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Obstructive Sleep Apnea: A Review of Literature

SanchitaThapliyal[1], Vishal Bansal, Apoorva Mowar

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I. Introduction

Obstructive sleep apnea (OSA) is defined as the presence of at least five obstructive events (apneas and hypopneas) per hour during sleep.1 It is characterized by periodic complete or partial upper airway obstruction during sleep, causing intermittent cessations of breathing (apneas) or reductions in airflow (hypopneas) despite ongoing respiratory effort. The obstructive sleep apnea–hypopnea syndrome, defined as the presence of at least five obstructive events per hour with associated daytime sleepiness.1 There is collapse of the pharyngeal airway resulting in repeated episodes of airflow cessation, oxygen desaturation, and sleep disruption.2 Apnea is cessation of airflow for at least 10 seconds whereas hypopnea is partial loss of breath (more than or equal to 4% desaturation) that last 10 seconds or longer.3 Normal sleep has breakdown fewer than 5 events/hr, mild sleep apnea has 5-14 events/hr, moderate has 15-29 events/hr, severe apnea has 30 or more events/hr.

An apneais considered obstructive if there is continued respiratory effort despite cessation of airflow. The various indices related to this disease are Apnea hypopnea index (AHI) that is averaged frequency of apnea and hypopnea events per hour of sleep, Respiratory distress index (RDI) is averaged frequency of apnea, hypopnea, Apnea hypopnea index(AHI) expressed as the number of apneas and hypopneas per hour of sleep, respiratory effort related arousal (RERA) per hour of sleep, obtained using polysomnography where RERA is reduction in airflow with resultant arousal but not meeting desaturation criteria for hypopnea.3,4 OSA is diagnosed by polysomnography, an overnight sleep study that requires entire night of recording by trained personal using sophisticated equipment.5 The concept described by Burstein and colleagues includes four airway zones- Zone 1 :extends from the naris to the velum, Zone 2 :extends from the lips to the hypopharynx but excludes all laryngeal structures, Zone 3 :extends from the epiglottis to the trachea and includes the larynx, and Zone 4 :extends from the subglottic region to the bronchi. The zone concept allows accurate evaluation and treatment at the appropriate level but in a systematic manner.6 The common symptoms are divided into nocturnal and daytime which are snoring, choking, dyspnea, diaphoresis, dry mouth, drooling and daytime sleepiness & fatigue, impaired memory & concentration, decreased dexterity, depression, anxiety respectively. There are some anatomical findings predictive of OSA like obesity, large neck circumference, retrognathia, overjet, macroglossia, elongated & low hanging soft palate etc.5 The clinical signs are due to sleep fragmentation and hypoxemia caused by the airway collapse during sleep.4 OSA affects almost every system resulting in an increased incidence of systemic diseases like hypertension, congestive heart failure, stroke, and cardiac arrhythmias.5 There is also increased risk of having an accident, presumably as a result of associated excessive daytime sleepiness.The Epworth Sleepiness Scale (ESS) is a subjective score used for knowing patient's tendency to fall asleep during specific non stimulating situations.7 The gold standard for the diagnosis of sleep apnea is an in-laboratory polysomnogram.9 The aim of the treatment is establishing a nocturnal normal oxygen and ventilation, elimination of snoring and disruption of sleep due to upper airway closure.10 OSAS in children and adults has been treated both medically and surgically.6 Surgical procedures are: Zone 1 - Nasal nasopharynx: Adenoidectomy, Septoplasty, Inferior turbinectomy, Maxillary advancement. Zone 2 - Oropharynx and Hypopharynx: Tonsillectomy, UPPP, Central tongue reduction, Mandibular advancement, Genioglossal advancement, Hyoid tongue suspension.6

II. Discussion

Sleep apneais defined as repeated episodes of obstructive apnea and hypopnea during sleep, together with dynamic sleepiness or altered cardiopulmonary function.9 Obstructive sleep apnea (OSA) is a disease characterized by collapse of the pharyngeal airway resulting in repeated episodes of airflow cessation, oxygen desaturation, and sleep disruption.2 Obstruction of the upper airway during sleep occurs due to inadequate motor tone of the tongue and/or airway dilator muscles as during natural sleep, upper airway muscle tone is reduced (Figure 1).3,4

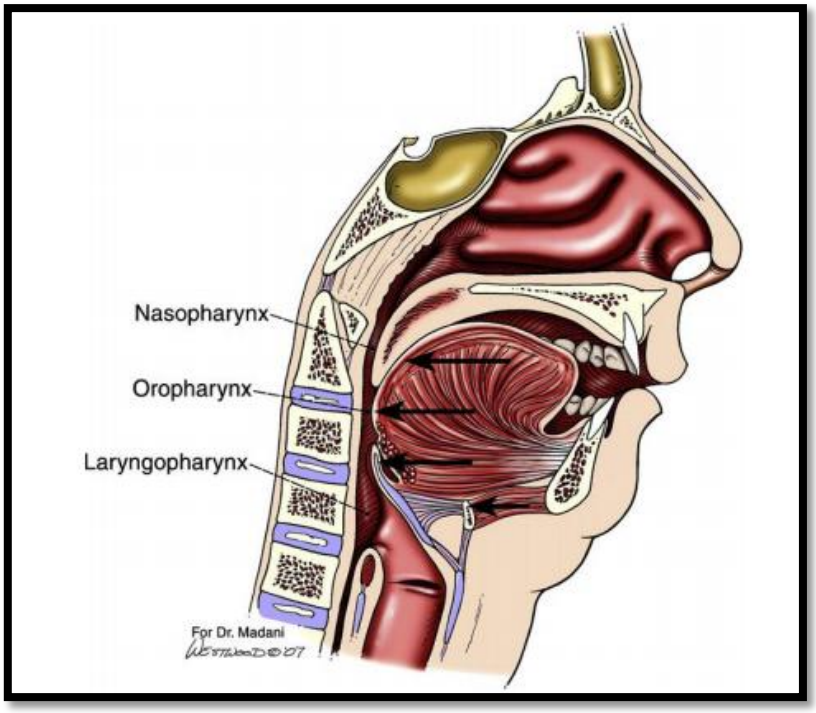

Figure. 1: Areas of Obstruction in Sleep Apnea

The apnea and hypopnea events of sleep-disordered breathing (SDB) have substantial negative consequences on health of a person.30 The effect range from immediate to long term, where intermittent hypoxia, sleep fragmentation, and exaggerated fluctuations in heart rhythm, blood pressure are intermediate effects and intrathoracic pressure effects including raised blood pressure, cardiovascular morbidities, cognitive impairment, mood and quality of life and premature death are long term effects.30 The risk factors of sleep disordered breathing are due to changes in upper airway structure affected by both hard and soft tissues by environmental and underlying medical conditions like allergic rhinitis, immunologic disturbances, recurrent infection of pharynx which can resultant in tonsil enlargement, besides other clinical risk factors.1,4 Clinical risk factors of obstructive sleep apnea are nasal obstruction, craniofacial abnormalities, mandibularretrognathia, micrognathia, narrowed, tapered and short maxillary arch, long soft palate, modified mallampati grade III or IV, enlarged tongue, hypertrophy of tonsils and with increased risk when neck circumference is greater than 17 inch formen and 16 inch for women (Table 1).23 The soft tissues mediating airway size are the tonsils, soft palate, uvula, tongue, and the lateral pharyngeal walls. The major craniofacial bony structures that determine airway size are the mandible and position of the hyoid bone.2

Table 1: Risk Factors of OSA

Clinical Risk Factors For Obstructive Sleep Apnea
Nasal obstruction
Macroglossia
Tonsillar hypertrophy
Mandibular retrognathia

DIAGNOSIS:

History and physical examination findings that should raise suspicion for obstructive sleep apnea3 - A) History: This involves taking thorough history about disruptive snoring, witnessed apnea or snorts, gasping/choking while sleeping, difficulty with concentration or short-term memory loss, excessive nocturia, difficulty with sleep maintenance, restless/unrefreshing sleep, loss of libido, morning headaches. The symptoms associated with OSA can be divided into night time and daytime symptoms. Night time symptoms includesonorous snoring, sweating, noisy breathing, mouth breathing, gasping, restless sleep. Daytime symptoms include irritability, excessive daytime sleepiness (EDS) behavioural problems and cognitive impairment (Table-2).5

Table-2: Symptoms And Signs Of Obstructive Sleep Apnea

Diurnal symptoms of OSA	Nocturnal symptoms of OSA
Daytime sleepiness; memory and concentration dysfunction; gastroesophageal reflux; behavioural imitability (imitability, depression, chronic fatigue, delerium); road traffic accident Signs associated with OSA Edamatus soft palate of uvula Long soft palate and uvula Decreased oropharyngeal dimensions Nasal obstruction Maxillary hypoplasia Retrognathia Central adiposity and increased neck circumference Hypertension and other cardiovascular consequences	Heavy persistent snoring, worse in supine position of after alcohol or sedatives; apnea with limb movement, eithnessed by bed partner; sudden awakening with noisy breathing; accidents related to sleepiness; nocturnal sweating; wake up with dry mouth; nocturnal epilepsy; nocturia

B) Physical examination: This follows elaborated history and involves examination of both soft and hard tissues. I. Soft tissue examination includes looking for enlarged tonsils, high-arched hard palate, nasal deformities/septal deviation. Enlargement of the palatine tonsils can lead to airway obstruction by decreasing airway calibre. In addition, tonsil and adenoid hypertrophy is the most common risk factor for sleepdisordered breathing in children and can be pathogenic in adults with OSA. It has been documented that obesity is a major risk factor for OSA, although not all obese individuals suffer from OSA and not all individuals with sleep apnea are obese. Obesity can result in deposition of fat within the parapharyngeal space and may lead to enlargement of the surrounding soft tissue structures. The various factors leading tomacroglossia could be hypothyroidism, amyloidosis, acromegaly that increases chances of OSA in an individual.2 Short stature and poor weight gain have also been attributed to OSAS in children, since slow wave sleep and growth hormone release are intimately related.6

III. Hard tissue

Bony structures affecting the size of the airway are mandible and position of the hyoid bone. A retrognathic mandible or micrognathia, adversely affect the airway size, and may predispose or lead to OSA. C) Comorbid conditions: OSA is associated with hypertension, recurrent atrial fibrillation, stroke, myocardial infarction and also pulmonary hypertension, chronic heart failure, although studies have indicated reduction in blood pressure in OSA patients when CPAP therapy is given.3,28 Recent meta-analysis, reports association between stroke (fatal or non-fatal), cognitive impairments and OSA.32,35 The long-term sequelae of OSAreported are due to potentially serious physiologic consequences leading to cognitive impairment, cardiovascular morbidity, hypertension, effect on quality of life and premature death.30,37 The clinical evaluations mentioned above includes examining both soft and hard tissues in an individual. Further approach is examination of chest and cervical spine with radiographs and tests to rule out gastroesophageal reflux. Investigations like 12 lead electrocardiograms (ECGs), 16 channelpolysomnogram, cardiac ultrasonography, upper airway endoscopy done during spontaneous ventilation, computed axial tomography (CT scan of the head and neck), and lateral cephalometric radiograph evaluation are routine.6 Diagnosis requires entire night of recording of polysomnography (PSG). PSG includes recordings of sleep stages and time, airflow and respiratory effort, cardiac rhythm, limb movements and oximetry.3 PSG requires overnight hospitalization and skilled personnel to perform it.5 Overnight oximetry is also used but is not sensitive and specific enough for the diagnosis of OSA, although it can be used for screening or follow up of CPAP therapy.3 Some questionnaire were proposed and used for initial diagnosis of OSA like Berlin, Epworth sleepiness scale (ESS) (Table-3).3,7

Table-3: Epworth Sleepiness Scale

THE EPWORTH SLEEPINESS SCALE

Name: _____

Today's date: _____ Your age (years): _____

Your sex (male = M; female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you. Use the following scale to choose the *most appropriate number* for each situation:

0 = would *never* doze
1 = *slight* chance of dozing
2 = *moderate* change of dozing
3 = *high* chance of dozing

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

Thank you for your cooperation

Berlin questionnaire includes 3 categories having 11 questions which separates patients based on severity of the symptoms as low and high risk of OSA.5 Patients with OSA who need to undergo surgery under general anesthesia are at risk of developing peri and post-operative complications like failed intubation, post-operative haemorrhage needing return to operation room, post-operative arrhythmia.17

TREATMENT-

Aim of the treatment is to establish normal nocturnal oxygenation and ventilation, cessation of snoring and elimination of sleep disruption.9 There are three categories under which treatment of OSA can be grouped-behavioural, medical and surgical.

Behavioural - This involves counselling after diagnosis has beenestablished. Counselling is about explaining importance of weight loss, sleep deprivation symptoms, avoiding alcohol consumption before sleep, nocturnal position that is helpful to patient that is lateralrecumbent.9 Weight loss is recommended in all patients with OSA. Medical intervention - includes continuous positive airway pressure (CPAP), oral appliance and medications for affected patients.9 CPAP is the most effective method of OSA management.23 CPAP acts as a pneumatic splint by elevating and maintaining constant pressure in upper airway during respiration. There are reports of severely affected patients by OSA being most benefited by use of CPAP therapy.23 Oral appliances-As early as 1902, the concept of oral appliances for treatment of upper airway obstruction and mandibular deficiency came.18 The role of oral appliances was first advocated by Pierre Robin for the management of airway obstruction in 1923.4 The aim with such appliances is modification of upper airway position in a way that either enlarge the airway space or reduce the airway collapse (Figure 2).23

Figure-2 : Oral Appliance For OSA

Proper impressions and fabrication for adequate fit is required for these appliances. Patient education about use of appliances during sleep is must for the success of therapy for proper treatment.8 Classification of oral appliances includes, tongue retaining and mandibular repositioning devices.23 Tongue retaining devices enlarge the upper airway volume and are indicated in patients with large tongue, few or no teeth or in those who cannot adequately advance their mandible. Mandibular repositioning devices reposition the mandible and related structureslike tongue and hyoid anteriorly and increase the dimensions of upper airway both laterally and antero-posteriorly in OSA affected patients. It is reported that protruded mandible results in upper airway changes in anteroposterior width, hyoid and third cervical vertebral position.4 Medications for OSA treatment: Protriptyline is the principal medication that has been used in OSA treatment. Proptriptyline is a tricyclic antidepressant with non-sedating properties and reported improvement in drowsiness. The proposed mechanism of action of Proptriptyline is that amine pump is blocked in pre-synaptic nerve endings that helps in decreasing apnea.38 Fluoxetine is also used for mild to moderate disease but not effective in severe OSA cases.37,38 Hanzel et al reported that Protriptyline and fluoxetine reduced rapid eye movement (REM) during sleep time and beneficial effect of these medications was reduced apnea andhypopnea index during sleep.37 Modafinil has been used to treat residual sleepiness in OSA patients despite use of continuous positive pressure airway (CPAP). It causes improved subjective and objective daytime sleepiness as it is wakefulness promoting drug. As risks of long-term exposure from the drug are unclear in the available trials, hence Modafinilhas been withdrawn by the European Medicines Agency (EMA) to be used in residual sleepiness in OSA.36

Surgical treatment - Surgical intervention is indicated when polysomnography diagnosed OSA patient has failed CPAP therapy.4 Tracheostomy bypasses the upper airway obstructions and was the first treatment for the patients with OSA. It could be considered insevere OSA in CPAP intolerant and when all other surgical treatments fail.23 It has serious complications like aesthetic compromise, tracheal stenosis, recurrent bronchitis, speech difficulties.6 Surgical procedures includes Stage-l and Stage-ll (Table-4).

Table-4: Surgical Techniques For OSA Treatment

Technique	**Location**
Stage I surgery (site-specific Techniques) Nasal surgery (eg, turbinectomy) Uvulopalatopharyngoplasty (UPPP) Base of tongure surgery Genioglossal advancement modified genioplasty radiofrequency ablation hyoidmyotomy	Nose Oropharynx (retropalatal airway) Oropharynx (retrolongual airway) +Hypopharynx + Hypopharynx
Stage II Surgery (upper airway reconstruction) Advancement (MMA)	Nasopharynx/oropharynx/ hypopharynx
other Tracheostomy Nonairway surgery Bariatric surgery	Trachea Gastric

Stage -l surgery is site specific and planned in patients with RDI score minimum of 15-20 and unsuccessful or intolerable non-surgical procedures. Stage l surgical procedures includes nasal surgery, uvulopalatopharyngoplasty, base of tongue surgery.
Stage -ll surgery includes maxillomandibular surgery. Surgery is planned and done in accordance with zones of involvement.

NASAL SURGERIES FOR OSA

About 45% of patients suffering from sleep disordered breathingcomplains of nasal obstruction. It is documented that approximately two third of the total airway obstruction is due to nasal airway resistance as anterior part of nasal cavity has more effect on nasal airway resistance than posterior part of nasal cavity. In recumbent position during sleep, there is increased nasal mucosal congestion as well tissue congestion, as veins of head are valve less the venous pressure in the nose increases.4Nasal surgery involves septoplasty which is straightening the nasal septum under general or local anesthesia where reduction in few millimetres of septal deviation has produce significant improvements in nasal airway.4

Turbinate reduction in which size of inferior or middle turbinate are reduced by partial resection, radiofrequency, laser, submucosal cautery. However, there are reports stating that septoplasty and turbinate reduction significantly reduced AHI.4 Nasal valve reconstruction is the procedure of internal or external nasal valve reconstruction using many techniques like auricular cartilage grafts, flaps or suturing of intra or extra nasal tissue. Cases reported by Friedman and colleagues showed that patients who underwent nasal surgery had improved snoring, nasal breathing, daytime sleepiness and reduction in optimal CPAP.4

PALATAL SURGERIES FOR OSA TREATMENT

Uvulopalatopharyngoplasty (UPPP) was first described by Fujita in 1981, a procedure involving tonsillectomy, soft palate and uvula trimming and suturing of tonsillar pillars.29 Conway et al reported 50% success rate by UPPP in a one year follow up study.39 The associated complications in post-operative period are airway obstruction, dysphagia, nasal reflux, hyper nasal speech and compromised nasal CPAP efficacy.23 UPPP has low success rate and considerable morbidity involved hence many modifications have been proposed byvarious authors.29 Uvulopalatal flap (UPF) reported by Powell and colleagues is advancement of uvula and distal part of palate by creating a flap that was reversed and sutured close to hard palate. A modification of UPF is described, that is extended uvulopalatal flap (EUPF). This includes additional removal of submucosal adipose tissue from soft palate and supratonsillar region. Based on reduction in AHI, success of 81.8% has been reported.4 An original research in USA multicentre trial, placed palatal implants for treatment of mild to moderate OSA as they alter elasticity of soft palate. These implants were made of Polyethylene terephthalate (PET) which on implantation causes permanent alteration in tissue site. Adverse events related to palatal implantation are partial exposure of implant, irritation and ulcerations in mucosa.21

Radiofrequency surgery for palatal reduction is a minimally invasiveand effective procedure in mild to moderate OSA patients who do not have nasal obstruction. The procedure stiffens the pharyngeal tissue and enlarge the upper airway. There are reports of improved objective and subjective measurements after radiofrequency for palatal and tongue base reduction.25

TONGUE SURGERIES IN OSA

Posterior airway space is decreased in macroglossia, which is caused by systemic diseases like amyloidosis, hypothyroidism etc that can predispose the person to OSA. Glossectomy is a treatment option in macroglossia that involves removing the anterior and middle portion of tongue.11 Partial glossectomy can be performed that helps in increasing oropharyngeal patency in macroglossia.23

MANDIBULAR PROCEDURES FOR OSA

Surgeries for OSA treatment that have a potential for cosmetic enhancement also include mandibular osteotomies with tongue advancement, bimaxillary advancement, cervical lipectomy etc. Maxillomandibular advancement (MMA) is a surgical procedureinvolving Le fort l osteotomy with bilateral mandibular sagittal split osteotomy and repositioning of maxilla and mandible leading to increased anteroposterior and lateral dimensions in upper airway at all the levels in maxillomandibular deficiency (Figure-3).23

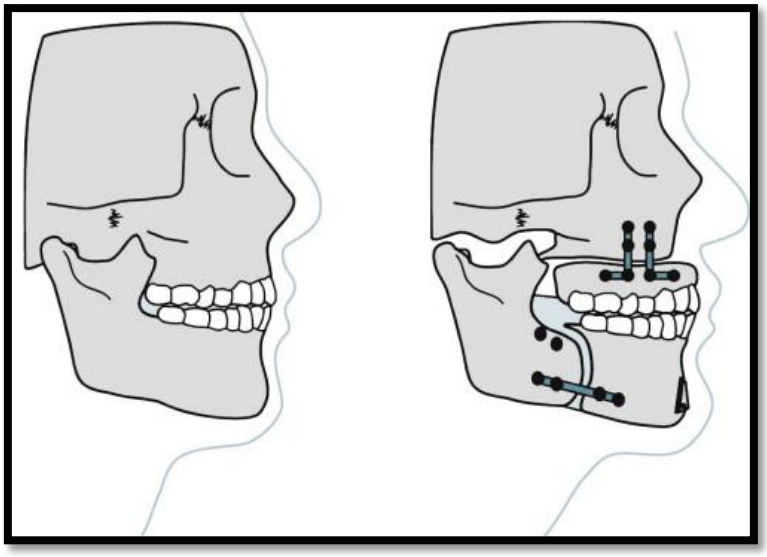

Figure-3 : Maxillomandibular Advancement

It is the onlysurgical procedure that creates more space for tongue to be advanced anteriorly as it advances the midface and provide more space for tongue.44,41 Reported complications are 10% cases with mandibular nerve neurosensory deficit.23 Genioglossal advancement with hyoid myotomy (GAHM) and MMO (Maxillomandibular osteotomy) advances the mandible and hyoid, hence pharyngeal muscles and base of tongue are also advanced causing expansion of airway.12 MMA is considered most effective method of pharyngeal airway expansion in improving or eliminating OSA and is considered best alternative to tracheostomy.Presurgical orthodontics helps in better post-operative outcomes by improving occlusion and eliminates pre-existing dental compensations. Although it is suggested that MMO has the highest cure and success rate of all the surgical procedures, still it should be tailored for each patient.44 Orthognathic procedures are successful with good prognosis if the requirement of corpus lengthening is less than 8mm, in case 8mm or more advancement is required, distraction osteogenesis is considered which involves simultaneous distraction histogenesis of soft tissues like nerve, muscles so that proper hard and soft tissue changes takes place.Patients suffering from acquired disorders like bilateral TMJ ankylosis with micrognathia and OSA (triad) are treated according to the new protocol where pre-release distraction osteogenesis is advisable which not only improves width of posterior airway space, AHI, but also aesthetics.27 The hypothesis behind the proposed protocol is occurrence of bradycardia via Trigemino-cardiac reflex and drop in oxygen saturation which leads to non-compliance for active mouth opening in management of TMJ ankylosis without pre-release distraction. Pre-release distraction osteogenesis helps in compliancefor active mouth opening exercise without respiratory distress in postrelease phase which further reduces the chances of re-ankylosis. Tsui WK et al in their review stated that success rate of mandibulardistraction osteogenesis (MDO) in OSA patients is 90% to 100%.41 It is concluded by review that MDO was highly effective in resolving OSAS in children having Pierre robin syndrome and adults with retrognathic mandible (Figure-4).

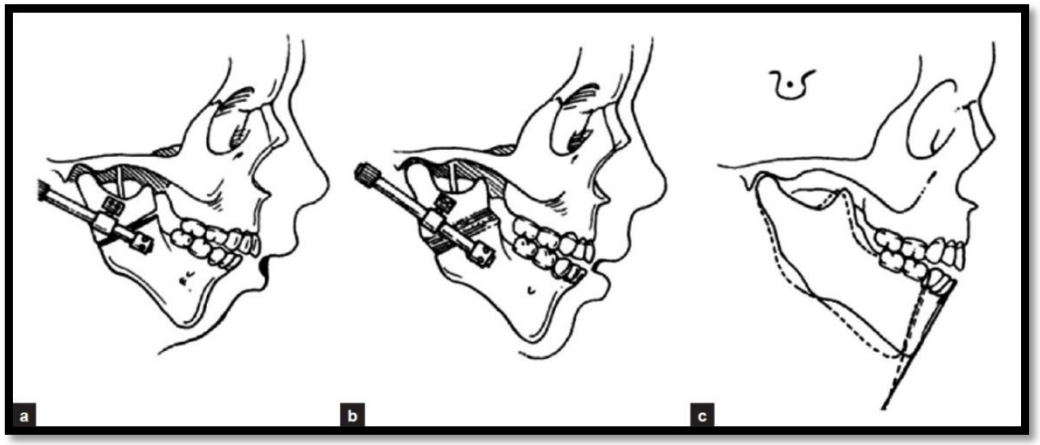

Figure-4 : Distraction Osteogenesis

Genioglossal advancement was first described by Riley and co-workers in 1984 which involved genial tubercle advancement and thereby genioglossal muscle for treatment of hypopharyngeal obstruction.4 A success rate between 39-78 % is reported by a meta-analysis for genioglossal advancement. In this inferior border of mandible is sectioned and advanced anteriorly leaving the dentoalveolar process intact. There are many variations in the surgical procedure involving alterations in osteotomy and altered positioning of bone segment like 900 rotation of bony window segment, laterally slanting the vertical cuts (Figure-5).44

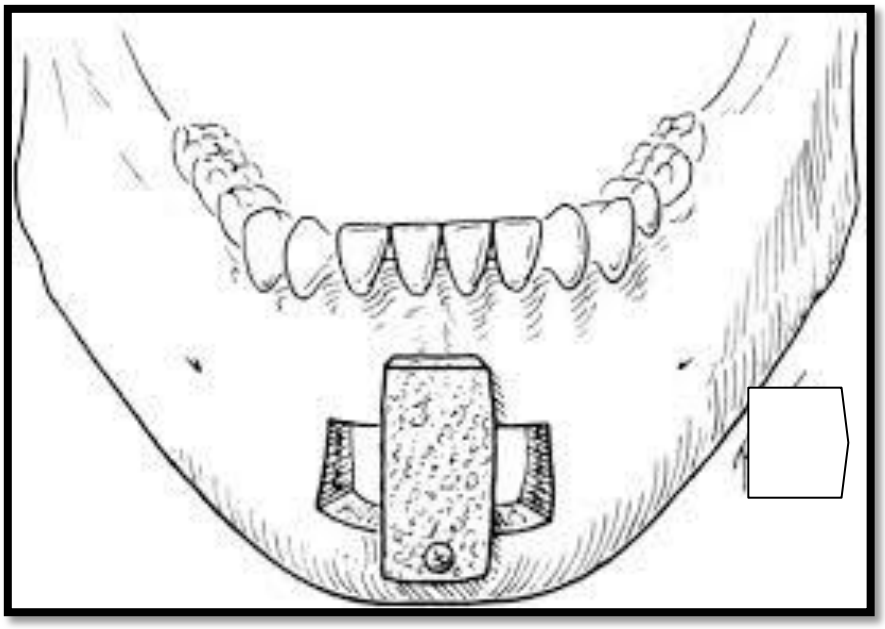

Figure-5: Genioglossal Advancement

In patients with severe micrognathia like bird facedeformity and OSA, mandibular advancement with advancement genioplasty increases posterior airway space which improves not only airway but also aesthetics.45 Genioplasty consists of osteotomy of anterior mandible with genioglossal and inferior border advancement. This is a minimally invasive procedure that results in both functional and aesthetic changes.11Sliding genioplasty is a procedure of advancement genioplasty done in microgenia cases due to foreshortened mandible or retrognathia.44 Riley et al observed in their study that following hyoid myotomy/suspension along with mandibular osteotomy / genioglossus advancement, improvement in apnea was observed to be between 60-70%.16 There are some soft tissue procedures that are performed that includes submental hump reduction. In this procedure, excessive fat is removed from submental region with closed or open technique. Obstructive sleep apnea affected patients are often obese and may have fat deposition in the neck leading to increased neck circumference, so improved OSA symptoms and aesthetic after cervical lipectomy and or submentoplasty can be achieved.42 In patients suffering from OSA, apart from conservative treatment like CPAP therapy, surgical treatments are also considered. There are further methods which are adjuvant to the standard non-surgical or surgical methods like, chest physiotherapy, yoga, breathing exercises and many more.There is an increased prevalence of obesity throughout the world, hence related disorders like OSA have increased, bariatric surgery is a newer method of surgery that helps obese patients to decrease fat. Bariatric surgery is reported effective in morbidly obese patients, those with basal metabolic index (BMI) of 40 or more or with coexisting medical ailments and BMI 35 or more.23 Bariatric surgical procedures have reported improvement and resolution of obesity-related comorbidities. Greenburg et al in a recent meta-analysis, reported a significant reduction in AHI score in patients after bariatric surgery.46 However, authors have reported and discussed that it is not known that how much weight loss would be required for treatment of OSA but a26% decrease in AHI has been predicted with 10% weight loss.31

IV. Conclusion

Sleep disordered breathing is one of the most common and yet manyatimes unappreciated condition associated with craniofacial abnormality. A practicing personnel who is responsible for diagnosing is either a primary care physician, otolaryngologist, general dentist, or oral and maxillofacial surgeon, who should follow a sequence of steps for clinical diagnosis by taking in-depth history, physical examination, using diagnostic aids and keeping oneself updated about the risk factors. It is the need of the hour to recognize OSA as an

independent factor that can affect morbidity and mortality if not treated. The disorder must be suspected in obese patients or people who snore as reported by bed partners. Patients with OSA may present with recurrent atrial fibrillation, stroke or resistant hypertension. Due to wide variety of anatomical and physiological causes of OSA, it is important to tailor the treatment for each patient to get the best possible outcome.

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