPHY IN ADULT OBSTRUCTIVE SLEEP APNEA
HYPOPNEA SYNDROME PATIENTS”** submitted by **Dr. P. CINDHUJA**
to the faculty of Department of ENT, The Tamil Nadu Dr. M.G. R Medical
University, Chennai in partial fulfillment of the requirement for the award of
MS Degree Branch IV OTORHINOLARYNGOLOGY research work
carried out by her under our direct supervision and guidance from November
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This is to certify that this dissertation work “ **A STUD ABOUT PRE AND POSTOPERATIVE EVALUATION OF APNEA HYPOPNEA INDEX IN POLYSOMNOGRAPHY IN ADULT OBSTRUCTIVE SLEEP**

APNEA- HYPOPNEA SYNDROME PATIENTS ” is a bonafide research work done by **Dr. P.CINDHUJA**

for the award of degree of M.S. Branch of otorhinolaryngology, post graduate student, Dept of ENT, Madurai Medical college and Govt. Rajaji Hospital, Madurai under my guidance and supervision

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**“A STUDY ABOUT PRE AND POST OPERATIVE EVALUATION
OF APNEA HYPOPNEA INDEX IN POLYSOMNOGRAPHY IN
ADULT OBSTRUCTIVED SLEEP APNEA-HYPOPNEA
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Department Of Otorhinolaryngology, Madurai Medical College and Govt
Rajaji Hospital, Madurai during the period of November 2020 to
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Date:

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ABBREVIATIONS

OSA	:	Obstructive Sleep Apnea
OSAHS	:	Obstructive sleep apnea hypopnea syndrome
AHI	:	Apnea Hypopnea Index
TFT	:	Thyroid Function Test
EEG	:	Electroencephalogram
EMG	:	Electromyogram
EOG	:	Electro oculogram
REM	:	Rapid Eye Movement
NREM	:	Non Rapid Eye Movement
SWS	:	Slow Wave Sleep
RERA	:	Respiratory Effort Related Arousal
CPAP	:	Continuous Positive Airway pressure

BMI	:	Body Mass Index
UPPP	:	Uvulopalatopharyngoplasty
DISE	:	Drug Induced Sleep Endoscopy
ESS	:	Epworth Sleep Scale
PSG	:	Polysomnography
TFT	:	Thyroid Function Test
SDB	:	Sleep Disordered Breathing
AASM	:	American Academy of Sleep Medicine
SL	:	Sleep Latency
TST	:	Total Sleep Time
SE	:	Sleep Efficiency
VPI	:	Velopharyngeal Insufficiency
TRT	:	Total Recording Time
RAS	:	Reticular activating system
HMS	:	Hyoidmyotomy with suspension
MMP	:	Maxillo-mandibular advancement procedures

INDEX

S.No.	Content	Page No
1.	Introduction	1
2.	Aims and Objectives of the Study	3
3.	Review of literature	4
	History	4
	Sleep	5
	Anatomy of structures contributing collapse	13
	Pathophysiology	19
4.	Clinical examination of OSA Patients	30
5.	Treatment	44
6.	Materials and Methods	56
7.	Statistical Analysis	63
8.	Results	64
9.	Analysis and Discussion	86
10	Conclusion	95
11.	Annexure Ethical Committee Anti Plagiarism Screen Shot Patient Information Sheet Patient Consent Form Proforma Master Chart	

INTRODUCTION

Obstructive sleep apnoea (OSA) can be regarded as a condition characterized by repetitive upper airway obstruction leading to sleep fragmentation, cardiovascular stimulation and oxygen desaturation during sleep. These together lead to symptoms such as snoring, unrefreshing sleep, excessive daytime sleepiness (EDS) and the increased risk of cardiovascular disease, hypertension, insulin resistance and road traffic accidents. Any individual with time may oscillate within a spectrum of sleep disordered breathing, from intermittent simple snoring, to chronic heavy snoring, to upper airway resistance syndrome (UARS), to mild OSA, to moderate OSA, to severe OSA or to obesity hypoventilation syndrome (OHS).

OSA is characterized by complete breath-holds (apnoeas) and partial breath-holds (hypopnoeas) – With the present obesity epidemic, ear, nose and throat (ENT), hypertension, obesity, diabetes and sleep specialists are witnessing a large increase in the prevalence of OSA in their practice. The medical management of OSA, especially associated comorbidities, diagnosis and continuous positive airway pressure (CPAP), all relevant to the work environment of ENT specialists. Eventhough the OSA patients are classically treated with lifestyle modifications and medical management, poor compliance in these patients warranting surgical management

In our study after vigorous investigations in OSA patients, various surgeries has been done addressing the level of obstruction and improvement after surgery both in terms of subjective and objective results were analysed.

AIM AND OBJECTIVES OF THE STUDY

- 1) To study about the pre and post operative apnea hypopnea index in polysomnography in adult obstructive apnea hypoapnea syndrome patients

- 2) To study the effectiveness of various surgeries for obstructive sleep apnea.

HISTORY

Historical background

Disordered breathing during sleep and the resultant excessive daytime sleepiness as long have been an enigma in modern medicine. Charles Dickens, a reward English novelist he introduced a character named as Joe, the fat boy, who was obese, always sleepy and huge snorer in his work titled The Posthumous Papers of the Pickwick Club in 1836. In later years medical fraternity adopted the term Picwickian Syndrome to denote syndromic association of obesity, somnolence, and hypoventilation.

Obesity hypoventilation syndrome was first reported by Burwell In 1956 Following this article, physicians presumed that alveolar hypoventilation was responsible for the excessive daytime somnolence.

Later in 1966, Gastaut documented that the upper airway obstruction fragmented sleep by brief arousals with the use of Polysomnography. He thus proved that the reason for the excessive day time sleepiness was sleep fragmentation in obstructive sleep apnea1970 –Elio Lugaresi described about “OSA syndrome”.

1983 – Riley described cephalometric evaluation for OSA.

1985 – Fujita described “Uvulopalatopharyngoplasty (UPPP)”.

1986 – Colin Sullivan, a young Australian medical researcher developed nasal “Continuous Positive Airway Pressure”.

1990- Dr.Murray John developed ESS

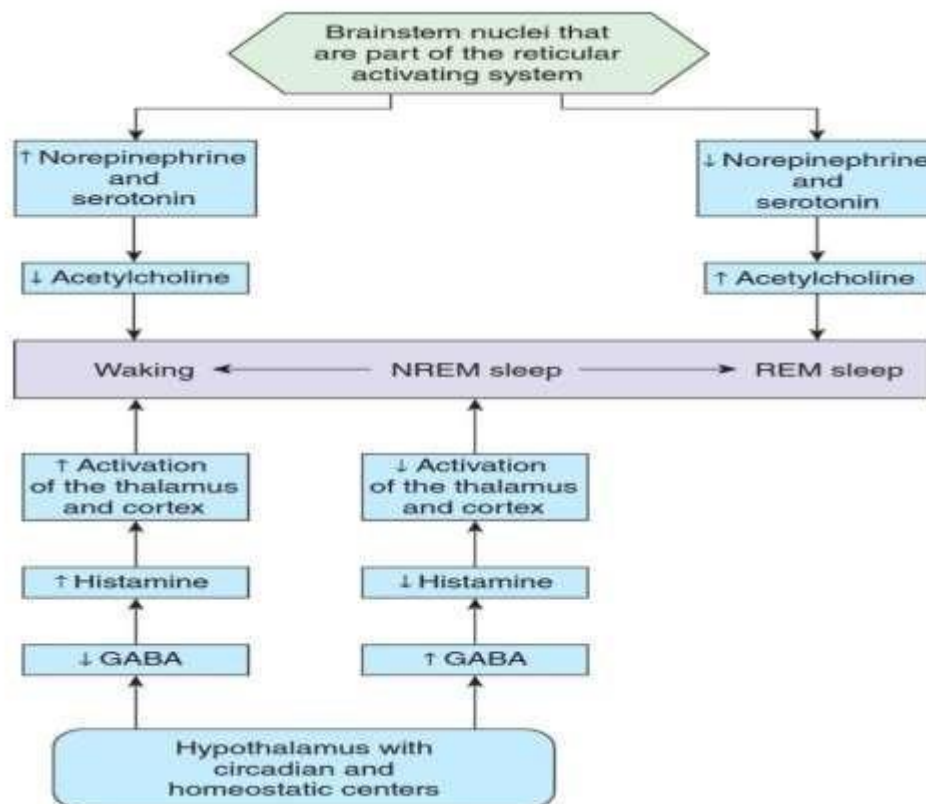
SLEEP

Sleep : There is a “temporary state of unconsciousness from which subject can be arouse by appropriate sensory or other stimuli and it is regulated by RAS”..

Theory of sleep and wakefulness

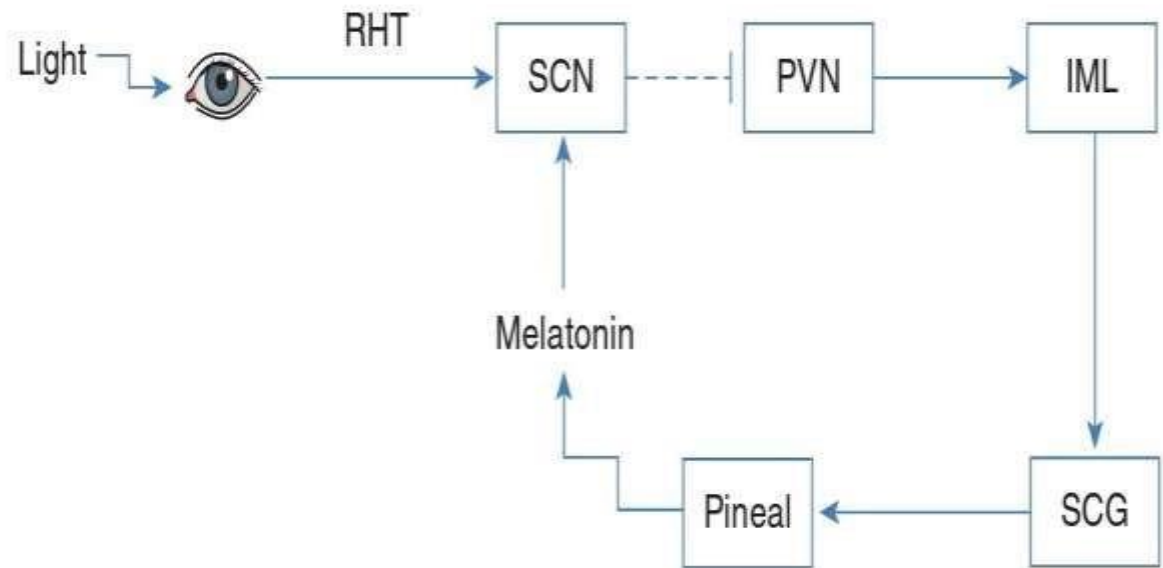
1) Passive theory:

“Discharge from RAS during prolonged hours of wakefulness leading to fatigue of RAS thereby inducing sleep.”



2) Active theory:

“Serotonin from raphe fibres” inhibits RAS thereby “Melatonin from pineal gland” inhibits reticular activating system promoting sleep .



3. CIRCADIAN RHYTHM- Light Dark Cycle

Suprachiasmatic nuclei (SCN) plays a major role in the entrainment process Secretion of melatonin. This rhythm is entrained to light/dark cycles by neurons in the retina. Light signals are relayed via the retinohypothalamic (RHT) fibers to the SCN. GABAergic neurons in the SCN inhibit neurons in the hypothalamic paraventricular nucleus (PVN) which then reduces the activity of sympathetic preganglionic neurons in the spinal intermediolateral nucleus (IML). These sympathetic preganglionic neurons innervate

postganglionic neurons in the superior cervical ganglion (SCG) that regulate release of melatonin from the pineal gland.

SLEEP is Alternating reciprocal activity of different groups of neurons in the ascending arousal system. wakefulness and rapid eye movement (REM) sleep are at opposite extremes. When the activity of norepinephrine- and serotonin-containing neurons (locus coeruleus and raphé nuclei) is dominant, activity in acetylcholine-containing pontine neurons is reduced.

This pattern of activity contributes to the appearance of the awake state. The reverse of this pattern leads to REM sleep. When there is a more even balance between the activity of the aminergic and cholinergic neurons, non-REM sleep occurs.

The orexin released from hypothalamic neurons may regulate the changes in activity in these brainstem neurons. An increased release of GABA and reduced release of histamine increase the likelihood of nonREM sleep via deactivation of the thalamus and cortex. Wakefulness occurs when GABA release is reduced and histamine

STAGES OF SLEEP

During sleep The EEG recorded varies in a cyclic fashion, which repeats in around every 90 minutes. There are four cycles in normal 6 to 8 hours of sleep. In normal individuals, sleep cycle begins with slow-wave sleep or Non-REM sleep. There are four stages of slow wave sleep (stages 1 to 4). A person

when falls asleep, passes sequentially through these four stages and increasingly to deep sleep and then into REM period. With completion of REM phase, sleep cycle completes. The REM phase is followed by the next new cycle, i.e. with stage 1 of non-REM sleep. Thus, the cycle repeats in every 70 to 90 minutes. Throughout the night, people wake up briefly (called stage W) but are typically unaware of being awake.

EEG FEATURES IN SLEEP

During Awake, EEG usually shows desynchronized, high frequency, low amplitude wave. That is known as beta waves in the range of 14 – 30 Hz. During quiet rest with eyes closed, waves range from 8 to 12 Hz, that is called alpha waves. When we were asleep, we encounter into several different states including theta & delta waves which are much slower than those in the wakeful state.

NREM SLEEP:

When we fall asleep it enters into NREM sleep which is further subdivided into four stages.(fig 2)

Stage 1(transitional stage): characterized by

- a) Loss of alpha activity
- b) Appearance of a low voltage mixed frequency EEG pattern with prominent theta activity (3-7 cps) and occasional vertex sharp waves may also appear.

Eye movements become slow and rolling

Relaxation of tone of skeletal muscle occurs

Motor activity may persist for a number of seconds. Occasionally sudden muscle contractions can occur.

Stage 2:

After a few minutes of stage 1, sleep usually progresses to stage 2 which is the first stage of real sleep occurring about 20 min. Characterised by

- **K complexes** - A typically large and slow (2Hz or slower) EEG wave starting with a negative sharp wave followed immediately by a positive wave.
- **Sleep spindles** - Short bursts of 12-14Hz lasting between 0.5 and 1.5 seconds

These phenomenon occur with a background of mostly theta and some scattered delta wave activity.

Stages 3 and 4:

- a) Stage 2 is generally followed by a period comprises of stages 3 and 4. Slow waves (< 2 cps in humans) appear during these stages, which are subdivided according to the proportion of delta waves in the epoch.
- b) In stage 3, there is a minimum of 20% and not more than 50% of the epoch time occupied by slow waves.
- c) In stage 4 there is greater than 50% of the epoch showing slow wave activity.

Stage 3 and 4 are also referred to as slow wave sleep (SWS), delta sleep, or deep sleep, since arousal threshold increases incrementally from stages 1 through 4. Eye movements cease during stages 2-4, and EMG activity decreases further.

REM SLEEP:

Also called as paradoxical sleep. In infants it is called as active sleep because of phasic muscle twitches. Characterized by

- “Activated” or “desynchronized” EEG (relatively low voltage mixed frequency).
- Bursts of rapid eye movements.
- Suppression of EMG activity in skeletal muscles.

REM sleep can be divided into

a) Tonic(persistent) b) Phasic (episodic) component Tonic aspects of REM sleep include “the activated EEG similar to that of stage 1, which may exhibit increased activity in the theta band (37 cps) and a generalized atonia of skeletal muscles except for the extraocular muscles and the diaphragm.”

Phasic features of REM include “irregular bursts of rapid eye movements and muscle twitches.”

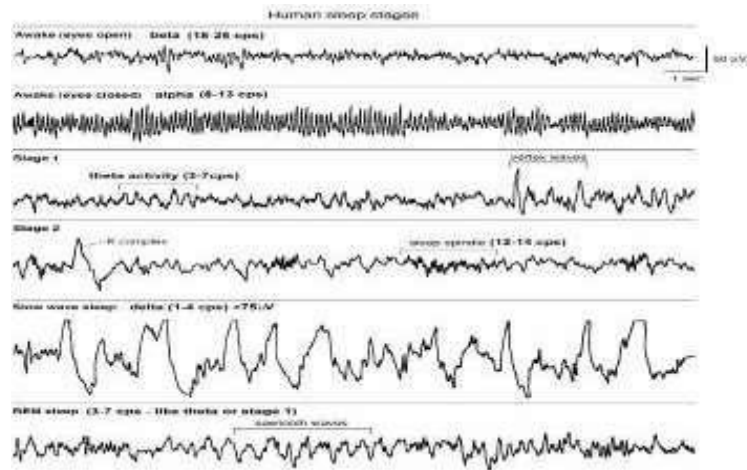


Figure : EEG in different stages of sleep

Distribution of sleep stages

Sleep architecture varies with age. REM sleep occurs in 80% of total sleep time in premature infants and 50% in full-term neonates. In elderly, it is about 25% to 20%. Children have more total sleep time (8–10 h) compared to adults. Healthy adults sleep around 7 to 9 hours per day, which decreases progressively as age advances. A young adult first enters non-REM sleep, then passes through stages 1 and 2. After this, they spend 70–100 min in stages 3 and 4. Then sleep becomes lighter, and the next REM period follows. This cycle is repeated at intervals of about 90 min throughout the night. Thus, 4–6 REM periods occur per night, alternating with NREM & REM.

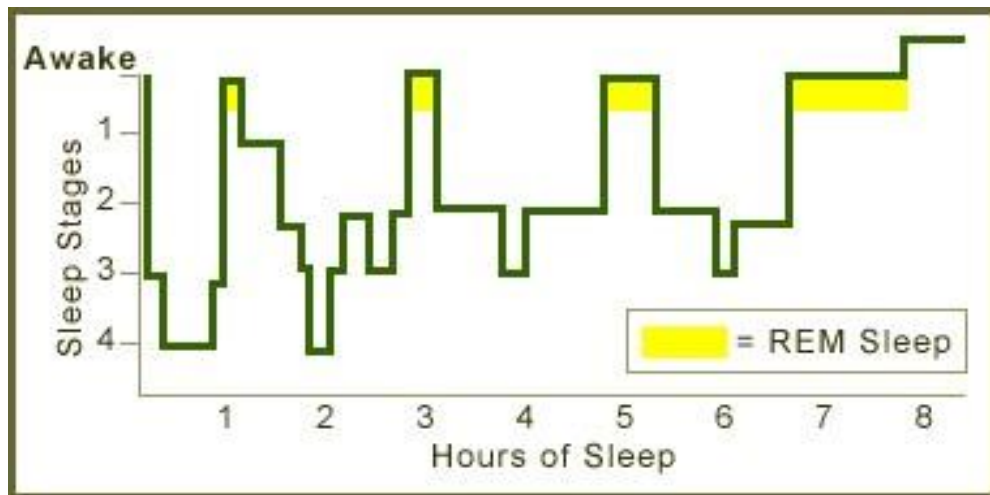


Figure: Times spent in different stages of sleep

Importance of sleep

Sleep is needed to maintain the metabolic-caloric balance, thermal equilibrium, and immune competence. If humans are awakened every time they show REM sleep and then permitted to sleep without interruption, they show a great deal more than the normal amount of REM sleep for a few nights.

ANATOMICAL STRUCTURES CONTRIBUTING

UPPER AIRWAY OBSTRUCTION

Pharyngeal level

There is collapse of various pharyngeal soft tissue structures especially that of velopharynx, oropharynx, and hypopharynx in addition to soft palate vibrations in OSA patients. Based on the different sites of pharyngeal collapse, “OSA patients are structurally classified as :

Type-1 collapse and narrowing in the retropalatal (velopharyngeal) region.

Type-2 collapse and narrowing in both retropalatal and retroglottal regions .

Type-3 collapse and narrowing in the retroglottal region alone” .

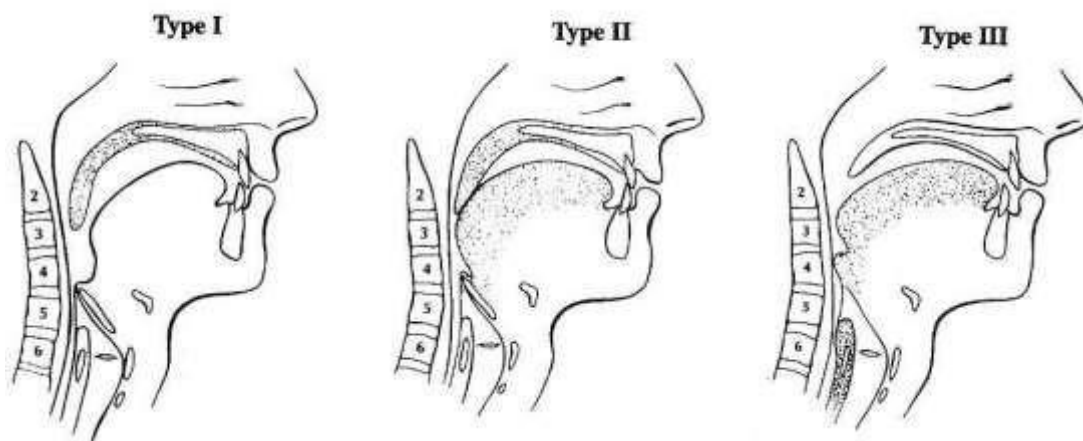


Figure : Sites of collapse

Soft palate

Soft palate (velum) is more complex anatomical structure comprising of palatal aponeurosis, fat, lymphatic tissue, and muscles .

- 1) Palatoglossus
- 2) Musculus uvulae
- 3) Superior fibres of superior constrictor muscle
- 4) Tensor veli palatine
- 5) Levator veli palatine

Its role in deglutition is swallowing, blowing, speech(vowels, non nasal consonants).During deglutition soft palate become taut which makes the tongue to press against the soft palate so it allowing food bolus to squeezed into oropharynx. Then soft palate is elevated posterosuperiorly to press against the posterior pharyngeal wall due to the action of levatorveli palate muscle to prevent bolus enter into nasal cavity. In addition to it closure is augmented by medial convergence of lateral pharyngeal wall.

Action of muscles

- **Tensor Veli Palati:** Tensor of soft palate and opens ET during swallow and yawn
- **Levator Veli Palati:** Elevate the soft palate.
- **Palatoglossus:** Elevate the posterior part of tongue and approximates soft palate and tongue.
- **Palatopharyngeus:** Elevation of the pharyngeal wall anterosuperiorly and medially during deglutition.

- **Musculus Uvulae:** by this action uvula pulled up and shorten it. “All the muscles of soft palate were innervated by pharyngeal plexus except tensorveli palate which is innervated by trigeminal nerve.

PALATOPHARYNGEAL SPHINCTER(PASSAVANT’S MUSCLE)

“It formed by anterosuperior surface of palatine aponeurosis to blend with upper border of superior pharyngeal constrictor to encircle the pharynx as a sphincter. It acts along with palatopharyngeus and levator palate to help in closure of pharyngeal isthmus ”.

TONGUE MUSCLE

Intrinsic muscles:

- 1) Superior longitudinal muscles
- 2) Inferior longitudinal muscles
- 3) Transversus linguae
- 4) Verticalis linguae

Extrinsic muscles

- 1) Pulls tongue upwards and backwards.- Styloglossus
- 2) Palatoglossus -Muscles of both side acting together bring the palatoglossal arches together closing the aperture from oral cavity to pharynx.
- 3) Tongue Protrudes (safety muscle of tongue)- genioglossus

4) Depresser of tongue- Hyoglossus

“All muscles of tongue are supplied by hypoglossal nerve except palatoglossus which is supplied by cranial part of accessory nerve”.

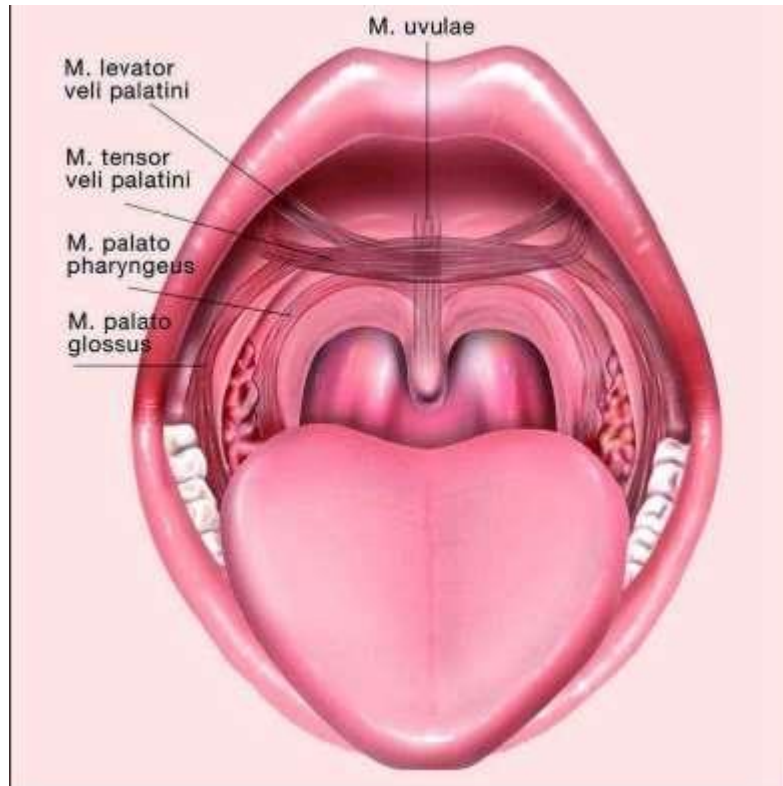


Figure : Muscles of soft palate

At Nasal level

Importance of nasal breathing in respiration is increasing the resistance about 50% .Nasal airway resistance mainly contributed by inferior turbinate , nasal valve and nasal septum.

Inferion turbinate

It composed of concha which is a separate bone and covered by respiratory epithelium and the sub epithelium of infturbinate contains a rich cavernous venous plexus (erectile tissue)

Nasal valve

External and internal nasal valves which are critical to nasal airflow.

The external nasal valve

Comprised of the septum medially, alar rim (lateral crus, sesamoid complex and fibrofatty tissue) laterally and nasal sill inferiorly. Anatomical abnormalities or compromise in the structural integrity of these components can cause external valve narrowing, stenosis or dynamic valve collapse that is exacerbated during inspiration.

The internal nasal valve,

Narrowest portion of the nasal cavity and is bounded by the septum medially, caudal edge of the upper lateral cartilage and head of the inferior turbinate laterally and nasal floor inferiorly. The apex of the internal nasal valve is approximately 10–15 degrees. Changes in the relationship of any structure within this space can cause symptoms of nasal obstruction

The nasal septum
The nasal septum serves many functions, including separation of the nasal airway into two nasal cavities, support of the nasal dorsum, and maintenance of the nasal tip and forms part of the nasal valves. Deviation of the nasal septum can lead to significant nasal airway obstruction and cosmetic deformity. The nasal septum consists of a bony, cartilaginous and membranous portion.

PATHOPHYSIOLOGY

Obstructive sleep apnea/hypopnea syndrome (OSAHS) prevalent disorder affecting 2–4% in men and 2% in women. It is characterised by “recurrent sleep induced collapse of the pharyngeal airway leading to hypercapnia, hypoxaemia with arousal from sleep leads to re-establish the patency of airway”. The pathophysiology of OSAHS is believed that OSAHS patients have an anatomical predisposition to collapse of airway and their patency is maintained during awake by the mechanisms that lead to increasing the activity of pharyngeal dilator muscles. During sleep these protective mechanisms fail thus leading to collapse of the pharyngeal airway behind the palate, tongue, or both.

MECHANICS OF THE PHARYNX:

The pharyngeal airway is a complex structure with functions of swallowing, speech and respiration. To accomplish all these functions the evolution of the structure and function of the pharynx has required certain important revolutionary changes. It has been studied that for production of speech in man, the rigid support of the hyoid bone to the pharynx which is present in many mammals is lost since during evolution of speech, because speech required laryngeal mobility and depending mainly upon on pharyngeal dilator muscle activity to ensure its patency.

However, the Dilator muscle activity of the pharynx is highly variable in each individual which is a product of the intrinsic anatomy and collapsibility of their airway. Thus individuals with an anatomically large airway are less dependent on pharyngeal muscles to maintain airway patency than those individuals with a smaller airway. When the pharyngeal collapse does occur, it usually occurs either in the velopharynx (behind the soft palate), the oropharynx (from the tip of the soft palate to the epiglottis), or both.

Human pharynx can be designed as a collapsible tube, the patency can be described using a „**balance of pressures**” concept. The upper airway size depends on the balance between two following forces.

“Force that collapses the airway tissue (such as negative intraluminal pressure and increased extraluminal pressure). Force that maintains the patency of airway (contraction of pharyngeal dilator muscles)”. The transmural pressure of the pharynx (P_{tm}) is thus “equal to the pressure in the lumen (P_l) minus the surrounding pressure in the tissue (P_{ti}), with the airway lumen becoming smaller as P_{tm} decreases”.

The change in area for a given change in pressure gives the effective elastance of the pharynx. The P_{tm} at which the area of the pharynx equals zero is the closing pressure of the pharynx. Using this model it is measured that normal individuals have the critical closing pressure (P_{crit} , pressure at zero flow) below 28 cm H₂O during sleep while those with mild OSAHS/snoring have a slightly negative P_{crit} value and those with severe

disease have a P_{crit} of 0^{10} . This suggests that in patients with apnea an anatomically predisposed smaller airway is more prone to collapse during sleep when muscle activity may be low.

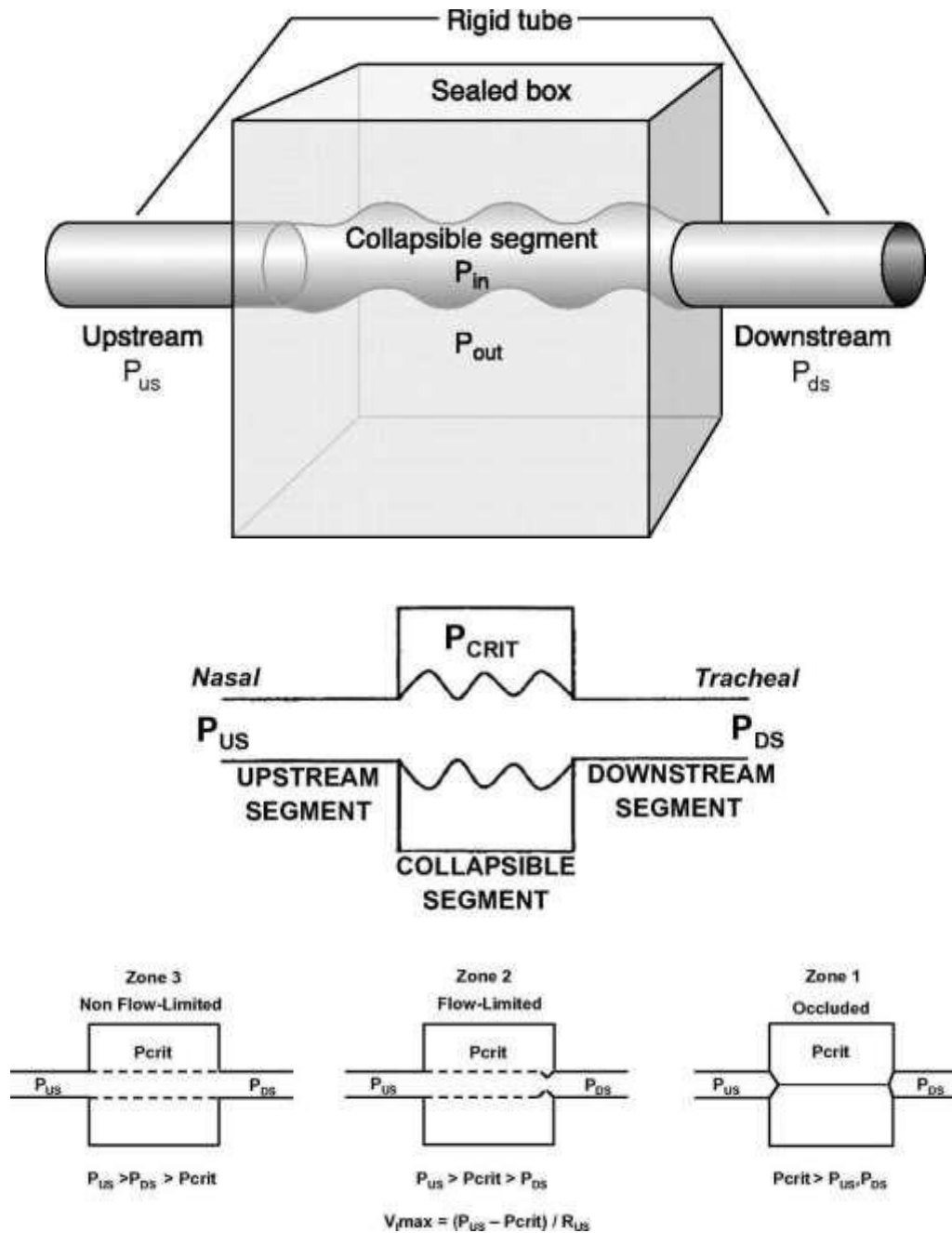


Figure: Balance of pressure concept

VARIANT ANATOMY IN OSA PATIENTS:

Normal individuals and patients with OSAHS have considerable difference in the anatomy of pharyngeal airway which predisposes them to have increased airway resistance and more chance of airway collapsibility. OSAHS patients have

- 1) Reduction in the length of the mandible
- 2) Inferiorly positioned hyoid bone
- 3) Retroposition of the maxilla
- 4) Soft tissue abnormalities like increase in the volume of the tongue, soft palate, parapharyngeal fat pads, and the lateral walls surrounding the pharynx.
- 5) Long axis of airway is directed anterior-posterior rather than laterally.
- 6) Increased length between hyoid and mandible

All the above factors are either genetically determined or due to obesity related changes.

PHARYNGEAL MUSCLE FUNCTION AND THE EFFECT OF SLEEP:

The muscles controlling airway patency fall into three groups:

1. Muscles influencing hyoid bone position (geniohyoid, sternohyoid)
2. Muscle of the tongue (genioglossus)
3. Muscles of the palate (tensor palatini, levator palatini).

These muscles can be divided into two groups

- PHASIC: Muscles which have increased activity during inspiration and so stiffening and dilating the upper airway thus counteracting the collapsing influence of negative airway pressure. Of which genioglossus is the most important phasic muscle. In expiration when the pressure becomes positive the activity of these muscles is substantially reduced (although not eliminated).¹¹
- TONIC: Muscles have a relatively constant level of activity throughout the respiratory cycle called tonic or postural muscles. Ex: tensor palate. They also play a role in the maintenance of airway patency¹².

The activity of the pharyngeal dilator muscles depends on number of variables

- 1) Motor nuclei in ventral medulla receive input from central respiratory pattern leading to activation of the genioglossus muscle (inspiratory phasic activation) before the onset of diaphragmatic contraction or inspiratory airflow, thus „„preparing““ the upper airway for negative pressure.
- 2) Rising PCO₂ and falling PO₂ can augment the activity of these muscles through respiratory premotor nuclei
- 3) Wakefulness drive to these muscles may exist
- 4) Negative intrapharyngeal pressure is the most important local stimulus to activation of the pharyngeal muscles during wakefulness. There is linear

relationship between negative pressure and genioglossus activity(GG EMG and Pepi) which is more pronounced in apneic individuals.

Action of pharyngeal dilator muscle is thus controlled by all the above factors in AWAKE STATE to maintain ventilation. Of which in patients with apnea negative pressure reflex is exaggerated in order to compensate for anatomically deficient airway (Neuromuscular Compensation) During transition from AWAKE TO SLEEP

Phasic neurones largely maintain their activity during sleep, while tonic neurones have large decrements in activity and thus the inspiratory phasic activity of muscles falls initially and then recovers to waking state but tonic activity of muscle (the tensor palatini) lose activity at sleep onset which continues to fall as sleep deepens, reaching levels of 20–30% of waking values

2) As of all reflexes which gets reduced in sleep negative pressure reflex also get decreased in sleep.

Above factors puts the airway in compromised state during sleep. In OSAHS(anatomically compromised airway) patients in addition to above factors, there is loss of neuromuscular compensation leading to greater incidence of airway collapsibility thus leading to apneic episodes.

OTHER FACTORS

LUNG VOLUME

Upper airway size increases at higher lung volumes, because increased lung volume leads to increased „tracheal tug“ and thus increasing the size of upper airway and so decreasing resistance to airflow. OSAHS patients have a “greater change in upper airway dimensions with changes in lung volume, that is, GREATER LUNG VOLUME DEPENDENCE OF UPPER AIRWAY SIZE”. In NREM sleep as the lung volume and end expiratory volume falls patients with OSAHS tends to have airway collapsibility as the airway is already compromised.

VENTILATORY STABILITY

Ventilator stability is measured by loop gain. Loop gain is “the measurement of the tendency of the ventilatory control system to amplify respiration in response to a stimulus or perturbation.” Ventilation will become unstable if the loop gain is >1 will stabilise if the loop gain is <1 Studies shows that patient with apnea have high loop gain ¹⁴ and thus highly unstable respiratory control system.

Role of nasal pathology in OSAHS

Nasal obstruction plays an important role in the pathogenesis of OSA. According to Bernoulli's principle, intraluminal pressure in the compromised space decreases dramatically if the inspired air speeds up to keep the ventilation volume constant. This effect collapses the airway even more.

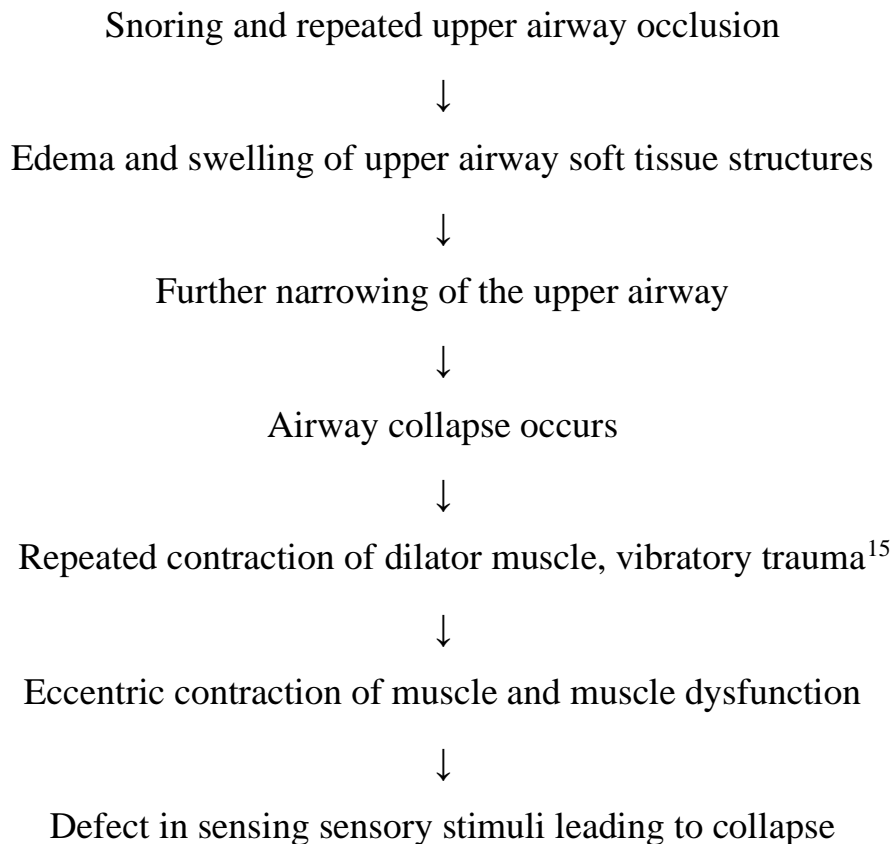
When the negative inspiratory pressure reaches a certain critical point in an effort to overcome the resistance of the upper airway. The combination of redundant soft tissue and lack or loss of muscle tone of the pharyngeal muscles during sleep causes a collapse in the same with inspiration.

On the other side, any patient who cannot breathe adequately through the nose during sleep should open his mouth to allow adequate ventilation. When this happens, the muscles of the tongue fall back, obstructing the airway at the level of the hypopharynx

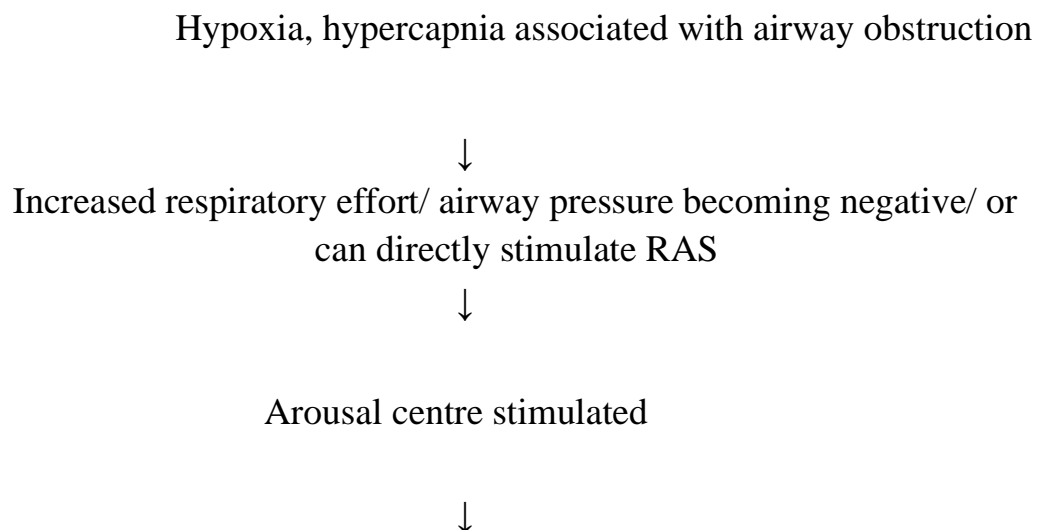
PROGRESSION OF SLEEP APNEA/HYPOPNEA

Sleep apnea which is initially mild in individuals worsens over time

because of the following reasons:



APNEA TERMINATION



Upper airway muscle activity increases followed by relief of obstruction associated with loud snort and hyperventilation.

Then the patient goes in for sleep and the cycle continues. In REM sleep arousal threshold is higher and so the duration of apnea will be longer in REM sleep.

SLEEP RELATED BREATHING DISORDERS

Sleep disordered breathing is a wide spectrum ranging from simple snoring to obesity hypoventilation syndrome¹⁶

SNORING

Defined as “sound generated by the vibration of pharyngeal soft tissues”. Usually it is more during inspiration than expiration and it may also present as a separate symptom without any association to day time sleepiness.

UPPER AIRWAY RESISTANCE SYNDROME

Is classified as a variant of OSA, characterized by increased airway resistance to breathing during sleep, without cessation of breathing. The primary symptoms are similar to those in OSAS although snoring may not be noted.

OBSTRUCTIVE SLEEP APNEA

Apnoea Absence of breathing for at least 10 seconds

Hypopnoea: 30% reduction in breathing amplitude with at least 3% oxygen desaturation;(OR) Respiratory-related arousal (**RERA**) via EEG or nasal flow limitation pattern

Obstructive sleep apnoea / hypopnoea syndrome (OSAHS)

OSA is defined as “five or more respiratory events (apneas, hypopneas, or RERAs) in association with excessive daytime somnolence, waking with gasping, choking, or breath-holding, or witnessed reports of apneas, loud snoring or both”.

Comprises: >5 obstructed breaths / hour AND excessive daytime sleepiness not better explained by other factors OR 2 or more of the following: choking or gasping in sleep, recurrent awakenings, unrefreshed sleep, daytime fatigue, impaired concentration.

Sleep-related hypoventilation disorders

Obesity hypoventilation syndrome (OHS) is the combination of obesity (BMI 30 kg/m²), with hypoxia during sleep, and hypercapnia during the day, due to hypoventilation.

Central sleep apnoea (CSA)

CSA comprises recurrent episodes of apnoea during sleep despite a patent airway . CSA may be associated with periodic breathing in left ventricular cardiac failure with Cheyne-Stokes respiration (CSR) in which there is temporary loss of ventilatory effort or unstable ventilatory control

(CSR-CSA)CLINICAL EXAMINATION OF OSA

THE CHIEF COMPLAINT of osa patients are

ADULT (should ask From patients and sleeping partner)

- Snoring
- Excessive day time sleepiness
- Witnessed apnea
- Disturbed sleep
- Morning headache
- Poor memory

History taking

For snoring : From the partner (i.e. questions that are not easy or possible for patient to answer): How severe is the snoring (audible outside the room, downstairs, by next door neighbour)? Does the patient snore with mouth open?

Is snoring positional?

Are there episodes of crescendo snoring, apnoea, choking or gasping?

Is the patient restless and are there any other limb movements?

Does the patient sleep walk or sleep talk

From patient

Do you feel that your sleep is refreshing

Do you get morning headaches?

Do you suffer from night sweats or palpitations

Do you have vivid dreams or nightmares?

Do you feel that your nose is blocked

Do you smoke and drink alcohol? How much?

Do you feel that your cognitive functions such as concentration and memory are impaired?

Have you had any driving accidents?

Have you had any surgery previously? (Assess fitness or problems with general anaesthesia.)

For sleepiness is assessed by the ESS

EPSWORTH SLEEPINESS SCALE

Epworth Sleepiness Scale was developed in 1991 by Dr. John W. Murray. He intended to “quantify the likelihood of someone to fall asleep in 8 different common situations”. The scores range from 0 to 24 and 10 is the normal limit. The higher the ESS score, the more severe the condition.

- 1) Sitting and Reading
- 2) Watching TV
- 3) Sitting, inactive in a public place (e.g., waiting room, a theater or a meeting)
- 4) As a passenger in a car for an hour without a break
- 5) Lying down to rest in the afternoon when circumstances permit
- 6) Sitting and talking to someone
- 7) Sitting quietly after lunch without alcohol
- 8) In a car, while stopped for a few minutes in traffic

- 0- would never doze off
- 1- slight chance of dozing off
- 2- moderate chance of dozing off
- 3- high chance of dozing off

History : about systemic hypertension, diabetics, thyroid ,Known smoker or alcoholic, regarding diet and weight gain regarding Treatment for same complaints lik weight reduction, withdrawal or reduce alcohol consumption, steroid nasal spray treatment for comorbidity like hypertension, cardio vascular disease,diabetes status, thyroid status

General examination of osa patient

Anemia (tiredness), polycythemia, Cyanosis, clubbing,
dypnea, pedal edema

BMI (Body mass index) >30 – significant

Neck circumference – High risk if >17 inch in Male and >15.5 inch in female

Blood pressure

Enlargement of Thyroid

Examination of face

Assessment of the mandible :

The Vertical line drawn from Lower border of lower lip to pogonion .
if it is > 2mm behind that indicates – Retrognathism

Assessment the maxilla

From Soft tissue of Nasion to subnasale- if it forms the vertical line is normal. if sub nasal is posterior to the vertical line is called maxillary retrusion

Assessment of dentition; by occlusion line

Class1 occlusion:

Maxillary buccal cusps fitted into mandible buccal cusp of first molar

Class 2 occlusion:

Mandibular buccal cusp of first molar Posterior to maxillary first molar-

Retrognathism

Class 3 occlusion:

Mandibular buccal cusp of first molar anterior to maxillary first molar-
mandibular prognathism

Examination of Nose & Nasopharynx:

Obstruction in this level can produce increases airway resistance and

Contribute to apnea and snoring, so have to examine

External nasal framework tip, supratip, ala ,columella .

Nasal valve collapse Ant& post rhionoscopy like Deviated nasal septum,

Turbinate hypertrophy, Mass, Polyps, Sinusitis and adenoids.

Examination of oral cavity and oropharynx

Tongue should be examined in resting position. The Normal size of the tounge it sits below or within the occlusal plane of mandibular teeth. The

Large tongue sits above the occlusal plane

Modified Mallampatti Scoring

Mallampatti grading system is to predict difficult intubation by evaluating the relationship between the tongue and with oropharyngeal structures.

Mallampatti scoring is assessed with the maximum protruding of tongue.

Friedman modified the mallampatti scoring by assessing without protrusion of tongue as it is the most natural position during sleep to asses the relation of oropharynx in relation to tongue and palate .The MMP has a highly predictive value for assessing the severity of OSA and its outcome after surgical procedures.

“Modified Mallampati classification by Friedman et al



Figure : Friedman tongue position

- A. FTP -I visualizes the uvula and tonsils/pillar.
- B FTP- II a visualizes most of the uvula but not the tonsils/pillar.
- C FTP – II b visualizes the entire Soft palate to the uvular base.
- D FTP III- shows some of the soft palate with the distal end absent.

E FTP -IV vis

In hard Palate should look for high arched and in Soft palate & uvula should examined for either Anteriorly or posteriorly placed, loose mucosa.

In uvula, edematous, short or webbed and elongated uvula

Examination of Tonsil by grading

A Size 0 – of tonsillar tissue.

B Size 1-within the pillars.

C Size 2, extended to the pillars.

D Size 3, extended past the pillars.

E Size 4, extended to the midline. palate. **Lingual hypertrophy grading** a

LTH 0, no lymphoid tissue.

b LTH 1, scattered lymphoid tissue.

c LTH 2, lymphoid tissue covering the entire tongue base, limited vertical thickness.

d LTH 3, lymphoid tissue covering the entire tongue base, significant vertical thickness of approximately 5–10 mm e LTH 4, lymphoid tissue covering the entire tongue base, rising to or above the tip of the epiglottis, approximately 1 cm in height.

Muller Maneuver

The muller's maneuver help us to evaluate the level and degree of collapse of upper airway . Procedure involves “the upper airway nasopharyngoscopy helps to evaluate the dynamic examination of nose, naso, oro and hypopharynx at rest and with the maximal inspiratory effect

against a closed nose and mouth. Soft tissue collapse especially at tongue base, palate ,lateral pharyngeal walls are examined. This severity is rated from 0(minimal collapse) to 4+ (complete collapse)”.Muller maneuver is beneficial technique for preoperative evaluation of severity. However, the repeatability of this maneuver is very low. Palatal and lateral wall collapse has a greater correlation, but with base of tongue it is low.

Polysomnography

It is the gold standard investigation in assessing the sleep disordered breathing and assessing the effectiveness after surgery.

Parameters

PSG monitors many physiological signals that is required for the assessment of sleep disordered breathing. These signals are displayed in graph in organized waveform.

- a) Sleep staging
- b) Respiratory airflow
- c) Respiratory effort
- d) Pulse oximetry
- e) Ventilation
- f) Cardiac rhythm

Sleep staging

ECG, EMG, EEG are the parameters monitored in sleep staging. Sleep staging helps in

- 1) Assessing the severity of the disease(it adjusts the total no of respiratory events based on total sleep time)
- 2) Helps in identification of respiratory events associated with arousal

Respiratory Airflow

It is monitored by two methods

- a) Oronasal thermistometry
- b) Nasal pressure transduction.

a) Oronasal thermistometry

Helps in identification and scoring of apneas. It is the superior modality in detecting low levels of airflow.

Mechanism: Exhaled air is warmer than the environmental air since the air is heated in the lungs. Oronasal thermistometry uses this mechanism and

“measures fluctuations in air temperature at nose and mouth” and thus measures even modest level of airflow.

b) Nasal pressure transduction

It is superior in measuring “subtle changes in airflow as seen in hypopneas”

Mechanism: Negative pressure fluctuation determines inspiration and positive pressure fluctuation determines expiration. Nasal pressure transduction uses this mechanism and detects airflow by measuring variations in air pressure.

Respiratory effort

Measuring respiratory effort helps in distinguishing central and obstructive apnea. It can be measured in following ways(RIP, Piezoelectric belts, PVDF, Esophageal manometry)

Pulse oximetry

It measures arterial oxyhaemoglobin saturation. Mechanism: Uses “DUAL WAVELENGTH”(red and infrared). Light transmitter and receiver is placed around the arterial bed and the “ratio of red and infrared transmission” is used to calculate spo₂.

Ventilation

It is required only in children. In adults, only if hypoventilation is suspected ventilation is monitored.

METHODS

- 1) Transcutaneous co₂ measurement
- 2) End tidal co₂ monitoring

Cardiac rhythm

Monitored by ECG. Single lead is sufficient to detect arrhythmias.

Usually lead 2 is used.

TYPES OF PSG

Depending upon the parameters measured PSG are of four types ¹⁹.

Level I - “Standard PSG with minimum of seven parameters measured (EEG, EOG, chin EMG, ECG, airflow, respiratory effort, and oxygen saturation) in a specialist unit”.

Level II - “Comprehensive portable PSG same, except that a heart rate monitor can replace the ECG and a technician is not in constant attendance”

Level III - “Modified portable sleep apnea testing is a cardiorespiratory study, minimum of four parameters ventilation (at least two channels of respiratory movement, or respiratory movement and airflow), heart rate or ECG, and oxygen saturation”. Done at home.

Level IV - “Continuous (single or dual) bioparameter recordings where devices that measure a minimum of one parameter, usually oxygen saturation are utilized”

Identification and Scoring of Events General Principle

- 1) Identification of apneic events on PSG requires measurement of airflow and confirmation by EEG that the event occurred during sleep.
- 2) Respiratory effort measured to differentiate central and obstructive type.
- 3) Oximetry measurement improves the reliability and also helps in scoring type of events.

Arousal:

Identified by “Abrupt shift of EEG frequency including alpha, theta frequency of >1 hz that lasts atleast 3 sec with atleast 10 sec of preceding stable sleep”.

In REM sleep there should also be increase in the activity of submental EEG.

AROUSAL INDEX: Total no of arousal divided by total sleep time.

Apnea :

Apnea is defined as cessation/ near cessation of airflow. It is detected by recording event of “90% or greater reduction in airflow for a period of 10 sec”.

It is classified as

- a) Obstructive: associated with respiratory effort throughout the event
- b) Central: absence of respiratory effort
- c) Mixed: absence of respiratory effort in initial part later followed by appearance of respiratory effort.

Hypopnea:

Hypopnea is defined as “reduction of airflow to a degree that is insufficient to meet the criteria of apnea”.

According to AASM, hypopnea is

- a) “Airflow decreased by atleast 30% compared with previous baseline.
- b) Diminished airflow for atleast 10 sec.

c) Event is associated with either 3 oxygen desaturation/ EEG arousal.” It can either be central/ obstructive. When hypopnea is associated with features of upper airway narrowing like thoracoabdominal paradox, snoring etc it is obstructive type. Otherwise it is central hypopnea.

Respiratory effort related arousal(RERA)

Change in airflow that doesn't meet criteria of apnea and hypopnea is called as RERA. It is defined as “event lasting for atleast 10 sec and associated with flattening of nasal pressure waveform and/or there is evidence of increasing respiratory effort terminating in an arousal but not meeting criteria for apnea or hypopnea”.

Interpretation from polysomnography:

From the above parameters severity of the sleep disordered breathing can be derived by many indices.

- a) Apnea hypopnea index
- b) Respiratory disturbance index
- c) Oxygen desaturation index

Apnea-Hypopnea index (AHI)

It is calculated as “number of apneas and hypopneas per hour of total sleep time”.

“Normal : AHI <5

Mild OSA : AHI 5-15

Moderate OSA : AHI 16- 30

Severe OSA : AHI>30²⁰

Respiratory disturbance index (RDI)

It is derived by “adding total no. of apnea, hypopnea, RERA and divided by total sleep time in hours”.

Oxygen desaturation index (ODI)

It is defined as “total no of times per hour of sleep that a patient’s oxyhaemoglobin saturation drops by 4 percent from baseline”.

Sleep Hypnogram

A sleep hypnogram is a summary of the entire night’s PSG data in a graphic form. It gives a good snapshot of sleep architecture, distribution of respiratory events, and oxygen saturation trends in different sleep stages, sleep position, and at different times of the study night. It is helpful to open a window for the hypnogram simultaneously while reviewing the PSG The most important parameters measured in sleep hypnogram are:

- 1) Total recording time(TRT)-“ beginning and end of recording”
- 2) Total sleep time(TST)-“ actual sleep REM +NREM”
- 3) Sleep period time(SPT)- “ sleep onset to final awakwning”
- 4) Sleep efficiency (SE)-“ percentage of TST in total time in bed”.

SE>85%is normal.

- 5) Sleep latency (SL) –“time elapsed between lights out to the first epoch of sleep (usually stage 1)”.

Dynamic Magnetic Resonance Imaging

MRI provides anatomic definition of soft tissue structures, allows for multiplanar imaging, and does not expose patients to radiation. With newer technologies, the dynamic airway can be evaluated with rapid image acquisition; multiple images per second. The main disadvantage of dynamic MRI arises in regards to patient comfort, concurrent sleep evaluation, scanner noise with possible requirement of sedation, and examination expense.

DISE

Drug induced sleep endoscopy is a procedure which involves endoscopic evaluation of upper airway and identifying the level of obstruction in a pharmacologically sedated patients. Results are interpreted as VOTE classification.

TREATMENT

OSA Treatment under comes under three categories :

- 1) Life Style Modification
- 2) Devices that can be worn
- 3) Surgery

LIFE STYLE MODIFICATION

Weight reduction

Avoiding alcohol and smoking

Sleep posture therapy: lateral or prone position

Devices that can be worn

a) Positive airway pressure

Most effective treatment in moderate to severe grades of OSA in tolerant patients which can reverse the negative cardiac and neurocognitive consequences of untreated disease. It is available in many forms such as including continuous positive airway pressure (CPAP), adjustable positive airway pressure (APAP), and bi-level positive airway pressure (BI-PAP).

Each delivers positive pressure through a device worn on the face, and serves as an internal pneumatic splint for the airway. CPAP, the most commonly used form of PAP, typically uses between 5 and 15 cm of water pressure to maintain airway patency. Although CPAP has high efficacy, compliance is limited²² causing non-adherent to therapy (defined as > 4 hours of use per night)

b) Oral appliances

Mandibular Advancing Device: Advance the mandible anteriorly, which brings forward the tongue and other muscles of the oropharynx and hypopharynx. The position of the palate is also changed with the mandibular repositioning device through action of the palatoglossus muscle.

Tongue Retaining Device (TRD): “ It increases pharyngeal patency by pulling the superior aspect of the tongue forward, away from the posterior wall of the pharynx”

Surgical Management

The aim of surgery in OSAHS patients to eliminate the airway collapse and reduce the airway resistance during sleep and preserve to normal function and adjacent normal structure. Since the patients with OSAHS may have obstruction either nasal cavity, nasopharynx, retropalatal, retrolingual, hypopharynx . Various surgeries have evolved to address the specific sites of obstruction. In order to avoid unnecessary procedures and minimize the surgical interventions a protocol was formulated popularly known Riley-Powell– stand ford surgical protocol.

It is a two staged procedure addressing specific sites of obstruction (nasal cavity/nasopharynx, oropharynx and hypopharynx).

PHASE 1: Targets soft tissues of upper airway i.e., nose, palate, tongue

PHASE 2: Targets hypopharyngeal and pharyngeal airway by altering skeletal framework.

Powell–Riley protocol surgical procedures

Phase I

- ❖ Nasal surgery (septoplasty, turbinate reduction, nasal valve grafting)
- ❖ Tonsillectomy
- ❖ Uvulopalatopharyngoplasty (UPPP) or uvulopalatal fl ap (UPF)
- ❖ Mandibular osteotomy with genioglossus advancement

- ❖ Hyoid myotomy and suspension
- ❖ Temperature-controlled radiofrequency (TCRF)-turbinates, palate, tongue base

Phase II

- ❖ Maxillomandibular advancement osteotomy (MMO) ❖
- Temperature-controlled radiofrequency (TCRF)-tongue base
- ❖ TCRF is usually employed as an adjunct treatment.

INDICATIONS FOR SURGERY

- ❖ “Apnea/Hypopnea Index (AHI) >20 events/per hour of sleep
- ❖ Oxygen desaturation nadir <90%
- ❖ Esophageal pressure (PES) more negative than –10 cm H₂O
- ❖ Cardiovascular derangements (arrhythmia, hypertension)
- ❖ Neurobehavioral symptoms (excessive daytime sleepiness [EDS])
- ❖ Failure of medical management
- ❖ Anatomical sites of obstruction (nose, palate, tongue base)”

CONTRAINDICATIONS FOR AIRWAY SURGERY

- ❖ “Severe pulmonary disease
- ❖ Unstable cardiovascular disease
- ❖ Morbid obesity (BMI >40)
- ❖ Alcohol or drug abuse

❖ “Psychiatric instability”

Patients can be categorized into either responders or non responders after surgery.

DEFINITION OF SURGICAL RESPONDERS

- 1) “Decrease in RDI greater than or equal to 50% and a total of less than 20 events/h
- 2) Decrease in AHI greater than or equal to 50% and a total of less than 10 events/h
- 3) Oxygen desaturation nadir 90%
- 4) Excessive daytime fatigue (EDS) alleviated
- 5) Normalization of sleep architecture
- 6) Response equivalent to CPAP on full-night titration”

DEFINITION OF NON RESPONDERS

No change or deterioration in outcome are referred as non responders.

NASAL SURGERIES

Nasal obstruction causes increase in resistance causing increase in negative inspiratory pressure thereby causing airway collapse. Moreover nasal obstruction increases negative pressure and causing functional narrowing of the pharyngeal airway, leading onto hypoxia and sleep apnoea. But nasal surgeries rarely cures OSAHS. In patients undergoing nasal surgeries compliance to nasal CPAP is increased ²⁴ since it reduces CPAP pressures

from a mean of 11.9 down to 9.2 centimeters of water pressure by improving nasal breathing.

Septoplasty, septorhinoplasty, and turbinate reduction are the various procedures to correct the underlying nasal pathology.

ADENOTONSILLECTOMY

Adenotonsillectomy is the surgery commonly done in pediatric population to correct loud snoring and restless sleep. The tonsils and adenoids can be removed or reduced in a number of ways like standard cautery, snare, bipolar cautery, harmonic scalpel, coblation, temperature- controlled radiofrequency, or microdebrider-powered shavers. Complications include hemorrhage, pain.

VELOPHARYNGEAL SURGERY

Since 80% of the patients with OSAHS have obstruction at the level of velum many surgeries have evolved to modify the anatomy of velum to maintain airway patency. It can either be minimally invasive or invasive procedures depending upon the severity of the disease. It includes

- 1) Uvulectomy and laser assisted uvuloplasty
- 2) CAPSO (Cautery-assisted palatal stiffening operation)
- 3) Injection Snoreplasty
- 4) Palatal implants

- 5) UPPP(uvulopalatopharyngoplasty): UPPP is the most common procedure for the treatment of OSAHS described by Fujita ²⁵ which is a modification of Ikematsu procedure. It consists of “tonsillectomy, reorientation of anterior and posterior tonsillar pillars, and excision of the uvula and posterior rim of the soft palate”

STEP 1: The incision is marked on the anterior pillars and ventral surface of the palate with electrical cautery

STEP 2: Dissection begins at the right inferior tonsillar pole and anterior pillar and tonsil removed. Incision continued to softpalate leaving dorsal flap. Uvula resected leaving dorsal flap. Incision extended to opposite side in a similar fashion.

STEP 3: Incisions are made at the junction between the dorsal palatal and posterior pillar flaps.

STEP 4: The upper part of the posterior pillar flap is approximated to the ventral palatal mucosa. After removing redundant mucosa soft palate and uvula are sutured.

Complications includes transient/ permanent VPI, pharyngeal symptoms (tightness, dryness, FB sensation in the throat), bleeding, wound dehiscence, prolonged pain, taste and voice disturbance, nasopharyngeal stenosis, deterioration of OSA. Since the complications encountered are due to the

removal of large amount of tissues, newo procedures have evolved that reorganizes and preserves tissues and promoting increase in airway. These includes relocation pharyngoplasty, lateral pharyngoplasty, expansion sphincter pharyngoplasty, Z- palatoplasty, and palatal advancement.

- 6) Transpalatal Advancement Pharyngoplasty(TAP)
- 7) Lateral Pharyngoplasty
- 8) Z-Palatoplasty (ZPP)²⁶: Described by Friedman with the aim of removing anterior mucosa only and the splitting of the soft palate in the midline. The key features are the cutting of the palatoglossus muscle, and the sewing of the posterior palatal mucosa to the anterior resection margin, which retracts the midline

STEPS: Outline of the palatal flaps, marked before incision.



The mucosa over the palatal flap is removed and the palatal musculature is exposed



The uvula and palate are split in the midline with a cold knife



The uvular flaps along with the soft palate are reflected back and laterally,

over the soft palate.



Two-layered closure of the palatal flaps is done.

Complications include bleeding, temporary and permanent VPI, throat discomfort symptoms like globus sensation, mild dysphagia, dry throat, and inability to clear the throat.

- 9) Expansion Sphincter Pharyngoplasty: Described by Pang & Woodson²⁷ in which a “horizontal incision is made in the palatopharyngeus muscle (after tonsillectomy), superolateral incisions are made on the soft palate, in the inferior aspect of the palatopharyngeus muscle is then suspended superolaterally to the arching fibres of palatoglossus, partial uvulectomy performed and then closure of wound is done”. Complications are as with other palatal procedures but the incidence is much low.

HYPOPHARYNGEAL SURGERY

Apart from velum, many of the OSA patients have obstruction at the level of tongue base. To address retroglottal area, surgery is directed toward either a reduction in the volume of tongue mass or advancement of the tongue’s anterior attachments. Most of the procedures are done as a part of multilevel surgery and not as a solo procedure.

Tongue-Base Reduction Procedures

Tongue-base reduction procedures are intended to reduce the size of the tongue, so that the space between the posterior pharyngeal wall and the tongue base is increased. Procedures include radiofrequency ablation, partial midline glossectomy, lingualplasty, and lingual tonsillectomy, Submucosal minimally invasive lingual excision (SMILE) and transoral robotic glossectomy (TORS).

Radiofrequency tongue ablation:

Radiofrequency tissue ablation which is a common procedure involves the application of a temperature-controlled radiofrequency probe to multiple locations in the base of the tongue. The aim of this procedure is to reduce the tongue volume through scar tissue generation. *Tongue-Repositioning*

Procedures:

Tongue-repositioning procedures increases the space between the posterior pharyngeal wall and the tongue base by moving the tongue anteriorly by releasing its attachment. It includes tongue suspension, hyoid myotomy and suspension (HMS), and genioglossal advancement.

a) **Tongue base suspension:** It is designed to stabilize the tongue by anchoring the tongue to the mandible with a permanent suture thereby preventing retrolingual collapse.

b) **Hyoid myotomy and suspension (HMS):** HMS uses permanent sutures to suspend the hyoid bone to the thyroid cartilage or mandible thereby pulling the tongue anteriorly.

c) **Genioglossal advancement:** Genioglossus advancement involves mobilization and advancement of the genial tubercle of the mandible, with limited osteotomy and fixation or forward movement of the lower anterior mandible and attached muscles thereby displacing the tongue anteriorly.

4) *Hypoglossal nerve stimulation:*

It is a new entity which is neither a tongue base reduction nor a tongue advancement procedure. But it increases retrolingual area by placing an implantable stimulator that stimulates the hypoglossal nerve during inhalation to keep the retroglottal airway open during sleep. Components includes implantable pulse generator, respiratory pressure sensor and stimulation lead.

MAXILLARY MANDIBULAR ADVANCEMENT

Maxillomandibular advancement (MMA) is intended to widen the retroglottal and retropalatal airways by creating LeForte 1 osteotomy to the maxilla and a bilateral split sagittal osteotomy to the mandible to advance both the maxilla and mandible. Though it is recommended as a phase 2 surgery it can be done as a primary procedure in patients with obvious craniofacial issues and multiple sites of upper airway obstruction provided these patients fulfill clinicoradiologic criteria based on cephalometric measurements.

TRACHEOSTOMY

Permanent tracheotomy was the first treatment²⁸ formulated for OSA and it is still the gold standard surgery for OSA. Because of the morbidity associated with the surgery it is reserved for patients who are intolerant of mechanical measures or fail upper airway surgery and continue to have severe symptoms or physiologic changes related to obstructive sleep apnea

MULTILEVEL SURGERY

Because of the inconsistent results with various surgeries, Friedman proposed a staging system¹⁸.

Stage-I Friedman tongue position 1 and 2. Tonsil size 3 and 4. BMI <40.

Stage-II Friedman tongue position 1 and 2. Tonsil size 0, 1 and 2.

BMI <40. Friedman tongue position 3, 4 tonsil size 3 and 4. BMI <40.

Stage-III Friedman tongue position 3 and 4. Tonsil size 0, 1 and 2. BMI <40.

OSA patients of stage 2 and 3 have been identified to have obstruction not only at the single level but simultaneously at multiple levels²⁹ requiring surgery addressing various levels of airway. Therefore multilevel pharyngeal surgery is usually required to surgically overcome the several sites of obstruction.

SURGERY ON EPIGLOTTIS

Rarely patients with OSA have obstruction at the level of epiglottis making it to collapse posteriorly during sleep. To correct this retrodisplacement, epiglottopexy (suspending the epiglottis to the tongue base with a suture) and epiglottoplasty (removing a portion of the superior part of

the epiglottis) are the procedures employed. These procedures are rarely performed, but if performed, they are often done so as part of a multilevel surgery³⁰.

SURGICAL SUCCESS

DEFINED AS “as a decrease in respiratory disturbance index greater than or equal to 50% and a total of less than 20 events/h or a decrease in apnea index greater than or equal to 50% and a total of less than 10 events/h.³¹”

MATERIALS AND METHODS

STUDY PLACE

Government Rajaji Hospital, Madurai – 625020. Department of Otorhinolaryngology

STUDY DESIGN

Prospective study

STUDY PERIOD

One year

STUDY POPULATION

All patients with snoring and OSA who reported to the

.department of otorhinolaryngology of Madurai Medical College during the study period with the fulfillment of inclusion criteria.

INCLUSION CRITERIA

- 1) Age > 18years
- 2) Both sexes (male and female)
- 3) BMI <40
- 4) Patient with excessive daytime sleepiness
- 5) Witnessed apnea snoring

EXCLUSION CRITERIA

- 1) Age below 18 yrs and above 55yrs
- 2) Hypothyroidism and other metabolic disorders
- 3) BMI >40
- 4) Associated craniofacial abnormalities

INVESTIGATION

- 1) Thyroid Function Test (TFT)
- 2) CT Paranasal sinus
- 3) CT NECK
- 4) VIDEO LARYNGOSCOPY
- 5) DIAGNOSTIC LARYNGOSCOPY

ETHICAL COMMITTEE APPROVAL

Institutional Ethical Committee, Government Rajaji Hospital, Madurai Medical College, Madurai reviewed the experimental design and protocol as well as the letter of information and consent form. Full approval of the board was granted. All patients were given information outlining the experimental protocol and all patients signed a consent form prior to entering the study.

METHODOLOGY

This is a prospective study conducted Department of Otorhinolaryngology, Madurai Medical College from June 2020 to december 2021. All patients who attend our op with the complaints of snoring, frequent awakening at night, excessive day time sleepiness, choking in sleep are further evaluated. All the patients underwent clinical examination followed by blood investigations especially thyroid function test and BMI evaluation .Then polysomnography level is done



Figure : Recording of oxygen saturation in PSG

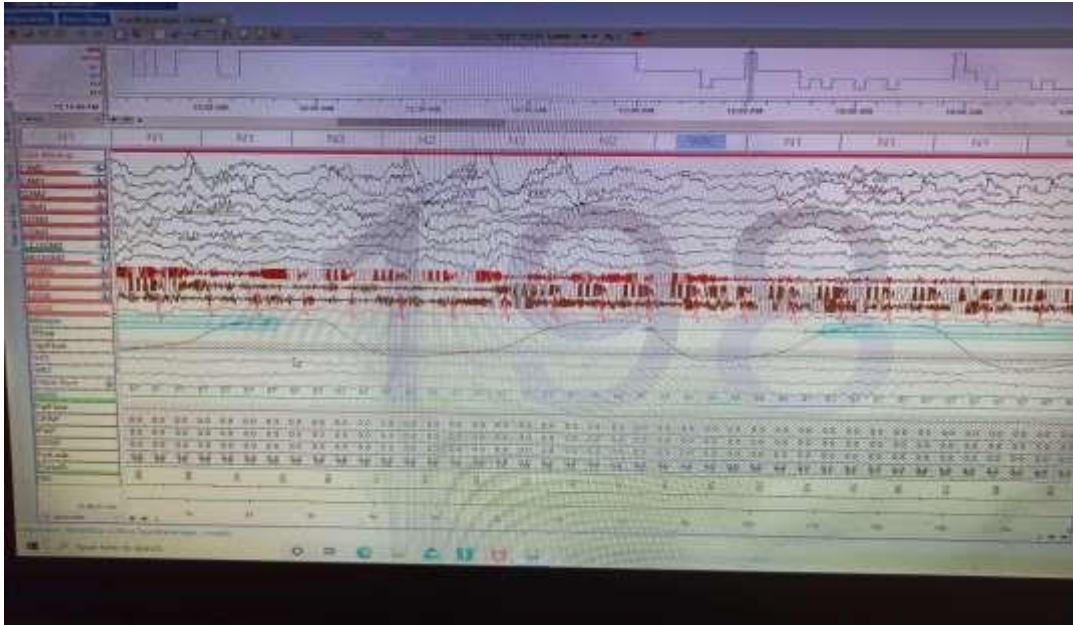


Figure :Polysomnographic recording

SURGERY

Selected patients for surgery were admitted in the ward and started with pre op antibiotics. Under general anaesthesia or local anesthesia depending upon level of obstruction patients underwent surgery accordingly.

Turbinate surgery

Inferior Turbinoplasty: an incision made along the inferior edge of the turbinate up to the anterior attachment. Using elevator the medial aspect of mucosa of inferior turbinate is elevated. Now the mucosa and bone on the lateral side of the anterior two third of the turbinate is excised. after that the residual bone is lateralised and mucosal flap is draped over the bony stump.

Powered inferior turbinoplasty : A tri cut turbinate shaver blade is introduced submucosally at the anterior end of the inferior turbinate. Mucosal tissue debrided from the anterior to posteriorly

Septal surgery

Hemi transfixation incision made, mucoperichondrial and mucoperiosteal flap elevated with freer's elevator on same side. Flap elevated inferiorly. Opposite mucoperiosteal flap elevated. Inferior strip of cartilage removed. Bony septum removed.

Tonsillectomy

In sister rose position, boyce davis mouth gag applied. Tonsillectomy done by enucleation done by dissection and snare method

Adenoidectomy

In sister rose position with boyce davis mouth gag, curettage done by st. clair thompson adenoid curette

Sinus surgery

Indication – choncha bullosa, chronic rhinosinusitis, sinonasal polypsis.

Infundibulotomy, uncinectomy, wide middle meatal antrostomy done.
Anterior and posterior ethmoidectomy done. Sphenoidectomy done.
Chonchoplasty done.

UPPP Indication: Antero posterior collapse at velum. Tonsillectomy done .

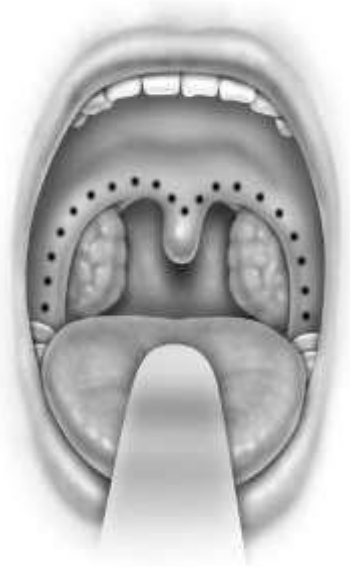
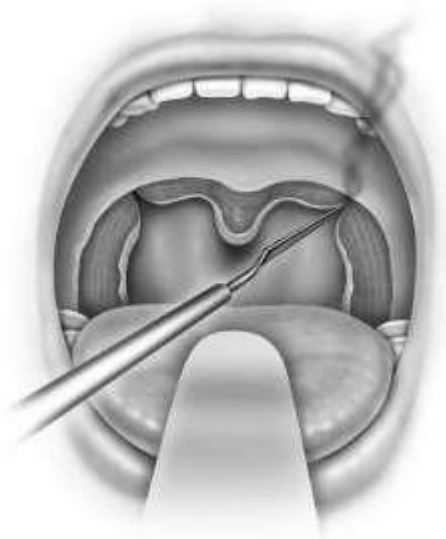


Figure : Incision

Incision made at the junction of post pillar and dorsal flap and pillar sutured to ventral flap.

Figure : Incision at posterior pillar



In fairbank's technique of UPPP, final wound will be rectangular in shape

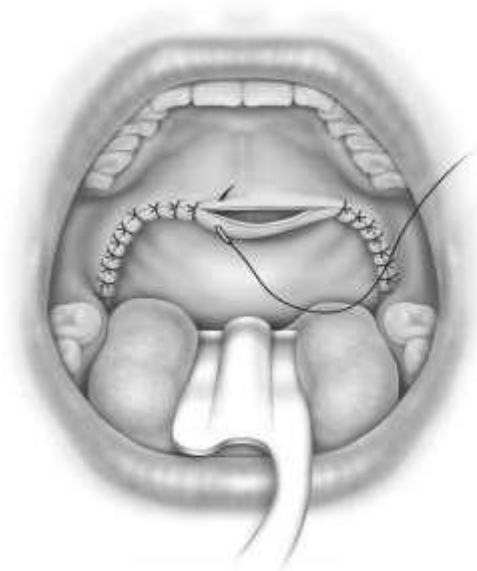


Figure : After wound closure

Post-Operative Follow Up

Patients were on intravenous antibiotics, oral or parenteral analgesics, mouth wash. Some Patients were on Ryle's tube feeding and discharged after 1 week.

All patients were followed up in weekly basis.

enquired about reduction in symptoms. After 2 months and polysomnography(level 1) is done and AHI is calculated. All parameters were compared with pre surgical values of the patients.

STATISTICAL ANALYSIS

Statistical analysis is done through SPSS 20 software. Descriptive statistical analysis done to summarize the baseline characteristics result.

Statistical analysis:

1. Descriptive stats: Mean, Standard Deviation, Frequency, %
2. t test - to find differences between quantitative variables.
3. Chi square test - to find association /differences for qualitative variables.
4. Spearman / Kendals correlation - to find relationships between variables.
5. one way ANOVA - - to find differences between quantitative variables >2 group

RESULTS

Table 1: Age Distribution of study participants

Age	Frequency	Percent
18-30	11	36.5
31-40	5	16.9
41-50	8	26.6
51-60	6	20
Total	30	100

Table 1 shows that majority of them were in age 18 to 30 years 36.5% followed 41-50 years 26.6% (Graph 1).

Graph 1: Age Distribution of study participants

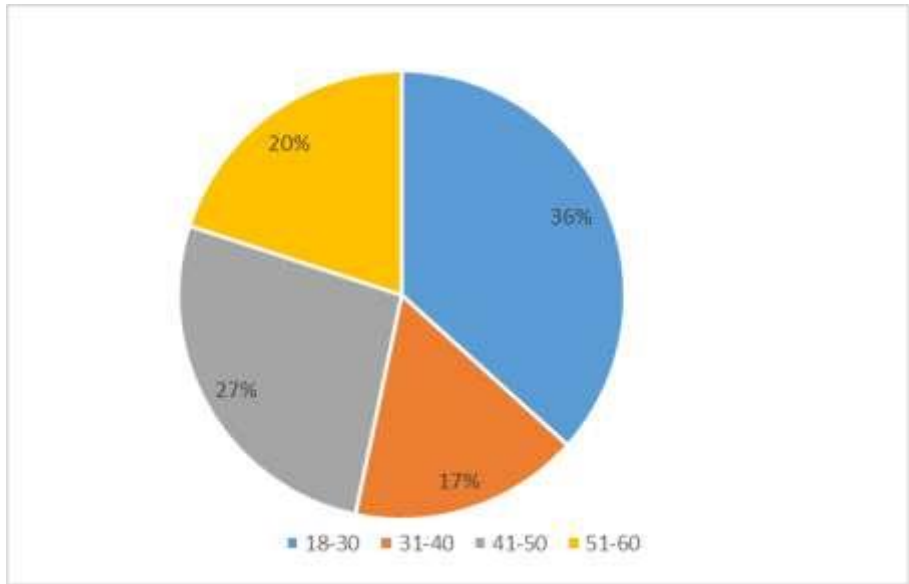


Table 2: Gender distribution of study participants

Sex	Frequency	Percent
Male	25	83.3
Female	5	16.7
Total	30	100

Graph 2: Gender distribution of study participants

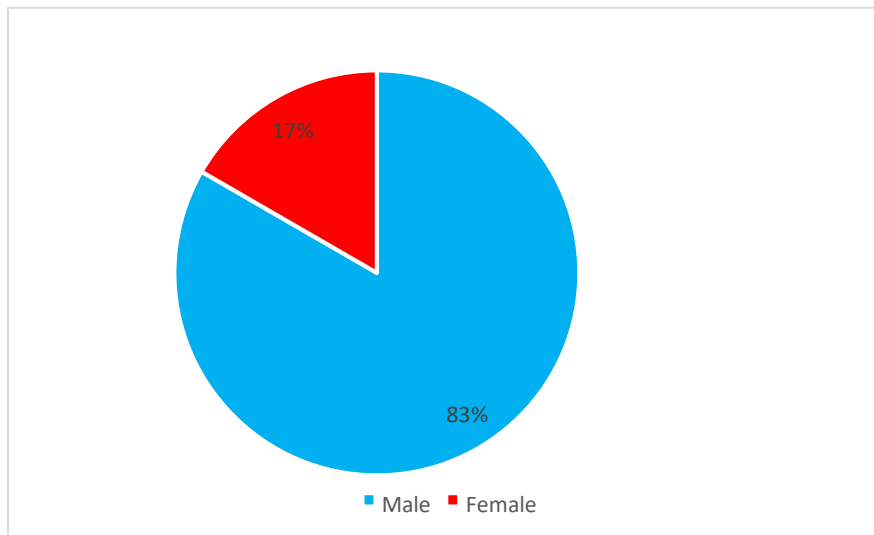


Table 2 shows that majority of them were male 83%, female were 17% (Graph 2).

Table 3: Co Morbidity status of participants

Comorbidity	Frequency	Percent
Diabetes	4	13.3
Hypertension	9	30

Table 3 shows that 4 had Diabetes and 9 had hypertension (Graph 3).

Graph 3: Co Morbidity status of participants

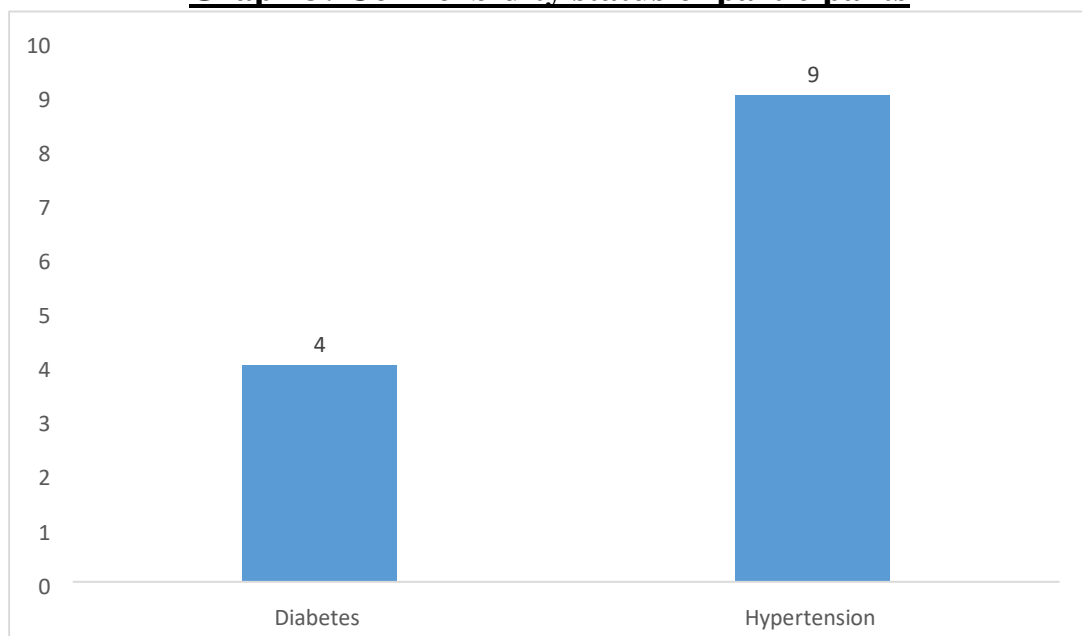


Table 4: Distribution of diagnosis

Diagnosis	Frequency	Percent
B/L grade tonsillar hypertrophy	2	6.6
chronic adenoid and tonsillar hypertrophy	2	6.7
Chronic rhinosinusitis	4	13.3
chronic tonsillitis	2	6.7
Deviated nasal septum with conchabullosa	2	6.7
Deviated nasal septum with inferior turbinate hypertrophy	4	13.3
Narrowing at velum and elongated Uvula	2	6.6
Narrowing at velum	4	13.4
Narrowing at velum. elongated edema uvula and soft palate	2	6.7
Narrowing in oropharynx and hypopharynx	2	6.6
Narrowing in oropharynx and velum	4	13.3
Total	30	100

Graph 4: Distribution of diagnosis

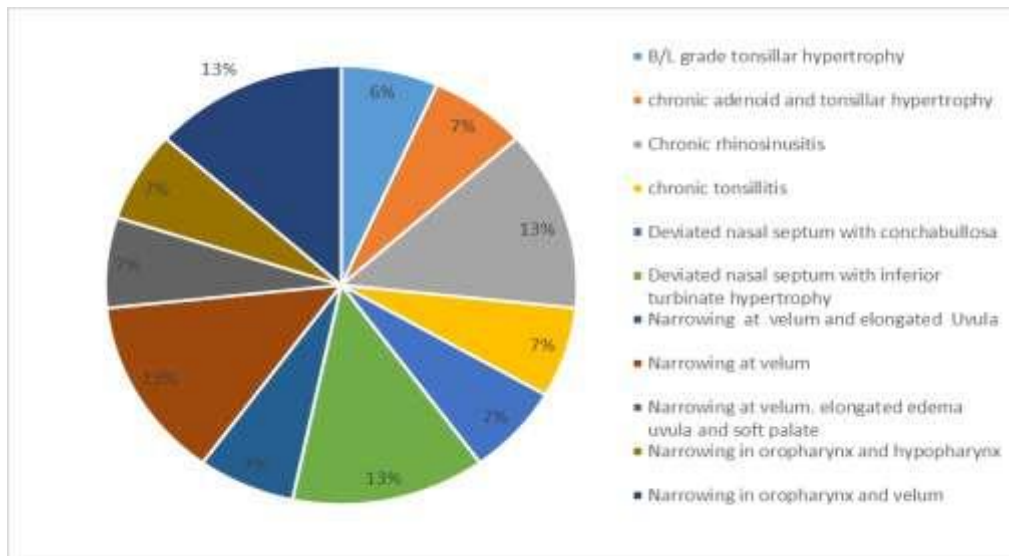


Table 5: Distribution of type of surgery

Type of surgery	Frequency	Percent
FESS	4	13.3
septoplasty with conchoplasty	2	6.7
SMR with b/l turbinoplasty	2	6.7
SMR with b/l conchoplasty	2	6.7
Tonsillectomy	4	13.3
Tonsillectomy and Uvuloplasty	2	6.7
Uuvlopalatopharyngoplasty	14	46.7
Total	30	100

Graph 5: Distribution of type of surgery

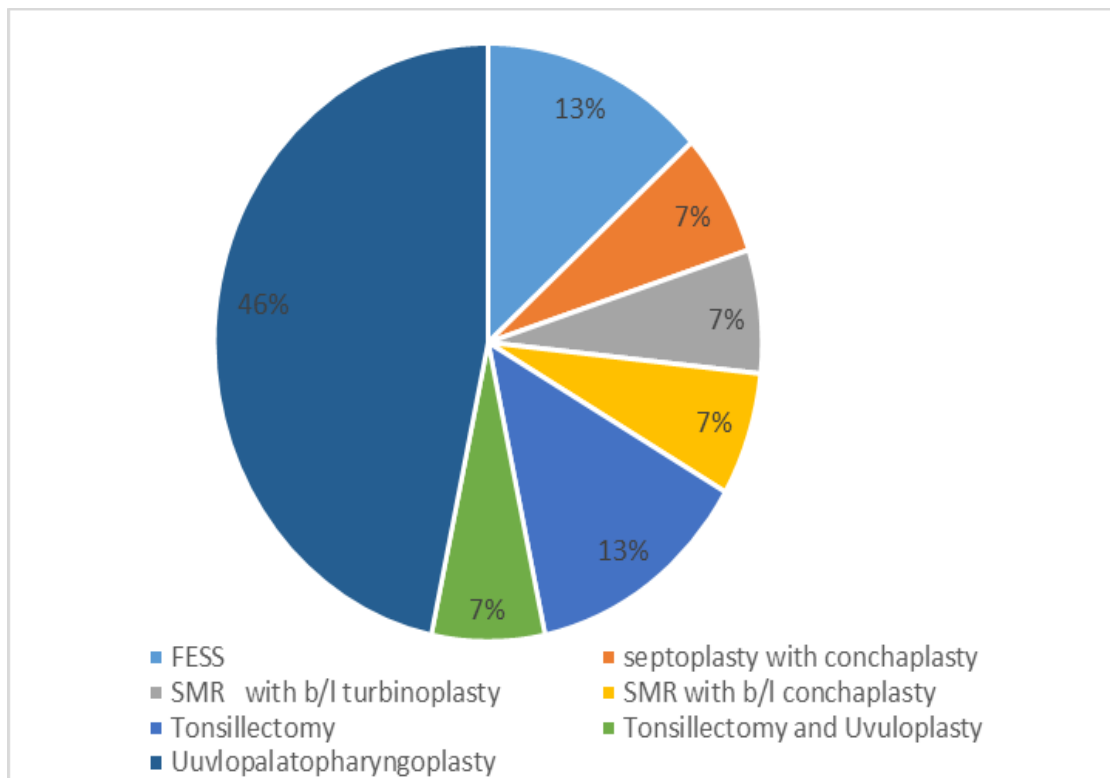


Table 6: Distribution of type of level

Level	Frequency	Percent
Level 1	10	33.3
Level 2	20	66.7
Total	30	100

Graph 6: Distribution of type of level

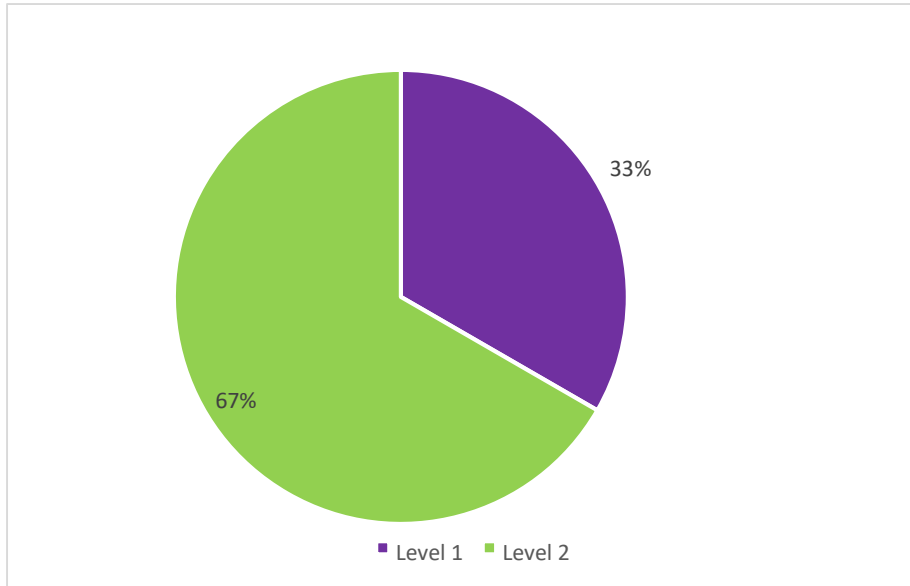


Table 6 shows that majority of them were 67% in level 2 followed 33% had level 1 (Graph 6).

Table 7: Distribution of postoperative snoring

Post op snoring	Frequency	Percent
Not reduced	5	16.7
Reduced	23	76.7
Stopped	2	6.7
Total	30	100

Table 7 shows that majority of them were 76.7% had reduced snoring after surgery. 16.7% were told it was not reduced and 6.7% were experienced no snoring after surgery (Graph 7).

Graph 7: Distribution of postoperative snoring

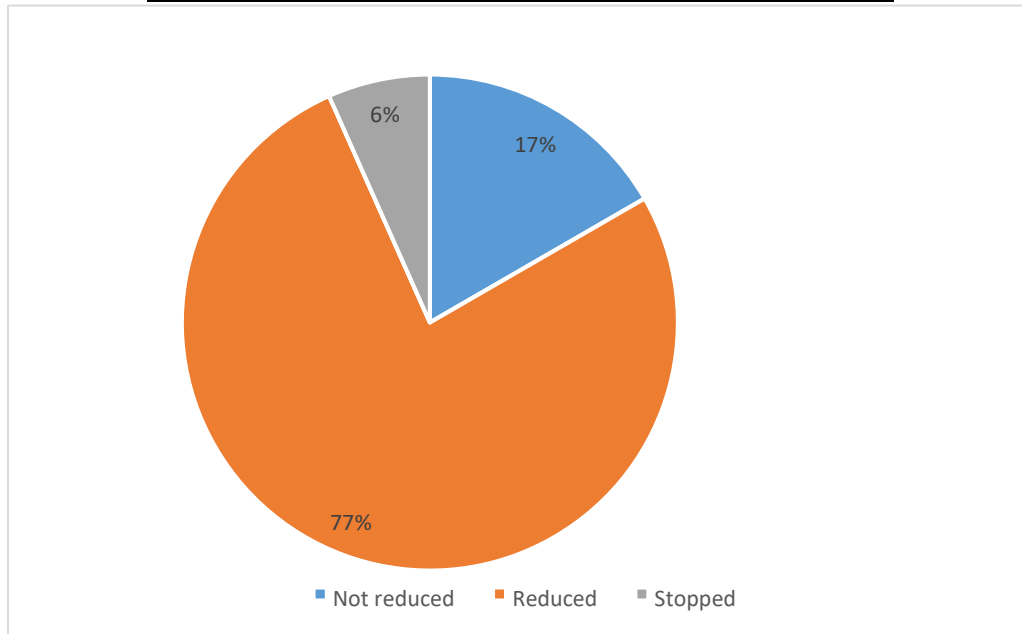


Table 8: Mean comparison of pre and postoperative AHI

	Mean	SD	t	p
Pre-op AHI	37.42	23.30	9.56	0.0001
Post-op AHI	12.72	9.94		

Table 8 explains that there was significant difference exists in mean value of AHI with pre and post-operative by paired t test with $p = 0.0001$. This implies

that mean AHI of pre op was higher and in post op AHI average was reduced (Graph 8).

Graph 8: Mean comparison of pre and postoperative AHI

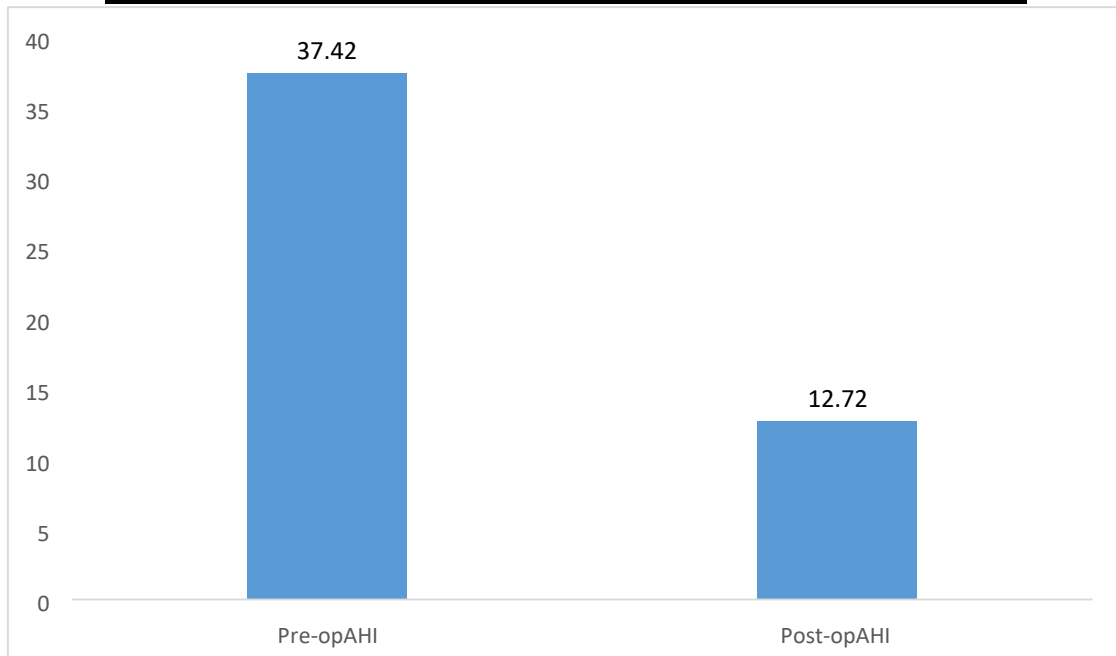


Table 9: Mean age comparison of levels & Postop snoring

	Level	Mean	SD	t	P
Age	Level 1	36.60	9.65	0.39	0.699
	Level 2	38.45	13.28		

	Post op snoring	N	Mean	SD	F	Sig.
Age	Not reduced	5	40	10.7	2.361	0.114
	Reduced	23	35.96	11.922		
	Stopped	2	54	1.414		
	Total	30	37.83	12.055		

Graph 9: Mean age comparison of levels& Postop snoring

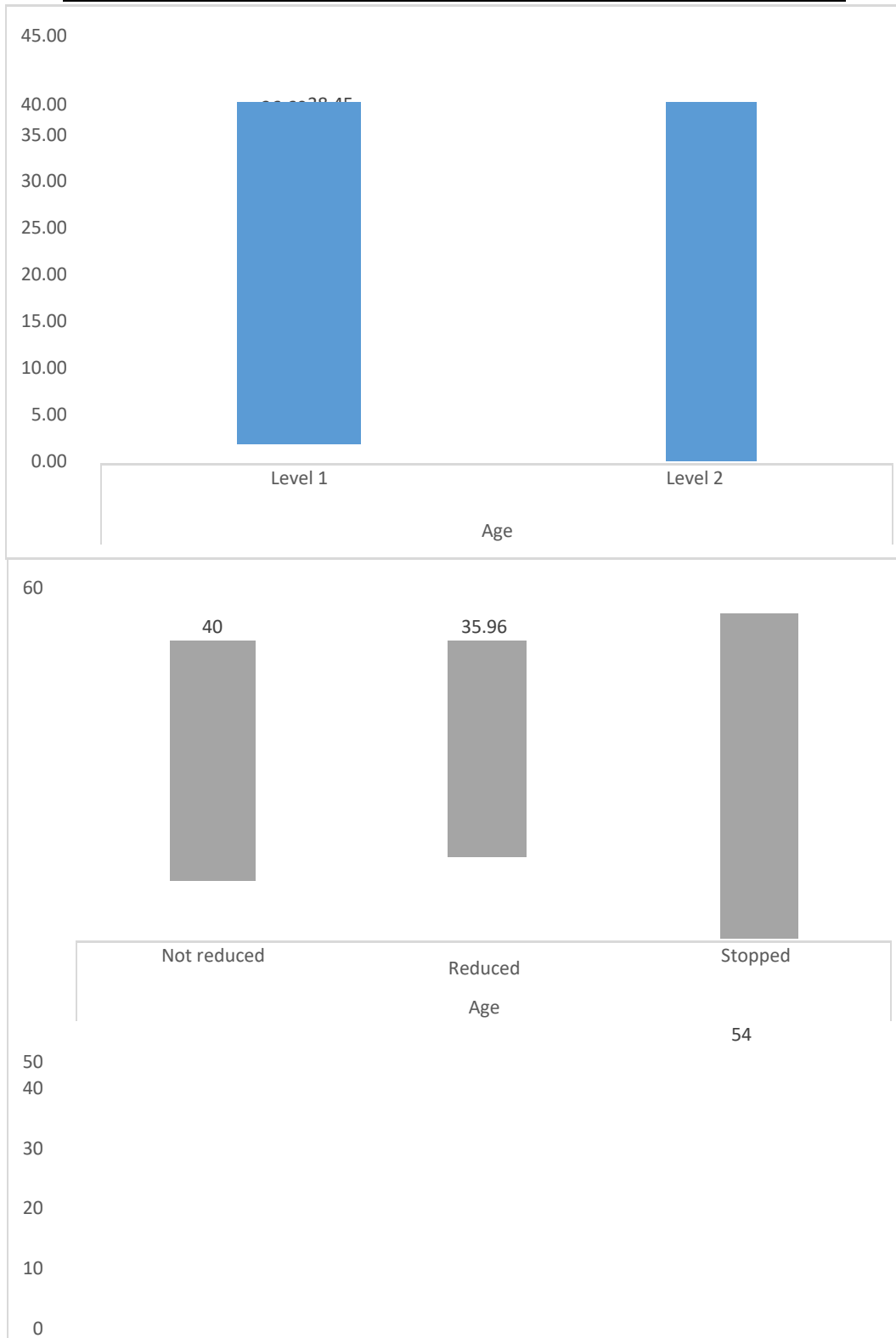


Table 9 explains that there was no significant difference exists in mean age of two levels by student t test with $p = 0.6$. Similarly, there was no significant

difference exists in mean age between post op snoring categories by one way ANOVA with $p = 0.1$ (Graph 9)

Table 10: Mean BMI comparison of levels

	Level	Mean	SD	t	P
BMI	Level 1	26.85	2.28	2.57	0.016
	Level 2	31.57	5.53		

Graph 10: Mean BMI comparison of levels

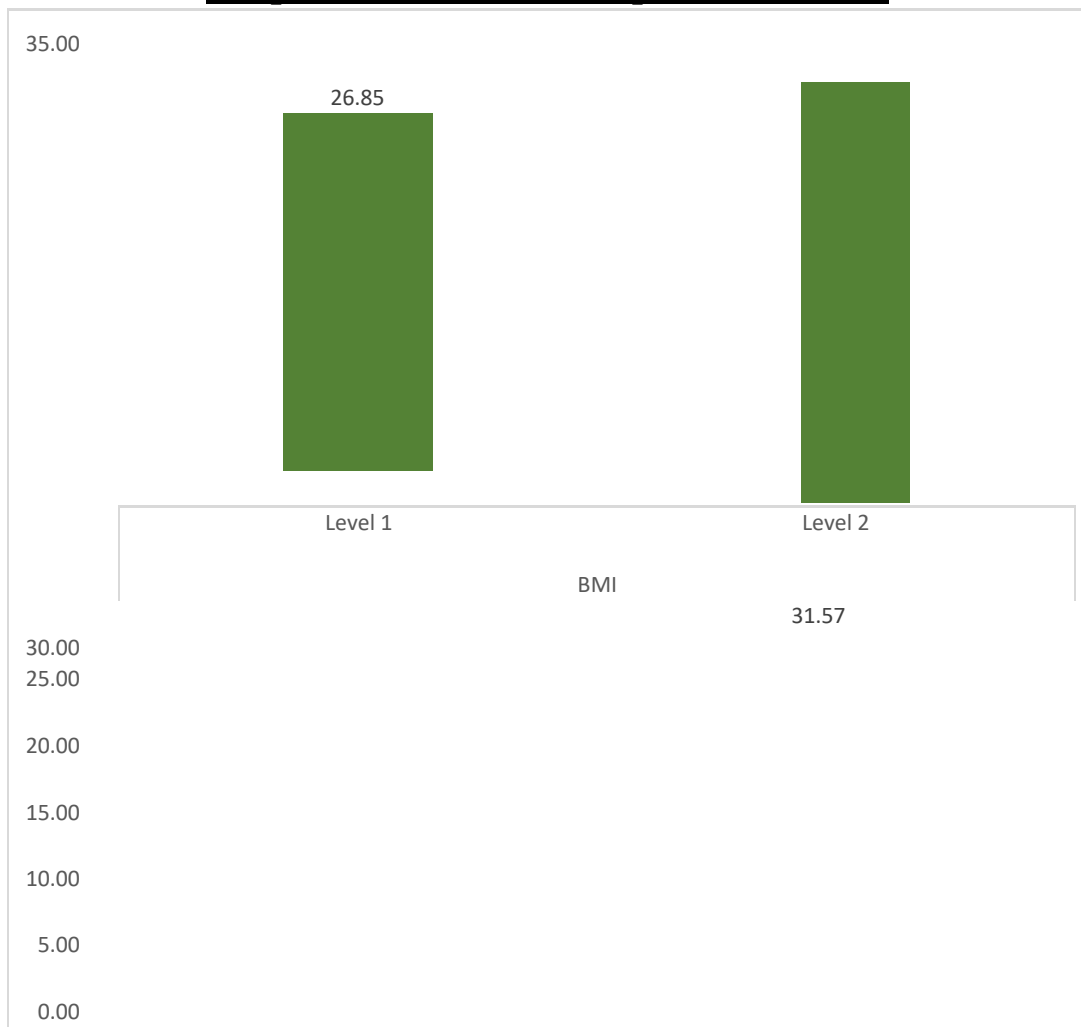


Table 10 explains that there was significant difference exists in mean BMI of two levels by student t test with $p = 0.01$. But, there was no significant difference exists in mean BMI between post op snoring categories by one way ANOVA with $p = 0.06$ (Graph 10).

Table 11: Mean Pre – Post op AHI comparison of levels

	Level	Mean	SD	t	P

Pre-opAHI	Level 1	20.48	4.98	3.25	0.003
	Level 2	45.89	24.30		
Post-opAHI	Level 1	6.99	2.39	2.41	0.023
	Level 2	15.59	11.05		

Graph 11: Mean Pre – Post op AHI comparison of levels

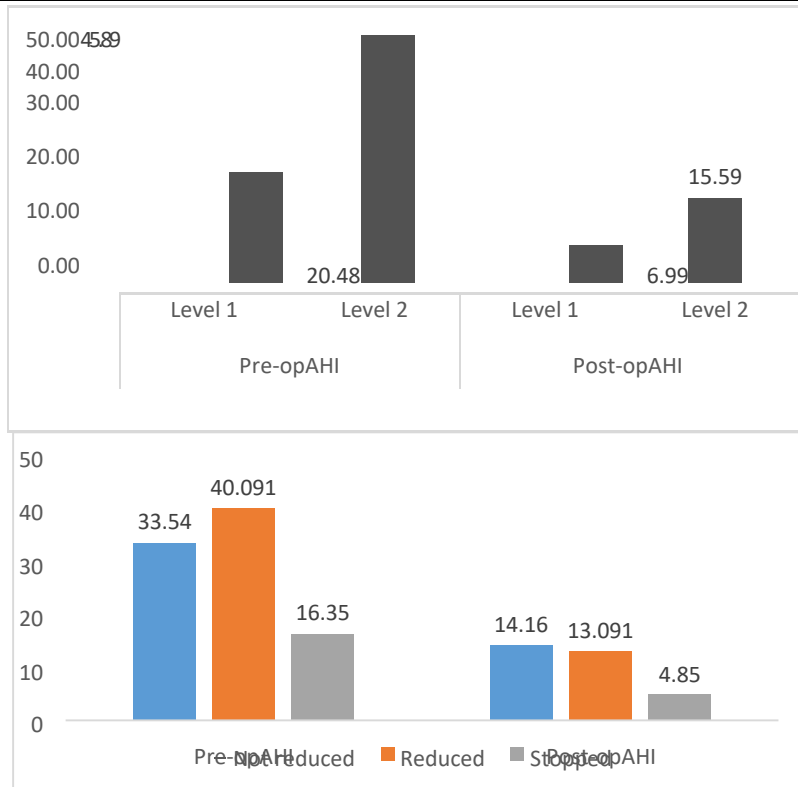


Table 11 explains that there were significant difference exists in mean Pre op AHI with two levels by student t test with $p = 0.003$; Post op AHI values with $p = 0.02$.

But, there was no significant difference exists in mean AHI between post op snoring categories by one way ANOVA for pre op AHI with $p = 0.3$ and for post op AHI with $p = 0.5$ (Graph 11).

Table 12: Gender wise comparison of levels

Sex	Level 1	Level 2	Total	Chisq	p
Male	8	17	25	0.12	0.7
Female	2	3	5		
Total	10	20	30		

Table 12 explains that there was no significant difference exists in levels due to gender by chi square test with $p = 0.7$ (Graph 12)

Graph 12: Gender wise comparison of levels

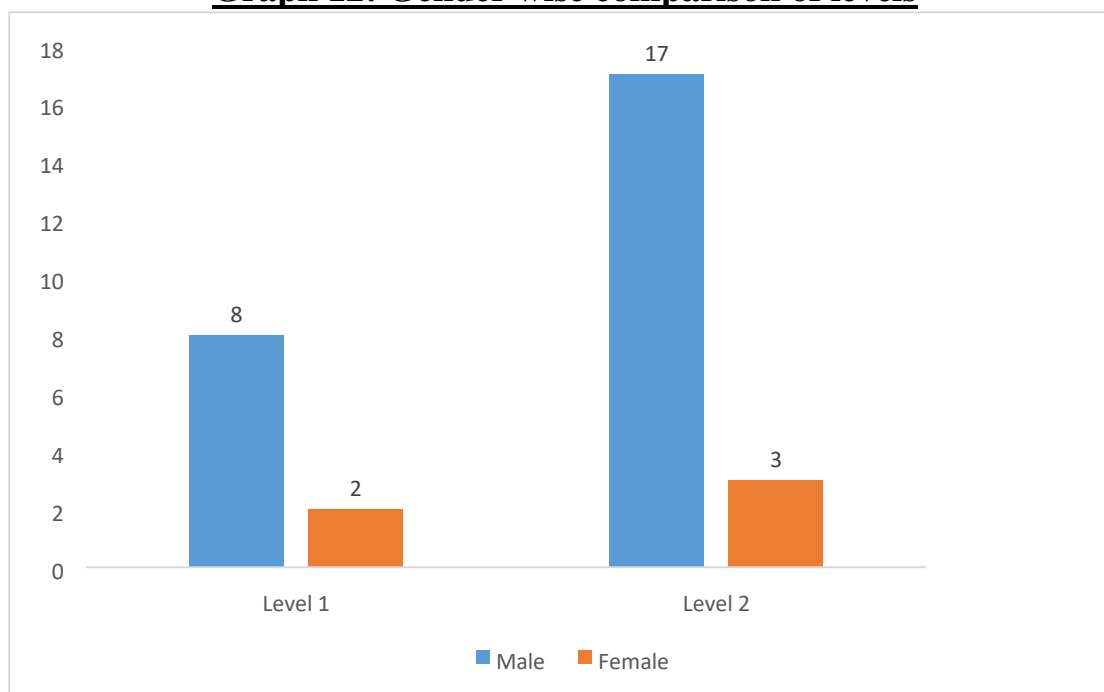


Table 13: Comorbidity comparison of levels

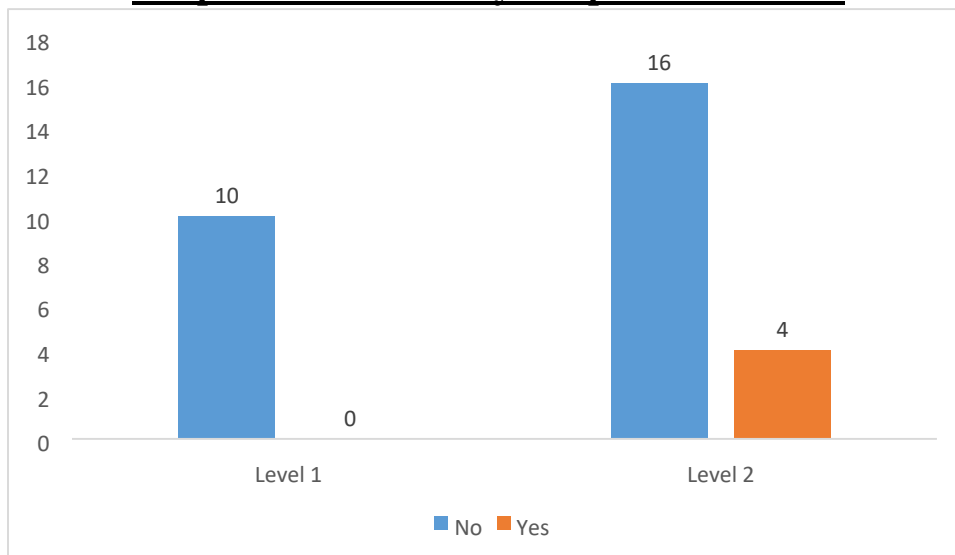
Diabetes	Level 1	Level 2	Total	Chisq	p
No	10	16	26	2.31	0.1
Yes	0	4	4		

Total	10	20	30		
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Hypertension	Level 1	Level 2	Total	Chisq	p
No	8	13	21	0.7	0.4
Yes	2	7	9		
Total	10	20	30		

Table 13 explains that there was no significant difference exists in levels due to diabetes by chi square test with $p = 0.1$ and hypertension with $p = 0.4$ (Graph 13)

Graph 13: Comorbidity comparison of levels



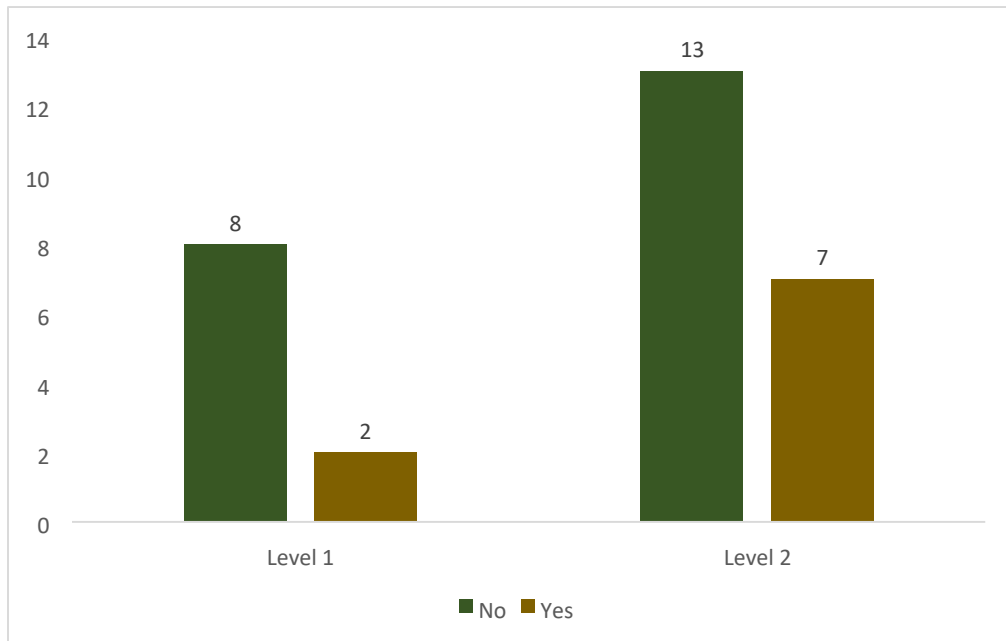


Table 14: Post op snoring comparison with levels

post op snoring	Level 1	Level 2	Total	Chisq	p
Not reduced	3	2	5	2.69	0.3
Reduced	7	16	23		
Stopped	0	2	2		
Total	10	20	30		

Table 14 explains that there was no significant difference exists in levels due to post op snoring by chi square test with $p = 0.3$ (Graph 14) **Graph 14: Post op snoring comparison with levels**

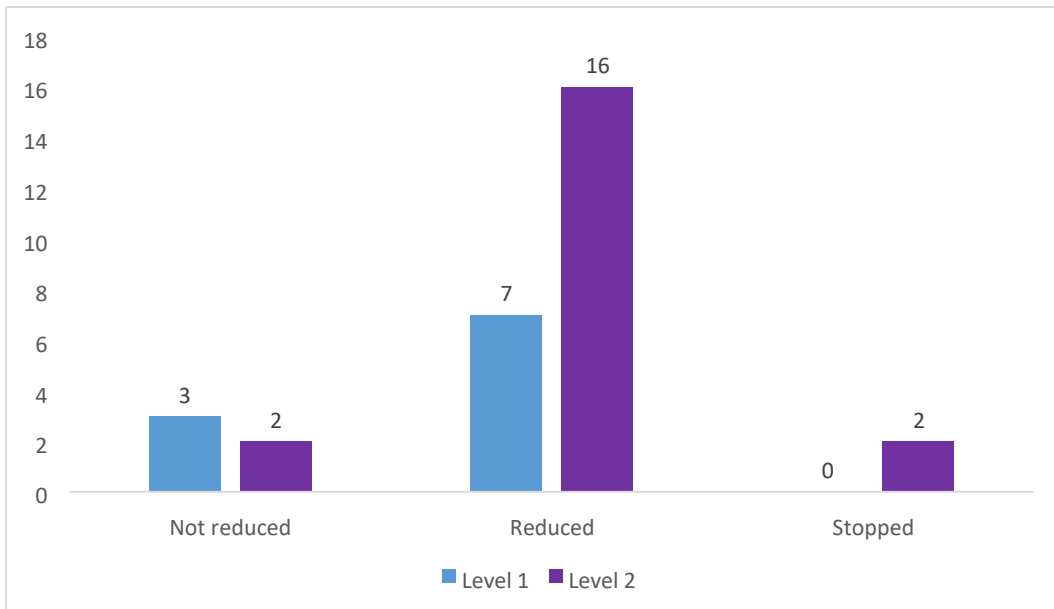


Table 15: Diagnosis wise comparison with levels

Diagnosis	Level 1	Level 2	Total
B/L grade3 tonsillar hypertrophy	0	2	2
chronic adenoid and tonsillar hypertrophy	0	2	2
Chronic rhinosinusitis	4	0	4
Deviated nasal septum with conchabullosa	2	0	2
Deviated nasal septum with inferior turbinate hypertrophy	4	0	4
chronic tonsillitis	0	2	2
Narrowing at velum and elongated Uvula	0	2	2
Narrowing at velum	0	4	4
Narrowing at velum. elongated edema uvula and soft palate	0	2	2
Narrowing in oropharynx and hypopharynx	0	1	1
Narrowing in oropharynx and velum	0	4	4
Total	10	20	30

Graph 15: Diagnosis wise comparison with levels

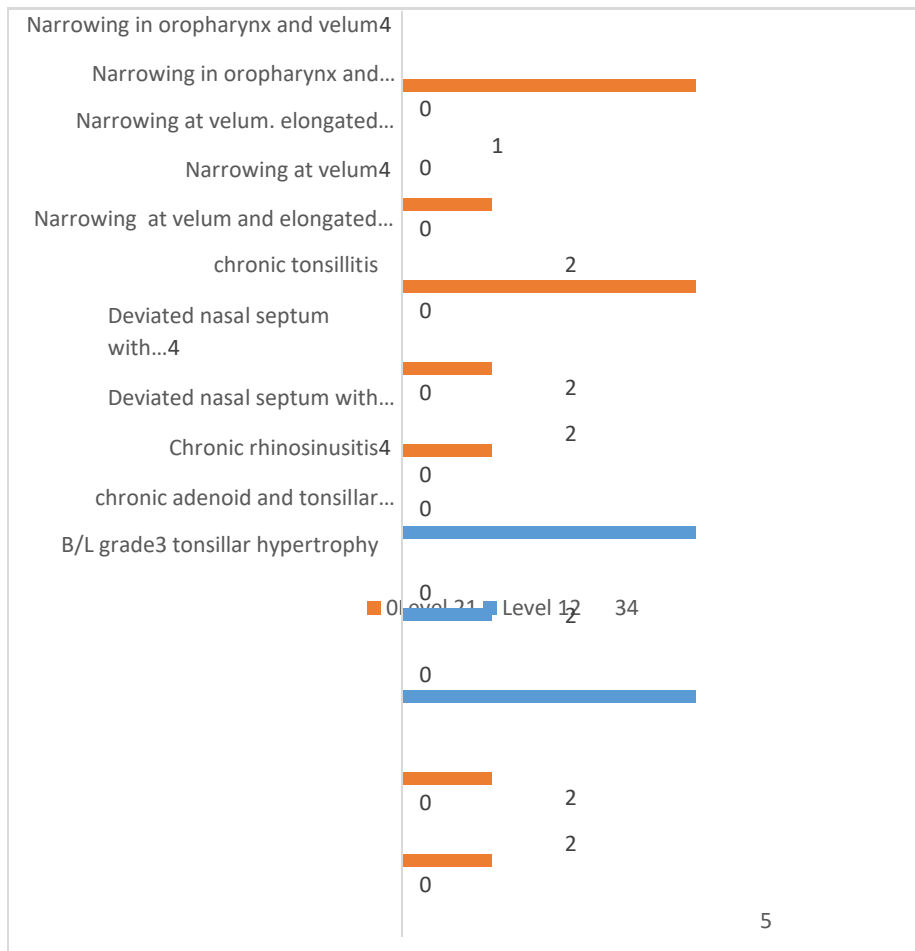


Table 16: Surgery wise comparison with levels

Type of surgery	Level 1	Level 2	Total
FESS	4	0	4
SMR with b/l turbinoplasty	2	0	2
SMR with b/l conchoplasty	2	0	2
Tonsillectomy	0	4	4
Tonsillectomy and Uvuloplasty	0	2	2
Uuvlopalatopharyngoplasty	0	14	14
septoplasty with conchoplasty	2	0	2

Total	10	20	30
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Graph 16: Surgery wise comparison with levels

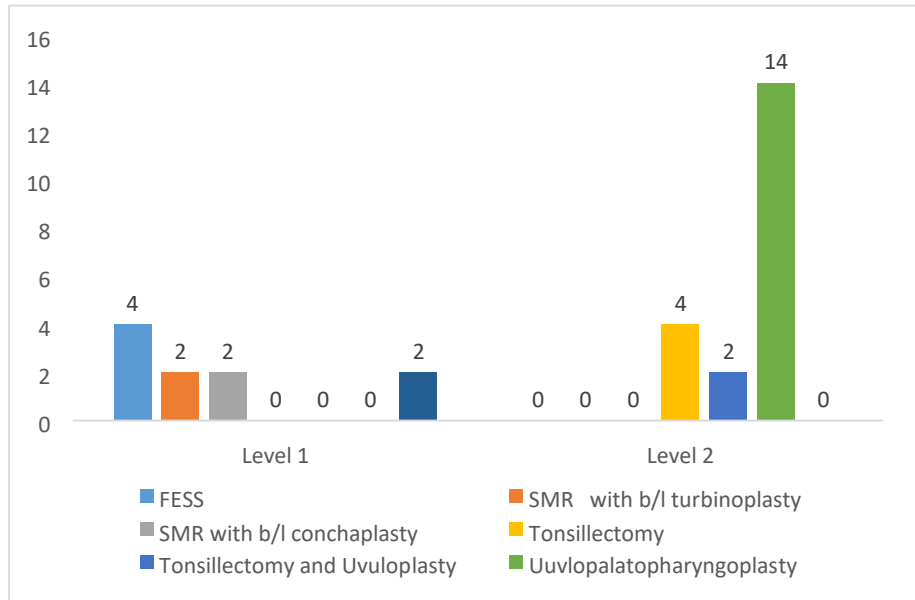


Table 17: Comparison of ESS with levels & Post op snoring

	Level	Mean	SD	t	P
ESS	Level 1	7.00	2.16	3.48	0.002
	Level 2	11.05	3.33		

Table 17 explains that there was significant difference exists in mean Ewsc of two levels by student t test with $p = 0.002$. But, there was no significant difference exists in mean ESS between post op snoring categories by one way ANOVA with $p = 0.09$ (Graph 17).

Graph 17: Comparison of ESS with levels , pre & Post op

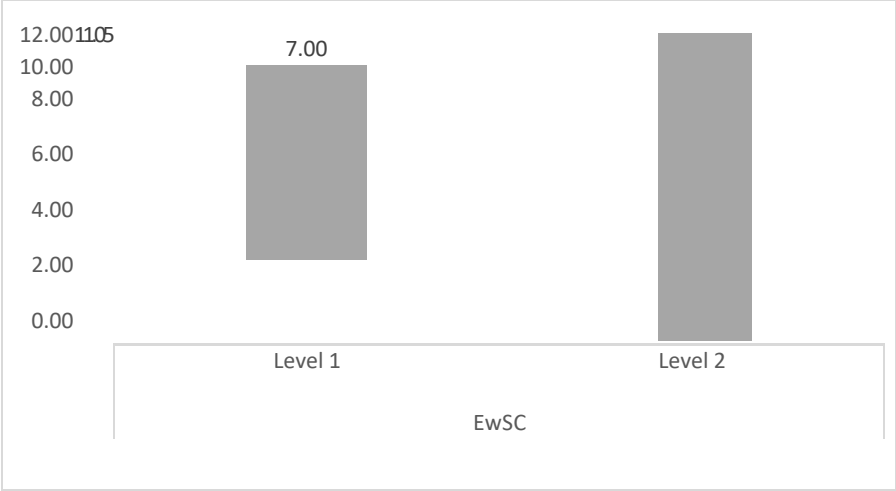


Table 18: Mean comparison of O2 with levels & Post op

	Level	Mean	SD	t	p
Pre op O2 saturation	Level 1	86.40	2.99	3.37	0.002
	Level 2	78.40	7.16		
post op O2	Level 1	94.20	1.32	1.19	0.244
	Level 2	93.10	2.75		

Table 18 explains that there was significant difference exists in mean O2 saturation of two levels by student t test with $p = 0.002$. But, there was no significant difference exists in mean post op O2 level of two levels by student t test with $p = 0.2$

Graph 18: Mean comparison of O2 with levels , Pre & Post op

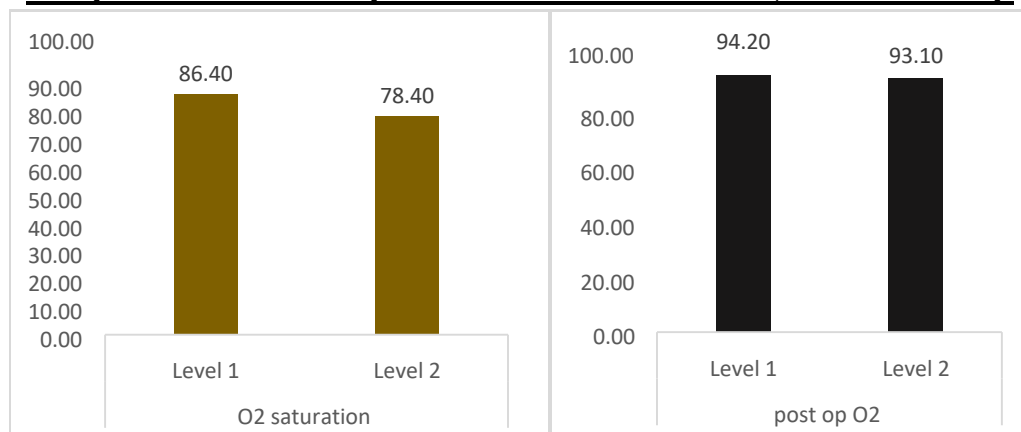


Table -19 Correlation analysis between study variables

		Sex	BMI	PreopAHI	PostopAHI	EwSC	Diabetes	Hypertension	O2 saturation	post op O2	Level
Age	Correlation	0.005	0.817	0.294	0.311	0.175	0.352	0.732	-0.67	-0.467	0.082
	p	0.978	0.0001	0.115	0.094	0.354	0.057	0.0001	0.0001	0.009	0.667
BMI	Correlation	-0.093									
	p	0.625									
Pre-op AHI	Correlation	-0.315	0.567								
	p	0.09	0.001								
Post-op AHI	Correlation	-0.171	0.513	0.815							
	p	0.367	0.004	0.0001							
EwSC	Correlation	-0.251	0.469	0.835	0.652						
	p	0.18	0.009	0.0001	0.0001						
Diabetes	Correlation	0.351	0.408	-0.045	-0.113	0.057					
	p	0.057	0.025	0.812	0.551	0.763					
Hypertension	Correlation	-0.098	0.694	0.475	0.467	0.298	-0.043				
	p	0.608	0.0001	0.008	0.009	0.11	0.822				
O2 saturation	Correlation	0	-0.788	-0.426	-0.372	-0.491	-0.43	-0.44			
	p	1	0.0001	0.019	0.043	0.006	0.017	0.014			

post op O2	Correlation	-0.053	-0.555	-0.48	-0.701	-0.349	-0.243	-0.558	0.446		
	p	0.782	0.001	0.007	0.0001	0.059	0.196	0.001	0.014		
Level	Correlation	-0.063	0.413	0.564	0.217	0.563	0.277	0.154	-0.542	-0.146	
	p	0.74	0.023	0.001	0.25	0.001	0.138	0.416	0.002	0.442	
Post op snoring	Correlation	0.259	0.256	-0.042	-0.287	-0.046	0.476	-0.023	-0.152	0.141	0.299
	p	0.167	0.172	0.827	0.124	0.81	0.008	0.905	0.424	0.457	0.109

Table 19 were explaining non parametric correlation analysis between the study variables at 5% level of significance. Age has positive correlation of 0.73 with hypertension ($p=0.0001$) and BMI; but age negatively correlated with O2 saturation (-0.67 , $p= 0.0001$) and post op O2 (-0.467 , $p =0.009$).

Body mass index had positive correlation with pre op AHI (0.567 , $p =0.001$), Post op AHI (0.513 , $p =0.004$), EwSc (0.469 , $p = 0.009$), diabetes (0.4 , $p =0.02$), hypertension (0.69 , $p =0.0001$) and levels (0.413 , $p=0.02$).

But, BMI had negatively correlated with O2 saturation (-0.788 , $p =0.0001$), post op O2 (-0.55 , $p= 0.001$). post op snoring had positive correlation with DM (0.476 , $p =0.008$).

Ewsc had positively correlated with pre op AHI (0.835 , $p =0.0001$) and Post Op AHI (0.65 , $p =0.0001$). Hypertension also had positive correlation with pre op and post op AHI $p<0.05$.

O2 saturation had negatively correlated with pre op AHI (-0.426 , $p =0.0001$), Post op AHI (-0.376 , $p =0.01$), Ewsc (-0.49 , $p=0.001$) DM (-0.43 , $p= 0.01$) and Hypertension (-0.44 , $p= 0.01$).

Post op o2 level had negatively correlated with pre op AHI (-0.555 , $p =0.001$), Post op AHI (-0.48 , $p =0.007$), Ewsc (-0.7 , $p=0.0001$) and hypertension (-0.55 , $p=0.001$). But had positive correlation with o2 saturation (0.446 , $p =0.01$).

DISCUSSION

A total of 30 patients were included in this study. All patients were evaluated clinically for sleepiness by epworth sleepiness scale and polysomnography in pre operatively and post operatively After 2 months.

UPPP, FEES, Tonsillectomy, Inferior turbinoplasty, septoplasty,

Adenoidectomy were done either alone or in combination.

Demographic details

- 1) 25 males and 5 females were recruited for study. Patients age ranged from 18 to 60 years. Most of the patients comes below 50 years. Mean age in between 36.60 to 38.45

Age distribution

- 2) Here majority of them were males 83%, female were 17%. But there is no significant difference in level of obstruction by gender.

Clinical details of patients

- 3) Hypertension (13.3%) was associated co morbidity in OSA patients even though obesity age, and diabetics are the confounding factors along with hypertension in OSA patient. Male patients are have more prevalence than female patients. But no significant difference correlation depending upon level of obstruction.
- 4) Mean BMI was 29.7% .In our study BMI has positively correlate with the pre operative AHI of 0.567

- 5) 66.7% patient of having obstruction by clinical diagnosis in oropharynx that means level 2 than the nasal cavity and nasopharynx 33.3% (level 1)
- 6) 66.3% patient undergone surgery at oropharyngeal level. Of which 46.7 % were uvulopalatoplasty, 13.3% of tonsillectomy . septal correction with conchoplasty and FESS were found 13.4 % . Turbinoplasty done for 6.7%.
- 7) In our study there is not much difference in age in comparison with level of obstruction. The mean age of level 2 was 38.45 with SD 13.28. and level 1 the mean age was 36.60 withSD 9.65
- 8) Mean value of AHI index in pre operatively was 37.42 with SD 23.30. That means most of the patients were coming under severe OSA.
- 9) In our study there was significant difference in Mean value of AHI IN PRE AND POST operatively. The mean pre op AHI is 37.42 and post op AHI was 12.72. And mean AHI in level 1 is 20.48 and 45. 89 in level 2 obstruction it indicates level 2 obstruction produce significant AHI index.
- 10) In Subjective assessments snoring in post operative was enquired of which tonsillectomy patients have almost snoring was stopped with compared with other surgeries and level of obstruction
- 11) There was significant difference showed in mean O₂ saturation of two level and more reduction of saturation in level 2 obstruction than nose and nasopharynx. But post-operatively no difference was noted that indicates improvement was observed in both level post-operatively.

12) The Epworth sleepiness scale was compared pre and postoperative period in both level patients. , it shows significant improvements in sleepiness after surgery. In level one patient having score of 7.00 wit 2.16 SD and level 2 score ranged from 11.05 to 3.33.

13)AHI and Epworth sleepiness scale were positively correlate with the BMI and negatively correlate with the O2 saturation. It indicate BMI it may the one of the co factor to produce sleep related breathing disorder. But it need long term post operative follow up need to suggest this.

DISCUSSION FROM OTHER STUDIES

The results of our studies were analysed and compared with other literature as follows

On statistical analysis on a study done in our institution, it was found that the study group comprised mainly male(85.3%). This data correlates with the study conducted by **Akram khan, MD et al** where the population comprised of 81% men. This suggests that incidence of OSA is more common in male population.

Akram khan ,MD et al³² “conducted a study from jan 1988 to aug 2006 in 63 patients and analysed PSG and BMI in patients with OSA who are more than 18 years of age the mean age of the population was 42.1 comprising primarily men(81%) and mean pre op AHI is 62 who underwent UPPP . He concluded that AHI reduced to 50% the previous value in 32 patients with a success rate of 51%”.

The mean age was 37.15 ± 5.8 and the mean AHI was 41.88 ± 15.35 . The mean AHI in our study correlated with **Janson C et al and PanKP** where the mean AHI was 40.1 and 47.3 respectively suggesting that most of the OSA patients falls under severe OSA category.

Yu Q, Yin G, Zhang P, Song Z, Chen Y, Zhang D Et all (2014)

distinct association between hypertension and OSA. Our study also correlate with this study.

The mean BMI in the study group comprises 29.7 coming under overweight category. This also correlates with **Pang KP and Woodson et al** where the study group had a BMI < 30.

And correlate with the **Mark A. Brown, M.D., JAMES L. Goodwin**, This study suggest that obesity and weight gain and correlate with severity of AHI index. Weight gain is arisk factor for developing and worsening Sleep Disorderded Breathing.

In this study, the data of 483 patients were reviewed retrospectively. The correlation between ESS and polysomnography (PSG) findings were assessed, The mean apnea-hypopnea index (AHI) was 27.71 ± 26.6 , the mean ESS score was 8.42 ± 4.88 . According to AHI, a statistically significant difference between ESS scores was detected . According to AHI (AHI ≥ 5 , AHI ≥ 15 and AHI ≥ 30) the best cutoff score for ESS score was found as 8 These results indicate that a score of 8 or higher on the ESS would seem a more appropriate cutoff score to suspect clinically relevant sleepiness .Kum,

R.O., Özcan, M., Yurtsever Kum, N. *et al.* A new suggestion for the Epworth Sleepiness Scale in obstructive sleep apnea. *Eur Arch Otorhinolaryngol* **272**, 247–252 (2015). <https://doi.org/10.1007/s00405-0143242-9>. Our study correlate with this study by ESS between 7 to 13. Almost 100% of individuals had snoring preoperatively which correlates with the study conducted by **Whyte KF et al.**

Snoring gets cured completely in 2 individuals and reduced in severity in 23 individuals after surgery as witnessed by partner. 5 patient not post op snoring not reduced. These patients need further evaluation. Sites of Obstruction in Obstructive Sleep Apnea in this study done by Anil N.RamaMD, MPH^aShivan H.TekwaniBS^aClete A.Kushida MD, PhD^a review was conducted by a MEDLINE search of the English literature published during the years 1980 to 2002. In review was conducted by a MEDLINE search of the English literature published during the years 1980 to 2002 the most common site of obstruction detected by these studies was at the level of the oropharynx, with extension to the laryngopharynx commonly observed..

Thomas verse et al³⁴ “conducted a study of tonsillectomy as a treatment of OSA in adults. The study was conducted in 11 patients between aug 1996 to aug 1999.. All were subjected to PSG. Of which 9 patients had OSA of varying degrees. After tonsillectomy he found that 100% improvement in OSA of mild degree and 80.8% improvement in severe OSA without any complications. He concluded that in carefully selected patients tonsillectomy

can be a safe option for OSA disorder”. **Janson C et al**³⁵ “conducted a study in 1997 in 25 patients and concluded that after UPPP Reduced prevalence of snoring and daytime sleepiness and reduction in AHI (mean [\pm SD], 40 \pm 26 to 21 \pm 21) at follow-up ($P < .001$). Sixteen patients (64%) were responders after 6 months and 12 (48%) at the long-term follow-up. Responders had a lower preoperative AHI (25 \pm 7) than non responders (48 \pm 29) ($P < .05$). None of the 7 patients with preoperative AHI of more than 40 were responders ($P < .01$). No difference was seen in preoperative body mass index, lung function, ventilatory response to carbon dioxide, computed tomography scan of upper airways, or change in body mass index between responders and nonresponders

Staurt et al “conducted a study in 48 patients. He compared surgical outcomes of efficacy of modified UPPP with coblation channeling of tongue. Patients were followed up for 3 months. At the end of 3 months PSG was taken. Post op clinical assessment questionnaire and BMI were compared. AHI reduced from 23.1 to 5.6 and ESS reduced to 5 from pre op value of 10.5”.

Pang KP and Woodson et al “conducted a prospective RCT to assess the efficacy of ESP in OSA patients. They did a study in 45 adults with small tonsil, BMI < 30 , friedman stage 2 and 3 with lateral wall collapse. They found that AHI reduced from 44.2 to 12 and lowest oxygen saturation

improved from 78 to 85. He also found that in UPPP group AHI reduced from 38.1 to 19.6 and lowest oxygen saturation from 75.1 to 86.6. they found that success rate was 82.6% in ESP and 68.1% for UPPP. They concluded that ESP offers benefits in selected group of patients". questionnaire and BMI were compared. AHI reduced from 23.1 to 5.6 and ESS reduced to 5 from pre op value of 10.5". The study conducted by **Den Herder et al.** He reported that, of 127 patients, 63% had single level obstruction while only 37% had multilevel disease. But study conducted by **Riley et al** shows 93.3% (223 patients) were identified as having multilevel obstruction. Another study by **Abdullah van Hasselt et al** showed higher incidence of multilevel disease (87% of their 893 patient populations had multilevel obstruction).

Post operative follow up PSG done after 2 months in our study, it was found that post operative AHI reduced t than the preoperative value in 30 patients which is statistically significant and show the success. In a study conducted by **Akram khan et al and Elshaug et al** success rate was correlate with our study.

In our study group AHI 8.82% of patients had 50-60% improvement in AHI. 29.4% patients had improvement in the AHI upto 60-70% of preoperative value and 38.23% of individuals had 70- 80% improvement in post op AHI.

On statistical analysis, two patients in our study underwent tonsillectomy with uvuloplasty as a sole surgical treatment for OSA. And these two patients improved well without any complications accounting a success rate of 100%.

This result correlates with the study conducted by **Thomas verse et al and Stow et al**, where the result was 100% in mild group and 80.8% in severe OSA group. In our study the study population had a BMI of average 25, therefore patients with moderate OSA also responds to tonsillectomy if the obstruction is due to enlarged tonsil alone. But the disadvantage is lack of adequate population group.

Lin et al “conducted a systemic review/ meta analysis on outcome of patients with OSA. He pooled 58 studies and the OSA patients were treated with multilevel surgery for upper airway. Patients were followed up for a period of mean 7.3 months. They found that success rate was 66.4% and complication rate was 14.6%”. questionnaire and BMI were compared. AHI reduced from 23.1 to 5.6 and ESS reduced to 5 from pre op value of 10.5”.

“**Keny P. Pang and Jin Keat Siow et al**” conducted a study in 487 patients from jan 2007 to may 2010 on safety of multilevel surgery on obstructive sleep apnea. The mean AHI was 47.3. The study population mainly comprised of men. Multilevel surgery comprising nasal surgery, palate surgery(UPPP, ESP and anterior palatoplasty), tongue surgery was done. The results are, 7.1% of overall complication has occurred.

Complications are postoperative desaturation, persistent hypertension, secondary hemorrhage, negative pressure pulmonary edema, upper airway obstruction requiring reintubation. They concluded that all OSA patients

should be monitored in post anaesthesia care unit after surgery and based on the outcome in this period, patients should be shifted to general ward”.

Among 30 patients, 28(82.4) patients had a postop oxygen saturation >90%, snoring reduced in 30 patients and ESS normalized(<10) in 27 patients. This suggests that after surgery there is both in subjective and objective improvement.

In our study in BMI index were there is no significant change between preop and post op BMI because lack of follow up duration and need continues follow up to suggests that weight reduction has a major role in the outcome of surgery.

CONCLUSION

Obstructive sleep apnea which is an entity of sleep disordered breathing leads on to various systemic consequences, if left untreated. Thus in the study conducted in our institution patients of OSA were clinically

evaluated and admitted in ward and perform pre op AHI in polysomnography. After identifying the site of obstruction, patients were channelized to different surgeries addressing velum, tonsil and uvula, turbinate, septum, sinuses. Significant proportion of patients improved both subjectively and objectively as determined by reduction in AHI from 37.42 to 12.72, reduction in ESS from 12.41 ± 3.09 to 5.14 ± 3.41 and reduction in snoring. Since the complications of surgeries were anticipated preoperatively and managed accordingly.

Thus nasal pathologies were produce significant O2 desaturation and significant AHI so addressing the nasal pathology by surgery in selected single level obstruction patient it may improve nasal patency and help to improve nasal resistance.

Level 2 obstruction produce more obstruction so effective with proper preoperative investigations, appropriate surgery, anticipation and management of complications was needed.

Lifestyle modifications should be consider to achieve better results.

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PATIENT CONSENT FORM

Title of the Project

A STUDY ABOUT PRE AND POAT OPERATIVE EVALUATION OF APNEA HYPOAPNEA INDEX IN POLYSOMNOGRAPHY IN ADULT OBSTUCTIVE SLEEP APNEA HYPONEA SYNDROME PATIENT”

Institution **Department** **of**
Otorhinolaryngology,
Madurai Medical College, **Madurai**
— 625020.

Name :	Date
Age	IP No.
Sex	Project Patient No.

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study.

Name of the subject

Signature

Date

Date

Name of the Investigator

Signature

INFORMATION SHEET

- We are conducting a prospective cohort study on

“A STUDY ABOUT PRE AND POST OPERATIVE EVALUATION OF APNEO AND HYPOAPNEA INDEX IN POLYSOMNOGRAPHY IN ADULT OBSTRUCTIVE SLEEP APNEA HYPONEA SYNDROME PATIENT”

at the . Department of Otorhinolaryngology, Madurai Medical College & Rajaji Government Hospital, Madurai.

- For snorers where the pathology is due to obstructive cause, surgery is one of the modality of treatment to make the patient free off symptoms and prevent the progression of disease and further complications.
- Polysomnography is a non invasive method of study which will help in identifying the severity of the disease and analyse the effectiveness of surgery using various parameters.
- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator

Signature of Participant

Date

ஆராய்ச்சி ஒப்புதல் படிவம்

பெயர் :

தேதி :

வயது :

நோயாளி எண் :

ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விபரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பெயரில்தான் பங்கு பெற்கிறேன். மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்த நேரமும் பின்வாங்கலாம் என்றும் அதனால் எந்த பாதிப்பும் எனக்கு ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் பங்கு கொள்ள சம்மதிக்கிறேன்.

PROFORMA

Name :

Date :

Age :

Ip / Op no :

Sex :

Occupation :

Presenting complaints :

History of presenting complaints :

S.no.	Complaints	yes	no	duration
1.	History of snoring			
2.	History of sleep awakening / insomnia			
3.	History of excessive day time sleepiness			
4.	History of impaired cognitive function			
5.	History of witnessed			
6.	History of increased weight gain			
7.	History of choking in sleep			
8.	History of nasal obstruction			
9.	History of mouth breathing			

Past history : Diabetes mellitus / Hypertension / Epilepsy / Asthma /
Jaundice / previous history of accidents

Personal history : Diet , appetite , smoking , alcohol , chronic drug intake ,
bowel and bladder habits

Family history : married / unmarried **General**

examination :

Built :

Height :

Weight :

BMI :

Neck circumference :

Waist circumference :

Temperature ; Pallor

:

Cyanosis :

Jaundice :

Pedal edema :

Vitals ;

Pulse : BP : Respiratory rate :

Systemic examination :

Respiratory system :

Cardiovascular system :

Central nervous system :

ENT Examination :

Throat :

Oral cavity :

Gums :

Oral mucosa :

Floor of mouth :

Anterior 2/3rd tongue :

Hard and soft palate :

MALLAMPATI score :

Oropharynx :

Antr. Pillar :

Postr. Pillar :

Tonsil :

Posterior pharyngeal wall :

Indirect laryngoscopy :

Posterior 1/3rd tongue :

Vallecula :

Epiglottis :

Vocal cords and mobility :

Posterior pharyngeal wall :

Nose :

External contour :

Antr. Rhinoscopy :

Postr. Rhinoscopy :

Ear :

Pinna :

External auditory canal :

Tympanic membrane :

Neck :

Laryngeal contour :

Abnormal veins / scars :

Accessory muscles of respiration :

Investigations :

Blood investigations :

Thyroid functions test :

Diagnostic nasal endoscopy :

Video direct laryngoscopy :

CT PNS

CT NECK

x- ray chest :

Patient name

: Diagnosis

:

Managem

ent :

- Investigations
- Anesthesia
- Procedure

S.No	Age	Sex	BMI	Preop AHI	Post-op AHI after 2 months	EwSC	Diabetes	Hypertension	O2 saturation	post op O2	Diagnosis	Type of surgery	Level	post op symptom snoring
1	23	F	23.4	21.6	11.2	9	No	no	89	93	Deviated nasal septum with conchabullosa	septoplasty with conchaplasty	1	not reduced
2	29	M	28.2	17.8	6.3	8	no	no	81	94	Deviated nasal septum with inferior turbinate hypertrophy	SMR with b/l conchaplasty	1	reduced
3	57	M	39.1	82.3	33.1	11	no	yes	69	88	Narrowing in oropharynx and	Uuvlopalatopharyngoplasty	2	reduced
4	53	F	38.4	16.1	4.9	6	yes	yes	65	93	B/L grade4 tonsillar hypertrophy	Tonsillectomy	2	stopped
5	51	M	30.1	29.3	7.4	8	no	yes	89	94	Chronic rhinosinusitis	FESS	1	reduced
6	42	M	25.9	18.9	5.2	7	no	no	87	95	Chronic rhinosinusitis	FESS	1	reduced
7	18	M	27.2	39.4	7.3	11	no	no	89	94	chronic adenoid and tonsillar hypertrophy	Uuvlopalatopharyngoplasty	2	reduced
8	41	M	33.6	66.3	27.1	14	yes	no	76	89	Narrowing in oropharynx and velum	Uuvlopalatopharyngoplasty	2	reduced
9	27	M	29.1	42.1	15.2	11	no	no	84	91	Narrowing at velum. elongated edema uvula and soft palate	Uuvlopalatopharyngoplasty	2	reduced
10	40	M	26.3	14.2	5.3	3	no	no	83	93	Deviated nasal septum with inferior turbinate hypertrophy	SMR with b/l turbinoplasty	1	not reduced
11	45	M	32.1	58.9	22.5	12	no	yes	77	90	Narrowing in oropharynx and velum	Uuvlopalatopharyngoplasty	2	not reduced
12	48	M	39.2	84.1	33.2	16	no	yes	70	89	Narrowing at velum	Uuvlopalatopharyngoplasty	2	reduced
13	22	M	24.9	16.9	5.4	5	no	no	88	94	chronic tonsillitis	Tonsillectomy	2	reduced
14	35	F	27.8	35.2	10.3	11	no	no	79	96	Narrowing at velum.	Uuvlopalatopharyngoplasty	2	reduced
15	28	M	26.5	24.2	5.1	12	no	no	80	96	Narrowing at velum and elongated Uvula	Tonsillectomy and Uvuloplasty	2	reduced
16	25	F	24.1	20.6	11.3	8	No	no	89	92	Deviated nasal septum with conchabullosa	septoplasty with conchaplasty	1	reduced
17	28	M	27.2	18.8	5.3	8	no	no	87	96	Deviated nasal septum with inferior turbinate hypertrophy	SMR with b/l conchaplasty	1	reduced
18	59	M	39.4	81.3	32.1	12	no	yes	70	91	Narrowing in oropharynx and hypopharynx	Uuvlopalatopharyngoplasty	2	reduced

19	55	F	37.4	16.6	4.8	6	yes	no	78	94	B/L grade3 tonsillar hypertrophy	Tonsillectomy	2	stopped
20	47	M	30.3	28.5	7.2	8	no	yes	89	94	Chronic rhinosinusitis	FESS	1	reduced
21	41	M	25.7	19.9	5.4	8	no	no	87	95	Chronic rhinosinusitis	FESS	1	reduced
22	19	m	24.2	25.4	5.2	11	no	no	89	96	chronic adenoid and tonsillar hypertrophy	Uuvlopalatopharyngoplasty	2	reduced
23	45	M	33.9	67.3	17.1	16	yes	no	76	93	Narrowing in oropharynx and velum	Uuvlopalatopharyngoplasty	2	reduced
24	31	M	28.1	32.1	11.2	11	no	no	84	96	Narrowing at velum. elongated edema uvula and soft palate	Uuvlopalatopharyngoplasty	2	reduced
25	40	M	27.3	15.2	5.3	3	no	no	83	96	Deviated nasal septum with inferior turbinate hypertrophy	SMR with b/l turbinoplasty	1	not reduced
26	52	M	32.3	57.8	26.5	12	no	yes	77	91	Narrowing in oropharynx and velum	Uuvlopalatopharyngoplasty	2	not reduced
27	46	M	38.9	82.1	30.2	16	no	yes	70	93	Narrowing at velum	Uuvlopalatopharyngoplasty	2	reduced
28	25	M	24.9	29.2	5.1	5	no	no	88	96	chronic tonsillitis	Tonsillectomy	2	reduced
29	35	M	27.8	36.2	10.3	11	no	no	79	96	Narrowing at velum.	Uuvlopalatopharyngoplasty	2	reduced
30	28	M	26.5	24.2	5.1	12	no	no	80	96	Narrowing at velum and elongated Uvula	Tonsillectomy and Uvuloplasty	2	reduced



INSTITUTIONAL ETHICS COMMITTEE
MADURAI MEDICAL COLLEGE & GOVT. RAJAJI HOSPITAL, MADURAI
CDSCO:Reg.No.ECR/1365/Inst/TN/2020 &
DHR Reg.No.EC/NEW/INST/2020/484

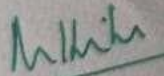
Study Title	: A Study about Pre and Post Operative evaluation of Apnea- Hypoapnea index in polysomnography in adult obstructive sleep apnea syndrome patients
Principle Investigator	: Dr.P.Cindhuja
Designation	: PG in MS., Otorhinolaryngology (2019-2022)
Guide	: Dr.Prof. Dr.Dinakaran, MS., ENT Professor and Head of ENT
Department	: Department of ENT, Government Rajaji Hospital & Madurai Medical College, Madurai

The request for an approval from the Institutional Ethics Committee (IEC) was considered on the IEC meeting held on **08.02.2021** Auditorium, Govt. Rajaji Hospital, Madurai at 10.00 AM.


The Members of the committee, the Secretary and the Chairman are pleased to inform you that your proposed project mentioned above is **Approved**.

You should inform the IEC in case of any changes in study procedure, methodology, sample size investigation, Investigator or guide or any other changes.

1. You should not deviate from the area of work for which you had applied for ethical clearance.
2. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions. If encountered during from study.
3. You should abide to the rules and regulations of the institution(s)
4. You should complete the work within the specific period and if any extension is required, you should apply for the permission again for extension period.
5. You should submit the summary of the work to the ethical committee on completion of the study.


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INTRODUCTION

Obstructive sleep apnoea (OSA) can be regarded as a condition characterized by repetitive upper airway obstruction leading to sleep fragmentation, cardiovascular stimulation and oxygen desaturation during sleep. These together lead to



Mobile view



Edit

