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RESEARCH ARTICLE

OBSTRUCTIVE SLEEP APNEA: PART II. TREATMENT MODALITIES.

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Abstract

Obstructive sleep apnea (OSA) can present serious health risks and must be diagnosed by a physician in conjunction with a sleep study. Of nonsurgical treatment alternatives, Nasal continuous positive airway pressure (nCPAP) has been shown to be more effective than oral appliance therapy in improving respiratory disturbances. Common difficulties associated with Continuous positive airway pressure (CPAP) therapy include sense of dryness in the mouth, rhinorrhea, nasal congestion and dryness, mask discomfort, claustrophobia, irritation from device noise, aerophagy, chest discomfort and partners's intolerance. Therefore many patients are unable to or unwilling to comply with the use of CPAP. This article discusses the various treatment modalities that have been investigated in the management of OSA

Introduction:-

Sleep apnea is a medical disorder that can be present in any age group.[1,2] It is characterized by temporary cessation of breathing during sleep.[3] It is estimated to affect approximately 2% to 4% of the adult population and is most commonly observed in middle-age overweight males. Strained respiration, decreased blood oxygen levels, and arousals that interrupt a normal sleep pattern characterize this syndrome. Many cases present a significant health risk and can result in excessive daytime sleepiness, early morning headaches, impaired concentration, social impairments, systemic and pulmonary hypertension, traffic and work related accidents, ischemic heart disease, and cerebrovascular disease.[4,5]

Nasal application of continuous positive airway pressure to treat OSA was first reported in 1981.[6] Since then it has become the most effective and widely used treatment option for OSA. CPAP acts as a pnuematic splint to force the upper airway open during sleep, and thus prevents OSA. Common difficulties associated with CPAP therapy include sense of dryness in the mouth, rhinorrhea, nasal congestion and dryness, mask discomfort, claustrophobia, irritation from device noise, aerophagy, chest discomfort and partners's intolerance.[3] Many patients are unable to or unwilling to comply with the use of CPAP. Upto 30% of patients with OSA reject CPAP treatment as a long-term treatment option.[7-9] Among those who accept CPAP, compliance is highly variable and often thought to be suboptimal.[10,11] This article discusses the various treatment modalities that have been investigated in the management of OSA.

Therapeutic strategies for patients with sleep apnea may be grouped into three general categories: behavioral, medical, and surgical. Treatment decisions should be based on the effect of the sleep disorder on daytime symptoms and cardiopulmonary function rather than on the absolute number of episodes of apnea or hypopnea. The goals of

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treatment are to establish normal nocturnal oxygenation and ventilation, abolish snoring, and eliminate disruption of sleep due to upper-airway closure.[12]

Behavioral:-
- ✧ Weight loss
- ✧ Avoidance of alcohol and sedatives
- ✧ Avoidance of sleep deprivation
- ✧ Nocturnal positioning

Medical:-
- ➢ First-line therapy
- ✧ Positive pressure through a mask
- ➢ Second-line therapy
- ✧ Oral appliance
- ✧ Other
- ✧ Fluoxetine or protriptyline
- ✧ Thyroid hormone (in hypothyroid patients)
- ✧ Nocturnal oxygen

Surgical:-
- ✧ Upper-airway bypass
- ✧ Tracheostomy
- ✧ Adenoidectomy
- ✧ Tonsillectomy
- ✧ Nasal Polypectomy
- ✧ Upper-airway reconstruction
- ✧ Uvulopalatopharyngoplasty
- ✧ Septoplasty
- ✧ Genioglossal advancement
- ✧ Maxillomandibular advancement

Weight loss:-
Obesity has been shown to be associated with OSA.[1,13] An increase in the body mass index of one standard deviation was associated with a four-fold increase in the risk of having an apnea-hypopnea index (AHI) of more than five per hour.[3] Peppard et al, have shown in normal volunteers that for each percentage of change in weight there was approximately a 3% change in the AHI (a 10% reduction in weight was associated with a 26% reduction in the AHI).[14]

Several studies have examined the short term effect of weight loss in OSA and have recently been reviewed.[15] Significant weight loss is associated with improvement in OSA and total resolution in some patients. However, the amount of weight loss achieved does not always correlate with the extent of improvement in the AHI. Alcohol consumption should be avoided in the evening as alcohol may relax the airway making the airway more prone to obstruction at susceptible sites.[3]

Positional Therapy:-
The frequency of apnea and hypopneas is influenced by body position in upto 50% patients with OSA.[16,17] The AHI is higher in the supine position and lower in the lateral position or with the head of the bed elevated to 30 to 60 degrees. The upper airway size has been shown to be larger in the seated position compared to the supine position.[18-20] However, there is no increase in the upper airway size when patients with OSA move from the supine position to the lateral position.[20]

Methods for avoiding sleeping in the supine position include positional monitor that triggers an alarm, wearing a backpack with a soft ball inside, pinning a tennis ball to the patients pajama top or placing a wedge pillow lengthwise in the bed.[15] The position-dependent therapy may be a reasonable alternative to nasal CPAP in patients with mild disease with a positional component. However, the data for positional therapy using randomized trials is sparse and the long term effects of positional therapy have not been evaluated.[3]

Pharmacological Therapy:-

Protriptyline is a non-sedating tricyclic antidepressant agent that was inadvertently found to reduce OSA in patients with narcolepsy. It's mechanism of action is not clear. However, protryptiline is well known to reduce the rapid eye movement (REM) sleep. OSA is often worse in REM sleep and therefore by decreasing REM time, the apnea time and severity of oxygen desaturation may be improved.[21] Patients with co-existing depression and female patients may be potentially preferable patients for this medication.

Serotonergic neurons exert an excitatory effect on upper airway dilator motor neurons.[22,23] Upper airway obstruction is associated with a decrease in the activity of upper airway dilator muscles such as the genioglossus. Maintenance or augmentation of activity in these muscles may prevent upper airway collapse. In an animal model of OSA, the systemic administration of serotonin antagonist led to reduction in upper airway cross-sectional area and oxygen desaturation[24] and the administration of serotonergic agents (trazadone and L-tryptophan) was effective in treating sleep disordered breathing.[25] However, current data show that this reduction is not clinically significant.

Currently there are no data to recommend the use of progesterone in the management of OSA. The data showing benefit of medroxyprogesterone in the treatment of obesity hypoventilation syndrome is limited by the small numbers of patients and the lack of long term results.[26-31]

Hypothyroidism has been associated with sleep disordered breathing.[32] Nine out of eleven consecutive patients with hypothyroidism were reported to have OSA.[33] However, the prevalence of hypothyroidism was only 3% in 65 consecutive patients diagnosed to have OSA.[34] CPAP therapy may be started in patients with severe hypothyroidism and severe OSA and in patients with an urgent need to treat OSA in combination with thyroid hormone replacement therapy.

As acetazolamide produces a metabolic acidosis and stimulates ventilation, it was hypothesized that it would improve sleep disordered breathing. In a blinded placebo-controlled study, there was a physiologic but no clinical improvement (reduction in AHI from 50 per hour to 26 per hour) in ten patients with OSA.[35]

The positive chronotropic action of theophylline may improve the stability of breathing by decreasing circulation time, in congestive heart failure.[36] Theophylline has been shown to be useful in central apnea and periodic breathing and does not appear to be useful in patients with OSA.

Systemic hypertension is common in patients with sleep apnea. Cilazapril, an angiotensin converting enzyme inhibitor and metoprolol, a beta-blocker have been shown to reduce AHI by about 30%.[37] Clonidine has been shown to reduce AHI in REM sleep. Whether the reduction in sleep apnea is because of decrease in blood pressure or due to the direct effect of the drug is not clear. However, there are also data showing that anti-hypertensive treatment may lead to an increase in sleep apnea and hence the effect of antihypertensive treatment on sleep apnea is not clear.[38]

Atrial overdrive pacing has been reported to substantially reduce the number of episodes of central and obstructive apnea in patients with pacemakers that had previously been implanted for the treatment of the sick sinus syndrome or the bradycardia-tachycardia syndrome.[39] However, further studies are needed to elucidate the mechanisms involved in achieving these reductions and to assess the precise role of cardiac pacing in preventing symptoms, disability, and death in the general population of patients with sleep apnea syndrome.[39]

Opioid antagonist and nicotine stimulate ventilation through generalized cortical stimulation. An increased opioid activity has been demonstrated in the cerebrospinal fluid of patients with OSA.[40] Although doxapram infusion has been shown to reduce the duration of apnea and the severity of oxygen desaturation, it did not reduce the number of episodes of apnea or oxygen desaturation. Nicotine gum chewed before bedtime has been shown to reduce the number of apneic episodes in the first two hours of sleep.[41] Transdermal nicotine patches did not influence the apnea episodes but was found to reduce the lowest oxygen saturation during sleep.[42]

Modafinil may be considered as adjunctive therapy in patients who are compliant with CPAP therapy but have persistent day time sleepiness. Although, modafinil has also been shown to improve objective measures of sleepiness in untreated patients with OSA, the concern is that it may not prevent the cardiovascular consequences of

OSA as it does not eliminate upper airway obstruction. Due to lack of controlled trials with long term follow up, modafinil is currently not recommended in untreated patients with OSA.[43,44]

Oxygen is sometimes used in patients with central apnea caused by heart failure. It is not used to treat obstructive sleep apnea. Oxygen administration during sleep in some cases can paradoxically lead to significant worsening of the apnea. Supplemental oxygen may be helpful in patients who have frequent and severe desaturations.[3]

Currently, there is no effective pharmacological treatment of OSA. Different drugs seem to affect different aspects of sleep apnea, such as central or obstructive or those occurring in REM or non-REM sleep. Stimulation of ventilation may be helpful in some patients with central apnea, periodic breathing or hypercapnea. Modafinil might be helpful as an adjunctive drug to CPAP in selected patients with OSA who continue to have day-time sleepiness despite use of CPAP.

Positive Airway Pressure Therapy:-

The first reported use of nasal continuous airway pressure (nCPAP or CPAP) for OSAHS in adults was by Sullivan and colleagues[6] in 1981. Their device consisted of intranasal tubes attached to a blower unit. In 1983, the nasal mask delivery system, similar to contemporary systems, was introduced.[45] Fundamentally, the application of a therapeutic level of CPAP results in immediate relief in the upper airway obstruction.

This benefit has been attributed to the CPAP functioning as a "pneumatic splint" for the upper airway.[46] Additional physiologic benefits of CPAP application have been shown to include improvement in the function of pharyngeal dilator muscles,[47] ventilator drive,[48] and upper airway morphology.[49] CPAP rarely results in serious side effects. However, about 25% of patients may develop nasal congestion with chronic use.[8]

The benefit of CPAP in treating the sleepiness associated with OSAHS has been well established.[50-54] The patient's perceived quality of life showed a significant increase after treatment.[51-53] Interestingly, the spouses of OSAHS patients also gained from CPAP therapy, as it eliminated the impairment of their own sleep due to the snoring and sleep disruption caused by bed partners.[55] More recently, emerging evidence suggests that CPAP therapy reduces long-term morbidity and mortality from cardiovascular causes.[56,57]

The introduction of automatically adjusting CPAP devices (auto-CPAP) over the past several years represents a significant advancement in CPAP technology since its inception in 1981. The device continuously adjusts the applied airway pressure to an "optimum" level throughout the night and appears to improve compliance.[58]

Common side effects of CPAP include rhinorrhea, nasal congestion and dryness, mask discomfort, conjunctivitis from air leak, skin abrasions, claustrophobia, irritation from device noise, difficulty exhaling, aerophagy, chest discomfort, and bed-partner intolerance.[3]

Patients who cannot tolerate CPAP because of discomfort from exhaling against high pressure can be treated with the addition of a pressure "ramp"or the use of bilevel positive-pressure therapy. Autotitrating CPAP was also introduced in the last decade with the premise that by continuously adjusting pressure to meet the patient's variable needs, the overall mean airway pressure will be reduced. This would hopefully allow better tolerance and therefore better compliance.[59]

BiPAP (variable/bilevel positive airway pressure) provides two levels of pressure: a higher inspiratory positive airway pressure (IPAP) and a lower expiratory positive airway pressure (EPAP) for easier exhalation. This modality appears to be better tolerated and individuals may find it easier to use; it is used in cases when patients cannot tolerate CPAP, for patients with chronic CO_2 retention as well as sleep apnea, and for patients with neuromuscular disease who need some assistance with nocturnal ventilation. BiPAP may also be more useful in CSA.[60]

Oral Appliances:-

In 1934, Pierre Robin first described the concept of advancing the mandible with a monoblock functional appliance to treat airway obstruction in infants with micrognathia. His method was not accepted to any extent and it was not until 1985 that Meier-Ewert and coworkers next described an intraoral protraction device for the treatment of sleep apnea. Many articles followed showing therapeutic efficacy in treating OSA with various one-piece, hard acrylic, nonadjustable advancement appliances.[61]

Dentists specializing in sleep dentistry can make a custom-made mouthpiece that shifts the lower jaw forward, thereby maintaining an open airway (in theory). This approach can be successful in individuals with mild to moderate OSA but has been proven less effective for severe cases.[62] In one study comparing CPAP to dental appliance in mild to moderate OSA, dental appliances decreased AHI from 21 (baseline) to 14, compared to a decreased AHI of 5 in patients using CPAP.[63]

Oral appliances (OA) find their greatest success when utilized for simple snoring, upper airway resistance syndrome and mild to moderate obstructive sleep apnea. Improvement of snoring occurs in high proportion of patient with complete resolution in smaller subset. A large literature review by Lowe showed that, as groups oral appliances were effective in mild to moderate OSA with 75 % compliance rate. Oral appliance therapy has been accepted by the American Sleep Disorder Association as an appropriate treatment modality for OSA patient.[59]

Oral appliances can lift the soft palate or advance tongue or mandible thus opening the airway. A combination of oral appliances and CPAP is also used in a few cases. Those that lift the soft palate are rarely used because of gag, discomfort and success of laser and radio frequency soft palate procedures. Tongue Retention/Tongue Retaining Devices (TRD) have an anterior hollow bulb, which creates a negative pressure vacuum when tongue is inserted. Tongue is held forward away from post pharyngeal wall, opening the airway. This appliance simultaneously modifies the position of the mandible.[59]

Mandibular Repositioning or Advancement Devices (MRD / MAD) function by engaging one or both of dental arches to modify mandibular protrusion and improve the velopharyngeal airway patency.[64] The most common mandibular repositioning dimension quoted is 50-75 % of maximal protrusion (approximately 5-7mm).[65] As these appliances hold the mandible in antero-inferior position, these indirectly bring the tongue forward as a consequence of muscle attachment and open up the posterior airway. The repositioning may also stretch and reduce the collapsibility of soft palate via its connection to the base of tongue and increase the superior airway space.[59]

Although tongue repositioning devices and mandibular advancement devices have been standard appliances for treatment of OSA, a recent study by Venket R et al describes the use of four new prosthodontic appliances for managing sleep apnea namely uvula lift appliance, uvula and velopharynx lift appliances, nasopharyngeal aperture guard and soft palate lift appliance and a conventional mandibular advancement appliance. He concluded that nasopharyngeal aperture guard appliances was the best among the five type of appliances. Further studies would be required in this direction.[65]

Till date, more than 40 different OAs have been patented. Design variations depend upon :

- ✧ Method of retention
- ✧ Flexibility of material
- ✧ Adjustability
- ✧ Vertical opening
- ✧ Freedom of jaw movement.

According to the material used these can be either polyvinyl vacuum formed thermoplastic appliances or those made of hard acrylic. According to adjustability these may be fixed or adjustable. Fixed oral appliances are usually one piece design that can be adjusted in the antero posterior plane.[59]

One of the accepted design is one-piece non adjustable soft vinyl vacuum formed mandibluar repositioning appliance consisting of thermoplastic material covering the maxillary and mandibular arches in the desired antero inferior position. The occlusal position is established and recorded by either a wax bite, silicon bite or anterior jig with inter occlusal registration.[66]

In the two part Herbst – style appliance, the arches are connected by pivoting bars that can be altered in length to titrate the protrusive mandibular position for effectiveness and comfort. The occlusal registration for these two part appliances is not as important because mandibular reposition can be titrated from the intercuspal position.[66]

TAP (Thornton Adjustable Positioner) appliance uses a hook on the maxilla to attach to the mandible in order to bring it forward. For edentulous patients with OSA, a Tongue Stabilizing Device (TSD) can be used which does not attach to teeth and acts as a pacifier. It is made of soft silicon and holds the tongue forward by gentle suction

preventing it from falling back. Implant retained mandibular repositioning device in the mandible is a viable treatment modality for edentulous OSA patients.[67]

The advantages of oral appliances over other sleep apnea treatment options include relatively low cost, good success rates (efficacy comparble to uvulopalatopharyngoplasty but less efficacious than CPAP), good compliances a more benign adverse-effect profile, rapid effect and easy termination without sequalae.[68] OA insertion can be performed as a single stage procedure in an out patient setting. These can be used effectively for simple snoring and mild to moderate OSA as recommended by American Academy of Sleep Medicine,[69] but a study by Jeffery Pancer concluded that oral appliances appear to be effective treatment alternative for selected patients of snoring and varying degrees of sleep apnea including those with severe OSA.[70]

OAs improve the blood oxygen saturation levels as they relieve the apnea in 20 -75 % of patients. They reduce the AHI to < 10 events per hour or bring about 50 % reductions in AHI. They also reduce AHI to normal in 50-60 % of the patients.[59] Side effects with oral appliances are generally minor and include excessive salivation, muscle and tooth discomfort and occasionally temporomandibluar joint discomfort. But symptoms usually improve over time.[71]

Surgery:-

When the nonsurgical therapies for OSA fail or are unacceptable to the patients, surgical options are considered. The first surgical treatment for OSA was tracheotomy in 1969 by Kuhol[72] for the treatment of upper airway obstuction in a patient with Pickwickian syndrome. Previously, in 1964, Ikematsu started treating snoring with a soft palate procedure known as uvulopalatopharyngoplasty (UPPP).[73] Building on that, Fujita published results on UPPP in OSA. However, Sher's review in 1996 showed the success rate to be close to 40%.[74] Since then, a whole host of procedures were developed to treat OSA. They are all designed to improve the posterior airway from the nasal aperture to the larynx.[75]

Fujita et al[76] simply categorized the upper airway obstruction as either retropalatal or retroglossal. The retropalatal level involves the soft palate, uvula, and palatine tonsils. The retroglossal level involves the tongue base and supraglottic structures. Type I obstruction is the presence of restriction only at the retropalatal level. Type II obstruction is the presence of restriction only at the retroglossal level. Type III is the presence of both obstructions at both levels. However, there is increasing support in a different concept proposed by Moore.[77] Moore considered the airway obstruction as a spectrum of disease, starting from primary snoring as the mildest form, to upper airway resistance syndrome (UARS) and then to the different degrees of OSA; mild, moderate, and severe.[78]

The various surgical techniques used for treatment of OSA Are:-

- Procedures in which the soft tissue is either removed or ablated

1. Uvulopalatopharyngoplasty (UPPP)
2. Laser assisted uvulopalatoplasty (LAUP)
3. Uvulopalatopharyngo-glossoplasty (UPPGP)
4. Laser midline glossectomy
5. Radiofrequency ablation of tongue base
6. Reduction of tongue base with hyoepiglottoplasty

- Procedures in which soft tissue is repositioned through skeletal alteration

1. Mandibular advacement (MA)
2. Maxillomandibular advancement (MMA)
3. Transpalatal advancement pharnygoplasty
4. Genioglossal advancement
5. Hyoid myotomy and suspension

A major limitation in the interpretation of the data for the role of surgery in OSA is the application of different criteria to define surgical success by different authors. It is clear that the definition for surgical success should be clearly defined so that data reporting is uniform to allow proper interpretation. Ideally, preoperative and postoperative PSG (polysomnography) should be performed and the assessment criteria should include RDI (Respiratory disturbance index), sleep architecture, time spent below 90% SpO_2 and symptoms.[3] Currently, the

commonly accepted definition for surgical cure is respiratory distress index or apnea-hypopnea index less than 20 with a reduction greater than 50% and few desaturations less than 90% with improvement of subjective symptoms.[78] In children having OSA, surgery to correct underlying abnormalities often results in an improvement in symptoms and in some cases, it is curative.[79]

Riley et al[81] found the success rate for UPPP, genioglossus advancement (GGA), and hyoid suspension (HS) to be 61% and Friedman, et al achieved a 41% success for UPPP and radiofrequency volume reduction procedure (RFQ). Using the uvulopalatal flap with RFQ, Verse et al obtained a success rate of 51%. Hard tissue surgical procedures have shown better success rates but they require more preparation and may have higher morbidity. Maxillomandibular advancement (MMA) which is modeled after conventional orthognathic surgery has achieved remarkable success rates of 97% and 100%.[81] Therefore, it is important to examine the patients carefully before deciding on the most appropriate surgical procedures.

Uvulopalatopharyngoplasty (UPPP) enlarges the retropalatal airway by excision of the tonsils (if present), trimming and reorientation of the posterior and anterior tonsillar pillars, and excision of the uvula and posterior portion of the palate. The results are better in patients in whom the predominant site of obstruction is retropalatal. Complications include velopharyngeal insufficiency (2%), bleeding (1%), nasopharyngeal stenosis (1%) and death due to upper airway obstruction (0.2%). The true prevalence is difficult to estimate as many reports do not comment on the presence or the absence of the postoperative complications.[74]

Laser-assisted uvulopalatoplasty (LAUP) enlarges the retropalatal airway by ablation of the uvula and posterior margin of the soft palate with carbon dioxide laser. LAUP is often performed under local anesthesia. The success rate reported with this procedure ranges from 0 to 87 %. However, the definition of success rate also varied in these studies to include a postoperative reduction in the RDI or AI (Apnea index) by 50% with or without reduction of the AHI to less than 20 per hour.[82-87]

Saggital mandibular osteotomies are performed to effect anterior mobilisation of the insertion of the tongue at the genioid tubercle and thus enlarge the retrolingual space. There must be a significant antecedent mandibular deficiency and dental malocclusion to permit the requisite degree of anterior movement of the mandible and the mandibular teeth.[88,89] Although, the reports of this technique are few, they demonstrate the potential influence of mandibular deficiency as a cause of OSA and furthermore that mandibular advancement could treat OSA.[3]

The degree of mandibular advancement performed to treat OSA without maxillary advancement would lead to prognathism and dental malocclusion. Performing maxillary advancement permits mandibular advancement in patients with OSA having maxillomandibular deficiency but not maxillomandibular disproportion. The maxilla and the mandible are both advanced by sagittal osteotomies, which enlarge the retrolingual as well as some retropalatal space. It also improves the tension and collapsibility of the suprahyoid and velopharyngeal musculature.[90]

Nasal, Septal and Adenoid surgeries are sometimes performed in order to open the nasal breathing passages and permit easier breathing. Weak or malpositioned cartilages around the nostrils can impede nasal breathing as will a droopy nasal tip or excessively narrow nostrils. The nasal turbinates are horizontal ridges within the nose. These may become chronically enlarged usually as a result of allergies. Reduction in the size of the turbinates will improve nasal air flow. The nasal septum divides the nose into right and left nasal passages. A septal deviation is an alteration of the relatively straight and midline position of the septum. If the septum is crooked, it may cause blockage of the nasal breathing passage. It can be straightened in order to improve breathing and is called a septoplasty. An enlarged adenoid may occasionally interfere with breathing. The adenoid is located in the posterior surface of the nasal cavity above the soft palate. An adenoidectomy removes this excess tissue to allow for unrestricted airflow through the nasal passages and upper throat. Although this is most commonly performed in children, it may be indicated in teenagers or young adults.[79]

The tonsillectomy can be an important component of surgery for OSA, especially if the tonsils are at all enlarged. The removal of redundant tissue by tonsillectomy increases the caliber of the throat thereby reducing blockage to breathing. Since the quality and quantity of tissue of the throat changes after tonsillectomy there can be a subtle alteration in voice quality. In a mature adult, pain following tonsillectomy can be unpleasant, but is reasonably well controlled with prescription medication.[79]

Genioglossus Tongue Advancement procedure is done through the mouth through an incision below the gingiva in front of the mandibular anterior teeth. After creating a small rectangular bone window, the tendons that attach the tongue to the jaw are pulled forward on a small bone fragment. This produces a larger space between the back of the tongue and the throat thereby creating a wider airway. Complications resulting from this procedure are very uncommon. There is minimal if any alteration in facial appearance and many patients have not complained about this issue. This operation is often performed in tandem with at least one other procedure such as the UPPP or hyoid suspension.[78]

Distraction osteogenesis is now a viable treatment option for adults and children with unilateral or bilateral mandibular and maxillary hypoplasia. It is also helpful in patients with severe obstructive sleep apnea who are morbidly obese.[79]

Many patients with OSA have abnormalities in facial structure on cephalometry[91] and it has been suggested that correction of such factors by maxillofacial surgery will lead to cure in sleep apnea. However, the correlation of mechanical characteristics of the pharyngeal airway with cephalometry is only indirect and, moreover, such surgery is expensive, requiring several operative procedures. Virtually all cases have been reported by one group, and therefore efficacy will need to be shown in wider clinical trials using the expertise of several surgeons.[92]

Conclusion:-

There is a wide range of clinical disorders associated with OSA which affect the decision to treat patients. However, in some cases the link between OSA and these clinical disorders has not been proved convincingly. Studies clarifying these issues will allow better selection of patients requiring treatment. Nasal CPAP is the standard treatment for OSA and tracheostomy should only be considered in patients truly unable to tolerate CPAP.

Considering that obstructive sleep apnea greatly increases patients chances of heart attack, stroke and early death, dentist might be in a critical position to screen patients, refer patients and treat patients and assume a primary role in saving lives.

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