IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

ORTHODONTIC CONSIDERATION IN OBSTRUCTIVE SLEEP APNEA

1Aarthiga priyadharshini k, 2J.Dinesh kumar, 3NV.Adithya nair, 4Dr.MK.Karthikeyan, 5Dr.U.Vivekanandan
1Intern, 2MDS, 3Intern, 4Head of the department , 5Reader
1Thaimoogambigai dental college and hospit,
2Thaimoogambigai dental college,
3Thaimoogambigai dental college,
4Thaimoogambigai dental college,
5Thaimoogambigai dental college

ABSTRACT

A Common disorder called obstructive sleep apnoea OSA characterised by recurrent bouts of p breathing stoppage caused by upper airway collapse's is characterised by severe symptoms, including excessive daytime sleepiness, and is strongly linked to high cardiovascular morbidity and mortality. For the proper management of this disease, a variety of therapy alternatives are currently available. Continuous positive airway pressure (CPAP) is still regarded as the gold standard of care more than three decades after it was first used. Nasal CPAP (nCPAP) is very effective at treating sleep apnea-related clinical sequelae and controlling symptoms while also enhancing quality of life. For individuals who are unable to tolerate CPAP or require high amounts of positive pressure, there are other positive airway pressure treatment options. Mandibular advancement devices, especially if they are manufactured to order, are efficient in treating mild to severe OSA and offer a good substitute for patients who are unable to tolerate CPAP therapy. The function of surgery is still debatable. When CPAP therapy has failed, the well-established technique of uvulopalatopharyngoplasty may be considered, but patients with a craniofacial deformity may benefit from maxilla-mandibular surgery. There are some minimally invasive snoring treatments being looked at right now. All people with obesity benefit from weight loss in terms of symptoms and morbidity, and bariatric surgery is a possibility for those with extreme obesity. A multidisciplinary approach is required for effective disease management.

INTRODUCTION

Obstructive sleep apnoea (OSA) is a widespread condition marked by recurrent bouts of nocturnal breathing stoppage brought on by upper airway collapse. Significant cardiovascular morbidity and death are linked to OSA, which has severe symptoms such increased daytime sleepiness. Different degrees of nightly monitoring of respiratory, sleep, and cardiac parameters (polisomnography or nocturnal cardio-respiratory poligraphy), designed to detect obstructive events and the ensuing changes in blood oxygen saturation (SaO2), are used to diagnose OSA(1). The apnoea/hypopnea index (AHI), computed as the number of obstructive episodes per hour of sleep and determined by nocturnal cardiorespiratory monitoring, is the indicator that is most frequently used to describe the severity of OSA. There are now a variety of therapy alternatives accessible for this illness management. Continuous positive airway pressure (CPAP) is still regarded as the gold standard of treatment more than three decades after it was first used. Nasal CPAP (nCPAP) is very effective at treating sleep apnoea-related clinical sequelae and controlling symptoms while also enhancing quality of life. For individuals who cannot tolerate CPAP or need high amounts of positive pressure, other positive airway pressure techniques are available. For patients who are intolerant to CPAP therapy, mandibular advancement devices—especially those that are tailored to order—are useful in treating mild to severe OSA(2). We will talk about various orthodontic treatment options for obstructive sleep apnea in this review.

HISTORY

Early in 1837, the "posthumous writings of the pickwick club" reported obstructive sleep apnea. The phrase "pickwick syndrome," coined in 1914 to describe obesity and sleep patients in homage to Charles Dickens' character Joe, was first used by a British author to describe JOE, the primary character, as a large youngster who falls asleep easily and involuntarily.(Figure-1) The Pickwick syndrome and severe obesity and alveolar hypertension were linked, according to a 1956 research by Beckmann et al(3). In an effort to better understand its defining trait, recent study on OSA has been done. A cycle of five phases controls sleep mostly in adults. Rapid eye movement sleep, also known as paradoxical sleep, occurs during the fifth period, which follows the first four non-rapid eye movement sleep cycles. There are four to six sleep cycles per night, each of which includes the movement from the first stage to the RAM and involves the activation of the brain, body muscles, and the heart and breathing systems. OSA is brought on by a reduction or stoppage of airflow due to partial or total obstruction of numerous levels of the upper airway. O2 saturation decreases and carbon dioxide partial pressure may occasionally rise as a result of inadequate alveolar ventilation, which is why multidisciplinary management is mostly needed for OSA treatment.(4)

PATHOPHYSIOLOGY

OSA is defined by recurrent partial or total collapse of the upper airway while sleeping, leading to episodic decrease (hypo-nea) or stoppage (apnea) of airflow despite respiratory effort. To keep the airway open during inspiration, the upper airway dilator muscles must be contracted. (Figure-2)The levator and tensor palatini muscles (which advance and elevate the soft palate) as well as the Geniohyoid and stylopharyngeus muscles aid the genioglossus muscle, which contracts with each inspiration to prevent posterior collapse of the tongue (opposing medial collapse of the lateral pharyngeal walls). It has been suggested that people who have OSA have impaired genioglossus muscle function, a tongue muscle. When there is an effort to inhale while sleeping, this results in the tongue prolapsing against the posterior pharyngeal wall.(7) 3 Most OSA sufferers have a restricted upper airway, which is primarily brought on by fat buildup in the parapharyngeal fat pads and pharyngeal muscles, or alterations in the craniofacial structure. Although obesity is the main risk factor for OSA, between 20% and 40% of OSA patients do not have obesity. Non anatomic variables which include lower airway dilator muscle dysfunction, increased chemosensitivity, and low arousal threshold (i.e., early awakening from sleep leading to instability in ventilatory control), are significant in these patients and define diverse phenotypes of OSA.(6) These abnormalities range from clinically obvious anatomical anomalies like micrognathia and retrognathia to subtle radiographic findings like inferior location of the hyoid bone and shorter mandibular and maxillary length, which lead to a small maxilla-mandibular volume. For example, for a given level of OSA, Caucasians tend to be more overweight while Chinese people have more craniofacial bony restriction. The relative contribution of soft tissue and bony abnormalities to OSA varies between individuals and populations. 18 Upper airway collapse is avoided when a person is awakened by the activity of the pharyngeal dilator muscles when there is a small pharyngeal airway. During sleep, the tone of the compensatory and basal dilator muscles decreases, allowing the airway to close.(5).Fluid around the upper airway is another risk factor that has recently attracted attention. When supine in people with lower-extremity edoema, extra fluid may build up in the pharyngeal region and translocate cephalic from the lower extremities to the neck area, making the upper airway vulnerable to collapsing while they sleep. According to the discussion in the text that follows, redistribution of fluid to the lungs may potentially potentiate CSA. OSA and CSA have been demonstrated to be improved by therapeutic methods such diuretics, stockings, and exercise.(6). When the muscles supporting your tongue and soft palate, as well as other soft tissues in your throat, momentarily relax, obstructive sleep apnea develops. Your airway narrows or closes as a result of these muscles relaxing, briefly stopping breathing.(10)

AETIOLOGY

Obesity and excess weight, which are linked to the soft tissue of the mouth and throat, are the most common causes of obstructive sleep apnea in adults. This soft tissue can result in the airway becoming clogged when you're sleeping since your throat and tongue muscles are more relaxed.Body mass index, or BMI, of 25 to 29.9, or obesity, affects more than half of those with obstructive sleep apnea (BMI of 30.0 or above).(8)

When your throat muscles relax too much, they block normal breathing, causing obstructive sleep apnea. The tongue, tonsils, soft palate, and the triangular bit of tissue that hangs from the soft palate (the uvula) are just a few of the structures that are supported by these muscles.

Your airway narrows or closes as you breathe in when the muscles relax, which can make breathing difficult for up to 10 seconds. Your blood's oxygen content may decrease as a result, and carbon dioxide may accumulate.(10) Those who have obstructive sleep apnea make up more than half of the population and are either fat or overweight (BMI of 25 to 29.9) (BMI of 30.0 or above).(11) Your brain notices this breathing difficulty and momentarily wakes you up so you can reopen your airway. Typically, this awakening is so fleeting that you don't recall it.

Breathlessness that quickly resolves itself after just one or two deep breaths can awaken you. You might snort, choke, or gasp.(Figure-3)

This process can repeat itself five to thirty times or more per hour, all night long. You won't be able to experience deep, restorative sleep as a result of these disturbances, and you'll likely experience sleepiness during the day.

People with obstructive sleep apnea might not be aware of their disrupted sleep. Many people who suffer from this kind of sleep apnea are unaware that they haven't had a good night's sleep.(10) Large tonsils or adenoids as well as dental issues like an overbite are common causes of obstructive sleep apnea in youngsters. A tumour or growth in the airway, as well as congenital abnormalities such Down syndrome and Pierre-Robin syndrome, are less frequent causes.(8) Some medications and drugs, especially opiates like hydrocodone or fentanyl, can cause CSA.

<u>SYMPTOMS</u>

- Hypertrophy of the adenotonsilla
- Mandibular malformation
- Macroglossia Tumors of the Upper Airway (rare)

Muscle tone loss

- Alcohol
- Drugs that are sedative.
- Snoring (9)
- Dry moth
- Increase in caries
- TMJ disorders
- Retrognathic mandible (12)

PREVALENCE

Although obstructive sleep apnea is frequently left undiagnosed and untreated, it is estimated that 4–9 percent of middle-aged adults have the condition. Over 65-year-olds are thought to have the syndrome in at least 10% of cases.
A narrowing or collapsing of the airway is more likely as we age because the brain's capacity to keep the muscles of the upper airway and throat rigid during sleep is affected.(8) When characterised as an ongoing obstruction of the upper airway during sleeping, OSA is a relatively widespread condition. The apnea-hypopnea index (AHI), which measures the frequency of full (apneas) or incomplete (hypopneas) obstructive episodes per hour of sleep, is typically used to determine the severity of sleep apnea. In the Wisconsin Sleep Cohort, which defined OSA as an AHI 5 events/hour, the prevalence of OSA was 24% in men and 9% in women aged 30 to 60.(11)

ROLE OF OSA IN OTHER DISEASE

- **Cardiovascular disease**

Numerous research have looked at the function of OSA as a pathogenetic factor in cardiovascular and cerebrovascular disorders as well as the potential preventive effects of CPAP therapy.(12) Intermittent hypoxia, excessive sympathetic nerve activity, systemic hypertension, endothelial cell failure, oxidative stress, inflammation, and accelerated atherosclerosis are just a few of the intermediate pathways by which OSA may raise cardiovascular risk. Contrarily, persistent intermittent hypoxia may also trigger some protective mechanisms, such as the growth of coronary artery collaterals in individuals with ischemic heart disease.(13)

- **Systemic hypertension**

Systemic hypertension, a cardiovascular comorbidity in OSA, has received the most research. Respiratory events during sleep are linked to higher mean nocturnal blood pressure, more variability in blood pressure, and hypertensive peaks at the conclusion of apneas and hypopnea. Blood pressure and OSA severity have been linked in a dose-response manner.(14)In the OSA population, 24-hour blood pressure monitoring is strongly advised since OSA patients may exhibit higher blood pressure values only while sleeping, or while sleeping and being awake. Additionally common in OSA is resistant hypertension, which is inadequate blood pressure management while taking three antihypertensive medications.(15)

- **Arrhythmias**

Patients with OSA frequently experience arrhythmias, particularly atrial fibrillation (AF). The greater likelihood of recurrence of AF in individuals with OSA compared to non-OSA participants and the protective effect of CPAP treatment point to a permissive role for OSA in the arrhythmogenic mechanism of AF.(16,17) Compared to non-OSA patients or patients with chronic heart failure and central apneas, studies in patients with implanted cardioverter-defibrillators (ICD) have shown a significant frequency of nocturnal discharge in OSA.(18)

- **Cerebrovascular disease**

The use of CPAP therapy may lower the risk of stroke, although the majority of studies have used composite cardiovascular outcomes, which include stroke, rather than collecting data for individual event types. The RCTs that are currently available on the effects of CPAP in patients with stroke and OSA are often short-term, and the low acceptance of CPAP treatment in patients with OSA and prior stroke is another challenge to be taken into account. In the latter category, a recent meta-analysis of RCTs showed that CPAP users had improved neurological function(19)

- **Metabolic disease**

The connection between OSA and metabolism is very intricate. One the one hand, obesity, which in and of itself is characterised by impaired energy metabolism and adipose tissue inflammation, is frequently linked to OSA. OSA, on the other hand, may independently contribute to the aetiology of metabolic diseases as nocturnal intermittent hypoxia has been found to influence glucose metabolism.(20,21)

- **Diabetes**

Particularly intriguing from a therapeutic perspective is the bidirectional link between OSA and diabetes.(22) Treatment for OSA may lessen the likelihood of serious diabetic complications. Although meta-analyses have shown that CPAP treatment has no effect on glycemic control, this may still be the case.(23) If diabetic patients with untreated OSA have higher rates of neuropathy, peripheral artery disease, diabetic retinopathy, and diabetic nephropathy(24). There are few studies on how CPAP affects diabetic complications. (30)Optic nerve function improved in severe OSA patients with good CPAP compliance compared to patients with poor compliance(25). A recent post-hoc analysis of SAVE study data revealed that diabetic patients had a higher risk of negative outcomes than non-diabetic patients did(26), and that diabetic patients with OSA who had good CPAP treatment adherence(27)—at least 4 hours per night during the study's first two years—had a protective effect on recurrent cardiovascular events(28,29). In conclusion, OSA may exacerbate metabolic problems, and OSA treatment with enough adherence may provide protection, particularly when combined with lifestyle changes and weight loss. Since CPAP therapy for at least 4 hours per night may be protective, especially when diabetes problems are also present, it is important to routinely screen diabetic patients for OSA.(31,32)

- **Renal disease**

Risk factors for both renal illnesses and OSA include arterial hypertension, diabetes mellitus, obesity, and advanced age(33). Each of these factors may independently contribute to the development and progression of the other. OSA may endanger the kidney through a number of interrelated mechanisms, including endothelial dysfunction, sympathetic hyperactivity, intrarenal renin-angiotensin system hyperactivity, nocturnal intermittent hypoxemia, and recurrent nocturnal blood pressure peaks. A relationship between nocturnal hypoxemia and hyper-activation of the intrarenal renin-angiotensin system has been experimentally demonstrated(34).

- **Asthma**

Obstructive sleep apnea (OSA), a disease that is extremely common and frequently correlated with asthma, also goes by the name of. In addition to daytime tiredness, poor asthma control, and a lower quality of life, OSA symptoms are frequently reported by asthmatic patients.(35) The Wisconsin Sleep Cohort's longitudinal data indicated that having asthma at baseline increased the chance of developing OSA over time. OSA has been linked to a higher frequency of asthma exacerbations, according to sleep studies, which verified that asthmatics are more likely to have it than controls. Of individuals with asthma that was difficult to treat, 49% had mild to moderate OSA.(36,37)

DIAGNOSIS OF OSA

The following criteria are used to make the diagnosis:

1) Physical examination of the chest, abdomen, and head and neck, as well as clinical signs such snoring, sleeping patterns, mouth breathing, etc.(38)

2) Apnea/hypopnea index (AHI) or respiratory disturbance index: The number of breathing pauses per hour can be used to determine the severity of OSA syndrome (RDI). It is most frequently employed(39).

3) The Epworth Sleepiness Scale (ESS) is a valid tool for assessing a patient's likelihood of nodding off. Clinically, it classifies patients into 4 groups according to their perceived levels of daytime sleepiness.(39)

4) The Multiple Sleep Latency Test (MSLT): In this test, the time it takes for someone to fall asleep is assessed with the aid of an ECG. This is done in a dark room during the day on at least four distinct occasions. Sleep latency is the word for this amount of time. (40)

5) The gold standard and most trustworthy method for identifying sleep apnea and other sleep disorders is polysomnography (PSG). The patient is asked to stay overnight in a lab during the study, and his or her sleep is monitored.Airflow, body position, sleep duration and phases, ECG, respiratory effort, movements, oxygen saturation, etc. are only a few of the variables that are monitored throughout the study.(38) figure 16

EPWORTH SLEEPINESS CYCLE figure-4

Tools used in diagnosis of OSA

- Polysomnography
- Oximetry

POLYSOMNOGRAPHY(PSG)

Sleep and breathing patterns are both captured by polysomnography. With the assistance of a technician, PSG is carried out overnight in a sleep centre and commonly includes oxygen saturation, respiratory airflow, thoracoabdominal movement, EEG, electromyogram, and electrooculogram tracings (oximetry).

Before going to bed, polysomnography needs to be set up for 30 to 60 minutes, and it also needs to be detached for 30 minutes in the morning. To conduct and oversee this test, personnel should be on hand for at least 10 hours at night.(41)

OXIMETRY

Due to the low cost and accessibility of recording pulse oximeters, oximetry is employed as the initial OSAHS screening method. These spectrometers are employed in the detection and computation of the differential light absorption caused by the presence of oxygenated and deoxygenated haemoglobin in blood. This is a technique for measuring blood oxygen saturation.(41)

TREATMENT PLANNING

- Behavioural interventions
- Conservative or medical treatment
- CPAP
- Orthodontic appliance
- Surgical procedure

BEHAVIOURAL INTERVENTIONS

Patients should be advised to undergo weight reduction therapy since it improves the symptoms of OSAHS and other related problems because patients with obstructive sleep apnea are fat. For the sake of their general health, smokers should be counselled to quit.

Alcohol, narcotics, and sleeping pills should not be used because they may reduce the function of the airway dilator and exacerbate OSAHS. For people with mild OSA, positional treatment is recommended. In order to lessen symptoms, patients should be encouraged not to sleep on their backs and have their bed's head lifted.(41)

CONSERVATIVE/MEDICAL

Beginning with certain lifestyle modifications is necessary for the treatment of obese people with moderate OSA. Exercise, calorie restriction, alcohol abstinence, quitting smoking, rearranging sleeping positions, and nasal continuous positive airway pressure are a few of these adjustments. Certain medications should not be taken because they may make OSA worse. For example, alcohol depresses the genioglossus' tone and makes the upper airway more prone to collapsing.(43) Then there are opioids, which slow down and deepen breathing, cause tightness in the chest and abdomen, impair the potency of the upper airways, and dull the respiratory response to hypoxia and carbon dioxide. Additionally, additional CNS depressants like benzodiazepines diminish the muscle tone in the upper airways and the ventilatory response to hypoxia, which may increase the AHI and prolong apnea episodes.(42) Atypical antipsychotics, antidepressants, anticonvulsants, etc. are examples of medications that should be avoided since they can lead to weight gain. Additionally, a number of endocrine disorders may manifest as OSA or may worsen OSA. Therefore, it is important to assess whether a patient exhibits clinical signs and symptoms of hypothyroidism, acromegaly, or Cushing syndrome during all first patient evaluations.(42)

The tricyclic antidepressant Protriptyline is the most effective drug studied in the treatment of OSA(43)

In order to reduce the more serious REM-related apneas, protriptyline decreases the proportion of time spent in REM sleep and stimulates the tone of the upper airway muscles.

Another remarkable medication is modafinil, a non-amphetamine CNS stimulant used to improve alertness and decrease daytime drowsiness. However, it's important to note that modafinil cannot replace CPAP or an oral appliance. Additionally, the carbonic anhydrase inhibitor acetazolamide increases respiration by causing metabolic acidosis, which lessens the severity of oxygen desaturations and reduces the number of apneas in OSA patients. Progesterone's respiratory stimulating action is beneficial for some OSA patients, notably those who have obesity-hypoventilation syndrome.(42)

Another remarkable medication is modafinil, a non-amphetamine CNS stimulant used to improve alertness and decrease daytime drowsiness. However, it's important to note that modafinil cannot replace CPAP or an oral appliance.(44) Additionally, the carbonic anhydrase inhibitor Acetazolamide increases respiration by causing metabolic acidosis, which lessens the severity of oxygen desaturations and reduces the number of apneas in OSA patients. Progesterone's respiratory stimulating action is beneficial for some OSA patients, notably those who have obesity-hypoventilation syndrome. According to several recent research, bariatric procedures cause the patient's OSA, as determined by the AHI, to resolve or improve.(45)

CONTINUOUS POSITIVE AIRWAY PRESSURE

The most effective non-surgical treatment for OSA at the moment is CPAP. While the patient is sleeping, a snug-fitting mask is used to provide the nasal CPAP.

The passive collapse of soft tissues while breathing while sleeping is prevented by a CPAP of 7 to 15 cm of water acting as a pneumatic splint of the upper airway. Another proposed mode of action involves stimulating the genioglossus muscle's mechanoreceptors, which results in higher airway tone.(42)Figure 5

Major side effects of CPAP are significant epistaxis, paranasal sinusitis but they are rare (46,47)

additional airway pressure instruments. If a CPAP machine doesn't work for you, you can try an auto-CPAP machine, which regulates the pressure as you sleep. Another choice that offers your body more pressure when you inhale and less pressure when you exhale is BPAP units, which provide bilevel positive airway pressure.(48)figure 6

When using CPAP, flow tracings show occasional periods of missing airflow that significantly improve. It has been shown to be successful in lowering daytime drowsiness levels in OSA patients by decreasing apneas and hypopnea. The first successful use of nasal CPAP to treat sleep apnea was documented in 1981 by Sullivan et al.(42)figure 7

Bi-level positive airway pressure (Bi-PAP) systems, which permit independent regulation of the inspiratory and expiratory pressures, and Auto-CPAP, the newest addition to CPAP systems, have recently been used to treat obstructive sleep apnea more successfully while also improving tolerance and compliance. Notably, Auto-CPAP devices modify the CPAP throughout the night as opposed to maintaining a constant pressure. Additionally, the more recent models offer patients adjustable-size masks for comfort. Although CPAP therapy been proved very-successful, patient’s compliance is low. According to one study compliance rates at 12 months have been reported as low as 54%. On the other hand, disadvantages of CPAP are nasal dryness, congestion, sore throat,dryness of skin, eyes and nose.stephen A. Marsh recently created the Airing tiny CPAP device, which he claims to be the first mask-free, hose-less, cordless micro CPAP device in the world. Although he asserts that this gadget has a ground-breaking design and will function similarly to existing CPAP machines so that patients won't have to struggle with bulky, uncomfortable masks, it must first receive FDA approval and undergo clinical trials before it can be released.(49)figure 8

ORAL APPLIANCE

Another method of treatment is the application of various prosthetic devices. Oral appliances may be utilised in individuals with primary snoring, mild obstructive sleep apnea, or those with moderate to severe obstructive sleep apnea who refuse or are unable to tolerate nasal continuous positive airway pressure, according to the American Sleep Disorders Association. By shifting the lower jaw and stabilising the tongue, these devices aid in maintaining an open airway. Various oral appliances, including a tongue-retaining device, a Klearway appliance, a Herbst appliance, an elastic mandibular advancement, and elastomeric sleep appliance, and an equaliser airway device, are offered in the market.(50,51) Excessive salivation, xerostomia, soft tissue irritations, temporary discomfort of the teeth, and temporomandibular joint are side effects of oral appliance therapy.

A review of OSA (TMJ) treatment, small occlusal alterations, and stiffness or pain in the masticatory muscles was conducted; however, the literature reveals few examples of tooth loosing during long-term appliance therapy.(42)Figure-9,10

SURGICAL TREATMENT

Numerous studies have shown that more than half of patients will not adhere to conservative treatment for an extended period of time or patients would seek surgical modalities if conservative treatments are unable to sufficiently relieve their snoring. Currently, tracheostomy, nose surgery, uvulopalatopharyngoplasty, and a number of orthognathic surgical techniques are employed in the surgical treatment of obstructive sleep apnea.

The first successful tracheostomy for the treatment of obstructive sleep apnea was performed by Kuhlo et al. in 1969. Obstructive sleep apnea symptoms and indications are virtually completely eliminated by it.(52) Having the least acceptance due to difficulties, they should only be utilised as a last resort. Surgery, albeit regarded as a rapid fix for OSA, has its own indications and restrictions.

SURGICAL INDICATIONS

- Oxygen desaturation <90%
- Esophageal pressure (Pes) more negative than -10 cm of H2O
- Apnea – hypopnea index (AHI) ≥ 20 events/hr of sleep
- Cardiovascular derangements (arrhythmia, hypertension)
- Neurobehavioral symptoms [excessive daytime somnolence (EDS)]
- Failure of medical management
- Anatomical sites of obstruction (nose, palate,base of the tounge)

If significant daytime weariness is present, surgery may be suggested even with an AHI of 20. Riley and Powell presented a surgical approach in 1993 for the treatment of obstructive sleep apnea syndrome using dynamic upper airway reconstruction. They presented a two-phase technique in accordance with their analysis of 239 instances.

Uvulopalatopharyngoplasty and/or mandibular resection procedures were part of Phase I, which took a cautious approach.(53)

POWELL–RILEY OR STANFORD PROTOCOL
SURGICAL PROCEDURES

PHASE -1

- Nasal surgery (Septoplasty, turbinate reduction,
- nasal valve grafting)
- Tonsillectomy
- Uvulopalatopharyngoplasty (UPPP) or
- Uvulopalatal flap (UPF)
- Mandibular osteotomy with genioglossus
- advancement
- Hyoid myotomy and suspension
- Temperature-controlled radio-frequency (TCRF)—
- turbinates, palate, tongue base

PHASE-2

- Maxillomandibular advancement osteotomy
- (MMO)
- Temperature-controlled radio frequency (TCRF)—
- tongue base

NASAL SURGERIES

For healthy breathing and rest, a patent nasal airway is necessary. Every obstacle has the potential to raise airway resistance and cause mouth breathing. When the mouth is opened, the jaw rotates posteriorly, allowing the tongue to protrude into the posterior airway space (PAS) and constrict the hypo-pharyngeal airway. Nasal-obstruction can be brought on by larger turbinates, ineffective nasal valves, or septal abnormalities. There are numerous methods (including Septoplasty, alar grafting, and turbinate reduction) for treating nasal blockage (54).

THE PILLAR PROCEDURE (SOFT PALATE IMPLANTS)

The Pillar treatment is a minimally invasive method that can also treat mild to moderate sleep apnea sufferers and chronic snoring. Three to five polyester rods are inserted into the soft palate as part of the procedure.Each implant measures 1.5 mm in diameter and 18 mm in length(Figure-11). The rods cause an inflammatory reaction in the nearby soft tissues, which stiffens the soft palate. As a result, as the muscles relax during deep periods of sleep, the stiffer soft palate is less likely to make contact with the rear wall of the pharynx, decreasing snoring and apnea. Additionally, this surgery can be carried out in the clinic under local anaesthetic.(55)

UVULOPALATOPHARYNGOPLASTY

To open up the posterior airway space, a surgical operation called uvulopalatopharyngoplasty (UPPP) is performed to remove extra tissue from the upper airway. Shortening of the uvula, removal of certain tonsils, adenoid, and pharyngeal tissue are the main surgical goals of this procedure.(42) There are three different types of UPPP: conventional, laser-assisted, and temperature-controlled radio-frequency (TCRF). Changes in voice pattern and GERD worsening are long-term negative effects of UPPP.(57,58)
Figure 12

TONGUE PROCEDURE

As the base of the tongue is one of the most frequent obstruction locations in OSA, reducing the tongue's mass—especially its base—has been proven to be an effective way to manage the condition.(42) Uvulopalatopharyngoglossoplasty (UPPGP), which includes modified Uvulopalatopharyngoplasty (UPPP) with minimal resection of the tongue base, was described by Djuperlandet al. (1992) and Midjejeig (1992).(59)figure 13

In a different investigation, Fugita et al used the carbon dioxide laser to conduct midline glossectomy on 12 patients in order to increase the hypo-pharyngeal airways, and they saw encouraging outcomes.(60) While in a short trial with only 10 patients, Chabolle et al. (1999) combined tongue base reduction with hyoepiglossoplasty and observed a significant improvement. However, the intra-operative consequences that could arise during such treatments are those of any oral and pharyngeal surgical intervention, namely haemorrhage and airway blockage. Additionally, surgical complications such as tongue numbness, temporary alterations in taste, dysphagia, and infection are possible.(61,62)

MANDIBULAR OSTEOTOMY WITH GENIOGLOSSUS ADVANCEMENT

Patients with verified hypo-pharyngeal blockage should consider genioglossus advancement (Fujita III). It can be applied alone or in conjunction with other surgical techniques.(42)

HYOID MYOTOMY AND SUSPENSION

By moving the hyoid complex in an anterior direction, hyoid myotomy and suspension are intended to relieve hypopharyngeal blockage. Initially, the hyoid was suspended from the mandible during this procedure via fascia lata. To harvest the fascia lata and other tissues, more incisions and dissection were necessary.

However, in order to harvest the fascia lata and reveal the mandible, more incisions and dissection were necessary. The method has been adjusted to suspend the hyoid bone to the superior border of the thyroid cartilage in order to minimise the scope of surgery.(42)

MAXILLOMANDIBULAR ADVANCEMENTS

The most current and effective surgical approach for treating obstructive sleep apnea is combined advancement of the maxilla and mandible with or without hyoid suspension, and it is regarded as Phase II of the Powell Riley protocol. The success rate for skeletal surgery in treating obstructive sleep disorder is 65-100 percent, according to Kuo et al., Bear and Priest, and Bear. By enlarging the skeletal face framework, MMO increases the anteroposterior and lateral dimensions of the hypo-pharyngeal and pharyngeal airways.(63-66).figure 14

It is the only procedure in the protocol that really expands the mouth cavity to give the tongue more room. In order to prevent their posterior collapse, it also applies additional pressure to the velopharyngeal and supra-hyoid muscles. Complications include intraoperative haemorrhage, airway obstruction or post-operative TMJ dysfunction, and injury to the inferior alveolar neurovascular bundle are drawbacks.(67,68) figure 15

RECOMMENDATIONS

- OSA management calls for comprehensive, multidisciplinary care. The patient should receive education first. Patients need to be made aware of the dangers of untreated OSA, particularly daytime sleepiness and auto accidents.
- Most OSA patients should make lifestyle and behavioural changes, which include losing weight, getting more physical activity, avoiding alcohol, and abstaining from certain drugs.
- For mild to moderate OSA, we advise starting with CPAP. However, an oral appliance can be a viable choice as first-line therapy for a patient who anticipates issues with CPAP adherence

.

- For patients for whom CPAP or an oral appliance is either denied, not an option, or unsuccessful, surgical therapy is an alternative. However, surgical intervention can be the primary line of treatment for patients whose OSA is brought on by an obstructive lesion that can be removed through surgery.

CONCLUSION

Studies on OSA have noticed its prevalence over the past few decades and revealed its different risk factors, accompanying symptoms, successful diagnostic techniques, and efficient treatment options. More clinical research and trials on OSA are advised in order to better understand the nature of its impacts due to the condition's numerous harmful side effects. In order to aid in the early identification and treatment of OSA, it is also crucial to improve awareness of this illness among community members and its acknowledgement in the health sectors.

References

1. Obstructive sleep apnoea syndrome and its management
Lucia Spicuzza, Daniela Caruso and Giuseppe Di Maria. DOI: 10.1177/ 2040622315590318
2. Br. J. Anaesth. (1989), 63, 477-488 OBSTRUCTIVE SLEEP APNOEA,
C. D. HANNING
3. Alami, Sana et al. "Orthodontic Considerations in Obstructive Sleep Apnea— State of the Art". A Textbook of Advanced Oral and Maxillofacial Surgery Volume 2, edited by Mohammad Motamedi, IntechOpen, 2015. 10.5772/59086.
4. Diagnosis and Management of Obstructive Sleep Apnea
A Review Daniel J. Gottlieb, MD, MPH; Naresh M. Punjabi, MD, PhD
5. Sleep Apnea Types, Mechanisms, and Clinical Cardiovascular Consequences
VOL. 69, NO. 7, 2017 ISSN 0735-1097
6. Role of Orthodontist in Obstructive Sleep Apnea - An Orthodontic Review
Luv Agarwal and Ankit Gupta Published Date: 2016-08-25 DOI10.21767/2469-2980.100027

7. Causes of Sleep Apnea By Rick Ansorge Medically Reviewed by Carol DerSarkissian, MD on May 12, 2021
8. Obstructive sleep apnea in orthodontics: An overview DOI: 10.4103/2349-5243.192536
9. https://www.mayoclinic.org/diseases-conditions/obstructive-sleep-apnea/symptoms-causes/syc-20352090#:~:text=Obstructive%20sleep%20apnea%20occurs%20when%20the%20muscles%20in%20the%20back,the%20tonsils%20and%20the%20tongue
10. Epidemiological aspects of obstructive sleep apnea doi:10.3978/j.issn.2072-1439.2015.04.52
11. Obstructive sleep apnea in orthodontics: An overview DOI:10.4103/2349-5243.192536
12. Does OSA Upregulate Cardioprotective Pathways to an Ischemic Insult DO1: https://doi.org/10.1016/j.chest.2017.07.036
13. Oxidative stress in obstructive sleep apnea and intermittent hypoxia – Revisited – The bad ugly and good: Implications to the heart and brain https://doi.org/10.1016/j.smrv.2014.07.003
14. https://doi.org/10.2147/NSS.S148543 Blood-pressure variability in patients with obstructive sleep apnea: current perspectives
15. The Impact of Continuous Positive Airway Pressure on Blood Pressure in Patients With Obstructive Sleep Apnea Syndrome Evidence From a Meta-analysis of Placebo-Controlled Randomized Trials doi:10.1001/archinte.167.8.757
16. Treating obstructive sleep apnea with continuous positive airway pressure reduces risk of recurrent atrial fibrillation after catheter ablation: a meta-analysis https://doi.org/10.1016/j.sleep.2018.02.013
17. Effect of obstructive sleep apnea and its treatment of atrial fibrillation recurrence after radio frequency catheter ablation: A meta-analysis https://doi.org/10.1111/jebm.12313
18. Obstructive Sleep Apnea and the Risk of Sudden Cardiac Death: A Longitudinal Study of 10,701 Adults https://doi.org/10.1016/j.jacc.2013.04.080
19. Disrupted day–night pattern of cardiovascular death in obstructive sleep apnea https://doi.org/10.1016/j.sleep.2017.06.027
20. Obstructive Sleep Apnea and Diabetes
A State of the Art Review Sirimon Reutrakul, MD Babak Mokhlesi, MD
Published:May 17, 2017DOI:https://doi.org/10.1016/j.chest.2017.05.009
21. Zhu, B., Ma, C., Chaiard, J. et al. Effect of continuous positive airway pressure on glucose metabolism in adults with type 2 diabetes: a systematic review and meta-analysis of randomized controlled trials. Sleep Breath 22, 287–295 (2018). https://doi.org/10.1007/s11325-017-1554-x
22. CPAP in patients with obstructive sleep apnea and type 2 diabetes mellitus: Systematic review and meta-analysis Gonzalo Labarca, Tomas Reyes, Jorge Jorquera, Jorge Dreyse, Lauren Drake First published: 02 August 2018 https://doi.org/10.1111/crj.12915
23. Obstructive Sleep Apnea and Diabetic Neuropathy A Novel Association in Patients with Type 2 Diabetes Abd A. Tahrani 1,2, Asad Ali 3,4, Neil T. Raymond 5, Safia Begum 3, Kiran https://doi.org/10.1164/rccm.201112-2135OC
24. Association of sleep-disordered breathing with severe chronic vascular disease in patients with type 2 diabetes https://doi.org/10.1016/j.sleep.2018.05.001
25. Obstructive Sleep Apnea and Retinopathy in Patients with Type 2 Diabetes. A Longitudinal Studyhttps://doi.org/10.1164/rccm.201701-0175OC
26. Hypoxemia and Glycemic Control in Type 2 Diabetes Mellitus With Extreme Obesity https://doi.org/10.1210/jc.2014-1260
27. Leong WB, Jadhakhan F, Taheri S, Thomas GN, Adab P. The Association between Obstructive Sleep Apnea on Diabetic Kidney Disease: A Systematic Review and Meta-Analysis. Sleep. 2016;39:301-8. https://doi.org/10.5665/ sleep.5432.
28. Nishimura A, Kasai T, Kikuno S, Nagasawa K, Okubo M, Narui K, Mori Y. Effect of sleep-disordered breathing on albuminuria in 273 patients with type 2 diabetes. J Clin Sleep Med. 2018;14:401-7. doi.org/10.5664/jcsm.6986.
29. Liguori C, Placidi F, Palmieri MG, Izzi F, Ludovisi R, Mercuri NB, Pierantozzi M. Continuous positive airway pressure treatment may improve optic nerve function in obstructive sleep apnea: an electrophysiological study. J Clin Sleep Med. 2018;14:953-8. https://doi.org/ 10.5664/jcsm.7158.
30. Quan W, Zheng D, Douglas McEvoy R, Barbe F, Chen R, Liu Z, Loffler K, Lorenzi-Filho G, Luo Y, Mukherjee S, Tripathi M, Woodman R, Li Q, Wang X, Arima H, Xiao Y, Zhang X, Anderson CS; SAVE Investigators. High Risk Characteristics for Recurrent Cardiovascular Events among Patients with Obstructive Sleep Apnoea in the SAVE Study. EClinicalMedicine. 2018 Sep 21;2-3:59-65. doi: 10.1016/j.eclinm.2018.09.002. PMID: 31193555; PMCID: PMC6537527.
31. Borel A-L, Tamisier R, Böhme P, Priou P, Avignon A, Benhamou P-Y, Hanaire H, Pépin J-L, Kessler L, Valensi P, Darmon P, Gagnadoux F. Obstructive sleep apnea syndrome in patients living with diabetes: Which patients should be screened? Diabetes Metab. 2018. pil:S1262-3636(18)30163-0. doi.org/ 10.1016/j.diabet.2018.08.006
32. Abuyassin B, Sharma K, Ayas NT, Laher. Obstructive Sleep Apnea and Kidney Disease: A Potential Bidirectional Relationship? J Clin Sleep Med. 2015;11:915-24. https://doi.org/10.5664/icsm.4946.
33. Zalucky AA, Nicholl DD, Hanly PJ, Poulin MJ, Turin TC, Walji S, Handley GB, Raneri JK, Sola DY, Ahmed SB.Nocturnal hypoxemia severity andrenin-angiotensin system activity in obstructive sleep apnea. Am J Respir Crit Care Med. 2015;192:873-80. https://doi.org/10.1164/ rccm.201502-03830C.
34. Teodorescu M, Barnet JH, Hagen EW, Palta M, Young TB, Peppard PE. Association between asthma and risk of developing obstructive sleep apnea. JAMA2015;313:156-64. https://doi.org/10.1001/jama.2014.17822.

35. Taillé C, Rouvel-Tallec A, Stoica M,Danel C, Dehoux M, Marin-Esteban V,

Pretolani M, Aubier M, d'Ortho MP.Obstructive sleep apnea modulates
airway inflammation and remodelling in severe asthma. PLoS One.
2016;11:0150042. https://doi.org/10.1371/journal.pone.0150042.
36. Julien JY, Martin JG, Ernst P, Olivenstein R, Hamid Q, Lemiere C, Pepe C, Naor N, Olha A, Kimoff RJ. Prevalence of obstructive sleep apnea-hypopnea in
severe versus moderate asthma. Allergy Clin Immunol. 2009;124:371-6.
https://doi.org/10.1016/jjaci.2009.05.016.
37. Obstructive sleep apnea in orthodontics: An overview
Rohit Kulshrestha^, Ragni Tandon2, Saniya Kinger1, Abhimanyu Rohmetra?
Raahat Vikram DO: 10.4103/2349-5243.192536
38. Role of Orthodontist in Obstructive Sleep Apnea - An Orthodontic Review
DOI10.21767/2469-2980.100027
39. Obstructive Sleep Apnea: An Overview on Features, Diagnosis & Orthodontic Management Dr. Vini Akanksha Jai1, Dr. Vaibhav Khare2, Dr. CH. Sudheer Kumar3, Dr. Renuka B1, Dr. AtulVasan1 E-ISSN: 2349-9788; P-ISSN: 2454-2237
40. Role of Orthodontist in Obstructive Sleep Apnea - An Orthodontic Review
Luv Agarwal and Ankit Gupta DOI: 10.4172/2469-2980.100027
41. Simou E, Britton J, Leonardi-Bee J. Alcohol and the risk of sleep apnoea: a systematic review and meta-analysis. Sleep Med. 2018 Feb;42:38-46. doi: 10.1016/j.sleep.2017.12.005. Epub 2018 Jan 3. PMID: 29458744; PMCID: PMC5840512.
42. Schwartz JR, Hirshkowitz M, Erman MK, Schmidt-Nowara W. Modafinil as adjunct therapy for daytime sleepiness in obstructive sleep apnea: a 12-week, open-label study. Chest. 2003 Dec;124(6):2192-9. doi: 10.1378/chest.124.6.2192. PMID: 14665500.
43. Valentino RM, Foldvary-Schaefer N. Modafinil in the treatment of excessive daytime sleepiness. Cleve Clin J Med. 2007 Aug;74(8):561-6, 568-71. doi: 10.3949/ccjm.74.8.561. PMID: 17708127.
44. Weaver TE, Maislin G, Dinges DF, Bloxham T, George CF, Greenberg H, Kader G, Mahowald M, Younger J, Pack AI. Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. Sleep. 2007 Jun;30(6):711-9. doi: 10.1093/sleep/30.6.711. PMID: 17580592; PMCID: PMC1978355.
45. Patel SR, White DP, Malhotra A, Stanchina ML, Ayas NT. Continuous positive airway pressure therapy for treating sleepiness in a diverse population with obstructive sleep apnea: results of a meta-analysis. Arch Intern Med. 2003 Mar 10;163(5):565-71. doi: 10.1001/archinte.163.5.565. PMID: 12622603.

46. Sleep Apnea By Alexandra Benisek
Medically Reviewed by Neha Pathak, MD on September 07, 2021
https://www.webmd.com/sleep-disorders/sleep-apnea/sleep-apnea
47. Airing: the first hoseless, maskless, micro-CPAP https://www.indiegogo.com.
48. Lowe A, Fleetham J, Ryan F, Mathews B. Effects of a mandibular repositioning appliance used in the treatment of obstructive sleep apnea on tongue muscle activity. Prog Clin Biol Res. 1990;345:395-404; discussion 405. PMID: 2377643.
49. Shadaba A, Battagel JM, Owa A, Croft CB, Kotecha BT. Evaluation of the Herbst Mandibular Advancement Splint in the management of patients with sleep-related breathing disorders. Clin Otolaryngol Allied Sci. 2000 Oct;25(5):404-12. doi: 10.1046/j.1365-2273.2000.00411.x. PMID: 11012655.
50. Friedman M, Ibrahim H, Joseph NJ. Staging of obstructive sleep apnea/hypopnea syndrome: a guide to appropriate treatment. Laryngoscope. 2004 Mar;114(3):454-9. doi: 10.1097/00005537-200403000-00013. PMID: 15091218.
51. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea syndrome: a surgical protocol for dynamic upper airway reconstruction. J Oral Maxillofac Surg. 1993 Jul;51(7):742-7; discussion 748-9. doi: 10.1016/s0278-2391(10)80412-4. PMID: 8509912.
52. Ishii L, Roxbury C, Godoy A, Ishman S, Ishii M. Does Nasal Surgery Improve OSA in Patients with Nasal Obstruction and OSA? A Meta-analysis. Otolaryngol Head Neck Surg. 2015 Sep;153(3):326-33. doi: 10.1177/0194599815594374. Epub 2015 Jul 16. Erratum in: Otolaryngol Head Neck Surg. 2015 Sep;153(3):478. PMID: 26183522.
53. Friedman M, Vidyasagar R, Bliznikas D, Joseph NJ. Patient selection and efficacy of pillar implant technique for treatment of snoring and obstructive sleep apnea/hypopnea syndrome. Otolaryngol Head Neck Surg. 2006 Feb;134(2):187-96. doi: 10.1016/j.otohns.2005.10.032. PMID: 16455363.
54. Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic azbnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg. 1981 Nov-Dec;89(6):923-34. doi: 10.1177/019459988108900609. PMID: 6801592.
55. Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic azbnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg. 1981 Nov-Dec;89(6):923-34. doi: 10.1177/019459988108900609. PMID: 6801592.
56. Djupesland G, Schrader H, Lyberg T, Refsum H, Lilleås F, Godtlibsen OB. Palatopharyngoglossoplasty in the treatment of patients with obstructive sleep apnea syndrome. Acta Otolaryngol Suppl. 1992;492:50-4. doi: 10.3109/00016489209136809. PMID: 1632251.
57. Djupesland G, Schrader H, Lyberg T, Refsum H, Lilleås F, Godtlibsen OB. Palatopharyngoglossoplasty in the treatment of patients with obstructive sleep apnea syndrome. Acta Otolaryngol Suppl. 1992;492:50-4. doi: 10.3109/00016489209136809. PMID: 1632251.
58. Fujita S, Woodson BT, Clark JL, Wittig R. Laser midline glossectomy as a treatment for obstructive sleep apnea. Laryngoscope. 1991 Aug;101(8):805-9. doi: 10.1288/00005537-199108000-00001. PMID: 1865726.
59. Mickelson SA, Rosenthal L. Midline glossectomy and epiglottidectomy for obstructive sleep apnea syndrome. Laryngoscope. 1997 May;107(5):614-9. doi: 10.1097/00005537-199705000-00011. PMID: 9149162.
60. Woodson BT, Fujita S. Clinical experience with lingualplasty as part of the treatment of severe obstructive sleep apnea. Otolaryngol Head Neck Surg. 1992 Jul;107(1):40-8. doi: 10.1177/019459989210700107. PMID: 1528601.
61. Maxillofacial Surgery and Obstructive Sleep Apnea Syndrome. https://doi.org/10.1016/S0030-6665(20)31254-8

62. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea syndrome: a review of 306 consecutively treated surgical patients. Otolaryngol Head Neck Surg. 1993 Feb;108(2):117-25. doi: 10.1177/019459989310800203. PMID: 8441535.

63. Prinsell JR. Maxillomandibular advancement surgery in a site-specific treatment approach for obstructive sleep apnea in 50 consecutive patients. Chest. 1999 Dec;116(6):1519-29. doi: 10.1378/chest.116.6.1519. PMID: 10593771.

64. Li KK, Riley RW, Powell NB, Guilleminault C. Maxillomandibular advancement for persistent obstructive sleep apnea after phase I surgery in patients without maxillomandibular deficiency. Laryngoscope. 2000 Oct;110(10 Pt 1):1684-8. doi: 10.1097/00005537-200010000-00021. PMID: 11037825.

65. Efficacy and safety of maxillomandibular advancement in treatment of obstructive sleep apnoea syndrome PMCID: PMC3631807 PMID: 23620639

66. Ronchi P, Novelli G, Colombo L, Valsecchi S, Oldani A, Zucconi M, Paddeu A. Effectiveness of maxillo-mandibular advancement in obstructive sleep apnea patients with and without skeletal anomalies. Int J Oral Maxillofac Surg. 2010 Jun;39(6):541-7. doi: 10.1016/j.ijom.2010.03.006. PMID: 20434311.

Figure-1(joe the fat boy)

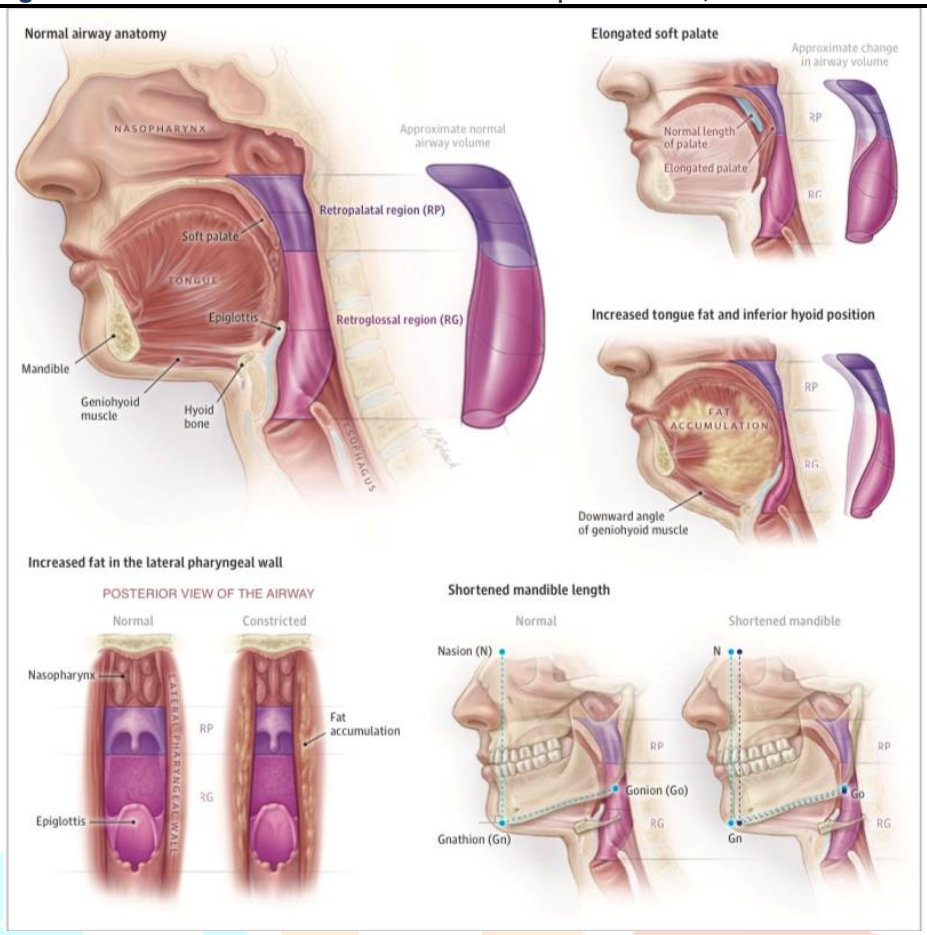


Figure-2(normal and elongated airway anatomy

1	Normal range	ESS <11
2	Mild	ESS =11
3	Moderate	ESS =16
4	Severe	ESS >18

Figure- 3(snoring daigram)

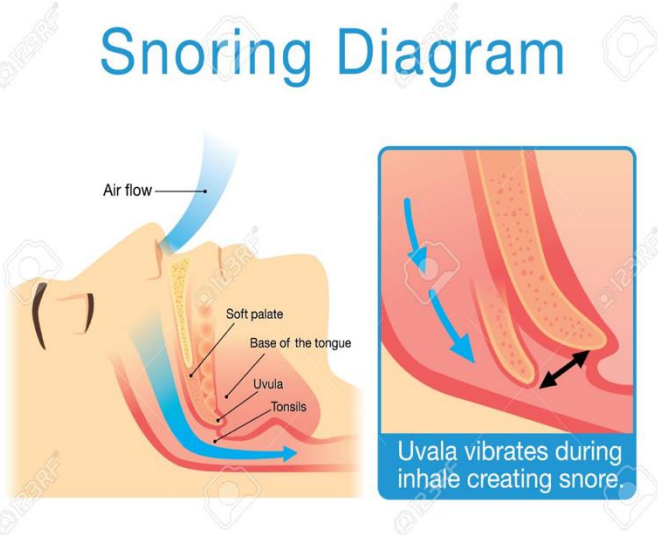
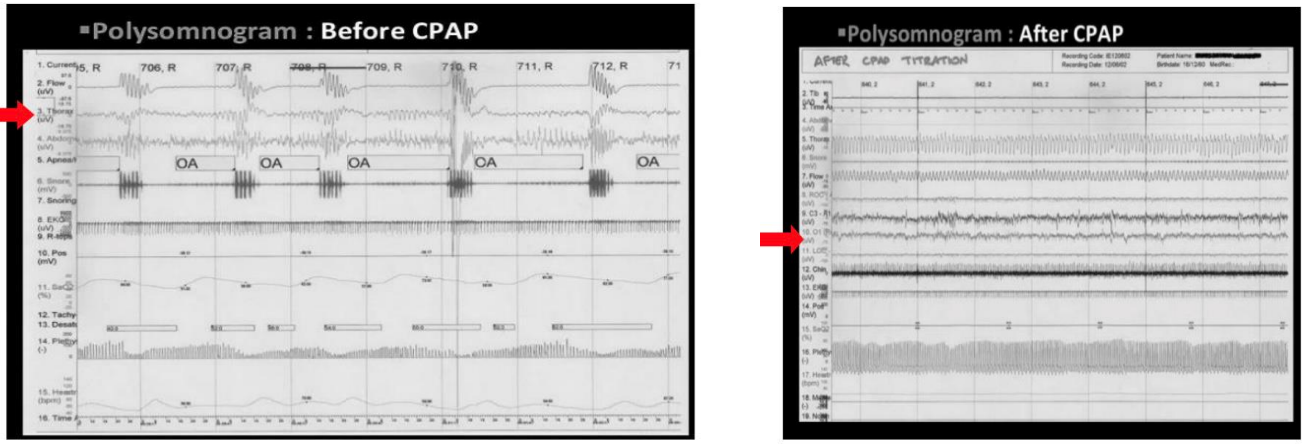


Figure 4(Epworth Sleepiness cycle)



Figue 5 (cpap- continues positive airway pressure)
Figure 6 CPAP Machine

Figure 7,8(polysomnogram- Before and after)

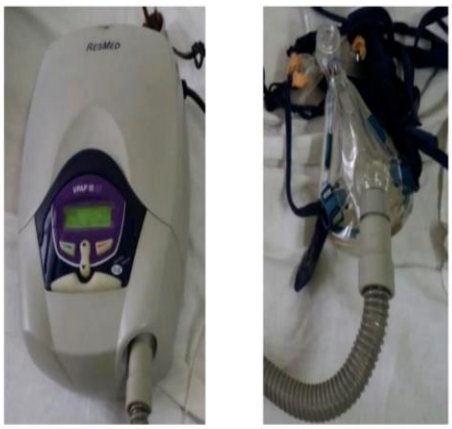
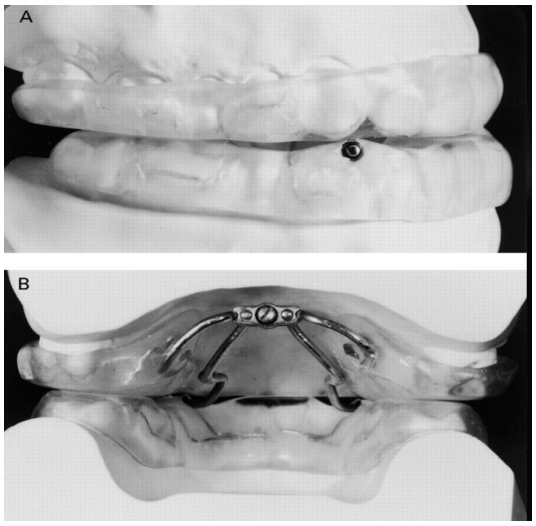
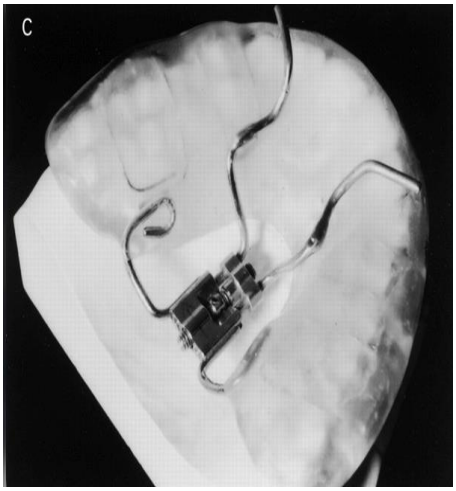
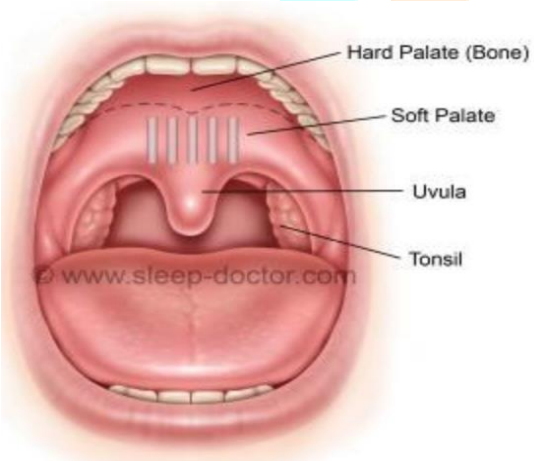


Figure 9, 10(oral applaince)







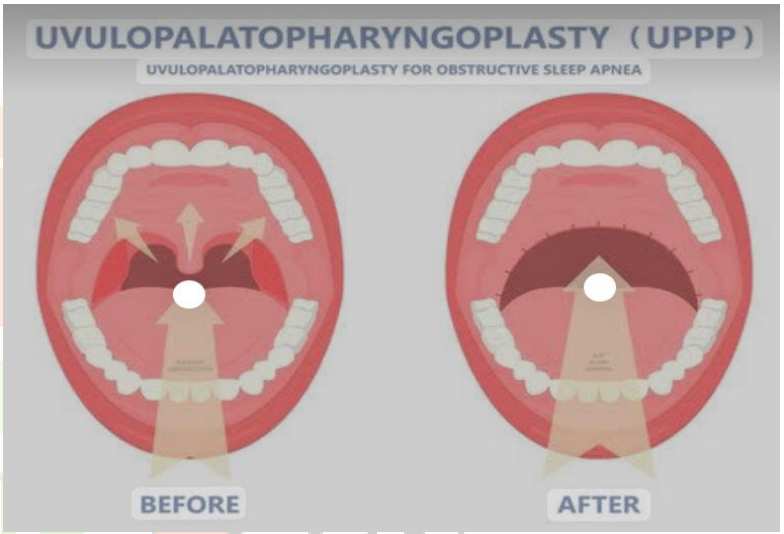


Figure 11(pillar procedure)

Figure 12 UPPP

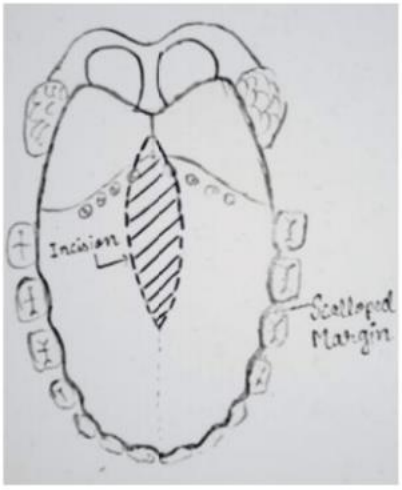


Figure 13 tongue procedure

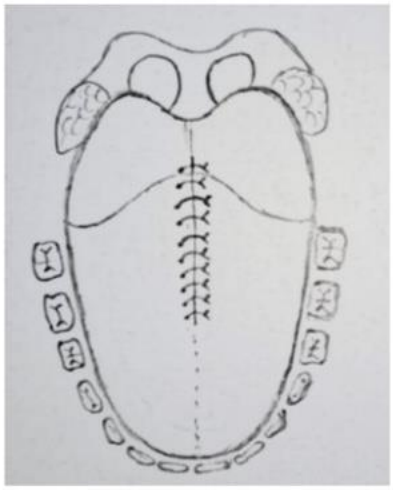
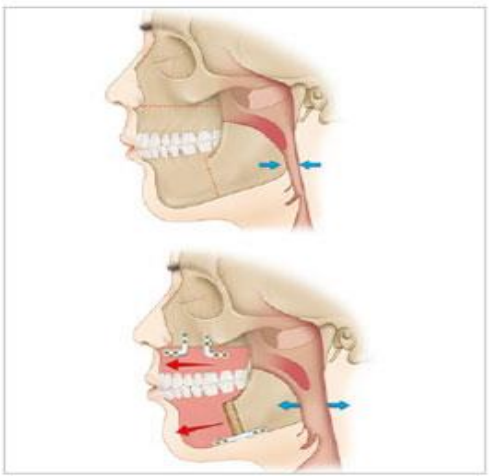


Figure 14 maxillomandibular advancement



In maxillomandibular advancement surgery, the upper and lower jaws are surgically advanced to open the airway.

Figure 15(surgical procedure of maxillomandibular advancement)

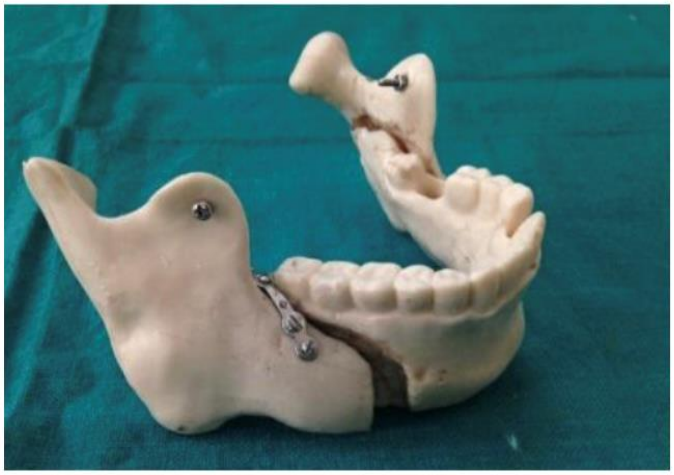


Figure 16 Diagnosis chart