Causes of unsatisfactory results in treatment of snoring and obstructive sleep apnea

Essay

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By

Ahmed Ahmed Mohamed Kamel

M.B., B.Ch.

Supervised by

Prof. Dr Ahmed Abd-Elaal Elsalamawy

Professor of otorhinolaryngology

Faculty of medicine

Cairo University

Lect. Dr Mohamed Mosleh Ibrahim Khalil

Lecturer of otorhinolaryngology

Faculty of medicine

Cairo University

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Abstract

Obstructive sleep apnea (OSA) is a disorder caused by repetitive occlusion of the upper airway during sleep. It can be treated by multiple therapeutical options including medical, behavioral and surgical.

Nasal continuous positive airway pressure (CPAP) is the treatment of choice of OSA however; the compliance of the patients is very low. Surgery is used as an alternative to CPAP. There are many surgical modalities in the treatment of OSA but neither one of them have accomplished complete success. The success rate of uvulopalatopharyngoplasty which is the most famous operation in the treatment of snoring and obstructive sleep apnea does not exceed 40%.

In this study we aim at highlighting the different causes of failure of the different treatment modalities of OSA.

Keywords: Obstructive sleep apnea, snoring, CPAP, surgical treatment, uvulopalatopharyngoplasty, success.

Abbreviations

OSA: Obstructive sleep apnea

OSAS: Obstructive sleep apnea syndrome

SAS: Sleep apnea syndrome

CPAP: Continuous positive airway pressure

UPPP; U3P: Uvulopalatopharyngoplasty

AHI: Apnea hypopnea index

AI: Apnea index

PSG: Polysomnography

REM: Rapid Eye Movement

EMG: Electromyography

CNS: Central Nervous System

ECG: Electrocardiography

ESS: Epworth Sleepiness Scale

SDB: Sleep disturbed breathing

QOL: Quality of life

MSLT: Multiple Sleep Latency Test

UARS: Upper Airway Resistance Syndrome

RDI: respiratory disturbance index

RERAs: respiratory event-related arousals

TRDs: Tongue Retaining Devices

MRDS: Mandibular Repositioning Devices

biPAP: Bi-level positive airway pressure

ESS: Endoscopic Sinus Surgery

LAUP: Laser-assisted uvulopalatoplasty

TCRFTA: Temperature-controlled radiofrequency tissue ablation

GA: Genioglossus advancement

MMA: Maxillomandibular Advancement

LMG: Laser midline glossectomy

EDS: Excessive Daytime Sleepiness

HM: Hyoid Myotomy

BMI: Body Mass Index

OHS: Obesity-hypoventilation syndrome

AN: Autonomic Neuropathy

(IGF)-I: Insulin-like growth factor

CT: Computed Tomography

FPP: Friedman Palate Position

LAST: Lowest Oxygen Saturation

LA-UPPP: Laser-assisted Uvulopalatopharyngoplasty

Contents

List of figures

List of tables

Introduction

Snoring is an undesirable sound that originates from the soft tissues of the upper airway during sleep ***(Young et al., 1993)***.

Obstructive sleep apnea (OSA) syndrome is a relatively common but fatal disorder affecting 4% of the adult male and 2% of female population and it represents a more severe aspect of snoring as it usually involves more than one level of obstruction ***(Young et al., 1993)***.

Obstructive sleep apnea is a condition characterized by repetitive episodes of complete or partial obstruction of the upper airway leading to cessation or reduction of airflow during sleep ***(Remmers et al., 1978)***.

Athough application of nasal continuous positive airway pressure (CPAP) is a safe and effective treatment and is prescribed for the treatment of OSA in approximately 80% of patients with OSA in United States, a low compliance rate of the therapy (50% at best) attenuates its usefulness ***(Aloia et al., 2005)***.

It was well established that the retropalatal region was diagnosed as the most common site of obstruction in patients with snoring and obstructive sleep apnea (OSA) ***(Metes et al., 1991)***. Consequently, uvulopalatopharyngoplasty (UPPP) that consists of removal of the palatine tonsil, uvula, a portion of the soft palate, and the lateral pharyngeal wall is the most common surgical procedure for the treatment of OSA ***(Shepard et al., 1990)***.

The hypopharyngeal space has been found to be another important area of obstruction. Traditionally, maxillary-mandibular advancement has been used to expand this level of airway obstruction. Given the morbidity of such a procedure, other operations have been proposed, including genioglossus advancement, hyoid advancement, and base of tongue reduction using radiofrequency energy ***(Riley et al., 2000)***.

Uvulopalatopharyngoplasty was first described by ***Fujita et al,*** in ***1981***. However, using this technique, failure rates are reported from 30% to 90% ***(Maisel et al., 1992; Cahali, 2003)***.

A systematic review of the literature in 1996 identified a 42% success rate in published studies using polysomnographic outcomes of an apnea hypopnea index (AHI) of less than 20 or an apnea index of less than 10 events per hour ***(sher et al., 1996)***. Better success rates remain elusive despite attempts to improve selection and alternative techniques.

Although subjective improvement of symptoms including excessive daytime sleepiness and snoring have been acceptable ***(Friberg et al., 1995)***, the response rate on objective assessment based on polysomnographic (PSG) results has been no greater than 50% ***(sher et al., 1996)***. To improve the response rate, many modifications have been attempted; however, the results have been mixed ***(O'Leary et al., 1991; Fairbanks, 1999).***

The multiplicity of surgical modalities described for OSA suggests that no clear surgical approach has been widely accepted for the management of this disease.

Most of the studies on various surgical approaches to OSA focus on a single surgical modality; most studies assume that the patients with OSA are a homogeneous group with the same type of pathology.

Kao et al., (2003) believe that OSA is a multilevel, multifactorial disease process creating a very heterogeneous patient population. To improve the surgical cure rates for OSA, the entire upper airway must be brought under inspection, and each level of obstruction should be identified. The treatment plan should potentially include nasal, oropharyngeal/palatal, and hypopharyngeal measures to improve the airway.

Many combinations of surgical procedures coexist in aim to improve the success rates including; 50% success rate in UPPP and tongue-base radiofrequency, 42-59% in UPPP and midline glossectomy, 35-77% in UPPP and genioglossus advancement with or without hyoid myotomy and suspension. The most impressive results of 90% or more occur with combined UPPP, genioglossus advancement, and maxillary-mandibular advancement ***(Vilaseca et al., 2002)***.

The different methods of identifying the level of collapse including fiberoptic airway endoscopy with the Müller maneuver and cephalometrics, etc all give contradictory data which is not surprising given the fact that these static measurements of bony and soft tissue anatomy most likely do not reflect the dynamic changes in pressure that result in airway collapsibility ***(Yao et al., 1998)***.

Multiple methodological issues hamper attempts to assess and improve surgical success rates. Differences in patient anatomy, surgical technique, and variable methods of patient selection make meaningful comparisons of equivalent patient groups difficult. Lack of comparative or randomized studies, in turn, contributes to uncertainty about surgery's effectiveness.

The etiology of surgical failure is multifactorial, including medical factors like obesity and hormonal disturbances, errors in the diagnosis of the site of the obstruction as there is usually more than one level of collapse and errors in the choice of the suitable surgical procedure that gives a higher and prolonged success rate as there are multiple choices of surgical procedures even for the same level of collapse.

In this study, we aim at spot lights on the different factors contributing to the failure in the treatment of OSA and conclude the different methods of assessment of surgical success.

Pathophysiology of sleep apnea syndrome

The almost continuous patency of the upper airway during wakefulness and sleep under normal conditions is attributed to the morphology of the upper airway. The nasal passage is a non-collapsible conduit due to its cartilaginous and bony architecture. Similarly, the larynx and extrathoracic trachea have cartilaginous framework. However, the pharynx lacks a strong structural support, and thus potentially collapsible. The stability of the pharynx and by implication the upper airway is significantly dependent on the action of the pharyngeal muscles, which are normally activated in rhythmical fashion during inspiration**.** ***(Strohl et al., 1980).***

The pathophysiology of snoring and obstructive sleep apnea is determined by a number of interrelated factors (table 1)

(Table 1) Known and suspected predisposing conditions for obstructive sleep apnea

Condition	Examples	Contribution
Obesity, body fat distribution	Adult obesity, Prader-Willi syndrome	Complex and ill-defined
Race/genetics		?Anatomical similarity
Age		?Tissue laxity
Male gender		Unclear
Alcohol, sedatives, analgesics, anaesthetics		Muscle relaxation, depressed arousal
Smoking		?Chronic nasal congestion, pharyngeal oedema
Nasal obstruction	Septal deviation, chronic nasal congestion	Increased pharyngeal negative pressure
Pharyngeal obstruction	Tonsillar and adenoidal hypertrophy	Increased pharyngeal negative pressure

Cranio-facial abnormality	Down's, Pierre-Robin, Treacher-Collins, Apert's, Crouzon's, Beckwith-Wiedemann, achondroplasia, acromegaly, fragile-X	Mid-face hypoplasia, macroglossia or micrognathia
Endocrine/Metabolic	Hypothyroidism, androgen therapy, Cushing's	Upper airway infiltration or myopathy, obesity
Neuromuscular disorders	Poliomyelitis, myotonic dystrophy, dysautonomia, tetraplegia	Disordered pharyngeal neuromuscular function
Connective tissue disorders	Marfan's	Abnormal upper airway connective tissue
Storage diseases		Macroglossia
Chronic renal failure		Unclear

(Loadsman and Hillman, 2001)

The onset of the inspiration triggers a reflex increase in the EMG activity of the pharyngeal dilator muscles (e.g. genioglossus, geniohyoid, palatoglossus, and medial pterygoids). These muscles are activated rhythmically during daytime respiration and hold the airway open preventing collapse. The tensor palatini retracts the posterior pharyngeal wall, thus maintaining pharyngeal patency during nasal breathing, the primary route during sleep ***(Gleeson et al., 1986).***

In common with other skeletal muscles they become hypotonic during sleep, especially rapid eye movement (REM) sleep. However, in patients with obstructive sleep apnea EMG activity is further decreased during an obstructive episode and can sometimes completely disappear. An excessive reduction in upper airway muscle tone or any incoordination between reflex EMG activity in these muscles and onset of inspiration results in an increased susceptibility of the upper airway to collapse ***(Wiegand, 1990).***

Upper airway resistance is determined by the diameter of the airway, which in turn is dependent on the patient's craniofacial morphology and anatomical defects of space occupying lesions. Anything narrowing the airway will increase the resistance. In order to pull air through the narrowing, greater respiratory effort will be needed resulting in the generation of a higher negative intra-luminal pressure which, in turn, encourages collapse. As the airway narrows, the rate of airflow through it increases and, on the basis of Venturi principle, there is a further drop in the intraluminal pressure again encouraging collapse.

Increased compliance of the pharyngeal tissues results in collapse at a lower negative pressure.

Despite the long list of causes (mentioned before) most adult patients with obstructive sleep apnea, have no evident predisposing abnormality.

Many adult patients with snoring and obstructive sleep apnea, while not having obvious skull base and facial skeletal anomalies, have more subtle anatomical variations which mean that they fall at one end of the normal distribution of pharyngeal dimensions. Thus, they suffer from obstructive sleep apnea despite having no evident craniofacial abnormality ***(Tangel et al, 1992).***

The impaired activities of the dilator muscles result in ineffective splinting of the upper airway, thus increasing the transmission of the subatmospheric intrathoracic pressure into the upper airway**.** The interplay of the impaired upper airway muscle activity and the unopposed subatmospheric inspiratory pressure into the upper airway resistance provides a favorable ground for airway collapse during sleep ***(Kuna, Sant's Ambrgio, 1991).***

The deposition of fat predominantly in the lateral walls of the pharynx results in decreased patency of the pharynx. This increases the risk of collapse particularly during sleep when there is relaxation of the pharyngeal dilator muscles. Similarly, increased intraluminal pressure due to superficially located fat masses, further worsens the delicate balance between transmural pressure and compliance of the pharyngeal wall ***(Delberto et al, 1994).***

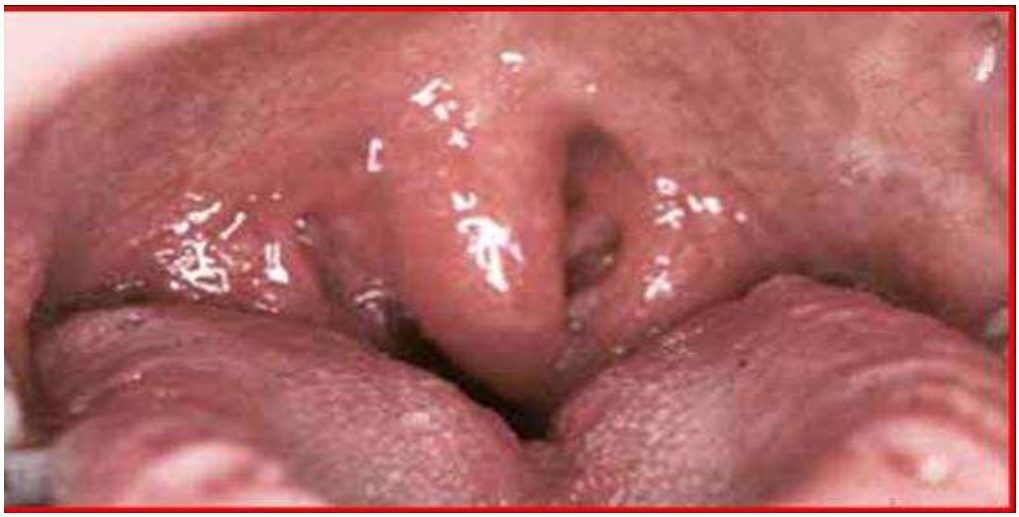


Fig (1): Obstructed airway (Helfear and Wilson, 1994)

The clinical features and complications associated with the obstructive sleep apnea syndrome essentially result from the two main effects of the obstructive episode, which are hypoxia and the generation of high negative intrathoracic pressure.

I-Hypoxia

During an apneic episode there is no air flow into the lungs, thus if the episode is long enough the patient will become hypoxic and hypercapnic. The degree of hypoxia will be determined by the duration of the apneic event, lung volume (which is reduced in obesity) and any coexistent neuromuscular or cardiopulmonary disorders ***(Carlson et al, 1994).***

Hypoxia is associated with a rise in sympathetic output and catecholamine production and the resulting peripheral vasoconstriction causes transient pulmonary and systemic hypertension. However, the relationship between night-time hypoxia and established systemic hypertension is controversial ***(Carlson et al., 1994).***

Long standing pulmonary hypertension was thought to develop only in patients with coexisting chronic obstructive airway disease but it has been found in patients without respiratory problems ***(Laks et al., 1997).***

Hypoxia can cause a variety of cardiac arrhythmias, which range from bradycardias to ventricular ectopies. During an obstructive episode the effort of trying to inspire against a closed glottis results in increased vagal activity with subsequent bradycardia. The presence of hypoxia significantly worsens this bradycardia. Polycythemia associated with prolonged hypoxia is thought to develop only in patients with coexistent daytime oxygen desaturation ***(Carlson J.T. et al., 1994).***

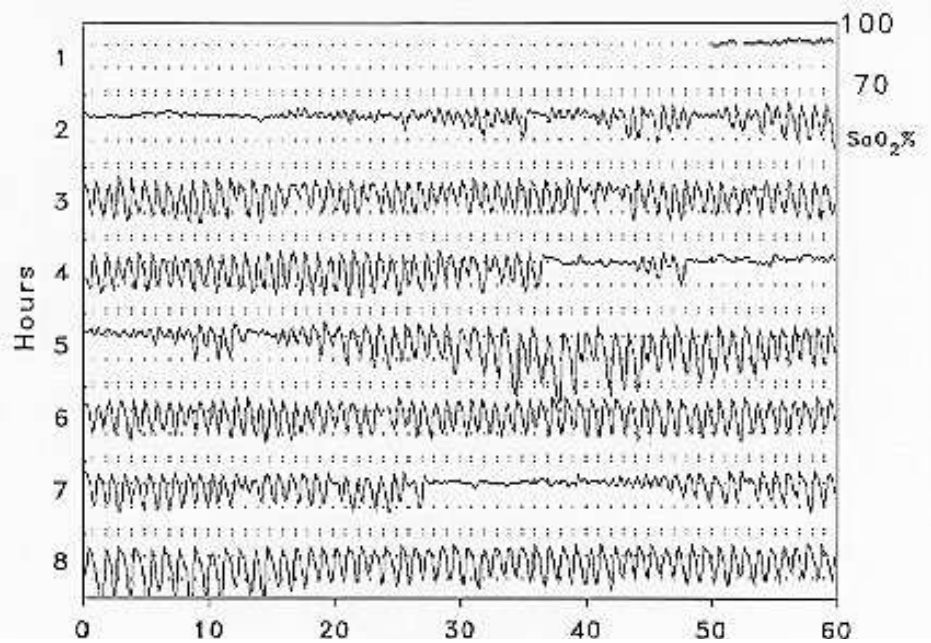


Fig (2) overnight arterial oxygen saturation monitoring.

A patient with severe obstructive sleep apnoea. Recording shows multiple dips in oxygen saturation ***(Carlson et al., 1994).***

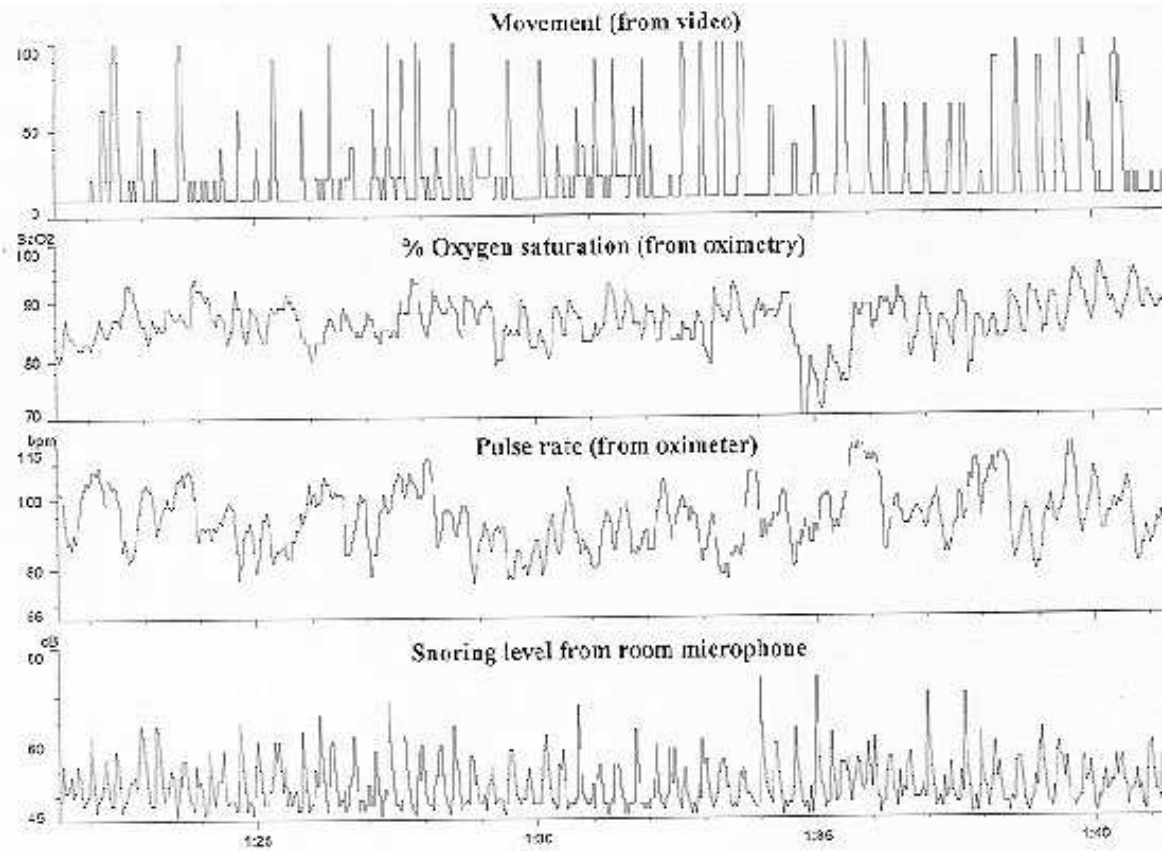
II-Negative intrathoracic pressure

A-Arousals

The efforts of the respiratory system to overcome upper airway obstruction result in the generation of high negative intrathoracic pressure. The main symptomatic consequence of this high negative pressure is that they stimulate arousals and the termination of an obstructive episode is thought to depend heavily on arousal. The place of hypoxia or hypercapnia as mechanism of arousal remains unclear. It was thought that arousal was caused by hypoxia stimulating the carotid body receptors or hypercapnia stimulating the CNS receptors. However, it is now recognized that obstructive episodes can result in arousal without there being any associated hypoxia or hypercapnia. The main factor proceeding arousal is the high level of negative intrathoracic pressure generated by increased respiratory effort associated with airway obstruction ***(Vinchen et al, 1987).***

Hypoxia and hypercapnia will contribute to this by stimulating increased ventilatory efforts. When the degree of inspiratory effort reaches a certain threshold in a given subject arousal occurs, resulting in a return of tone to upper airway muscles and the obstruction clears. The patient is often unaware of these arousals, which can occur many hundreds of times throughout the night (Fig.3). The effect of repeated arousals is a very disturbed sleep pattern, which is reflected in symptoms of excessive daytime sleepiness, one of the commonest symptoms of obstructive sleep apnea. Excessive daytime sleepiness also occurs in patients with higher upper airway resistance syndrome and in some patients who snore loudly without any obstructive episodes. Presumably these patients are generating sufficiently high negative intrathoracic

pressure from their partial obstruction to stimulate arousals frequently enough during the night to disturb sleep. The threshold for arousal is elevated during REM sleep such that obstructive episodes are more prolonged and severe during this sleep stage **(Bonnet, 1985).**



(Fig 3) Monitoring of Arousals

This patient with OSA shows the typical snore/silence/snore pattern in association with body movements and pulse rate rises indicating sleep disturbance, and falls in oxygen saturation showing that the obstruction has been bad enough to interfere with gas exchange ***(Bonnet, 1985).***

B-Cardiovascular Consequences:

The generation of high negative intrathoracic pressure has cardiovascular consequences. The heart has to work hard to pump against the subatmospheric pleural pressure as well as against an increased pulmonary and systemic arterial resistance. The dilating force on the heart will tend to aspirate venous blood into the right heart but at the same time, increases the preload of the left heart and there is evidence that the heart enlarges during an obstructive episode ***(Lavie et al., 1995).***

When the obstruction clears and the negative pressure is released the heart will continue to pump against the high peripheral resistance until this also returns to normal. In those patients left ventricular wall thickening has been observed. The increase in cardiac work may be reflected by ischemic changes that are sometimes seen as an ECG recording during obstructive episodes ***(Koskenvuo et al., 1987).***

Carlson et al., 1994; Hu et al., 1999, reported a high prevalence rate of hypertension among patients with obstructive sleep apnea**.**

` The most serious consequence of untreated obstructive sleep apnea is increased cardiovascular morbidity and mortality. This is the reason why it is so important to exclude or establish the diagnoses. There is also an increased incidence of angina and myocardial infarction in snorers ***(Hung et al., 1990).***

C-Social consequences

As well as daytime sleepiness the combination of disturbed sleep and chronic hypoxia is also responsible for other physiological and intellectual changes the patients with obstructive sleep apnea may exhibit. These include personality changes, poor memory and difficulty in

concentrating. These problems combined with loud snoring can result in serious social consequences. Examples include marital stress sometimes leading to divorce, interference with the performance of the patient's job possibly leading to loss of employment and an unwillingness to stay in hotels. The more serious is the increased incidence of accidents, especially road traffic accidents because of daytime sleepiness and poor concentration ***(Kales et al, 1985*).**

Clinical features and differential diagnosis

Snoring is the cardinal symptom of the obstructive sleep apnea syndrome as essentially all patients with this syndrome snore. Of course, not all patients who snore have obstructive sleep apnea syndrome. By far the commonest associated symptom is excessive daytime sleepiness ***(Young et al, 1993)*.**

1-Snoring and obstructive episodes

The classic snoring noise is due to partial upper airway obstruction causing turbulent airflow with resultant vibration of the soft palate and uvula, the faucial pillars, the lateral pharyngeal walls and sometimes the tongue base level or is associated with a high upper airway resistance syndrome in which the noise has a different, less vibrant quality. In the obstructive sleep apnea syndrome the bed partner will describe periods of very loud snoring interspersed with the periods of silence during apnic episodes. The apnic episodes unusually last 20-60 seconds but can last up to a couple of minuets. Most partners will not usually wait this long time and will rouse the patient. During an episode there is no airflow at the nose or mouth yet the patient can be seen to be making marked

respiratory efforts associated with tracheal tug, paradoxical abdominal and chest movements and suprasternal recession. Respiratory efforts become more vigorous as time passes until the silence is broken by a loud snoring, grunting noise followed by some deep breathes as the patient clears the airway. The snoring then resumes before the next apnic episode begins. Although, the minimum criteria for diagnoses of obstructive sleep is > 5 apnic episodes per hour some patients will experience many hundreds of episodes throughout the night and those that are symptomatic will usually have an apnic index of > 20 ***(Issa, Sullivan, 1982).***

2-Excessive daytime sleepiness

This symptom can vary from a feeling of being generally tired to actually falling asleep during important activities. In the mild form, patients will complain of falling asleep during activities such as watching television or trying to read a novel. More seriously they may fall asleep during important events such as meetings and even during face – to – face conversation and most seriously while driving or operating machinery. Patients are often not good at assessing their degree of daytime sleepiness and talking to partners may give a more accurate indication of the degree of the problem ***(Wittig, 1992).***

The Epworth sleepiness scale is a self – administered questionnaire which provides a measurement of the patient's general level of day time sleepiness. Epworth sleepiness scale scores increase with the severity of obstructive sleep apnea and are more closely related to the apnea index than the degree of hypoxemia ***(Johns, 1991)***.

Normal patients score between 2 and 10 whereas total Epworth sleepiness scale scores greater than 16 are found only in patients with

moderate or severe obstructive sleep apnea, narcolepsy or idiopathic hypersomnia. However, small minorities of patients with moderate or severe obstructive sleep apnea score in the normal range so a lower score alone cannot exclude obstructive sleep apnea. As with many assessments related to obstructive sleep apnea the results should not be interpreted in isolation. Other conditions should be considered in patients who fall asleep during the day. A rare condition causing daytime sleepiness is narcolepsy in which there are episodes of sudden onset of sleep usually lasting about 15 minutes. This disease usually starts between the ages of 10-20 years and affects both sexes equally. It can be associated with cataplexy, sleep paralysis and hallucinations at the onset of sleep or when waking up.Snoring is not usually a feature and the diagnosis is confirmed using EEG, which shows REM stage at the onset of sleep ***(Johns, 1992).***

The Epworth sleepiness scale questionnaire is applied in sleep centers as follows:

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired. This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you (table 2). Use the following scale to choose the most appropriate number for each situation

0 = would never doze

1 = slight chance of dozing

2 = moderate chance of dozing

3 = high chance of dozing

Table (2) Epworth sleepiness scale

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

(Johns, 1992)

3-Morning headaches

These are often included as a common symptom ***(Wittig, 1992).***

4-Intellectual deterioration and personality changes

As with excessive daytime sleepiness a more accurate picture will be obtained from the partner ***(Kales et al, 1985).***

Physical signs

- Obesity**:** most patients with symptomatic obstructive sleep apnea are obese however many patients with obstructive sleep apnea are not significantly overweight. Obesity is defined as body mass index >30 kg /m^2.
- Narrowing of the lateral airway walls.
- Enlarged (kissing) tonsils.

- Retrognathia or micrognathia.
- Long or thick uvula.
- High arched hard palate.
- Soft palate edema or erythema.

(Loadsman and Hillman, 2001)

DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA

A patient with a unilateral sensorineural hearing loss may have an acoustic neuroma, a potentially life-threatening condition which should be excluded. In the same way a patient presenting with snoring may have obstructive sleep apnea syndrome another possibly life-threatening condition either directly because of its long-term cardiovascular effects or indirectly because of excessive daytime sleepiness leading to road traffic accidents or other injuries. A patient presenting with snoring one should consider the possibility of obstructive sleep apnea which cannot be reliably excluded as the basis of history ***(Viner et aI., 1991)*** , examination ***(Viner et al., 1991)***, or observation during sleep ***(Haponik et al., 1984)*** alone. The extent of any investigations will obviously be influenced by resources and locally available facili1ies. However to perform surgery, such as uvulopalatopharyngoplasty (UPPP), on a snoring patient without any attempt to exclude obstructive sleep apnea will deprive some patients of an important warning sign (i.e. snoring) that they have potentially life-threatening condition.

A condition that may well not be cured by surgery but for which there are successful alternative treatments if the condition is identified ***(Charles and Michael, 1997).***

DIAGNOSIS

History:

Snoring is a noise generated from the upper airway due to partial airway obstruction.

An apnea is the cessation of airflow at the nostrils and mouth for at least 10 seconds. The apnea index (AI) is the number of apneas per hour of sleep. Hypopnea is a reduction in tidal volume. There is disagreement

as to the exact definition of hypopnea. ***Gould et al., (1988)***, suggested that it is a 50% reduction in thoracoabdominal movement lasting for 10 seconds in the presence of continued air flow. However, some authors describe it as a decrease in airflow associated with oxygen desaturation ***(Block et al., 1979).***

The sleep apnea syndrome (SAS): 30 or more apneic episodes during a 7-hour period of sleep or an apnea index equals to or greater than 5. This is an arbitrary definition, with increasing experience of the condition, others have suggested that an apnea index of 10 ***(Fletcher et al., 1985)***, or an "apnea - hypopnea" index of 15 ***(Gould et al., 1988)*** should be present before diagnosing the sleep apnea syndrome.

The American sleep association grades sleep apnea as follows:

- Mild =5-20 apneas per hour.
- Moderate = 20-40 apneas per hour.
- Severe = > 40 apneas per hour.

Important points in the history *(Charles, 1997):*

- **Partner must be present**
- **Snoring**
- **Positional**
- **Obstructive episodes**
- **Arousals/nocturnal choking**
- **Excessive daytime sleepiness**
- **Intellectual deterioration**
- **Abnormal motor movements**
- **Morning headaches**
- **Nocturnal enuresis/Impotence**
- **Nasal obstruction**
- **Drugs**
- **Alcohol intake**
- **Cardiovascular symptoms**
- **Respiratory symptoms**
- **Thyroid symptoms**
- **Social history**

Quality-of-life scales (general and disease-specific):

In early work, global measures of health were used to assess the effect of OSA. These measures originally were designed to measure aggregate health characteristics and to provide synoptic information regarding a patient's own perception of health. The Medical Outcome Survey Short Form (SF-36) includes eight domains to measure health and well-being ***(Stewart et al, 1991).***

Briones et al. 1996 showed a correlation among the Epowrth sleepiness scale (ESS) score and vitality, role-emotional and general

health domains. Another study using the SF-36 showed improvement in energy and vitality and mental and physical functioning domains, although another measure used in the study failed to identify these effects ***(Jenkinson et al, 1997)***. Mild to moderate sleep disturbed breathing (SDB) was associated with decreased vitality measure on the SF-36, whereas severe SDB was associated with a global decrease in quality of life (QOL) ***(Baldwin et al., 2001).*** All dimension of QOL were diminished significantly on the SF-36 in patients with OSA as compared with control patients ***(Baldwin et al., 2001).***

The Obstructive Sleep Apnea Patient-Oriented Severity Index (table 3) was designed for use in the OSA Treatment Outcome Pilot Study. It involves responses to questions regarding five subscales to which importance and magnitude of effects are assigned. A symptom impact score is generated from the product of the importance and the magnitude. The OSA Treatment Outcome Pilot Study study demonstrated worth QOL in all domains except body pain ***(Piccirello et al, 1998).***

Table 3

Items on the Obstructive Sleep Apnea Patient-Oriented Severity Index

Sleep problems

1. **Trouble falling asleep**
2. **Waking during sleep**
3. **Loud/excessive snoring**
4. **Restlessness during sleep**
5. **Waking "too early" in the morning**
6. **Waking up feeling tired**
7. **Bed wetting**

Awake problems

8. **Fatigue or tiredness**
9. **Frequent yawning**
10. **Sleepiness while driving**
11. **Memory and/or concentration problems**
12. **Productivity limited at certain times of day**
13. **Often late for meetings or appointments**
14. **Participation in community, volunteer or religious activities limited**

Medical problems

15. **Amount of medical care required for OSA**
16. **Interaction of OSA with other medical problems**
17. **Travel by car to other regions of country limited because of fear of medical problem**
18. **Unable to have sexual relations because of medical problem**
19. **Financial burden as a result of illness**

Emotional and personal problems

20. **Fear going to bed**
21. **Nerves are "right on surface"**
22. **Inability to relax, always anxious**
23. **Marital strain, stress and tension**
24. **"Foul" mood**
25. **Unable to experience closeness with spouse and/or others**
26. **Lack of desire for sexual relations**
27. **Feeling that future is hopeless**

Occupational impact

28. **Competence questioned**
29. **Reliability questioned**
30. **Inability or difficulty getting new job**
31. **Loss of job**
32. **Modification in job because of excessive sleepiness**

(Piccirillo et al, 1998)

Examination:

The patient's general appearance will indicate the extent of any obesity and may suggest condition such as acromegaly or myxoedema. Height, weight, neck-circumference and blood pressure need to be recorded. Craniofacial morphology is assessed looking for retrognathia or micrognathia. Nasal examination will allow assessment of the nasal airway and reveals causes of nasal obstruction. Oral cavity examination allows assessment of the size of the tongue, soft palate, uvula and tonsils. Patients with obstructive sleep apnea often have a classic picture of an enlarged swollen edematous uvula and soft palate. One must be very cautious about seeing this as an invitation to perform a uvulopalatopharyngoplasty as this appearance can represent part of a multisegmental upper airway problem for which treatment may be preferable, so a full assessment is required before any surgery. The nasopharynx examined to exclude adenoidal tissue, polyps, cysts or tumours. The oropharynx often appears congested and there may be redundant mucosa and prominent lateral pharyngeal bands. The hypopharynx and larynx must be examined to look for any lesions that may be narrowing the airway such as enlarged lingual tonsils, cysts or tumours. A normal glottis with mobile vocal cords must be confirmed. In most patients no obvious obstructive lesion is found and in a few patients with obstructive sleep apnea the oropharyngeal inlet appears completely normal and widely patent.

Important aspects of the examination *(Charles, 1997):*

- **General appearance**
- **Weight**
- **Height**
- **Blood pressure**
- **Craniofacial morphology**
- **Nasal airway**
- **Tongue size**
- **Soft palate/uvula/tonsils**
- **Nasopharynx-adenoids/polyps/cyst/tumour**
- **Hypopharynx-lingual tonsils/vallecula, epiglottic or supraglottic cysts/tumour**

Investigations:

The reasons for investigations in this condition are to assess the patient's general condition, to differentiate between simple snoring and sleep apnea and to determine the presence, type and severity of any apneas or hypopneas and to assess the site of obstruction. The investigations that assess the patient's general condition are:

- Complete blood picture: to look for polycythaemia/anaemia/mean corpuscular volume.
- Thyroid function tests: if hypothyroidism is suspected.
- Blood gases: if there is any suspicion of daytime hypoventilation and hypoxaemia then arterial PO2 and PCO2 need to be determined.

- Electro-cardiography: if there is any suggestion of associated cardiac disease.
- Lung function tests: performed if there is suspicion of a coexisting pulmonary disorder. The finding of a "saw-tooth" pattern on a flow volume loop is thought to be very suggestive that the patient has obstructive sleep apnea.
- Chest X-ray: to detect cardiomegaly or pulmonary disorders.

The investigations to differentiate between simple snoring and sleep apnea and determine the presence, type and severity of any apneas or hypopneas and to assess the site of obstruction can be divided into those that are performed on awake patients and those performed on sleeping patients.

The introduction of specific examination modalities as Muller's maneuver which performs an awake video nasopharyngoscopy under local anesthesia and the patient is asked to perform a reverse Valsalva maneuver in order to locate the site of airway collapse ***(Friedlander et al., 2000)***. Lateral cephalometry is one of many techniques that has been used to investigate the facial characteristics of obstructive sleep apnea patients. Lateral cephalometric characteristics of the bony structure in obstructive sleep apnea patients include a retruded mandible, a low hyoid bone and large overbite ***(Pae et al., 1999)***. Cephalometric analysis of the upper airway seemed promising in predicting the outcome of surgery ***(Riley et al., 1983).*** Objective evaluation of the nasal airway by acoustic rhinometry has been shown to be helpful tool for quantifying the symptom of nasal obstruction in case where nasal factor to assess the pharyngeal, tracheal and bronchial cross-sectional areas along the airway. This method has been used extensively, particularly for the comparative

assessment of the pharynx size among snorers, nonsnorers and patients with obstructive sleep apnea syndrome. It has the advantage of being quick and non-invasive technique which is based on analyzing sound waves reflected from the oropharyngeal cavity which can be helpful in localizing the site of airway obstruction ***(Huang et al., 2000)***.

Multiple sleep latency test

The multiple sleep latency test (MSLT) evaluates degree of impairment of daytime alertness. This test involves recording the time of sleep initiation for multiple naps separated by at least 2 hours during a patient's normal waking period. This instrument can be used to diagnose upper airway resistance syndrome (UARS) or an assessment of treatment effect (***Chervin et al, 1996)***.

In the absence of UARS, the MSLT is used to diagnose narcolepsy. It generally is considered the "gold standard" for evaluating daytime somnolence and sleep latency. Moderate correlation exists between "irresistible sleepiness," which describes the sensation of being overcome by sleep, and MSLT ; however "irresistible sleepiness" failed to identify pathologic MSLT in patients with sleep disturbed breathing (SDB). ***(Rinaldi et al, 2001).***

Muller maneuver: palate, base of tongue and lateral walls

The Muller maneuver originated from attempts to evaluate various levels of upper airway obstruction. The examiner views the upper airway through the nasopharyngoscope at rest and with maximal inspiratory effort against closed nose and mouth. The base of tongue, lateral pharyngeal walls and palate are examined for collapse. The examiner rates collapsibility of each structure from 0 (minimal collapse) to 4+

(complete collapse). Muller maneuver score was shown to be correlated moderately with preoperative SDB severity and its reproducibility was verified between examiners. ***(Terris et al, 2000).***

Collapse of the palate was correlated highly with respiratory disturbance index (RDI), whereas lateral wall collapse was correlated moderately, and base of tongue collapse was not correlated. ***(yao et al, 1998)***

P close

P close is the pressure at which the upper airway collapses. This value is a significant discriminating feature between normal subjects and patients with abnormal collapsibility as is seen in OSA ***(Isono et al., 1997).***

In apneics, P close tends towards higher values than in control subjects. Airway collapse can occur at the level of the palate or tongue base. Positive P close predicted treatment effect in patients with OSA. For patients with positive P close, nocturnal oxygenation was normalized after UPPP in 27%, whereas oxygenation corrected 73% of OSA in patients with negative P close ***(Rowley et al., 1996).***

Cephalometrics

Cephalometric radiographs are obtained and evaluated in standardized manner. Relationships of different structures to one another have been assessed for predictive value in diagnosing OSA and evaluating surgical outcome. Studies correlated postoperative outcomes with increased posterior airway length, increased hyoid-mandibular length and increased posterior airway space (PAS). ***(Rayan et al., 1990)***

Li et al, (2001) report an increase in pharyngeal length and depth of 48% and 52%, respectively, after maxillomandibular advancement and report a high surgical success rate for these procedures.

Conflicting data were described by ***Yao et al, (1998)***, who found that cephalometric radiographs reflect anatomic changes postoperatively but these changes did not correlate with efficacy as measured by improvement in the apnea hypopnea index (AHI).

Polysomnography

The polysomnogram (PSG) was first described in 1974 by Holland et al. since that time, PSG has become the "gold standard" in diagnosis and follow up of sleep apnea because it provides objective on sleep and respiratory status. Originally, the only events evaluated were apnea; however, analysis has expanded to include hypopneas and respiratory event-related arousals (RERAs). The diagnosis most frequently is made on the basis of the sum of these events per hour or RDI. Information gathered includes pulse oximetry, electrocardiography, nasal or oral airflow, respiratory effort, extremity electromyography, submental electromyography, electro-oculogram, positionally dependent sleep changes, and electroencephalographic evidence of arousals ***(Coleman, 1999)***

Despite collecting information on oxygen desaturation, arousals, limb movements, sleep architecture and cardiac events, diagnosis most often is made by RDI alone. With the pressures of medical economics, a variety of polysomnographic portable technologies have evolved to decrease and improve access and ease of assessment. These studies range from fully monitored home studies to overnight oximetry, although each has limitations in the data collected. ***(Sergi et al., 1998)***

Parra et al, (1997) showed 89% concordance between AHI measured by a home device and traditional PSG. ***Kapur et al, (2000)*** reported that unattended home sleep studies were acceptable for the evaluation and diagnosis of OSA in 90% of cases.

Fibreoptic nasoendoscopy:

Borowiecki et al. (1978) used simultaneous videoendoscopy and polysomnography as research tools to try to determine what happens in the upper airway during an obstructive event. Patients with moderate to severe obstructive sleep apnea had a nasoendoscope inserted into the upper airway and then supported on a frame above the bed while they fell asleep. Video recordings of the upper airway were made throughout the night. Different patients exhibited different levels of obstruction.

However, because of restricted movement of the suspended scope and the relatively limited view, this technique was not used routinely. ***Pringle and Croft (1991)*** have devised an outpatient technique known as "sleep nasoendoscopy" which involves inducing sleep with a small dose of sedative then examining the upper airway during obstructive episodes.

Experience with this procedure has allowed the formulation of a grading system based on site of obstruction (***Pringle and Croft, 1993***). Patients who obstruct primarily at the velopharyngeal level can be identified and offered uvulopalatopharyngoplasty whereas those who have multisegmental collapse or tongue base collapse can be offered alternative treatment. The investigation is best reserved for those in whom obstructive sleep apnea has already been diagnosed on overnight sleep study, as these patients often require much smaller doses of sedative to induce sleep.

TREATMENT OF OBSTRUCTIVE SLEEP APNEA

A number of different treatment modalities for snoring and obstructive sleep apnea have been advocated, each with varying degrees of success. No one type is effective for all patients.

Treatment modalities include:

A. Non-surgical treatment.

B. Surgical treatment.

A. NON-SURGICAL TREATMENT:

1. Weight Loss:

Obesity is considered one of the major risk factors of snoring and obstructive sleep apnea as it clearly worsens the severity of both conditions. Weight loss should be encouraged in all in all obese apnea patients to decrease the mass load placed on the respiratory system, to increase the resting lung volume and to improve gas exchange ***(Emirgil and Sobol, 1973).***

Weight reduction improves levels of arterial oxygen saturation while the persons are awake and asleep, and shortens apneic periods in addition to amelioration of snoring sounds in the majority of affected obese patients ***(Pasquali et al., 1990).***

Unfortunately, voluntary loss of large amounts of weight is difficult for patients and maintaining such a weight loss is a significant problem. Patients seem to be most successful with weight when they are carefully supervised in structured programs. Surgically induced weight loss (bariatric

surgery) including intestinal bypass, gastric stapling and the use of gastric bubble has also been reported to resolve the sleep apnea syndrome ***(Peiser et al., 1984).***

2-Mechanical devices:

Various approaches have been suggested for the treatment of snoring and OSA.

I. Sleeping position devices:

Patients and bed partners commonly describe greater snoring and more frequent apnea during sleep in the supine position. It was postulated that the supine position facilitates gravity-associated relapse of the tongue against the posterior pharyngeal wall ***(sanders, 1987)***.

An old idea of taping a marble on the snorer's back to force him to sleep on his side “snore ball”, a tennis ball is put into a socket that is pinned to the back of the snorer often between the shoulder blades ***(Moran, 1987)***.

Unfortunately, snoring is purely an involuntary phenomenon, and if these devices work, it is most likely because they kept the patient from going to sleep altogether ***(Fairbanks, 1987)***.

II. Oral devices:

An oral device for the management of snoring and OSA is a small plastic dental appliance. It is worn in the mouth during sleep to prevent the oropharyngeal tissues and base of the tongue from collapsing and obstructing the airway. Most of devices may be held in place by gripping the teeth with wire clasps or with flexible plastic material of which they are constructed. This is usually a methylmethacrylate, polyvinyl or other

thermoplastic material that has been FDA-approved for intraoral use ***(Strauss, 1994)***.

Oral devices essentially function in three ways. First, by bringing the mandible and base of tongue forward or by acting as scaffolding to support a dropping soft palate and uvula. Second, by stabilizing the mandible and preventing it from opening during sleep. This assists the geniohyoid muscle in dilating the airway through protraction of the hyoid bone. Third, by altering mandibular position through downward rotation, thereby causing an increase in baseline genioglossus muscle activity which is related to maintenance of patent airway.

There are four basic types of oral devices: the soft palate lifter, the tongue retaining devices (TRDs), the mandibular repositioning devices (MRDS) (fig 4), and the tongue posture training devices. Within these types there are variations in design that also affect the workability of each. There are differences in the way of each these devices function ***(Strauss, 1994)***.

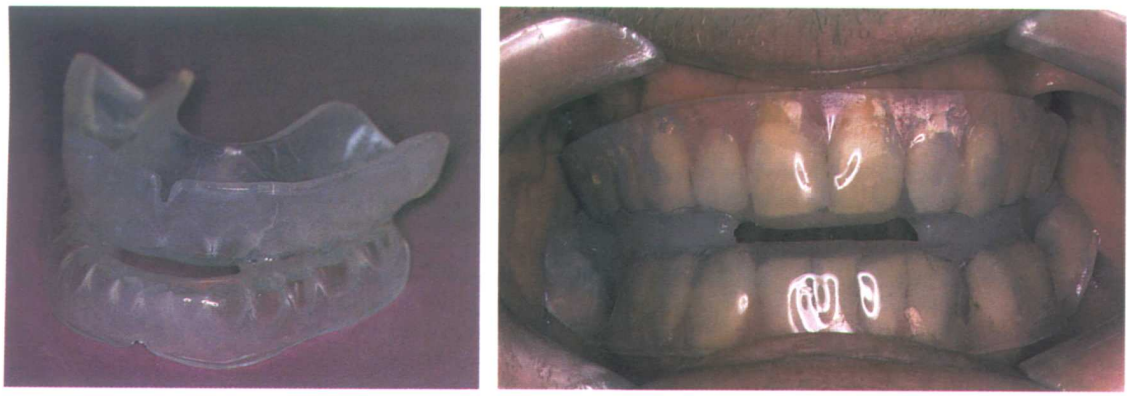


Fig (4): the mandibular repositioning devices.

III. Nasal Dilators:

The nasal valve area is the narrowest passage in the respiratory tract

causing more than half of the total resistance to nasal inspiration in healthy subjects ***(O'neill and Tolley, 1988).***

A nasal dilator applied to the area of nasal valve during sleep increase the nasal airflow and thus reducing snoring. The nostrils were dilated with a plastic nasal device (Nazovent), and the air flow through the nose increased and subsequently less negative intrathoracic pressure was needed for inspiration ***(Irvine et al, 1984)***

3. Drug thrapy:

pharmacotherapy has been suggested as treatment for those with mild sleep apnea

A. Protriptyline:

Protriptyline, a non-sedating tricyclic antidepressant, produces variable but generally beneficial results in patients with mild to moderate obstructive sleep apnea ***(Clark et al., 1979).***

Protriptyline decrease the percentage of time spent in REM sleep. Apnea is worse during REM sleep with its associated muscle relaxation. therapy during the severity of nocturnal hypoxamia by decreasing frequency of the more severe REM-related apneas ***(Brownwell et al., 1982)***.

Protriptyline has many side effects, largely because of its anticholinergic properties that frequently limit its use. These include dry mouth, urinary retention, constipation and impotence ***(Brownwell et aI., 1982).***

B. Progesterone:

Medroxy progesterone, a synthetic oral preparation, has been proposed as a possible treatment for central sleep apnea based on its central

respiratory stimulant properties. Even though it has limited effectiveness for those with central sleep apnea, it has no demonstrable effectiveness in patients with obstructive sleep apnea ***(Strohl et al., 1981).***

c. Acetazolamide:

Acetazolamide, a carbonic anhydrase inhibitor, has been shown to be helpful in treatment of central sleep apnea ***(White et al., 1982).***

Acetazolamide stimulates ventilation by increasing the hydrogen ion concentration of arterial blood which is reported to decrease apnea frequency. Apnea associated arousals and the severity of oxygen desaturation in patients with central sleep apnea ***(White et al., 1982)***.

D. Aminophylline Compounds:

Theophylline has been used effectively in children with apnea or periodic breathing, and is useful in patients with central apnea but again has not been shown to be effective for those with obstructive sleep apnea ***(Sahan et al., 1978).***

4. Electrical Stimulation of the upper airway:

Although electrical stimulation has not yet been proven to be an effective treatment of patients with obstructive sleep apnea, it is worth mentioning as a reflection of the innovative efforts that are currently on going to develop better therapies for this disorder.

One proposed mechanism for pharyngeal occlusion in obstructive sleep apnea is tongue prolapse into the pharynx, which may be due to diminished neuromuscular activity in the genioglossus muscle which is the principle tongue protrusor muscle ***(Remmers et al., 1978).***

It is possible to reduce airway resistance in anaesthetized dogs by

electrical stimulation of the genioglossus muscle. Subsequently, it is possible that application of submental electrical stimulation of the genioglossus muscle using surface electrodes and electrical stimulation of 0.5 msec (repetition rate, 50 Hz) at 15-40 volts reduce apnea frequency, percentage of apnea time, frequency of oxyhemoglobin desaturation and apnea duration ***(Miki et al., 1989).***

Indeed, this therapy was damped by a recent report indicating that transcutaneous electrical stimulation failed to augment upper airway size as evidenced on fast computerized tomographic scanning of awake patients with obstructive sleep apnea and that it did not improve sleep disordered breathing in these patients during sleep ***(Edmonds et al., 1992).***

5. Nasal Continuous Positive Airway Pressure (CPAP):

Sulivan et al., (1981) initially described the relief of obstructive sleep apnea by the administration of air under 4.2-10 cm H_2O pressure via nasal prongs sealed within the nares. Shortly thereafter, ***Rapoport et al., (1983)***, described the use of a self-sealing nasal mask through which air under pressure was delivered. The application of CPAP via nasal mask has been shown by ***Sanders, (1987)***, to provide significant amelioration of mixed as well as occlusive apnea with improvement in nocturnal oxygenation. Also, it was found to be effective in diminishing both the central and obstructive portions of mixed apnea and tended to be effective in reducing apneas (fig 5).

Nasal CPAP is generally, but not universally, well tolerated by sleep apnea patients. Several investigators using patient reported data have documented good compliance with home nasal CPAP by sleep apnea

patients. However, the potential inaccuracy of assessing patient reported data is obvious. Recently, studies have been conducted which employed timer on the CPAP units to measure the machine run time which ostensibly reflects the duration of patient's use ***(Fairbank, 1994)***.

A new modality becomes available for delivering positive airway pressure in the treatment of sleep apnea, namely, Bi-level positive airway pressure **(biPAP)** ***(Sanders, 1987)***.

This device allows independent adjustment of the positive pressure delivered during inspiration (inspiratory positive airway pressure), and expiration (expiratory positive airway pressure) permitting specific titration of the pressure required to keep the upper airway patent during inspiration and expiration. By definition, delivered pressures are the same during both phases of the breathing cycle during CPAP administration, the pressure during expiration must equal that during inspiration ***(Sanders, 1987)***.

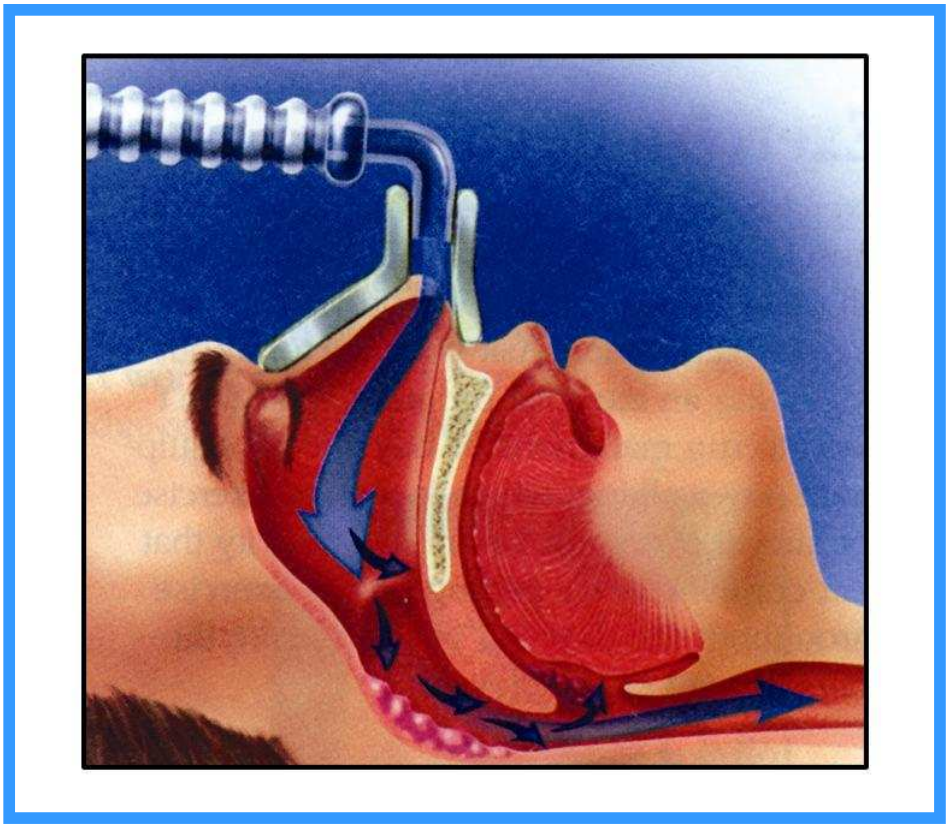


Fig (5): diagram showing the mechanism of action of CPAP.

B.SURGICALTREATMENT

Indication for surgical intervention:

The need for surgical treatment of obstructive sleep apnea and for snoring is determined basically by three factors:

- The severity of medical complications.
- Socioeconomic compromise due to disabling daytime sleepiness.
- Socially disturbing loud snoring.

Opinions differ on whether the severity of the sleep apnea syndrome should be based on the medical complications or the degree of hypersomnolence.

The degree of hypersomnolence should be considered

- Marked: The patient cannot stay awake even when motivated.
- Moderate: The patient frequently falls asleep whenever sedentary, job performance is suffering and driving is usually a significant concern.
- Mild: The patient can stay awake to work satisfactorily and complain little about problems in driving short distances (up to 30 min).

The disease is considered life threatening if anyone of the following conditions is met:

- Significant bradycardia (below 40/ min) with apnea.
- Asystole.
- Ventricular arrhythmias.
- CO2 faIling frequently below 50%.
- Cor-pulmonale.
- Extreme hypersmnolence as measured by multiple sleep latency test (MSLT).

Appropriate management decisions are most effectively achieved by multidisciplinary team that includes a sleep specialist, polysomnographer, pulmonary physician and otolaryngologist ***(Fujita, 1981)***.

There are many surgical procedures for treatment of snoring and OSA according to the level of obstruction:

- **Nasal surgery:**

  It includes septoplasty for deviated septum, turbinectomy for hypertrophied inferior turbinates and endoscopic sinus surgery (ESS) for nasal polypi ***(Kao et al., 2003)***.

- **Palatal surgery:**

- Uvulopalatopharyngoplasty (UPPP; U3P):

UPPP was first described by ***Fujita et al,*** in ***1981***. It consists of removal of the palatine tonsil, uvula, a portion of the soft palate, and the lateral pharyngeal wall is the most common surgical procedure for the treatment of OSA ***(Shepard et al., 1990)***.

- The uvulopalatal flap:

This procedure achieves anatomic results that are similar to those achieved with the UPPP but with less postoperative discomfort and fewer complaints of foreign body sensation (fig 6,7,8,9) ***(Powell et al., 1996)***.

Poor candidates for the uvulopalatal flap are those with a long, thick uvula and significant palatal redundancy ***(Powell et al., 1996)***.

- **Laser-assisted uvulopalatoplasty (LAUP):**

LAUP was initially designed for the management of snoring ***(kamami, 1990)***. Gradually, it has been extended to treating various degrees of obstructive sleep apnea (OSA). LAUP is an office procedure performed under local anesthesia and requires several sessions until satisfactory results are achieved.

During surgery, which has been extensively described by ***Krespi et al, (1994)*** vertical trenches are created on either side of the uvula into the soft palate, coupled with shortening and trimming of the uvula.

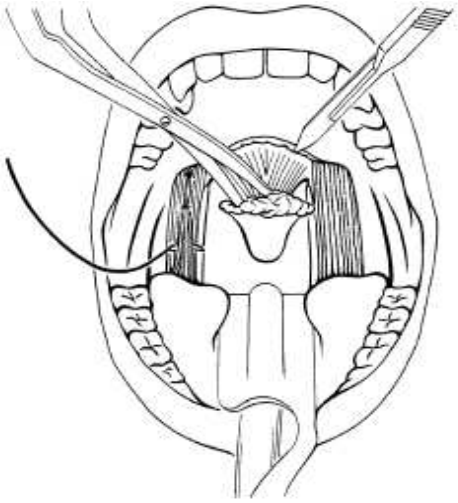


Fig (6) Left tonsillectomy was performed; a triangular incision (1 cm) from the upper pole of tonsillar fossa toward third molar was incised with removal of mucosa and submucosal adipose tissue. Right tonsillar fossa was accomplished with enlargement in upper and lateral dimension.

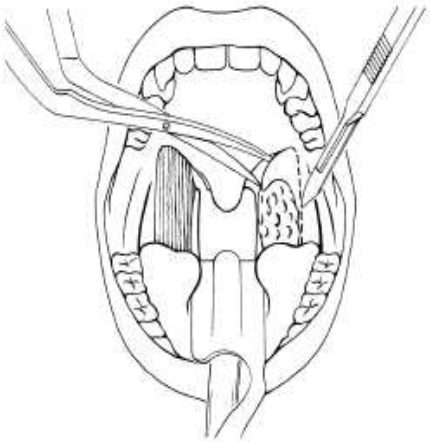


Fig (7) The mucosal web between uvula and posterior pillar was divided (0.5-1 cm) in an oblique direction along the uvula. An incision was made 5 to 10mm below the posterior end of hard palate to upper margin of the tonsillar fossa in both sides. Left-angle scissors were used to dissect the plane between submucosal adipose tissue and muscle layer from the incision line toward uvular tip (stripping technique). The uvular tip was excised within the specimen.

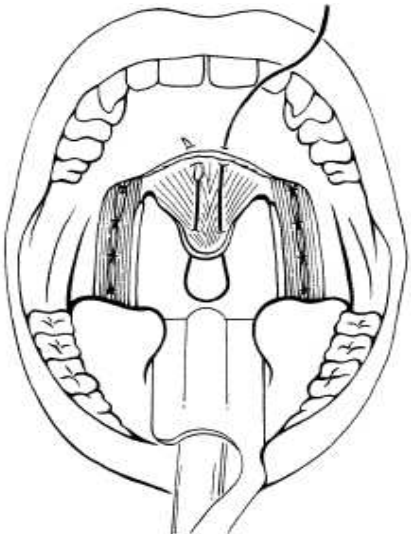


Fig (8)The denuded flap was imbricated and sutured to he proximal part of soft palate with 2-0 Vicryl. Bilateral tonsillar fossae were closed to decrease the dead space

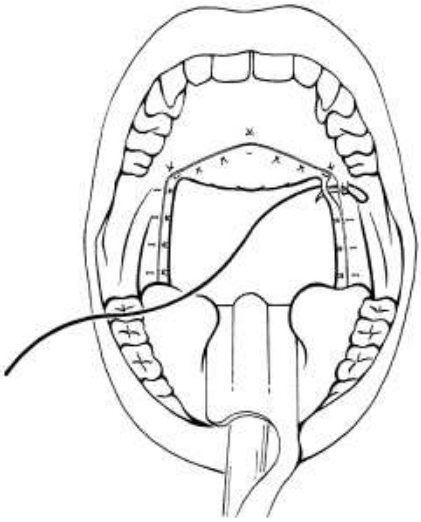


Fig (9) The approximation of posterior and anterior pillars was done in mattress suture with maximal lateralization.

-Temperature-controlled radiofrequency tissue ablation (TCRFTA):

High frequency alternating current, usually from 300 kHz to 3 MHz , flows from insulated tip of an electrode (uni or bipolar) into tissues. Ionic agitation is produced in the tissues about the electrode tip as the ions attempt to follow the changes in the direction of the alternating current ***(Powell et al., 1997)***.

Coblation techniques for treatment of snoring & OSA include:

A- Coblation ablation technique.

B- Coblation channeling technique.

Coblation (cold ablation or controlled ablation) is a bipolar radiofrequency used in soft tissue surgery ***(Timms and Temple 2002)***.

-Transpalatal advancement pharyngoplasty:

Pharyngoplasty using the technique of palatal advancement differs from earlier descriptions in a shorter palatal flap, more aggressive palate mobilization and palate advancement using an osteotomy leaving soft tissue attached to bone (fig10) ***(Tucker et al., 2005).***

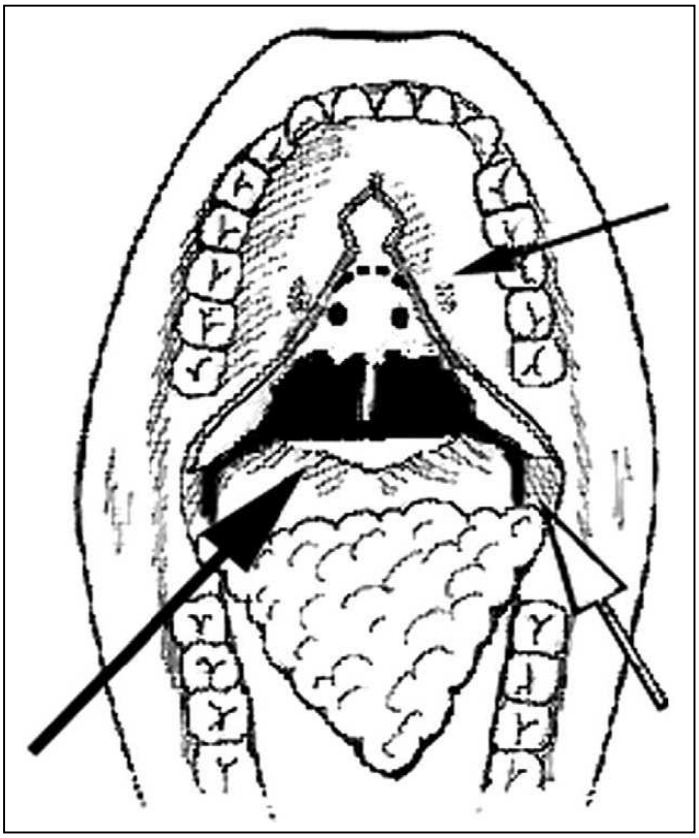


Fig (10)Intraoral view of pharyngoplasty with palatal advancement.
The palatal flap is constructed with its tip 1 cm. anterior to the level of bone removal (dotted line). The flap is medial to the greater palatine foramen (stippled area, small arrow). A vertical midline incision is extended anteriorly and this allows wider exposure. The location of the palatal drill holes are shown leaving a 5-mm margin of bone. The nasal septum is visible in the midline. The posterior osteotomized segment remains attached to the tensor tendon (large solid arrow). Laterally, the tensor tendon is incised medial to the hamulus (open arrow) ***(Tucker et al., 2005).***

- **Oropharyngeal procedures:**

Adenotonsillectomy:

The size of the tonsils and adenoids can be removed or reduced in a number of ways. The surgeon's preference, cost, and postoperative pain and complications dictate which methods are used in each institution, which are subject to change over time. The methods include use of convential adenotonsillectomy, bipolar cautery, harmonic scalpel, coblation, temperature-controlled radiofrequency, or microdebrider-powered shavers.

- **Surgeries for tongue base obstruction:**

o **Surgical procedures for tongue stabilization or advancement as:**

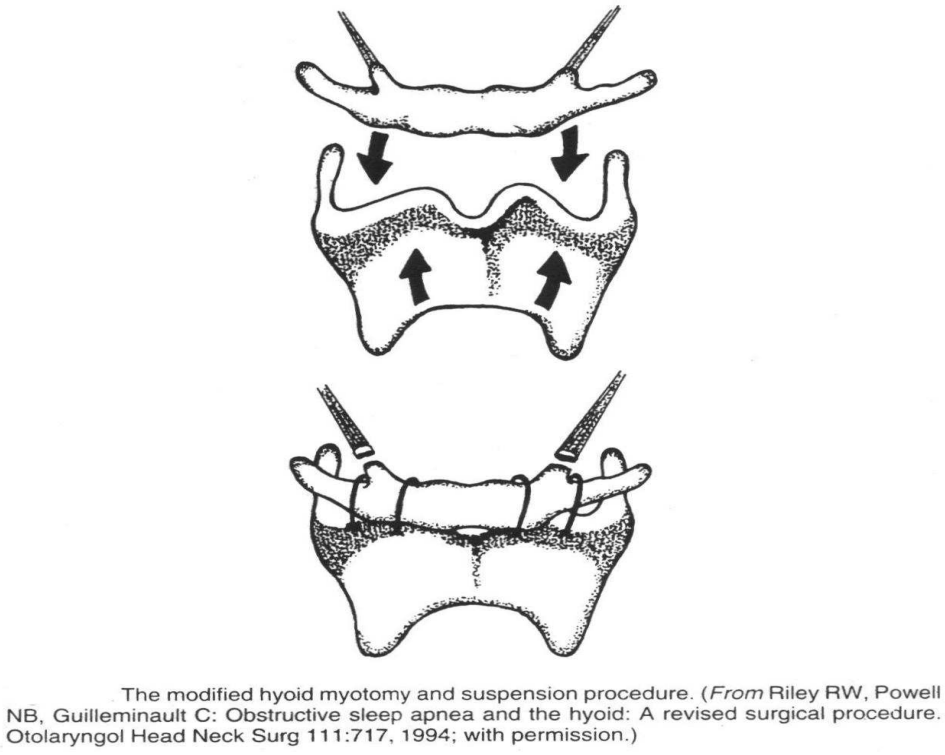
Tongue suspension:

After an intraoral incision is made in the frenulum, a titanium screw is placed at the lingual cortex of the geniotubercle of the mandible and a permanent suture is passed through the paramedian tongue musculature along the length of the tongue, through the tongue base, and back through the length of the tongue musculature. It is then anchored to the screw, pulling the tongue base anteriorly ***(Terris et al., 2002)***.

Hyoid myotomy and suspension:

A horizontal cervical incision over the hyoid bone is preferred, and the dissection is carried down to the suprahyoid musculature. The midline hyoid bone is isolated and then advanced over the thyroid ala. It

is secured with two medial and two lateral permanent sutures (fig 11) ***(Riley et al., 1994)***.



The modified hyoid myotomy and suspension procedure. (*From* Riley RW, Powell NB, Guilleminault C: Obstructive sleep apnea and the hyoid: A revised surgical procedure. Otolaryngol Head Neck Surg 111:717, 1994; with permission.)

Fig (11) the modified hyoid myotomy and suspension ***(Riley et al., 1994)***.

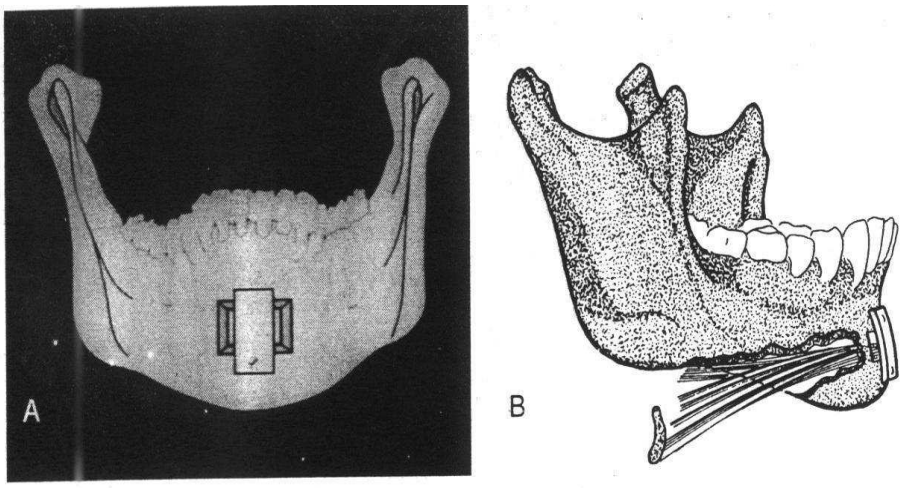
Genioglossus muscle advancement:

Window mandibular osteotomy is done to free the genial tubercle then forward fixation of the rectangle osteotomy after rotation then the tongue is pulled anteriorly by about 10-14mm (thickness of the respective mandible) (fig 12) ***(Riley et al., 1990).***

Maxillomandibular advancement (MMA):

Le Fort 1 osteotomy with advancement and a sagittal split ramus osteotomy with advancement is modified by maximizing the distances that the segments are moved and filling the defects with calvarial bone grafts.

The overall average advancement of the genial tubercle is 21 mm (fig 13). The response rate for this surgery is over 90% ***(Riley et al., 1990)*.**



The genioglossus advancement procedure. *A*, Anterior view of present technique. The rectangular geniotubercle osteotomy modification offers excellent tension on the genioglossus muscle with a minimum fracture risk and is technically reliable. The geniotubercle fragment presently is rotated only enough to allow bony overlap. A single inferiorly placed miniscrew is placed by a lag technique; occasionally, a superiorly placed microscrew is needed to adequately fix the fragment. *B*, Lateral view of present technique.

Fig (12) genioglossus muscle advancement ***(Troell et al., 1998)***.

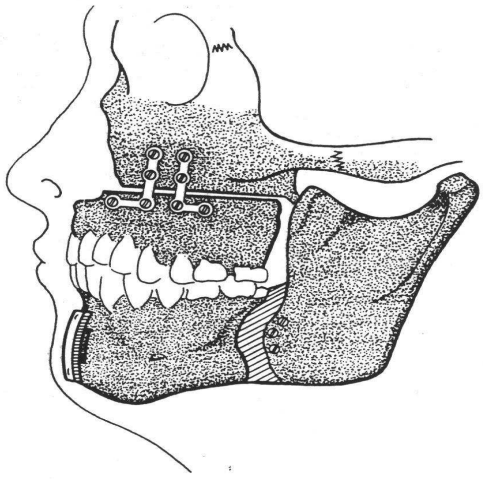


Fig (13) the maxillomandibular advancement ***(powell et al., 1994)***.

- **Surgical procedures for tongue bulk reduction as:**

Laser midline glossectomy (LMG):

This procedure, devised to tackle the problem of hypopharyngeal, collapse. It is an alternative to the maxillofacial procedures. After a covering tracheostomy, a CO_2 laser is used via an operating microscope and a rectangular area of the tongue base excised down to the valeculla. Further tongue base, hypertrophic lingual tonsils or redundant aryepiglottic folds are also excised using the laser ***(Fujita, 1991).***

Lingualplasty:

This is the same procedure as the LMG except that additional tongue tissue is removed posteriorly and laterally to the portion removed by LMG. Lingualplasty is usually combined with a tracheostomy for airway protection ***(Woodson et al., 1992).***

Radiofrequancy tongue base reduction:

This procedure involves an insulated probe delivering radiofrequency energy of 465 khz to reduce the volume of tongue tissue by producing coagulation necrosis and healing by scar and musculature contracture ***(Powell et al., 1997)***.

- **Tracheostomy:**

Tracheostomy was utilized as an effective treatment for obstructive sleep apnea, long before the disease was fully recognized as

an entity or even named. While never methods have displaced tracheostomy as the primary treatment for the disease, tracheostomy is not yet obsolete ***(Fairbanks, 1994).***

Tracheostomy can be performed as a permanent solution for sleep apnea or can be temporary, together with UPPP to protect the airway in the immediate post-operative period ***(Fairbanks, 1994).***

Indication for tracheostomy for OSA:

- Morbid / retrogenathia.
- Excessive hypopharyngeal/tissue obstructing by mirror examination of larynx.
- Oxygen desaturation below 50% on polysomnograph.
- Significant cardiac arrhythmias during apneas.

The immediate improvement in symptoms after tracheostomy is dramatic. Its effectiveness in controlling cardiac arrhythmias and improving symptoms of pulmonary hypertension and oxygenation is well documented ***(Thawely, 1993).***

Although tracheostomy is an effective therapeutic method for patients with significant obstructive sleep apnea, the performance, postoperative care, and long time follow up require an active understanding of the potential problems ***(Thawely, 1993).***

Failures in CPAP therapy

The standard therapy for obstructive sleep apnea is a nocturnal ventilation therapy with continuous positive-airway pressure (CPAP). In almost all cases, CPAP ventilation leads to a reduction of pathological respiratory events to a physiological level ***(Aloia et al., 2005)***.

CPAP provides a pneumatic stent for the upper airway, eliminating the airway collapse during inspiration. It is administered by a soft mask that covers the nose only. Sufficient pressure is introduced to eliminate apneas, hypopneas, and snoring ***(Aloia et al., 2005)***.

The criterion standard for determining the amount of pressure appropriate for each patient is the PSG. In some centers, this is performed as a split-night study, with data from the first half of the night used for diagnosis of SDB. Once this diagnosis is made, if the RDI is sufficient to suggest benefit from initiation of CPAP (usually an RDI of 20 or more), the second half of the night's study is used to determine the optimal amount of pressure. The disadvantage of the split-night approach is that the second half of a full night study often reveals more severe sleep apnea, so a diagnostic study limited to the first half of the night can underestimate disease severity ***(Doherty et al., 2003).***

The amount of pressure delivered is reported as cm H_2O. An average starting point for CPAP would be 8-10 cm H_2O. Patients report that pressures at these levels feel odd but are tolerable even when beginning treatment and become more tolerable as the patients become

accustomed to treatment. Higher levels (>15 cm H_2O) often are not well tolerated ***(Doherty et al., 2003).***

When a second overnight study is logistically difficult, some clinicians empirically start a patient on CPAP with a pressure of 8-10 cm H_2O. A new generation of CPAP machines can sense the amount of pressure needed to overcome upper airway resistance. Patients are sometimes started using these machines without a prior titration study. Alternatively, an autotitrating machine can be used for several nights, the record of amount of pressure required to suppress apneas and hypopneas can be downloaded and studied, and a suitable nightly pressure can be determined in this fashion. Also, the amount of pressure required to suppress snoring can be used as an audible guide to appropriate pressures ***(Stammnitz et al., 2004).***

A patient who routinely takes sedatives or ingests alcohol during the evening and does not intend to change this should probably be tested after continuing their usual nightly routine. CPAP titration without sedatives or alcohol is likely to lead to undertreatment of the SDB at home, when such patterns are resumed ***(Aloia et al., 2005).***

Most patients feel better during the daytime on the first day after beginning CPAP. During the first week of treatment, most experience rebound sleep with prolonged episodes of REM sleep. Sleep patterns become more normal after the first week. For these reasons, several weeks of CPAP use may be helpful for normalization of sleep patterns in patients with severe sleep apnea who plan to undergo surgery. Sleep

patterns should be normalized prior to the planned surgery ***(Kawahara et al., 2005).***

Regular use of CPAP improves both the patients' and their bed partners' quality of life. The treatment lessens depressive symptoms, and improves daytime functioning, blood pressure and insulin sensitivity. Asthmatic OSA patients have fewer nighttime symptoms ***(Kawahara et al., 2005).***

Most physicians agree that patients with a respiratory disturbance index (RDI) greater than 20 require treatment. CPAP can also be useful for patients with a lower RDI, especially if they experience daytime sleepiness or other symptoms. If the severity of the daytime symptoms and the Epworth Sleepiness Scale score are much greater than would be expected with a particular RDI, a trial of CPAP can help determine whether elimination of the SDB leads to improvement of the daytime symptoms, or if other factors contribute to the daytime symptoms ***(Douglas, 1998).***

Patients who are unlikely to benefit from CPAP include those with such severe nasal obstruction that CPAP cannot be used, patients with such extreme claustrophobia that they cannot tolerate a nasal mask, and patients in whom CPAP does not reliably eliminate apneas, hypopneas, and snoring ***(Douglas, 1998).***

Drawbacks for CPAP treatment include the following: Some patients have much difficulty adjusting to using CPAP. Overall compliance rates can be low (46% in one study defining use as at least 4

h/d, 5 d/wk) ***(Jean et al., 2005).*** The points below may assist the physician in improving treatment compliance.

Claustrophobia: Many patients report claustrophobia. They find that the sensation of covering the nose with a mask makes them so uncomfortable that they cannot tolerate wearing the n-CPAP. Sometimes this can be helped with a smaller or more transparent mask design. Use of nasal pillows (inserted into the nostrils) instead of a formal nasal mask may allow such patients to tolerate the CPAP ***(Douglas, 1998).***

Trouble tolerating initial pressure: Especially when higher pressures (>12-13 cm H_2O) are required for elimination of apneas and hypopneas, patients may find this level of pressure uncomfortable. Many CPAP machines have a built-in ramp or gradual increase in pressure. Using this feature, the mask can be placed and pressure begun at a very low and easily tolerated level. Over 30 minutes, the pressure gradually builds to the full amount necessary. Often, the patient can fall asleep during this ramp-up time. The full pressure is not used until the patient is actually asleep ***(Bachour et al., 2004).***

Nasal obstruction:

Evaluation by an otolaryngologist reveals whether this is predominantly a fixed skeletal obstruction or a soft tissue obstruction potentially modifiable without surgery. Marked septal deviation or turbinate hypertrophy usually requires surgery for resolution. Alar collapse may be adequately treated by internal or external dilators (eg, Breathe Right strip, Nozovent). Surgery is sometimes required for repair of marked alar collapse ***(McArdle et al., 1999).***

Mucosal edema may be due to allergic rhinosinusitis or to vasomotor or irritative rhinitis. Allergy testing and treatment and pharmacotherapy trials (eg, topical steroids or antihistamines, oral antihistamines or decongestants) may be beneficial ***(McArdle et al., 1999).***

One way to determine whether sufficient potentially reversible mucosal edema exists to pursue that avenue of treatment is the topical decongestant test. The patient uses a nasal topical decongestant (eg, oxymetazoline) at bedtime for several days, with the patient and bed partner observing for any improvements in snoring or apneas. A marked improvement suggests potentially reversible mucosal edema as a main contributor to the nasal obstruction. Failure to improve suggests a fixed skeletal obstruction that requires surgical correction ***(McArdle et al., 1999).***

Sometimes the dryness of the air or its temperature may be irritating to the patient. Use of in-line humidification and warming of the inspired air may alleviate patient discomfort ***(Neill et al., 2003).***

Facial or nasal pain: Sometimes this pain can be related to a poorly fitting mask. With the many different types of masks available now, different styles and sizes can be tried to select the optimal fit for each individual anatomy. Because the mask is pulled tight against the face, an edentulous anterior maxilla may not provide the resistance necessary for a good fit. Leaving dentures in at night can help with this. If the facial or nasal pain persists despite mask refitting, evaluation for nasal

obstruction or chronic sinusitis may be helpful. The CPAP Pro delivery method anchors the tubing to a platform based on an upper retainer, obviating the need for a forehead strap ***(McArdle et al., 1999).***

Dry eye or other eye discomfort: If the mask does not seal well, shift of pressurized air from the upper end of the mask toward the eye may occur, causing dry eye or even exposure keratitis. Mask refitting usually eliminates this problem ***(Bachour et al., 2004).***

Mouth falling open, awakening with dry mouth: Sometimes a chin strap is required to prevent the mouth from opening at night. A commercially available disposable adhesive bandage may used to pull the chin up toward the lower cheeks ***(Neill et al., 2003).***

Epistaxis: Epistaxis may be related to the high-flow dry air and may be helped by humidification and warming of the inspired air ***(Neill et al., 2003).***

Nasal drying: Forced dry air can be irritating to the nose, encouraging mucosal inflammation and crusting. Use of humidified air for CPAP usually eliminates this problem ***(Neill et al., 2003).***

Although CPAP provides good improvement in symptoms and physiologic parameters, compliance with treatment is not good, with regular use sometimes estimated as low as 30%. Rigorous patient education and early reinforcing follow-up may improve long-term use.

Variations of air pressure delivery can sometimes make CPAP use more comfortable for patients. Autotitrating positive airway pressure (APAP) continually adjusts the pressure to barely overcome the

collapsing forces. Bilevel positive airway pressure (BiPAP) provides higher pressure during inspiration (when the pneumatic splint is needed to prevent obstructive airway collapse) and lower pressure during expiration. C-Flex is another autoadjusting delivery method that increases pressure toward the end of expirations, as collapse would usually begin, and decreases pressure during early expiration. Patients who require higher pressures to overcome obstructive apneas may tolerate these devices better than the one-level n-CPAP, which delivers the higher pressure throughout the entire respiratory cycle ***(Victor, 2004).***

Measures of surgical success

Patients should undergo a postoperative polysomnogram three to four months after upper airway reconstruction, to determine the response to surgical therapy.

Many authors concluded that postoperative snoring level, Epworth Sleepiness Scale (ESS) and Quality of life questionnaire are necessary to assess the subjective symptoms elimination and they are obtained from both the patient and bed partner ***(Friedman et al., 2006)***.

However, it is well known that subjective improvement of patients′ symptoms often does not correlate well with objective measurement, many studies have reported subjective improvement rates that far higher than objective improvement rates ***(Senior et al., 2000)***.

Friedman et al; concluded that they have a significant improvement in both snoring levels and ESS scores with an overall subjective improvement in 73.9% of patients studied for the treatment with the Pillar Implant system after failed U3P operation and also quality of life scores demonstrate significant improvement in all domains.

However, their data indicated that objective cure was only achieved in 21.7% of patients undergoing the procedure.

Other authors concluded that postoperative fiberoptic nasendoscopy is necessary to assess the anatomical improvement in the airway especially in the retropalatal obstruction (fig 14) ***(Tucker et al., 2005)***.

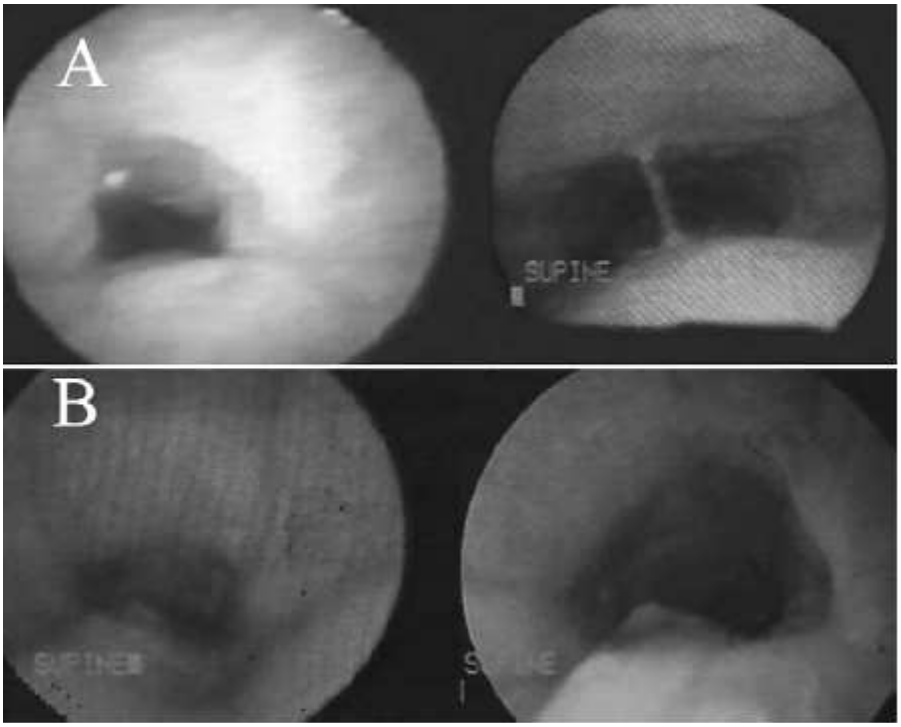


Fig (14) Preoperative and long-term postoperative photographs of the retropalatal airway in 2 patients (A and B) are shown. Preoperative (left) and postoperative (right) are shown with the posterior pharyngeal wall to the top and the soft palate to the bottom of the photographs. (A) There is significant enlargement in the lateral pharyngeal walls, which are now concave in appearance. (B) In this patient, the velopharynx is constricted with a small opening as well as proximal muscular posturing of the palate in the preoperative photo. Postoperatively, the enlargement of the velopharynx and muscular posturing is absent ***(Tucker et al., 2005)***.

The success of surgical intervention has been defined in a number of ways; a widely cited metaanalysis by ***Sher, Schectman and Piccirillo, (1996)*** described a threshold of success that allows for a 50% reduction in the apnea hypopnea index (AHI) (with a postoperative AHI of less than

20) or a 50% reduction in the apnea index (AI) (with a postoperative AI of less than 10).

A stricter definition has also been used that acknowledges only the AHI which must yield a 50% improvement, with a postoperative value less than 20.

In addition to objective polysomnographic improvement, patients should experience relief from their snoring and improved sleep hygiene.

The elimination of neurocognitive dysfunction, especially excessive daytime sleepiness (EDS), corresponds to reports of better-quality sleep, improved ability to concentrate, elimination of the necessity of naps and improved work performance ***(Troell et al., 1998)***.

If neurocognitive dysfunction exists, even with mild obstructive objective sleep parameters, additional treatment should be considered ***(Troell et al., 1998)***.

Many authors previously used an RDI (Respiratory Disturbance index) of 20 as such a level of success, this number is based on limited mortality data reported by ***He et al, (1988)***.

The success rate differs greatly according to the measures of surgical success, for example, the success rates for U3P when defined as a 50% decrease in the AI; the response rate is 83.3% for patients with type I narrowing or collapse versus 19.0% for patients with type II and type III narrowing or collapse.

But when defined as a 50% decrease in the RDI, the response rate is 67.1% for patients with type I narrowing or collapse versus 23.7% for patients with type II and type III narrowing or collapse (***Sher et al., 1996)***.

Johnson and Chinn, (1994) achieved a mean reduction of 44.1 points on the RDI (from a preoperative value of 58.7 to a mean postoperative value of 10.5) in patients undergoing U3P and genioglossus advancement without HM, when defining success as a RDI of less than 10, seven out of nine patients (80%) where successfully treated.

Troell et al, (1998) reported that seven of eleven patients (63.6%) who underwent a U3P with genioglossus advancement and HM were cured with cure defined as a postoperative RDI of less than 10.

Vicente et al, (2006) reported 78% success rate for patients with severe OSA who underwent U3P with tongue base suspension technique performed with the Repose system with the surgical success defined when the ESS dropped below 11 and the AHI decreased below the threshold of 20 events per hours of sleep and at least 50% from the preoperative value.

Waite et al, (1989) published results from 23 patients who underwent MMA with a surgical success rate (defined as RDI of less than 10) of 65%, their success rate increased to 85.9% when their threshold of response was lowered to a postoperative RDI of less than 20.

Obesity as a cause of failure in OSA surgery

Obesity is a major risk factor for obstructive sleep apnea syndrome (OSAS) and is present in approximately 60% of patients with OSAS ***(Young et al., 1993)***.

Although the exact pathophysiologic relationship between obesity and OSAS has not been defined, the effect on the airway size and the collapsibility from excessive fat deposits in the pharynx as well as the reduction in lung volume has been implicated ***(Strobel et al., 1996)***.

Obesity and sleep apnea are a chicken and egg problem. It is not always clear which condition is responsible for the other. For example, obesity is often a risk factor and possibly a cause of sleep apnea, but it is also likely that sleep apnea increases the risk for weight gain: Some studies indicate that sleep apnea disrupts rapid eye movement (REM) sleep, which, in turn, increases the risk for obesity. Research indicates that animals deprived of REM sleep tend to eat more. People with apnea may also become too tired to exercise and so put on weight ***(Wittels et al., 1990)***.

The evidence for an association between obesity and OSA is well documented, with obesity shown to be a risk factor for OSA in both adults and children ***(Gami et al., 2003)***. In obese adults, the reported prevalence of OSA is about 40%; in extremely obese adults (BMI ≥40 kg/m^2), the prevalence increases to 98% ***(Gami et al., 2003)***.

Few OSA studies have been conducted in the obese pediatric population. ***Marcus et al. (1996)*** reported that 36% of obese children and

adolescents had abnormal polysomnograms (PSGs), and they also showed a positive correlation between the degree of obesity and the severity of OSA. ***Silvestri et al. (1993)*** reported a 59% prevalence of OSAS in obese children and adolescents, similarly showing an increased trend as obesity became more severe. In a pediatric population referred for sleep problems, ***Mallory et al. (1989)*** found that 37% of children and adolescents suffering from severe obesity had abnormal PSGs, and of those patients, 24% were diagnosed with OSA.

Obesity has been found to be directly related to the severity of OSAS ***(Vgontzas et al., 1994)***. In addition, obesity has been implicated as a poor prognostic factor in sleep apnea surgery. Major weight gain was associated with surgical failures, although there was no negative effect from aging and minor weight gain ***(Li et al., 2000)***. Failures after UPPP was related to preoperative BMI and postoperative weight gain ***(Larsson et al., 1994).***

Consequently, massive weight loss via dietary or surgical methods seems to be a logical treatment option in patients with morbid obesity (BMI >40 kg/m2) and severe OSAS who are intolerant of nasal continuous positive airway pressure (CPAP) and refused tracheotomy ***(Yale, 1989)***.

Currently, bariatric surgical procedures including the vertical banded gastroplasty and the gastric bypass are used to produce malabsorption and restrict gastric volume for weight control ***(Yale, 1989)***. However both operations have associated mortality between 0.3% and 1.6%. In addition, significant perioperative complications present ***(Yale, 1989)***. Furthermore, poor long-term outcomes with significant weight

gain above the preoperative weight level have been reported ***(Wolfel et al., 1994)***.

Dietary weight loss may seem to be a simple method in the treatment of OSAS. However, the results are generally not as dramatic as the results from surgical methods. Furthermore, regardless of the method of dietary alteration, there is a high degree of relapses after weight loss ***(Wilson et al., 1994)***.

More importantly, although OSAS has been shown to improve in a majority of patients after significant weight loss by either dietary or surgical methods, many patients continue to have significant apnea ***(Charuzi et al., 1992)***. And a recent report has demonstrated the recurrence of sleep apnea despite maintenance of weight loss several years after bariatric surgery ***(Pillar et al., 1994).***

An important treatment consideration in the treatment of morbidly obese patients is the potential of the coexisting obesity-hypoventilation syndrome (OHS), characterized by obesity and chronic hypercapnea, OHS and OSA often coexist in morbidly obese patients. OHS has been shown to influence the outcomes of tracheotomy in the treatment of OSAS ***(Alexander et al., 1962)***.

Kim et al., (1998) performed tracheotomy in OSA patients that were divided into three different groups based on the presence or absence of concomitant cardiopulmonary or upper aerodigestive tract dysfunction. Interestingly, tracheotomy only achieved a less than 50% cure rate (RDI <20). This subgroup of patients was found to be significantly heavier and

the persistence of sleep-disordered breathing episodes was found to be associated with hypoventilation and hypoxemia due OHS.

Rapoport et al. (1986) treated eight patients with OHS and OSAS with either tracheotomy or nasal CPAP. Although apnea was improved in all of the patients, two patients continued to have an apnea index greater than 10 events per hour, and four of the eight patients remained hypercapnic, i.e., having persistent OHS.

OHS was found to be an important factor in the treatment failure of airway reconstructive surgery for the treatment of severe OSA in obese patients. Therefore, a thorough preoperative evaluation including a pulmonary function test to identify the coexistence of OHS must be considered before embarking on any surgical reconstruction in this patient population ***(Kim et al., 1998)***.

Patients with significant OHS and OSAS should undergo a tracheotomy or use nasal CPAP for airway protection. Polysomnography with the tracheotomy tube unplugged or with the nasal CPAP applied should be performed to identify the potential of persistent sleep-disordered breathing episodes due to OHS. This may avoid embarking on any surgical reconstruction that would be doomed to fail due to OHS despite a significant improvement of the OSAS ***(Kim et al., 1998)***.

However, ***Kasey et al., (2000)*** underscore the importance of weight reduction as a treatment option for OSAS in obese individuals as they performed airway reconstructive surgery i.e. phase I reconstruction (uvulopalatoplasty, genioglossus advancement, and /or hyoid suspension) and phase Π reconstruction (maxillomandibular advancement) on 21

patients . Seventeen (81%) of these patients were successfully treated (RDI <20). Although every patient in this study lost a minimum 15 kg immediately after the completion of their airway reconstruction, the majority of the patients regained all of their weight, with some patients weighing more than the preoperative level at the 6 months' postoperative polysomnography.

So, ***Kasey et al., (2000)*** stated that airway reconstruction for the management of severe OSA in the morbidly obese patients is a viable option, and the weight reduction was not likely a major factor since the majority of the patients remained morbidly obese.

Hormonal disturbances

Recognition of the influence exerted by other diseases and syndromes may contribute to the challenge of effective OSA treatment. "Disproportionate anatomy" among the base of the tongue, narrow mandible and hypoplastic mandible affects upper airway dynamics ***(Rojewski et al., 1984)***. This disproportion can be seen in syndromatic patients and in hormonal disturbances.

A number of hormones interact with sleep and breathing. SDB affects hormones *via* a number of mechanisms. Conversely, hormones and endocrine states induce, aggravate or alleviate SDB ***(Saaresranta et al., 2002).***

SDB and sleep disturbances may interact with hormones in several ways. Episodes of apnoea or hypopnoea cause sleep fragmentation and disturb sleep cycles and stages. Arousals may induce stress response resulting in increased levels of stress hormones ***(Späth-Schwalbe et al., 1991)***.

Hypoxia may also have direct effects on central neurotransmitters' which result in alterations in the hypothalamo-pituitary axis and in secretion of the peripheral endocrine glands ***(Semple et al., 1981)***. Hypercapnia alone or combined with hypoxia may increase levels of renin, adrenocorticotrophic hormone, corticosteroids, aldosterone and vasopressin ***(Raff et al., 1983)***.

Finally, disorganisation of sleep, sleep loss and naps disturb sleep-controlled endocrine rhythms resulting in endocrine and metabolic abnormalities ***(Raff et al., 1983)***.

The direct and indirect effects of hormones and endocrine disorders on sleep and breathing are mediated *via* several pathways.

Male gender and postmenopausal state, as risk factors, link sex hormones to the pathophysiology of SDB ***(Polo-Kantola et al., 2003)***,

Sleep apnoea is common in acromegaly ***(Weiss et al., 2000)***, hypothyroidism ***(Pelttari et al., 1994)*** or Cushing's syndrome ***(Shipley et al., 1992).***

The most recent studies suggest that SDB may not only complete the clinical picture but play a central role in the pathophysiology of obesity ***(Vgontzas et al., 2000)***. The prevalence estimates of sleep apnoea among various endocrine states and disorders are shown in table 4.

Table 4 Prevalence of sleep-disordered breathing in some endocrine disorders and states

	Prevalence of sleep apnoea %	***Sample size***
Diabetes type 1	31	16
	42	12
Diabetes type 2	1.9 (*versus* 0.3 in nondiabetics)	579
	36 (*versus* 14.5 in nondiabetics)	25
Diabetes with autonomic	37 (*versus* 0 in those without AN)	8 (and 8 without AN)
neuropathy	0 (*versus* 6 in those without AN)	8 (and 8 without AN)
	26 (*versus* 0 in those without AN)	23 (and 25 without AN)
Hypothyroidism	82	11 10 20 26
	100	
	25	
	7.7 (*versus* 1.9 in controls)	
Acromegaly	40 with active, 0 % with inactive disease	10 with active, 11 with inactive disease
	45	11
	81	53
	39	54
Cushing disease/syndrome	45	22
Polycystic ovary syndrome	17	53
	44	18
Postmenopause	2.7[#] (*versus* 0.6 in premenopausal females)	314

AN: autonomic neuropathy

Hypothyroidism

A link between SDB and hypothyroidism is suggested by the high prevalence of sleep apnoea among hypothyroid patients, particularly in rare myxoedematous patients (77–100%) ***(Pelttari et al., 1994)***. Therefore, symptoms of SDB should be routinely asked in all hypothyroid patients and sleep studies should be considered when symptoms present.

The increased prevalence of SDB appears to be related to obesity and male sex rather than hypothyroidism *per se* ***(Pelttari et al., 1994)***. However, decreased ventilatory responses, extravasation of albumin and mucopolysaccharides in the tissues of the upper airway ***(Orr et al., 1981)*** and hypothyroid myopathy ***(Grunstein et al., 1988)*** have been suggested as possible contributing factors for SDB in hypothyroidism.

The decreased ventilatory responses increase with thyroxin replacement ***(Zwillich et al., 1975)***, and episodes of apnoea may disappear. However ***Lin et al, (1992)*** concluded that after initiation of thyroxin replacement therapy, patients may snore more, suffer from nocturnal chest pain and ventricular arrhythmia.

A temporary worsening of SDB after onset of thyroxin therapy could be due to an increase in basal metabolic rate, increased oxygen consumption and increased respiratory drive, which could promote periodic breathing and upper airway instability. Prolonged episodes of apnoea and lower oxyhaemoglobin saturation could be risky in patients with pre-existing coronary heart disease. To avoid the possible complications, hypothyroid patients with SDB should be, at least, initially treated with nasal CPAP. When the steady state has been achieved and the

patient no longer has symptoms of hypothyroidism, the need for nasal CPAP therapy has to be re-evaluated ***(Lin et al., 1992)***.

In patients with OSAS, the prevalence of hypothyroidism is 1–3% ***(Winkelmann et al., 1996)***, which does not essentially differ from that in the general population. Screening for hypothyroidism in patients with sleep apnea does not seem necessary unless the patient is symptomatic or belongs to a risk group (*i.e.* females aged ≥60 yrs) ***(Winkelmann et al., 1996)***.

Catecholamines

In blood and urine, high levels of catecholamines and their metabolites reflect increased sympathetic activity. Muscle sympathetic nerve activity is greater in obese than in normal-weight subjects, and greater in sleep apnoeics than in age and BMI-matched controls ***(Narkiewicz et al., 1999)***.

Hypoxia and hypercapnia induce sympathetic nervous system overactivity. The sympathetic responses to hypoxia and hypercapnia are further potentiated during apnoea, when the inhibitory influence of the thoracic afferent nerves is eliminated ***(Narkiewicz et al., 1999)***.

Nocturnal noradrenaline levels correlate with OSAS severity and oxygen saturation ***(Eisenberg et al., 1990)***. Also sleep fragmentation leading to chronic partial sleep loss is likely to contribute to the increased sympathoadrenal activity and increased circulating catecholamine levels encountered in OSAS. This assumption is supported by observations in healthy male volunteers. One night of partial sleep deprivation resulted in increases in circulating noradrenaline and adrenaline levels ***(Irwin et al.,***

1999). Most studies report a positive relationship between episodes of obstructive apnoea and noradrenaline levels, whereas a minority of studies has found a relationship between adrenaline and episodes of obstructive apnoea ***(Coy et al., 1996)***.

Acromegaly

The association of snoring and daytime sleepiness and acromegaly was first reported more than a century ago (Roxburgh et al., 1896). Macroglossia and pharyngeal swelling are the most probable reasons for the high incidence of SDB in acromegaly ***(Mezon et al., 1980)***. Accordingly, sleep apnoea alleviates when tissue hypertrophy decreases with somatostatin analogue treatment ***(Ip et al., 2001)***.

Growth hormone and insulin-like growth factor (IGF)-I may also have a direct role in the pathogenesis of sleep apnoea but the observations are controversial ***(Grunstein et al., 1991)***. Some investigators report an association between the presence of sleep apnoea and high growth hormone and IGF-I levels ***(Rosenow et al., 1996; Perks et al., 1980)***, whereas the others fail to show any association between obstructive sleep apnoea and biochemical activity of acromegaly ***(Pekkarinen et al., 1987; Grunstein et al., 1991).***

The high IGF-I levels in acromegaly may drive breathing and result in increased hypercapnic ventilatory response measured during wakefulness , and increased frequency of central apnoea or periodic breathing with symmetric waxing and waning respiratory efforts during sleep ***(Ip et al., 2001)***.

Treatment of acromegaly with adenomectomy or octreotide may cure acromegaly related OSAS. The operative team should be aware of the risks of performing the trans-sphenoidal adenoma resection in acromegalic patients with sleep apnoea in whom upper airway oedema could potentially further aggravate gas exchange postoperatively ***(Guilleminault et al., 1980)***. Octreotide treatment may promptly alleviate OSAS, and thus its preoperative administration is recommended ***(Rosenow et al., 1998)***. Preoperative nasal CPAP therapy could also reduce the perioperative risks ***(Rosenow et al., 1998)***. Perioperative tracheostomy is the safest and sometimes the only alternation to secure breathing after surgery.

After adenomectomy, sleep apnoea persists in every fifth patient, in particular, in those whose growth hormone levels remain high. In addition to endocrine factors, the high prevalence of residual SDB after adenomectomy could be related to soft tissue hypertrophy, which remains unaltered. However, uvulopalatopharyngoplasty is not feasible in the treatment of acromegaly related OSAS. Nasal CPAP with new pressure titration is often needed after surgery ***(Rosenow et al., 1998)***.

Growth hormone deficiency

Not only excessive growth hormone production, but also growth hormone deficiency could link with sleep apnoea. Syndromes with hereditary growth hormone deficiency are often associated with obesity, craniofacial and pharyngeal abnormalities predisposing to SDB.

Sleep apnoea patients have low growth hormone levels without any specific causes of growth hormone deficiency ***(Grunstein et al., 1989)***.

Growth hormone secretion occurs mostly during sleep, and 70% of nocturnal growth hormone pulses are associated with slow-wave sleep ***(Van Cauter et al., 1992)***. In OSAS, growth hormone secretion is decreased not only due to obesity, but also because of sleep fragmentation resulting in decreased amount of slow-wave sleep ***(Issa et al., 1986)***.

In addition, repetitive hypoxaemia may affect growth hormone secretion. In animals, hypoxia inhibits growth hormone release or biosynthesis. Growth hormone deficiency in adults is associated with impaired psychological well-being, insulin resistance, endothelial dysfunction, increased visceral fat, increased cardiovascular mortality and accelerated ageing ***(Conceicao et al., 2001)***. Similar features are typical in OSAS, which raises the question of a possible link between OSAS-related growth hormone deficiency and the comorbidity seen in OSAS. Indeed, patients with severe OSAS have similar levels of IGF-I to adult patients with growth hormone deficiency ***(Grunstein et al., 1989)***. Low IGF-I may contribute to an increased risk for cardiovascular diseases among sleep apnoeics.

Two reports suggest that growth hormone replacement therapy may also affect sleep and breathing ***(Gerard et al., 1997; Nolte et al., 2002)***. Among 145 children on growth hormone replacement, four developed sleep apnoea; in three cases this was associated with tonsillar and adenoidal hypertrophy ***(Gerard et al., 1997)***. Sleep apnoea improved in one patient after cessation of growth hormone therapy, and in all patients following tonsillectomy and adenoidectomy. In five, male, middle-aged patients with postoperative pituitary insufficiency, cessation of growth hormone replacement for 6 months resulted in a decrease of obstructive

apnoeic events but in an increase of central apnoeic events. Following cessation of growth hormone replacement, slow-wave sleep decreased markedly ***(Nolte et al., 2002).***

At least in theory, an unfortunate coexistence of growth hormone deficiency and SDB would result in a potentially vicious interaction between two altered physiological functions, resulting in severe anatomical abnormalities. A primary growth hormone deficiency could predispose to SDB through short stature, craniofacial growth retardation and low respiratory drive. SDB would further aggravate growth hormone deficiency through sleep disturbance. A primary SDB could aggravate itself by affecting craniofacial and upper airway soft tissue growth through induction of secondary growth hormone deficiency ***(Nieminen et al., 2002)***.

In patients with growth hormone deficiency and with predisposing anatomical abnormalities for SDB, a systematic screening for SDB is encouraged. Nasal CPAP treatment and maxillomandibular surgery are feasible therapeutical approaches in these patients. Treatment of SDB may result in normalisation of growth hormone secretion and normal growth in children ***(Nieminen et al., 2002)***. Conversely, symptoms of SDB should also be monitored during growth hormone replacement therapy because of increased risk of SDB.

Diabetes mellitus

The prevalence of SDB in type-1 diabetes remains to be confirmed. Some authors have reported a prevalence rate of sleep apnoea as high as

42% ***(Mondiniet al., 1985)*** whereas others have not observed a difference from the general population ***(Catterall et al., 1984)***.

Small sample sizes and different diagnostic criteria for sleep apnoea may explain some of the discrepancy. Diabetic children (n=25) have more episodes of apnoea during sleep and the duration of apnoeic events is longer than in healthy controls ***(Villa et al., 2000)***. Further, the degree of severity of sleep apnoea correlates with the glucose control and the duration of diabetes.

Among ~13,000 Japanese hospital inpatients, the prevalence of sleep apnoea was 0.3% ***(Katsumata et al., 1991)***. In a subgroup of ~600 male type-2 diabetics, the prevalence of sleep apnoea was higher than in nondiabetics (1.9 *versus* 0.3%, respectively). In a Swedish 10-yr follow up study, snoring was a risk factor for diabetes, independent of other risk factors ***(O'Donnell et al., 1999)***.

Among hypertensive diabetics, the prevalence of sleep apnoea, defined as AHI ≥20, was 36% compared with 14.5% in nondiabetics. Autonomic diabetic neuropathy may be associated with sleep apnoea. Among 23 diabetics with autonomic neuropathy (one had type-1 diabetes), six had sleep apnoea, whereas none of the diabetics without autonomic neuropathy were affected ***(Ficker et al., 1998)***.

Sites of obstruction and their identification

The phenomenon of snoring originates from different sites in the head and neck region because of their size, position, or relationship with surrounding structures. Problems may arise from the nasal cavity (deviated septum, enlarged nasal turbinates, presence of nasal polyp, and nasal adhesions), the retropalatal area (elongated or floppy soft palate and uvula), hypertrophied or obstructive tonsils, the tongue base (retrolingual area), upper and lower jaw and chin deformities (retrognathism), posterior and lateral pharyngeal wall constriction with muscle hypertrophy, or hyoid and epiglottis positions (Fig 15) ***(Hudgel et al., 1988)***.

Any obstructions in these areas will cause greater negative inspiratory pressure and partial or complete collapse of the airway (hypopnea or obstruction apnea). Additional anatomical factors contribute to the development of obstructive sleep apnea, including decreased dilating forces of the pharyngeal dilators and negative inspiratory pressure generated by the diaphragm ***(Kuna et al., 1991)***.

The etiology of anatomic obstruction in OSA is believed to be an imbalance between the forces acting to maintain airway patency (the force of the pharyngeal muscles) and the negative inspiratory forces generated by the diaphragm. This mismatch may be due to a clear anatomic abnormality (i.e. micrognathia, macroglossia, or hypertrophy of the tonsils and adenoids) but more often is subtle ***(Hoffstein, 1996).***

It has been demonstrated that patients who have OSA have pharyngeal collapse that is more significant than in control subjects when the same amount of negative suction pressure is applied ***(Wetmore et al.,***

1986). Additionally, patients with sleep apnea have been shown to have failure of reflex activation of pharyngeal dilators in response to airway occlusion ***(Hoffstein, 1996)***.

In determining the site of obstruction, two problems come to light: Where is the pharyngeal area of greatest collapse and how can this area be determined accurately? The exact location of pharyngeal collapse is often difficult to ascertain with certainty. More confounding is the fact that the area is often not a single area at all but involves a combination of retro-palatal and retroglossal collapse.

Similarly, the collapse may be oriented in an anterior-posterior dimension or in a lateral-medial dimension (or a combination). Emphasizing this point, in one series of 200 patients with OSA, only three were found to have a single anatomic abnormality by routine otolaryngologic examination ***(Rojewski et al., 1984).***

Theoretically, accurate identification of the exact sites of collapse should aid the surgeon in procedure selection, thereby improving success rates. In most patients, however, it is difficult to identify the anatomic locations amenable to surgical correction. This challenge in identifying the areas of anatomic abnormality has led to the development of diagnostic techniques, including fiberoptic airway endoscopy with the Miiller maneuver and cephalometrics, etc ***(Yao et al., 1998).***

The role of cephalometrics in predicting the site of anatomic obstruction has led to contradictory data, which is not surprising given the fact that these static measurements of bony and soft tissue anatomy most likely do not reflect the dynamic changes in pressure that result in airway collapsibility ***(Yao et al., 1998)***.

The role of CT in the assessment of the site of anatomic narrowing has been similarly unsuccessful. There was clearly no difference in static cross-sectional area of the pharyngeal lumen in patients who responded to UPPP and those who did not ***(Launois et al., 1993)***.

Attempts at predicting the site of collapse using intrapharyngeal pressure recordings also have been unsuccessful. Various studies have failed to correlate palatal collapse by pressure manometry with a successful outcome from UPPP ***(Metes et al., 1991)***.

It has been hypothesized by at least one author that only the most proximal site of obstruction can be identified by these methods ***(Launois et al., 1993)***.

Methods that would identify distal sites of collapse will aid in predicting which patients actually will benefit from an isolated surgical intervention that addresses only the proximal palatal obstruction, or if additional surgical modalities may be warranted ***(Launois et al., 1993)***.

Using a related technique, somnofluoroscopy, several authors have attempted to radiographically localize the site of obstruction during sleep ***(Katsantonis et al., 1986)***. In one study, patients who had closure identified at the level of the soft palate were more likely than the population as a whole to improve after UPPP (67% versus 42%) ***(Katsantonis et al., 1986)***.

Müller's maneuver seems to offer the best and easiest analysis of dynamic airway collapsibility. This maneuver assesses the extent of anterior-posterior collapsibility and lateral collapsibility along various points of the upper aerodigestive tract (ie, retropalatal or retroglossal). With this information, the examiner should be able to begin the difficult

task of assessing which areas may be amenable to surgical correction in a multistage process. There are several studies that suggest that patients who demonstrate nasopharyngeal collapse on Muller's maneuvers are more likely to improve after UPPP ***(Sher et al., 1985)***.

Additional data have demonstrated that the maneuver may more accurately identify poor responders to surgical intervention with UPPP, although other studies state that the utility of the maneuver as a predictive technique is low ***(Aboussauan et al., 1995)***.

Owing to the difficulty in predicting a single site of obstruction with relative accuracy, most surgeons currently advocate a multiphase approach in the surgical treatment of OSA, with UPPP playing an integral role. Increased success rates are seen when multiple procedures are used that address various sites of obstruction ***(Li et al., 2000)***. Palatal obstruction is addressed with UPPP, and base-of-tongue obstruction frequently is addressed with genioglossus advancement and hyoid myotomy suspension or base-of-tongue reduction. This procedure then is followed by maxillary-mandibular advancement for treatment failures. The success of these approaches adds support for the argument that the limited success of UPPP when performed in isolation is due to its inability to adequately address the multiple sites of obstruction in patients with OSA ***(Li et al., 2000).***

Additional patient-selection criteria

Clearly, the difficulty in precisely determining the anatomic site of obstruction in patients with OSA leads to challenges in predicting which patients will benefit from palatal surgery in isolation. There is numerous other patient factors that have been implicated in contributing to OSA,

however, that are measured easily and theoretically could increase the likelihood of selecting patients who would benefit from UPPP ***(Senior et al., 2000).***

Unfortunately, for all of the data collected during a polysomnograph, none has been correlated consistently with a successful outcome for an isolated UPPP ***(Johnson et al., 1994)***.

Increasing body mass index or weight of the individual has been shown to decrease the likelihood of success of a UPPP ***(Larsson et al., 1994)***. However, It also has been shown that with more severe OSA (by both AHI and apnea index), UPPP tends to be less successful ***(Larsson et al., 1994)***. In light of these data, it was hypothesized that less severe OSA may be more amenable to surgical correction with UPPP.

In a study by ***Senior et al. (2000)***, only 40% of patients with mild OSA (AHI >5 and <20) who were treated with UPPP with or without septoplasty responded (response being defined as a decline of 50% in AHI). Interestingly, in the same series, the patients who did not respond had an elevation of their AHI from 16.6 ± 5 to 26.7 ± 18.4 ***(Senior et al., 2000)***. Based on these data, they concluded that the anatomy of mild OSA is also complex and is not always corrected with isolated UPPP.

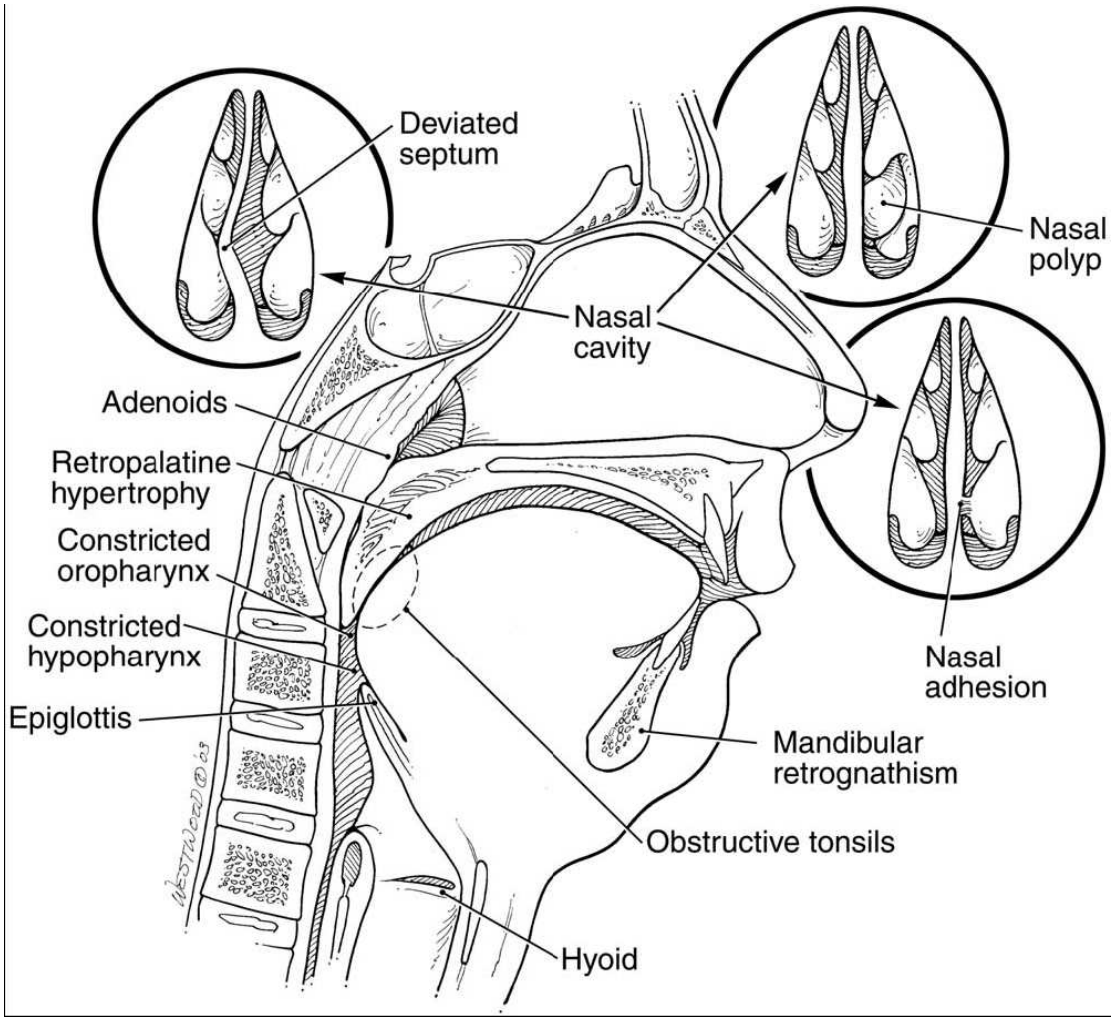


Fig (15) snoring and obstructive sleep apnea occurs because of at least 8 different components. They include nasal, tonsillar, adenoids, base of the tongue (oropharynx), hypopharynx, soft palate, and uvula. Additionally, the position of hyoid, mandible, and maxillae play an important role. ***(Mansoor Madani, 2004)***

Nasal obstruction

Nasal obstruction has frequently been mentioned as a possible risk factor in obstructive sleep apnoea syndrome (OSAS). Because increasing nasal resistance results in an increase in negative oropharyngeal pressure during inspiration, it predisposes to upper airway collapse ***(Olsen et al., 1981)***.

Correction of nasal obstruction has been reported to be an effective treatment of OSAS in patients with nasal obstruction but not those with craniomandibular abnormalities ***(Searieas et al., 1993)***. These data suggest that nasal obstruction may be a risk factor for OSAS and may deserve attention during the investigation of snorers referred for suspected OSAS.

However, previous studies using objective nasal resistance measurement in snorers decrease the importance of nasal obstruction alone to be a risk factor for OSAS ***(Miljeteig et al., 1992; Atkins et al., 1994)***.

Similarly, ***Young et al. (1997)*** failed to demonstrate any possible correlation between nasal resistance and sleep-disordered breathing in a sample of the general population.

On the other hand, there is clinical evidence supporting a role for acute or chronic nasal obstruction in OSAS. ***Lavie et al. (1981) and McNicholas et al. (1982)*** and demonstrated that nasal obstruction due to allergic rhinitis was associated with both sleep fragmentation and OSAS. The OSAS appeared to be reversible during remissions of the allergic symptoms.

Zwillich et al. (1981) and ***Lavie et al. (1983)*** compared apnoea during sleep experienced by normal subjects with and without

experimentally-induced nasal obstruction and found that nasal obstruction induced episodes of apnoea and episodes of arousal from sleep. Chronic abnormalities such as septal deviation and nasal valve obstruction have also been reported to result in sleep disturbance and OSAS that diminished significantly after relief of the nasal obstruction ***(Dayal et al., 1985).***

Searieas et al. (1992) have demonstrated that surgical correction of nasal obstruction is an effective treatment of mild OSAS in patients with nasal obstruction without cephalometric abnormalities. These data suggest that the list of the potential risk factors for OSAS includes not only BMI, male sex, and cephalometric abnormalities, but also nasal obstruction.

Lofaso et al. (2000) illustrate that daytime nasal obstruction, whatever the cause, is a risk factor for obstructive sleep apnoea syndrome, and its influence is less than that of obesity or cephalometric landmarks. However, because nasal obstruction can be treated, further studies are warranted to assess the clinical relevance of the findings.

Fitzpatrick et al. (2003) demonstrated that mouth breathing condition resulting from nasal obstruction causes a profound increase in OSA severity as compared with nasal breathing during sleep. No subject had any evidence of OSA during the nasal breathing condition, the normal pathway for ventilation during sleep. During wakefulness, nasal obstruction is associated with increased breathing through the mouth, but there are no published measurements correlating nasal resistance with oral fraction during sleep. Nonetheless, there is other evidence that links nasal obstruction (whether or not it is associated with mouth breathing during

sleep) with an increased tendency to sleep apnoea (fig 16, 17) ***(Ohki et al., 1996).***

A large epidemiological study demonstrated an increased prevalence of OSA among individuals with subjective nasal congestion due to allergy ***(Young et al., 1997)***. Similarly, a group of snorers with OSA were shown to have a higher nasal resistance than snorers without OSA ***(Lofaso et al., 2000)***.

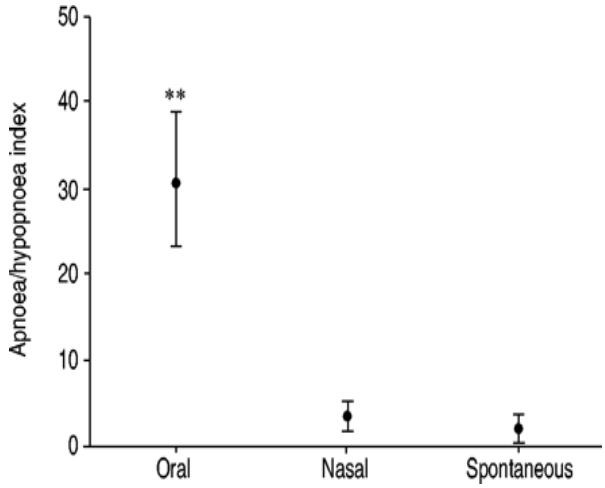


Fig (16): Effect of breathing route on sleep apnoea severity in the supine position. Data are presented as mean±sem. **: p<0.01 oral *versus* nasal breathing route ***(Fitzpatrick et al., 2003).***

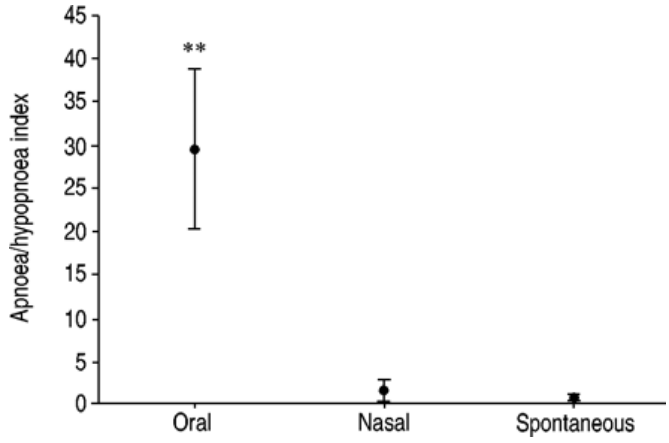


Fig (17): effect of breathing route on sleep apnoea severity in the lateral position. Data are presented as mean±sem. **: p<0.01 oral *versus* nasal breathing route ***(Fitzpatrick et al., 2003).***

Mouth opening has been documented during sleep in normal subjects and in patients with OSA ***(Miyamoto et al., 1998)***. Mouth opening, even in the absence of oral airflow, has been shown to increase the ability to upper airway collapse ***(Meurice et al., 1996)***.

The two most likely explanations for the latter finding are that jaw opening is associated with a posterior movement of the angle of the jaw and compromise of the oropharyngeal airway diameter, and that posterior and inferior movement of the mandible may shorten the upper airway dilator muscles located between the mandible and hyoid and compromise their contractile force by producing unfavorable length-tension relationships in these muscles ***(Meurice et al., 1996)***.

The marked increase in upper airway resistance during sleep while mouth breathing along with documentation during sleep in normal subjects of jaw opening and retroglossal airway patency might lead to speculatation that the most likely site of obstruction in the asleep normal subject breathing through the mouth may be the junction of the soft palate and tongue. However, the degree of jaw opening during sleep under conditions of forced breathing through the mouth in is unknown and may be quite significant, and it has long been known that jaw opening can profoundly affect the diameter of the retroglossal airway ***(Morikowa et al., 1961)***.

Hence, it is possible that the increased upper airway resistance observed during mouth breathing could be caused by compromise at both velopharyngeal and retroglossal levels ***(Fitzpatrick et al., 2003).***

Fitzpatrick et al. (2003) suggest a therapeutic role for improving the nasal airway in patients with nasal obstruction and OSA. However, published reports are inconsistent regarding the effects of attempts to improve nasal airway patency on sleep apnoea severity ***(Bahammam et al., 1999)***.

Meurice et al. (1996) have pointed out that patients with significant upper airway compromise at the retroglossal level (who may obstruct at that site independent of nasal obstruction) are less likely to respond to nasal surgery than those with a wider posterior airway space. And they anticipated that selected patients with OSA, those with severe nasal obstruction and normal retroglossal airway dimensions, might demonstrate substantial improvement in sleep apnoea severity after relief of nasal obstruction, but this has not been evaluated in a prospective study.

In another study done by ***Schönhofer et al. (2000)*** demonstrated that objective measurements of snoring and apneas during sleep were almost unaffected by the nasal dilator (Nozovent). In 4 of 21 patients, a decrease in respiratory disturbance index was found with Nozovent. However, in another 17 of 21 patients, it deteriorated. Obviously, patients with obstructive sleep apnea in common will not benefit from this kind of treatment

Schönhofer et al. (2000) reported that nasal obstruction may be the cause in the pathogenesis of the distal collapse of upper airway and this process of collapse proceeds in a consecutive manner even after correction of the nasal obstruction.

Kao et al., (2003) reported that the efficacy of nasal surgery alone in the treatment of moderate to severe OSA has been shown to be limited, with the success rate of less than 20% and they agree with the conclusions drawn by the authors that nasal obstruction is only a component of the etiology of OSA and that treating nasal airway alone will not cure the majority of patients with moderate or severe OSA.

However, the impact of a narrow nasal airway is significant by either causing an increase in the velocity of airflow through the nose and thus increasing the collapse of the airway through Bernoulli's forces or by causing mouth breathing, which is accompanied by posterior displacement of the base of tongue.

In the former case, the airway in the OSA can be thought of as a musical instrument. The palate is the reed; the hypopharynx is the instrument itself, and the nasal airway as the musician who is playing the instrument. The more congested the nose, the more rapid is the airflow through the rest of the instrument, the louder is the snoring, and the more significant is the obstruction. The improvement of airflow through the nose has a limited benefit. Further improvement of snoring or the OSA requires changes to the reed or the instrument itself (***Kao et al., 2003).***

The appreciation of the impact on nasal airflow has been blunted by inability to objectively access the degree of nasal obstruction in most of our patients. Patients themselves are poor estimators of their nasal breathing, and objective measures are not widely used by clinicians. In addition, it is unclear whether nasal breathing while awake correlates well with nasal breathing while asleep and whether sleep stages have any

impact on nasal airway as they do on the hypopharynx and oropharynx in the OSA patient (***Kao et al., 2003).***

Kao et al, (***2003***) concluded that correction of nasal obstruction must be done first before correction of distal airway collapse, but they decided to operate on patients only when they have significant complaints when it comes to nasal obstruction.

However, the approach to nasal breathing in the OSA patient demands a more objective assessment. Many patients will not believe that their nasal breathing is a problem. In approaching nasal obstruction in the OSA patient, the nasal breathing should be maximized as much as possible, independent of the patient's assessment of nasal airflow.

There are many dynamic measurements of nasal airflow and resistance including nasal peak flow, rhinomanometry, and acoustic rhinometry. Each procedure has its limitations, but most have diagnostic sensitivities between 80% and 95% for nasal obstruction ***(Dunagan et al., 2000).***

Nasal peak flow

Nasal peak flow test is inexpensive, easy to perform, and may have potential use in outpatient clinical trials or for home assessment of daily variations in nasal obstruction; however, it is highly effort dependent, and results may vary widely, especially between patients ***(Shelton et al., 1990).***

This technique involves measuring the peak inspiratory nasal airflow with a modified peak flow device. Although peak flows do not measure resistance, nasal peak flow measurements correlate well with measurements of resistance and have their greatest usefulness in the

detection of large changes in nasal patency in individual subjects ***(Hellgren et al., 1997)***.

Rhinomanometry

Since air flows from an area of high pressure to an area of low pressure, pressure gradients and flow measurements may be used to calculate nasal resistance.

Rhinomanometry, the measurement of nasal airway resistance, is probably the test most frequently performed because it measures both flow and resistance. It is classically divided into passive or active phases, and into anterior or posterior rhinomanometry ***(Holmstrom et al., 1990).***

Active rhinomanometry requires the subject to generate airflow through the nose by their own effort. Passive rhinomanometry utilizes external generation of a constant flow of air at a given pressure and requires no respiratory effort. Active rhinomanometry is a quick test to perform, and the International Committee on Standardization of Rhinomanometry recommends it for most studies ***(Clement. 1984)***. Anterior and posterior rhinomanometry primarily differ in the location of the transducer used to measure posterior pharyngeal pressure.

It may help to distinguish functional causes of upper airway obstruction from structural causes. For example, decongestants or exercise will improve airflow due to inflammation and vascular engorgement, whereas fixed abnormalities such as concha bullosa do not change after exercise or decongestants ***(Broms et al., 1982).***

Acoustic Rhinometry

Acoustic rhinometry, a technique used widely in Europe, evaluates nasal obstruction by analyzing reflected sound waves introduced through the nares. It is generally easy to perform, is noninvasive, and does not require patient cooperation like many of the other evaluation procedures ***(Pedersen et al., 1994).***

It produces an image that reflects variations in the cross-sectional dimensions of the nasal cavity and closely approximates nasal cavity volume and minimal cross-sectional area. The short measurement period (10 s) makes this procedure easy to use in all patients, even children ***(Naclerio et al., 1998)***.

Uvulopalatopharyngoplasty

Uvulopalatopharyngoplasty as first described by ***Fujita et al*** in **1981** is a common surgical rocedure applied for the treatment of obstructive sleep apnea syndrome (OSAS) (fig 18). However, using this technique, failure rates are reported from 30% to 90%. ***(Maisel et al., 1992; Cahali, 2003)***.

A systematic review of the literature in 1996 identified a 42% success rate in published studies using polysomnographic outcomes of an apnea hypopnea index (AHI) of less than 20 or an apnea index of less than 10 events per hour ***(Sher et al., 1996)***. Better success rates remain elusive despite attempts at improved selection and alternative techniques.

Assessing a surgical procedure's effectiveness for OSA is difficult. Outcomes are affected by many variables in addition to the procedure used; the most critical potentially being the population studied. Without being able to control this variability, attributing success to a procedure is difficult. To stratify a population undergoing surgery, pretreatment disease severity, body mass index, Mueller's maneuver, and cephalometry have been used**.** All have historically failed to accurately predict UPPP outcomes ***(Sher et al., 1985).***

Multiple methodological issues hamper attempts at assessing and improving surgical success rates. Differences in patient anatomy, surgical technique, and variable methods of patient selection make meaningful comparisons of equivalent patient groups difficult. Lack of comparative

or randomized studies, in turn, contributes to uncertainty about surgery's effectiveness ***(Shepard et al., 1990).***

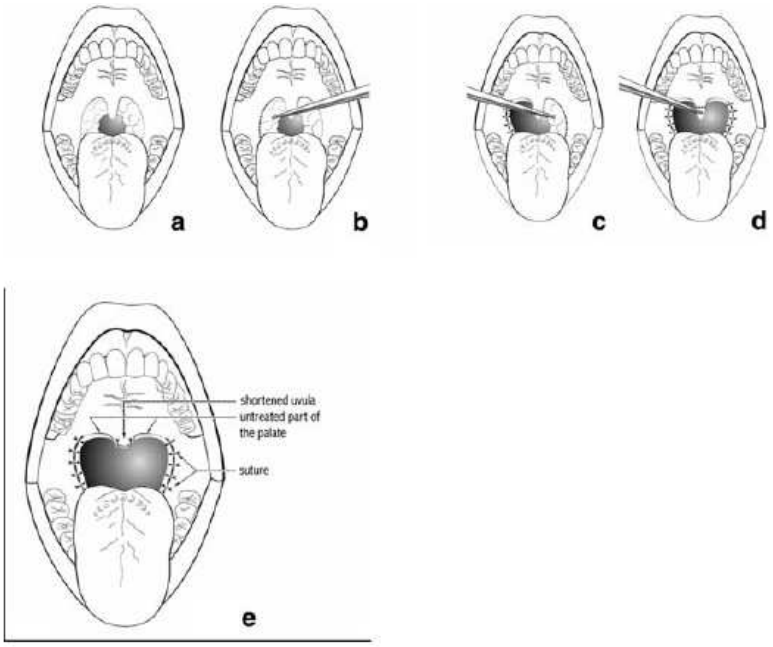


Fig (18) schematic representation of the nontraumatic surgical technique used in Bern {modified according to ***Fujita et al. (1981)***}

Recently, ***Friedman et al, (2002)*** reported on a simple reproducible clinical staging system that demonstrated power to stratify UPPP success rates into 3 defined groups. In their classification, stage I patients demonstrated a 70% success rate; stage II, a 50% success rate; and stage III, an 8% success rate. Because the system was based on common upper airway exam findings, it allows both prospective and retrospective staging of patients. Similarly staged patients may then be compared.

Staging system

Palate position had been previously studied and found to be a clinical indicator of OSAS. This palate classification is based on observations by ***Mallampati et al. (1985)*** who previously suggested palate position as an indicator of the ease or difficulty of endotracheal intubation by standard anesthesiologists' techniques.

Friedman et al, (2002) have incorporated two modifications into the Mallampati classification to create their own staging criteria: 1) the anesthesiologist's assessment is based on the patient sticking out their tongue and the observer then noting the relationship of soft palate to tongue. Their grading is based on the tongue in a neutral, natural position inside the mouth. 2) The original grading system had only 3 grades, and they believe that 4 grades are essential.

The Friedman Palate Position (FPP) grade was assessed as previously described. The procedure involves asking the patient to open their mouth widely without protruding their tongue. The procedure is repeated 5 times so that the observer can assign the most accurate level. At times there can be some variation with different examinations, but the most consistent position is assigned as the palate grade. Palate grade I allows the observer to visualize the entire uvula and tonsils or pillars. Palate grade II allows visualization of the uvula but not the tonsils. Palate grade III allows visualization of the soft palate but not the uvula. Palate grade IV allows visualization of the hard palate only (Fig 19).

Tonsil size was graded from 0 to 4. Tonsil size 0 implies previous tonsillectomy. Tonsil size 1 implies tonsils hidden within the pillars. Tonsil size 2 implies the tonsil extending to the pillars. Size 3 tonsils are

beyond the pillars but not to the midline. Tonsil size 4 implies tonsils that extend to the midline (Fig 20).

Weight and height were recorded at the initial visit, and the BMI (kg/m2) was calculated. The BMI was graded as grade 0 (_20 kg/m2), grade I (20 to 25 kg/m2), grade II (25 to 30 kg/m2), grade III (30 to 40 kg/m2), and grade IV (_40 kg/m2).

Stage I disease was defined as those patients with FPP I or II, tonsil size 3 or 4, and BMI of less than 40 kg/m2. Stage II disease is defined as FPP I or II and tonsil size 0, 1, or 2, or FPP III and IV with tonsil size 3 or 4 and BMI of less than 40 kg/m2. Stage III disease is defined as FPP III or IV and tonsil size 0, 1, or 2 and BMI less than 40 kg/m2. All patients with a BMI of 40 kg/m2 or greater and those with significant craniofacial or other anatomic deformities were classified as stage IV disease.

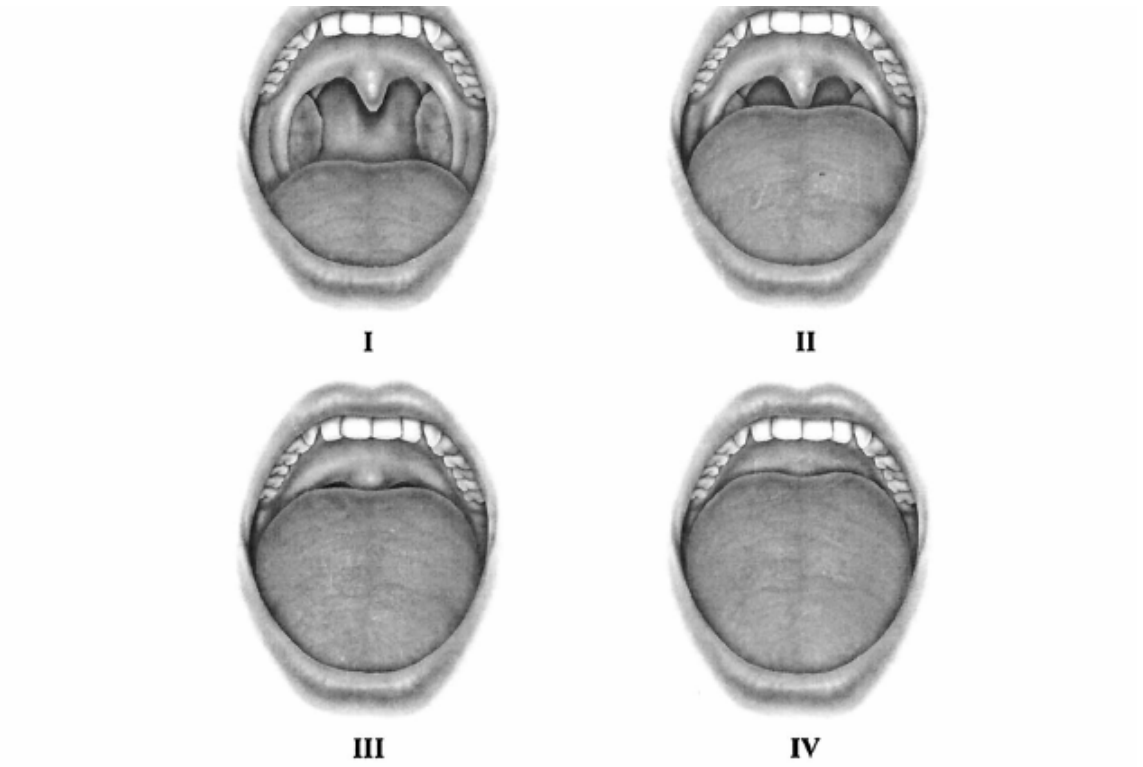


Fig (19): Friedman palatal position grading (***Friedman et al., 2002)***.

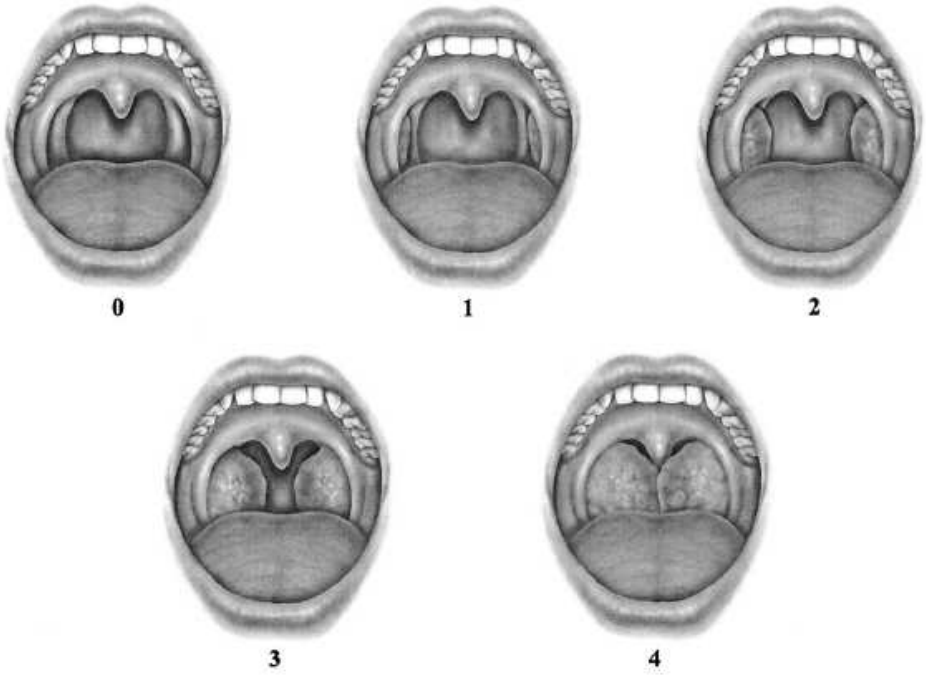


Fig (20): Friedman grading by tonsils size (***Friedman et al., 2002)***.

The cause of UPPP failure continues to be poorly understood. Undoubtedly, multiple causes exist. Although failure caused by obstruction at nonpalatal airway sites occurs, technical failure at the palate after UPPP failure is common. This tenet is supported by persistent obstruction proximal to the UPPP site, no change or worsening of closing pressures (a measure of airway collapsibility), and no improvement in retropalatal cross sectional area in UPPP nonresponders ***(Isono et al., 2003).***

UPPP decreased retropalatal airway closing pressure by 3.5 cm H2O; furthermore, a direct correlation between the severity of sleep-disordered breathing (as determined by nocturnal oximetry) and retropalatal airway closing pressure was revealed ***(Isono et al., 2003).***

UPPP failures revealed retropalatal airway closing pressure greater than atmospheric pressure. Reduced retropalatal airway collapsibility was maintained up to 1 year after UPPP, whereas few patients developed marked stenosis of the retropalatal airway with aggravation of sleep-disordered breathing after initial improvement of sleep-disordered breathing and retropalatal airway collapsibility ***(Isono et al., 2003).***

Manometry identifies the retropalatal site as a primary site of persistent airway obstruction. When successful, UPPP improves airway collapsibility and structure, and when unsuccessful, structural improvements are less ***(Isono et al., 2003).*** Overall, data suggest that persistent upper airway obstruction is associated with technical failure of the retropalatal segment proximal to the level of UPPP excision of the palate (***Isono et al., 2003).***

Mechanisms of Improvement of Passive Pharyngeal Collapsibility with Uvulopalatopharyngoplasty

As illustrated in Figure 21, the pharyngeal airway is surrounded by soft tissues, such as the soft palate and tongue, which are enclosed by bony structures, such as the mandible and spine. The balance between the amount of soft tissues and the size of the bony enclosure determines the airway size. In fact, higher P close values were demonstrated both in obese patients and in patients with small mandibular size ***(Watanabe et al., 2002)***.

In this model, soft tissues were removed to outside the bony enclosure, creating available room for the airway. However, the level of

the operation may have produced differing results. When the removed soft tissue amount was insufficient to create room for the airway, retropalatal closing pressure would not decrease to normal values, resulting in failure of UPPP. This speculation coincides with the previous findings of UPPP failures that the critical closing pressure presented abnormal values ***(Schwartz et al., 1992)*** and that the airway obstruction site remained at the retropalatal region ***(Woodson, 1997)***.

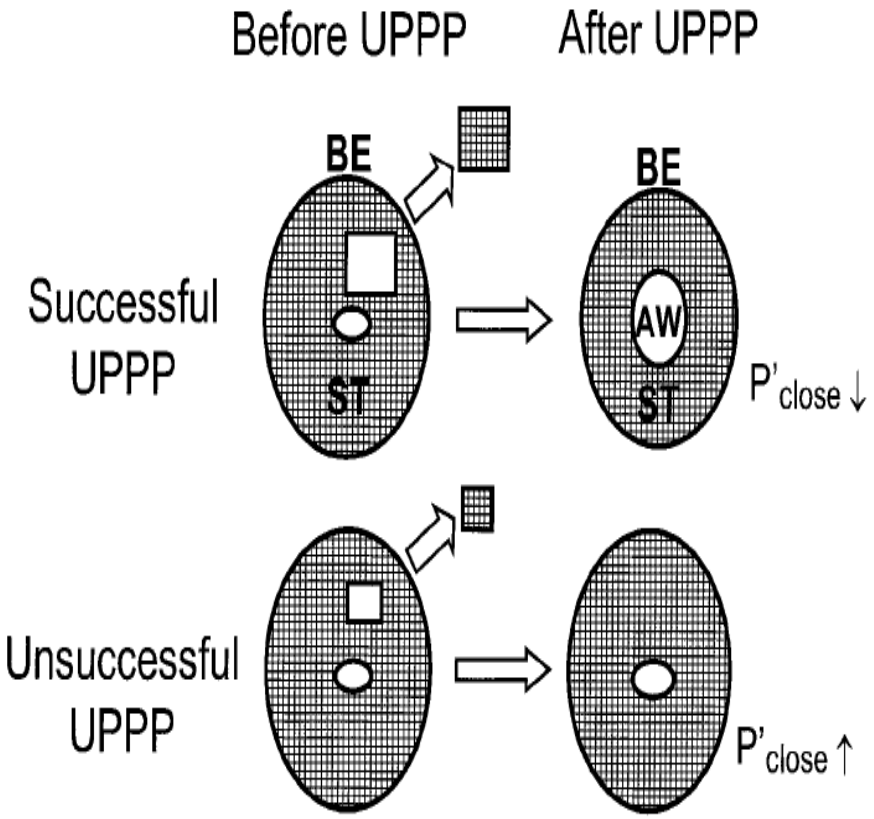


Fig (21): A mechanical model explaining effects of the surgery on collapsibility of the pharynx. The balance between the amount of soft tissue (ST) and the size of the bony enclosure (BE) determines the pharyngeal airway size (AW). Sufficient removal of the ST outside the bony enclosure results in reduction of closing pressure (P'close) and therefore in successful uvulopalatopharyngoplasty ***(Tucker et al., 2005).***

There are few studies evaluating long-term effects of UPPP on SDB in patients with OSA, and no study has evaluated long-term effects of UPPP on pharyngeal mechanics. ***Larsson et al. (1991)*** reported that 30% of the initial responders had relapsed to being nonresponders 21 months after UPPP in association with increases in BMI.

Similarly, ***Launois et al. (1993)*** reported that the success rate of UPPP decreased to 28% at 14 months after UPPP, although the rate was 86% at 4 months after UPPP. Interestingly, a significant increase in BMI was not evident during this period, suggesting that the aggravation of SDB after UPPP may have resulted from factors other than weight gain.

UPPP also may worsen upper airway structure. To better treat the airway, future options include more aggressive direct modifications of the distal soft palate, or alternatively, procedures that modify surrounding structures. Because more aggressive soft-tissue resection may be associated with increased complications, alternative techniques are required ***(Tucker et al., 2005)*.**

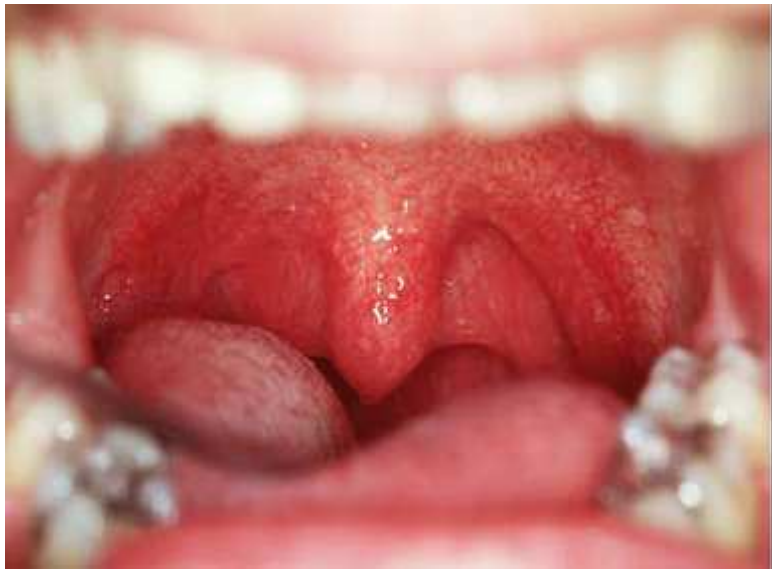


Fig (22): typical findings in the soft palate in patients with OSAS: uvular hyperplasia, depressed pillars of fauces with an extremely narrow transition into the rhinopharynx

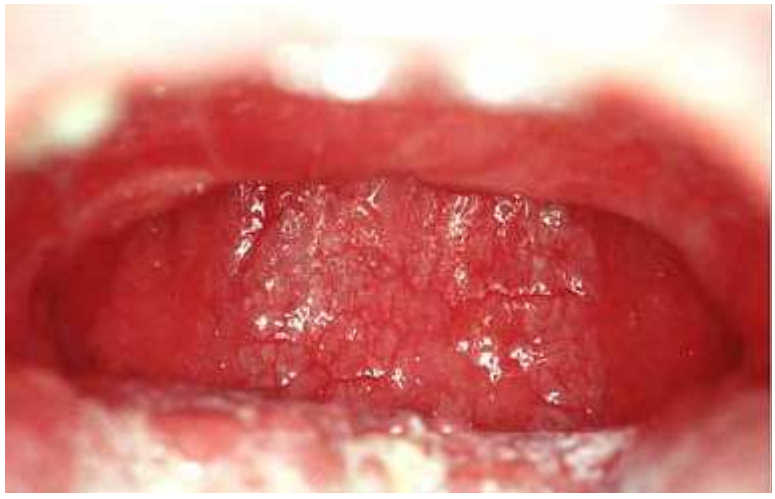


Fig (23): the postoperative site with wide pharyngeal opening.

Laser-Assisted Uvulopalatoplasty

Laser-Assisted Uvulopalatoplasty (LAUP) was initially designed for the management of snoring ***(kamami, 1990)***. Gradually, it has been extended to treating various degrees of obstructive sleep apnea (OSA). LAUP is an office procedure performed under local anesthesia and requires several sessions until satisfactory results are achieved.

During surgery, which has been extensively described by ***Krespi et al, (1994)*** vertical trenches are created on either side of the uvula into the soft palate, coupled with shortening and trimming of the uvula. Several studies ***(kamami, 1994; Walker et al., 1995, 1997, 1999; Utley et al., 1997)*** have examined the efficacy of the technique, recognized as standard LAUP, and reported comparable results to uvulopalatopharyngoplasty (UPPP).

However, other studies found that LAUP was ineffective as it is associated with a considerable number of subjective delayed failures as well as objective aggrevation of sleep parameters ***(Laurtano et al., 1997)***, had deleterious effects on the respiratory dynamics and may trigger the generation of OSA in formerly nonapneic patients who only snored ***(Berger et al., 2001)***, or lead to deterioration of the existing sleep apnea ***(Finkelstien et al., 2002)***.

Dickson and Mintz (1996) introduced a modified technique of LAUP, which they termed one-stage LAUP. This modified technique was designed to minimize the overall pain of the patients and the cost of the standard LAUP. During surgery, a curvilinear horizontal incision is made

under the palatal dimple, and ultimately the same amount of soft palate tissue is removed as in UPPP. The authors reported excellent short-term subjective results and a successful objective response. ***Seemann et al; (2001)*** also used one-stage LAUP and reported encouraging results. ***Ryan and Love, (2000)*** on the other hand, concluded that the response to this technique was varied and unpredictable, and only a few patients achieved a satisfactory response, a poor response in 34% and worsening in 30%.

Wareing and Mitchell; (1996) and ***Wareing et al; (1998)*** also pointed out that LAUP was associated with delayed failures in a sizeable number of patients, with reappearance of socially disruptive snoring in one fifth of the patients who earlier had benefited from the procedure.

Intraoral photographs demonstrated a substantial enlargement of the oropharyngeal isthmus immediately after surgery, causing temporary relief of signs and symptoms in a considerable number of patients ***(Finkelstien et al., 2002)***.

It is thought that the late decline in the improvement of snoring, aggravation of the sleep-related symptoms and the overall failure in the objective measures is attributable to progressive fibrosis inflicted on soft palate tissues by the thermal damage of the laser beam. LAUP, which is based on cutting and vaporizing palatal tissues, leaves a raw surface that subsequently undergoes scarring. These wounds take longer to heal than those created with a scalpel ***(Finkelstien et al., 2002)***.

The effectiveness of surgery, therefore, should be assessed months later, when the healing process has stabilized. Indeed, a study on the long-term histopathologic changes after LAUP disclosed that the various

components of the soft palate underwent extensive changes, with replacement of the loose connective tissue in the lamina propria by diffuse fibrosis that also extended to the central layer, on the expense of the seromucous glands and muscle fibers ***(Berger et al., 1999).*** Palatal fibrosis after LAUP was also encountered in 27% of the patients in the study by ***Carenfelt (1991).***

It was shown that the pharyngeal scar contracture occurred in centripetal direction and caused a curtain like medial traction of the posterior tonsillar pillars and a pulling of the lateral pharyngeal walls medially. Eventually, the pharyngeal cross-sectional area went through major anatomic changes that included narrowing of the lumen, increased rigidity, decreased compliance and loss of distensibility needed during inspiration (fig 24) ***(Berger et al., 1999).*** These deficiencies have deleterious effects on the respiratory dynamics and may aggravate existing OSA.

Finkelstien et al; (2002) concluded that the patients in their study experienced pain that lasted for an average of 9.8 days and was severe enough to keep patients away from work for an average of 7.2 days. These results are supported by the observations of ***Troell et al; (2002)*** who compared postoperative pain between various methods of palatal surgery in patients with snoring, upper airway resistance syndrome and mild apnea and reported a mean of 13.8 and 14.3 days with pain after LAUP and UPPP, respectively. To ease pain all patients also consumed narcotic analgesics.

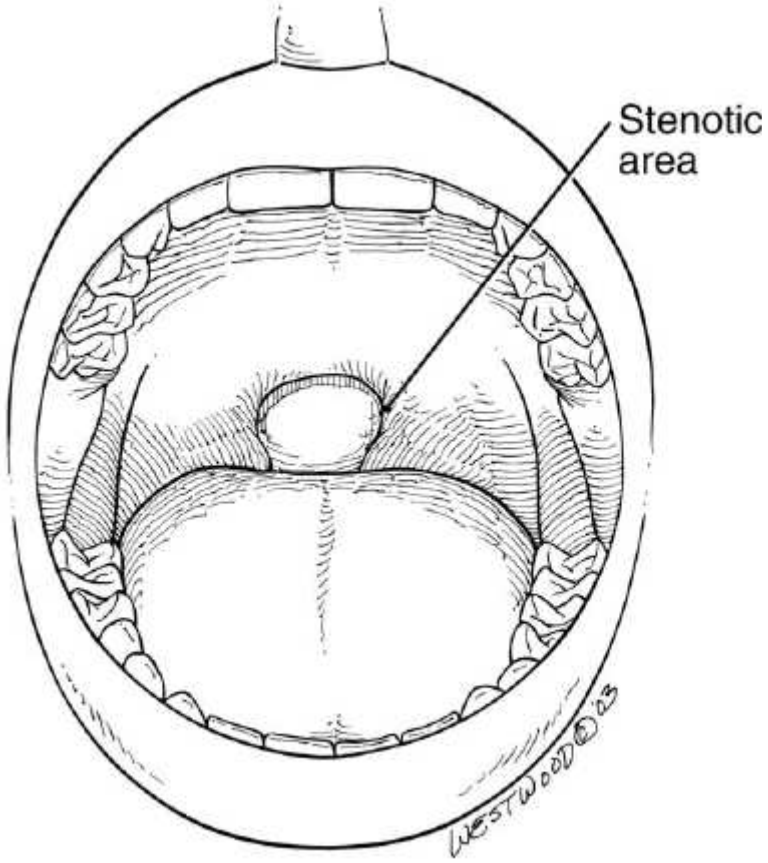


Fig (24) diagram showing palatal stenosis following LAUP (note the medial curtain like traction of the posterior pillars ***(Berger et al., 2003)***

Other reports also noted that excessive dryness of the mouth and discomfort in the throat were the most prominent complaints after LAUP ***(Ryan and Love, 2000; Berger et al., 2001)***.The reason for the sensation of dryness is the destruction during LAUP of multiple seromucous glands in the uvula and the posterior portion of the soft palate which provide continuous lubrication to the oropharynx and probably to the vocal cords. Indeed, any surgical intervention that diminishes the amount of glandular tissue may results in pharyngeal dryness and surface irritation of the vocal cords, especially LAUP, which is associated with a marked

decrease in the amount and function of the velar glands because of extensive palatal fibrosis and glandular destruction ***(Finkelstien et al., 1992).***

LAUP has gained much popularity in the last decade as a cure for OSA, a common yet potentially life-threatening syndrome. It is commonly accepted that the subjective, short-term outcome of LAUP is successful; however, the procedure has shown an inclination to aggravate patient′s pretreatment condition in the medium to long term. ***Berger et al; (2003)*** concluded that they found a significant worsening of the mean postoperative RDI, a surgical success in only one fifth of the patients (20%, 5/25) and a marked worsening of the postoperative RDI values in 9 patients (36%) in addition to a late worsening of the subjective initial results.

An American Sleep Disorders Association report published in 1994 withheld recommendation of LAUP as a suitable surgery to treat OSA, declaring it an experimental procedure because of insufficient data. An update for 2000 issued by the board of directors of the American Academy of Sleep Medicine stated that LAUP is not recommended for the treatment of sleep-related breathing disorders, including OSA. No specification has been given as to the type of LAUP technique being evaluated ***(Littner et al., 2001).***

Hypopharyngeal Surgery in Obstructive Sleep Apnea

Planning for surgical treatment of obstructive sleep apnea (OSA) begins with evaluation to determine the most likely site(s) of airway narrowing or collapse. By functionally dividing the pharynx into the retropalatal (corresponding to the portion of the oropharynx at the level of the soft palate and tonsils) and the so-called hypopharyngeal (actually corresponding to both the portion of the oropharynx at the level of the tongue base and the hypopharynx) regions, ***Fujita and Simmons (1987)*** described the following 3 patterns of obstruction: type I: retropalatal obstruction alone; type II: both retropalatal and hypopharyngeal obstruction; and type III: hypopharyngeal obstruction alone.

In ***1981, Fujita et al***. described uvulopalatopharyngoplasty (UPPP) as a treatment for patients with retropalatal airway obstruction. Although highly effective for simple snoring, with a control rate ranging from 75% to 87 %***(Hoffstein et al., 2000),*** a literature review by ***Sher et al. (1996)*** showed an overall response rate of 40.7% (with response defined as a 50% decrease in the respiratory disturbance index and a postoperative respiratory disturbance index of 20, or as a 50% decrease in the apnea index and a postoperative apnea index of 10) in patients with OSA treated with UPPP alone, regardless the site of obstruction. In patients with suspected retropalatal narrowing alone (type I obstruction), the response rate increased to 52.3%, but for those with a component of hypopharyngeal obstruction (types II and III), the response rate was only 5.3% ***(Sher et al., 1996).***

Several procedures have been developed to achieve higher response rates in patients with hypopharyngeal obstruction, and multiple

studies have reported the results of the new and older procedures. The procedures available to treat hypopharyngeal obstruction in OSA include genioglossus advancement, mortised genioplasty, tongue radiofrequency treatment, surgical reduction of the tongue base (midline glossectomy), hyoepiglottoplasty, hyoid suspension, and tongue base stabilization. These procedures can be performed alone or in combination.

Multiple studies have shown that these procedures improve outcomes in patients with suspected types II and III obstruction ***(Riley et al., 1994; Johnson et al., 1994)***.

Table 5. Genioglossus Advancement Results

Study	BMI, Mean	AHI, Mean		Success Rate, No. / Total No. (%) of cases*	LSAT
		Preoperative	Postoperative		
Riley et al, 1994	NR	NR	NR	9/23 (39)	
Johnson and Chlinn, 1994	NR	59	14§	7/9 (78)	Yes
Lee et al, 1999	NR	53	19§	24/35 (69)	
Miller et al, 2004	30	53	16§	16/24 (67)	yes

Abbreviations: AHI, apnea-hypopnea index, body mass index (calculated as weight in kilograms divided by square of height in meters; LAST, lowest level of oxygen saturation; NR, not reported.
***Defined as 50 % or more reduction in AHI and an AHI less than 20.**
§ p<.05.

Patients undergoing genioglossus advancement as the sole treatment of hypopharyngeal airway obstruction (**Table 5**) demonstrated, on average, severe OSA before surgery. after the procedure, there was significant improvement in the AHI in the three series that reported aggregate group data. Using the criteria for success based on AHI results, three studies ***(Johnson et al., 1994; Lee et al., 1999; Miller et al., 2004)*** reported success rates of more than 60%; while one series ***(Riley et al.,***

1994) had a lower rate. The 2 studies that considered the LSAT also showed an improvement in this metric.

Table 6. Tongue Radiofrequency Results

Study	BMI, Mean	AHI, Mean		Success Rate, No. / Total No. (%) of cases*	LSAT
		Preoperative	Postoperative		
Powell et al, 1999	30	40	18‖	7/18 (39)	Yes
Stuck et al, 2000	29	28	23	9/20 (45)	Yes
Woodson et al, 2001	31	41	33‖	11/55 (20)	No
Stuck et al, 2002	NR	25	17‖	6/18 (33)	Yes
Friedman et al,2003	32	44	28‖	(40)	No
Fischer et al, 2003	27.5	32.6	22	5/15 (33)	Yes
Kao et al, 2003		38.2	12.7	35/42 (83)	Yes
Riley et al, 2003	30	35	15‖	NR	Yes
Woodson et al, 2003	28	21	17	NR	No
Stuck et al, 2004	27.4	25.3	16.7	6/18 (33)	Yes
Verse et al, 2004	NR	27.8	22.9	5/15 (33)	No

Abbreviations: AHI, apnea-hypopnea index, body mass index (calculated as weight in kilograms divided by square of height in meters; LAST, lowest level of oxygen saturation; NR, not reported.

***Defined as 50 % or more reduction in AHI and an AHI less than 20.**

‖p<.05.

Eleven studies reporting tongue radiofrequency results are presented in **Table 6**. Overall, the patients were typically overweight or obese. The range of OSA severity included groups with moderate or severe OSA on the whole. Most of the studies reported a significant improvement in the AHI, and the proportion achieving a successful AHI outcome ranged from 20% ***(Woodson et al., 2001)*** to 83% ***(Kao et al., 2003)*** in separate series. Most of the studies that reported results showed an improvement in the LSAT, daytime somnolence, and quality of life. In individual studies, amount of local anesthetic and electrolyte solution mixture injected before treatment ***(Woodson et al., 2001)*** and the

Friedman stage ***(Friedman et al., 2003)*** were associated with outcomes. The evidence was mixed regarding the association between pretreatment AHI and successful outcomes ***(Friedman et al., 2003; Kao et al., 2003).***

Table 7. Midline Glossectomy Results

Study	BMI, Mean	AHI, Mean		Success Rate, No. / Total No. (%) of cases*	LSAT
		Preoperative	Postoperative		
Fujita et al, 1991	34.7	56	37	5/12 (42)	No
Woodson and Fujita, 1992	32.8	59	16§	17/22 (77)	Yes
Mickelson and Rosenthal, 1997	36.0	73	46§	3/12 (25)	Yes
Andsberg and Jessen, 2000	NR	35‖	18§‖	7/22 (32)	Yes
Li et al, 2004	NR	51	8§	5/6 (83)	Yes

Abbreviations: AHI, apnea-hypopnea index, body mass index (calculated as weight in kilograms divided by square of height in meters; LAST, lowest level of oxygen saturation; NR, not reported.
***Defined as 50 % or more reduction in AHI and an AHI less than 20.**
§ p<.05.
‖ Determined using the apnea index.

The case series for midline glossectomy are shown in **Table 7**. ***(Friedman et al., 2003; Fujita et al., 1991; Woodson et al., 1994; Mickelson et al., 1997; Andsberg et al., 2000; Li et al., 2004)*** these included groups that had a somewhat higher BMI and OSA severity (reflected by the AHI or the apnea index). Most had a statistically significant improvement in OSA severity. Again, the fraction of patients in whom a successful outcome by the sleep study criteria was achieved varied widely from 25% ***(Mickelson et al., 1997)*** to 83% ***(Li et al., 2004)***. Three of four series had improvement in the LSAT, but no results were reported for daytime somnolence or quality of life.

Hyoid suspension was originally described by ***Riley et al., (1984)*** with suspension of the hyoid bone to the inferior border of the mandible.

The technique was later revised to secure the hyoid arch anteriorly to the superior border of the thyroid cartilage ***(Riley et al., 1994)***.

Table 8. Hyoid Suspension Results

Study	BMI, Mean	AHI, Mean		Success Rate, No. / Total No. (%) of cases*
		Preoperative	Postoperative	
Vilaseca et al, 2002	27.8	48.3	29.0§	5/9 (56)
Neruntarat, 2003	29.3	44.5	15.2§	25/32 (78)
Den Herder et al, 2005	27.1	32.1	22.2	16/31 (52)
Bowden et al, 2005	34.1	36.5	37.6	5/29 (17)

Abbreviations: AHI, apnea-hypopnea index, body mass index (calculated as weight in kilograms divided by square of height in meters; LAST, lowest level of oxygen saturation; NR, not reported.
***Defined as 50 % or more reduction in AHI and an AHI less than 20.**
§ p<.05.

Four studies ***(Vilaseca et al., 2002; Neruntarat et al., 2003; Den Herder et al., 2005; Bowden et al., 2005)*** have considered the role of hyoid suspension as the sole procedure to treat hypopharyngeal airway obstruction (**Table 8**). The revised technique of hyoid suspension to the thyroid cartilage was used in all four series, and all patients had palate surgery performed previously or in the same setting. The patient populations are distinguished by their preoperative characteristics. Compared with the other two studies, the sample presented by ***Bowden et al, (2005)*** had a higher preoperative BMI and LSAT (82.1% vs. 72.7% in the study by ***Neruntarat et al, (2003)*** and not reported in the other 2 studies).

In turn, the postoperative results differ markedly in the raw improvements in AHI, as well as the proportion that achieved a successful outcome according to a sleep study; the population with the

highest BMI and higher LSAT had the worst outcomes. The series by ***den Herder et al, (2005)*** also showed that hyoid suspension performed in combination with palate surgery was associated with better outcomes (10 [71%] of 14 with successful AHI outcome) than for the procedure performed in isolation after previous, unsuccessful palate surgery (6 [35%] of 17 with successful AHI outcome). Finally, the series reported by ***Neruntarat et al, (2003) Vilaseca et al, (2003)*** and ***den Herder et al, (2005)*** showed an improvement in daytime somnolence, whereas the series reported by ***Bowden et al, (2005)*** did not.

Table 9. Hyoid Suspension in combination with Genioglossus

Study	Technique	BMI, Mean	AHI, Mean		Success Rate, No. / Total No. (%) of cases*
			Pre-operative	Post-operative	
Riley et al, 1994	Mandible	29	27	9.5§	133/233 (57)
Riley et al, 1994	Thyroid	30	45	13§	17/24 § (71)
Ramirez and Loube, 1996	Mandible	35	49	23	5/12 (42)
Utley et al, 1997	Thyroid	28.4	40.7	25	8/14 (57)
Bettega et al, 2000	Thyroid	27	47	47	5/21 (24)
Hsu and Brett, 2001	Thyroid	31	52.8	15.6	10/13 (77)
Vilaseca et al, 2002	Thyroid	27.8	70.5	57.4	2/11 (18)

Advancement or Mortised Genioplasty Results

Abbreviations: AHI, apnea-hypopnea index, body mass index (calculated as weight in kilograms divided by square of height in meters; LAST, lowest level of oxygen saturation; NR, not reported.
***Defined as 50 % or more reduction in AHI and an AHI less than 20 .**
§ p<.05.

Table 9 summarizes published results for the combination of genioglossus advancement with hyoid suspension. The patient populations, on average, were overweight or obese, and they demonstrated severe OSA. Postoperatively, the improvements in AHI were statistically significant in some but not all of the studies. The share

of patients achieving a successful AHI outcome ranged from 22% ***(Bettega et al., 2000)*** to 77% ***(Hsu et al., 2001)*** and most studies that reported data for daytime somnolence showed an improvement after surgery. Lower AHI and BMI were associated with a higher likelihood of successful outcomes in the largest study; successful AHI outcomes were achieved in 93 (74%) of 125 patients with an AHI less than 60 and LSAT less than 70 ***(Riley et al., 1993)***. Evidence from the group of studies also suggested that an SNB angle of more than 78° on pre- operative lateral cephalography was associated with better outcomes ***(Riley et al., 1993)*** and an SNB angle of less than 73.5° was associated with poorer outcomes ***(Ramirez et al., 1996).***

One study ***(Bettega et al., 2000)*** reported the combination of mortised genioplasty and hyoid suspension. Mean preoperative BMI was 25. The AHI did not change significantly (43 to 37), and 5 (22%) of 23 patients had a successful AHI outcome.

A single series also described the combination of tongue radiofrequency and hyoid suspension ***(Verse et al., 2004)***. The AHI improved from 39 to 21 after surgery, and there was also an improvement in LSAT. A successful outcome was achieved in 22 (49%) of 45 patients. The BMI was not reported.

Table 10 summarize all the above mentioned hypopharyngeal procedures and make a comparison between them and GA with HS.

Table 10. Comparison of Procedures and Combinations of Procedures

Procedure	Success Rate, No. / Total No. (%) of cases*	Compared with GA + HS (P Value)
Genioglossus Advancement	56/91 (62)	No difference (.26)
Mortised Genioplasty	16/33 (48)	No difference (.48)
Tongue Radiofrequency	95/269 (35)	Worse (<0.001)
Midline Glossectomy	37/74 (50)	No difference (.45)
Hyoid Suspension	51/101 (50)	No difference (.44)
Genioglossus Advancement & Hyoid Suspension	180/328 (35)	

Abbreviations :
GA Genioglossus Advancement, HS Hyoid Suspension
***Defined as 50 % or more reduction in AHI and an AHI less than 20.**

Radiofrequency ablation

During normal breathing, air passes through the throat on its way to the lungs. The air travels past the tongue, soft palate, uvula, and tonsils. The soft palate is the back of the roof of the mouth. The uvula is the prominent anatomic structure dangling downward visibly at the back of the mouth (pharynx). When a person is awake, the muscles in the back of the throat tighten to hold these structures in place preventing them from collapsing and/or vibrating in the airway. During sleep, the uvula and soft palate frequently vibrate causing the distinctive sounds of snoring ***(Boudewyns et al., 2000).***

The application of temperature-controlled radiofrequency (TCRF) for tissue ablation in the upper airway was first reported by ***Powell et al. (1997)*** in the in vitro bovine model and in vivo porcine model. At present the 3 most common radiofrequency devices are Somnoplasty (Somnus Medical Technologies Inc, Sunnyvale, CA), Coblation (Arthrocare Corporation, Sunnyvale, CA), and Ellman (Ellman International Inc, Hewlett, NY). These devices are capable of creating submucosal lesions (also known as radioablation) while simultaneously controlling bleeding. Radiofrequency procedures are generally performed in an outpatient setting with local rather than general anesthesia ***(Boudewyns et al., 2000).***

The application of radiofrequency to the throat tissues creates finely controlled localized burn-areas beneath the lining (mucosa) of the soft tissues of the throat. These burn- areas are eventually resorbed by the body, shrinking the tissue volume, opening the passageway for air, and thereby reducing symptoms of snoring ***(Boudewyns et al., 2000).***

TCRF has been shown by most centers to be safe and effective in treatment of the inferior turbinates, soft palate, and tongue in patients with snoring, sleep-disordered breathing (SDB) or nasal obstruction secondary to turbinate hypertrophy ***(Sher et al., 2001).***

Fig (25): treatment for snoring

fig (26): treatment for blocked nose

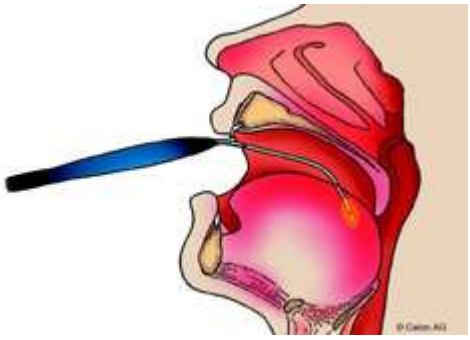


Fig (27): Tongue radiofrequency

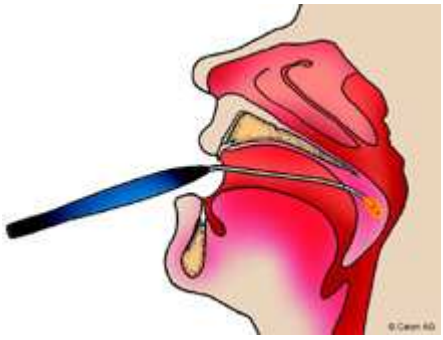


fig (28): palatal radiofrequency

The application of TCRF technology appears easy because the electrode is placed deep to the mucosa where there is essentially no visual evidence of the treatment effect below the surface. However, it is critical to have a basic understanding of the electrophysiological principle of

delivered radiofrequency energy to limit unwanted tissue damage and yet accomplish a positive treatment effect. This is accomplished more safely by carefully interpreting the real-time feedback information that is obtained concerning electrode temperature (degrees Celsius), power (watts, W), tissue impedance (ohms, Ω), treatment time (seconds), and total energy delivered (joules, J, or watts x seconds). Fortunately, with patience, perseverance, and experience, risks and complications may be limited ***(Bozkur et al., 2002).***

Complications after TCRF treatment may be related to several factors. These include the learning curve associated with the use of this technology and especially understanding of the electrophysiology of lesion generation for the various devices and treatment sites, the number and location of lesions created during each treatment session, and aspects of perioperative management other than corticosteroid use ***(Robinson et al., 2003).***

Regarding tongue radiofrequency, whether routine postoperative corticosteroids were used, it was concluded that the combined rates of moderate and major complications were 6.l% when corticosteroids were used and l.0% when steroids were not used. Student's t test for proportions revealed that this difference was statistically significant ($P < .001$) (***Riley et al., 2003)***.

A critical aspect of TCRF treatment is patient selection and tailoring treatment to patient anatomy. Treatment of the palate is markedly different from treatment of the tongue based in part on the anatomic character of the tissues (i.e., the tongue is thick, homogeneous

muscle, and the palate is relatively thin with diminutive muscle). Tissue bulk and consistency should be evaluated carefully, and one of the most important variables for palatal treatment is thickness. The midline palate is the safest region to treat because the musculus uvula provides the greatest bulk of the palate. The thickness of the palate decreases rapidly as one moves away from the median and paramedian region ***(Stuck et al., 2003).***

Palate thickness varies significantly in patients, if the palate is thin, the total energy delivery must be limited or damage to the mucosa will occur more frequently. Treatment to the lateral region of the palate should be undertaken only when it is established that the tissue thickness is appropriate. Lateral cephalograms can be used to measure the palate thickness in the midline, and the lateral regions of the soft palate are thinner to varying degrees and must be evaluated clinically. Clinical judgment must be used for optimal treatment planning ***(Stuck et al., 2003).***

There are obvious, important differences between various types of complications. ***Kezirian et al. (2005)*** classified these according to severity: minor (mucosal ulceration, mucosal crusting, or uvular sloughing), moderate (hemorrhage, palatal fistula, nerve paresis or paralysis, or significant dysphagia) or major (serious infection requiring drainage or other significant airway compromise)

One of the most common side effects of palatal radioablation, which occurs in 89% of patients, is swelling of the uvula. For a period of 24 to 48 hours after this procedure, the soft palate and uvula will undergo

extensive swelling and patients may have difficulty speaking, swallowing, or even breathing comfortably. Patients must be instructed to sleep in a recliner or with their head elevated while asleep. In general, swelling subsides on the third day after surgery ***(Madani, 2004).***

In extremely rare cases patients may have bleeding from the palatal radioablation. This is temporary and generally will stop without treatment. In fact 1 of the benefits of radioablation is its coagulation property. Accidental placement of the radiofrequency probes in or near palatal arteries may lead to such temporary bleeding. Simply remove the probe and inject the site with a small amount of lidocaine with epinephrine ***(Madani, 2004).***

During the healing phase of nasal radioablation patients may experience nasal bleeding. This side effect, although extremely rare, is caused by crust formation and subsequent dislodging of that crust. Patients must be advised to blow their nose very gently for up to 5 weeks after surgery. Keeping the nose packed with a small cotton roll for up to 24 hours prevents this bleeding. Patients can remove the packing from their nose after that, and there should be no need to replace it ***(Madani, 2004).***

The accidental overtreatment of a thin soft palate during palatal radioablation may lead to a palatal dehiscence and inability of the palatal musculature to seal the nasopharynx, with subsequent food and fluid reflux from the nasal cavity. This side effect is extremely rare and if noted at the time of surgery, such a dehiscence can be rectified by simply

placing a suture across the gap and the wound will heal without any problems ***(Madani, 2004).***

Failure to resolve or eliminate snoring or OSA is the most important complication to occur following palatal radioaplation. Because snoring and OSA are multifactorial, no single procedure will eliminate the patient's entire breathing problem. The most appropriate advice practitioners could give their patients is that these procedures will reduce the intensity of snoring but in no way will eliminate snoring altogether ***(Madani, 2004).***

Madani, (2004), concluded that palatal radioablation has a great tendency for relapse after 5 years. Of 1,200 patients initially treated for snoring by radiofrequency alone, 744 patients (62%) were re-treated with laser because of snoring relapse. Ninety-five percent of the 5,600 patients achieved a reduction of snoring intensity after LA-UPPP of 70%. Five to 10 years of follow-up shows that the procedure is stable as long as patients did not gain any additional body weight. Redevelopment of palatal flaccidity has been reported as a factor for relapse.

Nasal adhesions may occur following radiofrequency ablation of the inferior turbinates, the inferior turbinate and the nasal septum may develop a fibrous connection making breathing difficult and also causing nasal snoring (fig 29). The release of adhesion is performed under local anesthesia by using a simple electrocautery device such as the Ellman radiofrequency device. The inferior turbinate is then outfractured and an internasal splint or nasal pack is placed and kept in place for 2 to 5 days.

The potential of relapse is higher if the packing is removed prematurely ***(Madani, 2004).***

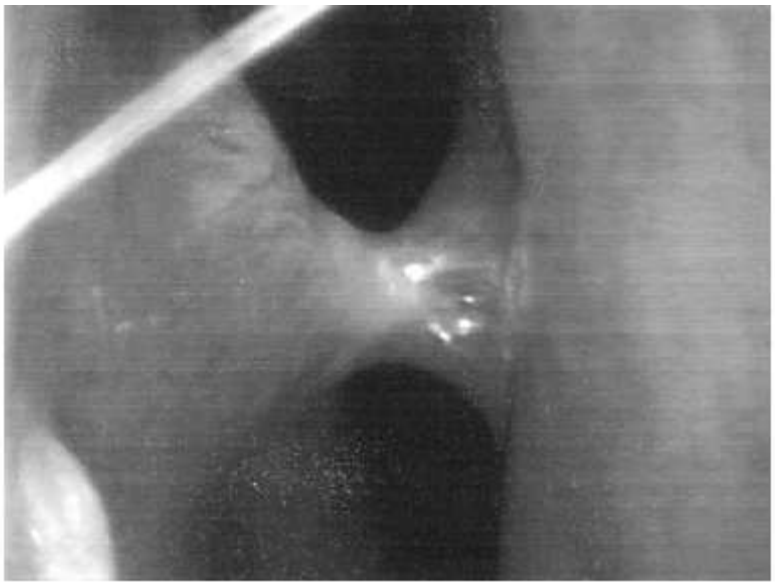


Fig (29) fibrous adhesion between the nasal septum on the left and inferior turbinate on the right could be a result of a traumatic accident to the nose or nasal surgery. This could easily be corrected by release of the adhesion and placement of an intranasal splint.
Mansoor Madani. Complications of UPPP Treatment of Snoring. J Oral Maxillofac Surg 2004.

Conclusion

The patients with OSA are not a homogeneous group with the same type of pathology. OSA is a multifactorial disease process creating a very heterogeneous patient population.

It is not one level causing the disease. To improve the surgical cure rates for OSA, the entire upper airway must be brought under inspection, and each level of obstruction should be identified.

Surgery chosen to OSA patient must be tailored for each patient alone as there are no two similar patients in all the circumstances.

There is no one surgical technique that is always fit for one level of obstruction, for example, U3P is not the only surgery that is fit for palatal obstruction or collapse.

The choice of surgery depends on different factors including:

Level of obstruction.

Skills of the surgeon

Availability of the different modern tools (Laser, radiofrequency, repose system, etc.)

Priority of which problem to be solved first, for example, nasal problems must be solved first.

Patients with mild obstructive disease are more likely to respond to surgical treatment than those with more severe disease.

Patients at less than 125% of their ideal body weight are most likely to have short-term and long-term benefits from surgical treatment of snoring and OSA.

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الملخص العربي

توقف النفس أثناء النوم يمكن تعريفه على أنه توقف حركة الهواء على الفم والأنف لزمن لا يقل عن١٠ ثواني مع وجود مجهود تنفس مستمر.

وفي الحالات الحرجة فإن فترات توقف التنفس تتكرر بمعدل يتراوح ما بين ٣٠ – ١٠٠ مرة في الساعة في مرحلة النوم الليلي .

وفي حالة حدوث توقف تنفس المريض فإن عملية تبادل الغازات بين الحويصلات الهوائية والهواء الجوي تتوقف مما يؤدي إلى هبوط احتياطي الأكسجين في الدم وبهذا قد يؤدي إلى نتائج خطيرة في الجسم وقد تكون مميتة .

وهذه الحالة لها عدة خيارات بالعلاج : علاج سلوكي وطبي وجراحي ويعتبر التنفس الصناعي الإيجابي (CPAP) هو أفضل أنواع العلاج وأكثرها أمنا ولكن معظم المرضى (أكثر من ٥٠ %) لا يستطيعون احتماله

وفي ما مضى كان يعتقد أن انسداد ما خلف سقف الحلق هو السبب الرئيسي لحالات الشخير وتوقف النفس أثناء النوم وبالتالي كانت عملية تجميل سقف الحلق هي أهم وأفضل العمليات الجراحية رغم أن نسبة نجاحها لا يتعدي ٤٠ % ولكن أظهرت الدراسات أن هناك مستويات أخرى للانسداد تشمل البلعوم ، ما خلف الحنجرة واللسان وبالتالي ظهرت عمليات أخرى لعلاج الشخير وتوقف النفس أثناء النوم.

ولكن كل هذه العمليات فشلت في الوصول إلى مستوى علاج كامل في حالات الشخير وتوقف النفس ولذلك ظهرت مجموعة من العمليات الجديدة التي تشمل أكثر من مستوى للانسداد في نفس الوقت وذلك أدى إلى ارتفاع نسب نجاح العمليات.

وأسباب فشل العلاج لا تتضمن عامل واحد بل عدة عوامل منها الجانب الطبي مثل السمنة المفرطة أو اضطرابات الهرمونات وتشمل أيضا أخطاء في تشخيص مستوى الانسداد حيث يكون هناك أكثر من مستوى للانسداد وتشمل أيضا أخطاء في اختيار العملية الجراحية المناسبة لكل مستوى في مستويا ت الانسداد.

وتخلص هذه الدراسة إلى :

أن المرضى يتوقف النفس أثناء النوم ليسوا مجموعة متجانسة ولكن توقف النفس أثناء النوم مرض متعدد العوامل يؤدي إلى خلق مجموعة غير متجانسة تماما من المرضى.

لكي نزيد مستويات النجاح في العمليات الجراحية لا بد من دراسة متأنية لكل الجهـــاز التنفسي العلوي لتحديد كل مستويات الانسداد.

لا بد أن تختار العمليات الجراحية لكل مريض على حـــدة لأنـــه لا يوجـــد مرضـــى متشابهون في كل الظروف .

لا توجد عملية جراحية واحدة لكل مستوى من مستويات الانسداد ولكن يوجد أكثر مـــن عملية لكل مستوى من مستويات الانسداد .

اختيار العمليات الجراحية يعتمد على عوامل عديدة ومنها :

مستوى الانسداد.

مهارات الجراح.

توافر مختلف الوسائل الحديثة مثل الليزر.

أي مستوى من مستويات الانسداد ويجب علاجه أولا.

دراسة الأسباب غير المرضيه فى علاج الشخير
و توقف النفس أثناء النوم

رسالة

توطئة للحصول على درجة الماجستير

فى الأذن و الأنف و الحنجره

مقدمة من

طبيب / أحمد أحمد محمد كامل

تحت اشراف

الأستاذ الدكتور/ أحمد عبد العال السلماوى

أستاذ الأذن و الأنف والحنجره

كلية الطب

جامعة القاهره

الدكتور/ محمد مصلح إبراهيم خليل

مدرس الأذن و الأنف والحنجره

كلية الطب

جامعة القاهره