3. LITERATURE REVIEW

It is generally believed that the most important advancement in the history of sleep medicine is the serendipitous discovery of sleep apnea in 1965 when Sleep medicine clinicians and researchers conducting continuous recording in subjects during nocturnal sleep reported long periods of apneic episodes, cyanosis, muscle relaxation with sudden flattening of EEG and slowing of alpha rhythm in certain specific group of patients [19]. These signs were exclusively noted in patients with Pickwickian Syndrome (Obesity Hypoventilation Syndrome). Later this condition came to be known as ‘Obstructive Sleep Apnea’.

During the initial years, obstructive sleep apnea (OSA) was considered to be a simple, intermittent closure of the upper airway; and therefore the treatments focused mainly on elimination or reduction of airway obstruction, without much regard to the three major factors – 1) dynamic relationship between muscles of upper airway and respiratory muscles; 2) mechanism of upper airway collapse during sleep; 3) region or zone of upper airway obstruction in each individual. Prior to the 1980s, the only reliable method in practice for management of OSA was tracheostomy.

The introduction of continuous positive airway pressure (CPAP) therapy using a nasal mask in 1981 was another important development that influenced a change in approach in management of OSA [20]. Following this, understanding of the upper airway dynamics during sleep and causes for OSA has progressed significantly.

Now it is established that OSA is a condition that occurs due a dynamic interaction between the respiratory muscles controlled by autonomic nervous system and upper airway muscles controlled by voluntary mechanism, the influence of sleep on both these systems and the resulting constriction of upper airway. It is also understood now that upper airway obstruction can occur at various levels depending on the interaction between these two systems and influences from various other factors like mucosal stickiness, position of the subject, muscle activity and size of
various muscles like tongue, tensor palate, etc. Despite of this clear understanding, the currently available modalities for managing OSA mild category are basically Mandibular advancement and tongue repositioning devices which act only at the retro-glossal level of obstruction. Such appliances may not be pertinent for the more common type- retro-palatal level of obstruction.

In order to affirm and assign the basic objectives for this research, a detailed systematic review of the available literature was done on the topics related to OSA – pathogenesis; mechanism of upper airway obstruction; modalities of management; oral appliances for OSA, with consultation of relevant cross references. The review of the literature is enumerated briefly in this section below.

Jung and Kuhlo (1965) [19] reviewed and investigated the various abnormal sleep disturbance syndromes with continuous recordings during nocturnal sleep. Special reference was made to Pickwickian Syndrome (Obesity Hypoventilation Syndrome). They stated that night sleep in Pickwickian patients showed long periods of apneic episodes, cyanosis, muscle relaxation with sudden flattening of EEG and slowing of alpha rhythm. Such marked sleep disturbances were first reported in Pickwickian patients by these authors. Later this condition came to be known as ‘Obstructive Sleep Apnea’.

Sullivan et al (1981) [20] reported that Obstructive sleep apnea can be effectively treated by means of positive airway pressure through the nares. Five patients with severe obstructive sleep apnea were treated by the authors with continuous positive airway pressure (CPAP) applied via a comfortable nose mask through the nares. It was stated that low levels of pressure (range 4.5-10 cm H2O) completely prevented upper airway occlusion during sleep in each patient and allowed an entire night of uninterrupted sleep. The authors hence stated that continuous positive airway pressure applied in this manner provides a pneumatic splint for the nasopharyngeal airway and is a safe, simple treatment for the obstructive sleep apnoea syndrome.
Bland et al (1969) [21] reported four cases of Cor pulmonale secondary to alveolar hypoventilation from chronic upper airway obstruction (enlarged tonsils and adenoids). It was suggested from the findings that, in addition to the chronically obstructed airway, a racial predilection, a defect of the central nervous system causing abnormal respiratory control, and a hyperreactive pulmonary vascular bed must also be present for the development of this unusual syndrome.

Holmberg and Aronson (1979) [22] reported a study that was carried out in an attempt to clarify the value of lateral skull and frontal radiographs as a means of evaluating nasal respiratory function. The study material consisted of 162 children between the ages of 6 and 12 years, with a gender distribution of 40 percent female and 60 percent male. The capacity of the nasal airway was both measured and subjectively evaluated, using lateral skull radiographs of twenty eight children between the ages of 8 and 12 years without adenoid vegetations at the posterior nasopharyngeal wall. The size of the adenoids was measured and evaluated in a similar fashion. In addition, they were graded clinically by posterior rhinoscopy. The nasal airflow was measured. In this way, simultaneous recordings of the airflow velocity and pressure gradient between the nasopharynx and nostrils were obtained. Subsequent correlation analyses gave the following results - a significant relationship between the size of the adenoids as measured on lateral skull radiographs and assessed clinically; a negative relationship between the size of the adenoids as measured on lateral skull radiographs and the nasal airflow; a significant relationship between the capacity of the nasal airway as measured on frontal radiographs and the nasal airflow; a reasonable assessment of the nasal airflow by subjective evaluation of airway capacity from frontal radiographs. It was concluded from these findings that lateral and frontal skull radiographs provide a satisfactory means of evaluating the dimensions of the nasopharynx and the capacity of the nasal airway, respectively.

William et al (1981) [23] reported three cases of hypersomnolence, snoring and documented sleep apnea. All three patients were stated to be profoundly myxedematous, both clinically and biochemically. Polygraphic studies during sleep documented the presence of repetitive episodes of obstructive sleep apnea in all three patients. These were accompanied by arterial oxygen desaturation. After becoming
euthyroid following the administration of the l-thyroxine all patients underwent a repeat evaluation in the sleep laboratory. This study revealed nearly complete resolution of obstructive sleep apnea in all patients. In addition, several sleep parameters showed marked improvement. It was stated from the data obtained that the presence of profound daytime sleepiness in hypothyroid patients could be indicative of a potentially lethal complication of myxedema, obstructive sleep apnea.

Perry et al (1981) [24] did a study to determine the relative selectivity of surface electrodes placed over the gastrocnemius and soleus muscles in the standard manner by comparing the electromyogram from these electrodes to the electromyogram from wire electrodes inserted into the soleus, gastrocnemius, and tibialis posterior muscles. Muscle activity was elicited in 11 normal subjects by performing six manual muscle tests. Three of the tests followed the standard technique and three were modifications designed to provide better differentiation of muscle action. All electromyographic data were quantified by computer integration and normalized to cancel out sampling inconsistency. None of the muscle tests totally restricted activity to the designated muscle, but the tests did determine which was the strongest participant. During the standard specific gastrocnemius and soleus muscle tests, the corresponding surface electrode provided a lower electromyogram than did the matching wire electrode; that is, the surface-to-wire-electrode ratio was less than one (gastrocnemius = 0.74, soleus = 0.58). This ratio was greater than one when the electromyogram was being recorded from one muscle while testing another muscle (gastrocnemius = 1.55, soleus = 1.92). The mathematical model relating surface-electrode values to the wire-electrode data from all three muscles identified 60 percent of the surface gastrocnemius electrode electromyogram as arising from that muscle, while only 36 percent of the soleus surface electrode data related to the activity of the soleus. It was thus concluded by the author that surface electrodes represent muscle group activity. If all muscles in reasonable proximity have a common function, such as plantar flexion, the information is a valid indicator. When antagonistic muscles are adjacent (such as the tibialis anterior and peroneus longus) surface electrodes can produce erroneous information. The electrodes would fail to differentiate by changes in amplitude which muscle was active. An EMG of lower muscular effort could not be separated from contamination by other muscles.
Doughlas et al (1982) [25] evaluated the respiratory volumes and timing in 19 healthy adults during wakefulness and sleep. It was observed that minute ventilation was significantly less (p < 0.05) in all stages of sleep than when the subject was awake (7.66 ± 0.34(SEM) l/min), the level in rapid-eye-movement (REM) sleep (6.46 ± 0.29 l/min) being significantly lower than in non-REM sleep (7.18 ± 0.39 l/min). The breathing pattern during all stages of sleep was noted to be significantly more rapid and shallow than during wakefulness, tidal volume in REM sleep being reduced to 73% of the level during wakefulness. Mean inspiratory flow rate (VT/Ti), an index of inspiratory drive, was significantly lower in REM sleep than during wakefulness or non-REM sleep. Thus it was stated that ventilation falls during sleep, the greatest reduction occurring during REM sleep, when there is a parallel reduction in inspiratory drive. It was also stated that similar changes in ventilation may contribute to the REM-associated hypoxaemia observed in normal subjects and in patients with chronic obstructive pulmonary disease.

David W and Devadatta (1984) [26] reported a research manuscript which was designed for the purpose of determining whether functional residual capacity (FRC) of lung does decrease and to what extent it decreases in normal humans during sleep. Using helium dilution in a closed system the authors measured FRC in 10 healthy males during wakefulness, stage 2, stages 3–4, and rapid-eye-movement (REM) sleep. It was concluded from the study that although the amount of the decrease in FRC identified during sleep was surely not large enough to impair ventilation distribution in normal humans, this degree of decrease might contribute to the hypoxemia seen in patients with severe airflow limitation.

Krieger et al (1985) [27] reported a study where 57 patients with suspected sleep apnea syndrome (SAS) underwent conventional spirometry, assessment of flow-volume curves, ENT examination, and polysomnography. It was observed that thirty patients had an obstructive SAS, four patients a central SAS, and 23 patients no SAS. Signs of upper airway fluttering (the sawtooth sign) were present in 61 percent of the patients with obstructive SAS and in 46 percent of the patients without obstructive SAS (central SAS or no SAS). Signs of extrathoracic upper airway obstruction (FEF50/FIF50 >1) were present in 67 percent of the patients with obstructive SAS and in 71 percent of the patients without obstructive SAS. From these results it was
inferred that upper airway abnormalities, as reflected by abnormal flow volume curves, are not always associated with obstructive SAS; they favor the hypothesis of a central component in the mechanism of upper airway occlusion during sleep.

David W (1986) [28] reported a study that was intended to determine whether the site of physiological narrowing within the upper airway was uniform or differed among patients with obstructive sleep apnea. Inspiratory pressures were measured with an esophageal balloon catheter and three catheters located at different sites along the upper airway: 1) supralaryngeal airway, 2) oropharynx, and 3) nasopharynx. Peak inspiratory pressure differences between catheters allowed assessment of pressure gradients across three airway segments: 1) lungs-larynx-retroepiglottal airway (esophageal-supralaryngeal pressure), 2) hypopharynx (supralaryngeal-oropharynx pressure), and 3) transpalatal airway (oropharynx-nasopharynx pressure). In five patients, hypopharyngeal obstruction was present, and in four patients no hypopharyngeal obstruction existed. In these four patients the site of obstruction was located at the level of the palate. In a given subject, the site of obstruction was the same during repeated measurements. The presence or absence of hypopharyngeal narrowing during sleep was not predictable from gradients measured across different segments of the upper airway during wakefulness. It was concluded based on the inferences that the site of physiological upper airway obstruction varies among patients with obstructive sleep apnea and is not predictable from pressure measured during wakefulness and henceforth it was speculated by the author that uvulopalatopharyngoplasty may not relieve obstructive apneas in patients with hypopharyngeal obstruction.

Stephen et al (1986) [29] did a Cine CT study to evaluate eight adult patients suspected of nonfixed upper airway obstruction. A method was developed by the authors for such imaging, which provided 10 images at each of 12 contiguous levels, extending from the soft palate through the extrathoracic trachea. These gave a dynamic view of the airway during one full respiratory cycle. Results were compared with similar studies in 10 normal volunteers. Seven of the patients showed intermittent obstruction on cine CT, while the eighth was judged normal. The pathology demonstrated included chondromalacia, laryngeal spasm, and polychondritis. Four patients were evaluated for sleep apnea and showed dynamic
abnormalities of the airway, although they were studied awake and asymptomatic, during normal quiet respiration. All were subsequently shown to have severe sleep apnea. It was concluded that cine CT has the potential to provide information quickly and noninvasively on upper airway dynamics and has certain definite advantages over conventional studies.

Suratt et al (1986) [30] conducted a study to investigate whether intranasal obstruction in humans produces predominantly central or obstructive apnea. To analyze this, the authors studied eight normal men by having them sleep in random order with their nose open or occluded with petrolatum gauze. Esophageal pressure was measured to detect respiratory effort, and standard techniques were used to monitor and score the stages of sleep. It was observed that intranasal occlusion increased both the number of apneas plus hypopneas per hour of sleep and the minutes of obstructive events per hour of sleep (p<0.05). The minutes of central events per hour of sleep also increased significantly but not to the degree that occurred with obstructive events. Nasal obstruction produced no immediate changes in pulmonary function. The subject with the highest resistance measured through the mouth with the pulse flow method had the most apneas following nasal occlusion. It was concluded that intranasal obstruction produces predominantly obstructive apneas and hypopneas during sleep.

Popper et al (1986) [31] did endoscopic analysis of the pharyngeal airway during treatment of Obstructive sleep apnea with nasal continuous positive airway pressure with a Pneumatic Splint. It was observed from this analysis that the obstruction to airflow in obstructive sleep apnea occurred at the level of the pharynx and that this obstruction can be successfully overcome with the use of nasal CPAP, which acts as a "pneumatic splint."

Carlos M. et al (1987) [32] reported a manuscript with case presentation of obstructive sleep apnea in an edentulous patient treated successfully with the use of orthognathic surgery. Through the manuscript, the authors stated the cephalometric method of analyzing the pre and post-surgical data. The authors also highlighted that obstructive sleep apnea is one disorder in which the proper identification of mandibular skeletal malocclusion is a concomitant factor can suggest a proper route to
follow for its possible correction and perhaps improve the surgical patient’s quality of life.

Stein et al (1987) [33] reported a study which was conducted to evaluate the upper airway in eight patients with obstructive sleep apnea by using a rapid sequential CT scanner (Imatron 0.100). Four patients also had simultaneous polysomnograms to determine the onset of sleep and apnea. The upper airway was scanned while the patient was awake (eight patients), asleep (four patients), and asleep and apneic (eight patients). Measurements of the cross-sectional area of the upper airway were correlated with the findings on sleep studies in four patients. During the awake state the airway was narrowed and showed increased collapsibility in all eight patients. Five of the eight patients had cross-sectional areas of ≤ 4 mm² at one or more sites at some time during the respiratory cycle while awake. During apnea all patients had obstruction at the uvula and oropharynx, but the length of the obstruction varied from one patient to another. In three of the eight patients the obstruction extended inferiorly to the hypopharynx. It was concluded that cine CT can be used to objectively evaluate patients with sleep apnea and may demonstrate the need to modify surgical treatment.

Parisi (1987) [34] stated a case report of a patient who had undergone bilateral carotid body resection five years earlier for palliation of chronic airflow obstruction who was also found to have severe obstructive sleep apnea. The case was stated to have hypercapnic respiratory failure, which improved after tracheostomy. A physiologic mechanism was proposed to explain this association.

Riley et al (1987) [35] documented a case report of two patients who underwent posterior repositioning of the mandible for prognathism and subsequently developed OSAS is presented. It was affirmed through the case reports that since OSAS can occur postoperatively as illustrated by the cases, it is important to question patients regarding sleep habits (snoring, observed apnea during sleep, and daytime sleepiness) prior to and following mandibular retropositioning procedures in an attempt to identify those patients potentially at risk for OSAS and its associated neurologic and cardiopulmonary consequences.
A. Leiberman et al (1988) [36] reported a manuscript presenting the clinical findings, diagnosis and treatment of young infants, less than 18 months of age, with Obstructive Sleep Apnea. Through the clinical report he suggested that surgical treatment resulted in the relief of the upper airway obstruction and improvement of the clinical condition.

Chaban R, Cole P and Hoffstein V (1988) [37] reportedly developed a technique to determine the site of upper airway obstruction in patients with idiopathic obstructive sleep apnea (OSA). This technique was based on the analysis of inspiratory airflow pressures at various levels of the pharyngeal airway during sleep. Pharyngeal pressure was measured by a moveable Millar catheter pressure transducer. The catheter's position in the airway was localized radiographically. Ten patients with OSA were tested: five patients were found to have upper airway obstruction at the level of the soft palate, and five had upper airway obstruction at the base of the tongue. Authors concluded that measuring airway pressures at multiple sites along the airway is useful in localizing the site of obstruction in patients with OSA, and may have important implications in terms of the patient's response to surgical treatment.

Rubinstein et al (1988) [38] reported a research in which they studied 10 overweight male patients with severe obstructive sleep apnea and low-normal pharyngeal collapsibility to determine the site of upper airway pathology in these patients. They stated that all 10 patients exhibited paradoxical inspiratory narrowing of the glottis during quiet tidal breathing. This phenomenon was not observed by them in a matched group of 10 snoring, non-apneic male controls. Based on their findings they concluded that paradoxical glottic narrowing may be a contributing factor in the pathogenesis of upper airway obstruction in patients with severe obstructive sleep apnea who have low-normal pharyngeal collapsibility.

Paul E Bonham et al (1988) [39] reported a study which they conducted using cephalometrics and overnight polysomnographic monitoring to analyze the effects of a modified functional appliance on airway, sleep, and respiratory variables in patients with obstructive sleep apnea (OSA). Twelve patients without overt anatomic or pathologic evidence of obstruction were selected on the basis of an initial single night
of polysomnographic monitoring, which confirmed the diagnosis of obstructive sleep apnea syndrome. The patients subsequently were fitted with a modified functional appliance designed to securely hold the mandible in an anterior-inferior position. A subsequent overnight polysomnographic study was obtained with each patient wearing the appliance. Lateral cephalometric radiographs with and without the appliance in place were also obtained. The mean vertical and horizontal changes in Mandibular position while wearing the appliance were 8.49 mm and 2.28 mm, respectively. The findings indicated that 10 of the 12 patients had decreases in the rate of complete airway obstructions from a mean of 28.86 to 18.69 events per hour, and in the total apnea index from a mean of 53.81 to 35.99 events per hour. A reduction in the rate of obstructive events was attributed to the effect of the appliance on the oropharyngeal structures. It was inferred from the study that the modified functional appliance proved to be a conservative, successful treatment alternative that could benefit patients with obstructive sleep apnea syndrome.

Aldrich (1988) [40] reviewed data on sleep-related accidents from 70 control subjects and 424 adults with four categories of sleep disorders: sleep apnea, narcolepsy, other disorders of excessive sleepiness, and sleep disorders without excessive sleepiness. Mean sleep latency test was performed and it showed that the results did not differ significantly in patients with accidents and those without. It was inferred that patients with a wide variety of sleep disorders appear to be at increased risk for sleep-related accidents.

Arthur W et al (1989) [41] reported a study which was designed to demonstrate a relationship between Obstructive sleep Apnea and speech by analyzing the perceptual characteristics of the speech quality, and determine the degree to which each of three descriptors of speech abnormality contributed to the perception of speech abnormality. Ten graduate students in speech pathology listened in two 1.5 hour sessions to 252 random speech samples presented on a master tape. There were 81 subjects comprised of 27 sleep apnea patients, 27 matched chronic obstructive pulmonary disease control patients, and 27 matched normal control subjects. Authors concluded from the data that analysis of abnormal speech resonance, articulation and phonation may identify obstructive sleep apnea or may provide insight into its pathology.
Wiegand et al (1989) [42] stated a study which was done to examine the influence of sleep on upper airway collapsibility. Inspiratory upper airway resistance (epiglottis to nares) and genioglossus electromyogram (EMG) were measured in six healthy men before and during inspiratory resistive loading. It was noted that UAR increased significantly from wakefulness to non-rapid-eye-movement (NREM) sleep. It was also noted that resistive load application during wakefulness produced small increments in UAR and during NREM sleep UAR increased dramatically with loading in four subjects. It was conclude from the findings that there is marked variability among normal men in upper airway collapsibility during sleep.

Riley et al (1989) [43] did a study on fifty-five patients with obstructive sleep apnea syndrome (OSAS) and evaluated following inferior mandibular osteotomy with hyoid myotomy and suspension. Patients were objectively examined by Polysomnography before and 6 months following the surgical procedure. It was inferred that thirty-seven patients (67%) had good response from surgery, and 18 patients (33%) were considered non-responders. Lung disease, mandibular deficiency and obesity were stated to be the factors that affected the success of surgical treatment.

Riley (1989) [44] stated about the merits of maxillomandibular advancement surgery in Obstructive Sleep Apnea Syndrome. The author affirmed that maxillofacial surgery has a significant role in the treatment of obstructive sleep apnea syndrome. He also stated that staging the surgical reconstruction is the best way to treat OSAS.

David A et al (1990) [45] reported a study that aimed to analyze whether reduction in Geniohyoid muscular support will lead to increased airway resistance in normal subjects. The authors studied seven normal men throughout a single night of sleep and recorded inspiratory supraglottic airway resistance, Geniohyoid muscle electromyographic (EMG) activity, sleep staging, and ventilatory parameters in these subjects during supine nasal breathing. Mean inspiratory upper airway resistance was significantly (P < 0.01) increased in these subjects during all stages of sleep compared with wakefulness, reaching highest levels during non-rapid-eye-movement (NREM) sleep. It was inferred from the data that sleep-related changes in Geniohyoid muscle activity may influence upper airway resistance in some subjects. It was also stated that
the relationship between Geniohyoid muscle activity and upper airway resistance was complex and varied among subjects, suggesting that other factors must also be considered to explain sleep influences on upper airway patency.

Douglas J et al (1991) [46] reported a study that intended to propose that a sleep-induced decrement in the activity of the tensor palatini (TP) muscle could induce airway narrowing in the area posterior to the soft palate and therefore lead to an increase in upper airway resistance in normal subjects. The authors investigated the TP to determine the influence of sleep on TP muscle activity and the relationship between changing TP activity and upper airway resistance over the entire night and during short sleep-awake transitions. Seven normal male subjects were studied on a single night with wire electrodes placed in both TP muscles. Sleep stage, inspiratory airflow, transpalatal pressure and TP moving time average electromyogram (EMG) were continuously recorded. In addition, in two of the seven subjects the activity (EMG) of both the TP and the genioglossus muscle simultaneously were recorded throughout the night. Upper airway resistance increased progressively from wakefulness through the various non-rapid-eye-movement sleep stages, as has been previously described. The TP EMG did not commonly demonstrate phasic activity during wakefulness or sleep. However, the tonic EMG decreased progressively and significantly (P < 0.05) from wakefulness through the non-rapid-eye-movement sleep stages: awake, 4.6 & 0.3 (SE) arbitrary units; stage 1, 2.6 t 0.3; stage 2, 1.7 * 0.5; stage 3/4, 1.5 k 0.81. It was concluded from the data that 1) tonic TP activity decreases during sleep; 2) this decrease in TP activity correlates with the increase in upper airway resistance during sleep, especially when evaluated over discrete sleep-wake transitions; and 3) this is not a generic upper airway muscle response to sleep in as much as Genioglossus EMG was well maintained during the night.

Rumbach et al (1991) [47] reported a study in which event-related potentials (ERPs) were recorded in 47 patients with obstructive sleep apnea (OSA) syndrome prior to and after 6 weeks of treatment with continuous positive airway pressure (CPAP). It was observed that compared with a control group, the OSA patients showed ERP abnormalities: lengthened P3 latencies and decreased N2-P3 amplitudes. After 6 weeks of CPAP treatment, there was a highly significant improvement in the abnormal ERPs: the P3 and N2 latencies were shortened, but remained longer than in
controls, and the N2-P3 and N1-P2 amplitudes were increased. The authors couldn’t establish any correlations with various sleep variables. Base on this it was stated that ERPs may be used as an electrophysiological marker of brain dysfunction; treatment of OSA with CPAP is probably responsible for functional brain modifications. It was also stated that, possible relationships between the ERP abnormalities and the neuropsychological disorders observed in OSA remain to be established.

Collop et al (1991) [48] documented a study in which the authors investigated 12 men with OSA to see if nasal CPAP used nightly for six weeks would improve their underlying sleep-disordered breathing. They also studied pharyngeal volumes measured using magnetic resonance imaging and a computer-controlled digitizing pad. They stated that patients with more severe OSA had improvement after six weeks; however, they still demonstrated significant OSA. Patients with less severe OSA did not have significant change. The study didn’t show any significant difference in any patient’s awake pharyngeal volumes. The authors concluded that patients with OSA should be encouraged to wear their nasal CPAP machines regularly.

Mahowald et al (1991) [49] emphasized the spectrum of sleep-disordered breathing (SOB), pitfalls in monitoring techniques and caveats in data interpretation. They stated that Otolaryngologists play an invaluable role in the assessment and treatment plans for patients with SOB as they are likely to see a patient population at high risk for SOB. They also stated that the complexities of Polysomnography monitoring and data interpretation in patients with SOB should encourage very close cooperation and active participation with sleep disorders specialists.

Powell et al (1991) [50] reviewed and briefly stated the specific surgical techniques for treatment of anatomic obstruction at the hypopharynx such as mandibular osteotomy with genioglossus advancement (phase I surgery) and the more complex bimaxillary advancement (phase II surgery). They stated that limited mandibular osteotomy does not create more room for the tongue base; however, it does place the tongue on tension; bimaxillary advancement creates additional room for the tongue physically as well as tension. Surgical techniques for each of the selected procedures were outlined by the authors in adequate detail. These include the
indications for each of the selected procedures such as limited anterior sagittal mandibular osteotomy with genioglossus advancement and hyoid myotomy with fascia lata suspension.

E. Sforza and J. Krieger (1992) [51] reported a study in which the modified maintenance of wakefulness test was performed in 58 patients with obstructive sleep apnea (OSA) syndrome before treatment and after long-term home therapy with nasal continuous positive airway pressure (CPAP). Before treatment the patients had shorter mean sleep latency than controls. After treatment, the mean sleep latency increased as compared to baseline but was still shorter than in controls. The incomplete normalization of the mean latency contrasted with the patients' claim that they no longer felt sleepy. The improvement in daytime alertness was significantly correlated with the reduction in sleep fragmentation after CPAP treatment and with the baseline mean sleep latency. The results of the study supported the hypothesis that sleep disruption related to respiratory events plays a role in the pathogenesis of daytime sleepiness.

Mezzanotte et al (1992) [52] reported a study in which they investigated the waking genioglossus (GG) electromyogram (EMG) activity in 11 OSA patients and 14 age-matched controls to determine if GG activity is higher in the awake state in apnea patients than controls. To make this determination, they developed a reproducible methodology whereby true maximal GG EMG could be defined and thus basal activity quantitated as a percentage of this maximal value. Therefore, direct comparisons of basal activity between individuals was possible. The authors that observed apnea patients had significantly greater basal genioglossal activity compared to controls (40.6±5.6% vs. 12.7±1.7% of maximum). This difference persisted when size-matched subsets were compared. It was also stated that the augmented GG activity in apnea patients could be reduced with positive airway pressure. The authors speculated that this neuromuscular compensation present during wakefulness in apnea patients may be lost during sleep leading to airway collapse.

Shellock et al (1992) [53] reported a study in which they evaluated ten patients with clinically proved obstructive sleep apnea using ultrafast spoiled gradient-recalled acquisition in the steady state (GRASS) MR imaging of the
pharyngeal airway to determine the presence of occlusions and/or narrowings. Twelve sequential images were obtained at one midsagittal plane and at eight transverse planes through the pharyngeal airway. The scans were obtained at the rate of one image per 1.04 sec while the patient was breathing quietly. Occlusions or narrowings of the pharyngeal airways were detected on MR images in all patients. It was stated that he site(s) of the occlusions and the site(s) and extent of the narrowings varied. It was also reported that six patients had occlusions and four had narrowings of one or more sites. It was inferred from the study that ultrafast spoiled GRASS MR imaging can be used to evaluate patients with obstructive sleep apnea during tidal breathing and is useful for determining the presence of occlusions and narrowings of the pharyngeal airway.

Koopmann (1993) [54] reported a review of a previous article entitled "Obstructive Sleep Apnea Syndrome: A Surgical Protocol for Dynamic Upper Airway 'Reconstruction" by Drs Riley, Powell, and Guilleminault. Reportedly, in the previous article the authors discussed a major problem with the surgical management of obstructive sleep apnea syndrome (OSAS): the obstruction of the airway due to collapse' of the lateral walls of the hypopharynx and/or obstruction of the supraglottic larynx by the tongue base, either from relaxation of the tongue musculature or excessive tissue in the hypopharynx. They also correctly identified what may be the major cause of failure of the uvulopalatopharyngoplasty (UPPP). Reportedly, in discussing the postoperative findings, the authors satisfactorily compared the presurgical and postsurgical results to the CPAP. The reviewing author concluded stated that electrical stimulation of the nerves of the hypopharynx may obviate the need for invasive procedures such as the mandibular advancement osteotomy.

Yuji Suto et al (1993) [55] reported a study which intended to use ultrafast MR imaging to examine the pharyngeal airway in patients with sleep apnea and to evaluate the usefulness of this technique for localizing the site of obstruction. Fifteen patients with sleep apnea and five healthy volunteers underwent ultrafast MR imaging while awake and during sleep induced with hydroxyzine hydrochloride. Sequential midline sagittal images of the pharynx were obtained and displayed in the cine mode. It was stated that patients with sleep apnea were found to have sites of pharyngeal abnormality that were not present in healthy volunteers. Nine sites of narrowing in
seven patients were detected with the patient awake; 21 sites of obstruction in 13 patients were diagnosed with the patient asleep. It was also noted that six patients showed only one obstruction, and seven had several obstructions: five had obstructions at the velum palatinum and at the oropharynx; one had obstructions at the velum palatinum, oropharynx, and hypopharynx; one had obstructions at the velum palatinum and the hypopharynx. The sites of narrowing during wakefulness and the sites of obstruction during sleep were the same in only four of the patients with pharyngeal airway obstruction. It was concluded from the findings that Ultrafast MR imaging is useful for localizing the sites of pharyngeal airway obstruction in patients with sleep apnea.

R. I. Raine (1993) [56] reviewed on the topic- ‘Investigation and treatment of sleep-related breathing disorders’. The author affirmed that accurate diagnosis with assessment of severity of the condition is mandatory before embarking on treatment. It was also stated that clinical suspicion, physical examination and a clinical score, coupled with nocturnal oximetry are useful screening tests if they indicate the presence of OSA. It was further stated that diurnal and half-night polysomnography are useful in confirming the diagnosis of OSA, but are also inadequate in excluding other diagnoses.

Alexandros et al (1994) [57] reported a study that intended to describe the frequency and severity of sleep apnea in obese patients without a primary sleep complaint and to assess the sleep patterns of obese patients without apnea and compare them with the sleep patterns of non-obese controls. The study was designed as a Prospective case series with historical controls in an obesity and sleep disorders clinic. The Subjects included two hundred obese women and 50 obese men (mean body mass index, 45.3) consecutively referred for treatment of their obesity and 128 controls matched for age and sex. Eight-hour sleep laboratory recording, including electroencephalogram, electro-oculogram, electromyogram and respirations were Analyzed. Subjectively reported sleep-related symptoms and signs were also recorded. It was concluded based on the obtained data that severely or morbidly obese men are at extremely high risk for sleep apnea and should be routinely evaluated in the sleep laboratory for this condition, while for severely or morbidly obese women the physician should include a thorough sleep history in the clinical assessment.
Pracharktam N et al (1994) [58] reported a study that was conducted to determine whether craniofacial morphology differs between subjects with Obstructive sleep apnea syndrome (OSAS) and heavy snorers, and to investigate how change in posture from upright to lying down affects the upper airway passage. Lateral head radiographs of ten persons diagnosed with OSAS and ten snorers matched for age, height and weight without any history of daytime sleepiness, doctor-diagnosed OSAS, and no evidence of significant desaturation on overnight oximetry were obtained in both upright seated and awake supine positions. It was observed that the posterior superior pharyngeal space in both the OSAS and snorers was reduced when changing from upright to supine posture. Significant differences in cranial base alignment, ramus width relative to the middle-crani al fossa, position of the maxilla relative to the cranial base in the seated position were noted between subjects with OSAS and subjects with snoring and less severe apnea. In addition, differences in the posterior superior pharyngeal space, tongue length, tongue to intermaxillary area ratio and hyoid position were demonstrated both in the upright and in the supine positions in the OSAS compared to the snoring group. It was inferred from these results that anatomic factors may predispose some snorers to develop OSAS. Measurements made from awake supine position lateral head radiographs revealed no additional differences between OSAS and snoring subjects when compared to measurements made on radiographs taken in the upright position.

Toshio Sugahara et al (1994) [59] reported a case of Rheumatoid arthritis (RA) with micrognathia caused by destruction of the temporomandibular joint (TMJ) leading to obstructive sleep apnea (OSA). The authors stated performing a bilateral total replacement of the TMJs in a tracheotomized OSA patient with RA using alloplastic TMJ components. They stated that this treatment resulted in dilation of the upper airway, permitted closure of the tracheal stoma and improved masticatory function.

Douglas NJ & Polo O (1994) [60] reported a manuscript in which they gave a brief outlook on pathogenesis of sleep apnea/hypopnea syndrome (SAHS). They stated the effect of sleep on upper airway narrowing. They also discussed briefly on the effects, factors predisposing and clinical effects of SAHS.
Garfinkel D (1995) [61] reported a manuscript that investigated the effect of a controlled-release formulation of melatonin on sleep quality in 12 elderly subjects (aged 76 [SD 8] years) who were receiving various medications for chronic illnesses and who complained of insomnia. In all 12 subjects the peak excretion of the main melatonin metabolite 6-sulphatoxymelatonin during the night was lower than normal and/or delayed in comparison with non-insomniac elderly people. In a randomized, double-blind, crossover study the subjects were treated for 3 weeks with 2 mg per night of controlled-release melatonin and for 3 weeks with placebo, with a week’s washout period. Sleep quality was objectively monitored by wrist actigraphy. Sleep efficiency was significantly greater after melatonin than after placebo (83 [SE 4] vs 75 [3]% p<0.001) and wake time after sleep onset was significantly shorter (49 [14] vs 73 [13] min, p<0.001). Sleep latency decreased, but not significantly (19 [5] vs 33 [7] min, p=0.088). Total sleep time was not affected. The only adverse effects reported were two cases of pruritus, one during melatonin and one during placebo treatment; both resolved spontaneously. It was concluded from these inferences that melatonin deficiency may have an important role in the high frequency of insomnia among elderly people. And that controlled-release melatonin replacement therapy effectively improves sleep quality in this population.

Danuta et al (1995) [62] reported a study designed to investigate the influence of surgical treatment of the Waldeyer’s ring for Obstructive sleep apnea on the morphology of dental arch. 90 patients aged 3 to 14 were included for the study, 60 of them were diagnosed with Obstructive sleep apnea. After the surgical treatment, interviews; Pediatric, Laryngologic and Orthodontic follow-ups; laboratory and gasometric tests; Pulsoxymetry during sleep; Rhinomanometry before and 8 days after surgery; dental arch analysis before and one year post-operatively were performed. It was concluded from the data that early performance of surgery on hypertrophied tonsils greatly influenced the development of dental arch, in children of age 6 to 7 years especially. Authors emphasized that a multi-disciplinary approach between Otorhinolaryngologists and Orthodontists is needed during surgical treatment planning for children of that age.
Vavrina (1995) [63] reported a study which was carried out on 110 children undergoing tonsillectomy or adenotonsillectomy to evaluate the usefulness of computer assisted pulse oximetry (POM) as a screening tool for nocturnal obstructive sleep apnea episodes. Twenty-one healthy age matched children served as a control group. A self-designed software (CAP0 version 1.0) was used to analyse collected oximetric data. It was stated that pre-operatively up to 25% of children showed a characteristic pattern of repeated oxygen desaturations related to partial or complete airway obstruction, which was not seen in the matched group. Thirty-one percent had an oxygen desaturation index (ODI) of more than 2 phases/h, being significantly higher than in the matched group. It was concluded that computer assisted POM is useful in predicting and grading nocturnal obstruction and adds decision making data for the treatment in children suspected of suffering from obstructive sleep apnea.

Tucker (1996) [64] demonstrated a case in which Uvulopalatopharyngoplasty was performed for Obstructive Sleep Apnea (OSA). Following surgery Upper Airway Resistance Syndrome (UARS) was demonstrated in the patient. The differences between OSA and UARS were also reviewed by the author. By means of the case report, the author summarized that Obstructive apneas and hypopneas that are defined by decreases in airflow, continued respiratory effort, and decreases in oxyhemoglobin saturation traditionally are used to determine OSA severity. In UARS, arousals and sleep fragmentation may occur without apnea, hypopnea, or snoring. Physicians treating snoring and OSAS need to be aware of UARS as a possible contributor to persistent excessive daytime sleepiness after surgical treatment of OSAS or snoring.

Stanescu et al (1996) [65] reported a study which investigated whether expiratory flow limitation occurs during sleep in heavy snorers (HS) and in patients with obstructive sleep apnoea (OSA). The authors studied four non-apnoeic, heavy snorers and five OSA patients. Airflow was measured with a Pneumotachograph attached to a tight-fitting mask, and supraglottic pressure with a catheter placed at the supraglottic level. Scoring for flow limitation was achieved by visual inspection of 200 breaths recorded during sleep. About 20% of the respiratory cycles presented isolated inspiratory flow limitation. From the data, approximately the same percentage was observed in heavy snorers and OSA patients. Isolated expiratory flow limitation was less frequently recorded. Coupled inspiratory and expiratory flow limitations
were more numerous, especially in heavy snorers. Authors concluded that both in heavy snorers and obstructive sleep apnoea patients, inspiratory flow limitation is associated with expiratory flow limitation thereby suggesting that upper airway obstruction during sleep is both an inspiratory and expiratory event.

Whitney and Gannon (1996) [66] reported a case who presented to the emergency department with acute delirium. It was further noted that an abnormal respiratory pattern with periods of apnea associated with oxygen desaturation were also present. The observation that the patient had episodes of apnea while sleeping led to the suspicion that this patient had OSA, and formal polysomnography confirmed the diagnosis. Other causes for acute delirium were ruled out. The delirium resolved after the OSA was treated.

Schwartz et al (1996) [67] evaluated seven patients with histories of snoring and OSA in a pilot study to assess the effects of electrical stimulation to the soft palate. Each patient slept with a palatal appliance that delivered a weak electrical stimulus to the soft palate on activation. A 3 milliampere stimulus in the range of 9 to 10 volts was found to be effective in terminating snoring without causing patient arousal. It was noted that the effects of the stimulus on OSA were variable. The results of this study indicated that electrical stimulation of the soft palate may be effective as a treatment for snoring and OSA and warrants further investigation.

Schmitz et al (1996) [68] documented a manuscript in which they discussed about hyoid bone suspension with inferior myotomy as a treatment technique for obstructive sleep apnea. They discussed about the regional anatomy, surgical technique, and materials for suspension of the hyoid bone, along with newer modifications of the procedure. Potential risks and complications are also discussed.

Thornton and Roberts (1996) [69] reviewed the various non-surgical options for management of Obstructive sleep apnea. The authors stated the following- that the practitioner must develop a new paradigm for diagnosis and treatment of this disorder; this paradigm must recognize the available modalities and prescribe a logical sequence for treatment; Oral appliance therapy (OAT) should become a part of this paradigm; most patients seek treatment for their snoring because of social
repercussions and only a few recognize the life-threatening consequences of OSA. They concluded by stating that OAT offers a cost effective, user-friendly method for management of this condition.

Mayer et al (1996) [70] reported a study that aimed to evaluate in a large population of snorers with or without OSA, the relationship between body mass index (BMI), age and upper airway morphology. One hundred and forty patients were referred for assessment of a possible sleep related breathing disorder and had complete polysomnography, cephalometry and upper airway computed tomography. It was noted that for the whole population, OSA patients had more upper airway abnormalities than snorers. When subdivided for BMI and age, however, only lean or younger OSA patients were significantly different from snorers as regards their upper airway anatomy. The shape of the oropharynx and hypopharynx changed significantly with BMI both in OSA patients and snorers, being more spherical in the highest BMI group due mainly to a decrease in the transverse axis. On the other hand, older patients whether snorers or apnoeics, had larger upper airways at all pharyngeal levels than the youngest group of patients. For the total group of patients, upper airway variables explained 26% of the variance in apnoea/hypopnoea index (AHI), whereas in lean or youngest subjects upper airway variables explained, respectively 69 and 55% of the variance in AHI. It was concluded that, in lean or young subjects, upper airway abnormalities explain a major part of the variance in apnoea/hypopnoea index and are likely to play an important physiopathogenic role. The authors also suggested that the shape of the pharyngeal lumen in awake subjects is more dependent on body mass index than on the presence of obstructive sleep apnoea.

Guilleminault et al (1996) [71] evaluated a population of 110 subjects (58 men) diagnosed as having UARS. The study investigated acute systolic and diastolic blood pressure (BP) changes seen during sleep in two different samples. First, six patients from the original subject pool were found to have untreated chronic borderline high BP and were subjected to 48 hours of continuous ambulatory BP monitoring before treatment and another 48 hours of BP monitoring 1 month after the start of nasal continuous positive airway pressure (N-CPAP) treatment. A second protocol investigated seven normotensive subjects drawn from the initial subject pool. Continuous radial artery BP recording was performed during nocturnal sleep with
simultaneous polygraphic recording of sleep/wake variables and respiration. BP changes were studied during periods of increased respiratory efforts and at the time of alpha EEG arousals. Increases in systolic and diastolic BP were noted during the breaths with the greatest inspiratory efforts without significant hypoxemia. A further increase in BP was noted in association with arousals. It was inferred from this study that, in the absence of classic apneas, hypopneas, and repetitive significant drops in oxygen saturation (below 90%), repetitive increases in BP can occur as a result of increased airway resistance during sleep. It was also that, in some patients with both UARS and borderline high BP, high BP can be controlled with treatment of UARS. It was conclude that abnormal upper airway resistance during sleep, often associated with snoring, can play a role in the development of hypertension.

Boudewyns et al (1997) [72] reported a study designed to determine the site(s) of Upper airway (UA) obstruction and the influence of sleep stage on the pattern of obstruction. Twenty eight obstructive sleep apnoea patients underwent UA pressure measurements during polysomnography. Solid-state pressure sensors were located at the nasopharynx, oropharynx, tongue base, hypopharynx and oesophagus and the lower limit of UA obstruction was determined relying on the observed pressure pattern. The site of UA obstruction varied among consecutive apnoeas in all but two patients. The lower limit of UA obstruction was predominantly located at the naso and oropharynx. Rapid eye movement (REM) sleep was associated with a tendency for obstruction to extend towards lower levels of the UA and nasopharyngeal occlusion was significantly less observed during REM compared to oropharyngeal obstruction. It was inferred from the data that Upper airway obstruction involves more than one specific site of the upper airway in the majority of sleep apnoea patients. Obstruction at lower levels of the upper airway is more likely to be observed during rapid eye movement sleep.

Numa et al (1997) [73] reported a manuscript in which they measured arterial blood gases, functional residual capacity (FRC), respiratory system resistance and compliance (Rrs, and Crs) in supine and prone positions in 30 patients under neuromuscular blockade with lung disorders including moderately severe restrictive and obstructive disease and control subjects without significant lung disease. Prone positioning was not associated with a significant increase in FRC in the cohort of 30
patients, nor in any of the subgroups. Although individual patients demonstrated large improvements in oxygenation, a statistically significant (but clinically insignificant) increase in AaPO2 ratio was observed only in the subgroup of patients with obstructive disease. There was no correlation between changes in FRC and changes in AaPO2. A significant improvement in Rrs occurred in the prone position compared to supine in patients with obstructive lung disease. No significant changes in Crs were seen in the prone position. It was concluded from these inferences that prone positioning has no effect on FRC but a significant decrease in Rrs in patients with obstructive lung disease was also observed.

Aboussouan et al (1997) [74] reported a manuscript which focussed on the diagnosis and therapeutic options in OSA with emphasis on their limitations. The modalities discussed included nasal CPAP with titration of pressure to adequate levels, oral appliances, UPPP in selected patients, UPPP with mandibular osteotomy and genioglossus advancement, maxillofacial surgery (with maxillary, mandibular and hyoid advancement) and the choice of last resort, tracheostomy.

Martin et al (1997) [75] reported a study which was designed to test the hypothesis that increasing age and the male sex predispose to upper airway narrowing in normal subjects. The study measured upper airway calibre using acoustic reflection in 60 men and 54 women (median 35, range 16–74 yrs) both seated and supine. All upper airway dimensions, except oropharyngeal junction (OPJ), decreased with increasing age in both men and women while supine. Men had greater changes in airway area at OPJ on lying down. Men had greater body mass indices and larger neck circumferences than women. For any body mass index, neck circumference was larger in men than women. It was concluded from this study that upper airway size decreases with increasing age in both men and women, and that men have greater upper airway collapsibility on lying down at oropharyngeal junction than women.

Gregory et al (1997) [76] reported a study with retrospective case series of pre and post operative polysomnograms (PSG) of pediatric patients with obstructive sleep apnea (OSA), which was done to evaluate the effectiveness of surgical treatment of obstructive sleep apnea in a diverse population of children. It was concluded from the findings of the study that tonsillectomy, adenoidectomy and UPPP are effective in the
treatment of OSA in a diverse group of pediatric patients. Patients with asthma, cerebral palsy, Down syndrome, morbid obesity, and hereditary syndromes all improved significantly with surgical management.

Kuna and Smickley (1997) [77] reported a study in which respiratory-related superior pharyngeal constrictor (SPC) muscle activity was determined in 18 obstructive sleep apnea (OSA) patients during wakefulness and sleep. Hooked-wire electrodes were implanted into the SPC muscle via a nasopharyngoscope. It was observed that during quiet breathing in wakefulness, phasic expiratory SPC-muscle activity was consistently present in six subjects and intermittently present in 12 subjects, particularly following a swallow. The SPC muscle showed two patterns of activation during spontaneous obstructive apneic episodes in non-rapid-eye-movement (NREM) sleep: (1) activation during airway reopening, with or without waning phasic or tonic activation in the first half of the ensuing apnea; and (2) absence of activation during apneas or arousals. Activation on airway reopening following spontaneous apneas usually occurred on inspiration. When the apneic episodes were eliminated by application of nasal continuous positive airway pressure (CPAP), SPC-muscle activity was absent in NREM sleep in 16 subjects, but phasic expiratory activity persisted in two subjects. It was inferred from the results that SPC-muscle activation is similar to that of pharyngeal dilator muscles during spontaneous and induced apneas, and is not necessary to induce upper-airway closure during NREM sleep in OSA subjects.

Ruhle et al (1997) [78] documented a manuscript in which they stated that obstruction of the upper airway may cause arousals resulting in daytime sleepiness and cardiovascular disturbances. They also stated that Upper airway resistance syndrome may easily be overlooked because conventional measurements of oronasal airflow and thoracic and abdominal efforts are not sensitive enough. By measuring esophageal pressure even small disturbances can be detected, but the esophageal gauge may disturb sleep. The authors concluded that other, less invasive methods like measurements of impedance by forced oscillation technique or flattening of the inspiratory flow contour could be valid alternatives in the diagnosis of the upper airway resistance syndrome.
Lorino et al (1998) [79] reported a study that was designed to determine whether oscillatory respiratory resistive impedance at 16 Hz (RFO) might be proposed as an alternative index to Esophageal pressure amplitude (DPes), inspiratory pulmonary resistance (RLI) and inspiratory flow limitation score (FS) are used as indices of upper airway obstruction for the titration of nasal continuous positive airway pressure (nCPAP) in patients with obstructive sleep apnea syndrome (OSAS). Eleven OSAS patients were studied during a night of polysomnography - controlled nCPAP titration. Nasal flow, airway opening and esophageal pressure were continuously measured during nasal breathing, and forced-flow oscillations (FO) were applied for 5 min at each nCPAP level. RFO was obtained by linear regression analysis of respiratory resistive impedance versus frequency. The study demonstrated the applicability of the FO technique in sleeping patients receiving nCPAP, and the reliability of RFO for assessing pulmonary resistance. The authors concluded stating that RFO might be proposed as a quantitative index of airway obstruction for nCPAP titration.

Barbe et al (1998) [80] stated a study which was done to investigate the association between sleep apnea syndrome (SAS) and automobile accidents, and to evaluate potential underlying mechanisms. In this study, the authors prospectively recruited 60 consecutive patients with SAS (apnea–hypopnea index, 58 ±3) and 60 healthy control subjects, matched for sex and age. The number of automobile accidents during the past 3 year was obtained from participants and insurance companies. The authors quantified the degree of daytime sleepiness (Epworth scale), anxiety and depression (Beck tests), and assessed the level of vigilance and driving performance. It was observed that patients had more accidents than control subjects and were more likely to have had more than one accident. The study did not show any correlation between the degree of daytime sleepiness, anxiety, depression, the number of respiratory events, nocturnal hypoxemia, level of vigilance, or driving simulator performance and the risk of automobile accidents among SAS patients. It was concluded through the study that patients with SAS have an increased risk of automobile accidents; none of the clinical or physiological markers commonly used to define disease severity were able to discriminate those patients at higher risk of having an automobile accident.
Lorenz et al (1998) [81] reported a study that intended to introduce an ultrafast MR imaging technique of the pharynx as a diagnostic tool for viewing the mechanism of obstruction in patients with obstructive sleep apnea. Six healthy volunteers and 16 patients with obstructive sleep apnea were examined on a 1.5-T whole-body imager using a circular polarized head coil. Ultrafast two dimensional fast low-angle shot sequences were obtained in mid-sagittal and axial projections during transnasal shallow respiration at rest, during simulation of snoring, and during performance of the Muller maneuver. All patients underwent physical examination, transnasal fiberoptic endoscopy, and polysomnography. It was concluded through the study that ultrafast MR imaging is a reliable non-invasive method for use in the evaluation of obstructive sleep apnea.

Popovic and White (1998) [82] conducted a study which tried to determine the level of awake genioglossus electromyogram (EMGgg) and upper airway resistance in 12 pre- and 12 postmenopausal women under basal conditions and during the application of an inspiratory resistive load. In addition, a subgroup of eight postmenopausal women were studied a second time after 2 weeks of combined estrogen and progesterone replacement in standard doses. It was concluded from the findings of this study that female hormones (possibly progesterone) have a substantial impact on upper airway dilator muscle activity.

Piccirillo et al (1998) [83] reported a study which intended to serve as a demonstration project for a multicenter treatment outcomes research project for patients with obstructive sleep apnea. A clinical-severity staging system was created to control for important differences in the severity of sleep apnea among the enrolled patients. A disease- specific quality-of-life measure was used in this project to measure, from the patient’s perspective, important pretreatment and posttreatment physical, functional, and emotional aspects of obstructive sleep apnea. Adults with apnea indexes greater than 5 who had not previously undergone uvulopalatoplasty were considered for this study. In total 142 patients were enrolled from eight otolaryngology practices. The mean age was 48 years, 112 were men, and 114 were white. The mean pretreatment apnea index was 40.0, and the mean respiratory distress index was 60.5. Seventy-one patients received continuous positive airway pressure, and 48 patients received surgery. Outcomes were assessed from scores on patient-
based general and disease-specific health status measures 4 months after enrolment. The authors stated that the study represented a step forward for the support of future outcomes research projects by organized otolaryngology, in spite of the limitations such as short duration follow-up and limited number of patients undergoing post-treatment polysomnograms which prohibited any analysis of treatment effectiveness.

Waite (1998) [84] reported a manuscript that reviewed the pathophysiologic nature of obstructive sleep apnea (OSA) and its management for the oral and maxillofacial surgeon. The manuscript discussed the basics in upper airway resistance, medical considerations in OSA, specific problems in sleep apnea related to age, pediatric sleep disorders, methods of examining the upper airway, the value of cephalometric analysis, nonsurgical therapy (such as nasal continuous positive airway pressure [CPAP]), surgical treatment planning, uvulopalatopharyngoplasty (UPPP), maxillomandibular advancement (MMA) surgery, and the application of orthodontic treatment. It was concluded that MMA is safe and effective, and it can be considered as an alternative option to CPAP.

Mogayzel et al (1998) [85] reported a study which intended to characterize sleep-disordered breathing in 88 children with achondroplasia aged 1 month to 12.6 years. It was inferred from the study that children with achondroplasia often have sleep-related respiratory disturbances, primarily hypoxemia; The majority do not have significant obstructive or central apnea; however, a substantial minority are severely affected; Tonsillectomy and adenoidectomy decreases the degree of upper airway obstruction in most but not all children with achondroplasia and obstructive sleep apnea; Restrictive lung disease can present at a young age in children with achondroplasia.

Fry et al (1998) [86] reported a study which tried to evaluate unattended full polysomnography (PSG) recorded in the home by the DigiTrace Home Sleep System (DHSS) and to assess the ability to acquire, store and analyze polysomnographic data using the DHSS compared to standard paper PSG. Participants in the study included all adult patients who required standard clinical PSG as part of their clinical evaluation, regardless of suspected diagnosis, except patients requiring video recording for abnormal behaviors. It was concluded that using the DHSS, unattended
full PSG can be performed in the home with reliable and high quality recordings; full PSG can be extended to a larger patient population, because it is no longer limited by the number of beds, and there is a reduction in cost due to elimination of overnight staff and facility cost.

Meissner et al (1998) [87] reported the case of an adult who developed recurrent pulmonary edema as a result of unrecognized chronic upper airway obstruction due to polyarticular juvenile rheumatoid arthritis. The report highlighted the importance of considering upper airway involvement in the differential diagnosis of sedentary patients with arthritic joint disease and breathing difficulties.

Cohen et al (1998) [88] reported a study which was conducted with the purpose to present in detail the types of skeletal osteotomies performed by the authors, focusing on a variety of modifications that have enabled their application in children with OSA. It was concluded from this study that skeletal expansion in conjunction with soft-tissue reduction in the pediatric population permits substantial increase in the volume of both the nasopharynx and oropharynx; Creative use of conventional osteotomies and the application of distraction osteogenesis enable surgeons to apply maxillofacial and craniofacial techniques in treating children with obstructive sleep apnea.

Trudo FJ et al (1998) [89] stated a study which evaluated the state-dependent changes in upper airway caliber using with magnetic resonance imaging (MRI) techniques. 15 normal subjects were studied during wakefulness and sleep. Sleep was facilitated by one night of sleep deprivation prior to MRI. It was concluded by the authors that the lateral pharyngeal walls play an important role in upper airway narrowing during sleep in normal subjects.

Caballero et al (1998) [90] reported a study which intended to evaluate if the caliber of the upper airway, measured by CT, allows to distinguish patients with obstructive sleep apnea syndrome (OSAS) from healthy people. 16 OSAS patients and 39 healthy volunteers were studied. Polysomnography and CT of the upper airways during awake periods were performed in both groups. The area of the nasopharynx, oropharynx, and hypopharynx (in inspiration and expiration), the uvula diameter, and retropharyngeal tissue were evaluated. The simultaneous identification
of the variables that differentiate between control and OSAS groups was determined by a multivariate discriminant model. It was concluded from the results that CT could play an important role in studying the upper airway in patients with OSAS and that the determination of the retropharyngeal tissue by CT could be a useful procedure to evaluate OSAS.

Daniel and Teotimo (1999) [91] reported a study which was designed to compare respiratory nocturnal polysomnography (NPSG) characteristics between matched cohorts of upper airway resistance syndrome (UARS) and obstructive sleep apnea syndrome (OSAS) patients. All patients received 13-channel NPSG, including esophageal pressure (Pes) manometry. By definition, OSAS patients had an apnea-hypopnea index (AHI, number of apneas/hypopneas per hour total sleep time) > 15, and UARS patients had an AHI < 5. Respiratory effort-related arousal (RERA) was defined as the absence of apnea/hypopnea with > 10 s duration of progressive negative Pes, culminating in an arousal or microarousal. UARS patients, by definition, had > 15 RERAs per hour. Fifteen consecutively diagnosed UARS patients were matched with OSAS patients on the basis of body mass index (BMI) and gender. It was concluded from the data obtained that with the exception of AHI, respiratory NPSG parameters were the same for UARS and OSAS patients when BMI and gender were controlled.

Elliott N and Nancy A (1999) [92] reported a manuscript that reviewed the literature highlighting on the upper airway resistance syndrome (UARS). The authors, based on the review stated that UARS is a form of sleep-disordered breathing in which repetitive increases in resistance to airflow within the upper airway lead to brief arousals and daytime somnolence. The review described the chronological progression of general understanding of UARS within the broader context of sleep-disordered breathing. The primary symptom - daytime somnolence, as reported by the authors, result directly from repetitive EEG arousals. The level of negative intrathoracic pressure is the most likely stimulus for arousal, possibly mediated by mechanoreceptors in the upper airway. A general consensus regarding the exact clinical definitions and the physiologic measurement techniques leading to a diagnosis does not exist, although esophageal manometry and pneumotachographic airflow measurements taken during polysomnography are the “gold standard.” Less invasive
diagnostic modalities have been proposed, but none of them have been well-validated according to this review manuscript. Aside from daytime somnolence, hypertension is also reported as an important sequel of this disorder, likely resulting from autonomic and cardiovascular changes induced by increased negative intrathoracic pressure. Nasal continuous positive airway pressure is the most efficacious form of therapy, according to this manuscript, although reportedly low patient compliance may limit its practical application. Palatal tissue reduction by radiofrequency ablation and the use of oral appliances hold promise as safe and effective modalities, but these treatments require further study, as reported in the manuscript.

H Sakakibara et al (1999) [93] reported a study which aimed to comprehensively evaluate the cephalometric features in Japanese patients with obstructive sleep apnoea (OAS) and to elucidate the relationship between cephalometric variables and severity of apnoea. Forty eight cephalometric variables were measured in 37 healthy males and 114 male OSA patients, who were classed into 54 non-obese and 60 obese groups. Diagnostic polysomnography was carried out in all of the OSA patients and in 19 of the normal controls. It was concluded that in obese patients, upper airway soft tissue enlargement may play a more important role in the development of obstructive sleep apnoea, whereas in non-obese patients, bony structure discrepancies may be the dominant contributing factors for obstructive sleep apnoea.

Takuya Watanabe et al (1999) [94] stated a study in which polysomnographic findings and clinical symptoms were investigated in 14 cases of upper airway resistance syndrome. The mean scores of the Epworth sleepiness scale and self-rating depression scale in eight cases were 13.5 and 38.6, respectively. The mean sleep latency of the multiple sleep latency test in four cases was 10.2 min. Seven cases were treated with continuous positive airway pressure (CPAP), and one with hormone replacement therapy. The most common symptom was daytime sleepiness. Five cases had hypertension. CPAP reduced increasing negative esophageal pressure (Pes) and frequency of EEG arousals, and improved hypertension in one case. It was stated that hormone replacement therapy improved increasing negative Pes and clinical symptoms.
Henderson and Strollo (1999) [95] reviewed the various modalities of medical management of Obstructive sleep apnea. They stated that although medical therapies have provided the avenue for the diagnosis and treatment of this common medical condition, it requires active patient participation, to achieve the desired outcomes of improved sleep continuity, daytime functioning and quality of life. They also stated that conservative therapies, such as weight loss and patient positioning; and pharmacological therapies are not effective. The authors stressed that positive pressure therapy has become the treatment of choice for the vast majority of OSA patients and Oral appliances offer an acceptable treatment alternative for select patients. They stated that these mechanical approaches can produce significant decreases in the frequency and severity of sleep-disordered breathing and nocturnal oxyhemoglobin desaturation and these interventions will reduce long-term morbidity and possibly mortality.

Yantis (1999) [96] presented an overview of Obstructive sleep apnea (OSA) with focus on the identification of risk factors and common symptoms of OSA in children. The author highlighted the statement- understanding that children with OSA commonly manifest symptoms that are different than those of adults and that special considerations in the diagnosis of children should be practiced is important. It was also stated that all persons with habitual childhood snoring should be evaluated for the presence of OSA.

Cohen et al (1999) [97] reported a manuscript in which the authors stated the various aggressive surgical treatment protocols for several patients by which all sites of upper airway obstruction were treated simultaneously by a combination of craniofacial skeletal expansion and soft-tissue reduction. The authors concluded stating the efficacy of an aggressive surgical approach to the treatment of OSA in children, avoiding the necessity for tracheostomy or permitting decannulation of permanent T in the majority of cases.

Malhotra et al (2000) [98] assessed the relationship between two dilator muscle electromyograms (EMG) - genioglossus (GG), tensor palatine (TP) Fifteen normal subjects were studied, during wakefulness and stable non-rapid eye movement (NREM) sleep. The GGEMG and TPEDMG were assessed during basal breathing and
during inspiratory resistive loading. There was a strong correlation between stimuli and GGEMG during wakefulness in most subjects. It was concluded from the study that intrapharyngeal pressure may modulate genioglossus activity during wakefulness, with a fall in muscle responsiveness during sleep. The activity of the TP was not clearly influenced by any measured local stimulus either awake or asleep.

Hui et al (2000) [99] reported a manuscript emphasizing on treatment modalities for Obstructive sleep apnea. The authors stated that Obstructive sleep apnoea syndrome is a common but under recognized disorder with associated substantial morbidity and mortality. Excessive daytime sleepiness caused by the disorder leads to poor work performance and increases the risk of an individual having an automobile accident, as reported by the authors. The main objective of treatment for sleep apnoea, according to the authors, is the relief of disabling daytime sleepiness and the improvement of quality of life. Conservative measures such as weight reduction and the avoidance of alcohol should be initiated when appropriate. Nasal continuous positive airway pressure devices, as stated by the authors, have remained the standard treatment since it was first introduced in 1981. The authors summarized stating that oral appliances provide an alternative treatment choice in mild-to-moderate cases, whereas surgery is useful in selected cases.

Zallek and Chervin (2000) [100] stated a case report of a 60-year-old man with cluster headaches (CH), refractory to many different medications for 9 years, found to have obstructive sleep apnea (OSA). Treatment with nasal continuous positive airway pressure (CPAP) was associated with substantial reductions in the frequency and severity of cluster headaches. It was inferred through these observations that obstructive sleep apnea may trigger CH during susceptible periods.

Don et al (2000) [101] reported a study that intended to examine the mechanics of infantile obstructive sleep apnea (OSA) by measuring airway pressures using a triple-lumen catheter in 19 infants with concurrent overnight polysomnography. Pressure measurements from a triple-lumen catheter indicated that the soft palate is the most common site of obstruction in infants with clinically significant OSA, suggesting that palatal dysfunction contributes to the disease in infants. It was also noted that during spontaneous airway obstruction of infants less
than 1 yr of age, the first respiratory effort had reduced amplitude compared with baseline, but unlike preterm infants the amplitude of subsequent respiratory efforts returned to and then exceeded baseline. Termination of apnea was associated with greater amplitude of respiratory efforts in the treatment group compared with infants with few events. However, apnea termination was accompanied by arousal in the minority of events and there was no evidence of increased amplitude of respiratory efforts between the discrete events.

Caputo et al (2001) [102] documented a manuscript that was aimed at analyzing the potential for prenatal magnetic resonance imaging to predict pulmonary hypoplasia in congenital diaphragmatic hernia. A Prospective observational study was carried out with thirteen cases of congenital diaphragmatic hernia without associated anomalies and 74 controls as participants. Measurements by magnetic resonance imaging of fetal lung volume were achieved. It was conclusion by analyzing the data obtained that in isolated congenital diaphragmatic hernia, fetal lung volume measurement by magnetic resonance imaging is a potential predictor of pulmonary hypoplasia and postnatal outcome.

Vahid (2001) [103] reported a study that aimed to examine the effect of obesity on pharyngeal size in both men and women and to determine the role of upper airway dimensions in the expression of sleep-disordered breathing (SDB) and its relationship to gender. 78 male patients and 52 female patients were included for the study and they underwent in-laboratory polysomnography with measurement of upper airway size using the acoustic reflectance method. It was concluded from the study that the static properties of upper airway in awake men but not women correlate with the severity of sleep apnea. This suggested inherent structural and functional differences in upper airway during sleep between men and women with more favourable airway mechanics in women.

James et al (2001) [104] evaluated the effect of gender on two measures of upper airway mechanics and function: pharyngeal resistance and critical closing pressure (Pcrit). Pharyngeal resistance at two points, fixed flow of 0.2 l/s (RL) and peak flow (Rpk), were measured in 33 men and 27 women without significant sleep-disordered breathing. Pcrit was measured in eight men and eight women without
sleep-disordered breathing. From the findings the authors concluded that there are no significant differences in collapsibility between men and women.

Sanders (2001) [105] reported a study which was designed to test the hypothesis that patients with obstructive sleep apnea (OSA) have impairment of endothelial cell function. Eight men with OSA and nine overweight control subjects without OSA were included for the study. Endothelial cell function was tested by measuring the change in forearm blood flow (FBF) with infusion of acetylcholine (Ach), sodium nitroprusside (SNP) and verapamil (VER). It was concluded that there is abnormal endothelial mediated resistance vessel vasodilatation in OSA patients.

Salah et al (2001) [106] reported a study which aimed to test the hypothesis that episodic hypoxic exposure activates long-term facilitation (LTF) in OSA patients during stable non-rapid eye movement (NREM) sleep. The authors induced repetitive hypoxia in OSA patients using nasal continuous positive airway pressure (CPAP) to maintain upper airway patency and stable sleep state for the duration of the experiments. It was concluded that that episodic hypoxia during sleep in OSA patients elicits LTF of ventilator motor output. It was also stated that the thoracic pump muscles do not demonstrate LTF and nasal CPAP did not alter the ability of OSA patients to elicit LTF at the thoracic pump muscle.

Ciscar et al (2001) [107] reported a study in which Ultrafast magnetic resonance imaging (one image per 0.8 s) was used to study the upper airway and surrounding soft tissue in 17 patients with OSA during wakefulness and sleep, and in eight healthy subjects whilst awake. The major findings of this investigation in the 25 subjects were as follows: 1) the velopharynx (VP) was smaller in apnoeic patients, only during part of the respiratory cycle; 2) the variation in VP area during the respiratory cycle was greater in apnoeic patients than in controls, particularly during sleep, suggesting an increased compliance of the VP in these patients; 3) VP narrowing was similar in the lateral and anterior-posterior dimensions, both in controls and apnoeic patients while awake; apnoeic patients during sleep have a more circular VP upon reaching the minimum area; 4) there was an inverse relationship between dimensions of the lateral pharyngeal walls and airway area, probably indicating that lateral walls are passively compressed or stretched as a result of
changes in the airway calibre; and 5) soft palate and parapharyngeal fatpads were larger in apnoeic patients, although their role in the genesis of OSA is uncertain. It was concluded that changes in the velopharynx area and diameter during the respiratory cycle are greater in apnoeic patients than in normal subjects, particularly during sleep. This suggested that apnoeic patients have a more collapsible velopharynx, this being the main mechanism of obstruction.

Goldberg (2002) [108] stated that Obstructive sleep apnea (OSA) is a common disease in the United States, with a 4% prevalence in men and a 2% prevalence in women. Three principal options are available to treat patients with OSA: behavioral modifications, devices that can be worn, and surgical options. All causes of sleepiness and physiologic and anatomic airway obstruction should be identified initially, including nonobstructive causes for sleep disturbance. A treatment plan can be individualized based on patient preference, anatomy, and severity of apnea. He stated that surgical treatment for obstructive sleep apnea can achieve a good result and resolution of symptoms in a high percentage of patients who are appropriately selected and who will follow the surgical treatment plan to its conclusion.

Gold et al (2002) [109] reported a study that intended to compare upper airway collapsibility during sleep between patients with upper airway resistance syndrome (UARS), normal subjects, and patients with Obstructive sleep apnea/hypopnea syndrome (OSA/H). One hundred six adult patients with sleep-disordered breathing and 12 adult subjects without habitual snoring or daytime sleepiness and with an apnea/hypopnea index (AHI) < 5/h were evaluated. It was concluded from the study that UARS is a syndrome of increased upper airway collapsibility during sleep. The upper airway collapsibility during sleep of patients with UARS is intermediate between that of normal subjects and that of patients with mild-to moderate OSA/H.

Feldman and Quan (2002) [110] reported a case of Obstructive Sleep Apnea (OSA) found to be caused by tonsillar lymphoma presenting as asymmetric tonsillar hypertrophy. The authors stated that tonsillar lymphoma is rare, but can present as hypertrophied tonsils and/or adenopathy and lead to the development of OSA. The
report emphasized the importance of a thorough upper airway examination of all patients undergoing evaluation for OSA.

Zucconi (2002) [111] evaluated whether patients with obstructive sleep apnea syndrome have indicators of increased risk factor for atherosclerosis in the carotid arteries. The study included male patients with severe OSA, with normal neurological examination and normal computed tomography scan, compared to age matched subjects without OSA chosen for similar co-morbidity and vascular risk factors. It was concluded that increased carotid wall thickness, considered a valid marker of the risk of stroke in middle-aged subjects, occurred in severe OSA patients much more than controls. Thus it was inferred that the OSA patients may be at greater risk for cerebrovascular diseases, regardless of the association with other vascular risk factors.

Sanders (2002) [112] reported a study which was conducted to determine the effects of 6 weeks treatment with continuous positive airway pressure (CPAP) on objective sleepiness, quality of life, cognitive function and systemic arterial blood pressure in patients with a severely elevated Apnea / Hypopnea Index (AHI more than 30) who are not subjectively sleepy. The investigators concluded that the results of this study do not support the initiation of CPAP therapy in Obstructive Sleep Apnea/Hypopnea (OSA/H) patients who are not complaining of sleepiness.

Roland et al (2002) [113] reported a study which intended to examine links between obstructive sleep apnea (OSA), insulin resistance, and dyslipidemia. Obese children who snored underwent polysomnography and metabolic studies. It was concluded that the severity of OSA correlated with fasting insulin levels, independent of BMI. Insulin levels may be further elevated as a consequence of OSA in obese children.

Aboudara (2003) [114] investigated how well lateral cephalometric head films depict three-dimensional upper airway structures. Subjects included were 11 normal adolescent children, of 7–16 years old. Airway information over the same anatomic area in the nasopharynx was compared between lateral cephalometric head films and three dimensional cone beam computed tomography (CT) scans. It was concluded
that intra-subject proportion of airway volume to area showed moderate variability. CT airway volume showed more variability than corresponding head film airway area.

Oliven et al (2003) [115] analyzed the upper airway response to electrical stimulation of genioglossus in Obstructive Sleep Apnea Patients. Airflow was measured at multiple levels of nasal pressure, and upper airway collapsibility was defined by the nasal pressure below which airflow ceased. The findings of the study showed that responses in apnea severity to Hypoglossal nerve-electrical stimulation can be predicted by characterizing the patient’s baseline pressure-flow relationships and response to Genioglossus electrical stimulation.

Schwartz et al (2003) [116] reported an editorial manuscript emphasizing the role of Upper airway surface tension as a significant cause of airflow obstruction during sleep. They affirmed that measurements of upper airway surface tension might guide clinicians in deploying surfactant therapy and might be used as a marker for mucosal injury and sensory receptor dysfunction in sleep disordered breathing. They stated that further work research is required to correlate surface tension in upper airway lining fluid with altered structural changes and sensory-neural defects in upper airway control.

Beth A Malow et al (2003) [117] reported a study that intended to determine the effect of treating obstructive sleep apnea (OSA) on seizure frequency in adults and children with epilepsy in a prospective study. Adult patients and the parents of pediatric patients seen in the University of Michigan Epilepsy and Pediatric Neurology Clinics were given validated questionnaires. Thirteen adults (aged 20–56) and 5 children (aged 14–17) were selected for Polysomnography (PSG) based on frequency of seizures and risk for OSA. Seizure frequency was compared during 8-week baseline and treatment phases and AED levels were done to document stability in medication levels. It was concluded from the study that treatment of OSA in patients with epilepsy may improve seizure control.

Kao et al (2003) [118] reviewed the overall efficacy of a surgical methodology based on localizing the level of anatomic obstruction for each patient and surgical correction of the nasal, oropharyngeal, or hypopharyngeal obstruction. Forty-two
patients with a respiratory disturbance index (RDI) greater than 15 were included in the study. Surgery involved at least 2 levels of obstruction usually performed in 2 stages. It was concluded that the use of an anatomically based methodology in approaching patients with OSA seemed to offer a higher efficacy than a single procedure as reported in the literature.

Profant et al (2003) [119] examined the effect of continuous positive airway pressure (CPAP) treatment on quality of life (QOL) in patients with obstructive sleep apnea. Thirty-nine patients with sleep apnea were studied. Health-related quality of life was measured (HRQL) with the use of the Medical Outcomes Survey (MOS) instrument, before and after patients were randomized to receive either 1 week of CPAP or placebo-CPAP (CPAP administered at ineffective pressure). It was concluded that CPAP treatment does appear to improve several aspects of HRQL, however this improvement may reflect a nonspecific response (ie, placebo) because comparable improvements were observed in both the active treatment group and the placebo treatment group.

Carden and Malhotra (2003) [120] stated that OSA pathogenesis is related to three general components: pharyngeal anatomy, pharyngeal muscle activity, and ventilatory control stability. They also stated that for unclear reasons, men have increased risk of sleep apnea compared with women. In theory, men could be predisposed to sleep apnea based on anatomical compromise, impaired pharyngeal muscle function, ventilator control instability and/or a combination of factors. The authors also stated that despite these assumptions, the mechanism(s) underlying the male predisposition to OSA is still incompletely understood. It was further stated by the authors that the factors that may be playing a role included pharyngeal anatomy, pharyngeal muscle activity, hormonal influences and ventilatory control stability.

Richard (2003) [121] reported a study in which the response of the genioglossus to brief nasal negative pressure applications (NPAs) in early inspiration was compared between OSA patients and an age-matched group of normal subjects. It was concluded from the findings of the study that the response of the genioglossus to NPA during wakefulness is not impaired in OSA patients compared with normal subjects and is greater at low suction pressures.
McCowen and Malhotra (2003) [122] reported a study which intended to determine whether or not men with obstructive sleep apnea (OSA) have altered nocturnal luteinizing hormone (LH) and testosterone secretion compared with controls. Ten men with a prior diagnosis of severe OSA and five healthy control men were included in the study. It was inferred from the study that reduced pituitary stimulation of testicular testosterone secretion occurs in sleep apnea. Furthermore, it was also concluded that severity of sleep apnea is inversely correlated with pituitary-gonadal hormone secretion.

Stanchina et al (2003) [123] investigated the isolated effects of lung-volume changes on pharyngeal collapsibility and mechanics and genioglossus muscle activation during stable non-rapid eye movement sleep. It was concluded from the study that upper-airway muscles respond to changes in lung volumes but not adequately to prevent increased collapsibility. It was inferred from the results that lung volume has an important influence on pharyngeal patency during non-rapid eye movement sleep in normal individuals.

Levy and Pepin (2003) [124] stated that Sleep fragmentation, a common feature in many sleep disorders, may be related specifically to various abnormalities, such as respiratory events or periodic leg movements, and may also exist without any identifiable triggering event as in chronic insomnia. The authors briefly discussed the various autonomic markers used to assess and evaluate sleep fragmentation. They stated that the autonomic markers are much easier to use and can be implemented outside of highly specialized sleep laboratories and hence they may be useful in various clinical conditions, as the detection of sleep fragmentation is helpful for the management of sleep disorders.

Omran et al (2004) [125] reported a study to determine the severity of posthypoxic ventilatory decline in patients with sleep apnea relative to normal subjects during sleep. The authors measured EEG, electrooculogram, arterial O2 saturation, and end-tidal PCO2. They also compared the prehypoxic control (C) with posthypoxic recovery breaths. It was inferred from the data obtained that posthypoxic ventilatory decline occurred after termination of hypocapnic hypoxia in normal
subjects and patients with sleep apnea and manifested as decreased tidal volume which was more pronounced in sleep apnea patients.

Marcus et al (2004) [126] reported a study that was based on the hypotheses that children have increased neuromotor activation of their pharyngeal airway during sleep compared with adults. And also that infants would have an upper airway that was resistant to collapse. The study compared the upper airway pressure-flow ($V^*$) relationship during sleep between normal infants, prepubertal children, and adults. The upper airway response to 1) intermittent, acute sub-atmospheric pressure administration (PNEG) (infants, children, and adults), and 2) hypercapnia (children and adults) were evaluated. Results showed that adults had a more collapsible upper airway during sleep than either infants or children. The children exhibited a vigorous response to both PNEG and hypercapnia during sleep ($P \leq 0.01$), whereas adults had no significant change. Infants had an airway that was resistant to collapse and showed a very rapid response to PNEG. We conclude that the upper airway is resistant to collapse during sleep in infants and children. Normal children have preservation of upper airway responses to PNEG and hypercapnia during sleep, whereas responses are diminished in adults. Infants appear to have a different pattern of upper airway activation than older children. Authors speculated based on the findings that the pharyngeal airway responses present in normal children are a compensatory response for a relatively narrow upper airway.

Edwin et al (2004) [127] reported a systematic review on functional MRI imaging of lungs using hyperpolarized 3-Helium gas. According to the review manuscript, lung imaging traditionally relied on x-ray methods, since proton MRI is limited to some extent by low proton density in the lung parenchyma and static field in-homogeneities in the chest. The relatively recent introduction of MRI of hyperpolarized noble gases, as reported in the review, has led to a rapidly evolving field of pulmonary MRI, revealing functional information of the lungs, which were previously unattainable. The manuscript briefly described the physical background of the technology, and subsequently focuses on its clinical applications. Four different techniques that were commonly used in various human investigations were discussed: ventilation distribution, ventilation dynamics, and small airway evaluation using diffusion imaging and oxygen uptake assessment.
Tryon (2004) [128] stated a study which intended to identify and critically evaluate several theoretic and methodologic issues that are central to the divergent views regarding the valid use of actigraphy for sleep assessment. From the findings, following points were concluded - (1) Coefficients of the validity of actigraphy exceeded those associated with common medical tests and the best psychological tests. (2) Reasons why actigraphy should be held to a substantially higher empirical standard than common medical tests and the best psychological tests have yet to be advanced. (3) Differences between actigraphy and polysomnography are not random and can be reduced. (3a) Sleep onset is a gradual rather than a discrete process. Actigraphy keys on an earlier phase of the sleep-onset process than does polysomnography, resulting in systematic rather than random differences. (3b) A sleep switch device can be used to substantially increase the accuracy of sleep-onset times. (3c) The residual unreliability of polysomnographic data explains a portion of the differences between actigraphy and polysomnography. Actigraphy cannot be expected to agree more completely with polysomnography than polysomnography does with itself. (4) Complete agreement between actigraphy and polysomnography has been presumed, but achieving such a limit is theoretically impossible. Some lower maximum agreement limit should be identified. (5) Conclusions that actigraphy was not an accurate sleep-wake indicator and that it is inappropriate to infer sleep from actigraphy data are not supported by the findings.

Emmanouel et al (2004) [129] documented a study which intended to evaluate if administration of nasal corticosteroids for 4 weeks to snoring children with only mild elevation in their apnea-hypopnea index would improve both polysomnography findings and symptoms of sleep-disordered breathing. It was concluded from the study that four weeks of nasal corticosteroid improved both polysomnography findings and symptoms in children with mild sleep-disordered breathing. The clinical effects were maintained for several months after treatment.

Warunk (2004) [130] reported a manuscript which systematically reviewed the sleep medicine literature with special attention to the articles about randomized, controlled studies. The author discussed on terminology, diagnostic imaging, clinical procedures, patient communication, contraindications and complications of therapy with different types of adjustable and nonadjustable oral appliances. It was
summarized from the review that oral appliances are a common treatment option in the management of sleep apnea syndromes. While many patients experience a complete or partial resolution of their symptoms, some do not improve or may even become worse. It is therefore imperative that physicians conduct progress evaluations while the respective dental care provider continues to make adjustments to optimize the effectiveness of the chosen appliance.

Walsleben et al (2004) [131] reported a study which aimed to describe the distribution of nocturnal sleep characteristics and reports of daytime sleepiness in a large well-defined group of healthy adults. 470 subjects were selected as a ‘normative’ group based on screening of health conditions and daily habits that could interfere with sleep. It was concluded from the study that a clear lessening in the quantity and quality of sleep with age that appeared to be more rapid in males compared to females.

Yucel et al (2005) [132] examined the changes of the upper airway cross-sectional area in each phase of respiration in different degrees of severity of Obstructive sleep apnea syndrome (OSAS) with dynamic CT and investigated whether these changes have any correlation with sleep apnea severity parameters, including polysomnography (PSG) and cephalometry. It was concluded from the study that patients with severe OSAS had significant differences in the parameters. Measurement of the cross-sectional area of oropharynx in expiration can especially be useful for diagnosis of severe OSAS as a new key point.

Arie Oksenberg (2005) [133] reported a manuscript in which he classified Obstructive Sleep Apnea (OSA) patients into Positional Patients (PP) - patients with a supine Apnea-Hypopnea Index (AHI) at least twice higher than the lateral AHI, and Non-Positional Patients (NPP), with a supine AHI not reaching double value of lateral AHI. It was stated that AHI is the most significant factor that predicts the positional dependency. Further it was stated that PP are common in mild to moderate OSA. Severe OSA patients are mostly NPP. Positional therapy - avoidance of the supine posture during sleep – was affirmed as a suitable form of therapy for PP, particularly. For moderate-severe non-positional OSA patients, positive airway pressure was recommended as the logical first therapeutic option.
Amy S et al (2005) [134] reported a study which compared Loop Gain (LG) and Pharyngeal critical closing pressure (Pcrit) between men and women with OSA to determine whether the factors contributing to apnea are similar between genders. The first group of 11 men and 11 women were matched for OSA severity (mean _ SE apnea-hypopnea index = 43.8 _ 6.1 and 44.1 _ 6.6 events/h). The second group of 12 men and 12 women were matched for body mass index (BMI; 31.6 _ 1.9 and 31.3 _ 1.8 kg/m2, respectively). All measurements were made during stable supine non-rapid eye movement sleep. LG was determined using a proportional assist ventilator. Pcrit was measured by progressively dropping the continuous positive airway pressure level for three to five breaths until airway collapse. It was inferred from the results of the study that women may be protected from developing OSA by having a less collapsible upper airway for any given degree of obesity.

Clodagh and Bradley (2005) [135] reported a manuscript on the pathogenesis of obstructive sleep apnea (OSA). The authors stated that structural/anatomic factors that constrict space for the soft tissues surrounding the pharynx and its lumen are crucial to the development of OSA in many patients. Enlargement of soft tissues enveloping the pharynx, including hypertrophied tonsils, adenoids, and tongue, is also an important factor predisposing to UA collapse, inasmuch as this can impinge on the pharyngeal lumen and narrow it during sleep. Other factors, including impairment of UA mechanoreceptor sensitivity and reflexes that maintain pharyngeal patency and respiratory control system instability have also been identified as possible mechanisms facilitating UA instability. The authors suggest that OSA may be a heterogeneous disorder, rather than a single disease entity. Therefore, the extent to which various pathogenic factors contribute to the phenomenon of repetitive collapse of the UA during sleep probably varies from patient to patient. Finally the authors summarize stating that further elucidation of specific pathogenic mechanisms in individuals with OSA may facilitate the development of new therapies that can be tailored to individual patient needs according to the underlying mechanism(s) of their disease.

Heinzer et al (2005) [136] conducted a study to determine the influence of lung volume on the level of continuous positive airway pressure (CPAP) required to prevent flow limitation during non-REM sleep in subjects with sleep apnea.
Seventeen subjects were studied during stable non-REM sleep in a rigid head-out shell equipped with a positive/negative pressure attachment for manipulation of extrathoracic pressure. An epiglottic pressure catheter plus a mask/pneumotachometer were used to assess flow limitation. It was concluded from the study that relatively small changes in lung volume have an important effect on the upper airway in subjects with sleep apnea during non-REM sleep.

Pevernagie et al (2005) [137] reported a manuscript which reviewed the existing scientific knowledge regarding the relationship between nasal function, breathing and sleep including: the effect of sleep on breathing, nasal anatomy and function, physiological adaptations of the nose to sleep, the effect of nasal pathology on breathing during sleep, the role of nasal obstruction in snoring and sleep-disordered breathing. It was summarized from the review that there is evidence for an interaction between nasal and pharyngeal conditions in the pathogenesis of the obstructive sleep apnea syndrome.

Nayar and Knox (2005) [138] presented a clinical report of fabrication of a mandibular advancement splint for an edentulous patient without an increase in the vertical dimension of occlusion. The authors described the management of obstructive sleep apnea in an edentulous patient with a mandibular advancement splint. The method of splint fabrication and rationale were discussed.

Lee Shing et al (2005) [139] stated a case report in which they described a case of obstructive sleep apnea secondary (OSAS) to pharyngomalacia and laryngomalacia in a neonate with Down syndrome. It was stated that pharyngomalacia is an under-diagnosed but important condition in bronchosopic examination of patients with Down syndrome. It was further stated by the authors that pharyngomalacia is a newly described clinical entity, which if unnoticed is associated with failure of supraglottoplasty in relieving symptoms of airway obstruction of laryngomalacia.

Kristo et al (2005) [140] reported a manuscript that describes a subset of patients with Upper airway resistance syndrome (UARS) diagnosed by polysomnography who do not manifest snoring, which the authors defined as silent
Upper airway resistance syndrome (SUARS). It was concluded from the study that UARS may occur in the absence of clinically significant snoring and may be an occult cause of Excessive daytime somnolence (EDS). It was also stated from the study that there is a prevalence of SUARS of 9% among UARS patients and nearly 1% of all patients studied for hypersomnolence by polysomnography.

Aarnoud Hoekema (2006) [141] did a systematic review of the literature to determine the evidence base with respect to the efficacy and co-morbidity of the oral appliance therapy in Obstructive sleep apnea syndrome (OSAS). The preliminary results of a randomized parallel trial were also reported on the effectiveness and specific indication of, respectively, the oral appliance and continuous positive airways pressure therapy in OSAHS. It was concluded from the study that oral appliance and CPAP therapy are competitive in a substantial proportion of OSAHS patients.

Kanbay (2006) [142] reported a study that was done to determine the prevalence of metabolic syndrome in Turkish patients with Obstructive sleep apnea syndrome (OSAS) and the metabolic syndrome related cardiovascular diseases. It was concluded from the data obtained that metabolic syndrome is associated with an increased prevalence of OSAS. In addition, the prevalence of MS is significantly high in patients with moderate-severe OSAS patients.

Diez-Montiel et al (2006) [143] reported a study that measured the long-term impact of tonsillectomy and/or adenoidectomy (T&A) on children with obstructive sleep apnea (OSA). A controlled study was done on 101 OSA children, operated between June 1999 and January 2001. It was concluded from the data obtained through the study that children with OSA who undergo surgery show a significant long-term improvement in quality of life.

Bhimaraj et al (2006) [144] reported a study analyzing the Anti-hypertensive medications and insulin requirements post tracheostomy in a patient with severe obstructive sleep apnea syndrome. Through the study they concluded there will be a significant reduction in blood pressure and blood sugar levels following tracheostomy for severe Obstructive Sleep Apnea. This severe reduction in blood pressure and
hypoglycemia could mimic sepsis and clinicians need to be vigilant to these dramatic effects following tracheostomy and appropriately adjust baseline medications.

Bharati et al (2006) [145] studied the structural and functional cardiac alterations in obstructive sleep apnea (OSA), their relationship to the severity of OSA and the effects of treatment with continuous positive airway pressure (CPAP). Left and right ventricular morphology and function were studied using echocardiography before and after treatment with CPAP in symptomatic patients (Epworth sleepiness score, 10 _ 4.8) with severe OSA (apnea-hypopnea index [AHI], 42 _ 24). The patients (n _ 43, 32 men) had no known cardiac disease and were obese (body mass index, 31.6 _ 5.4 kg/m2). The same echocardiographic parameters were studied in age-matched overweight patients (n _ 40; body mass index, 26.4 _ 2.3 kg/m2). It was concluded from the study that the structural and functional consequences of OSA on the heart are influenced by the severity of AHI. These effects are reversible if the apneic episodes are abolished.

Hayes (2006) [146] reported a manuscript with case report of a six-year-old healthy female with cystic fibrosis (CF) and pancreatic sufficiency presenting with cough, weight loss, and lung function decline. Reportedly, history suggested obstructive sleep apnea, and nocturnal Polysomnography (NPSG) confirmed this. Adenotonsillectomy resulted in resolution of clinical symptoms with return of normal lung function. This case manuscript emphasized that obstructive sleep apnea syndrome (OSAS) may be a potential cause of lower airway inflammation and resulting weight loss in the young CF population. Based on the case report, the author states that upper airway inflammation from snoring and OSAS in children with CF may cause or potentiate lower airway inflammation leading to cough and depressed pulmonary function, culminating in weight loss. He summarized stating that resolution of clinical symptoms with return of normal lung function correlated with the treatment of OSAS. And also physicians should consider OSAS in the clinical presentation of worsening respiratory symptoms and weight loss in young CF patients who are unresponsive to appropriate antibiotic therapy and airway clearance.

White (2006) [147] reported a manuscript that provided an overview of the disorder Obstructive sleep apnea (OSA). Four general topics were addressed:
The author stated that obstructive sleep apnea is a common disorder characterized by repetitive collapse of the pharyngeal airway during sleep. The disorder results primarily from an anatomically small upper airway in conjunction with pharyngeal dilator muscles that can compensate for the anatomic deficiency awake, but not asleep. Ventilatory control instability and a low arousal threshold may contribute to the disorder as well. The consequences of sleep apnea, according to the author, fall into two domains: (1) neurocognitive dysfunction (sleepiness and decreased quality of life) resulting from sleep fragmentation and (2) cardiovascular disease (hypertension, stroke, myocardial infarction, and heart failure) likely resulting from the intermittent hypoxia. The disorder, as reported by the author, is generally diagnosed in the sleep laboratory over the course of a night, although alternative approaches in the home are also utilized reportedly. A number of treatment options are available, as reported by the author. Continuous positive airway pressure remains the most consistently effective approach according to the author, although oral appliances (generally mandibular-advancing devices) and a number of surgical procedures have some demonstrated efficacy, as reported in the manuscript. The author summarizes stating that, therapy must be individualized to the patient’s desires and the severity of the apnea.

Eun Yeon et al (2006) [148] reported a study that investigated the structural abnormalities in male patients with severe obstructive sleep apnea–hypopnea syndrome (OSAHS). Twelve male patients with severe OSAHS and 12 age-matched male normal subjects underwent a volumetric brain MRI. The MRIs were spatially normalized to standard T1 template and segmented into gray matter (GM), white matter (WM) and CSF. The segmented images were smoothed using 12-mm FWHM isotropic Gaussian kernel. Optimized voxel-based morphometry (VBM) protocol was used for the analysis of the brain tissue concentration and regional volume change using SPM2. The analysis showed structural brain abnormalities in patients with severe OSAHS, which were not detected by visual inspection. According to the authors, decreased Grey matter concentration (GMC) in right hippocampus may be related to memory impairment in OSAHS patients and GMC reduction in right cerebellum may be associated with the in-coordination of upper airway muscles.
Somoza et al (2006) [149] reported a study which intended to evaluate the usefulness of Helical CT-generated three-dimensional images of upper airway UA) in assessing adjustable mandibular advancement device (MAD) effectiveness in OSA patients; and second, to study the efficacy and tolerability of Herner MAD. It was concluded that patients responding to MAD had smaller UA dimensions, especially at hypopharynx. The narrower the hypopharynx, the greater the improvement in AHI wearing MAD. It was also concluded that helical CT provides three-dimensional evaluation of upper airways and may be useful to select OSA patients likely to benefit from MAD treatment.

Jason et al (2006) [150] examined the dynamic modulation of upper airway (UA) function during sleep, by devising a novel approach to measuring the critical pressure (Pcrit) within a single breath in tracheostomized sleep apnea patients. The authors examined tidal pressure-flow relationships throughout the respiratory cycle to compare phasic differences in UA collapsibility between closure and reopening. It was concluded by the authors that UA collapsibility varies dynamically throughout the respiratory cycle and that both local mechanical and neuromuscular factors may be responsible for this dynamic modulation of UA function during sleep.

Philip et al (2006) [151] reported a study which intended to assess the short-term efficacy and compliance of a novel mandibular advancement device for obstructive sleep apnoea (OSA) patients in routine care setting. The study validated efficacy of Optimized Retention of the Mandible (O.R.M.) device on respiratory and sleepiness parameters in routine standard of care setting. The authors stated that majority of patients responded to treatment without systematic titration protocol. Rapid improvement on quality of life and sleep was stated by most patients as well as high overall compliance, as reported by the authors.

Katsuhisa et al (2006) [152] reported a case of hyperthyroidism in a patient who was initially suspected to have Obstructive sleep apnea syndrome (OSAS). The authors stated that the prevalence of hyperthyroidism is approximately 2.9% in patients with OSAS. In the case presented, sleep study showed obstructive apneas but the findings of excessive sweating, slow sweat-like artifacts and atrial fibrillation lead to the suspicion of hyperthyroidism, as according to the authors.
Heinzer et al (2006) [153] conducted a study that was intended to determine the effect of a constant lung volume increase on sleep disordered breathing during non-REM sleep. Twelve subjects with OSA were studied during non-REM sleep in a rigid head-out shell equipped with a positive/negative pressure attachment for manipulation of extrathoracic pressure. The increase in lung volume due to CPAP (at a therapeutic level) was determined with four magnetometer coils placed on the chest wall and abdomen. It was concluded that an increase in lung volume causes a substantial decrease in sleep disordered breathing in patients with OSA during non-REM sleep.

Harlid Richard and Frostell Claes (2006) [154] investigated to find out if mandibular advancement device (MAD) can be used as an effective treatment for patients with severely elevated sleep related respiratory disturbances/severe OSAS. The results of the study indicated that good, if not sufficient, effect of treatment of OSAS with MAD can be expected also in patients with severe elevation of respiratory disturbances during sleep.

Thomas Verse et al (2006) [155] reported a study which was done to determine the efficacy of a multilevel surgical protocol for obstructive sleep apnea (OSA). Sixty patients with moderate to severe OSA because of multilevel pharyngeal obstruction were enrolled into this prospective, controlled clinical trial after clinical examination, endoscopy, and polysomnography. Surgery included uvulaflap, tonsillectomy, hyoid suspension, and radiofrequency treatment of the tongue base (group A). A second group did not receive hyoid suspension (group B). In both groups, nasal surgery was performed if necessary. Polysomnography and Epworth Sleepiness Scale (ESS) were recorded at baseline and 2 to 15 months after surgery. It was concluded from the study that the presented protocol including the hyoid suspension was effective in the treatment of OSA, whereas surgery without hyoid suspension was less successful.

Tamay et al (2006) [156] stated a study which was intended to elucidate the knowledge and attitude physicians have about pediatric OSA, using the Obstructive
Sleep Apnea Knowledge and Attitudes in Children (OSAKA-KIDS) questionnaire. The first section of the OSAKA-KIDS questionnaire, which includes 18 items presented in a true-or-false format, was developed to assess the knowledge physicians have about pediatric OSA. The second section, including five items, was developed to assess attitudes and was measured on a five-point Likert scale ranging from 1 to 5. It was inferred from the study that among physicians there are deficits in knowledge about childhood OSA and its treatment.

Nuckton et al (2006) [157] documented a study which aimed to assess the clinical usefulness of the Mallampati score in patients with obstructive sleep apnea. One hundred thirty-seven adult patients were evaluated for possible obstructive sleep apnea. Prospective determination of the Mallampati score, assessment of other variables for multivariate analysis, and subsequent overnight polysomnography were done. It was concluded from the findings that Mallampati scoring is a useful part of the physical examination of patients prior to polysomnography. The independent association between Mallampati score and presence and severity of obstructive sleep apnea in the study suggested that this scoring system has a practical value in clinical settings and prospective studies of sleep-disordered breathing.

Lundkvist and Friberg (2006) [158] evaluated the treatment of uvulopalatopharyngoplasty (UPPP) in patients failing or not accepting Continuous positive airway pressure (CPAP) and/or mandibular advancement device treatment. It was concluded from the study that UPPP is an effective and safe method of treating OSAS in patients failing CPAP and/or dental device treatment.

Aiman et al (2007) [159] highlighted the importance of the connections between Obstructive sleep apnea (OSA) and asthma. Based on literature review, they stated that pathophysiology of the two conditions overlapped significantly. Furthermore, they stated that a collateral rise in prevalence of both OSA and asthma has been noticed during in the past years. The authors proposed a hypothetical OSA–asthma relationship that has implications on the diagnosis and management of patients presenting with either condition singly. They stated that clinicians should be aware that OSA might complicate asthma management. Based on this hypothesis, they
suggested that the treatment of the individual patient who experiences both asthma and OSA needs to be multidisciplinary and comprehensive.

Padma et al (2007) [160] reported a review on sleep disordered breathing. They reviewed the basic patho-physiology stated in the literature and also the different modalities of management. Based on the literature review, they stated that oral appliances improve the blood oxygen saturation levels as they relieve apnea in 20-75% of patients. Appliance therapy also reduces the apnea-hypopnea index (AHI) by 50% or to < 10 events per h. Oral appliances also reduce the AHI to normal in 50-60% patients.

David and Moris (2007) [161] presented a manuscript of case report with a modification of the phase I of the surgical treatment for Obstructive Sleep Apnea (OSA), encompassing genioglossus advancement and hyoid suspension. The modified mandibular anterior window Osteotomy, followed in this technique, provides greater bone preservation, gives increased mandibular strength by design, and uses autologous bone for grafting. After elaborating the technique, the authors summarized stating that this hyoid procedure offers many advantages, including the ability to be easily accomplished through the same incision and to provide for a superior and anterior vector of tension that augments it of genioglossus advancement and also that it gives a stable and predictable outcome.

Zhi-Hui Zhao et al (2007) [162] reported a study in which one hundred twenty-six eligible consecutive Chinese heart failure (HF) patients were included and underwent historic data collection and a sleep study. Seventy-one percent of HF patients were diagnosed with sleep apnea (SA), of which 65% were central sleep apnea (CSA) and 35% were obstructive sleep apnea (OSA). Higher body mass index (BMI), metabolic syndrome, habitual snoring, and nocturia were independent risk factors for OSA; It was observed that there was a high prevalence of SA in Chinese patients with HF. HF patients with obesity, metabolic syndrome, snoring and nocturia were more susceptible to OSA and CSA.

Wendy et al (2007) [163] documented a case report that presented 3 cases in which patients with OSA had significant corneal and anterior segment complications
after beginning CPAP therapy. These cases highlighted the importance for eye care professionals to ask about OSA and knowing the potential complications of CPAP therapy. It was highlighted by the authors that patients with serious ocular complications related to CPAP might be considered for oral appliances or surgical options.

Rowley et al (2007) [164] reported a study which was conducted to analyze the influence of long-term facilitation on upper airway collapsibility as measured by the critical closing pressure (Pcrit) model and to determine whether changes in upper airway resistance (Rua) correlated with changes in collapsibility. The authors studied 13 subjects (10 men, 3 women) with a mean apnea-hypopnea index of 43.9 _ 24.0 events/h. It was concluded from based on the findings that long term facilitation of upper airway dilators is not associated with changes in upper airway collapsibility in subjects with obstructive sleep apnea. The authors stated that the results corroborated with previous evidence that changes in upper airway resistance and caliber can be dissociated from changes in upper airway collapsibility.

Petros et al (2007) [165] intended to assess the improvement of concurrent erectile dysfunction (ED) in men with overlap syndrome (obstructive sleep apnea and chronic obstructive pulmonary disease), treated with continuous positive airway pressure (CPAP) and bronchodilators. The authors evaluated 48 men of a mean age of 52.8±10 years suffering from both obstructive sleep apnea (OSA) and chronic obstructive pulmonary disease (COPD), and concurrent ED. It was concluded that conventional treatment for OSA and COPD, has a positive effect on concurrent ED on the minority of patients. The authors stated that this effect may be possibly due to the improvement of respiration during sleep with CPAP and of oxygenation with bronchodilators continuously.

Vsevolod and Christopher (2007) [166] documented a review which focused on the most common and well-studied syndrome of Obstructive sleep apnea. The authors, through the review, tried to determine how genetic, genomic, and proteomic studies have provided new insights into the (1) pathogenesis, (2) expression, and (3) sequelae of OSA. Based on the review, the authors summarized the following- It is unlikely the genetic basis underlying the pathogenesis and expression of OSA will be
significantly advanced by the technologies of genomics and proteomics; Rather, more traditional approaches of human and animal genetics and targeted gene technologies hold the most promise for exploring the “causes” of OSA; In the clinical arena, genomic and proteomic approaches hold promise as a screening tool for diagnosis of OSA using blood or urine.

Jonas et al (2007) [167] reported a study that evaluated regional lung aeration during wakefulness and sleep. Ten healthy subjects underwent spirometry awake and with polysomnography, including pulse oximetry, and also CT when awake and during sleep. Lung aeration in different lung regions was analyzed. Another three subjects were studied awake to develop a protocol for dynamic CT scanning during breathing. It was concluded through the study that aeration is reduced in dependent lung regions and increased in ventral regions during NREM and REM sleep. Ventilation is more uniformly distributed between upper and lower lung regions than has previously been reported in awake, upright subjects. The authors further stated that reduced respiratory muscle tone and airway closure are likely causative factors for the above mentioned observations.

Patil et al (2007) [168] documented a research manuscript which intended to determine the relative contribution of mechanical loads and dynamic neuromuscular responses to pharyngeal collapse during sleep. Sixteen obstructive sleep apnea patients and sixteen normal subjects were matched on age, sex, and body mass index. Pharyngeal collapsibility, defined by the critical pressure, was measured during sleep. It was concluded from the findings that increased mechanical loads and blunted neuromuscular responses are both required for the development of obstructive sleep apnea.

Moore (2007) [169] reported a review manuscript in which the author stated that Oral appliances (OA) are a recognized first-line treatment for snoring and mild-to-moderate OSA and can be a legitimate alternative (or rescue therapy) for some severe OSA patients who fail first-line therapies (CPAP or UPPP). Short-term side effects to Mandibular repositioning appliance (MRA) are common but typically are transient in nature, and long-term side effects seem to be common but typically mild. Although airway response varies between individuals, overall, 52% of all comers
respond to OA therapy. It was further stated that an appropriate working knowledge of this field and relationship with knowledgeable and experienced dental sleep medicine providers is needed for otolaryngologists seeking this therapy for their patients.

Kenneth and Ana (2007) [170] stated that OSA is challenging and there has been greater recognition by the medical and dental disciplines. By understanding the rationale, indications, benefits, risks and success of the various treatment options available, clinicians will be able to make more informed treatment recommendations in patient management. The authors briefly discussed about the epidemiology, pathophysiology, treatment protocol and treatment strategies.

Gilles et al (2007) [171] conducted a study to demonstrate an inflammatory process at the bronchial level in patients with OSA and to analyze effects of continuous positive airway pressure (CPAP) application and humidification on bronchial mucosa. The study was conducted by using sequential induced sputum for cell analysis and IL-8 production, nitric oxide exhalation measurement, and methacholine challenge before and after CPAP. It was concluded from the findings by the authors that Obstructive sleep apnea syndrome is associated with bronchial inflammation. It was inferred that a spontaneous bronchial inflammation in OSA and the development of a CPAP-related airway hyperresponsiveness (AHR) may require a long-term follow-up to evaluate consequences.

Bradley and Thomas (2007) [172] reported a case of cricoarytenoid dysfunction leading to acute respiratory insufficiency requiring tracheostomy in the immediate postoperative period after total knee arthroplasty in a patient with severe rheumatoid arthritis. The authors summarized stating that cricoarytenoid involvement should be suspected in orthopedic patients with longstanding rheumatoid arthritis. Absence of symptoms does not rule out cricoarytenoid involvement because chronic disease may be exacerbated by trauma of intubation. It is important for the surgeon and anesthesiologist to be aware of the potentially fatal complication of airway obstruction in these patients. Tracheostomy remains the definitive treatment, and patients at risk should be counselled on this infrequent complication.
Torbjörn et al (2007) [173] presented a review paper that will summarized some of the studies on manipulated sleep to find out what are the physiological characteristics of “a good nights sleep”. The authors also stated some recent work on the characteristics of sleep during burnout, as well as some studies of individual characteristics of “intolerance” to night work. The authors also stated a number of disparate observations related to sleep, stress and wakefulness which according to them formed a cluster of research around an interesting and probably important sequence of putative causality, from stress, via disturbed sleep and its consequences for metabolic and mental functioning, to their possible endpoints in stress-related disorders and accident risk.

Walter (2008) [174] presented a review manuscript discussing the clinical assessment of patients with suspected OSAS and also the potential added value of structured questionnaires and clinical prediction models that seek to improve the diagnostic value of clinical assessment from the formalized evaluation of selected clinical features. The author stated that although the traditional “gold standard” for objective assessment is laboratory-based polysomnography, there is growing evidence that limited sleep studies focused on respiratory and cardiac variables are adequate in most cases, and are particularly suited to home-based assessment. It was further stated by the author that the choice between home versus sleep laboratory studies should be decided by taking into account resource limitations and the clinical index of suspicion for OSAS. It was stated by the author that patients with either a low or high clinical index of suspicion for OSAS appear most suited to home based investigation, whereas those with intermediate levels of clinical suspicion, or who present with atypical clinical features, may best be assessed by full polysomnographic studies in the first instance.

Liu A et al (2008) [175] reported a study analyzing the neuromechanical control of the isolated upper airway of mice. They characterized the passive structural and active neuromuscular control of pharyngeal collapsibility in mice and hypothesized that pharyngeal collapsibility, which is elevated by anatomic loads, is reduced by active neuromuscular responses to airflow obstruction. They examined the dynamic control of upper airway function in the isolated upper airway of anesthetized C57BL/6J mice. Pressures were lowered downstream and upstream to the upper
airway to induce inspiratory airflow limitation and critical closure of the upper airway, respectively. The findings in mice implied that anatomic and neuromuscular factors interact dynamically to modulate upper airway function.

Andrew et al (2008) [176] reviewed the literature for non positive airway pressure treatment modalities. The two modalities commonly used in clinical practice, mandibular advancement devices (MADs) and positional therapy were analyzed based on a literature review. Based on this review, it was stated that a better understanding of the range of OSA phenotypes and predictors of response to different treatment modalities is required to allow physicians to tailor the choice of treatment to the individual patient.

Alan et al (2008) [177] stated that obesity and central adiposity are potent risk factors for sleep apnea. They can increase pharyngeal collapsibility through mechanical effects on pharyngeal soft tissues and lung volume, and through central nervous system–acting signaling proteins (adipokines) that may affect airway neuromuscular control. They stated that a variety of behavioral, pharmacologic and surgical approaches to weight loss may be of benefit to patients with sleep apnea, through distinct effects on the mass and activity of regional adipose stores. They also affirmed that examining responses to specific weight loss strategies will provide critical insight into mechanisms linking obesity and sleep apnea, and will help to elucidate the humoral and molecular predictors of weight loss responses.

Amy and David (2008) [178] reported a review manuscript that emphasized on comprehensive understanding of the neural regulation of the muscles of upper airway. In the manuscript, the dilator muscles were classified in two broad categories; those that have respiratory related activity and those that fire constantly throughout the respiratory cycle. It was stated that the activity of both muscle groups is reduced shortly after sleep onset, indicating that both receive input from brainstem neurons involved in sleep regulation. It was also stated that in sleep apnea patient, this may lead to pharyngeal airway collapse. The review briefly described the currently proposed sleep and respiratory neural pathways and how these circuits interact with the upper airway dilator muscle motor neurons. It was summarized based on the literature review that upper airway dilator muscles activity is reduced during sleep,
likely due to loss of the wakefulness drive and decrements in respiratory neural drive and negative pressure responsiveness.

A Ysunza (2008) [179] presented a study that aimed to investigate whether Videonasopharyngoscopy (VNP) can be useful for identifying severe Obstructive Sleep Apnea (OSA) in a population of children with craniofacial anomalies and children with adenoid and tonsils hypertrophy. Eighty children with adenoid and tonsils hypertrophy and 72 children with craniofacial anomalies who were present with clinical data suggestive of sleep-disordered breathing were studied. All the parents completed a questionnaire concerning the children’s sleeping habits and sleep complaints before consultation. Each child underwent a general pediatric examination and an evaluation of craniofacial features, including assessment of upper airway permeability. In all children, a PS was performed. Also, all children underwent a VNP. It was concluded based on the results that in children with craniofacial anomalies and tonsils and adenoid hypertrophy, airway obstruction as assessed by VNP seems to be a significant risk factor for OSAS. VNP is a safe and significantly reliable procedure for the evaluation of sleep breathing disorders in children.

McGinley BM et al (2008) [180] reported a study designed based on the hypothesis that subjects without sleep apnea are more capable of mounting vigorous neuromuscular responses to upper airway obstruction than subjects with sleep apnea. To address this hypothesis the authors lowered nasal pressure to induce upper airway obstruction to the verge of periodic obstructive hypopneas (cycling threshold). Ten patients with obstructive sleep apnea and nine weight-, age-, and sex-matched controls were studied during sleep. Responses in genioglossal electromyography (EMGGG) activity (tonic, peak phasic, and phasic EMGGG), maximal inspiratory airflow (VImax), and pharyngeal transmural pressure (PTM) were assessed during similar degrees of sustained conditions of upper airway obstruction and compared with those obtained at a similar nasal ressure under transient conditions. Control compared with sleep apnea subjects demonstrated greater EMGGG, VImax, and PTM responses at comparable levels of mechanical and ventilatory stimuli at the cycling threshold, during sustained compared with transient periods of upper airway obstruction. Furthermore, the increases in EMGGG activity in control compared with sleep apnea subjects were observed in the tonic but not the phasic component of the
EMG response. It was concluded from these inferences that sustained periods of upper airway obstruction induce greater increases in tonic EMGGG, Vlmax, and PTM in control subjects. The neuromuscular responses protect individuals without sleep apnea from developing upper airway obstruction during sleep.

Babak et al (2008) [181] reported a manuscript that reviewed the clinical characteristics of the Obesity hypoventilation syndrome (OHS), Epidemiology, clinical presentation, pathophysiology, morbidity and mortality associated with it and currently available treatment. The authors summarized stating that prevalence of OHS is likely to increase because of the global obesity epidemic. A high index of suspicion can lead to early recognition of the syndrome and initiation of appropriate therapy. The treatment options other than positive airway pressure have been poorly studied and further research is needed to better understand the long-term treatment outcomes of patients with OHS. If positive airway pressure fails to achieve the desired results, weight reduction surgery or tracheostomy with or without pharmacotherapy with respiratory stimulants should be considered.

Kushida CA et al (2008) [182] reported a systematic review on positive airway pressure devices and titration techniques used to treat patients with sleep related breathing disorders (SRBDs), including obstructive sleep apnea (OSA). Based on this review, the authors stated these recommendations for conducting Continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BPAP) titrations. (1) All potential PAP titration candidates should receive adequate PAP education, hands-on demonstration, careful mask fitting, and acclimatization prior to titration. (2) CPAP (IPAP and/or EPAP for patients on BPAP) should be increased until the following obstructive respiratory events are eliminated (no specific order) or the recommended maximum CPAP (IPAP for patients on BPAP) is reached: apneas, hypopneas, respiratory effort-related arousals (RERAs), and snoring. (3) The recommended minimum starting CPAP should be 4 cm H2O for pediatric and adult patients, and the recommended minimum starting IPAP and EPAP should be 8 cm H2O and 4 cm H2O, respectively, for pediatric and adult patients on BPAP. (4) The recommended maximum CPAP should be 15 cm H2O (or recommended maximum IPAP of 20 cm H2O if on BPAP) for patients <12 years, and 20 cm H2O (or recommended maximum IPAP of 30 cm H2O if on BPAP) for patients ≥12 years. (5)
The recommended minimum IPAP-EPAP differential is 4 cm H2O and the recommended maximum IPAP-EPAP differential is 10 cm H2O (6) CPAP (IPAP and/or EPAP for patients on BPAP depending on the type of event) should be increased by at least 1 cm H2O with an interval no shorter than 5 min, with the goal of eliminating obstructive respiratory events. (7) CPAP (IPAP and EPAP for patients on BPAP) should be increased from any CPAP (or IPAP) level if at least 1 obstructive apnea is observed for patients <12 years, or if at least 2 obstructive apneas are observed for patients ≥12 years. (8) CPAP (IPAP for patients on BPAP) should be increased from any CPAP (or IPAP) level if at least 1 hypopnea is observed for patients <12 years, or if at least 3 hypopneas are observed for patients ≥12 years. (9) CPAP (IPAP for patients on BPAP) should be increased from any CPAP (or IPAP) level if at least 3 RERAs are observed for patients <12 years, or if at least 5 RERAs are observed for patients ≥12 years. (10) CPAP (IPAP for patients on BPAP) may be increased from any CPAP (or IPAP) level if at least 1 min of loud or unambiguous snoring is observed for patients <12 years, or if at least 3 min of loud or unambiguous snoring are observed for patients ≥12 years. (11) The titration algorithm for split-night CPAP or BPAP titration studies should be identical to that of full-night CPAP or BPAP titration studies, respectively. (12) If the patient is uncomfortable or intolerant of high pressures on CPAP, the patient may be tried on BPAP. If there are continued obstructive respiratory events at 15 cm H2O of CPAP during the titration study, the patient may be switched to BPAP. (13) The pressure of CPAP or BPAP selected for patient use following the titration study should reflect control of the patient’s obstructive respiration by a low (preferably <5 per hour) respiratory disturbance index (RDI) at the selected pressure, a minimum sea level SpO2 above 90% at the pressure, and with a leak within acceptable parameters at the pressure. (14) An optimal titration reduces RDI <5 for at least a 15- min duration and should include supine REM sleep at the selected pressure that is not continually interrupted by spontaneous arousals or awakenings. (15) A good titration reduces RDI ≤10 or by 50% if the baseline RDI <15 and should include supine REM sleep that is not continually interrupted by spontaneous arousals or awakenings at the selected pressure. (16) An adequate titration does not reduce the RDI ≤10 but reduces the RDI by 75% from baseline (especially in severe OSA patients), or one in which the titration grading criteria for optimal or good are met with the exception that supine REM sleep did not occur at the selected pressure. (17) An unacceptable titration is one that does not meet any one of the above
grades. (18) A repeat PAP titration study should be considered if the initial titration does not achieve a grade of optimal or good and, if it is a split-night PSG study, it fails to meet American Academy of Sleep Medicine (AASM) criteria (i.e., titration duration should be >3 hr).

Christine et al (2008) [183] reported a manuscript which aimed to optimize the success of Upper airway surgical procedures by identifying proper candidates for surgery, as well as to develop new invasive procedures for OSA treatment. The surgical procedures included in the manuscript were uvulopalatopharyngoplasty, uvulopalatal flap, laser-assisted uvulopalatoplasty, and Radiofrequency ablation (RF) of the soft palate with adenotonsillectomy, surgeries aimed at reducing the bulk of the tongue base or providing more space for the tongue in the oropharynx including genioglossal advancement, hyoid-suspension, distraction osteogenesis, tongue RF, lingualplasty, and maxillomandibular advancement. The authors elucidate the specific surgical procedures, discuss about the medical evaluation for surgery, possible complications and risk management of the surgical procedures.

Dai and Douglas (2008) [184] reported a manuscript with emphasis on Cheyne-Stokes respiration in central sleep apnea (CSR-CSA). The authors stated that this is a form of periodic breathing, commonly observed in patients with heart failure (HF), in which central apneas alternate with hyperapneas that have a waxing-waning pattern of tidal volume. Uniform criteria by which to diagnose a clinically significant degree of CSR-CSA have yet to be established, as stated by the authors. CSR-CSA is caused by respiratory control system instability characterized by a tendency to hyperventilate. Central apnea occurs when PaCO2 falls below the threshold for apnea during sleep due to ventilatory overshoot. Patients with CSR-CSA are generally hypocapnic, with a PaCO2 closer than normal to the apneic threshold such that even slight augmentation in ventilation drives PaCO2 below threshold and triggers apnea. According to the authors, the factors contributing to hyperventilation in HF include stimulation of pulmonary irritant receptors by pulmonary congestion, increased chemoreceptor sensitivity, reduced cerebro-vascular blood flow and recurrent arousals from sleep. Controversy remains as to whether CSR-CSA is simply a reflection of HF severity, or whether it exerts unique adverse effects on prognosis. The main adverse influence of CSR-CSA on cardiovascular function appears to be excessive
sympathetic nervous system activity due to apnea-related hypoxia and arousals from sleep. A number of studies have examined the potential relationship between CSR-CSA and mortality in HF. Most studies, as reviewed by the authors, reported that CSR-CSA was associated with an increased risk for mortality, but these studies were small. After discussing on the diagnosis, pathophysiology, and clinical significance in cardio-vascular system, the authors summarize stating that further research is needed to elucidate mechanisms which contribute to the pathogenesis of CSR-CSA and to determine whether its treatment can reduce morbidity and mortality in patients with HF.

Danny and Atul (2008) [185] reported a review manuscript on pathophysiology of Obstructive sleep apnea (OSA). Authors stated that OSA is a disorder characterized by repetitive narrowing or collapse of the pharyngeal airway during sleep. The disorder is associated with major morbidities including excessive daytime sleepiness and increased risk of cardiovascular disease. The underlying pathophysiology, according to authors, is multifactorial and may vary considerably between individuals. Important risk factors include obesity, male sex, and aging. The authors added a note on the current understanding of OSA pathophysiology in adults and highlighted the potential mechanisms underlying the principal risk factors. In addition, some of the pathophysiological characteristics associated with OSA that may modulate disease severity were illustrated. Finally, the potential for novel treatment strategies, based on an improved understanding of the underlying pathophysiology, were also discussed.

Esra and Mary (2008) [186] reported a manuscript emphasizing on Obstructive sleep apnea and metabolic syndrome. As reported by the authors, metabolic syndrome (MS), the commonly used term for the clustering of obesity, insulin resistance, hypertension, and dyslipidemia, affects millions of people worldwide, and is associated with an increased risk of cardiovascular disease and type2 diabetes. Authors, based on literature review, suggested that obstructive sleep apnea (OSA) may contribute to the development of MS and diabetes. In the authors’ view, Obesity, particularly visceral obesity, is an important factor in the assessment of adverse metabolic outcome in OSA. Reportedly, further prospective and interventional studies, with adequate sample sizes and longer follow-up, rigorous
control for adiposity, and, ideally, randomization and control for any therapeutic intervention, are clearly needed to address the direction of causality. The authors highlight the multiple mechanistic pathways involved in the interaction between OSA, obesity, and metabolic derangements. Chronic intermittent hypoxia and sleep fragmentation with sleep loss in OSA are likely key triggers that initiate or contribute to the sustenance of inflammation as a prominent phenomenon, but their complex interplay remains to be elucidated according to authors. The authors summarized stating that OSA may represent a novel risk factor for MS and diabetes, and thus clinicians should be encouraged to systematically evaluate the presence of metabolic abnormalities in OSA and vice versa.

Emmanuel et al (2008) [187] documented a manuscript that emphasized on the association between Chronic obstructive pulmonary disease (COPD) and sleep apnea-hypopnea syndrome (SAHS). Based on literature review, the authors stated that Chronic obstructive pulmonary disease (COPD) and sleep apnea-hypopnea syndrome (SAHS) are both common diseases affecting respectively 10 and 5% of the adult population over 40 years of age and their coexistence, which is denominated overlap syndrome, can be expected to occur in about 0.5% of this population. As reported by the authors, in an epidemiologic study it was shown that the prevalence of SAHS is not higher in COPD than in the general population, and that the coexistence of the two conditions is due to chance and not through a pathophysiologic linkage between these two diseases. Patients with overlap, reportedly have a more important sleep-related O2 de-saturation than do patients with COPD with the same degree of bronchial obstruction. They have an increased risk of developing hypercapnic respiratory insufficiency and pulmonary hypertension when compared with patients with SAHS alone and with patients with “usual” COPD. In patients with overlap, hypoxemia, hypercapnia, and pulmonary hypertension can be observed in the presence of mild to moderate bronchial obstruction, which according to the authors is different from “usual” COPD. The authors summarize adding a not on the therapy of the overlap syndrome consists of nasal continuous positive airway pressure or nocturnal noninvasive ventilation (NIV), with or without associated nocturnal O2. Patients who are markedly hypoxemic during daytime (PaO2 , 55–60 mm Hg) should be given conventional long-term O2 therapy in addition to nocturnal ventilation.
Eliot and Carolyn (2008) [188] reported a review of literature on the pathophysiology of Pediatric Obstructive Sleep Apnea. The authors stated that sleep-disordered breathing is a common and serious cause of metabolic, cardiovascular, and neurocognitive morbidity in children. The spectrum of obstructive sleep-disordered breathing ranges from habitual snoring to partial or complete airway obstruction, termed obstructive sleep apnea (OSA). Breathing patterns due to airway narrowing are highly variable, including obstructive cycling, increased respiratory effort, flow limitation, tachypnea, and/or gas exchange abnormalities. As reported by the authors, these may lead to disturbance of sleep homeostasis. Increased upper airway resistance is an essential component of OSA, including any combination of narrowing/retropositioning of the maxilla/mandible and/or adenotonsillar hypertrophy. Reportedly, in addition to anatomic factors, the stability of the upper airway is predicated on neuromuscular activation, ventilatory control, and arousal threshold. The authors state that during sleep, most children with OSA intermittently attain a stable breathing pattern, indicating successful neuromuscular activation. At sleep onset, airway muscle activity is reduced, ventilatory variability increases, and an apneic threshold slightly below eupneic levels is observed in non-REM sleep. Airway collapse is offset by pharyngeal dilator activity in response to hypercapnia and negative lumenal pressure. Ventilatory overshoot results in sudden reduction in airway muscle activation, contributing to obstruction during non-REM sleep. Arousal from sleep exacerbates ventilatory instability and, thus, obstructive cycling. Authors summarized stating that understanding of pathophysiology of pediatric OSA may permit more precise clinical phenotyping, and therefore improve or target therapies related to anatomy, neuromuscular compensation, ventilatory control, and/or arousal threshold.

Pillar and Shehadeh (2008) [189] stated that Obesity is probably the most important risk factor for the development of OSA. Several mechanisms responsible for the increased risk of OSA with obesity include reduced pharyngeal lumen size due to fatty tissue within the airway or in its lateral walls, decreased upper airway muscle protective force due to fatty deposits in the muscle, and reduced upper airway size secondary to mass effect of the large abdomen on the chest wall and tracheal traction. Conversely, OSA may itself predispose individuals to worsening obesity because of sleep deprivation, daytime somnolence, and disrupted metabolism. OSA is associated
with increased sympathetic activation, sleep fragmentation, ineffective sleep, and insulin resistance, potentially leading to diabetes and aggravation of obesity. Furthermore, the authors stated that OSA may be associated with changes in leptin, ghrelin, and orexin levels; increased appetite and caloric intake; and again exacerbating obesity. Thus, the authors concluded stating that obesity and OSA form a vicious cycle where each results in worsening of the other.

Richard (2008) [190] documented an editorial review by searching the literature for any evidence of Obstructive sleep apnea (OSA) causing or leading to cognitive deficits in subjects. Based on the review, it was surmised that is important to recognize the association between OSA and cognitive impairment so that patients with mild cognitive impairment (MCI) undergo appropriate screening (and treatment) for OSA and to address the cognitive and psychological issues that adversely affect the quality of life in patients with OSA.

Terri and Ronald (2008) [191] in their manuscript, reviewed the nature of CPAP adherence, the evidence regarding salient predictors, and described the interventions that have been tested to improve adherence. Based on the review, the authors suggested that use of CPAP for longer than 6 hours decreases sleepiness, improves daily functioning, and restores memory to normal levels. It was also suggested that various behavioral interventions stated in the literature may be effective in improving CPAP adherence.

Naresh (2008) [192] documented a review article in which various epidemiologic aspects of adult obstructive sleep apnea were considered, with a particular emphasis on issues related to the population prevalence, natural history, and factors that increase the predisposition for the disorder. Based on the literature search, it was stated that untreated obstructive sleep apnea is associated with an increased risk of fatal and nonfatal cardiovascular event, a higher propensity of sudden death during sleep, and a greater risk for stroke and all-cause mortality. The mechanisms by which obstructive sleep apnea increases medical morbidity are complex and remain a focus of intense basic and human research. It was further stated by the author that, given the high prevalence and public health burden of obstructive sleep apnea, the implications
of untreated disease for the individual and society are enormous and cannot be ignored.

Julie and Ronald (2008) [193] systematically reviewed studies on the epidemiology of conditions considered part of a pediatric sleepdisordered breathing (SDB) continuum, ranging from primary snoring to OSA. The authors highlighted a number of methodologic challenges, including widely variable methodologies for collection of questionnaire data about symptomatology, definitions of habitual snoring, criteria for advancing to further diagnostic testing, and objective diagnostic criteria for SDB or OSA. Based on the literary evidence, it was suggested by the authors that SDB is more common among boys than girls, and among children who are heavier than others, with a higher prevalence among African Americans.

Somers VK et al (2008) [194] published a document that was intended to highlight concepts and evidence important to understanding the interactions between sleep apnea and cardiovascular disease, with particular attention to recent advances in patient-oriented research. It was summarized by the authors through this extensive literature review that in the context of epidemics of obesity, hypertension, atrial fibrillation, and heart failure, the prevalence and consequences of both OSA and CSA are likely to increase. Numerous hurdles face the cardiovascular community in the development of consensus regarding best practice. Objective of this document was to help develop the platform from which, in collaboration with specialists in sleep medicine and related disciplines, such consensus may emerge.

Walsh et al (2008) [195] conducted a study that intended to determine the effect of head posture on upper airway collapsibility and site of collapse of the passive human upper airway. Pharyngeal critical closing pressure (Pcrit) and site of airway collapse were assessed during head flexion, extension and rotation in individuals undergoing propofol anesthesia. Fifteen healthy volunteers (8 male), including 7 who were undergoing surgery unrelated to the head or neck were included as participants in the study. It was concluded from the findings that head posture has a marked effect on the collapsibility and site of collapse of the passive upper airway indicating that controlling head posture during sleep or recovery from anesthesia may alter the propensity for airway obstruction. Further, manipulating head posture during propofol
sedation may assist with identification of pharyngeal regions vulnerable to collapse during sleep and may be useful for guiding surgical intervention.

Sachin et al (2008) [196] documented a study that was conducted to investigate the long-term compliance of patients who were provided with a mandibular advancement device. Records of 180 patients who were provided with a mandibular advancement device in 1996 were reviewed. It was concluded by the authors that the mandibular advancement device can be an effective long-term solution for a significant number of patients with problem snoring and also those with mild to moderate obstructive sleep apnea.

Riccardo et al (2008) [197] conducted a study to investigate clinical features of upper airway resistance syndrome (UARS), comparing them to those in patients with Primary snoring (PS), obstructive sleep apnea / hypopnea without daytime sleepiness (OSAH) and obstructive sleep apnea/hypopnea syndrome (OSAHS). Retrospective chart analysis of 157 patients with PS, 424 patients with UARS, 562 patients with OSAH, and 1610 patients with OSAHS were done. It was concluded from the study that UARS patients share some clinical features of patients with OSAHS and PS, although these two groups differ in their presentation of clinical sleepiness. Patients with UARS were most impaired in terms of their daily functioning and perception of sleep quality. This finding could not be corroborated by objective measures.

Helene et al (2008) [198] reported a study which was conducted to determine whether peak Pdi (trans-diaphragmatic pressure) swings and peak Poes (oesophageal pressure) swings, during maximal sniff manoeuvres and during maximal static inspiratory manoeuvres, are comparable or, instead, supply complementary information for assessing diaphragmatic and global inspiratory muscle strength. It was concluded from the study that peak Pdi and peak Poes are complementary rather than interchangeable and should therefore be used in combination with non-volitional tests for a complete sequential assessment of diaphragmatic strength in patients with suspected diaphragmatic dysfunction and/or inspiratory muscle disease.
Jason et al (2008) [199] reported a literature review that was focussed on pathophysiologic mechanisms and explored concepts regarding the impact of OSA and its treatment on selected clinical disease states.

Sangal et al (2008) [200] reported a study which was performed to evaluate whether the selective norepinephrine re-uptake inhibitor atomoxetine improves sleepiness, the clinical global impression (CGI) of severity of illness and the respiratory disturbance index (RDI) in patients with mild to moderate obstructive sleep apnea with excessive sleepiness. It was concluded from the findings that atomoxetine improved sleepiness and the CGI in patients with mild to moderate obstructive sleep apnea with sleepiness. However, it did not improve the RDI.

Yuko et al (2008) [201] conducted a study that was planned evaluate the retroglossal airway configuration quantitatively and to make clear the relationship between Body mass index (BMI) and airway configuration. This retrospective study included 15 OSA patients (male=11; female=4) and 14 normal controls. Maximum anterior–posterior diameter (AP) and lateral width (LW) of the airway were measured, and the square area (SA) was calculated using Retroglossal airway slice. The airway cross-section area (AWA) was also measured, and then the AWA/SA ratio was calculated. It was inferred from the findings that AP, LW, and AWA were not statistically significantly different between controls and OSA patients. On the other hand, the AWA/SA ratio in OSA patients was 8.8% statistically significantly smaller than in controls after adjusting for sex, age and BMI.

Braga et al (2009) [202] reported a study that was performed to evaluate the frequency of Obstructive Sleep Apnea in a sample of chronic atrial fibrillation (AF) compared to a sub-sample of the general population. Fifty-two chronic AF patients aged (60.5 ± 9.5, 33 males) and 32 control (aged 57.3 ± 9.6, 15 males). All subjects were evaluated by a staff cardiologist for the presence of medical conditions and were referred for polysomnography. The differences between groups were analyzed by ANOVA for continuous variables, and by the Chi-square test for dichotomous variables. It was concluded from the analysis that sleep-disordered breathing is more frequent in chronic persistent and permanent AF patients than in age-matched community dwelling subjects.
Carole et al (2009) [203] reported a manuscript that aimed (1) to review the state of science in pediatric upper airway disorders; (2) to make recommendations to the Institute to fill knowledge gaps; (3) to prioritize new research directions; and (4) to capitalize on scientific opportunities. The authors stated that upper airway serves three important functions: respiration, swallowing, and speech. During development it undergoes significant structural and functional changes that affect its size, shape, and mechanical properties. Abnormalities of the upper airway require prompt attention, because these often alter ventilatory patterns and gas exchange, particularly during sleep when upper airway motor tone and ventilatory drive are diminished. The report provided recommendations that facilitate translation of basic research findings into practice to better diagnose, treat, and prevent airway compromise in children.

Dantas et al (2009) [204] reported a study that intended to quantify the components of the extracellular matrix in the lateral pharyngeal muscular wall and compare them between obstructive sleep apnea (OSA) and non-OSA groups of non-obese patients. Specimens from the superior pharyngeal constrictor muscle were obtained during pharyngeal surgeries (tonsillectomy or palatopharyngoplasty) of 51 non-obese adult patients divided to 13 controls and 38 OSA patients. The fractional area of elastic fibers, collagen type I and III, matrixmetalloproteinases 1 and 2, versican and fibronectin in the endomysium were determined by histochemical and immune-histochemical analyses. Based on the data obtained, it was concluded that increased amounts of collagen type I within pharyngeal muscles may be related to the upper airway muscle dysfunction found in OSA. And it is possible that fibronectin plays a protective role in the female pharynx against OSA.

GLA Diaféria et al (2009) [205] reported a study that aimed to describe the possible alterations in the structure and function of the orofacial motricity in Obstructive Sleep Apnea Syndrome (OSAS) patients through the use of a phonoaudiological evaluation model. Orofacial structures and functions were examined in male OSAS patients with respect to anthropometric, otorhinolaryngological, and phonoaudiological parameters. The study population included 22 male OSAS patients with an average age of 47 ± 11 years, a mean body mass index of 26.2 ± 4.9 kg/m2, and a cervical circumference of 43.9 ± 3.4 cm. It was concluded from the study that phonoaudiological model adopted produced relevant
findings with respect to modifications in orofacial motricity, which demonstrates the importance of this kind of assessment in the treatment of OSAS patients.

LF Drager (2009) [206] reported a study that was designed to evaluate the characteristics and predictors of Obstructive sleep apnea (OSA) in patients with hypertension. Consecutive patients with established diagnosis of hypertension were included for the study. All participants were submitted to the Berlin Questionnaire, daytime sleepiness somnolence (Epworth Sleepiness Scale), full Polysomnography and clinical evaluation. Authors analyzed the sensibility and specificity of the Berlin Questionnaire, Epworth Sleepiness Scale, traditional risk factors for OSA (including age, male sex and obesity), presence of Metabolic Syndrome and Resistant Hypertension to predict the presence of OSA. Based on the inferences, it was concluded that Berlin Questionnaire, age and the presence of Metabolic Syndrome were the independent predictors of OSA in patients with hypertension. These findings, according to the authors, may help to increase recognition, diagnosis and treatment of hypertensive patients with concomitant OSA.

Yumino et al (2009) [207] reported a study to determine, in patients with HF, the prevalence and predictors of obstructive and central sleep apnea (OSA and CSA respectively) and the influence of changes in heart failure (HF) therapy on prevalence. A total of 218 HF patients with left ventricular ejection fraction (LVEF) ≤45% underwent sleep studies in this study and were classified as having moderate to severe sleep apnea (apnea-hypopnea index ≥15/hours of sleep, either OSA or CSA), or mild to no sleep apnea. The prevalence of moderate to severe OSA was 26% and of CSA was 21%. Predictors of OSA were older age, male sex, and greater body mass index, and of CSA were older age, male sex, atrial fibrillation, hypocapnia, and diuretic use. It was concluded from the data that OSA and CSA remain common in patients with HF, despite increases in b-blocker and spironolactone use.

Danny et al (2009) [208] reported a manuscript that focused on the underlying mechanisms contributing to sleep-disordered breathing. According to the authors, Obstructive sleep apnea (OSA) is the most common sleep-related breathing disorder and is characterized by repetitive narrowing or collapse of the pharyngeal airway during sleep. Conversely, central sleep apnea (CSA), highly prevalent in congestive
heart failure, is distinguished by a lack of drive to breathe during sleep, resulting in repetitive periods of insufficient ventilation. The brief review summarized the etiology and understanding of OSA and CSA pathophysiology and the role that the cardiovascular system may play in contributing to disease pathology and highlighted the likely substantial overlap that exists between the various forms of sleep-disordered breathing. The authors summarized stating the importance of understanding the underlying mechanism of apnea.

Debra et al (2009) [209] reported a study that was designed to determine if age specific differences in obstructive sleep apnea are present in children. The retrospective chart review study included the records of children (1–18 years of age) with obstructive sleep apnea diagnosed by overnight Polysomnography. Children included in the study also had evidence of adenotonsillar hypertrophy and had no other co-existing medical problems. Overnight Polysomnography was performed in all children. Apnea–hypopnea index (AHI), baseline and lowest O2 saturation, baseline and peak end tidal CO2, and total number of obstructive apneas, hypopneas, central apneas and mixed apneas were measured during each polysomnogram. Children were subdivided into the following age groups: 1–2, 3–5, 6–11 and 12–18 years. Polysomnograms were classified into normal, mild, moderate and severe categories. It was concluded from the data obtained that there is a predilection for children less than 3 years of age to have more severe obstructive sleep apnea as documented by Polysomnography. Central apnea also appears to be more common in this age group. These findings were explained by the author as due to anatomic and physiologic differences related to age necessitating a period of observation following adenotonsillectomy in younger children.

Deborah et al (2009) [210] reported a manuscript that reviewed the literature on pediatric OSA, its pathophysiology, current treatment options, and recognized approaches to perioperative management of these young and potentially high-risk patients. The authors stated that Obstructive sleep apnea syndrome (OSA) affects 1%-3% of children. Children with OSA can present for all types of surgical and diagnostic procedures requiring anesthesia, with adenotonsillectomy being the most common surgical treatment for OSA in the pediatric age group. Thus, it is imperative, according to the authors, that the anesthesiologist be familiar with the potential
anesthetic complications and immediate postoperative problems associated with OSA. The significant implications that the presence of OSA imposes on perioperative care have been recognized by national medical professional societies, as reported by the authors. The American Academy of Pediatrics, reportedly, published a clinical practice guideline for pediatric OSA in 2002, and cited an increased risk of anesthetic complications, though specific anesthetic issues were not addressed. In 2006, the American Society of Anesthesiologists, reportedly, published a practice guideline for perioperative management of patients with OSA that noted the pediatric-related risk factor of obesity, and the increased perioperative risk associated with adenotonsillectomy in children younger than 3 yr. However, management of OSA in children younger than 1 yr-of-age was excluded from the guideline as stated by the author, as were other issues related specifically to the pediatric patient. Hence, authors summarized stating that many questions remain regarding the perioperative care of the child with OSA.

Evelyn et al (2009) [211] reported a study that intended to determine if Obstructive Sleep Apnea - quality-of-life questionnaire (OSA-18) is an accurate measure for the detection of moderate-to-severe OSA. Children who were referred for evaluation of suspected OSA and who had a nocturnal pulse oximetry study were included in the cross-sectional study. The results of the oximetry study were interpreted by using the McGill oximetry score (MOS). Abnormal scores were consistent with moderate-to-severe OSA. Demographic and medical data were analyzed in addition to the OSA-18 results. Sensitivity and negative predictive values for the OSA-18 to detect an abnormal MOS were estimated. Logistic regression analyses with MOS as the dependent variable and the OSA-18 score, age, gender, co-morbidities, and race as independent variables were also done. It was concluded from the data obtained that among children who are referred to a sleep laboratory, the OSA-18 does not accurately detect which children will have an abnormal MOS and cannot be used to exclude moderate-to-severe OSA. The OSA-18 should not be used in the place of objective testing to identify moderate-to-severe OSA in children.

Glenn (2009) [212] conducted a study to evaluate the efficacy of a protocol designed to prevent post-adenotonsillectomy airway obstruction in small children with obstructive sleep apnea. Children with sleep study proven obstructive sleep
apnea or children under the age of 3 years with clinically suspected obstructive sleep apnea were treated according to a protocol that included: (1) rapid bloodless tonsillectomy; (2) repeated release of the tonsillar retractor; (3) avoidance of uvular edema; (4) routine intra-operative intranasal oxymetazoline, and placement of nasal airway; (5) extended recovery room observation. Primary outcome measures were (1) avoidance of unexpected intensive care unit admission; (2) post-extubation pulmonary edema; (3) aspiration pneumonia. It was concluded from the study that most cases of post-extubation pulmonary edema and pneumonia can be avoided in young children and those with mild-to-moderate obstructive sleep apnea following a protocol that anticipates and avoids precipitating causes of upper airway obstruction.

Richard (2009) [213] reported a review that discussed the potential mechanisms by which sleep apnea might directly lead to nocturnal arrhythmias because of its associated acute physiologic effects on the cardiovascular system during sleep. It was summarized from the review that sleep apnea may immediately modulate the autonomic nervous system during sleep in a manner that is likely proarrhythmic and may be linked to nocturnal sudden cardiac death.

Wojciech et al (2009) [214] evaluated the microvascular endothelial function (MVEV) in OSAS and the impact on MVEF of 2 months of treatment with continuous positive airway pressure (CPAP) and mandibular advancement device (MAD). Microvascular reactivity was assessed using laser Doppler flowmetry combined with acetylcholine (Ach) and sodium nitroprusside (SNP) iontophoresis in 24 OSAS patients and 9 control patients. In 12 of the 24 OSAS patients, microvascular reactivity was reassessed after 2 months of CPAP and MAD using a randomized cross-over design. It was concluded that an impairment of MVEF in OSAS is related to OSAS severity. Both CPAP and MAD treatments were associated with an improvement in MVEF that could contribute to improve cardiovascular outcome in OSAS patients.

Luiza et al (2009) [215] evaluated a retrospective group of upper airway resistance syndrome (UARS) patients to determine the evolution of UARS over time and the relationship between clinical evolution and subjects phenotype. Investigations were performed in 30 patients, in whom UARS was diagnosed between 1995 and
2000 by the use of full polysomnography (PSG) without oesophageal pressure (Pes) measurement. It was concluded that that UARS is part of a clinical continuum from habitual snoring to obstructive sleep apnoea–hypopnoea syndrome (OSAHS). Progression from UARS to OSAHS seems to be related to an increase in the BMI.

Lora and Fiona (2009) [216] reported a study that focused on the muscular actions leading to tongue protrusion under conditions where protrusion is either impeded (i.e., quasi-isometric contractions) or unimpeded (simple displacement). The authors recorded electromyographic (EMG) and single motor-unit activities from the extrinsic protruder muscle, the genioglossus, as well as the EMG of intrinsic protruder muscles in healthy human subjects that performed impeded or unimpeded protrusion tasks. It was concluded that that protrusion of the human tongue requires activation of the genioglossus and intrinsic protruder muscles, with the former more important for establishing anterior–posterior tongue location and the latter playing a greater role in the generation of protrusive force.

Görkem and Eric (2009) [217] hypothesized that there is a vicious cycle between Obstructive sleep apnea (OSA) and laryngopharyngeal reflux (LPR) disease and intended to evaluate the association between the two. It was concluded based on the study that OSA and LPR disease may be related through a vicious cycle whereby increased respiratory effort specifically contributes to reflux of gastric contents that, in turn, contributes to progression of OSA by causing inflammation that directly narrows the upper airway and produces changes in upper airway mechanics through mucosal damage and sensory dysfunction.

Norman et al (2009) [218] reported a case series in which three cases highlighting perioperative anesthetic management issues occurring prior to and after implementation of an OSA protocol were presented. The first case documented an adverse outcome, the second represented a near miss and the third illustrated the benefit of utilizing an OSA protocol.

Reginald (2009) [219] presented a report that highlighted the scientific rationale for considering maxillomandibular advancement as the surgical treatment of choice in selected patients with obstructive sleep apnea syndrome; reviewed the
treatment planning that will identify those patients who would benefit from this procedure; reviewed the surgical techniques; and reviewed the patient outcomes after maxillomandibular advancement surgery. The author stated that patients with obstructive sleep apnea syndrome who have demonstrable retropositioning of the maxilla and mandible should be informed of maxillomandibular advancement as the primary surgical treatment for obstructive sleep apnea syndrome.

Albert et al (2010) [220] reported a study which was done to evaluate circulating adipokines concentration in children with and without obstructive sleep apnea (OSA) and to determine the effects of treatment of OSA on their plasma concentration. Children with habitual snoring and symptoms suggestive of OSA were consecutively recruited. Their parents completed a sleep apnea symptom questionnaire, and the subjects underwent physical examination and an overnight polysomnography (PSG). OSA was diagnosed if they had an obstructive apnea index > 1. Fasting serum adiponectin, leptin, and lipid profiles were taken after overnight PSG. The subjects were divided into groups as obese, nonobese, and with and without OSA for comparison. It was concluded from the study that BMI rather than OSA was the main determinant of adipokines in children.

Chan et al (2010) [221] evaluated and reported the effect of mandibular advancement splints (MAS) on upper airway anatomy during wakefulness in obstructive sleep apnoea (OSA). Patients commencing treatment for OSA with MAS were recruited. Response to treatment was defined by a ≥50% reduction in the apnoea/hypopnoea index. Nasopharyngoscopy was performed in the supine position. It was inferred that Mandibular advancement caused an increase in the calibre of the velopharynx with relatively minor changes occurring in the oropharynx and hypopharynx. An increase in crosssectional area of the velopharynx with mandibular advancement occurred to a greater extent in responders than nonresponders. The results of the study indicated that velopharyngeal calibre is modified by Mandibular advancement treatment and this may be useful for predicting treatment response.

AM Li et al (2010) [222] reported a manuscript in which natural history of mild childhood obstructive sleep apnea (OSA) was examined and factors associated with disease progression were identified. Subjects were recruited from an
epidemiological study which examined the prevalence of OSA in Chinese children aged 6–13 years. The first 56 consecutive children identified with mild OSA (apnoea-hypopnea index 1–5) were invited for a repeat assessment 2 years after the diagnosis. It was concluded based on the study that mild Obstructive Sleep Apnea in the majority of children does not resolve spontaneously. Subjects with tonsillar hypertrophy, especially boys, should be closely monitored to allow early detection of worsening OSA. Weight control should be stressed in the management of childhood OSA.

Carole L (2010) [223] reported a manuscript emphasizing on treatment indications for childhood obstructive sleep apnoea (OSA) The author stated that in children with OSA, clinical symptoms during wakefulness tend to be vague and non-specific (eg, behavioural issues) and are often attributed to other problems; excessive daytime sleepiness is relatively uncommon. Relying on diagnostic polysomnography is also problematic. An apnoea-hypopnoea index (AHI) ≥1.5/h is considered statistically abnormal.3 4 5 However, this does not mean that every child with an AHI ≥1.5/h will benefit from treatment. There is a paucity of data on the clinical outcomes of children with OSA, and virtually no data on the clinical correlates of polysomnographic abnormalities. The usual treatment for OSA in young children is adenotonsillectomy according to the author. The author summarized adding a note on clinical outcome of mild OSA and the treatment indications for the same.

Daniel (2010) [224] reported an editorial manuscript emphasizing on Obstructive Sleep Apnea (OSA) and snoring. The author added a brief note on the clinical presentations and manifestations of OSA, prevalence in general population by reviewing the literature, risk factors and predictors as stated in the literature. Guidelines for performance of diagnostic procedures and other medical treatment and surgical procedures in OSA patients were discussed. Author finally summarized with a cautionary note that there does exist the possibility of progression to substantial airway collapse with increasing and sustained levels of deep sedation in a patient population who would ostensibly be difficult to intubate. In patients with known or suspected OSA, according to the author, it would be better to avoid placing the patient in the supine position; using ventilation monitoring or capnography; avoiding the use
of combination long-acting agents for sedation induction and using single agents for sedation maintenance.

Choi et al (2010) [225] reported a study which was conducted to evaluate the effects of mandibular advancement on oropharyngeal dimension and collapsibility and reveal the predominate site of change produced by mandibular advancement in patients with Obstructive sleep apnea (OSA). Sixteen adults (13 males and 3 females) with symptomatic mild to severe OSA participated. Custom made silicone mandibular positioners were used to keep the mandible at 67% of maximum advancement. Changes in the oropharyngeal size and collapsibility with mandibular advancement were evaluated using ultrafast computed tomography taken during wakefulness and midazolam-induced sleep. Cross-sectional areas were assessed using electron beam tomography at 4 levels: high retropalatal (HRP), low retropalatal (LRP), high retroglossal (HRG), and low retroglossal (LRG). It was concluded from the findings that Mandibular advancement increases oropharyngeal diameter and decreases oropharyngeal collapsibility during midazolam-induced sleep respiration at the retropalatal as well as the retroglossal region in most patients with OSA.

Owens et al (2010) [226] reported a study that was designed to 1) measure the change in end-expiratory lung volume (EELV) that occurs during the brief pressure drops of passive Pcrit (pharyngeal critical closing pressure) measurement, 2) determine if the EELV change is different between obese Obstructive sleep apnea (OSA) subjects and controls, and 3) assess the effect of a given change in EELV on the Pcrit in both groups. It was concluded from the findings that important lung volume changes do occur during the passive Pcrit maneuver. However, on average, there was no difference in lung volume change for a given Continuous positive airway pressure (CPAP) change between obese OSA subjects and controls. Pcrit was altered substantially by changes in lung volume. The authors further stated that the findings of their study supported the role of lung volumes in the pathogenesis of OSA and suggested that lung volume changes should be a consideration during assessment of pharyngeal mechanics.

Lisa et al (2010) [227] reported a review manuscript in which they stated that Obstructive sleep apnoea (OSA) is a highly prevalent condition with proven neurocognitive and cardiovascular consequences. The authors briefly reviewed the
existing literature for OSA pathogenesis in adults and highlighted the potential role of genetics in influencing key OSA pathophysiological traits.

Jee & Christian (2011) [228] conducted a study that aimed to use clinical scales in a standardized fashion in evaluating the frequency of a high and narrow hard palate and/or small and retroplaced mandible in children with polysomnographically demonstrated sleep-disordered breathing (SDB). Data on demographics, reason for referral, sleep history, Mallampati scale, size of the tonsils (Friedman scale), bite occlusion (dental positioning), and correlating clinical presentation and comparative physical exam of nasomaxillary and Mandibular features (using subjective grading scales) were collected, as were results of pre- and post- treatment polysomnography. Data from 400 children were analyzed. It was concluded from the study that non-obese children with SDB had different initial clinical complaints based on age. Independently of age, facial anatomic structures limiting nasal breathing and those considered to be risk factors for SDB were commonly seen in the total group. Furthermore, it was stated by the authors that clinical assessment of craniofacial features considered as risk factors for SDB and more particularly a Mallampati scale score of 3 or 4 can be useful in identifying children who may be more at risk for limited response to adenotonsillectomy, suggesting a subsequent need for post-surgery Polysomnography.

Amir et al (2012) [229] reported a study that was conducted to evaluate an automated analysis for Sleep-disordered breathing (SDB) detection using signals derived from a pulse oximeter attached to the patient’s finger. The authors compared event detection and classification obtained by full polysomnography (“the gold standard”) and by the automated new algorithm - sleep apnea algorithm (SAA) system in 74 subjects. It was inferred from the study that the SAA can conveniently provide an acceptable analysis of sleep related and/or cardiac-related breathing disorders, thereby suggesting that it may serve as a future platform for screening and diagnosis of SDB in patients with significant cardiovascular disease.

Eiseman et al (2012) [230] analyzed polysomnography and clinical features available from the Sleep Heart Health Study. It was inferred by the authors based on the findings from the study that clinical prediction of sleep apnea may be feasible with easily obtained demographic and electrocardiographic analysis, but the utility of the Epworth is questioned by its minimal relation to clinical, electrocardiographic, or polysomnographic.