Introduction

Definitions:

Obstructive sleep apnea: the cessation of airflow in the presence of continued respiratory effort.

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.

An hypopnea is a reduction in tidal volume. There is disagreement as to the exact definition of an hypopnea. **Gould et al (1988)** suggest it is a decrease in a 50% reduction in thoracoabdomenal movement lasting for 10 seconds in the presence of continued airflow. However, some authors describe it as decrease in airflow associated with oxygen desaturation **(Guilleminault et al., 1984).**

The sleep apnea syndrome (SAS): 30 or more apneic episodes during a 7-hour period of sleep or an apnea index equal to or greater than 5. This is an arbitrary definition and, 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 should be present before diagnosing the sleep apnea syndrome **(Gould, et al., 1988)**.

The American Sleep Association grade sleep apnea as follows:

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

Obstructive sleep apnea (OSA) is a disease process that was recognized 100 years before any definitive corrective therapy was attempted, with increased awareness of the disease in recent decades and with better diagnosis and understanding of the anatomy and pathology of the disease a wide array of treatment modalities has emerged ***(*Friedman, 1991*).***

The prevalence of OSA in the general population is at least affecting 2 to 4% of middle-aged adults ***(*Ip, et al., 2001*).***

Stradling and Davies (2004) found that OSA affects 24% of middle-aged men and 9% of women in the United States.

Obstructive sleep apnea syndrome (OSAS) has a multifactorial origin, each Factor acts with its own preponderance in each subject. Opening or closure (Partial or complete) of the upper airway depends on a subtle balance of forces among the following:

(1) Respiratory drive that determines negative suction pressure.
(2) Upper airway caliber.
(3) Respiratory control stability that influences synchrony (timing and activation intensity) between upper airway muscles and diaphragm .
(Bruno, et al., 1993)

In OSA, the apneic episodes produce aberrations of gas exchange, systemic and pulmonary homodynamic, and sympathetic neural activity **(Livinson and Millman, 1991).**

OSA is associated with decreases in the electromyographic activity of the pharyngeal dilators allow the subatmosphic pressures generated during inspiration to collapse the upper airway, during these episodes of upper airway occlusion, arterial oxyhaemoglobin saturation (SaO_2) decreases in association with concomitant elevation in systemic and pulmonary blood pressure **(John . et al., 1988).**

Reports suggest that (OSA) is associated with cardiac arrhythmias, conduction abnormalities, frequent arousals, and coronary artery disease (CAD**).** So that obstructive sleep apnea (OSA) is a serious medical disorder, which may be associated with sudden death during sleep **(Fichter , et al., 2002)**.

Conservative treatment of OSA has taken several directions, as weight loss; avoid alcohol and sedatives, nasal medication, drug treatment as protriptyline, Nosovent nasal splint, Mandibibular positional devise, Tongue retaining devise and Nasal continuous positive airway pressure **(Flemon, 2002).**

Surgical therapy of OSA is designed either to bypass the obstructive area or to prevent collapse of the soft tissues at the obstructive site **(Zohar, et al., 1993).**

Surgical techniques in use for treatment of OSA include tracheostomy, Uvulopalatopharyngoplasty (UPPP), maxillary, mandibular and hyoid advancement **(Zohar, et al., 1993).**

Also, laser-assisted uvulopalatoplasty (LAUP) is a technique using local anesthesia for treatment of snoring and mild to moderate obstructive sleep apnea (**Kamami, 1996**).

Also, Somnoplasty is an effective and minimally invasive choice for treatment of OSAS.Delivering radiofrequency energy submucosally to the soft palate and base of the tongue, Somnoplasty creates limited zones of coagulation beneath the tissue surface **(Guilleminault , et al., 1999).**

AIM OF THE WORK

The aim of this work is to detect the benefit of the

Surgical correction of obstructive sleep

apnea syndrome for reversal of the cardiopulmonary disorders associated with

it to the normal values.

Anatomical Considerations

ANATOMY OF THE PHARYNX

The pharynx forms the crossroads of the air and food passages. A muscular sphincter can close each major road from the pharynx. It opens anteriorly into the nasal cavity, the mouth and the larynx. The pharynx consists of the three parts: nasal, oral and laryngeal **(Gray, 1992).**

Nasopharynx

The nasopharynx or post-nasal space lies behind the nasal cavities and above the soft palate. The anterior wall is formed by the openings into the nasal cavities. A collection of lymphoid tissue, the nasopharyngeal tonsil (the adenoids) is found in the mucous membrane over-lying the basi-sphenoid. The nasopharyngeal tonsil has an oblong, rectangular shape, similar to a truncated pyramid, is situated submucosally at the junction of the roof and posterior wall of the nasopharynx. On each lateral wall of the nasopharynx is the opening of the pharyngotympanic tube. The inferior wall of the nasopharynx is formed by the superior surface of the soft palate **(Last, 1990).**

Oropharynx

The oral part of the pharynx extended from the junction of the hard and soft palates to the level of the floor of the valleculae. It opens anteriorly, through the oropharyngeal isthmus, into the mouth. Its lateral wall presents the palatopharyngeal arch and tonsil **(Beasley, 1997).**

The palatopharyngeal arch lies behind and projects further towards the median plane than the palatoglossal arch, it runs downwards, laterally, and backwards from the margin of the uvula to the side of the pharynx, and is formed by the projection of the palatopharyngeus muscle, covered with mucous membrane. On each side the palatopharyngeal and palatoglossal arches are separated by a triangular recess, the tonsillar sinus, in which the tonsil is lodged **(Gray, 1992).**

Laryngopharynx (Hypopharynx)

This part of the pharynx extends from the upper border of the epiglottis to the level of the cricoid cartilage. Its main features are the opening in the larynx, which bulges backwards into third part of the pharynx, and the pyriform recesses on either side. The three overlapping constrictors down to the level of the vocal folds form the posterior wall.

Pyriform fosse is small recesses lying on each side of the laryngeal inlet. Each is bounded by:

* Ary-epiglottic folds medially.
* Thyroid cartilage and thyrohyoid membrane, laterally.

Valleculae are paired shallow recesses lying between the base of the tongue anteriorly and the anterior surface of the epiglottis posteriorly.They are separated by the midline glosso-epiglottic fold and is bounded laterally by pharyngo-eoiglottic folds **(Last, 1990).**

The Nose:

The nasal passages are not static rigid structures, they are somewhat dynamic, being able to change intermittently and thereby increase and decrease resistance to airflow. A certain degree of resistance is a functional necessity. Resistance serves to slow and disperse airflow allowing the nasal mucosa to function more effectively. Nasal resistance accounts for 30 to 50 percent of total air resistance **(Ballinger, 1987)**.

The human nose contains several valves that regulate the airflow. The external nasal valve, which is dynamic structure, consists of four distinct airflow-resistive components. (i) The vestibule terminates in an airflow-resistive aperture between the septum and the caudal end of the upper lateral cartilage. Its cross-sectional area is stabilized by the cartilaginous structures and by inspiratory isometric contractions of alar dilator muscles. Its walls are devoid of erectile tissues that might otherwise affect its cross-sectional area and airflow resistance. By contrast, (ii) the bony entrance to the cavum is occupied by erectile tissues of both (iii) lateral (inferior turbinate) and (iv) septal nasal walls that modulate the cross-sectional area of the airway and airflow resistance. The body of the cavum offers little resistance to airflow. Valve constrictions induce "orifice flow" of inspiratory air as it enters the body of the cavum, disrupting laminar characteristics and thereby enhancing exchanges with the nasal mucosa of heat, water, and contaminants **(Cole, 2003**).

Normally, tins angle ranges between 10 and 15 degrees and the entire nasal valve area averages only 55 mm square. The nasal valve is regarded as the most crucial inflow regulator, accounting for most of the inspiratory resistance to the nasal airflow, about 50 percent of this resistance. In wide noses, the angle is less acute and the valvular effect is less. Disturbance of the nasal valve can produce symptoms of nasal airway obstruction, an important consideration in any nasal problem **(Frileck, 1986).**

The nasal turbinates, containing erectile tissues exert a significant valvular effect on the nasal airflow via their vasoconstriction and vasodilatation. The inferior turbinates exerts their valvular effect by increasing or decreasing turbulence. In the wide noses, they are the primary regulators of airflow **(Courtiss , 1983).**

The nasal septum, forming the septal valve is rigid and therefore, exerts a constant effect. Septal spurs and deviations can impair airflow and increase resistance to it. These deflections may develop at any of the septal articulations and spurs may be found where the quadri- lateral cartilage sends small processes between the ethmoid and vomer bones **(Lang , 1989)**.

The soft palate:

The soft palate is mobile, flexible partition between the nasopharyngeal airway and the oropharyngeal passage, and it can be likened to a set of points on a railway track, movement of which opens one line and close another. It extends posterior from the edge of the hard palate, and laterally it blends with the lateral walls of the oropharynx **(Schwab , et al., 1995).**

The base of the soft palate is the palatine aponeurosis formed by the expanded tendons of the tensor palati muscles that join in the median raphe. The aponeurosis is attached to the posterior edge of the hard palate and to its inferior surface behind the palatine crest, it is thicker in the anterior two thirds of the palate but very" thin further back. Near the midline, it splits enclose the uvular muscle, all the other muscles of the soft palate are attached to it. The anterior part of the soft palate is less mobile and more horizontal than the posterior part and it is principally this part that the tensor palati muscle acts **(Schwab , et al 1995).**

From the posterior edge of the soft palate hang the uvula in the midline. From the base of the uvula on each side, a fold of mucous membrane containing muscle fibers sweeps down to lateral wall of the oropharynx, this is the palatopharyngeal fold and the two folds together form the palatopharyngeal arch. More anteriorly, a smaller fold also containing muscle fibers, passing from the soft palate to the side of tongue, this is the palatoglossal fold, and with its opposite one, it forms the palatoglossal arch, which marks the junction of the buccal cavity and the oropharynx **(Schwab , et al., 1995)**

Muscles of the soft palate:

Tensor palati: It is a thin triangular muscle, which arises from the scaphoid fossa of the pterygoid process of the sphenoid bone, the lateral lamina of the cartilage of the pharyngotympanic tube and the medial aspect of the spine of the sphenoid bone. As it descends on the lateral surface of the medial pterygoid plate, its fibres converge to form a small tendon, which passes round the pterygoid hamulus of the medial plate before piercing the attachment of the buccinator to the pterygomandibular raphe and spreading out to form the palatine aponeurosis. The two-tensor palati muscles acting together tighten the soft palate primarily in its anterior part and depress it by flattening its arch. Acting alone, one tensor palati muscle will pull the soft palate to one side. This muscle is also the principal opener of the pharyngotympanic tube, assisted by the levator palati muscle **(Beasly, 1997).**

Levator palati: It is a cylindrical muscle arising by a small tendon from a rough area on the inferior surface of the petrous temporal bone; it also arises by a few fibres from the inferior surface of the cartilaginous part of the pharyngotympanic tube. The muscle passes downwards, forwards and inwards over the upper edge of the superior constrictor muscle where it pierces the pharyngobasilar fascia, and descends in front of the salpingopharyngeus muscle to be inserted into the upper surface of the palatine aponeurosis between the two bundles of the palato pharyngeus muscle.

The muscle fibrous tissues blend with those of the levator palati from the opposite side. The action of the muscle is to raise the soft palate upwards and backwards. Its action coupled with that of some of the upper fibres of the superior constrictor muscle, plays an important role in the closure of nasopharyngeal isthmus during deglutition. It also assists in opening the pharyngotympanic tube by elevating the medial lamina of the tubal cartilage **(Beasly , 1997).**

Palatoglossus: It is a small fleshy bundle of muscle fibres arises from the oral surface of the palatine aponeurosis where it is continues with the muscle of 'the opposite side. It passes anteroinferiorly and laterally in front of the tonsil where it forms the palatoglossal arch. It is inserted into the side of the tongue where some of its fibres spread over the dorsum of the tongue while others pass deeply into its substance. The action of the two muscles, together with the horizontal intrinsic muscle fibres of the tongue, is to close the oropharyngeal isthmus by approximation of the palatoglossal arches and elevation of the tongue against the oral surface of the soft palate **(Beasly , 1997).**

Palatopharyngeus: It arises in the palate as two bundles separated by the levator palati. The anterior bundle, the thicker one, arises from the posterior border of the hard palate and from the palatine aponeurosis. It passes back between the levator and tensor palati.The posterior bundle is thinner and arises from beneath the mucous membrane of the palate and passes medial to the levator palati.The two bundles unit at the postero-lateral aspect of the palate to descend in the palatopharyngeal fold before spreading out to form the inner vertical muscle layer of the pharynx and to be inserted into the posterior edge of the lamina of the thyroid cartilage. The action of the muscle is to pull the walls of the pharynx upwards, forwards and medially, so shortening the pharynx and elevating the larynx during deglutition, acting together, the two muscles approximate the palato-pharyngeal arches to the midline and direct food and fluid into the lower part of the oropharynx **(Beasly , 1997).**

Uvular Muscle: This is a small paired muscle arising from the palatine aponeurosis just behind the hard palate. Its fibres lie adjacent to the midline between the two lamina of the aponeurosis. It passes backwards and downwards to be inserted into the mucous membrane of the uvula. Its action is to pull up and shorten the uvula and to add bulk to the dorsal surface of the soft palate which assists in closure of the nasopharyngeal opening (velopharyngeal closure) in speech and deglutition **(Beasly , 1997).**

Nerve Supply: All the muscles of the soft palate except the tensor palati are supplied by way of the pharyngeal plexus. The tensor palati is supplied by the trigeminal nerve through nerve to medial pterygoid, a branch of mandibular nerve. The sensory supply to the palate is derived from the greater and lesser palatine branches of the maxillary division of the trigeminal nerve which pass on to the surface of the palate via greater and lesser palatine foramina **(Beasly , J997).**

Stages and architecture of normal sleep

Thousands of articles about sleep and sleep disorders appear each year in medical and psychological journals, and to date, more than 80 different sleep disorders have been described. However, despite the intensive efforts that these publications reflect, there is still no definitive explanation for why we sleep. Sleep is not a passive process, but rather an active state that is as complex as wakefulness.

The brain is not "at rest" during sleep; it is involved in a wide variety of activities. This card discusses the normal physiology of sleep, including sleep stages and architecture. The effect of sleep deprivation is discussed elsewhere.

OVERVIEW OF SLEEP STAGES –

Modern sleep researchers and clinicians analyze sleep using a standardized manual prepared by a committee chaired by Drs. A.Rechtshaffen and A. Kales.
This manual, first published in 1968, describes both recording methods and techniques for describing sleep **(Rechtshaffen, and Kales, 1968.)**.

Because of the large volume of data produced in a typical 8-hour sleep study, all sleep is analyzed in 20 or 30 second epochs, with each epoch being assigned a single sleep stage.
The Rechtshaffen and Kales (abbreviated as R & K) system subdivides sleep into two general states: rapid eye movement sleeps (REM) and non-rapid eye movement sleeps (NREM). NREM sleep is further subdivided into four NREM stages.

REM SLEEP - Rapid eye movement sleep was first described in 1953 when researchers observed periods of relatively fast electroencephalogram (EEG) activity recurring every 90 to 120 minutes (**Aserinsky and Kleitman, 1953**)
It is now generally accepted that REM sleep is characterized by three main features:

* A low voltage, fast frequency EEG pattern, which in many ways resembles an active, awake EEG pattern. For this reason, REM sleep is sometime called paradoxical sleep.

*An atonic electromyogram (EMG), consistent with inactivity of all voluntary muscles except the extraocular muscles. For all intents and purposes, the individual is paralyzed during REM sleep. The atonia of REM sleep is known to result from direct inhibition of alpha motor neurons. Atonia during REM sleep is incomplete or

absent in some humans; when absent, both humans and other animals appear to act out their dreams **(Hendriks, et al.,1982**).

In humans, the lack of appropriate muscle atonia during REM sleep is considered abnormal, causing a potentially dangerous sleep disorder called REM Behavior Disorder (RBD) **(Schenck, et al.1986.**)

*The presence of rapid eye movements. The chances are high that the patient will report dreaming if awakened during this period.

Phasic and tonic REM sleep –

All three primary characteristics of REM sleep are not always present simultaneously. REM sleep can be thought of as consisting of two somewhat different states, based on the occurrence of rapid eye movements in phasic bursts (phasic REM sleep) interspersed between periods of tonic REM sleep.

REM sleep is a predominantly parasympathetic (vagal) state, but during phasic REM sleep, there are sudden bursts of sympathetic nervous system (SNS) activity associated with rapid eye movements. These bursts of SNS activity have been reported to be associated with sudden increases in arterial blood pressure, cardiac or cerebral ischemia, cardiac arrhythmias, and sudden changes in heart and respiratory rates **(Hornyak, et al.,1991**). Short central apneas and hypopneas are also common during phasic bursts of rapid eye movements.

Long cardiac asystoles have been noted to occur in otherwise healthy young individuals during phasic bursts of REMs **(Guillimenault, et al., 1984).** It has been suggested that REM sleep, and phasic REM sleep in particular, may be a trigger for cardiovascular and cerebrovascular ischemic events that are reported to occur more frequently in the early morning hours **(Somers, et al.,1993).**

Function of REM sleep – The function of REM sleep is uncertain, although some data suggest an important role for REM sleep in memory consolidation (see Functions of sleep below). Following REM sleep deprivation by medication or frequent awakenings, REM sleep rebounds to quantities higher than normal. In addition, rats die after several weeks of total deprivation of REM sleep **(Rechtshaffen, et al., 1989**).
This suggests that REM sleep plays some essential, but not yet fully understood function.

Clinical issues related to REM sleep - Several aspects of REM sleep have relevance to clinical sleep disorders:

*Abnormalities in REM sleep are thought to be the cause of narcolepsy.

*The voluntary muscle atonia of REM sleep, as well as associated autonomic and metabolic changes, may exacerbate sleep apnea.

*REM sleep may be suppressed by alcohol, monoamine oxidase inhibitors, tricyclic antidepressants, stimulants, and some hypnotic/sedative drugs. Medications with prominent anti-cholinergic effects may also delay or suppress REM sleep.

NREM SLEEP - NREM sleep consists of four sleep stages, which are defined primarily by the frequency and amplitude of the EEG.

Stage I sleep Stage I sleep is the transition from wakefulness to deeper sleep, and is characterized by relatively fast EEG frequencies in the theta range (4 to 7 Hz). It is the lightest stage of sleep, and some sleep researchers do not consider it to be a true physiologic sleep stage. Additionally, patients awakened from stage I sleep typically do not perceive that they were actually asleep. Stage I sleep typically accounts for two to five percent of total sleep time in young adults. An increased amount or percentage of stage I sleep typically suggests sleep fragmentation due to a sleep disorder.

Stage 2 sleep - Stage 2 sleep, sometimes-called intermediate sleep, is a true physiologic stage of sleep. It typically accounts for 40 to 50 percent of total sleep time. Stage 2 sleep is characterized by a slowing of EEG frequency and an increase in EEG amplitude. Two distinct features of NREM sleep appear on the EEG for the first time in this sleep stage:

Sleep spindles. These are transient "spindle" shaped features of the EEG with a frequency of 12-14 Hz lasting at least 0.5 seconds. They are most prominent at the vertex of the scalp.

K- complexes. These consist of a high amplitude negative wave followed by a negative wave. They are usually thought to be associated with arousal.
Benzodiazepines typically increase stage 2 sleep at the expense of stages 3 and 4 sleep.

Stages 3 and 4 sleep - Stages 3 and 4 sleep, frequently referred to as deep sleeps or slow wave sleep, typically account for 20 percent of total sleep time in young adults. They are characterized by a transition to an EEG with high amplitude delta EEG waves (1.5 to 3Hz).

Function of NREM sleep - The function of NREM sleep is also uncertain, although stages 3 and 4 sleep have been reported to be associated with restorative functions of sleep, i.e., restoration of alertness and energy. The arousal threshold is highest during these stages. Some data indicate that deep sleep is increased in athletes after significant physical effort.

SLEEP ARCHITECTURE - Sleep stages occur in cycles lasting 90 to 120 minutes each. Four to five such cycles occur during a typical night of sleep. During the first half of the night, the individual typically passes from wake fullness briefly into stage I sleep and then to stages 2, 3, and 4. Stages 3 and 2 reappear, after which REM sleep is observed for the first time. During the second half of the night, stage 2 and REM sleep alternate.

Abnormalities of sleep architecture, such as REM sleep occurring earlier than 90 to 120 minutes, may be suggestive of sleep disorders such as narcolepsy, or may result from irregular sleep/wake organization, withdrawal from certain medications (e.g., tricyclic antidepressants, MAO inhibitors), or depression. Sleep disorders will increase the number of sleep stage changes and may completely disrupt the normal cycling of sleep.

Changes with age - Both the quantity and quality of sleep change significantly with aging **(Hirshkowitz, et al.,1992**).
In general, deep or slow wave sleeps (stages 3 and 4) declines with age, while light sleep (stage 1) increases.
The number of arousal's and the amount of wakefulness also increase in later years.
Physiologic functions- Physiologic functions, such as reflexes, physiologic feedback, and control systems, may vary with the state of consciousness. Differences occur during sleep compared to wakefulness, as well as during different stages of sleep, such as NREM, tonic REM, and phasic REM sleep (show figure 1) **(Orem and Barens ,1980**).

Hemodynamic and Ventilatory Changes in Normal Sleep

	NREM	Tonic REM	Phasic REM
Blood pressure	Decreases	Decreases	Increases
Heart rate	Decreases	Decreases	Increases
Vessel size	Vasodilatation	Vasodilatation	Vasoconstriction
Cerebral blood flow	Variable	Increases	Further increases
Respiratory rate	Decreases	Decreases	Increases
Minute ventilation	Decreases	Variable	Variable
Ventilatory responses to hypercapnia	Intact	Intact	Impaired
Respiratory muscle tone	No change/small decrease	Intercostals atonic, upper airway muscles hypotonic, diaphragm tonic	Diaphragm transiently inhibited, other respiratory muscles atonic or hypotonic
Airway smooth muscle	Decreases	Decreases	Increases
Arousal response	Faster than in REM	Slower than in NREM	Slower than in NREM

Orem J 1980 (Figure 1)

MEASUREMENT OF SLEEP AND SLEEPINESS –

Modern sleep medicine has a variety of objective tools for the measurement of sleep and sleepiness. Performing sleep studies as part of a diagnostic evaluation is common and is accepted by almost all-insurance carriers.

Polysomnography - Sleep is generally measured during an all night sleep study or polysomnogram (PSG), which involves simultaneous recording of several physiologic variables.

Sleep studies require attachment of electrodes to the scalp for recording the EEG, to the face near the right and left eyes for measurement of rapid eye movements, and to the chin for measurement of muscle tone **(Carskadon, et al., 1989).**

Other physiologic parameters, such as airflow and breathing effort, oxyhaemoglobin saturation, leg movements, electrocardiogram, and body position may also be measured depending on the purposes of the sleep study. All measurements are performed continuously throughout the patient's usual sleep period. All physiologic tracings are then analyzed, and a final report of sleep quantity and quality is prepared and interpreted.

Sleep studies are most often performed in specially designed and equipped laboratories, which are staffed by trained polysomnographic technologists. Sleep studies may also be performed in the home using ambulatory equipment. Ambulatory studies are not as comprehensive as studies in the sleep laboratory, but may be sufficient for more limited purposes. Significant controversy remains about the validity and reliability of ambulatory sleep studies in the patient's home, especially if a technician is not present.

Multiple sleep latency test - The multiple sleep latency test (MSLT) is an objective measure of daytime sleepiness that has been in common use for more than 15 years **(Carskadon, et al., 1986**.)
It was developed because patients frequently appear to be unaware of just how sleepy they are. The test is based upon an operational definition of sleepiness: the sleepier an individual is, the faster (s) he will fall asleep. The following protocol is typically used:

*The patient is given four to five opportunities to nap, usually at two-hour intervals during the day.

*On each occasion, the individual is asked to lie down on a bed in a quiet, darkened sleep room and fall asleep as quickly as possible. The EEG, eye movements, and muscle tone are measured during the test.

*The latency from wakefulness to sleep onset is measured to determine the "sleep latency." Each session is terminated after 15 minutes of sleep.

*The process is repeated during each of the four to five naps, and means sleep latency across all the naps is computed.Generally, a mean sleep latency of 5 minutes or less is considered indicative of severe daytime sleepiness, while a mean sleep latency of 15 minutes or longer is consistent with normal alertness.
The abnormal appearance of REM sleep during two or more of the four to five naps is also thought to be consistent with a diagnosis of narcolepsy.

Maintenance of wakefulness test - The maintenance of wakefulness test (MWT) is a variant of the MSLT **(Mitler, et al., 1982).** However, it differs in its goals as well as in the nature of the instructions given to the patient. Patients are typically tested while reclining in a quiet, darkened room. Instead of being asked to fall asleep as quickly as possible, they are requested to stay awake for as long as possible.

Conceptually, the MWT is a test of the individual's ability to stay awake, and it is therefore believed to be a more practical test of whether a person's sleepiness is likely to impair the ability to drive or work. The MWT also allows the element of motivation to enter into the equation, as is likely to be the case in real life situations.

Epworth sleepiness scale - The Epworth sleepiness scale is a paper and pencil scale that has recently come into wide use, typically as part of an office evaluation **(Johns, 1991).**

It describes a number of situations (e.g., as a passenger in a car, sitting quietly after lunch, etc) and asks the patient to rate how likely it would be for him or her to become drowsy or fall asleep in those situations (show figure 2). Although there are no well-established norms, a score >12 is usually thought to be abnormal.

Epworth Sleepiness Scale

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

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

Situation	Chance of dozing
Sitting and reading	_____
Watching TV	_____
Sitting inactive in a public place (eg, a theater or meeting)	_____
As passenger in a car for an hour without break	_____
Sitting and talking to someone	_____
Sitting quietly after lunch without alcohol	_____
In a car, while stopped for a few minutes in traffic	_____

(Redrawn from Johns, MW, Sleep 1991; 14:40.)

Johns,MW 1991
(Figure 2)

FUNCTIONS OF SLEEP – There are no general agreement about the possible function(s) of sleep, or even about whether there is one or more than one function of sleep. Numerous theories have been raised over the years **(Horne, 1988).** One, some, all, or none of these theories may be true.

* The most prominent theory is the Restorative Theory of Sleep, which, in its simplest form, states that some process during sleep restores tissue and prepares the body for the next day. This theory certainly appears to have validity on face value, as a night without sleep does not leave one prepared for the next day.

* The Adaptive Theory of Sleep proposes that sleep increases survival. In its simplest form, it states that sleep immobilizes animals during the most dangerous time of the day, decreasing their chances of becoming another animal's prey.

* The Energy Conservation Theory states that a low metabolism for energy conservation is the function of sleep.

Pathophysiology of OSAS

The obstructive sleep apnea syndrome (OSAS) arises from a sequence of primary events, which, in the fully developed case, repeat itself, hundreds of times each night. With the onset of sleep, the upper airway occludes, resulting in cessation of airflow despite continuing respiratory efforts.
As a result of the apnea, the patient becomes progressively asphyxiated until the apnea is terminated by a brief arousal from sleep, restoration of upper airway patency and resumption of airflow. With the return of breathing and relief of asphyxia, the patient typically returns to sleep quickly, only to have the sequence of events repeated over and over again **(Bradly, 1985).**

Mechanisms and anatomic sites of upper airway obstruction in obstructive sleep apnea

A variety of factors contributes to a decrease in upper airway diameter during sleep, even in normal individuals without obstructive sleep apnea (OSA).

An understanding of the mechanisms that underlie OSA requires consideration of these factors as well as the pathophysiologic features that distinguish patients with OSA from their normal counterparts.

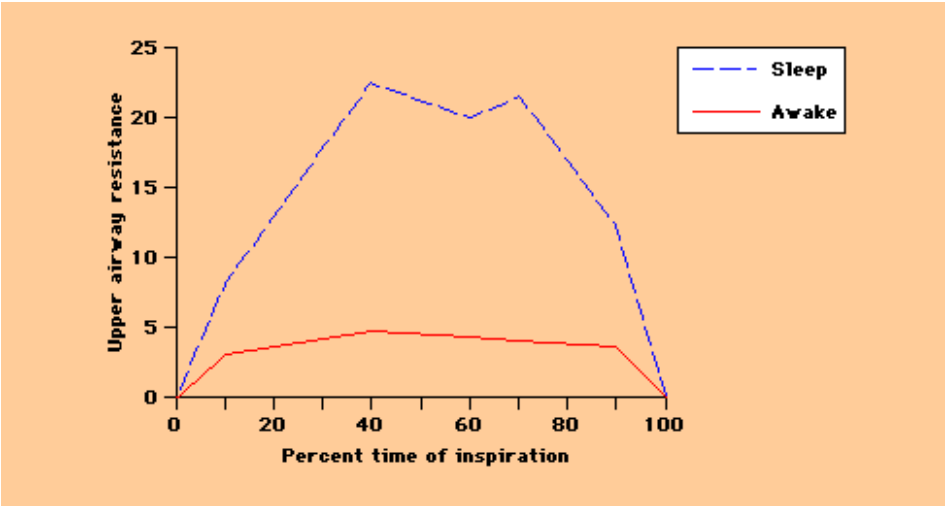
FACTORS INFLUENCING UPPER AIRWAY DIAMETER DURING SLEEP
Airway size is dependent upon a balance of the following factors:

*Those factors exerting an outward force, thereby maintaining luminal diameter and patency, e.g., contraction of the dilator muscles of the upper airway.

*Those factors exerting an inward force, thereby decreasing luminal diameter and patency, e.g., reduced intraluminal pressure.

During wakefulness, the upper airway remains patent regardless of size, except in some rare cases. However, the balance between these factors changes during sleep and many individuals are at risk for partial or complete upper airway obstruction. The video demonstrates upper airway occlusion occurring during inspiration in an individual with OSA during sleep. However, airway narrowing and increased upper airway resistance also occur in normal, non-snoring individuals during sleep **(Kay , et al., 1995.)**

The relative change in upper airway resistance over the course of inspiration, averaged for a group of normal, non-snoring subjects during wakefulness and sleep, is shown in Figure 3.



Upper airway resistance Averaged data from a group of non-snorers. Plotted is upper airway resistance versus percent time of inspiration in wakefulness (solid line) and non-REM sleep (dashed line). (Redrawn from Kay, A, Trinder, J, Kim, Y, J Appl Physiol 1995; 79:411).

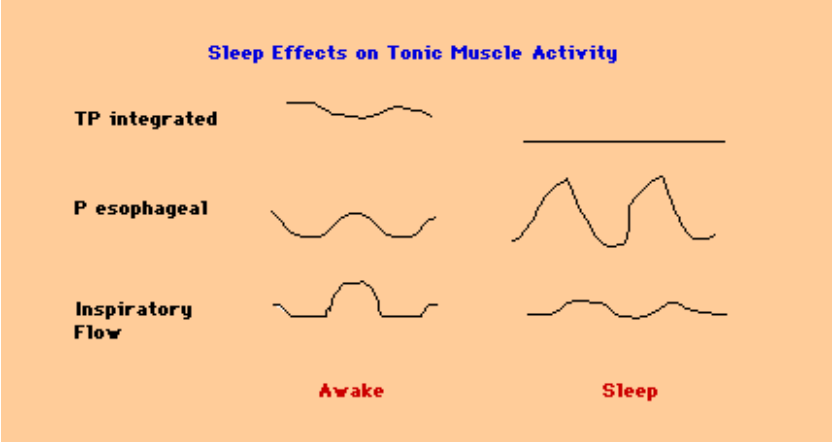
(Figure 3)

Several factors related to the loss of wakefulness may explain these changes:

Decreased drive to the muscles of the upper airway Many investigators believe that a reduction in drive to the dilator muscles of the upper airway out of proportion to the reduction in drive to the chest wall or pump muscles is the primary cause of airway narrowing and obstructive events during sleep. This disproportionate change may be a direct result of a decrease in medullary respiratory activity, particularly in those neurons that have a weak correlation with respiration **(Orem, et al., 1985).** This may translate into a reduction in activity of those respiratory muscles with both respiratory and postural functions, such as the muscles of the upper airway.

Activity of the upper airway muscles can be divided into two components: tonic and phasic. Most of the phasic activity, which is relevant to this discussion, is inspiratory.

Tonic activity refers to the activity during expiration or the level of activation in muscles without any phasic activity. The tonic component is reduced with the loss of wakefulness as seen with the tensor palatini (show figure 4) **(Tangel, et al., 1991**).



Effect of sleep on tonic muscle activity Integrated tensor palatini (TP) EMG activity, esophageal pressure (P esophageal) (negative is up in this figure), and inspiratory flow in a nonsnorer during wakefulness and sleep. Sleep is associated with a reduction in TP activity, an increase in esophageal pressure, and a reduction in airflow. (Redrawn from Tangel, DJ, Mezzanotte, WS, White, DP, J Appl Physiol 1991; 70:2574).

(Figure 4)

This figure also demonstrates that the reduction in tonic tensor palati activity is accompanied by an increase in esophageal pressure and a reduction in flow, i.e., an increase in inspiratory resistance. In contrast, phasic upper airway muscle activity shows no change or may even increase during sleep **(Tangel, et al., 1992).**

These findings suggest that the loss of tonic, rather than phasic activity, is the important factor, which compromises airway patency during sleep.

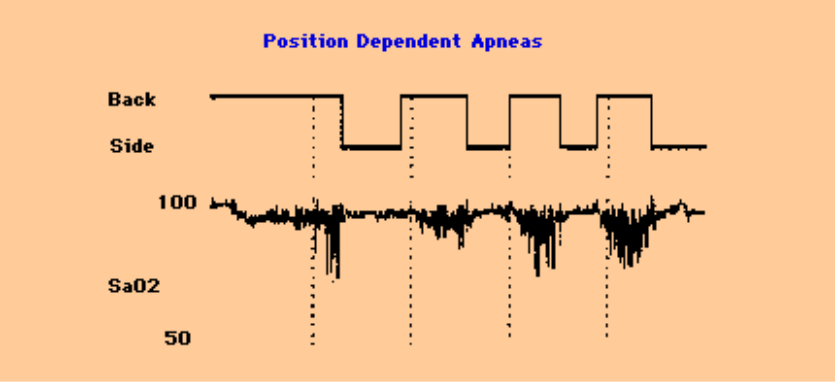
Loss of compensatory reflexes - There is substantial evidence that the ability to respond immediately to increases in negative pressure is lost or attenuated with sleep onset. Sleep appears to reduce the magnitude of the genioglossus' response to negative pressure and to increase the latency of the response **(Horner, et al 1994).**

This may be important because upper airway muscle activity during wakefulness is higher in patients with obstructive sleep apnea than in non-snorers, suggesting that this activity may compensate for a smaller airway in such patients **(Mezzanott e, et al 1992).**

Loss of this compensatory dilating activity with sleep onset would therefore put the patient at risk for obstruction.

Posture - Although not a primary mechanism for upper airway obstruction, body position plays an important role in the genesis of obstructive events **(Martin, et al., 1995).**

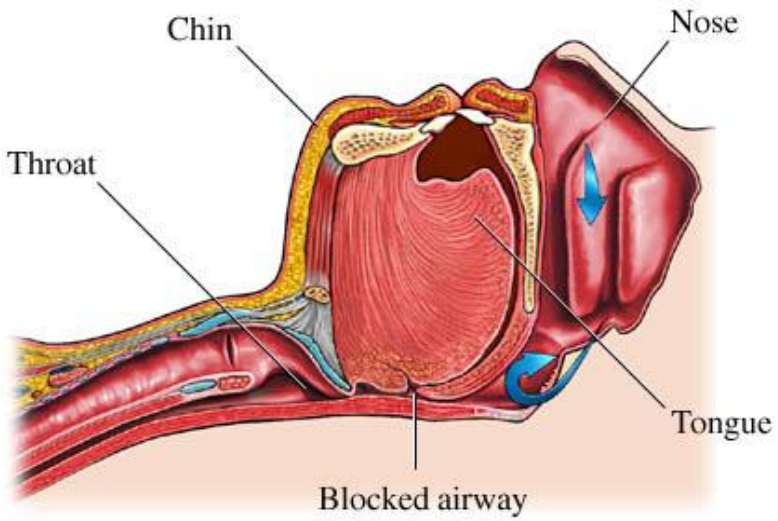
Some individuals with position-dependent sleep apnea obstruct exclusively in the supine position (show figure 5).



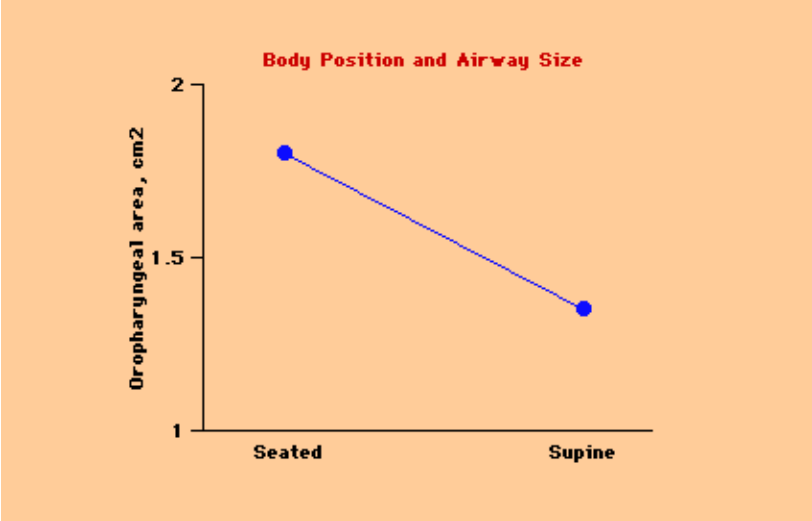
Position-dependent apneas A portion of the oximetry and body position data from an overnight polysomnogram in a man with position-dependent OSA. Oxygen desaturation occurs only when the patient is on his back.

Martin et al ,1995 (Figure 5)

When an individual is in the supine position, soft structures such as the tongue and soft palate can be drawn into the pharyngeal airway by the effects of gravity. As a result, the area behind the tongue and soft palate becomes a site of airway narrowing, which can lead to upper airway obstruction (show figure 6 A,B).



Martin et al ,1995 (Figure 6 A)



Airway size falls in supine position Oropharyngeal area in the seated and supine positions during wakefulness. (Redrawn from Martin, SE, Marshall, I, Douglas, NJ, Am J Respir Crit Care Med 1995; 152:721).

(Figure 6 B)

Another effect of the horizontal sleep posture is a decrease in lung volume. Reductions in lung volume have been shown to cause upper airway narrowing, possibly by reducing caudal traction on the airway **(Hudgel, et al., 1984).**

However, upper airway narrowing has not been reported in-patients with reduced lung volumes due to intrinsic lung restriction. This may be due to the differential effects of intrinsic lung restriction on diaphragm position and pleural pressure **(Van De Graff, 1988).**

Alcohol and sedative/hypnotics - Alcohol, benzodiazapines, and other similar agents have been shown to cause or worsen sleep-disordered breathing in non-snorers, snorers, and patients with obstructive sleep apnea **(Issa and Sullivan, 1982).** These substances may increase sleep-disordered breathing by depressing respiration and/or preferentially inhibiting upper airway muscle activity.

Features distinguishing patients with OSA from normal –

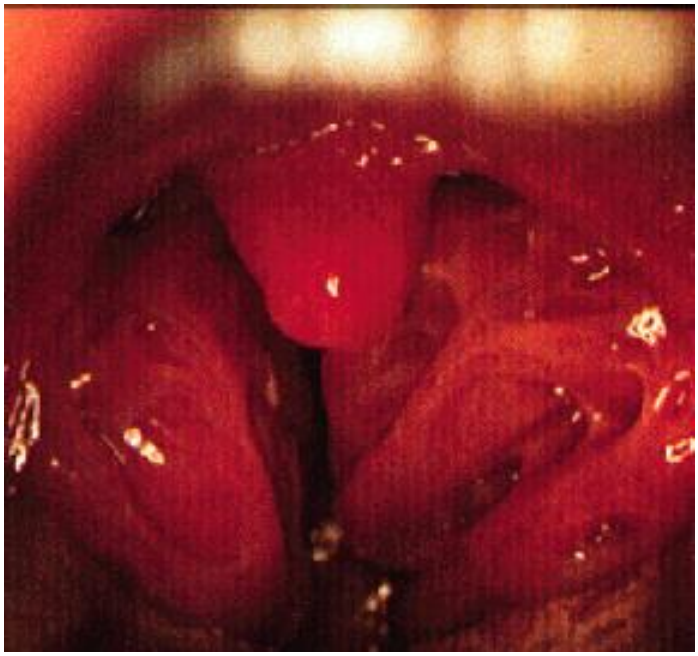
As noted above, the upper airway narrows in all individuals during sleep. However, in normal, non-snoring adults, the upper airway is generally not at risk for closure even after alcohol ingestion or experimentally induced airway occlusion. The pathophysiologic factors, which predispose the upper airway of the patient with OSA to closure, can be classified as either anatomic or neural.

Anatomic factors - Any factor that causes a section of the upper airway to narrow can contribute to airway collapse.

The increased resistance will cause a pressure drop across the narrowed site. The downstream section of the airway will then be at risk for collapse when muscle

tone is reduced during sleep. Some anatomic factors such as obesity can also contribute to airway collapse by increasing the transmural pressure in the collapsible segment of the airway.

It has been demonstrated that the upper airway of the patient with OSA is smaller than the airway of a non-snorer. Thickened lateral pharyngeal walls have been shown to account for most of the narrowing of the upper airway in individuals with OSA (**Schwab, et al., 1995**)
Airway narrowing can also be caused by:
*Nasal obstruction.
*Physical obstruction, e.g., with tonsillar hypertrophy (show figure 7).
*Conditions, which cause macroglossia and facial malformations.
*Obesity.



Tonsillar hypertrophy in OSA patient

Schwab et al ,1995 (Figure 7)

There are at least three explanations by which increased nasal obstruction is thought to be related to OBS. These are alteration in airflow dynamics, stimulation of neural reflexes, and genetic predisposition **(Metes et al 1992).** When nasal obstruction substantially alters airflow dynamics, a significant increase in pressure gradients

across the upper airway occurs. This results in pharyngeal collapse and soft tissue change in the area of the hypopharynx and oropharynx. This alteration in airway dynamic is believed to be the predominant mechanism by which nasal obstruction is linked to the development of OBS. The neural reflex mechanism is explained by alterations in airflow that are detected by nasal receptors. These in turn initiate autonomic changes in respiratory rhythm. The third mechanism is related to a genetic predisposition toward sleep induced respiratory instability that is incited by increased nasal resistance **(Deegan , 1996).**

NASAL OBSTRUCTION AND THE PATHOGENES1S OF OBSTRUCTED BREATHING DURING SLEEP

Altered Airflow Dynamics

Airflow into the lungs begins once diaphragmatic muscle contractions establish negative endothoracic pressure. A negative pressure gradient is generated across the upper airway. Increases in nasal resistance to airflow elevate the strength of muscle contraction required to inspire. This is related to the physiologic principle that by increasing the load (nasal resistance) applied to a muscle (the diaphragm)/ up to a point/ the strength of muscular contraction (inspiration) is increased. Under normal circumstances the pharynx is collapsible and a major limiting factor to inspiratory flow. The resultant elevations in negative pressures within the pharynx promote its tendency to collapse **(Peter , 1985).**

This tendency toward pharyngeal collapse is exacerbated by turbulent airflow. Increased nasal airflow/ secondary to greater pressure gradients/ also is associated with increased turbulent airflow. This turbulence can reduce further the pressures within the pharynx by creating subatmospheric pockets of air. Snoring results when the soft palate or pharyngeal tissues vibrate in response to the pressure gradients and turbulent airflow. Obstructive apneas finally develop when the critical airway collapsing pressure is exceeded **(Lugaresi , 1983).**

Neural Reflex Mechanism

Upper airway obstruction can affect the respiratory system through disturbed reflex mechanisms that may be trigeminally or vagally mediated. There is clinical evidence to show that when the afferent input from the nasal sensory receptors is acutely decreased/ respiratory rhythm is disturbed and leads to apnea. **White et al (1985)** conducted an experiment comparing the impact of nasal lidocaine and oxymetazoline (decongestant) to the effects of oxymetazoline alone in 10 sleeping normal men. This study design permitted the elimination of the sensation to airflow without the additive effect of nasal congestion known to occur with the topical administration of nasal anesthetics.

There were statistically significant increases in disordered breathing during sleep (central and obstructive apneas combined) by comparison to control nights where decongestant alone was administered (**Hoffstien ,1993).**

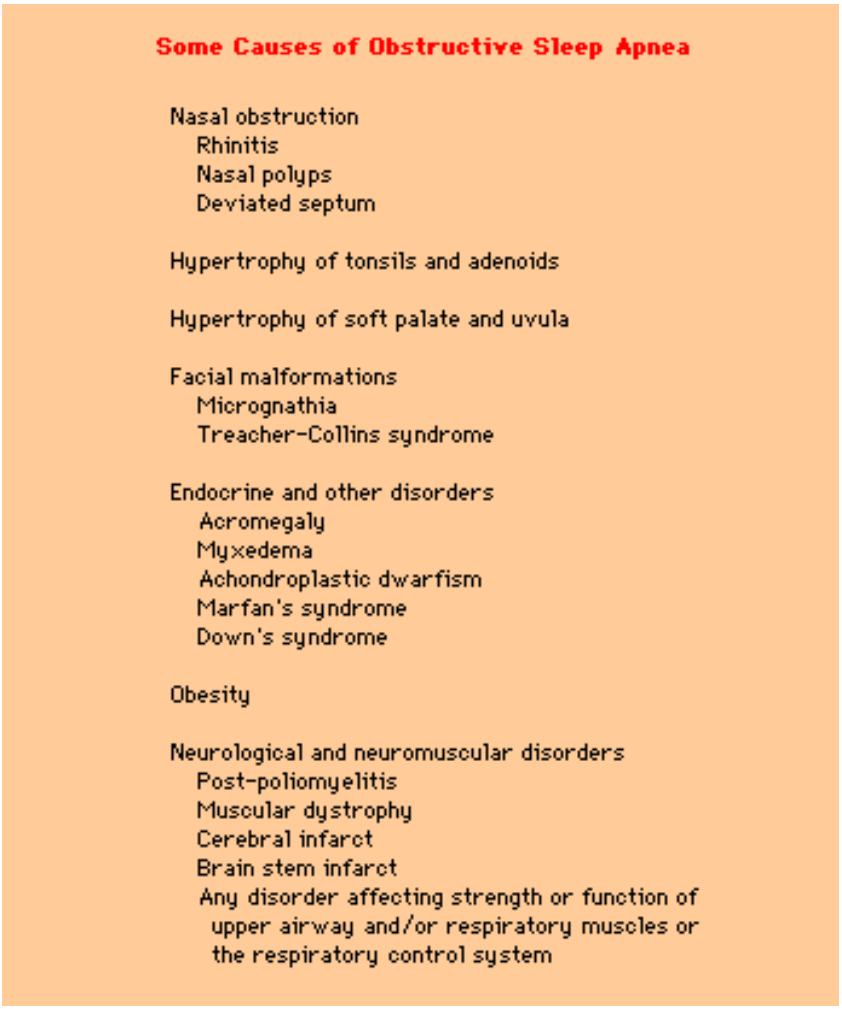
The effects of nasal resistance changes also have been documented to affect diaphragmatic breathing, pulmonary compliance, and the cardio-respiratory system. One explanation for decreases in arterial oxygen tension (PaO_2) seen with nasal obstruction has been the so-called **nasopulmonary reflex**. There is no uniformity of opinion regarding the specific mechanism and/or existence of this reflex. It has been suggested that the nasopulmonary reflex is induced when certain nasal stimulation (trigeminal) has the direct effect of increasing pulmonary resistance and decreasing pulmonary compliance (autonomic). These changes in the intrinsic function of the pulmonary system then lead to decrease ventilation of the alveoli with decreased oxygenation **(Wheker et al, 1978).**

Genetic Predisposition to Sleep Induced Respiratory Instability

Increased upper airway resistance, excessive weight, or pathologic decrease in central nervous system (CNS) excitation result in sleep irritability. According to this explanation, breathing disorders during sleep can be seen as a continuum ranging from OSAS to snoring. Patient's location along this continuum is determined by the presence of a genetic predisposition for sleep induced respiratory instability and the existence of any one or a combination of factors. **Lavie and Rubin (1984)** found that nasal occlusion resulted in significantly higher frequency of apneic episodes in six sons of OSAS patients when compared with those of four age-matched control subjects without familial history of OSAS.

Obesity contributes to airway narrowing through fatty infiltration in the tongue, in areas surrounding the airway, and/or because of enlarged fat pads lateral to the airway (**Winter, et al., 1995).**

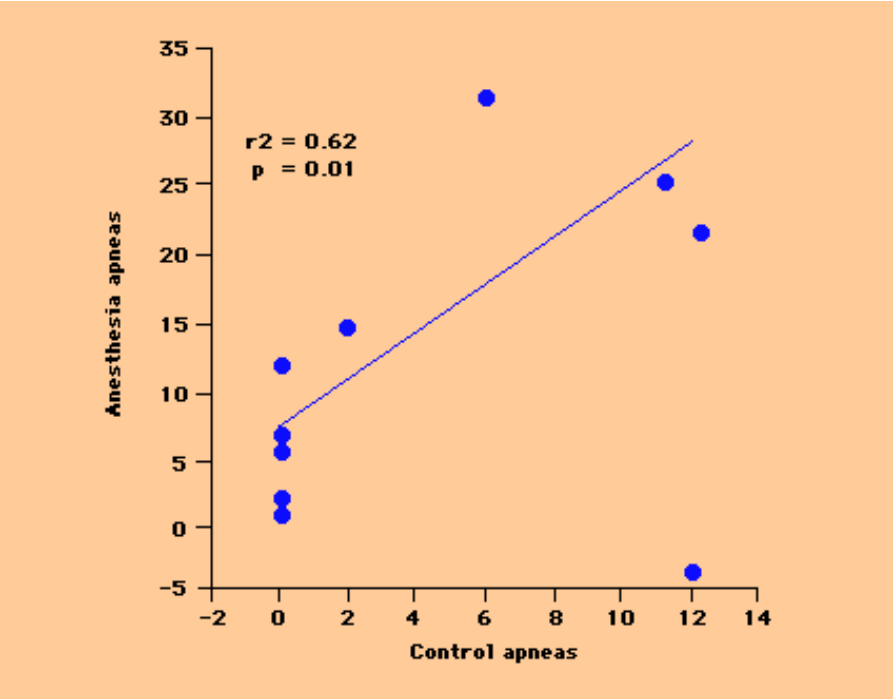
Figure (8) shows a more inclusive (but certainly not complete) list of factors contributing to OSA.



(Figure 8)

Neural factors it has been suggested that neuromechanical impairment of the upper airway can contribute to upper airway obstruction in-patients with OSA.This impairment could be due to a reduction in the sensitivity of upper airway flow and/or pressure receptors or to inappropriate activation of the musculature, either interims of timing or strength. Studies in which upper airway anesthesia caused or worsened sleep-disordered breathing have raised the possibility that impairment in upper airway receptors may be a factor in OSA (show figure 9) **(Mcncholas, et al., 1987**).

Also Friberg et al (1997) identified frequent focal degeneration of myelinated nerve fibers and axons by electron microscopy of moderate and severe apneic patients. So degenerative changes in peripheral nerves that they identified on electron microscopy might contribute to airway instability and the development of obstructive apnea by impairing pharyngeal reflexes.



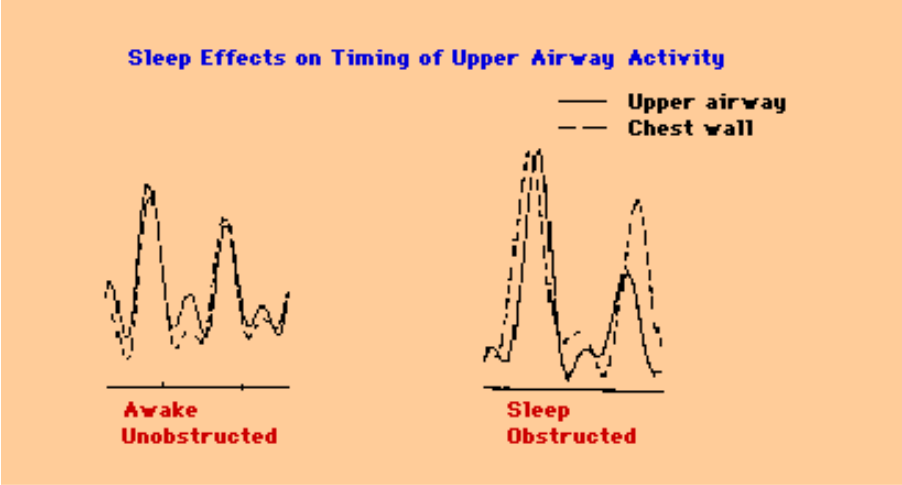
Anesthesia apneas correlate with control apneas The number of apneas which occurred during sleep after upper airway anesthesia is plotted against the number of apneas during the control study in a group of non-snorers. Note the increase in respiratory events with upper airway anesthesia which positively correlates with the number of events which occurred during the control study. (Redrawn from McNicholas, WT, Coffey, M, McDonnell, T, et al, Am Rev Respir Dis 1987; 135:1316).

(Figure 9)

However, there is currently little direct evidence for a subsensitivity of airway receptors in individuals with OSA.

Maintenance of upper airway patency depends upon appropriate timing and activity of upper airway muscles during inspiration, as upper airway dilator activity must be sufficient to hold the airway open against the suction forces created by the inspiratory pump muscles.

If inspiratory forces are generated before the upper airway muscles are activated, the patency of the upper airway is compromised. It has been demonstrated that, during apneic and high resistance breaths, activation of the inspiratory pump muscles precedes genioglossus activation (show figure 10) (**Hudgel, et al., 1990).**



Sleep effects on timing of airway muscle activity
Moving time average of upper airway muscle and chest wall EMG. During wakefulness (left panel), chest wall activation is slightly preceded by upper airway muscle activation. During obstructed breathing in sleep (right panel), upper airway activation **follows** chest wall activation, leaving the upper airway vulnerable to collapse. (Redrawn from Hudgel, DW, Harasick, T, J Appl Physiol 1990; 69:443).

(Figure 10)

This imbalance of activation is probably due to the periodic nature of the breathing pattern in OSA. Furthermore, sleep deprivation itself may reduce upper airway muscle activation **(Leiter, et al, 1985).**

This may be important in the progression of obstructive sleep apnea, since sleep disruption is an integral part of the syndrome. A vicious cycle is created, in which sleep disruption leads to worsening of sleep-disordered breathing, which then leads to further sleep disruption.

Upper airway muscle fatigue has also been suggested as a factor in OSA. At this time, however, there is little convincing evidence for this hypothesis.

Sites of upper airway obstruction in OSA - Although anatomical narrowing can occur at any site from the nares to the larynx, airway closure does not necessarily occur at the site of narrowing.

The narrowest site may have a regional compliance that prevents upper airway closure at that site, but can cause a sufficient fall in downstream pressures to promote airway closure at a more caudal site. Upper airway closure most commonly occurs in the nasopharynx **(Morrison, et al., 1993).**

However, this primary site is not necessarily the sole site of closure. In one study, for example, 80 percent of the patients studied had either multiple primary closure sites or one primary and one or more secondary closure sites **(Morrison, et al., 1993**).

These sites of closure were identified by the use of pressure catheters inserted at different levels of the upper airway and by visual inspection via fiberoptic endoscopy. The multiple pressure catheter technique has been used clinically in an attempt to identic individuals who may benefit from uvulopalatopharyngoplasty**.**

CARDIOVASCULAR EFFECTS OF OSA

Patients with obstructive sleep apnea (OSA) experience repetitive hemodynamic oscillations during the night. Changes in systemic arterial blood pressure, pulmonary arterial blood pressure, heart rate, and cardiac function occur in association with alterations in sleep state and in respiration.

These hemodynamic changes may be dramatic, with post-apneic systolic arterial pressures exceeding 300 mm Hg in patients who are normotensive while awake during the day. Because of these extreme changes after upper airway obstruction during sleep, recent investigations have attempted to examine the relationship of OSA to cardiovascular morbidity and mortality.

Questions commonly raised by physicians caring for patients with sleep apnea include:

* Is sleep apnea a cause of systemic hypertension that is difficult to control?
* Should sleep apnea be investigated in all patients with pulmonary hypertension?
* Does untreated sleep apnea contribute to myocardial infarction and cerebrovascular disease?

While these questions are not yet completely answered, some recent studies can guide clinicians caring for these patients. **(Podzus , et al., 1986**.).

Relationship of sleep apnea to systemic hypertension -

Sleep-disordered breathing (SDB) plays a causal or' contributing role in the development of comorbidities such as hypertension and cardiovascular events. Snoring, SDB, and obstructive sleep apnea (OSA) have been reported to be associated with hypertension since the early 1980s .There is substantial epidemiological and pathophysiological evidence to suggest that SDB causes hypertension. Moreover, an association between sleep apnea and hypertension that is independent of age, sex, and body weight has been reported **(Kales , et al., 1984)**.

Systemic hypertension is seen in up to 70% to 90% of cases of OSA **(Lugaresi , et al., 1978).** On the other hand, OSA is detected in about 30% to 35% of individuals with a primary diagnosis of essential hypertension **(Williams , et al.,1985**).There is epidemiological and pathophysiological evidence that OSA is an independent risk factor for essential hypertension **(Fletcher ,2000)**.

Patients with OSA are also at increased risk of developing pulmonary hypertension, coronary heart disease, and cerebrovascular accident. Inadequate nocturnal and diurnal blood-pressure control, structural vascular changes, altered homeostatic mechanisms such as thrombogenic factors (increased platelet

aggregability), and adverse metabolic effects all contribute to increased risk of latent cardiovascular complications **(Mary ,et al.,2000).**

Evidence

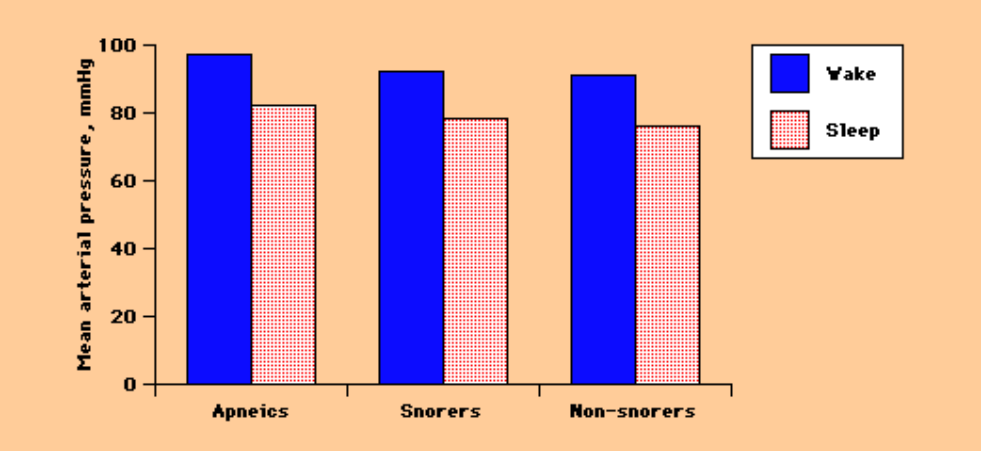
In their large cross-sectional study**, Nieto , et al., (2000)** studied healthy middle-aged and older adults who demonstrated that SDB is associated with prevalent hypertension. After controlling for age, sex, and body-mass index **(BMI)**, adiposity, alcohol intake, and smoking, they found that high apnea-hypopnea indices and oxygen saturations of less than 90% were associated with greater odds of hypertension in a dose-response fashion. **Peppard , et al., (2000)** In contrast, self-reported snoring had little or no association with hypertension. **Nieto , et al., (2000)** found that association between SDB and hypertension was seen in both sexes and in all ethnic groups and was slightly stronger among obese individuals.

The hypothesis that there is a causal association between sleep apnea and hypertension is farther supported by evidence from intervention trials. Successful treatment of sleep apnea using continuous positive airway pressure (CPAP) is accompanied by significant decreases in both daytime and nighttime blood pressures. **(Hedner , et al., 1995)**.

In a case-controlled study, **Davies , et al., (1993)** reported 24 hours of ambulatory blood-pressure measurements in 45 OSA patients and came to the conclusion that, when compared with a closely matched control population, OSA patients have significant elevation of systemic blood pressure, and that this contributes to an increased risk of cardiovascular morbidity and mortality.

Early clinical series of patients with OSA reported a high prevalence of systemic hypertension. However, these early series failed to account for co-morbid conditions that may also be associated with elevated arterial pressure, most notably obesity. The most compelling evidence that OSA can elevate systemic pressure comes from the Wisconsin Sleep Cohort Study, a prospective investigation of apparently healthy state workers without a previous diagnosis of OSA **(Hla, et al., 1994).**

Subjects with an apnea index of five or more events per hour of sleep had significantly higher blood pressures than did subjects with snoring but without apnea or subjects with neither snoring nor apnea. The increase in blood pressure found in the apneic patients was present during both wakefulness and sleep (show figure 11).



Mean arterial pressure is increased in sleep apnea syndrome Mean arterial pressure during wakefulness and sleep in subjects enrolled in Wisconsin Sleep Cohort Study. Subjects with polysomnographically demonstrated sleep apnea had higher blood pressures than either snorers without apnea or non-snoring individuals. (Redrawn from Hla, KM, Young, TB, Bidwell, T, et al. Ann Intern Med 1994; 120:382).

(Figure 11)

The effect of OSA on blood pressure during wakefulness was evident even when the investigators controlled for weight and gender.

These data have been subsequently confirmed by two large population studies. In 1,865 mostly middle aged adults referred to a sleep center for suspected sleep apnea, on no antihypertensive medication, increases in the apnea-hypopnea index (obstructive versus central not specified) were significantly related to increase in awake systolic and diastolic blood pressure, and an apnea hypopnea index of 10 events per hour increased the risk for systemic hypertension being present by 11 percent. Decreases in oxygen saturation of 10 percent during sleep were also linearly associated with increasing risk of systemic hypertention.These associations were independent of other variables such as age and body habitus, although such variables, particularly neck circumference, contributed to the linear regression model **(Lavie , et al ,.2000).**

A second community-based study included 3,670 predominantly middle aged and older participants, in the multicenter Sleep Heart Health Study on no antihypertensive medication. Apnea hypopnea index (obstructive versus central not specified) and percentage of sleep time with oxygen saturation below 90 percent were each linearly related to both systolic and diastolic blood pressure values independent of body mass index, although here too the body habitus measurement contributed to the linear regression model. The risk for awake systemic hypertension was significantly related to increasing severity of sleep apnea in the entire group of 6,132 participants, 40 percent of whom were on antihypertensive medications. For the severest degrees of sleep apnea studied (apnea hypopnea index > 30 in 373

subjects or > 12 percent of sleep time with oxygen saturation below 90 percent in 493 subjects) the mean odds ratio for hypertension adjusted for body mass index, neck circumference, waist to hip ratio, alcohol use, and smoking was 1.37 to 1.45. These data included subjects of both genders and diverse ethnic backgrounds **(Nieto, et al.,2000).**

Despite the evidence provided by this well conducted study, the contribution of OSA to systemic hypertension remains controversial.
A community-based study in Newcastle, Australia, also examined the impact of OSA on blood pressure. In this population, the odds ratio for hypertension was 3.8 when individuals with sleep apnea were compared with non-snoring individuals, but adjustment for age, gender, body mass index, current alcohol consumption, and smoking reduced the odds ratio to 1.5, and the confidence limits included 1.0 (ie, no increase in risk) **(Olson, 1995)**.

However, because these community-based studies do not capture large numbers of individuals with severe OSA, they may not answer the clinician's question about whether may be those investigations that have monitored blood pressure in OSA patients (both hypertensive and normotensive) before and immediately after initiation of therapy for OSA. Such studies have shown that acute therapy, with either tracheostomy or nasal CPAP may contribute to systemic hypertension that is difficult to treat. More pertinent continuous positive airway pressure, results in a significant decrease in systemic pressure **(Mayer , et al., 1991).**

In one report, for example, long-term therapy with nasal CPAP in 12 patients led to a reduction in the apnea index from 58 to 2 and a fall in arterial blood pressure from 147/82 to126/69; these changes occurred without a decrease in body weight **(Mayer ,et al., 1991).**

Furthermore, treatment of OSA may facilitate management of hypertension in patients whose blood pressure is difficult to control hypertension with obstructive sleep apnea. Whilst a causal association has been suspected for some time, the day-to-day variability of both blood pressure and sleep apnea severity, and clustering of confounding cardiovascular risk factors in sleep apnoea patients has made this association difficult to prove. There is unassailable evidence that obstructive apneas raise blood pressure acutely in both animal models and humans, through a combination of autonomic and state dependent arousal with some mechanical influences, and these rises can be controlled by nasal continuous positive airway pressure. Thus, although repetitive apneas alter the blood pressure variability and raise sleeping blood pressure in-patients with OSA and sophisticated animal models have demonstrated increases in daytime blood pressure after the onset of OSA in the short term, such effects on diurnal BP have yet to be proven in humans. Recent

rigorously designed large epidemiological studies have proven an independent association between OSA and systemic hypertension in both general and sleep clinic populations, with closely matched case control series also reporting raised blood pressure in OSA patients. A direct temporal causal association between the onset of obstructive sleep apnea and raised blood pressure is expected to be confirmed by longitudinal data from the continuing epidemiological population studies. **(Justim , et al., 2002).**

Sharabi , et al (2003) found that diastolic blood pressure is higher early in the course of OSAS. We identified 121 subjects newly diagnosed in a sleep study as having OSAS, and 29 matched control subjects in which screening for OSAS was negative. All had a medical interview, physical examination and routine laboratory. **Results**: Subjects who had OSAS had a higher, body mass index (3-kg/m $_{(2)}$ difference) and a higher diastolic blood pressure (4-mm Hg difference) value, without elevation in systolic blood pressure. There was no metabolic difference (lipids profile and fasting glucose levels) between groups.

Peppard, et al (2000) performed a prospective, population-based study of the association between objectively measured sleep-disordered breathing and hypertension (defined as a laboratory-measured blood pressure of at least 140/90 mm Hg or the use of antihypertensive medications). They found good relation between OSAS and hypertension.

Lavie , et al (2000) recently reported findings in 2677 adults who underwent polysomnographic sleep studies. Multiple regression analysis of blood pressure in subjects not on medication showed that the apnea index predicted both systolic and diastolic blood pressure following adjustment for age, body mass index, and sex. Indeed, multiple logistic regressions showed that, for every one event/h apnea index, the odds ratio of hypertension increased by 1%. Furthermore, episodic hypoxia as an aetiological mechanism in the apnea-hypertension relationship gained further support since a 10% decrease in apnea related saturation increased the odds of hypertension by 13%.

In the current issue of ***Thorax***, **Davies , et al (2000**) have carefully matched 45 patients with symptomatic apnea with 45 non-apnoeic controls taken from a primary care setting with regard to age, body mass index, alcohol and cigarette consumption, hypertension treatment, and the presence of ischaemic heart disease. They examined their 24 hours ambulatory blood pressure and found that daytime and night-time diastolic as well as night- time systolic blood pressure was significantly higher in patients with obstructive sleep apnea than in the controls. Body fat distribution was not different between the groups, minimising the possibility that upper versus central body fat distribution played a role in determining differences in blood pressure.

Results: After adjusting for age and body mass index, the mean nocturnal systolic and diastolic BP changes after CPAP treatment in the SDB group were significantly different from those in the no-SDB group: -7.8 vs +0.3 mm Hg (p = 0.02), and -5.3 vs -0.7 mm Hg (p = 0.03), respectively. There was a similar, although statistically insignificant, difference in the adjusted mean daytime systolic and diastolic BP changes after CPAP treatment between the two groups (-2.7 vs +0.4 mm Hg and -2.3 vs -1.7 mm Hg, respectively).

Hla , et al (2002) studied the effect of nasal CPAP on hypertension in patients with SDB They found that after three weeks of nasal CPAP treatment of SDB in hypertensive men caused the lowering of nocturnal systolic and diastolic BP values, suggesting that increased nocturnal BP in persons with hypertension was causally related to the apnea and hypopnea events of SDB.

***Also,* Heinrich , et al (2003)** studied the effective of nasal CPAP treatment in patients with moderate to severe OSA leads to a substantial reduction in both day and night arterial blood pressure. The fact that a 50% reduction in the apnea-hypopnea index did not result in a decrease in blood pressure emphasizes the importance of highly effective treatment. The drop in mean blood pressure by 10 mm Hg would be predicted to reduce coronary heart disease event risk by 37% and stroke risk by 56% .

How does OSA raise systemic blood pressure acutely?

During normal non-REM sleep systolic blood pressure is about 5-14% lower than during relaxed wakefulness.

The pressures are not constant but show smooth oscillations with 20-30 second cycles. Blood pressure is higher during REM than non REM sleep but not reach the wake levels.

With the onset of obstructed inspiratory efforts, the recurrent falls in pleural pressure (sometimes as low as -80cm H2O) are reflected to a varying extent as recurrent dips in the systolic blood pressure.

Because the heart is in the chest, pleural pressure are added to and subtracted from the systolic blood pressure (pulsus paradoxus) **(Shepard, 1985).**

As the apnea progresses systolic blood pressure rises and this is thought to be due to the concomitant fall in arterial oxygen saturation (SO_2). The systolic blood pressure rise mirrors the fall in SO_2, as does the accompanying bradycardia. Whether the rise in blood pressure is a direct consequence of the hypoxaemia, or the hypercapnic acidosis, or is due to the increased respiratory effort that these provoke is not clear **(Hanly , 1989).**

At the moment of arousal and termination of an episode of apnea, there is a further rise in systolic blood pressure, probably as a result of the release of the

bradycardia (by stretch receptor activity), the rise in sympathetic output occurring with arousal, and the continuing fall in arterial oxygenation - which, of course, takes a further 10-12 seconds to reserve at the carotid body (and brain) owing to the circulation time. On average systolic blood pressure usually rises about **I** mmHg for every 1% fall in S O_2, and the diastolic pressure rises about **0.5** mmHg **(McGinty et al, .1988).**

The fall in SO_2 and rise in systolic blood pressure are associated with a rise in sympathetic nervous output and catecholamine production. Thus much of the rise in systolic blood pressure is likely to be due to peripheral vasoconstriction, as cardiac output has been shown to fall during the episodes of apnea, a result mainly of the bradycardia, though stroke volume may also fall **(Parish and Shepard , 1990).**

Pathophysiology

The mechanisms underlying the association between SDB and hypertension are not entirely clear. Several pathophysiological mechanisms have been proposed. These include hemodynamic disturbances resulting from intermittent negative intrathoracic pressure during apneic episodes, recurrent episodes of hypoxemia and hypercapnia resulting in abnormal activation of arterial chemoreceptors and increased sympathetic activity, and an increase in sympathetic activity associated with repeated arousals during sleep **(Fletcher ,.2000).**

Excess sympathetic activity is a consistent finding in patients with sleep apnea syndrome and is presumed to contribute to the high incidence of hypertension. Prior studies have shown relationships between the severity of sleep apnea and sympathetic activity or blood pressure **(Hender , et al,.1988).** A linear relationship has been found between compliance with CPAP and sympathetic activity **(Silverberg and Oksenberg ,.1997).**

Urinary catecholamine excretion has been shown to decrease after long-term use of CPAP and following tracheostomy for severe sleep apnea **(Fletcher , .1987).**

Acute apnea is associated with many autonomic responses, including a significant elevation of blood pressure at apnea termination, bradytachyarrhythmias, high sympathetic output, and increased intracranial pressure. Repetitive apneas contribute to a crescendo rise in mean arterial pressure. Sympathetic activity during acute apnea may extend beyond apnea termination **(Morgan ,et al ,1995).**

Chronic treatment of OSA with nasal CPAP or tracheostomy ameliorates or eliminates essential hypertension **(Silverberg and Oksenberg , 1997)**. The acute blood-pressure response to apnea is probably initiated by arousal (which can evoke an acute presser response, even in the absence of apnea) **(Daves , et al., 1993)** episodic

hypoxemia; excessive muscular effort; and intrathoracic blood-volume shifts. Recurrent arousal at the termination of apneas provokes a chronic sympathetic response, leading to sustained hypertension **(Fletcher , 2000).** Acute hypoxia and excessive muscular effort to overcome upper-airway obstruction may induce acute hemodynamic changes. Intrathoracic blood volume shifts associated with negative inspiratory pressure may contribute to elevated blood pressure during apnea. Airway obstruction during sleep can increase blood pressure without arousal (**Fletcher , 2000).**

Episodic Hypoxia and Sympathetic Output

Acute hypoxia contributes to an acute rise in blood pressure during and following apnea. In fact, the level of oxygen-hemoglobin desaturation during acute apnea is directly related to the magnitude of blood pressure change associated with apnea **(Shepard , 1992)** .

Supplemental oxygen provided to subjects with simulated recurrent apneas ameliorates the blood-pressure increase in response to apnea **(Van den Aardweg , et al., 1992**).

Human and experimental studies **(O,Donnell , et al.,1996)** have shown that hypoxia and arousals are major stimulants of acute sympathetic output, which increases vascular resistance via a-adrenergic receptors in peripheral vasculature, as well as cardiac receptors (increased heart rate and cardiac output). These events cause acute blood-pressure elevation associated with apnea. At apnea termination, the sudden release of intrathoracic pressure allows right ventricular filling and increase in cardiac output, accounting for the sharp rise in blood pressure at apnea termination **(O,Donnell ,et al.,1996)** .

Hypoxia also stimulates epinephrine secretion from the adrenal medulla, which can further magnify the peripheral sympathetic response **(Fletcher , 2000).**

Hedner , et al (1988) showed increased motor nerve sympathetic activity during apnea using perineal nerve electrodes in patients with sleep apnea. Respiratory effort against obstruction produced increased sympathetic nerve activity, which was progressive throughout the apnea and peaked at apnea termination. Apnea is a more potent stimulus toward increased sympathetic activity than hypoxia alone. Furthermore, at the same level of hypoxia and hypercarbia, apnea leads to a much stronger acute sympathetic response than hypoxia or hypercarbia alone**-(Sommers , et al., 1989).**

Patients with OSA have been shown to have increased vascular reactivity to a hypoxic challenge (compared with subjects without apnea) whether they have elevated diurnal blood pressure or not (**Hender , et al., 1992**). Resting sympathetic

activity (measured as muscle nerve activity) is higher in apnea patients than in matched controls (**Somers , et al., 1995**).Increased sympathetic activity, vasoconstriction, and alterations in cardiac output are likely mechanisms for elevated blood pressure sustained output are likely mechanisms for elevated blood pressure sustained beyond the apnea period **(Fletcher ,2000).**

Urinary catecholamine levels are elevated in patients with sleep apnea. Urinary norepinephrine and normetanephrie levels are elevated in patients with OSA.Following tracheostomy, catecholamine levels returned to the normal levels **(Fletcher ,2000).**A significant correlation between urinary norepinephrine the respiratory disturbance index (apneas per hour) has been demonstrated **(Dimsdale , et al., 1995)**.

Plasma norepinephrine was shown to be higher in patients than in controls and correlated with the level of muscle nerve sympathetic activity. It may take several years for heightened sympathetic activity in sleep apnea patients to result in a sustained, diurnal blood- pressure increase **(Fletcher , 2000).**

Overactivity of the Sympathetic Nervous System

Overactivity of the sympathetic nervous system may be a fundamental mechanism in hypertension. Plasma norepinephrine in the heart and kidneys is elevated in young individuals with hypertension **(Goldstein , et al., 1983).** Heightened activity of the renal sympathetic nerves contributes to increased renal vascular resistance in essential hypertension **(Fletcher ., 2000**). Increased muscle nerve sympathetic overactivity is seen in mildly hypertensive patients **(Anderson , et al., 1989).** Sympathetic overactivity in the early stages of hypertension is followed by renal vascular disease or vascular remodeling Hypoxia-driven arterial chemoreceptors are potent stimulators of sympathetic activity. Recurrent episodic hypoxia stimulates carotid chemoreceptors and, thus, sympathetic activity .Subsequently, adrenal and renal sympathetic nerves maintain this heightened sympathetic activity. Local endothelial factors may play a role in blood pressure **(Fletcher , 2000).**

Hormonal factors

There is increased release of arterial natriuretic peptide (ANP) during apneas due to increased right atrial filling and hypoxia, facilitating new synthesis and release of ANP **(Krieger , et al.,1989)**. It has a modest vasodilatory action, besides its main effect on blood volume**(Mary ,et al.,2000).** Episodic hypoxia causes a progressive increase in blood pressure mediated, in part, through renal sympathetic nerves acting to increase renin-angiotensin activity through angiotensin-I receptors **(Ehlenz , et al.,1990)**. The activity of the renin-angiotensin-aldosterone system may be suppressed in patients with OSA **(Krieger ,et al.,1990)** . Angiotensin-ll activity

may contribute to vascular hypertrophy and remodeling. Hence, reduced angiotensin II in OSA may be a beneficial effect **(Mary , et al., 2000).** Angiotensin- converting-enzyme inhibitors may be helpful in individuals with hypertension and sleep apnea, but b-adrenergic antagonists have been shown to be more beneficial by some investigators **(Mayer , et al., 1990).**

Endothelium Derived Factors

The vascular endothelium secretes vasodilators and vasoconstrictors, thus modulating vascular tone. Increased endothelin activity is seen in OSA. With nasal CPAP therapy, there is reduced renal excretion of endothelin **(Ehlenz , et al., 1991)**. Hypoxemia increases endothelin gene expression and endothelin release **(Horio , et al., 1991)**. Altered eicosanoid activity has the potential to increase vasoconstrictor tone in OSA. Endothelin-derived nitric oxide is a potent mediator of vasodilatation; thus, it regulates blood pressure **(Moncada , et al., 1993).** Nitric oxide is generated from the amino acid L-arginine by nitric oxide –synthase. Impaired nitric-oxide-dependent vasodilatation in OSA patients has been reported in experimental studies. It is also speculated that reduced vasodilatation in hypertensive OSA patients suggests an attenuated effect of nitric oxide **(Mary , et al.,2000).**

Syndrome Z

The features of syndrome Z include hypertension, central obesity, insulin resistance, hyperlipidemia, and OSA.The factors influencing the relationship between blood pressure and cardiovascular risk include systolic blood pressure, diastolic blood pressure, circadian blood pressure patterns (dippers vs nondippers), blood pressure variability, and cardiac and vascular hypertrophy **(Wilcox , et al.,1998**).

Abnormal vascular endothelial function has been reported in hypertension, diabetes mellitus, and hyperlipidemia. This may precede the onset of cardiovascular disease symptoms by many years.

As discussed above, abnormal endothelial function may be present in patients with sleep apnea and hypertension **(Carlson , et al., 1996).** Whether sleep apnea affects endothelial function independently of hypertension and insulin resistance requires farther research.

OSA is closely linked to the cluster of cardiovascular risk factors known as syndrome X (a cluster of risk factors including systemic hypertension, insulin resistance, hyperlipidemia, and central obesity) and the converse is also likely but has not yet been proven (syndrome Z). These relationships should help physicians consider that patients with sleep apnea may have coexistent modifiable cardiovascular risk factors and, conversely, that sleep apnea should be suspected in patients with hypertension, central obesity, insulin resistant diabetes, or dyslipidemia.

There are specific effects of untreated sleep apnea that increase the cardiovascular consequences as discussed above. Hence, syndrome X may include sleep apnea and could better be defined as syndrome Z. defined as syndrome Z **(Wilcox , et al., 1998**).

The effective treatment of sleep apnea eliminates recurrent episodes of hypoxemia, reduces blood pressure and variability, and may reduce insulin resistance and, thus, triglycerides. **(Brooks , et al., 1994)** demonstrated an improvement in insulin sensitivity in most patients with type II diabetes mellitus and sleep apnea treated with nasal CPAP.

What is the evidence that an important proportion of patients with essential hypertension have appreciable sleep apnea?

Many studies have investigated whether there are occult cases of sleep apnea among patients initially diagnosed as having essential hypertension.

Lavie et al (1993) found that 30-40% of essential hypertension have unexpected sleep apnea and this was advanced as a possible explanation for their hypertension.

Kales et al (1984) compared 50 hypertensive patients and 50 control subjects matched for age and sex but not obesity. Thirty per cent of the hypertensive patients had sleep apnea but on average, it was not severe. The amount of sleep apnea was related to obesity and 40% of the patients were more than 20% overweight.

Fletcher et al (1985) compared 48 men with essential hypertension (most having treatment) with 34 age matched and nearly weight matched control subjects (117% versus 112% over weight). Fourteen hypertensive men (30%) and three control subjects (9%) had more than 10 episodes of apnea an hour, but were symptoms free. The subjects with apnea were heavier than the rest. Treatment for sleep apnea lowered diastolic pressure by 5mmHg 'but there is no untreated control group.

Williams et al (1985) studied 23 hypertensive subjects (all having treatment) and eight age and weight matched control subjects. Three- hour morning nap studies in hospital were used to examine respiratory movements and arterial oxygen saturation alone, after as much sleep deprivation as possible the night before. Thirty five percent of the hypertensive subjects were classed as having sleep apnea and they were much more obese than the non apneic patients.

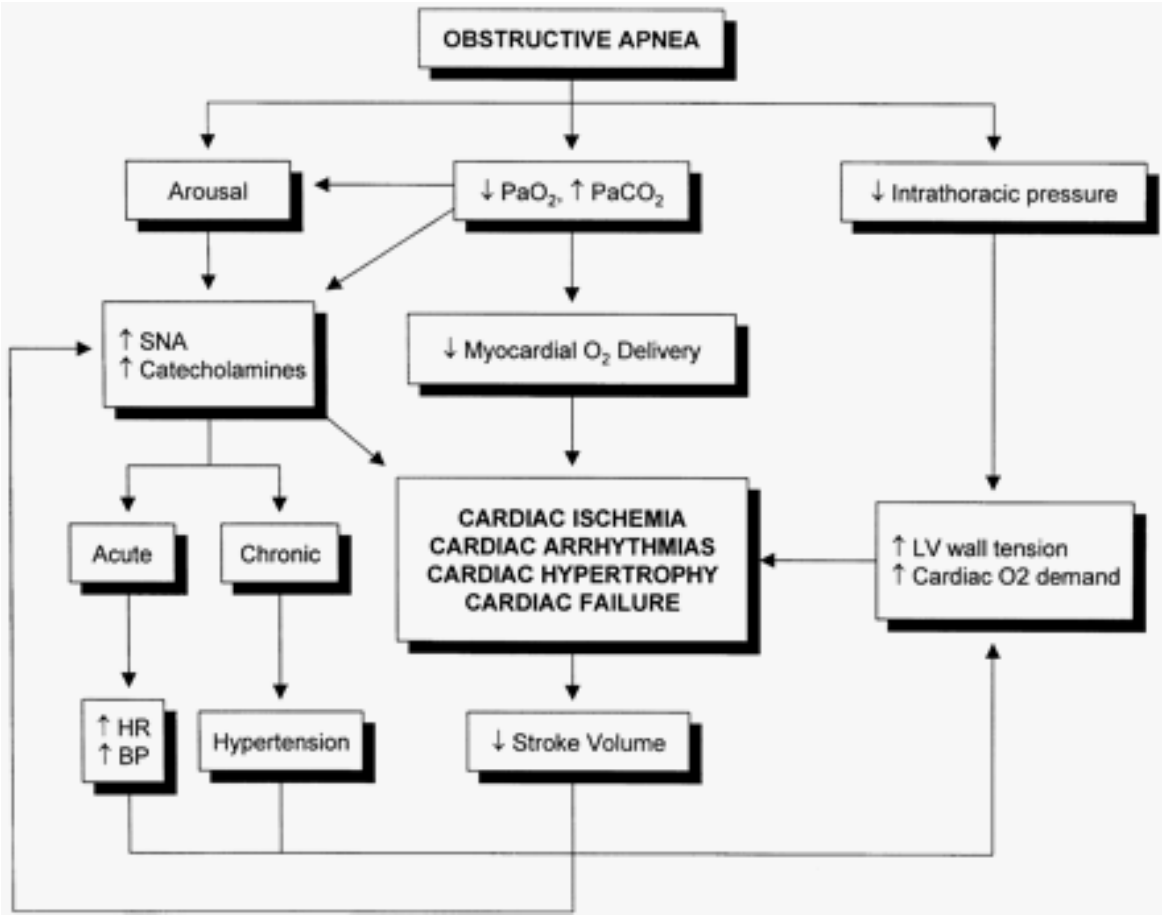
Relationship OSA to Ischemic Heart Disease and myocardial infarction

OSA has been associated with several cardiovascular diseases, most notably hypertension, ischemic heart disease, heart failure, stroke, cardiac arrhythmias, and pulmonary hypertension. With the exception of hypertension, evidence-implicating OSA in these disease conditions is presently circumstantial, and cause-effect relationships remain to be proven **(Robert Wolk , et al.,2003).**

Epidemiology- In a cross-sectional analysis of the Sleep Heart Health Study cohort, OSA was found to be an independent risk factor for coronary artery disease (CAD) **(Shahar , et al .,2001).** However, the association was modest; the odds ratio for CAD of the highest AHI quartile (AHI > 11) was 1.27 times (CI 0.99-1.62) that of the lowest quartile (AHI < 1.4). Prospective follow-up of the cohort should provide additional insights into the relationship between OSA and CAD. Because it has been established that OSA increases the risk for hypertension, it seems likely that it would also increase the risk for disorders, such as CAD, that are associated with hypertension.

Pathophysiology - As described above, and illustrated in (**Figure 12)** OSA exerts several acute physiological effects that could predispose to myocardial ischemia during sleep. In dogs with experimentally induced coronary artery stenosis, obstructive apneas can lead to myocardial ischemia even in the absence of hypoxia **(Chen and Sharf , 1998).** However, in the absence of a coronary stenosis, myocardial ischemia was not observed. Others have reported that electrocardiographic signs of ischemia in patients with OSA with CAD were more closely linked to increases in HR and BP related to apneas than to O_2 desaturation. These observations suggested that the main trigger of ischemia was an increase in O_2 demand rather than a reduction in supply **(Peled , et al., 1999).** In another study, Mueller maneuvers, which simulated the effects of obstructive apneas, caused more pronounced reductions in LV ejection fraction (LVEF) in humans than in those without CAD (**Scharf , et al., 1981)**. These findings emphasize that the diseased myocardium is more susceptible to the adverse effects of obstructive apneas than is the normal myocardium.

Clinical significance- Nocturnal ST-segment changes consistent with myocardial ischemia are quite common among patients with OSA and coexisting CAD. Various studies have reported prevalence's of such ischemic changes ranging from 20 to 100% **(Goldman , et al., 1993).**

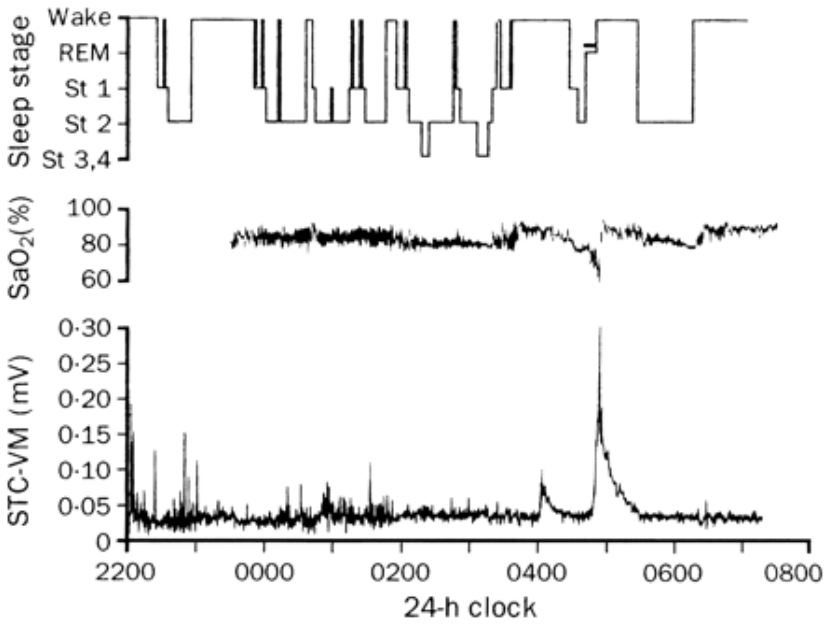


(Figure 12)

Schematic representation of the pathophysiological effects of OSA on cardiovascular system

ST-segment depressions are more frequent in those with more severe OSA or prior complaints of nocturnal angina **(Philip , and Guilleminault , 1993).** Ischemic episodes have been related both to O_2 desaturation and to the postapneic surges in HR and BP and can provoke awakening with complaints of angina **(Franklin , et al., 1995) (Figure 13).**

Myocardial ischemia during sleep in patients with OSA may also be asymptomatic (i.e., silent). However, to date, there are no reports of the prevalence of OSA among patients with CAD with silent nocturnal ischemia.
Studies in patients with OSA without coexisting CAD have been less consistent. In some, no evidence of nocturnal ischemic episodes in such patients was reported **(Richard , et al., 2001),** whereas in another, ST-segment depressions during the night occurred in 30% of patients **(Hanly , et al., 1993).**



(Figure 13)

ST segment depressions, expressed as ST segment change vector magnitude (STC-VM) in a patient with coronary artery disease during recurrent obstructive apneas. Increased STC-VM indicates ST segment depression. The most pronounced fall in oxyhemoglobin saturation (Sa_{O2}) is accompanied by the greatest degree of ST segment depression during which the patient woke up with angina

Acute application of CPAP to these patients significantly reduced the total duration of ST-segment depression. The reason for discrepancies between these studies remains unclear. Accordingly, there is uncertainty as to whether OSA causes nocturnal myocardial ischemia in the absence of CAD. In patients with CAD, OSA may be a poor prognostic indicator. Among 62 patients with CAD followed prospectively for 5 year, **(Peker , et al., 2000)** found that those with OSA had a significantly higher mortality rate (38%) than those without OSA (9%, p = 0.018), even after controlling for potentially confounding factors.

The clinical importance of OSA in ischemic heart disease is twofold. First, epidemiological evidence supports the concept of OSA being etiologically linked to the development of atherosclerosis. There is a high prevalence of OSA in patients with coronary artery disease, and several case-control or prospective studies suggest OSA as an independent predictor of coronary artery disease. Although the exact mechanisms of any atherogenic effects of OSA have not been established, one intriguing possibility is the involvement of inflammatory processes. C-reactive protein (CRP), a biomarker of systemic inflammation and of an increased risk for coronary events, may also play a direct role in atherogenesis. CRP is elevated in OSA a finding that supports the role of inflammation as a mechanism of OSA-

related atherogenesis. Consistent with this hypothesis, elevated plasma levels and cell expression of several adhesion molecules, as well as evidence of increased oxidative stress, have also been noted in OSA **(Shamsuzzaman , et al.,2002)**.

Second, there is evidence that, in patients with or without a history of coronary artery disease, OSA may trigger acute nocturnal cardiac ischemia with ST-segment depression that is often resistant to traditional therapy. Several OSA-related mechanisms, such as oxygen desaturation, high sympathetic activity, increased cardiac oxygen demand (due to tachycardia and increased systemic vascular resistance), and a prothrombotic state, may contribute to the onset of these ischemic episodes. Whether the same mechanisms may also lead to coronary plaque rupture and an acute coronary event, remains to be established (**Robert , et al., 2003)**

Harald , et al (1997) studied 21 patients with OSA, defined by an apnea index of 10 or greater, for six consecutive nights in the sleep laboratory.Classified into two groups, in Group I, 14 patients had both OSA and CHD, the latter proven by coronary angiography that showed the arteries of the heart to be narrowed by at least 50%. In Group II were 7 patients with OSA who had coronary angiography that proved negative. All patients had this test done because of a history of chest pain or abnormal findings in the electrocardiogram during an exercise test. The patients' average age was 56 years (range 47 to 67) and their average weight was 179 lbs (range 158 to 283). Sleep architecture was similar in the two groups, with overall an excess of stages 1 and 2 (67%) and a deficit of stages 3 and 4 (15%), along with 18% REM. Their Apnea Indices averaged 33/hr (SD=+/-15/hr) with a minimum of 11/hr and a maximum of 80/hr. Minimum blood oxygen saturation averaged 73, ranging from 45 to 89.Two days before the six nights of sleep testing; all patients were taken off medication for chest pain, calcium channel blockers (generally used for hypertension) and beta blockers (for hypertension and slowing heart rate). During the first two nights, they received no medication. During the remaining four nights, they received two consecutive nights of a slow-release form of nitrate, 100 mg of isorbide dinitrate, related to nitroglycerine, for suppression of myocardial ischemia. The other two nights they received a placebo (an inactive substance that looks like the active drug. Six patients had episodes of myocardial ischemia during the sleep studies. Five of these six patients came from Group I; the sixth came from Group II. These six patients had a total of 144 episodes of myocardial ischemia during three nights (the second night under each of the conditions: no meds, nitrate, and placebo). This means an average of 8 episodes of myocardial ischemia per patient per night, but two patients had very few episodes (4 altogether in one, only one in another), whereas others had many more, up to 59 (about 20 per night). The episodes lasted from 10 seconds to 96 seconds. They occurred mainly (85%) during periods of high apnea activity and oxygen desaturation. Nitrate had no effect.

The authors mentioned a prior study which found similar signs of myocardial ischemia in 7 of 23 (30%) of patients with OSA ***not*** previously believed to have

coronary heart disease. The supply of blood oxygen to the heart is decreased by hypoxemia; this may be one cause of myocardial ischemia. However, obstructive sleep apneas also increase the demand for oxygen to the heart because of the reduction of pressure within the chest as the obstructed patient attempts to breathe. During REM sleep, when ischemic and apneic events were most frequent, there is an increase in heart rate with increased demand for blood to the heart, related to the autonomic activation typical of REM sleep **(Harald, et al, .1997)**.

The authors also remarked that the patients said to be free of coronary heart disease were not devoid of some signs of cardiac problems, as with exercise testing. On the other hand, medical lore is filled with stories of patients who go to the doctor for a routine physical with electrocardiogram, are declared in the best of health, and drop dead the next day! We sleep apneics, it seems, and have special reason to fear the night. **(Harald , et al.,1997)**

One study reported that male snorers had an elevated odds ratio for angina pectoris (OR = 2.0, $p < 0.01$), even after adjustment for age, body mass index, and the presence of hypertension Female snorers did not demonstrate this increased risk. **(Koskenvuo , et al.,1985).**

Another study reported an increased odds ratio (OR = 1.8) for myocardial infarction in snorers, but this increase in risk was not statistically significant **(Schmidt- Nowara , et al.,1990).**One study comparing community-dwelling individuals who had sleep apnea with non-snoring individuals reported a crude odds ratio of 3.5 for coronary heart disease and 3.7 for occlusive vascular disease . However, these ratios fell to 1.4 (coronary disease) and 1.5(occlusive vascular disease) after adjustment for age, sex, body mass index, smoking and current alcohol consumption. Confidence intervals for these odds ratios included 1.0 after adjustment (ie, they were not statistically significant) **(Olson , et al., 1995)**. A single case control study supports the relationship between OSA and vascular morbidity. This study examined the occurrence of OSA in a group of individuals hospitalized for myocardial infarction and compared them to a group of community dwelling control subjects**(Hung, et al.,1990**).

The highest quartile for apnea index was found to have an odds ratio of 23 for myocardial infarction, even after adjustment for coronary disease risk factor**.** This study has been criticized; however, for the potential bi-directional nature of the relationship of myocardial infarction to OSA, since myocardial infarction, particularly if complicated by congestive heart failure, may contribute to the development of sleep apnea, presumably by increasing the tendency to periodic breathing. There was also a potential for bias in the selection of control individuals **(Hung , et al., 1990).**

Obstructive sleep apnoea (OSA) is associated with a range of cardiovascular sequelae and increased cardiovascular mortality **(Shaefer , et al., 1999**). Potentially fatal systemic illnesses frequently associated with this disorder include systemic hypertension, pulmonary hypertension, heart failure, nocturnal cardiac dysrhythmias, myocardial infarction and ischaemic stroke **(Friedlander , et al., 2000)**. A population-based study of 441 middle-aged subjects has indicated that patients with irregular breathing at night have a 3.5 fold risk of coronary artery disease. In yet another study, coronary artery disease patients were found to have OSA in 30.5%, whereas OSA was found in 19.7% among control subjects **(Shaefer , et al., 1999**). **(Talib , et al., 2001)** reported a case of fully involved anteroseptal myocardial infarction who turned out to be a patient of OSA on evaluation.

OSA and pulmonary artery hypertension

As clinical prescriptions of OSA were disseminated, an association between OSA and cor pulmonale became widely accepted and was fostered by the obvious exposure of these patients to nocturnal hypoxia. Thus, it seemed likely that nocturnal hypoxia caused hypoxic pulmonary vasoconstriction, eventually leading to sustained pulmonary hypertension. However, the causal connection of OSA to right heart failure was questioned by Bradley and colleagues, who reviewed their experience and found that all patients with OSA who had clinical features of cor pulmonale also had daytime (presumably awake) hypoxia **(Bradly , et al., 1985)**.

They suggested that OSA alone was inadequate to cause pressure overload of the right ventricle, and they proposed that coexisting chronic obstructive lung disease was necessary. **(Bradly , et al., 1985.)**

Subsequent studies have examined the relationship between OSA and pulmonary hypertension. They have confirmed that daytime hypoxia is required in addition to (or instead of) OSA to cause sustained elevation of pulmonary arterial pressure. This daytime hypoxia may be a consequence of obstructive lung disease or, in some patients, may be caused at least in part by obesity.

An Australian study attempted to isolate the effect of OSA on pulmonary artery pressure by investigating only patients without coexisting lung disease **(Sajkv , et al., 1994).**

Pulmonary pressures were determined noninvasively. In this report, 11 of 27 patients (41 percent) had pulmonary hypertension; however, the magnitude of the effect was quite small (mean pulmonary arterial pressure < 26 mmHg in all patients, making it unlikely that OSA is a common occult cause of pulmonary hypertension.

Thus, OSA should not be sought as a cause of sustained, severe pulmonary hypertension in the absence of combined conditions producing daytime hypoxemia **(Sajkv , et al., 1994).**

Laks (1995) found in his research the prevalence of pulmonary hypertension in populations of patients with OSA was documented to be around 40% .

Zerah , et al (1997) evaluated pulmonary function abnormalities associated with the sleep apnea syndrome (SAS) in 170 habitual snorers without SAS (n = 62, apnea-hypopne index [AHI] < 10 per hour of sleep), with moderately severe SAS (n = 56, AHI < 30) or with severe SAS (n = 52, AHI > 30). The three groups were similar regarding obesity (BMI ~ 30 kg) and smoking history (~ 20 pack-years). Pulmonary function was assessed by spirometry, forced oscillation mechanics, and gas exchange studies. Forced expiratory flows decreased as the SAS severity increased ($p < 0.001$, $p < 0.02$, and $p < 0.05$ for FEF50, FEV1 and FEV1/VC, respectively).

Multiple regression analysis showed that the correlation between FE**V**, and the AHI persisted when smoking history was taken into account ($p < 0.05$), suggesting that SAS may be an independent risk factor for small airway disease. A highly significant correlation was found between specific respiratory conductance (sGrs) and the AHI ($p < 0.0001$). In a multiple regression analysis ($p < 0.0001$), variables that influenced sGrs were distal airway obstruction as assessed by FEV50, ($p < 0.05$), morphological upper airway abnormalities as assessed by cephalometric parameters ($p < 0.02$), and the AHI ($p< 0.0005$). SAS appears to be highly correlated to lower and upper airway obstruction, as demonstrated by a reduction in specific respiratory conductance, which adds to the increase in breathing load due to obesity **(Zerah et al., 1997)**.

Kang , et al (1997) investigated the effect of state-specific changes associated with REM sleep on pulmonary artery pressure in patients with obstructive sleep apnea (OSAS). Six male patients with OSAS (age; 40 +/- 12 SD yrs, BMI; 39.0 +/- 8.6 kg/m2, AHI; 51.5 +/- 28.5) were examined throughout the night by polysomnography, while monitoring pulmonary artery pressure via right cardiac catheterization. All patients had pulmonary hypertension (PH) during periods of wakefulness, and their mean pulmonary artery pressure (PAPm) was 31.1 +/- 7.4 mmHg.

Sajkov , et al (1999) measured awake pulmonary hemodynamics, pulmonary gas exchange, and small airways function in 32 patients with OSA (apnea-hypopnea index, mean, 46.2 +/- 3. 9/h) who had normal screening lung function. C and cardiac output were measured by Doppler echocardiography at three levels of inspired oxygen (FIO2 0.50, 0.21, and 0.11) and during incremental increases in pulmonary blood flow (10, 20, and 30 microgram/kg/min dobutamine infusions) while breathing 50% oxygen. Eleven patients had PH (mean Ppa >/= 20 mm Hg.

Sajkov , et al (2003) measured pulmonary hemodynamics (Doppler echocardiography) in 20 patients with OSA (apnea-hypopnea index [AHI] 48.6 +/- 5.2/h) before and after 1 and 4 months of CPAP treatment (compliance 4.7 +/- 0.5 h/night). Patients had normal lung function, and no cardiac disease or systemic hypertension. Doppler studies were performed at three levels of inspired oxygen concentration (11%, 21%, and 50%) and during incremental increases in pulmonary blood flow (10, 20, and 30 microg/kg/min dobutamine infusions). Treatment resulted in a decrease in pulmonary artery pressure (Ppa, 16.8 +/- 1.2 mm Hg before CPAP versus 13.9 +/- 0.6 mm Hg after 4 mo CPAP, $p < 0.05$) and total pulmonary vascular resistance (231.1 +/- 19.6 versus 186.4 +/- 12.3 dyn. s. cm(-)(5), $p < 0.05$).

Relation of OSAS to cardiac arrhythmias

The most prominent and significant rhythm disturbances associated with OSA include extreme bradycardia and ventricular asystole lasting longer than 10 seconds **(Miller , et al., 1982).**

Smolensky , et al (1972) have presented data that a circadian rhythm exists in human mortality and the highest death rates occur between 4:00 and 6:00 AM, when REM sleep is particularly prevalent. The results of this large scale epidemiological study have stimulated considerable interest in the relationship between disordered breathing, hypoxaemia and cardiac arrhythmia's as a physiological mechanism contributing to nocturnal mortality.

Guilleimnault , et al (1984) have reported serious bradyarrhythmias in four apparently healthy 27 to 35 years old subjects. In these individuals, 42 episodes of sinus arrest of 2 to 9 seconds duration were observed, and all occurred during REM sleep.

Zwillich, et al (1982) demonstrated a strong positive correlation between the level of arterial oxygen desaturation during an apnoeic event and the degree of bradycardia. It is axiomatic that longer apnoeic episodes will produce a greater degree of oxygen desaturation, and according to this study, a predictable decline in heart rate. In fact, they state that virtually every apnea is associated with some degree of bradycardia. The mechanism for this appears to be enhanced vagal tone, probably due to a decrease in reflex respiratory inhibition of vagal tone. Ventricular ectopy is not so manifestly predictable.

Shepard , et al (1985) have provided excellent data which substantially enhance the understanding of this complex relationship. In their study, sleep-associated decreases in the electromyographic activity of the pharyngeal dilators allow the subatmospheric pressures generated during inspiration to collapse the upper airway. During these episodes of upper airway occlusion, arterial oxyhaemoglobin saturation (SO_2) decreases in association with concomitant elevations in systemic and pulmonary blood pressures. Throughout the apnoeic period, heart rate slows in proportion to the duration of apnea and the degree of oxyhaemoglobin desaturation. Increased vagal efferent activity plays a significant role in mediating these reductions in heart rate, as atropine usually ameliorates the apnea-related bradycardia. The resumption of ventilation is associated with rapid cardio-acceleration, which is considered to result from a decrease in vagal tone, probably combined with hypoxia mediated increases in sympathetic neural activity.

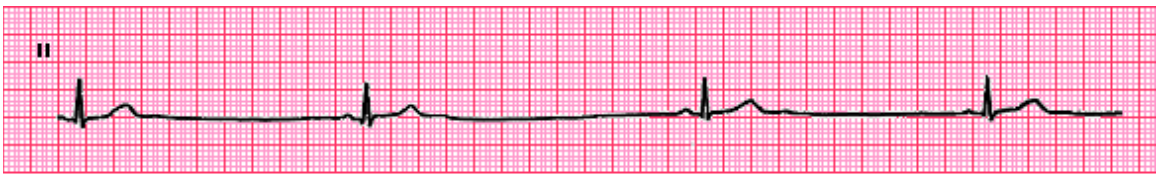
	Miler (1982)	Guilleminault et al. (1983)	Shepard et al. (1985)
Sinus bradycardia (less than 30 beat/min.) (Patients No.)	23	400	31
Sinus pauses (2 to 13 sec.) (%)	9	7	10
Second degree AV block (%)	4	8	6
Ventricular ectopy (%)	57	-	74
Any complex (%)	9	-	55
Ventricular Tachycardia (%)	-	3	3

It summarizes the data on the prevalence of the more serious arrhythmias in-patients with sleep apnea **(Shepard, 1985)**

(Table 1)

This repetitive sequence of events is responsible for the prominent sinus arrhythmia, which is frequently observed during sleep in these patients.

Marked sinus bradycardia (heart rate less than 40 beats per minute) and sinus pauses lasting from 2 to 17 seconds have been reported by different investigators to occur in as few as 9 % to as many as 30% of the patients with OSA .While severe bradyarrhythmias are a potential mechanism of sudden death during sleep, repetitive ventricular ectopy degeneration to ventricular fibrillation is considered to be the more common dysrhythmias leading to sudden death **(William, .1986).**



Sinus bradycardia Marked sinus bradycardia at a rate of 25 to 30 beats/min. The normal P waves (upright in lead II) and PR interval and consistent with a sinus mechanism with normal atrioventricular (AV) conduction. Courtesy of Ary Goldberger, MD.

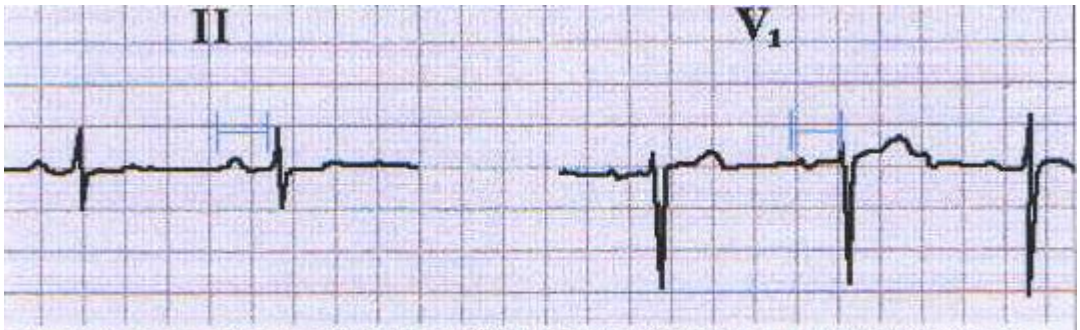
(Figure 14)

Although the contribution of asystole to the increase in mortality and sudden death described in OSA remains unclear, cases of aborted sudden death due to prolonged a systole have been described in patients without OSA **(Milstein , et al.,1989).**

When present, bradycardia and asystole in patients with OSA appear to result from enhanced vagal tone, not structural disease of the conduction system. As an example, an electrophysiologic study in-patient with prolonged asystole occurring exclusively during OSA found that most patients were normal; some had mild sinus and atrioventricular nodal abnormalities that were reversed with atropine(**Grimm , et al.,1996**).

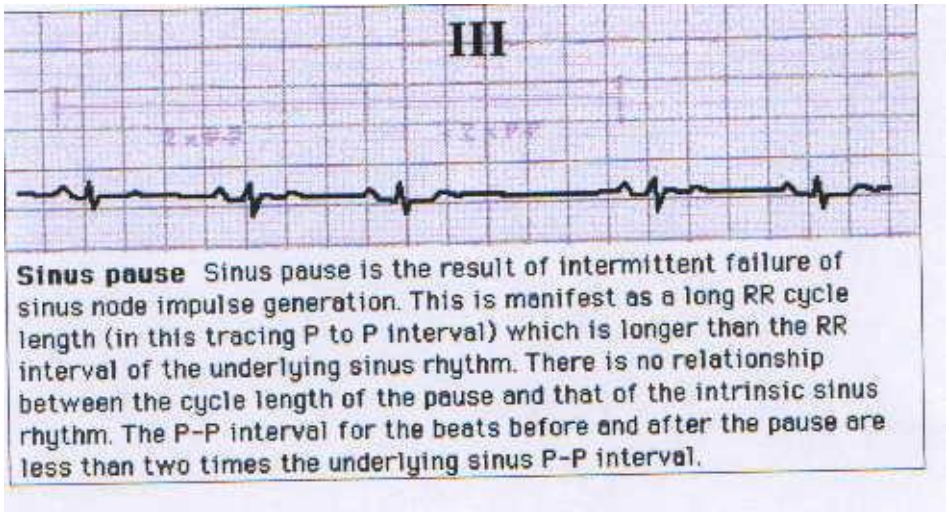
Activation of the parasympathetic nervous system in this setting can result from multiple physiologic abnormalities including hypoventilation, hypoxemia, respiratory acidosis and vigorous inspiratory effort against a closed airway (Mueller's maneuver). Therapy with nasal continuous positive airway pressure (CPAP) abolishes all episodes of ventricular asystole in most such patients (**Grimm , et al., 1996**.).One study of 10 patients with sleep apnea induced asystole, for example, found that effective nasal CPAP restored normal cardiac rhythm in night **(Becker , et al., 1993).**

The most frequent arrhythmias reported in association with OSA are sinus arrest, sinoatrial block, or atrioventricular block, all of which may lead to ventricular asystole. The mechanism of these bradyarrhythmias is usually a reflex increase in vagal tone triggered by a combination of apnea and hypoxemia (diving reflex) (**Robert , et al., 2003)**.



First degree atrioventricular (AV) block First degree AV block is caused by a prolongation or delay in impulse conduction through the AV node. It is defined as a PR interval >0.20 seconds. In this case the PR interval (blue lines) is approximately 0.22 seconds.

(Figure 15)



(Figure 16)

In addition to bradyarrhythmias**,** **Loun, et al (1976)** have reported increased ventricular ectopy during REM sleep in a 39-year-old man without organic heart disease.

The relationship between premature ventricular contractions **(PVC)** frequency and the severity of oxygen desaturation has been carefully examined, and no significant relationship between ventricular ectopic activity and oxyhaemoglobin saturation was detected provided SaO_2 remained above 60 per cent. However, patients desaturation below 60 per cent showed a significant two-fold to three-fold increase in PVC frequency as well as multiple episodes of ventricular bigeminy when SaO_2 was less than 60 per cent compared to when it was greater than 60 per cent **(Shepard, et al., 1985).**

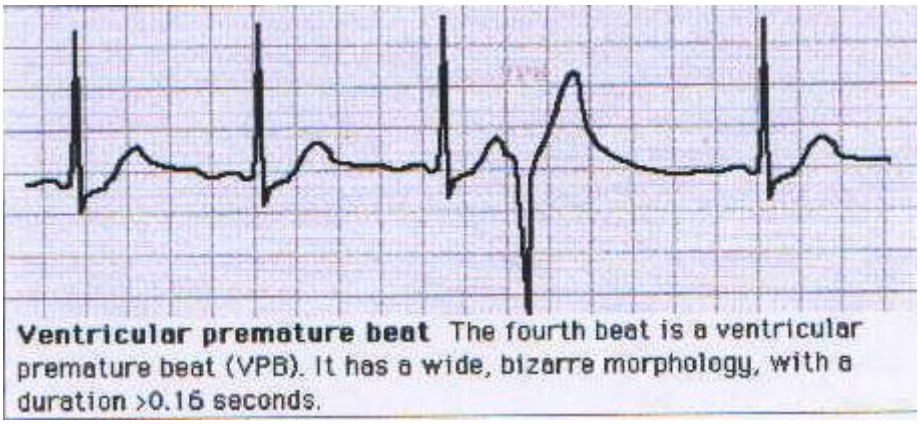
Consequently, patients who have desaturation below 60 percent appear to be at increased risk for ventricular arrhythmias, and a more aggressive approach to therapy is probably indicated. Individual patients who develop serious hypoxaemia associated arrhythmias at higher values of SaO_2 showed also have their sleep apnea effectively treated. While ventricular ectopy unrelated to oxygen desaturation can obviously persist following effective therapy for sleep apnea **(Guilleminault et Al., 1983).**

To even the casual observer, there is some relationship between hypoxia and cardiac dysfunction. However, the specific parameters of this relationship appear to be anything but simple and pellucid. Patients with OSA exhibit profound hypoxaemia during apnoeic episodes, as well as a wide variety of ventricular and supraventricular arrhythmia's **(Koskenvuo et al., 1987).**

Obstructive sleep apnea has been associated with various cardiac arrhythmias, however. This case report describes a patient who developed episodes of

supraventricular tachycardia during periods of apnea and oxygen desaturation. With the initiation of nasal continuous positive airway pressure during sleep, the arrhythmia was abolished. The etiology and possible mechanisms responsible for the supraventricular tachycardia are discussed
(Randazzo ,et al.,1996).

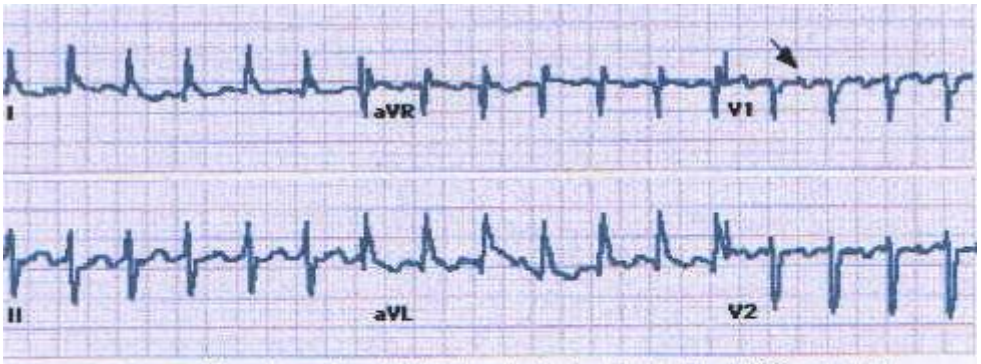
Koehler , et al (1993) found that the Patients with sleep apnea and nocturnal brady- and tachyarrhythmia are considered to be patients at especially high risk within the group of all apnea patients, the study include 13 patients with sleep apnea (apnea-index > 10 events/h).In a study suspected coronary heart disease and known increased frequency of nocturnal premature ventricular contractions (PVC) were studied. Polysomnography, long-term ECG and six-lead ECG were performed. RESULTS: Within the studied period (1.00 to 6.00 o'clock), an average of 47 PVC per hour was recorded (range 4 to 337/h). In two patients 24 episodes of nocturnal myocardial ischemia were observed, but were not accompanied by PVC. Interestingly only 387 of 1371 premature ventricular contractions (28.2%) were associated to apnea/hyperventilation episodes. Arrhythmia occurred mainly during sleep stages I/II and REM (n.s.). Patients with coronary heart disease, obstructive sleep apnea and severe hypoxemia are at higher risk of developing nocturnal PVC because reduced hypoxic tolerance of the heart may lead to electrical instability.**(Koehler ,et al.,1993)**.



(Figure 17)

Both bradycardia and a trend to tachycardia have been reported in obstructive sleep apneas (OSA). Because heart rate (HR) behavior may yield information on parasympathetic activity during OSA, we analyzed HR in samples of consecutive apneic cycles in non-rapid eye movement (NREM) sleep, recorded in normotensive patients breathing room air (n = 7) and supplemental O_2 (n = 4). In air, the patients showed different HR trends during apnea, as HR decreased (HR decreased), remained constant (HR=), or increased (HR increased). By multiple

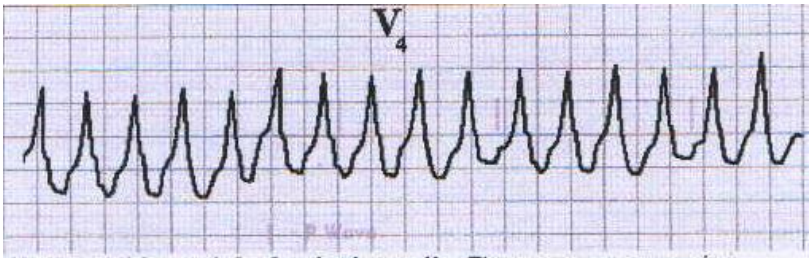
regression analysis, development of HR trends correlated with the HR fall in the late interapneic period, HR at first effort, the decrease in esophageal pressure, and the lengthening of inspiration during apnea (R2 = 0.42). O_2 abolished HR decreased-OSA, whereas HR= and HR increased-OSA still occurred but at higher HR than in air. In both the air and O_2 series, the HR fall preceding apnea correlated significantly with the degree of hypoxia reached in the previous apneic cycle. These data indicate a complex modulation of HR during OSA, with the HR fall in the late interapneic period possibly reflecting the effectiveness of parasympathetic cardiac control in OSA patients during sleep (**Bonsignore ,et al.,1997).**



Sinus tachycardia at a rate of 150 beats/min. Note the difficulty in separating the P waves from the T waves in the standard leads. The P waves are most evident in lead V1 (arrow) where the terminal negativity suggests left atrial enlargement. Courtesy of Morton Arnsdorf, MD.

(Figure 18)

Fichter , et al (2002) show that ventricular arrhythmias occurred significantly more often-in association with disordered breathing in patients at high risk for arrhythmias and reduced LVEF.

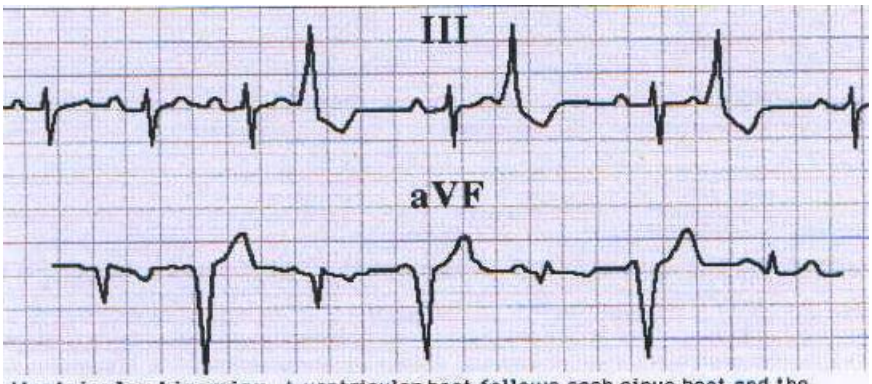


Monomorphic ventricular tachycardia Three or more successive ventricular beats are defined as ventricular tachycardia (VT). This VT is monomorphic since all of the QRS complexes have an identical appearance. Although the P waves are not distinct, they can be seen altering the QRS complex and ST-T waves in an irregular fashion, indicating the absence of a relationship between the P waves and the QRS complexes, i.e. AV dissociation is present.

(Figure 19)

Cutler ,et al (2002) found in the research that the OSA can result in a multitude of systemic manifestations. Structural changes occur in the airway to obstruct airflow during OSA, and the resulting apnea activates hypoxic and hypercapnic reflexes, which in turn lead to profound elevation in sympathetic nerve activity and cyclical changes in parasympathetic nerve activity. These autonomic effects are thought to contribute to the associated cardiovascular diseases (eg, hypertension) and frequently observed brady- and tachyarrhythmias.

Several reports also suggest that OSA may be associated with both supraventricular and ventricular tachyarrhythmias, although the latter are more likely to occur in the setting of other cardiac comorbidities, such as ischemic heart disease or heart failure **(Robert , et al., 2003).**



Ventricular bigeminy A ventricular beat follows each sinus beat and the coupling interval between the ventricular beat and the previous sinus QRS complex is constant (fixed coupling interval).

(Figure 20)

DIAGNOSIS OF OSA

History:

In order to obtain an accurate history, it is extremely important for the physician to recognize the need to obtain information from people other than the patient when dealing with a suspected apneic patient **(Croft and Pringle, 1991**).

In the case of children, the parents, the teachers and even the siblings can be helpful. In the case of adults, the bed partner as well as close associates at work will be helpful **(Moran , J987**).

The details that need to be obtained in the history include inquiry about snoring whether positional or not i.e. does this only occurs when the patient is supine or in any position (**Craft and Pringle, 1991**).

It is important to inquire about nasal obstruction, which may be persistent because of septal deviation or nasal polyps, or fluctuating due to rhinitis, which is often worse at night because of bedroom allergens. It is essential to determine alcohol intake as it is well recognized that alcohol can induce snoring in non-snorers, cause snorers to be obstructed and exacerbates established obstructive sleep apnea **(Milter, et al., 1988).**

Any sedative drugs such as barbiturates, hypnotics, anticonvulsants, narcotics or antihistaminic may have similar effects. Hypothyroidism is an important cause of, or contributing factor to, snoring and OSAS (**Fairbanks, et al., 1987**).

Similarly, a number of disorders of muscular weakness can cause or contribute to OSAS such as poliomyelitis, Guillian-Barre Syndrome, myotonic dystrophy, myasthenia gravis and cerebral palsy. Any co-existent cardiovascular or respiratory disorder needs to be recorded **(Fairbanks et al., 1987).**

The detailed history should also include inquiring about the obstructive episodes, arousal's and nocturnal shocking. Excessive daytime sleepiness, intellectual deterioration, personality changes, abnormal motor events, morning headaches, nocturnal, enuresis and impotence are all important points that should be included in the history **(Fairbanks et al., 1987).**

Finally, one should bear in mind the possibility that snoring is being used as an excuse by the spouse to leave the marital bed such that when the patient is investigated, minimal or no snoring is found **(Croft and Pringle 1997).**

Examination:

During physical examination, the physician may suspects snoring or obstructive sleep apnea on the basis of a collection of observations that are called " Soft Signs "These signs are anatomical features of the nose, pharynx, soft palate, uvula., tongue and body type that are often seen in OSAS but are not necessarily pathognomonic for it.

These anatomical variations might be insignificant if they appeared singly, but when several signs appear together in one patient, they create a composite picture of airway resistance **(Moran, 1987).**

The patient's general appearance will indicate the extent of any obesity and may suggest conditions such as acromegally or myxoedema. Height, weight, neck circumference and blood pressure need to be recorded. Craniofacial morphology has to be assessed looking for retrognathia or micrognathia. Nasal examination will allow assessment, of the nasal airway to reveal any cause of nasal obstruction. Oral cavity examination allows assessment of the size of the tongue, soft palate, uvula and tonsils. Patients with OSA often have a classic picture of an enlarged, swollen edematous uvula and soft palate **(Moran, 1987).**

The nasopharynx is examined to exclude adenoid hypertrophy, polyps, cysts or tumors. 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 reason that may narrow the airway such as enlarged lingual tonsils, cysts or tumors **(Croft and Pringle, 1997).**

The physical findings in OSAS can be summarized as follow:

Nose
Deviated septum
Polyposis
Septal hematoma
Septal dislocation
Rhinitis
Turbinate hypertrophy

Nasopharynx
Carcinoma
Adenoidal hypertrophy
Lymphoma
Stenosis
Pharyngeal flap
Papillomatosis

Mouth and oropharynx
Hypertrophied tonsils
Elongated and/or thickened palate and uvula
Lymphoma of tonsils
Lingual Cyst
Lingual tonsillar hypertrophy
Macroglossia - Acromegally
Micrognathia - Congenital or acquired
Lipoma of the neck
Hunter syndrome
Hurler syndrome
Head and neck burns
Papillomatosis

Larynx
Edema of epiglottis
Vocal cord paralysis
Laryngomalacia
Collapse of aryepiglottic folds

Investigations for OSA

It seems reasonable to assume that surgical and non-surgical therapies could be more widely planned if one could determine the precise location(s) of airway obstruction in each patient, thus allowing treatment to be individualized. Several diagnostic tests are potentially useful in assessing the anatomy and function of the upper airway in OSA, for selecting therapeutic options, and for monitoring responses to therapy, these investigations include:

Polysomnography in the diagnostic evaluation of sleep apnea

Polysomnography, a multi-channel recording of sleep and breathing, has been widely used in the sleep laboratory to diagnose obstructive sleep apnea (OSA) and other causes of sleep disruption, such as periodic limb movements (PLMs). Complete in-laboratory polysomnography traditionally involves monitoring multiple physiological variables with a technologist present throughout the study. These studies are generally performed at night, since abbreviated daytime nap studies are unreliable for the evaluation of sleep architecture and may underestimate the severity of the sleep apnea, especially if rapid eye movement (REM) sleep is not recorded **(Series, et al,.1991)**

However, daytime studies are potentially useful, if not clearly indicated, in shift workers who normally sleep during the daylight hours.

Data collection during polysomnography.

Full polysomnography typically monitors sleep stages, respiratory effort, airflow, oxygen saturation (SaO_2), an electrocardiogram (ECG), body position, and limb movements.

Sleep stages - Sleep stages can be monitored by using two electroencephalograms (EEG), right and left electrooculograms (EOG), and a submental electromyogram (EMG) to detect the hypotonia typically seen in REM sleep. **Rechtschaffen and Kales (1968**) have traditionally scored sleep from these variables using modifications of the rules developed

In addition, arousals from sleep can be determined using the recent criteria set by the American Sleep Disorders Association **(American sleep Disorders Association 1992)**.

Respiratory effort - Respiratory effort can be recorded using a number of different types of devices. Qualitative measures of chest and abdominal movement include strain gauges, piezo electrodes, impedance devices, and a band of EMGs spread across the chest wall and abdomen to detect ventilatory muscle activity. More sensitive and potentially quantitative means of detecting respiratory effort include an esophageal balloon to reflect intrathoracic pressure swings, and carefully calibrated respiratory inductive plethysmography.

Airflow changes. Changes in airflow can be detected using thermistors those sense alterations in heat exchange, an end-tidal CO_2 monitor, or a snoring microphone at the neck. Nasal prongs, connected to a sensitive pressure transducer, may be the most accurate method to assess subtle degrees of inspiratory flow limitation, since they can provide an accurate quantitative measure of inspiratory flow **(Montserrat, et al., 1997).**

Changes in arterial oxygen saturation - Alterations in SaO_2 can be detected using pulse oximetry attached to the finger or the ear.

Other parts of the polysomnogram include: The electrocardiogram is monitored to detect arrhythmias.

It is important to continually monitor body position, since some patients only have apnea and hypopnea in the supine position. Anterior tibialis EMG activity of both legs is monitored to detect periodic limb movements. End-tidal carbon dioxide measurements may also be obtained and are now considered important in the diagnosis of obstructive sleep apnea in children.

Derived information from collected data - From the collected data on the polysomnographic records, one can determine the following parameters, which then can be used to diagnose specific sleep disorders:

Sleep stage distribution - Sleep stage percentages and distribution can be measured, as well as latencies to each of the various stages:

Total sleep time (TST) can be determined by adding the amount of light sleep (stages 2 and I), deep sleep (stages 3 and 4), and REM sleep.

Sleep efficiency can be calculated by dividing total sleep time by total time in bed.

Arousal's - The total number of arousal's, including both three-second transient EEG shifts to a lighter stage and fall awakening can be counted from the record.

An arousal index can be calculated by dividing the total number of arousal's by TST.

Apneas - The number, average length, and type of apneas can also be counted. An apnea has been arbitrarily defined as a total absence of airflow for 10 seconds or longer in adults.

In a central apnea, there is a total absence of respiratory effort. This can typically be determined with absolute certainty only if an esophageal balloon or a carefully calibrated respiratory inductive plethysmograph is used to detect respiratory effort.

In an obstructive apnea, respiratory effort persists but does not result in inspiratory airflow.

In a mixed apnea, there appears to be an absence of airflow initially because of lack of inspiratory effort. Subsequently, however, an obstructive pattern develops, as apnea persists despite resumption of ventilatory effort. Mixed events are generally classified with obstructive apneas, since both involve upper airway collapse.

Hypopneas - The number and average length of hypopneas are also determined. The definition of a hypopnea has not been well standardized in the literature, and may vary depending upon whether one is performing full polysomnography or just cardiopulmonary recording (ie, recording only parameters of breathing and oxygenation, but not sleep or limb movements).

An event is usually counted as an obstructive hypopnea if two of the following criteria are observed on a full polysomnographic recording.

* A decrease in airflow lasting 10 seconds or longer.
* An arousal from sleep.
*Oxygen desaturation of at least two to four percent (depending on the laboratory).

Apnea index - One can calculate an apnea index (Al) by dividing the number of apneas by TST. One can also calculate an apnea plus hypopnea index (AHI) in the same manner by dividing the number of apneas plus hypopneas by TST. The AHI has also been referred to as the respiratory disturbance index (RDI). It is important to recognize that values of RDI may differ depending upon the type of equipment used to record the data.

With full polysomnography, the RDI is the number of events per hour represents the number of events of sleep. With equipment that does not include a measure of sleep, the RDI per hour of the total study.

The AHI or RDI that is used to diagnose sleep apnea remains controversial and varies with the age of the patient and the laboratory.

In young children, the presence of one obstructive apnea per hour may be sufficient to make the diagnosis of obstructive sleep apnea (OSA). Some laboratories use AHI values of five, 10, or even 15 to define significant disease in adults.

Snoring - The amount and intensity of snoring can be determined with a sound monitor. Some centers calculate a snoring index, which represents the number of snores per TST.

Body position - With observation of body position, one can determine whether obstructive apneas, hypopneas, and snoring are dependent upon body position.

Oxygen saturation - The baseline SaO_2 can be determined from the oximetry recording. Other variables that are typically measured include mean oxygen saturation, average oxygen desaturation, and nadirs of oxygen saturation during both non-REM and REM sleep. Studies may also report the number of times there is a fall in oxygen saturation of four percent or more.

Periodic limb movements - Periodic limb movements may be counted both as isolated events and in association with arousal's. As with other variables, one can calculate the number of events per hour of sleep and potentially quantify disease severity.

Validity of polysomnography –

Though full polysomnography is considered the "gold standard" for the diagnosis of sleep disorders, there are no studies assessing the validity of polysomnography for making a diagnosis of OSA in adults. Furthermore, a negative polysomnogram does not conclusively exclude the diagnosis of OSA in adults if there is a high pretest clinical suspicion of the disease. In one report, for example, 11patients who were suspected of having OSA based on obesity, hypertension, or a history of observed apneas during sleep, had a negative result on all-night polysomnography **(Meyer, et al, 1993).**

Six of the 11 had a positive second sleep study, during which an increase in the mean AHI from 3.1 - + l.0 to 19.8 - + 4.7 reflected a change from normal to clearly abnormal values.

The investigators postulated that the degree of nasal patency, body position, or disruptive environmental factors might all be important factors in producing this night-to-night variability.

Polysomnographic diagnosis of upper airway resistance syndrome

The upper airway resistance syndrome (UARS) has recently been described **(Guilleminault, et al., 1993)**

This syndrome involves arousal's from sleep that are associated with flow limitation from increased airway resistance. UARS is frequently, but not always, associated with snoring; it can also result in excessive daytime sleepiness, which resolves with the nocturnal use of nasal continuous positive airway pressure (CPAP).UARS is now felt not to represent a distinct syndrome, but rather subtle sleep apnea.

Although standard polysomnography can detect arousal's associated with increased snoring, diagnosis of the UARS may require an esophageal balloon to identify augmented respiratory effort and the increased airway resistance that accompany the arousal from sleep. Since esophageal balloons are not universally practical in a clinical setting, sleep centers have turned to qualitative respiratory inductive plethysmography or nasal pressure transducers to detect subtle hypopneas.

The multiple sleep latency test.

Full polysomnography may be used in conjunction with a multiple sleep latency test (MSLT) performed on the following day. This test assesses the severity of the patient's sleepiness and helps in the diagnosis of narcolepsy **(American sleep Disorders Association 1992**).

Procedure for multiple sleep latency testing - When the patient awakens after Night time polysomnography, the leads on the extremities and body are removed, but the EEG, EOG, and submental EMG leads are left in place. Two hours later and every two hours thereafter, the patient goes back to bed and is instructed to try to fall asleep; the patient is instructed to get out of bed if sleep has not occurred within 20 minutes.

If the patient does sleep, then the nap is terminated 15 minutes after sleep onset. The patient is generally tested over four to five naps. The mean sleep latency is calculated for all the naps, and the patient is felt to be pathologically sleepy if the mean sleep latency is under five minutes.

Interpretation of data from multiple sleep latency testing.

If REM sleep is seen in two or more naps, then the MSLT may be consistent with a diagnosis **of narcolepsy.**

REM sleep will also appear in two or more naps if:

* The patient is sleep deprived.
* The patient has an abnormal sleep wake cycle, such as that seen with shift work.
* The patient is on REM-suppressing medications.

The patient has been withdrawn from these medications in the last three weeks.

Severe OSA, for example, may cause enough sleep disruption to cause pathological sleepiness and REM sleep in one nap. Thus, clinical correlation is needed to interpret MSLT data.

MSLT is not routinely performed after polysomnography for OSA unless there is a need to document the severity of the sleepiness or the patient has symptoms suggestive of coexistent narcolepsy

Polysomnography and the assessment of treatment modalities –

Besides its utility as a diagnostic tool, full polysomnography has been used to titrate nasal CPAP as well as bilevel pressure therapy for OSA. The patient is usually fully monitored, and the pressure applied via nasal mask, full face mask, or nasal adapters is increased in response to persistent snoring, apneas, hypopneas, desaturation, or arousals. The optimum pressure determined from this type of evaluation is used for the home prescription of nasal CPAP Newly developed self-adjusting CPAP machines that monitor inspiratory flow appear to be as effective as sleep technicians in determining the CPAP prescription for home use **(Lioberes, et al., 1996).**

However, when these auto-titrating machines are used outside the sleep laboratory, their accuracy in generating CPAP prescriptions has yet to be determined. Full polysomnography can also be used to test the longer-term effectiveness of other treatment modalities, such as weight loss, surgery, or oral appliances.

Split night polysomnographic studies - There has recently been a trend to perform split night studies, in which the diagnosis of OSA is established in the first half of the night and the optimal CPAP pressure is determined during the second half. An MSLT should not be performed after a split night study, since the patient will not be in a steady state condition. The patient is generally studied for a baseline period of two to three hours.

If moderate to severe OSA is diagnosed, the remaining four hours of the study are used to titrate the level of CPAP according to the patient's therapeutic requirements.

The first half of the night has been shown to provide a fairly accurate appraisal of disease severity **(Sanders, et al.,1990)**And single split night study can also establish the correct pressure in a majority of patients **(Yamashiro, et al., 1995).**

Patient compliance does not appear to be affected by a half night versus a full night of titration in the laboratory **(Fleupry, et al., 1994)**.

This methodology appears to be a realistic approach to diagnosis and the initiation of therapy. It helps to decrease health care costs and minimize scheduling delays due to limited laboratory resources.

Full polysomnography versus unattended monitoring at home –

The increasing evidence that OSA is a very common condition has made it apparent that it may not be possible to diagnose all current cases of OSA using laboratory polysomnography. This has led to the development of new recording devices for the diagnosis of OSA at home.

Radiographic imaging in the diagnostic evaluation of sleep apnea

Although obstructive sleep apnea (OSA) is a major public health problem, affecting 2 to 4 percent of the middle-aged population, its pathogenesis has not been fully elucidated.

Upper airway imaging is a powerful technique that has significantly advanced our understanding of the pathogenesis of sleep apnea, allowing us to evaluate the structure and function of the upper airway and the surrounding soft tissues. In addition, upper airway imaging has become clinically useful in evaluating patients for upper airway surgery and dental appliances. This part will first review upper airway anatomy as a background for considering the various imaging modalities. It will then consider:

*The advantages and disadvantages of different upper airway imaging modalities.

*Clinical indications for upper airway imaging.

Upper airway anatomy.

The upper airway is a complicated structure that performs several different physiologic functions, including respiration, vocalization, and deglutition. The biomechanical relationships of the more than 24 muscles that allow the upper airway to perform these functions are not well understood **(Fleetham, 1992)**

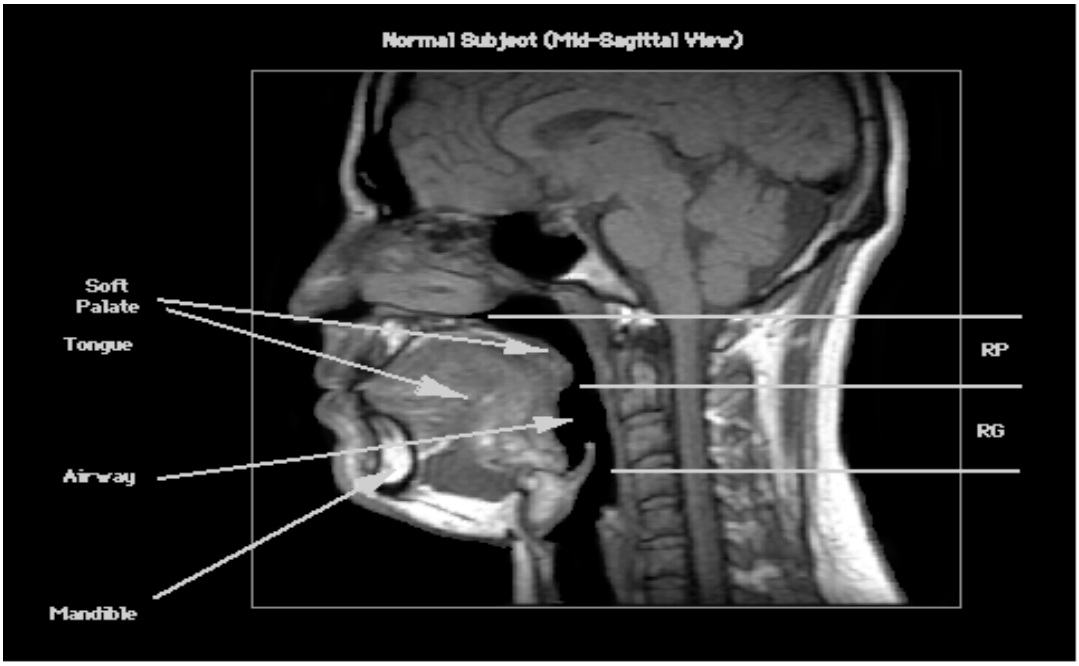
The upper airway can, however, be subdivided anatomically into three regions **(Schwab, et al.,1995):**

*The nasopharynx (the region between the base of the skull above and the soft palate below).

*The oropharynx, which can be subdivided into the retropalatal region (also called the velopharynx) and the retroglossal region.

*The laryngopharynx (the region from the upper border of the epiglottis above and the lower border of the cricoid cartilage below).

The upper airway is smallest in the oropharynx in both normal subjects and patients with OSA, particularly in the retropalatal region (**Galvin, et al.,1989)** Airway closure during sleep occurs in the retropalatal region in the majority of patients with sleep apnea (**Homer, et al,.1989).** The relevant anatomical structures of this region (tongue, lateral pharyngeal walls, lateral parapharyngeal fat pads) can be imaged radiologically (show radiograph IA-IB) (Figure 21).



Normal upper airway Mid-sagittal MR image in a normal subject demonstrating the anatomic regions of the upper airway and relevant soft tissue structures. The retropalatal (RP) region is defined from the level of the hard palate to the distal margin of the soft palate; the retroglossal (RG) region is defined from the distal margin of the soft palate to the base of the epiglottis. Courtesy of Richard J Schwab, MD.

(Radiograph IA-IB).

(Figure 21)

Advantages and disadvantages of different upper airway imaging modalities

The ideal upper airway imaging modality in OSA is one, which is inexpensive, noninvasive, does not involve radiation, and is performed in the supine position. It should also provide:

*High resolution with anatomical representation of the airway and soft tissue structures.

*Imaging during sleep.

*Dynamic imaging, in order to visualize apneic events.

Although such an ideal modality does not exist, MR imaging and nasopharyngoscopy are the best choices among the available options.

Acoustic reflection - Acoustic reflection is a technique that allows measurement of airway caliber on the basis of reflected soundwaves. The advantages of acoustic reflection are that it is noninvasive, has no associated radiation, and can be repeated. However, it is performed in the sitting, not the supine position, and it does not provide high resolution anatomical representation of the airway or soft tissue structures. It has been used primarily as a research tool, and its clinical utility has not been assessed.

Fluoroscopy - Fluoroscopy provides dynamic upper airway imaging during wakefulness and sleep. However, it is associated with significant radiation exposure, and it is not sensitive enough to measure changes in airway size or the detailed motion of structures surrounding the airway.

Cephalometry - Cephalometry is a lateral radiograph of the head and neck. Cephalometry is a widely available, easily performed study that is not as expensive as CT or MR imaging. However, it requires specific, standardized radiographic equipment and techniques as well as interpretative skills.

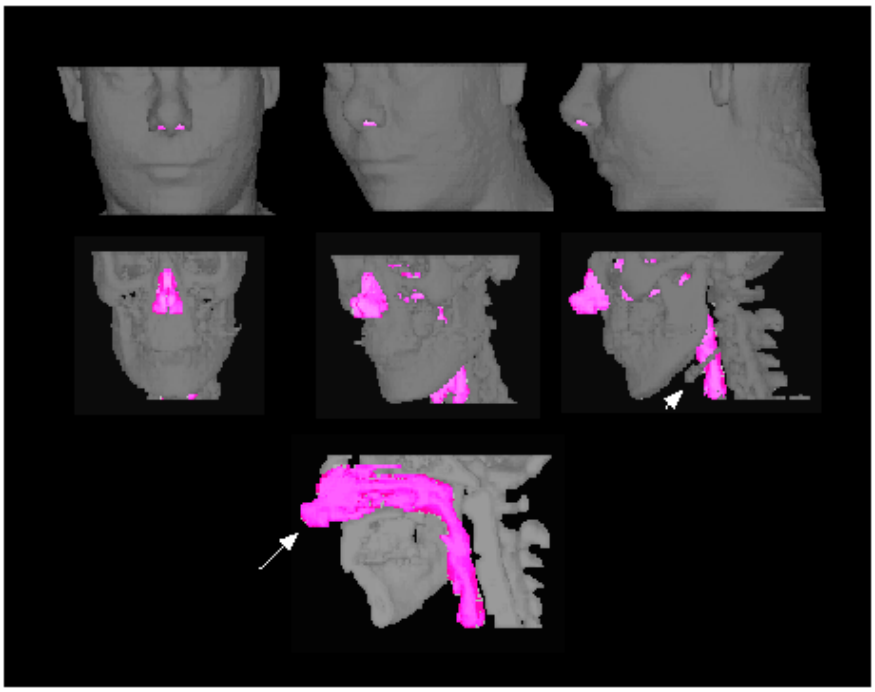
In addition, it is performed only while the patient is sitting or standing, and cannot be performed when the patient is asleep.

Cephalometry provides two-dimensional evaluation of skeletal and soft tissue structures, giving limited information about anterior-posterior structures but no information about lateral soft tissue structures. It is useful in the evaluation of patients with bony facial abnormalities, such as retrognathia, and in the evaluation of dental appliances.

Computed-tomography- CT scanning is a widely available but expensive study that can only be performed when the patient is in the supine position. Radiation exposure limits its usefulness for performing studies during wakefulness and sleep.

CT permits accurate determination of upper airway cross-sectional area and volume, with excellent resolution of the airway and bony structures.

Images are acquired only in the axial plane, but spiral CT provides direct three-dimensional volumetric reconstruction of images, allowing reconstruction of bony structures (cranium, mandible, and hyoid) and the airway **(show radiograph 2)** (Figure 22)**.**



Upper airway anatomy Three dimensional surface, bony and airway reconstruction of contiguous axial CT images in a normal subject. Top row demonstrates surface reconstruction. Middle row demonstrates relationships between bony skeleton and airway. Bottom row demonstrates skeleton airway relationships in the midsagittal plane. Note the relationship between hyoid bone and airway. Arrow: airway; Arrow head: hyoid bone. Courtesy of Richard J Schwab, MD.

(Radiograph 2)

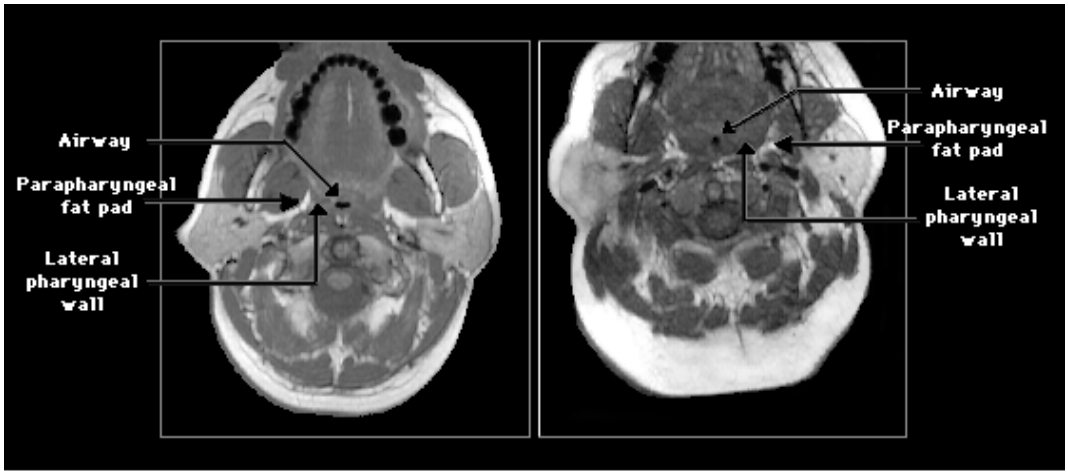
(Figure 22)

CT scanning is also useful in the evaluation of patients with sleep apnea who have bony manipulations (dental appliances and maxillomandibular advancement).

Electron beam or cine CT allows collection of images in 50 msec, and it provides dynamic imaging with excellent temporal and spatial resolution.

Magnetic resonance imaging –

MR imaging is now a readily available but expensive technique that is limited to imaging in the supine position. It provides excellent resolution of the airway and soft tissues, with precise definition of the soft tissue boundaries of the upper airway (show radiograph IA-IB and show radiograph 3A-3B).



Reduced airway size in obstructive sleep apnea Comparison of an axial image at the minimum airway area (retropalatal region) of a normal subject (left) and a patient with sleep apnea (right). Note the smaller airway size and airway width in the patient with sleep apnea. In addition, the thickness of the lateral pharyngeal wall (distance between the airway and parapharyngeal fat pads) is larger in the patient with sleep apnea. Courtesy of Richard J Schwab, MD.

Radiograph 3A-3B

(Figure 23)

Upper airway cross-sectional area and volume can be accurately determined, and because the study involves no radiation, it can be performed and repeated during both wakefulness and sleep.

Some technical advantages provided by MR imaging include:

*Sagittal, coronal, and axial images can be obtained.
*The ability to provide three-dimensional reconstruction of the airway and soft tissue structures, such as the tongue, soft palate, fat pads, and lateral pharyngeal walls.
*Dynamic imaging is possible with echoplanar and ultrafast MR imaging. *The technique facilitates spectroscopic imaging for quantitation of fat and water in soft tissues.

MR is useful in the evaluation of patients with OSA who have undergone surgical procedures altering airway and soft tissue configuration, such an uvulopalatopharyngoplasty or geniohyoid advancement.

Insights from upper airway imaging regarding the pathogenesis of OSA –

Use of upper airway imaging has provided insights about upper airway structure and function during wakefulness, sleep, and dynamically during the respiratory cycle. In addition, it has afforded the opportunity to assess upper airway and soft tissue changes induced by continuous positive airway pressure (CPAP).

Upper airway imaging during wakefulness:

Imaging studies with CT **(Galvin, et al,.1989) , (Ryan, et al,.1991)** and MR**(Suto, et al.,1993)** accurately measure upper airway cross-sectional area and volume as well as the surrounding soft tissue structures. These studies have provided insights about differences in upper airway anatomy between normal and patients with OSA during wakefulness (radiograph show 3A-3B):

*Upper airway caliber is significantly less in-patients with sleep apnea compared with normal subjects. In both normal and patients with OSA, airway narrowing is greatest in the retropalatal region.

*The upper airway in patients with OSA has its major axis oriented in an anterior-posterior dimension (lateral narrowing), whereas the normal upper airway has its major axis oriented in a horizontal dimension.

*Lateral narrowing of the upper airway in OSA is explained by larger lateral pharyngeal walls. Therefore, the lateral pharyngeal walls (not the soft palate, tongue, or parapharyngeal fat pads) are an important anatomic factor causing airway narrowing in this setting.

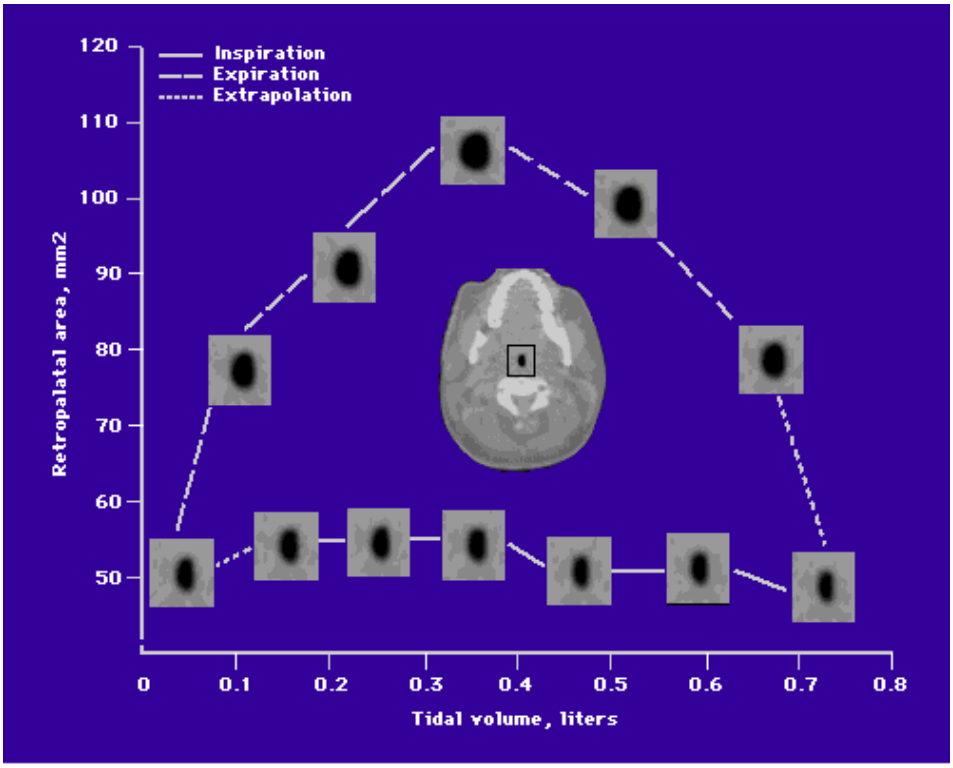
*The total volume of parapharyngeal fat is a greater in-patient with OSA.

*The soft palate and tongue are larger in-patients with OSA.

Dynamic upper airway imaging during respiration –

The upper airway should not be considered a static structure, as studies have demonstrated significant changes in upper airway caliber during the respiratory cycle **(Schwabvs, et al., 1993).**

Imaging studies with electron beam (cine CT) have provided insights about the following respiratory-related changes in upper airway caliber (show radiograph 4 and show figure 24).



Respiratory variation in upper airway caliber in obstructive sleep apnea Axial CT images during respiration in an apneic patient at the retropalatal region demonstrating respiratory variation in upper airway caliber. Airway caliber remains relatively constant during inspiration, enlarges in early expiration and narrows towards end-expiration. Note that the apneic airway has an anterior-posterior configuration. (Solid line = inspiration, dashed = expiration, dotted line = extrapolation between end of inspiration and the beginning of expiration, and between end of expiration and the beginning of inspiration). Courtesy of Richard J Schwab, MD.

(Radiograph 4)

(Figure 24)

*Upper airway caliber remains relatively constant during inspiration in both normal subjects and patients with OSA.

*Upper airway caliber increases in size during early expiration, more in-patients with OSA than in normal.

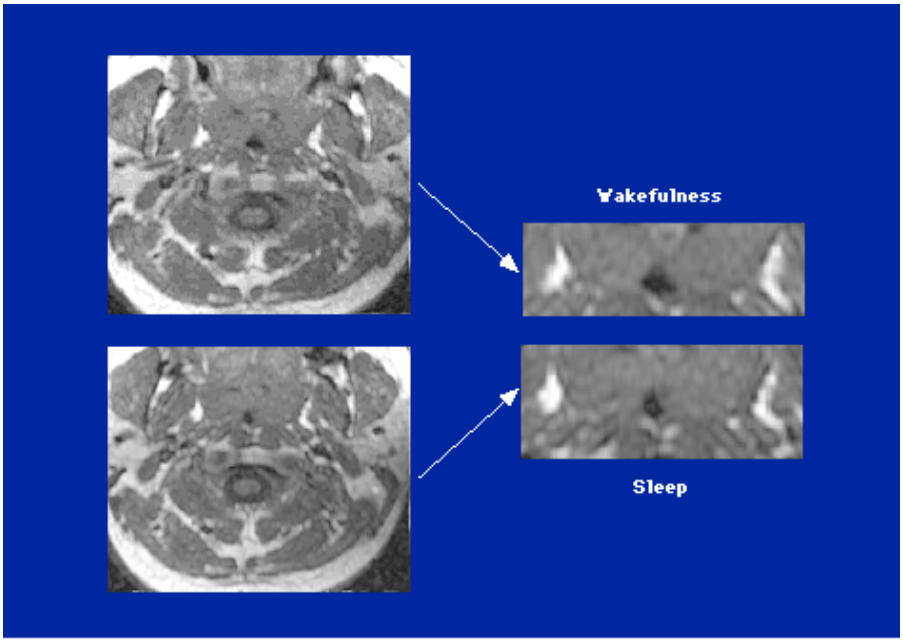
*Upper airway caliber decreases significantly at the end of expiration, indicating that the apneic airway may be headed to a closed position at end-expiration.

* Airway dimensional changes are greater in the lateral than the anterior-posterior direction during respiration, emphasizing the importance of the lateral pharyngeal walls in mediating upper airway changes.

State-dependent upper airway imaging –

Upper airway narrowing during wakefulness does not exactly correlate with the site of obstruction during sleep, although the site of narrowing in both instances is in the retropalatal region **(Homer, et al.,1989) (Suto, et al.1993.)**

As a result, it is useful to study changes in the airway and surrounding soft tissues during both wakefulness and sleep. MR imaging has been used to study these state-dependent changes in the upper airway (show radiograph 5A-5B) (Figure 25) (**Schwab, et al.1995**).



State-related changes in soft tissues in OSA State-related soft tissue changes at the retropalatal region in a patient with obstructive sleep apnea. Thickening of the lateral pharyngeal walls occurs during sleep, reducing airway cross-sectional area. Courtesy of Richard J Schwab, MD.

Radiograph 5A-5B
(Figure 25)

*The airway is narrowest in the retropalatal region during both wakefulness and sleep.

*The minimum airway area decreases by approximately 30 percent during sleep in both normal and patients with OSA.

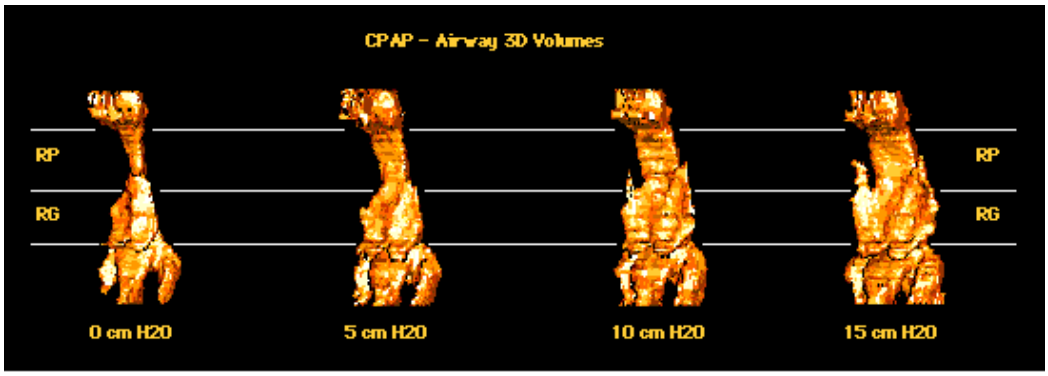
*The lateral pharyngeal walls thicken during sleep as the airway decreases in size. This suggests that the lateral pharyngeal walls may be important in the genesis of airway closure during apnea.

Upper airway and soft tissue changes induced by continuous positive airway pressure –

Nasal continuous positive airway pressure (CPAP) is an effective therapy for patients with OSA.

Imaging studies with CT and MR have provided important information regarding the effects of CPAP on upper airway caliber and the surrounding soft tissue structures **(Collop, et al., 1991)**

- Airway cross-sectional area and volume increase significantly with CPAP. The increase in airway area with CPAP occurs in both the retropalatal and retroglossal regions (show radiograph 6) (Figure 26).
-

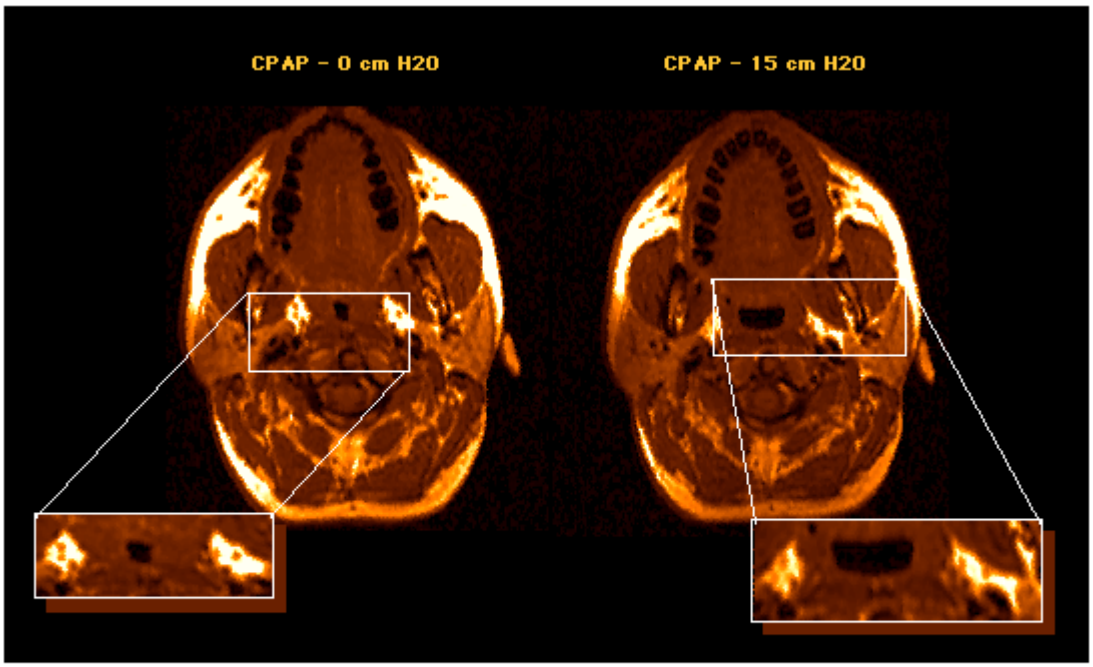


CPAP increases upper airway volume Three dimensional surface renderings of the upper airway demonstrating the effect of incremental levels of CPAP pressure (0 to 15 cmH20) on upper airway volume. CPAP significantly increases airway volume in the retropalatal (RP) and retroglossal (RG) regions in this normal subject. In addition, note the lateral airway widening as CPAP pressure increases. Courtesy of Richard J Schwab, MD.

(Radiograph 6).
(Figure 26)

*Airway dimensional changes with CPAP are greater in the lateral than the anterior-posterior dimension.

*CPAP predominantly affects structures lateral to the airway, i.e., the lateral pharyngeal wall and parapharyngeal fat pads, rather than the tongue and soft palate (show radiograph 7A-7C) (Figure 27).



Lateral enlargement of airway with CPAP Axial MR image at the retropalatal level in a normal subject with 0 cmH20 and 15 cmH20 of CPAP. Lateral airway enlargement is demonstrated with the application of 15 cmH20 of CPAP. The anterior-posterior airway dimension is relatively unchanged with 15 cmH20 of CPAP. Courtesy of Richard J Schwab, MD.

(Radiograph 7A-7C).
(Figure 27)

There is an inverse relationship between pharyngeal wall thickness and the level of CPAP.

Nasopharyngoscopy - Nasopharyngoscopy is widely available, easy to perform, and is not associated with radiation. It can be performed during wakefulness or sleep, and the patient can be in either the sitting or the supine position. A Mueller maneuver performed during the procedure may simulate an obstructive apnea and therefore provide insight into the location of upper airway obstruction. However, nasopharyngoscopy is an invasive procedure, and it evaluates the airway lumen, not the surrounding soft tissue structures

Advantages of nasopharyngoscopy

* It does not involve radiation exposure. Performance of dynamic tests is possible.
* It is useful to evaluate the obstruction at the retropalatal and retroglossal levels.
* It is easily reproducible, being possible to perform the test preoperatively and postoperatively.
* The test may be performed with the patient in sitting or supine position.
* The test may be performed with the patient awake or sleeping.
* The test is widely available and relatively cheap.

Disadvantages of nasopharyngoscopy

* It is an invasive technique. It can produce some discomfort when introducing the nasopharyngoscope into the nose.
* It gives an approximate idea of the pharynx, as it is not possible to make any measurements.
* The evaluation depends on the experience of the examiner.

Conclusions - Several of the findings discussed above have major clinical implications for the treatment of patients with OSA.
The two most important findings appear to be:

- Demonstration of the anatomic significance of the lateral pharyngeal walls in determining airway caliber in normal and patients with OSA.Because of this finding, upper airway surgery may need to involve the lateral pharyngeal walls to be successful.

- Characterization of the changes in upper airway caliber during the respiratory cycle, emphasizing the importance of end-expiratory airway narrowing. As a result, the end of expiration appears to be the key time to apply positive airway pressure to prevent airway closure (**Sanders, et al.,1983).**

Clinical indications for upper airway imaging - Upper airway imaging is primarily a research tool at the present time.It is not indicated in the evaluation of most patients with OSA and is not necessary if a patient with sleep apnea is being successfully treated with CPAP.

Prior to uvulopalatopharyngoplasty - There are some circumstances in which upper airway imaging may have clinical value in the evaluation of patients with sleep apnea. As an example, patients undergoing upper airway surgery (e.g., uvulopalatopharyngoplasty) may benefit from awake upper airway imaging. MR imaging is the preferred modality, since upper airway soft tissue resolution is excellent and there is no radiation exposure.

Uvulopalatopharyngoplasty (UPPP) is the most common surgical procedure for patients with obstructive sleep apnea **(Shepard and Olsen ,1990).**
The success rate is related to the site of obstruction, with patients demonstrating retropalatal obstruction having better results than those with retroglossal obstruction (**Shepard, et al., 1989**).

Thus, MR imaging of the upper airway should be considered prior to UPPP. The MR scans will identify patients with retroglossal collapse, who are not ideal candidates for this form of surgery.

It should be noted, however, that there are at present no data demonstrating an improved outcome in those patients undergoing UPPP who were selected based upon the results of MR imaging. If the UPPP is unsuccessful in treating the patient's sleep apnea, the MR scan should be repeated to elucidate the site of airway narrowing and determine whether further surgery is indicated. Although controversial, it is also reasonable to perform nasopharyngoscopy with a Mueller maneuver prior to UPPP, in order to determine if collapse occurs in the retropalatal or retroglossal regions.

There are no data at present to demonstrate that the Mueller maneuver accurately simulates apneic events during sleep. Nevertheless, surgery directed at advancing the tongue (e.g., geniohyoid advancement or maxillomandibular advancement) should be considered rather than UPPP if MR imaging demonstrates primarily retroglossal narrowing, and nasopharyngoscopy with the Mueller maneuver demonstrates retroglossal collapse.

Prior to maxillomandibular advancement

A CT scan should be considered prior to maxillomandibular advancement. CT scanning is the preferred imaging modality for Evaluating bony structures and the scan can provide three-dimensional image reconstruction of the airway and bony skeleton, which will help in designing the surgical approach (show radiograph 2).

If three-dimensional CT scans are unavailable, cephalometrics should be considered prior to maxillomandibular advancement **(Deberry, et al., 1988).**

Cephalometric studies have certain limitations, since they are two dimensional and provide information only about anterior-posterior structures, not lateral structures. However, cephalometric studies have demonstrated multiple soft tissue and bony abnormality in-patients with OSA **(Deberry, et al., 1988).**

Prior to the application of oral appliances - Cephalometrics provides information about the posterior airway space, retrognathia, micrognathia, hyoid and mandibular position, and tongue and soft palate size. As a result, cephalometric studies have been used prior to the application of oral (dental) appliances for the treatment of OSA.

Three-dimensional CT scans can also be considered before and after use of an oral appliance to evaluate relationships between the airway and bony skeleton.

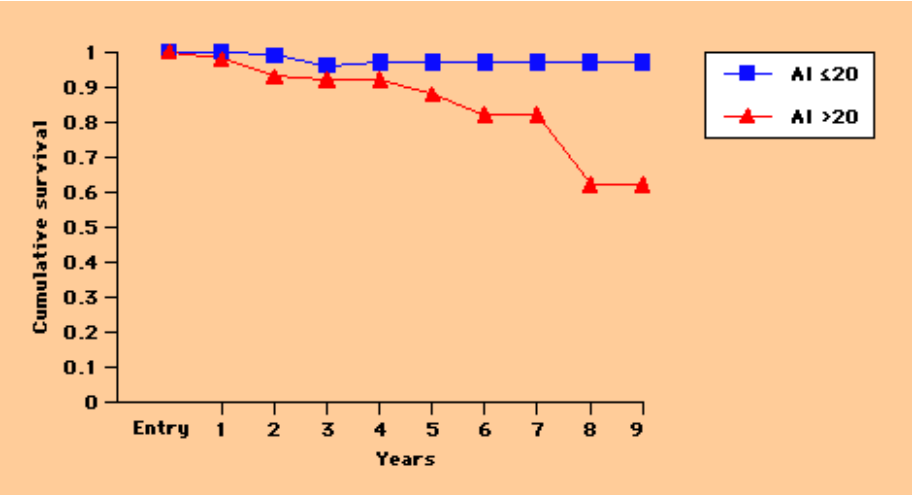
Overview of the management of obstructive sleep apnea

Obstructive sleep apnea (OSA) is a disorder that can be found in many diseases, the most common being obesity.

The optimal approach to therapy requires that the clinician determine the cause of the disorder, establish the severity of the sleep apnea, and then match the patient with the most appropriate treatment **(Guilleminault, et al., 1995).**
There are many compelling reasons to treat sleep apnea patients, including the resultant physiologic improvements and an enhanced quality of life
(Yamashiro and Kryger, 1994).

This part will review the basic principles underlying the management of OSA.

Role of apnea index the apnea index is defined as the average number of apneas/hour of sleep. There is compelling evidence to recommend treatment in all patients with an apnea index exceeding 20. This frequency of apneas appears to be associated with increased mortality. In one study of 3 85 men with OSA, for example, the probability of cumulative eight year survival was 96 percent for patients with an apnea index below 20 compared to only 63 percent for those with an index of greater than 20 (show figure 28) **(He, et al., 1988).**



Effect of apnea index on mortality Probability of cumulative survival for patients with obstructive sleep apnea and an apnea index (AI) that is either equal to or less than 20 (top line, 142 patients) or greater than 20 (bottom line, 104 patients). All the patients were men and were untreated. Survival was significantly higher in the patients with an AI ≤20. (From He, J, Kryger, MH, Zorick, FJ, et al, Chest 1988; 94:9.)

(Figure 28)

Furthermore, research suggests that treatment reduce this increased mortality. There were no deaths in the above study in-patients treated with tracheostomy or nasal continuous positive airway pressure (CPAP) **(He, et al., 1988).**

Given the low mortality associated with an apnea index below 20, we recommend treatment only when there are complications directly attributable to apnea, such as excessive daytime sleepiness or premature cardiovascular disease. This would include patients who have the upper airway resistance syndrome (**Guilleminault, et al,. 1991).**

Children with obstructive sleep apnea may have very different clinical features as well as polysomnographic findings from those seen in adults **(Carroll and Loughlin, 1995).** Many children do not have excessive daytime sleepiness as the major presenting complaint and the apnea index in such children seldom reaches the levels seen in adults.

GENERAL THERAPEUTIC MEASURES - It is useful to review the general therapeutic measures that may be helpful in-patients with OSA before discussing modalities directed at correcting the obstructive events.

Weight control -Obesity appears to play a major role in the vast majority of patients with OSA. As a result, weight control is an important therapeutic focus in obese patients, since it can lead to resolution of the syndrome **(Wittels and Thomson, 1990).**

In one study of 15 hypersomnolent patients with moderately severe OSA, for example, moderate weight loss from 106.2 kg to 96.9 kg was associated with a fall in apnea index from 55 to 29 as well as a decrease in oxyhemoglobin desaturation and an improvement in daytime hypersomnolence [II]. Unfortunately, weight loss is difficult to achieve and maintain further more; apnea may recur in spite of maintained weight loss **(Pillar, et al., 1994).**

Weight gain can exacerbate symptoms even in-patients in whom excess weight was not the primary factor responsible for OSA. One setting in which this occurs is an in-patient with anatomic abnormalities, such as craniofacial malformation.

Alcohol avoidance - Alcohol is known to exacerbate sleep apnea, worsen pre-existing sleepiness, and promote weight gain **(Issa, et al., 1982).** Thus, patients with sleep apnea should abstain from alcohol intake, even during the daytime.

Treatment of coexisting disease - Treatment of coexisting disease, such as hypertension or ischemic heart disease, may have to be modified once more definitive therapy for OSA is instituted **(Rauhala, et al., 1991).**
For example, dosages of antihypertensive medications may need to be reduced following successful treatment of OSA.

Matching the patient to treatment - Specific treatment measures for OSA depend upon the age of the patient and the presence or absence of anatomic airway obstruction. Regardless of the modality used, successful therapy leads to a rapid

reduction in apneic episodes and improvement in associated symptoms, such as hypersomnolence and personality changes.

Young age Most children with sleep apnea have an anatomic abnormality that may require surgery. Examples include enlarged tonsils and adenoids, and skeletal abnormalities involving the facial structures.
Young adults may not accept positive airway pressure therapy by mask (e.g., CPAP) as a long-term treatment. Thus, referral to an Otolaryngologist or maxillofacial surgical specialist or consideration of an oral appliance is often necessary.

Anatomic airway obstruction - The first step in the specific management of documented OSA is to consider the role of anatomical airway obstruction that may be present in each individual patient:
Enlarged tonsils or adenoids - Markedly enlarged tonsils or adenoids should be removed in children and adults with OSA **(Mangat, et al.,1977)**. Occasionally, other mass lesions causing apnea may require resection.
Skeletal abnormalities involving the face - Physical examination plus lateral cephalometric radiographs may provide insight into the potential contribution of abnormal craniofacial configuration to upper airway obstruction during sleep.

These radiographs may be important particularly in non-obese patients and in those with an examination suggesting abnormal facial or jaw structure.

Nasal obstruction

Treatment is indicated if nasal obstruction is thought to play major role in the genesis of apnea **(Mcnicholas, et al., 1982).**

In patients with allergic rhinitis, for example, a trial of inhaled nasal steroids (such as beclomethasone or fluticasone) may be tried for one month.

Topical vasoconstrictors and evaluation by an Otolaryngologist may also be helpful. It must be emphasized, however, that surgical correction of nasal obstruction may lead to amelioration of the apnea.

Treatment in the absence of anatomic airway obstruction -Therapeutic options in patients without anatomic causes of OSA include nasal CPAP, surgery, oral appliances and, in rare cases, drugs.

CPAP IN THE ADULT POPULATION

Indications for CPAP - CPAP is effective in the treatment of patients with clinically important obstructive sleep apnea/ hypopnea syndrome. Treatment is indicated when there is documented sleep-related apnea/hypopnea and evidence of clinical impairment.

CPAP may be effective in the treatment of patients with clinically significant Cheyne Stokes respiration or central sleep apnea syndromes. Treatment may be indicated if there is documented central apnea and clinical impairment.

CPAP is not routinely indicated in individuals with simple snoring that is not associated with pauses in respiration or with clinical impairment.

Technology - Reliable equipment that can sustain pressures of up to 20 cm is commercially available.Routine testing of the maximal pressure delivered by the device can be monitored with a simple water manometer. The optimal pressure should remain relatively constant throughout respiration.
Inspiratory and expiratory pressures in the nasal mask should be monitored during initial and follow-up evaluations and not vary more than 2 cm H20 between inspiration and expiration. Supplemental oxygen or humidity can be administered through the mask.

Compliance - Patients generally state that they are compliant with CPAP, but actual use of the device varies widely. Higher levels of compliance are most often associated with the relief of daytime sleepiness, fatigue and restoration of alertness.

Monitoring Routine outpatient clinical follow-up is recommended to determine the response to treatment and the level of compliance. Because compliance is difficult to estimate based on clinical assessment, it is recommended that some objective assessment must be made.

Indications for nasal CPAP

Obstructive sleep apnea - When an adequate CPAP is applied to the nasal inlet, obstructive apneas are eliminated (**Sullivan, et al.,1981**).

The mechanism of action is due to the mechanical effects of raising the intralummal upper airway pressure above the positive critical transmural pressure of the pharynx or hypopharynx **(Smith, et al.,1988**).

It has also been noted that with the passive distention in the upper airway there is a concomitant reduction in phasic electrical activity of the genioglossal muscles **(Strohl and Redline, 1986**).

After upper airway, patency has been established. If the upper airway pressure is reduced, the apneas immediately return **(Smith, et al.,1988)**.

The immediate clinical effectiveness of CPAP for patients with obstructive apnea has been well documented. Studies have consistently demonstrated that the symptoms and signs associated with the obstructive sleep apnea have been reduced as evidenced by improved cognition and psychological function
(Bearpark , et al.,1987) , improved symptomatic daytime hypersomnolence **(Frith and Cant, 1985)**, reductions in waking and resting carbon dioxide and reduction in heart rate and pulmonary artery pressure **(Marrone, et al.,1987)**.Moreover, long-term treatment with CPAP has been associated with decreased hematocrit and improved ventricular ejection fraction **(Krieger, et al., 1990)** . Chronic use of CPAP therapy has been associated with increased waking upper airway dimensions probably because of decreased soft tissue edema **(Ryan, et al., 1991)**.In the most recent study of 30 d of CPAP use even one night without treatment was associated with immediate return to pretreatment levels of sleepiness and psychomotor vigilance **(Kribbs, et al.,1993)**.

Finally, there had been two retrospective series that suggest an increased mortality from obstructive sleep apnea that may be reduced by treatment with CPAP **(He, et al., 1988)**. However, there have been no prospective studies to confirm these data.

Consensus statement CPAP is effective in eliminating obstructive sleep apnea, oxyhemoglobin desaturation, and respiratory event-related arousal from sleep. CPAP is also associated with improved morbidity as manifested primarily by reductions in daytime sleepiness and improved cardiopulmonary function. Although the long-term effects of nasal CPAP have not been fully determined, available data suggest a possible reduction in mortality.

Snoring - Simple snoring is the result of a partially collapsed and flow-limited airway **(Lilstro, et al., 1991)**.

Physiologic studies have shown that very loud snoring can be associated with normal oxygenation and sleep architecture **(Lilstro, et al.,1991)**. Moreover, it has also been shown that the transmural critical pressure in simple snorers falls in an intermediate range between normal individuals and patients with obstructive hypopnea and apnea **(Glesdhill, et al., 1991)**. Therefore,
CPAP can be applied in a fashion similar to patients with obstructive sleep apnea and identical results are obtained **(Glesdhill, et al., 1991).**

Nasal CPAP has also been used to eliminate snoring that is associated with obstructive sleep apnea **(Berry, et al., 1984).**
In the first study, both the snoring and associated apneas were eliminated **(Berry, et al., 1984)**.

In the second, study that examined snorers with evidence of hypopneas and recurrent arousals. CPAP effectively restored normal breathing and eliminated arousals. However, no clinical changes were detected other than a marginal increase in the multiple sleep latency test **(Guilleminault, et al.,1991**).
Nasal CPAP has also been successfully used in a select subset of patients with asthma that is associated with obstructive apnea and heavy snoring (**Chan, et al.,1988).**

There have been no studies examining the clinical efficacy of nasal CPAP in individuals who only have evidence of snoring without any evidence of obstructive hypopneas or apneas.

Consensus statement - CPAP is effective in eliminating snoring associated with apneas/hypopneas and arousal from sleep. There are no clinical studies that demonstrate the health benefits of CPAP in the treatment of patients with simple snoring.

Central sleep apnea - In contrast to obstructive sleep apnea, much less is known about the effectiveness of CPAP on periodic breathing and associated central apneas. In patients with central apnea or Cheyne Stokes respiration. CPAP causes a variable reduction in the numbers of apneas **(Hoftstein, et al,.1987**).

In part, this variable response stems from our lack of understanding of the underlying mechanism of action. Currently, several mechanisms have been proposed including reflex activation of upper airway receptors **(Issa and Sullivan ,1986**) improved oxygenation **(Hanly, et al,.1989**) and improved circulatory mechanics **(Takasaki, et al,.1989**).

The few published studies suggest there is either an immediate direct reduction in apnea frequency or possibly an indirect time-dependent reduction in apnea that results from improved cardiac function **(Bradley, et al., 1992).**

The clinical effectiveness of nasal CPAP in central sleep apnea is less well documented. In one study examining the effects of CPAP in patients with congestive heart failure and Cheyne Stokes respiration, 3-mo follow-up demonstrated improved sleep, less restlessness, and subjective alleviation of fatigue and hypersomnolence (**Takasaki, et al., 1989**).

In addition, nasal CPAP led to improved overall left ventricular function with decreased symptoms of heart failure and a change from class III/IV (New York Heart Association Classification) to class II. However, in a more recently published 2-wk controlled trial of nasal CPAP there was no overall benefit **(Davies, et al., 1993**).

In fact, it was felt to be detrimental in certain patients with congestive heart failure **(Davies, et al., 1993)**. To date, there have not been any long-term studies on clinical effectiveness of CPAP.

Consensus statement - CPAP may be effective in eliminating polysomnographic central sleep apnea. There are inadequate data determining whether CPAP will improve the clinical symptoms associated with central sleep apnea.

Adjunctive therapy - In some patients with obstructive sleep apnea, significant oxyhemoglobin desaturation will persist in spite of the elimination of apneas. In these patients, supplemental oxygen can be added to the CPAP system directly into side port either of the mask or into the inspiratory flow through the tubing leading to the mask **(Demerozu, et al., 1991).**

In general, it should be appreciated that high levels of O_2 flow will be necessary if the inlet for O_2 is not in the mask itself and that this will significantly increase the cost of therapy. O_2 added in the tubing leading to the mask at a site proximal to the venting site must mix with a relatively high flow of room air, whereas O_2 added directly in the mask is diluted only by the patient's ventilation. The amount of oxygen flow necessary to restore normal arterial oxygen saturation can be determined during the sleep study by monitoring arterial saturation, but will vary widely with changes in leak at the mask and with the pressure of CPAP delivered.

It is also important to note that oxyhemoglobin desaturation may be due to alveolar hypoventilation and, thus, forms of assisted mechanical ventilation may need to be considered **(Bach and Alba 1990).**

Because the low humidity can cause vasomotor rhinitis, in-line humidification systems are now available positioned either at the intake of the blower unit or by the addition of an in- line cascade type humidifier **(Rodenstein and Stanescu 1986)**. In addition, effective humidification of inspiratory airflow has also been achieved by hygroscopic condenser humidifier **(Parra, et al., 1991)**, although these features do add to cost.

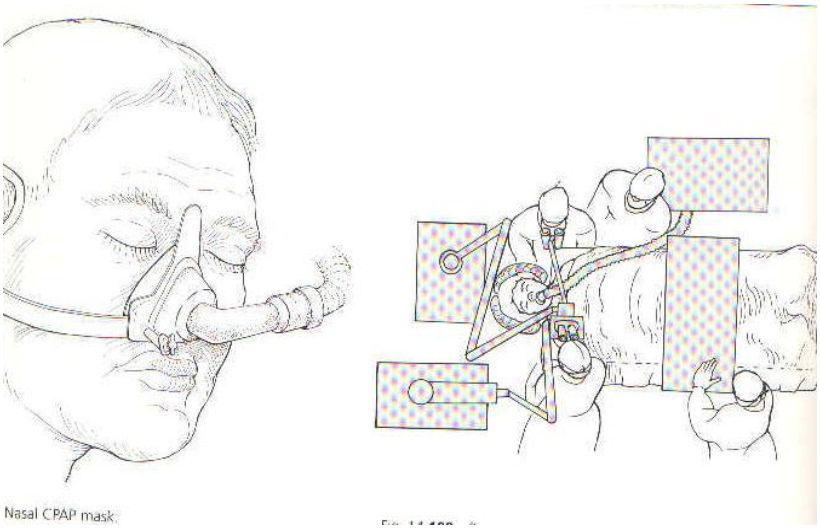
COMPLICATIONS - At first, seemingly minor complications were associated with the CPAP devices, although it was initially thought that these complaints might be significantly reducing compliance. Data that are more recent however, suggest that the lack of clinical benefit, not minor discomfort. Is the major factor influencing compliance (see below). Local skin irritation, drying of the nasal and pharyngeal membranes (50 percent),nasal congestion /rhinorrhea (25 percent), and eye irritation (25 percent) are the most common .

On-line humidification with a standard mist humidifier can significantly reduce the mucosal systems and newer, better sealing masks or nasal prongs can decrease eye irritations.

Rare case reports of major complications with the device have been reported and include pneumocephalus **(Janour and Wilson,1989**), bacterial meningitis (**Bamford and Quan, 1993**), conjunctivitis (**Stauffer, et al., 1984**) **,** massive epistaxis and atrial arrhythmia (**Meurice, et al., 1992**). There are no reports of pneumothorax.

Consensus statement - CPAP is a safe form of therapy with relatively few recorded major complications. Nevertheless, minor discomfort and complaints regarding the mask interface remain relatively common. Severe facial skin irritation due to nasal masks may be avoided by using ADAM nasal pillows

Relative contraindications include patients with bullous lung disease and recurrent sinus or ear infections. There are no absolute contraindications.



(Figure 29)

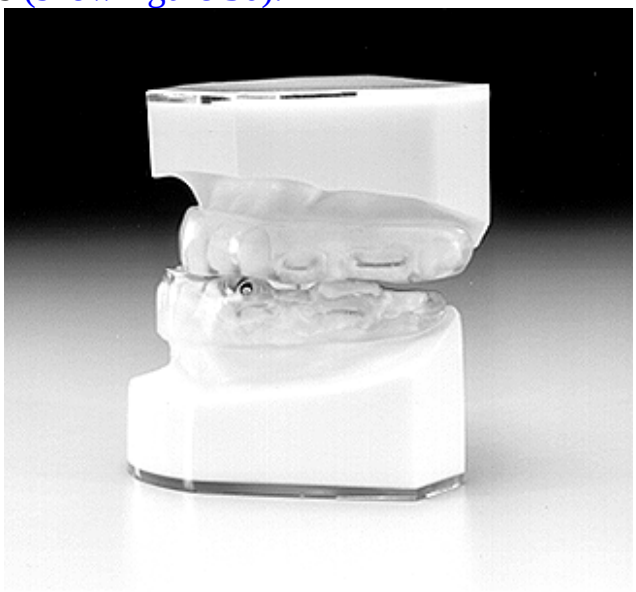
Oral appliances in the treatment of obstructive sleep apnea

Oral appliances are increasingly used as a treatment modality for patients with obstructive sleep apnea (OSA). The recent publication of practice guidelines by the American Sleep Disorders Association (ASDA) Standards of Practice Committee represents a milestone in the acceptance of this form of therapy by sleep medicine clinicians **(Thorpy, 1995).**

This part will address the types, effectiveness, and indications for use of oral appliances in the treatment of OSA. The problems with oral appliances will also be discussed, and a practical approach to the use of these devices will be presented.

Types of oral appliances - An oral appliance is a device inserted into the mouth in order to increase the size of the upper airway during sleep and thereby relieve obstruction. Two major types are in current use:

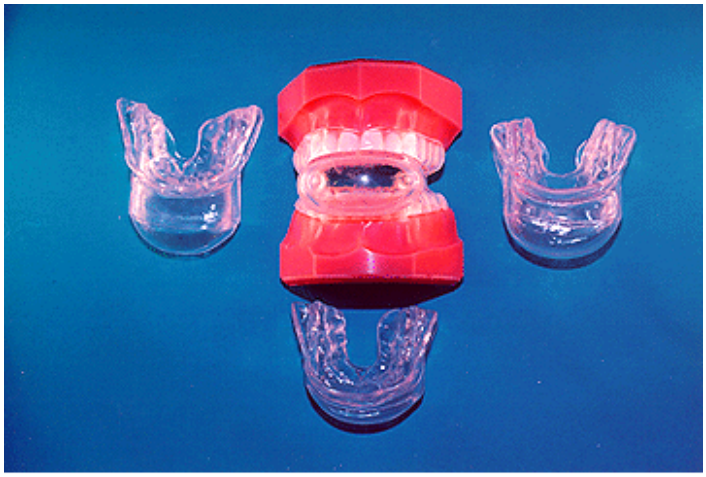
The great majorities of devices modify mandibular posture to a forward and slightly opened position. These types of devices are secured to the teeth by using orthodontic techniques (show figure 30).



Mandibular repositioning appliance This mandibular repositioning oral appliance is manufactured in a laboratory from dental impressions (Klearway appliance, Great Lakes Orthodontics, Inc, Tonawanda, NY). Special features stressed by the manufacturer are the full dental coverage, the low profile of the appliance, and adjustability of mandibular position by means of a screw.

(Figure 30)

The second type of device uses a suction cavity to hold the tongue in an anterior position (show figure 31).



Tongue retaining device (four views) When inserted into the suction cavity of this oral appliance, the tongue is held forward to maintain airway patency. (Tongue Retaining Device, Professional Positioners, Inc, Racine, WI).

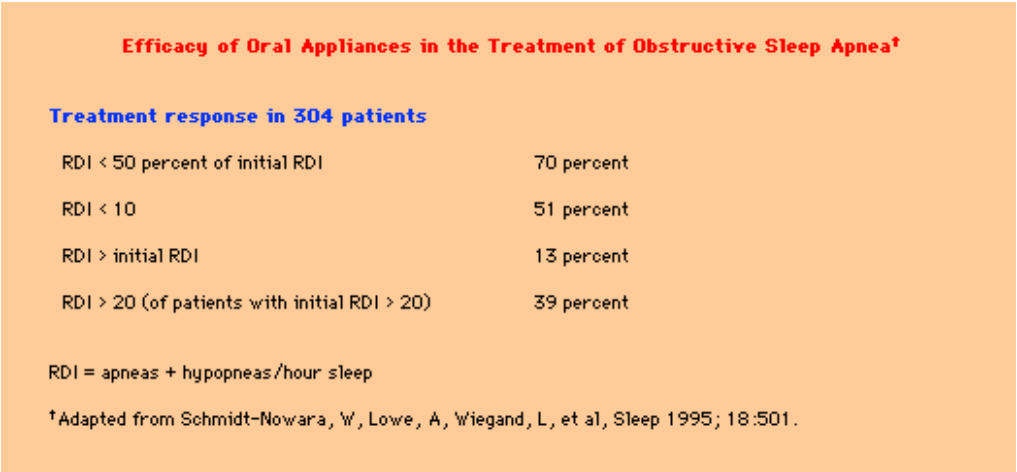
(Figure 31)

Mandibniar repositioning devices - The mandibular repositioning appliances differ by size, the type of material (hard or soft), the degree of mandibular mobility permitted while worn, and the presence of an opening to permit oral breathing. Most of these devices are fabricated in a dental laboratory from traditional dental impressions, but prefabricated versions can be fitted directly to the patient. Newer designs allow adjustment of the degree of mandibular advancement.

The importance of these various features is not well defined, but they allow the adaptation of the device to a patient's specific needs. The capacity to adjust the mandibular position is a particularly attractive development, since the degree of advancement necessary for successful treatment is difficult to predict prospectively in an individual patient.

Effectiveness - A review of therapy with oral appliances summarized the data from 21 papers and 320 patients in whom efficacy was investigated by polysomnography and other methods **(Schhhmidt, et al., 1995)**.

The findings were remarkably consistent, even though a variety of oral appliance designs was used. However, the patients described in these papers had less severe apnea and obesity than typical clinic patients with OSA, suggesting some selection by the investigators (show figure 30).



(Figure 32)

Snoring was improved in 98 percent of patients, usually to a clinically satisfactory degree. Improvement was assessed by patients' reports but was also documented by **objective recording (O,Sullivan, et al., 1995).**

Sleep apnea was treated effectively in most, but not all patients. The mean respiratory disturbance index (RDI = apneas + hypopneas per hour of sleep) in 304 patients decreased from 43 to 19. The majority of patients were substantially improved, including a 51 percent "cure" rate (RDI <10). However, 13 percent had an increased RDI when oral appliances were used, and a substantial number had an RDI that remained above 20 (show figure 32)

Predictors of treatment success were a lower baseline RDI (initial RDI above 40 to 50 was associated with significant residual RDI. positional apnea, and certain cephalographic indicators. The correlation of cephalography with treatment success, however, is not sufficiently strong to be clinically useful **(Eveloff, et al., 1994)**.

Sleep quality by ploysomnography and reports of sleepiness improved, but objectively measured sleepiness has not been assessed.

When reported, oxygenation improved, but by a relatively small amount.
One controlled crossover study of 20 patients with mild to moderate OSA compared the results of treatment with an anterior mandibular positioner and nasal CPAP **(Ferguson, et al,.1997).**

Nasal CPAP resulted in more treatment successes (70 versus 55 percent) and a significantly lower apnea/hypopnea index (4.2 versus 13.6), but patient satisfaction was greater with the oral appliance.

Indications for use - the ASDA. Standards of Practice Committee has declared oral appliances to be indicated for patients with:

*Primary snoring, i.e., patients without significant sleep apnea.

*Mild OSA who do not select or do not respond to weight loss or position change as their principal therapy.

*Moderate to severe OSA if nasal continuous positive airway pressure (CPAP) is refused or cannot be tolerated, and if surgery is refused or not indicated.

* Oral appliances should be considered a second choice of therapy in such patients, because CPAP is generally more effective.

We also believe that use of oral appliances may be appropriate in the following circumstances:

*The upper airway resistance syndrome, because of the substantial effectiveness of oral appliances for treatment of snoring.

*Failure of uvulopalatopharyngoplasty surgery **(Thorpy, 1995)**.

Contraindications - There are several contraindications to the use of oral appliance therapy:

*Poor dentition and temporomandibular joint problems are contraindications to the use of mandibular advancing appliances.

*Severe nasal obstruction.

*Severe hypoxemia during sleep.

Problems - All oral appliances require some patient training to overcome the initial aversion to sleeping with an object in the mouth. Excessive salivation is a common problem that abates with steady use. Some discomfort of the teeth and jaw on awakening can also be expected initially; adjustment of the appliance is necessary if the discomfort persists.
The mandibular repositioning appliances can potentially damage the teeth and the temporomandibular joint (TMJ) because of the mechanical stress imposed on these structures.

However, these problems are minimized by:

*Careful dental evaluation before treatment to exclude patients with poor dentition or TMJ problems.
*Careful instruction to patients to report pain and malocclusion.
*Periodic dental follow-up.

Compliance with the use of oral appliances is imperfect, as it is with other volitional treatments for OSA. Reported compliance rates vary from 50 to 100 percent, the lower rates occurring in case series with longer follow-up **(Schhhmidt, et al., 1995**).

However, compliance is based upon patient reports, and by analogy with CPAP, may be lower if measured objectively.
The primary reason for patients to discontinue treatment is inadequate treatment effect; discomfort and occlusive changes have been factors in other patients.

Recommendations - The initial diagnostic evaluation requires a medical assessment for OSA. This should include some kind of respiratory monitoring during sleep, since questionnaires are not adequately sensitive to exclude OSA in snoring patients even in the absence of other symptoms **(Kump, et al., 1994).**

The overall choice of therapy should include consideration of the severity of the patient's OSA as well as patient preference for a particular form of therapy. For patients with moderate severity OSA, patient preference may favor oral appliance therapy over CPAP **(Ferguson, et al.,1996).**
Once therapy with an oral appliance has been chosen, a coordinated approach becomes important, involving a physician familiar with OSA and a dentist with special knowledge of oral appliances.

The selection of the type of oral appliance will be influenced by the dental examination and the dentist's experience with different types of devices.
For mandibular repositioning appliances, an initial judgment must be made about the necessary degree of mandibular advancement, and consideration of this goal must be incorporated into the design and fitting of the appliance.

After the initial fitting of the oral appliance, the patient must be examined for response, initially by symptoms and ultimately by repeat objective monitoring. Adjustment of the appliance may be required on the basis of these observations. Follow-up sleep testing should be deferred until adequate symptom response and patient comfort have been achieved. Long-term follow-up should be planned by the dentist and physician, with attention to compliance, comfort, dental complications, and evidence for recurrent OSA.

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Pharmacologic therapy

Many drugs with CNS effects also can alter patterns of sleep and wakefulness, both during periods of acute administration as well as during periods of withdrawal. Several sleep disorders are caused by dependence on psychoactive drugs. In addition, some medications can exacerbate primary sleep disorders, thereby affecting sleep indirectly. The potential effects of medications or substance abuse on sleep should be assessed in any patient presenting with sleep complaints, as drug effects can be a primary or contributing cause of sleep disturbances. Sleep alterations caused by commonly used medications and recreational drugs are summarized.

The data presented here are based primarily on polysomnographic evaluations rather than on self-reports. Although many studies have evaluated the effects of hypnotic agents on sleep using electroencephalography (EEG), relatively few studies have assessed the effects of other drugs on sleep and wakefulness systematically **(Buysse, 1991).**

Some widely used drugs are available and the effects of their administration as follow:

Progestrone

Medroxy-progesterone acetate (MPA), a derivative of progesterone hormone, and recognized as a respiratory stimulant in male subjects and should be reserved for a small number of patients with the obesity-hypoventilation syndrome (sleep apnea, obesity, and awake hypoventilation) who are not controlled with CPAP or bi-level positive airway pressure devices. Such patients may, in rare cases, respond to MPA in a dose of 60 mg once daily. MPA does not improve patients with OSA who do not have awake hypoventilation. **(Fairbanks and Fairbanks 1992).**

Protriptylline

Protriptylline, is a non sedating tricyclic antidepressant, has been found to produce a beneficial clinical response in patients with mild to moderate OSA. The dose level appears to be in range of 5 to 30 mg taken at bedtime. Its mode of action is by the reduction of percentage of time spent in rapid eye movement sleep. The most serious side effects of treatment with protriptylline are cardiac arrhythmias, myocardial hypertrophy and heart failure **(Brownell, et al.,1982).**

Nicotine

Nicotine has been shown to posses' respiratory stimulant properties and has been tested as a treatment for sleep apnea. Eight patients with sleep apnea chewed gum containing 2 to 4 mg of nicotine for 20 minutes at approximately hourly intervals between 3 and 8 PM, for a total dose of 14 mg of nicotine. Nicotine did reduce apnea during the early hours of sleep, but it is impractical as a treatment agent for apnea due to its short duration of action and side effects **(Gothe *et al.* ,1985).**

Theophylline

Theophylline acts, as a respiratory stimulant and has been used successfully to treat apneas during infancy. In adult with OSA, theophylline has caused a significant decrease of the number of apnea, hypopnea episodes, and the duration of the hypoxic episodes. In-patient with pulmonary hypertension there was a marked tendency towards normalization of pulmonary pressure. These results support the positive effect of a long-term theophylline administration in patients with OSA **(Kaplan *et al.*, 1993).**

Clonidine Hydrochloride (Catpres)

Clonidine hydrochloride, an alpha 2-adrenergic agonist with REM-suppressant active, has been evaluated recently in the treatment of OSA patients. A dose of 0.2mg of clonidine administered orally at bed-time totally suppressed REM sleep.Clonidine had no effect on the frequency and duration of non REM breathing abnormalities **(Issa, 1992).**

Acetazolamide.

Acetazolamide, a carbonic anhydrase inhibitor, which stimulates ventilation by increasing the hydrogen ion concentration of arterial blood, has been reported to decrease apnea frequency, apnea associated arousals, and the severity of oxygen desaturation in-patient with central sleep apnea. Although the effects of acetazolamide in patients with OSA have not been reported, it may well produce beneficial therapeutic re-suits in patients who have coexisting hypoventilation or metabolic alkalosis based on its ability to decrease periodic breathing, apneas, and the severity of hypoaemia during sleep in high altitudes **(Moran and Orr, 1985).**

Oxymetazoline Hydrochloride (OXY)

Oxymetazoline hydrochloride, a long-acting vasoconstrictor in patients with OSA leading to improve upper airway patency during sleep and reduce the incidence of disordered breathing events 0.025% OXY was administered by gargling(5 ml) and transnasal instillation (one ml) prior to sleep.Mean apnea index decrease and respective apnea/hypopnea indices also decreased **(Hutt *et al.*, 1990).**

Thyroid Hormone

Very severe OSA is common in thyroid deficient patients and may be present in the majority of cases. There are several possible mechanism for OSA with hypothyroidism.These include obesity, macroglossia, impaired upper airway muscle function, deposition of mucopolysaccharides in tissues of the upper with newly diagnosed hypothyroidism had OSA.

Thyroxin used in patients found to be severely hypothyroid and/or myxoedematous has been shown to be successful in reversing the OSA and the accompanying symptoms **(Moran and Orr 1987).**

The critically ill patient - The critically ill patient with OSA (severe sleepiness and cardiorespiratory failure) should be hospitalized and monitored until stabilized from physiologic and symptomatic perspectives **(Miller, et al,.1992).** Such patients should be started on CPAP or bi-level pressure devices via mask as soon as feasible. Patients with less severe symptoms can be evaluated and managed in an out-patient setting.

FOLLOW-UP - All patients who undergo surgery for OSA should have a repeat evaluation during sleep to ensure that apnea is under control with adequate restoration of oxygenation and sleep continuity. One of the cardinal symptoms of apnea, snoring, may be ameliorated after surgery without necessarily abolishing the apneic episodes **(Krayger , 1994).**

Repeat sleep evaluation is also indicated in patients receiving nonsurgical therapy who donot improve or who have recurrent symptoms. The prescription may change if the patient's weight increases or if symptoms return. Possible causes of treatment failure include noncompliance with therapy, weight gain, an inappropriate level of prescribed positive pressure, or an additional disorder causing sleepiness, such as narcolepsy or periodic movements during sleep, which may require alterations in the therapeutic regimen.

SURGICAL TREATMENT OF OSA

Introduction

Obstructive sleep apnea is a complex neuromuscular syndrome with potentially serious respiratory – cardiovascular consequences. Obstructive sleep apnea is characterized by frequent apneas, habitual heavy snoring, and daytime sleepiness. Cessation of airflow lasting at least 10 seconds and occurring at least 30 times during night sleep is another indication of OSA **(Powell *et al,* 1991).**

It is now generally accepted that the site of obstruction in OSA may involve one or multiple segments of the upper airway. Surgical treatment of OSA bypasses the site(s) of obstruction or modifies the anatomy of the collapsible or obstructive segments.

The surgical therapies described are mainly applied as separate procedures or in various combinations.

Nasal Surgery

For patients in whom medical treatment fails, surgery of the nose is recommended**.**

Series et al.,(1992), Gustaut , et al.,(1966) and several others have studied the key role played by nasal resistance and have proven that nasal obstruction can destabilize the oropharyngeal airway.

Surgical treatment of the nose and nasopharynx is aimed at eliminating obstruction at these sites from increasing the airway resistance. Nasal surgery has been documented by numerous studies to successfully improve oxygen saturation, decrease arousal, and produce subjective improvement in snoring and daytime somnolence **(Hester, et al.,1985).**

To assess the nasal component of the airway problem in snoring and OSA a simple diagnostic test is recommended. Patients are asked to use an decongestant nasal spray (e.g., oxymetazoline) three sprays in each nostril half an hour before bedtime. Patient and family are then easily able to compare snoring on "spray nights" versus "non-spray nights."

If the snoring completely disappears on the nose spray nights it can be concluded that the nose contributed as a major factor in the sleep disordered breathing, and surgery would likely relieve the problem.

If, on the other hand, there was no improvement at all by the nose spray then nasal treatments would be unlikely to help significantly, unless there is a fixed obstruction, as septal disorders.

Fairbanks (1991) performed the nasal spray test on patients prior to surgery for obstructive sleep apnea. He and his colleagues observed more than 300 patients over 5 years and found that studying the effects of a vasoconstrictive nasal spray subjectively had an important predictive role in determining whether or not nasal surgery would benefit those with snoring or sleep apnea.

Nasal valve surgery

The nasal valve area is the slit-like opening formed by the septum,the caudal end of the lower lateral nasal cartilage, the soft tissue overlying the piriform aperture, the floor of the nose, and the head of the inferior turbinate. The nasal valve is stabilized by cartilage and bone, and modulation occurs by the nasal muscles and "pseudo-erectile" tissues of the nasal septum and inferior turbinate. Loss of stability of the lateral nasal wall with subsequent collapse may occur at the level of the nasal valve.

It may be the result of either nasal muscle dysfunction, for example, after a facial nerve palsy or loss of skeletal support, after over-aggressive surgery, or both. Correction of such collapse as shown by **Irvine et al., 1984** in two patients with severe sleep apnea may improve nasal airflow and decrease the arousals and oxygen desaturation of OSA. The use of "spreader grafts" sutured between the dorsal border of quadrangular cartilage of the septum and the upper lateral cartilage also can widen effectively the middle third of the nose and thereby improve nasal function **(Toriumi, 1993).**

Septal Surgery

Many different procedures are used to straighten and thin the septum. An incision is made through the mucosa and perichondrium on one side, and a flap is elevated. The cartilage is then cut through and the mucoperichondrium is then elevated on the opposite side until the septal cartilage and bones are freed of all attachments. Deviated parts of the cartilage and bone are then removed or replaced in a better position. This procedure can be done with the help of a headlight or endoscopically with rigid endoscopes.

Rubin , et al (1993) evaluated patients with moderate to severe sleep apnea syndrome, and 9 of the 16 treated by submucosal resection (SMR) reported a clinical improvement in the quality of their nocturnal sleep and diurnal hypersomnolence. Post-treatment polysomnographic recordings in responders disclosed significantly less apneas and waking within sleep.

In 1982 Lavie et a1 showed that 12 of the 14 subjects had significant subjective improvement in chronic fatigue and sleepiness after nasal septal surgery.

Similar studies by **Konno et al, (1980), Zwillich, et al,(1981) and Olsen, et al(1981)**have shown that mechanical occlusion of the nasal passages during sleep leads to a three- to fivefold increase in sleep disruption, an increase in arousals and awakenings, and desaturation up to I0-fold, ultimately interfering with an individual's sense of well-being both when awake and asleep.

Series, et a1(1992) evaluated the effects of nasal surgery on sleep-related breathing abnormalities in 20 adults with obstructive sleep apnea. Polysomnographic studies were done before (baseline), and 2 to 3 months after surgery (septoplasty, turbinectomy, and/or polypectomy). Nasal resistance-were measured at these visits in 14 patients.

Cephalometric measurements were obtained before surgery. Nasal resistance decrease significantly after nasal surgery. Interestingly, apnea and apnea plus hypopnea indices returned to normal values (<5 and 10, respectively) in four subjects with normal cephalometric indices

Turbinate surgery

Often septal surgery is combined with an operation to reduce the size of the fleshy internal nasal swellings called turbinates. Many operations have been devised to reduce the turbinates. These range from completely amputating the turbinate to shrinking the turbinate by radiofrequency probes or an electrically heated wire. Other methods include limited trimming of the turbinate by microdebrider, submucous diathermy (SMD) and laser excision

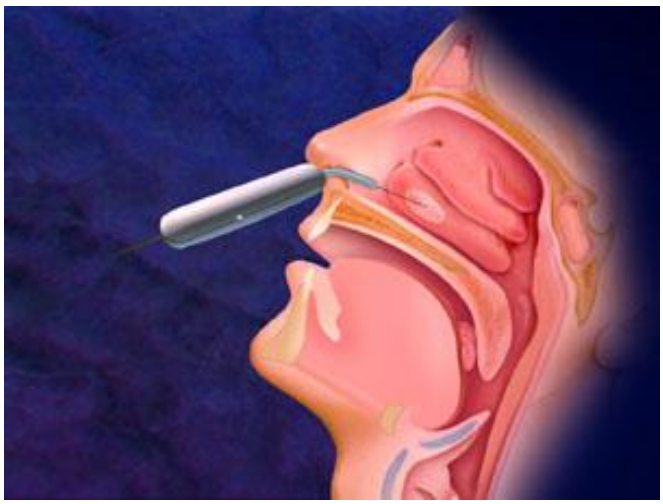
Turbinate Somnoplasty: Radiofrequency Ablation of Hypertrophied Nasal Turbinates

There are many potential causes for nasal obstruction. Some of the most common causes are nasal allergies, deviation of the nasal septum (the partition in the middle of the nose on the inside), or sinus or nasal infection.

The nasal passages can also be obstructed by enlarged turbinates. Enlarged turbinates can impair normal breathing, causing patients to breathe through the mouth. Enlarged turbinates may be treated with intranasal sprays and medications. If turbinate hypertrophy is chronic, surgical interventions may be considered.

Radiofrequency Tissue Volume Reduction (RFTVR) with temperature monitoring and control is a surgical method, which uses radiofrequency heating to create targeted coagulative submucosal lesions resulting in tissue volume reduction. RFTVR uses very low levels of radiofrequency energy to create finely controlled necrotic lesions in soft tissue structures. Following the general pattern of wound

healing, the necrosis leads to scar formation and retraction of tissue, resulting in an overall reduction of volume in the treated area. Over time, the scar tissue is partially resorbed by the body, causing further volume reduction.



(Figure 33)

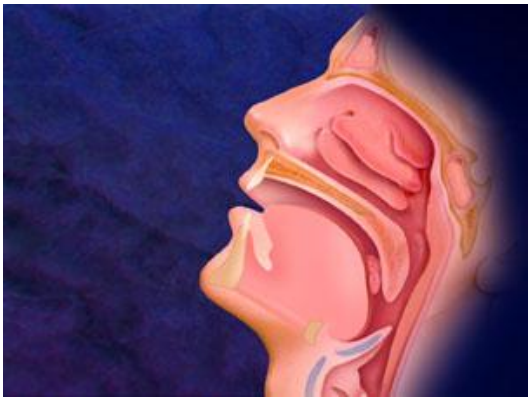
Frame 33: Submucosal Delivery of RF Energy

In this outpatient procedure, the patient receives a local anesthetic. Using direct vision, the physician inserts the SP 1100 electrode into the inferior turbinate. The Somnus radiofrequency generator delivers RF beneath the mucosa.

(Figure 34)

Frame34: Creation of Coagulative Lesion

Tissue is heated in a limited area around the electrode, creating a submucosal coagulative lesion. The patient does not feel discomfort during the procedure, and the mucosa is protected from thermal damage.



(Figure 35)

Frame 35: Tissue Volume Reduction

The lesion is naturally resorbed by the body, leading to tissue volume reduction and relief of nasal obstruction. This can be an effective treatment for patients who suffer from chronic turbinate hypertrophy

The advantage of radiofrequency volumetric tissue reduction (RFVTR) over electrocautery for turbinate hypertrophy is that the former can be performed as an office procedure with local anesthesia, whereas the latter is done in an outpatient surgical center under general anesthesia **Troell (2003)**.

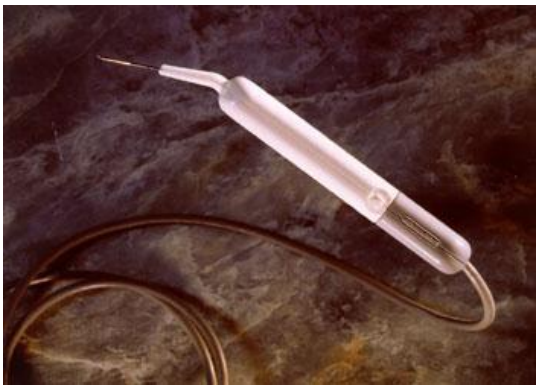
In a prospective, non-randomized study**, Black et al (2002)** evaluated the effectiveness and morbidity of bipolar radiofrequency thermal ablation of the inferior turbinates in patients with nasal obstruction caused by turbinate hypertrophy (n = 20). The authors concluded that the bipolar radiofrequency thermal ablation of inferior turbinates is a promising option, for inferior turbinate hypertrophy.

In a prospective, randomized clinical trial**, Sapci et al (2003)** compared nasal functions of patients with chronic nasal obstruction following treatment by (i) radiofrequency tissue ablation, (ii) laser ablation, and (iii) partial turbinectomy. The study was carried out on 45 adult volunteer patients with symptoms and signs of nasal obstruction and stuffiness related to enlarged turbinates (n = 15 in each group). These investigators found that that radiofrequency tissue ablation to the turbinate was effective in improving nasal obstruction objectively and in preserving nasal mucociliary function. Laser ablation of the turbinate was effective in improving the

nasal obstruction; however, it significantly disturbed the mucociliary function. Partial turbinectomy resulted in similar improvements as obtained with radiofrequency tissue ablation. These findings were based on a small sample size and a relatively short follow-up (12 weeks).

Thus, further follow-up studies with a larger sample size is needed to evaluate the long-term improvement of symptoms and maintenance of nasal functions.

In a prospective, non-randomized study, **Lin et al (2003)** assessed the effectiveness of turbinate surgery with radiofrequency for the treatment of allergic rhinitis that is unresponsive to medical therapy (n = 108). These researchers concluded that radiofrequency appears to be an effective and safe tool for treating allergic rhinitis with poor response to medical therapy. If further long-term studies confirm these findings, radiofrequency has the potential to be one of the most popular surgical modalities for the treatment of allergic rhinitis refractory to medical therapy.



(Figure 36)

(SP 1100) The SP 1100 handpiece is designed for the delivery of controlled thermal energy to the inferior turbinates.

Sinus Surgery/Polypectomy/Adenoidectomy

Surgery of the sinuses should be undertaken when medications have been tried and a CT shows evidence of persistent disease. Surgery is aimed at re-establishing ventilation and drainage.

Endoscopic sinus surgery, often referred to as sinoscopy, entails the use of a sinuscope for access and inspection of the sinus cavities and related structures. Sinoscopy can be either diagnostic (i.e. to determine the exact source of sinus symptoms), or involve the use of lasers or other instruments that allow the surgeon to actually treat the underlying cause of the symptoms. Small or isolated polyps can often be completely removed using a small mechanical suction device or a microdebrider — an instrument that cuts and extracts soft tissue. Also, a microdebrider can used for a turbinate reduction refers to any type of procedure to

reduce the size of the nasal turbinate, and consequently increase the size of the nasal cavity.

The microdebrider system designed for simultaneous resection and evacuation of soft and bony tissues and fluids in otorhinolaryngology and head and neck surgical procedures.

Advantage:

Unlike conventional non powered suction and cutting instruments, sinonasal soft tissue shavers have the advantage of evacuating the tissue from the surgical site without the need to remove the instrument, providing potentially continuous suction of blood and resected tissue from the field with the opportunity for improved visualization and precision and less frequent interruption during the procedure **(Becker, 1997).**

It has been found that microdebrider to be an excellent tool in functional endoscopic sinus surgery, because the device is used parallel to critical anatomical structures, such as the lamina papyracea and the skull base, the technique is safer and carries less risk to these area **(Setihff,1996).**

There is much less mucosal trauma as no stripping of the mucosa occurs with this device with this device/ and less denuding of the bone, as a result of this decreased trauma and more mucosal preservation, healing time is reduced and re-epithelization of the denuded surface occurs much more quickly **(Krause and Christmas, 1996).**

Disadvantage:

Disadvantage of the shavers is that the feel of palpation one gets with a conventional instrument is lost. Smaller, lighter hand pieces are designed to return this important feedback **(Becker, 1997).**

Biopsy specimens, however a still obtained best with a conventional instrument. **McGarry and Adamson** claim that the use of microdebrides does not preclude the submission of tissue for histological analysis. Tissue is collected at the time of surgery via an in-line specimen trap. They don't recommend the use of a microdebrider system in its present form when dealing with a known malignancy or in situations where resection margins are important **(McGarry et al., 1996).**

Functional endoscopic surgery generally can treat these conditions intranasally. This may involve removal of the uncinate process, infundibulotomy, ethmoidectomy, maxillary antrostomies, frontal sinusotomies, and sphenoidotomies, depending on the preoperative CT extent of the disease.

What advantages does sinoscopy offer over conventional sinus surgery?

- Local vs. general anesthesia
- Outpatient procedure vs. required hospital stay
- Ability to reach areas that previously could not be visualized directly
- Less trauma to surrounding tissue

Mucosal preservation is attempted throughout while marsupializing or exenterating disease in the affected sinuses. Polypectomies are performed if necessary.

Hypertrophy and infection of adenoids requires removal with a curette. Small amounts of residual lateral adenoid tissue also can be cauterized.The goal is to have patent choanae.

OROPHARYNGEAL SURGERY IN THE MANAGEMENT 0F OSA

Surgery to the oropharynx for sleep disordered breathing is probably the most common surgery done for this problem. This is because of two reasons.

First is that these types of procedures are taught universally in otolaryngology training programs throughout the United States, whereas many of the other types of procedures for tongue advancement or other hypopharyngeal surgery and skeletal surgery for airway correction are taught rarely.

Second, obstruction at the level of the oropharynx is the most common point of obstruction in the airway. Looking at the breakdown of airway obstruction, the oropharynx alone would appear to be involved by itself in roughly 25% of cases, and with the hypopharynx or retrolingual pharynx in about another 50% of cases, so that the total involvement of the oropharynx or retropalatal pharynx in obstructive breathing disorders during sleep is between two thirds and three fourths of all patients seen **(Coleman and Christofer ,1999.)**

The type of procedure done for patients with problems of soft tissue obstructing the airway at the level of the oropharynx or retropalatal air-way depends upon the type of problem being treated and the anatomy involved.

Procedures range from very conservative operations to very aggressive operations.

The more conservative operations usually are done in the office under local anesthesia and involve treatment to the uvula or middle section of the palate. Infrequently are the tonsils or lateral walls of the pharynx involved with these types of treatments.

The more aggressive treatments are the variations on uvulopalatopharyngoplasty that treats not only the uvula and middle section of the palate, but also the lateral portion of the palate, tonsil, or tonsillar fossa, and *the* lateral and posterior pharyngeal walls at that level **(Coleman and Christofer,1999).**

UVULOPALATOPHARYNGOPLASTY (UPPP)

Otolaryngologists recently have become actively practicing-uvulopalatopharyngoplasty (UPPP) is a procedure, which has gained popularity over the past few years **(Lusk, 1986).**

Uvulopalatopharyngoplasty is effective in the majority of carefully selected patients with OSA. The operation is also extremely successful in reducing or eliminating snoring.

The UPPP has resulted in greater than 50% reduction of the apnea/hypopnea index in approximately 70% of the patients.
Reduction or elimination of snoring was reported by 87% of the patients **(Katsantonis, 1991).**

Most articles published in recent years presented the results following UPPP without clearly specifying the surgical procedure performed. A classic surgical procedure has not been standardized yet, being modified by different teams of surgeons, most clinics treating patients with OSA develop their own UPPP technique **(Zohar *et aL*, 1993).**

Objectives of UPPP

Evolution in the operative technique in any new operation is driven by the need to achieve successful correction of pathological anatomy as well as the avoidance of complications. The desirable objectives of UPPP are:-

(I) maximize the lateralization of the posterior pharyngeal pillars including submucosal musculature, which would increase the lateral dimension of the oropharyngeal airway.

(2) Interrupt some of the sphincteric action of the palatal nasopharyngeal musculature, which would increase the patency of the nasopharyngeal airway.

(3) Maximize shortening of the soft palate in the lateral parts, while sparing midline musculature (resulting in a "squared off' soft palate appearance).

These objectives are important to prevent palatal tethering and nasopharyngeal stenosis and to preserve mobility land function of the palate for purposeful closure **(Fairbanks, 1991).**

Indications of Uvulopalatopharyngoplasty

1. Treatment of Snoring

Over the past decade, UPPP has become an acceptic treatment for chronic snorers. The operation can relieve 80-90% of snoring subjects **(Croft and Golding-Wood, 1990).**

2. Treatment of Moderate OSA

Uvulopalatopharyngoplasty should be offered to an individual with moderate OSA, to one who presents with endoscopic and cephalometric profiles characterized by the presence of obstruction at the level of the velopharyngeal sphincter, and to one in whom the hypopharyngeal airway is normal **(De Berry-Borowiecki *et al.*, 1985).**

3. Treatment of Severe OSA

Treatment of severe OSA with UPPP avoided the complications of tracheostomy and difficulties with long term CPAP. Morbid obesity and narrowing of the airway at more than one level are the main causes of the failure of UPPP in patients with severe OSA **(Walker *et al.*, 1989).**

Technique of UPPP.

Numerous surgical techniques have been applied in an attempt to accomplish the surgical objective technique obtained by the UPPP. **Fairbanks** described the following technique in **1991:**

* The patient was placed on an operating table in the tonsillectomy position. The oropharynx was exposed with a suitable self-retaining retractor.

*The uvula was grasped with forceps and pulled anteriorly.The crease on the ventral surface of the soft palate produced by this maneuver was used for marking a horizontal incision through the mucosa of the soft palate.

*The incision was extended laterally and downwards to the base of the tongue parallel with the free edge of the palate and palatoglossal fold.

* Submucous dissection with scissors or cutting diathermy was carried out towards the free edge of the palate and above the surface of the palate and pharyngeal muscles.

*The muscular uvula was transected and the palatine tonsils (if present) were dissected together with the mucosa of the posterolateral pharyngeal wall as a part of the specimen.

*The entire cuff of dissected tissue was placed on traction directed anteriorly and superiorly for the posterolateral pharyngeal mucosal flap.

*The redundant portions were then advanced and fixed in their respective new positions with interrupted 2-0 chromic mattress sutures **(Fairbanks, 1991).**

The use of the electro - surgical dissection technique is a superior method to reduce operative time as will as creates an essentially bloodless field **(Peters, 1986).**
In 1986 Lusk described the marking method for optimal resection of the soft palate with minimal risk of velopharyngeal incompetence. During Physical examination, the physician must carefully note the degree of lateral pharyngeal wall movement.

Aggressive reduction of the soft palate in patients with minimal lateral pharyngeal wall movement may result in velopharyngeal incompetence. This method described by **Lusk** has the advantage of individualizing the amount of tissue removed by referencing the incision to the point of contact between the soft palate and the nasopharynx; it takes into account the varying anatomy of the soft palate, the nasopharynx, and the degree of lateral wall movement **(lusk, 1986).**

The UPPP is generally a safe operation though not without some morbidity.

In a review of 101 UPPPS, **Fairbanks (1990)** found 56% of patients experienced nasopharyngeal regurgitation at sex - weeks post operatively, reducing to 24% at one year, and one patient died of airway obstruction. Difficulties in swallowing (10%) and speech (7%) also persisted at one year in their series whilst postoperative haemorrhage occurred in 6% of their patients.

Whilst not denying the role of UPPP in the management of snoring, the associated morbidity and mortality make it procedure not to be undertaken lightly and any alternative which can reduce the morbidity and mortality whilst still achieving satisfactory result should therefore be considered seriously **(Wibinney, et al., 1995).**

The place of UPPP in treatment of OSAS is still not fully defined and though UPPP may eliminate snoring in these patients, it has been shown not to improve the long - term mortality ***(He et al., 1988)***. This reinforces the importance of patient selection and late postoperative reassessment when using UPPP to treat patients with moderate OSAS **(Pringle and Croft, 1997).**

Post-Operative Management

(1) Intravenous antimicrobial prophylaxis is maintained for 48 hours, and if swelling, oedema begins to be apparent in the operating room or early in the post-operative period, short course of steroids is given intravenously.

(2) Pain medication is given more sparingly *(i.e.* low-dose parenteral morphine), with the recognition that apnea is aggravated by narcotics and life threatening loss of airway can be precipitated, especially in the post-anesthetic period or the period of post-operative oedema of the airway.

(3) Similarly, antiemetics, sleeping medications, and sedative-tranquilizers can be precipitating an apneic crisis **(Fairbanks, 1991).**

(4) Standardized post-operative routine, each patient is admitted to intensive care unit the first night post-operatively. Continuous ECG monitoring is performed, along with continuous nocturnal ear oximeter tracings. **(Macaluso *et al.*, 1989).**

(5) A nasopharyngeal airway tube is left in place for the first 48 hours, post-operatively **(Coleman, 1986).**

(6) After the first night in the intensive care unit, patients demonstrating adequate saturation are transferred to the ward **(Macaluso *et al.*, 1989).**

Complications of UPPP

A. Peri-Operative Complications

Regardless of the surgical procedure performed, patients with OSA are predisposed to specific complications because of anatomical abnormalities and the existence of their underlying systemic disease.

1. Peri-Operative Airway Obstruction

Airway obstruction represent approximately three quarters of all the immediate complications of UPPP.

Causes of peri-operative airway obstruction:-
(1) Uses of sedatives, narcotics, and strong analgesics.
(2) Unsuccessful intubation upon induction of anesthesia.
(3) Post-operative pharyngeal and base of tongue oedema.
(4) Post-operative haemorrhage **(Johnson and Sanders, 1986)**

2. Failed Intubation

Patients with OSAS have a dangerous and well-documented tendency to suffer exacerbation of apnea when given sedatives. This tendency of sedatives to acutely exacerbate sleep apnea is complicated by the common anatomical patterns of these patients. The majority of them will be over-weight and have short stock necks, and large tongue is almost present **(Esclamado *et al*, 1989).**

3. Cardiopulmonary Complications

Cardiopulmonary complications are primarily related to hypertension, cardiac arrhythmias, cor-pulmonale and congestive heart failure state. These conditions are commonly associated with severe OSA **(Burgess *et al,* 1992).**

4. Loss of Respiratory Drive Due to O_2 Administration

Loss of respiratory drive due to O_2 administration in hypoxemic and hypercapnic patients is a well-established phenomenon **(Johnson and Sanders, 1986).**

5. Emergence of Previously Undiagnosed Central Apnea (Eslamado, *et al,* 1989).

B. Surgical Complications

1. Early Complications

a.Pain

Generally, lasts 4 to 6 days, is analgesically treated, and poses no great difficulty to the patient.

b. H*aemorrhage*

Post-operative haemorrhage may be minimal or severe that required return to the operating room for control.

c. Local infection

Local infection may occur after the excision of the tonsil; a parapharyngeal swelling developed and septic fever ensued.

d.Dysphagia

Is usually during the first 2 to 4 post-operative days.

e. Nasal *reflux*

In the first 3 to 4 post-operative days.

f. Hypernasal Speech

Is the expression of velopharyngeal valve insufficiency and may last 7 to 14 days.

g. Eustachian Tube Dysfunction

Due to oedema of the pharyngeal opening of the eustachian tube or haematoma of the torus tubari.

(Croft and Golding-Wood, 1990)

II. Late Complications

a. Velopharyngeal Insufficiency

Transient early velopharyngeal insufficiency is manifested usually with nasal regurgitation and less commonly with hypernasal speech. The incidence of early post-operative regurgitation ranges from 20% to 60% and in the vast majority of cases, subsides within 2 to 3 weeks

Teflon paste injection in the submucosal layer of the posterior pharyngeal wall has an effective method for treating velopharyngeal insufficiency **(Zohar *et al,* 1991).**

b.Nasopharyngeal stenosis

Nasopharyngeal stenosis represents the most undesirable long-term Uppp complication because it results in significant disability and is extremely difficult to correct **(Macalusa, *et al,* 1989).**

Types of Nasopharyngeal stenosis

Nasopharyngeal stenosis develops within 4 to 6 weeks post-UPPP and can present with various degree of severity, a mild form of this complication is adherence of the lateral aspects of the palate to the posterior pharyngeal wall.

In moderate nasopharyngeal stenosis, only a small central section of the nasopharynx remains open. Symptoms of partial nasal airway obstruction and possibly velopharyngeal insufficiency occurred. The latter is a result of inability of the velopharyngeal sphincter to close successfully because of scar rigidity .The severe form of nasopharyngeal stenosis is characterized by total fusion of the palate to the posterior pharyngeal wall **(Pollo, et al., 1989).**

Treatment of Nasopharyngeal stenosis

Correction of nasopharyngeal stenosis is a formidable task. Techniques include pharyngeal, palatal, and combination flaps, Z-plasty, skin grafting and stenting **(Katsantonis, 1991).**

Laser Assisted Uvulopalatoplasty (LAUP)

Introduction

Laser Assisted Uvulopalatoplasty (LAUP) is a technique using local anesthesia for the treatment of snoring and mild to moderate Obstructive Sleep Apnea Syndrome (OSAS) in an office setting.

Indications

It has been shown that the majority of snorers can benefit from a new technique under local anesthesia, called LAUP, introduced by the author **(Kamami, 1990)**. In the late 1980s, and popularized in USA in 1992, by **Coleman, (1994)** and **Walker, et al., (1994)**. Many authors over the world have published their results with this technique **(Schwimmer, 1994)**.

For diagnosis, patients have pre- and post-operative questionnaires and physical examination (with a flexible fibreoptic nasopharyngolaryngoscope), to refine the selection of LAUP candidates and to characterize their symptoms. When OSAS is suspected, a full-night polysomnography is very important to assess its presence and severity. X-ray cephalometry, or CT of the head should also be carried out if necessary.

LAUP is contraindicated in snoring caused by such conditions as severe OSAS or severe nasal or maxillo-facial problems. In cases of OSAS, it is indicated mainly for the mild and moderate cases.

However, LAUP has a role to play in those patients with severe OSAS who do not respond to Continuous Positive Airway Pressure (CPAP) by modifying the upper airway.

Techniques

This author performs LAUP with a Sharplan CO_2 laser. The power is set at 20 to 30 watts, depending on the thickness of tissue that is to be incised, and is used in a continuous mode. A specific snoring handpiece is used with a spot size of 0.6mm to 3.5mm and a focus of 300mm.

This handpiece has a focus-defocus ring: focus to cut, and defocus to coagulate.

The patient is premedicated with an oral analgesic and anti-emetic. Blood pressure is checked before each session. At the time of the session, the patient is placed in a seated position with the mouth open and wearing eye-protectors. The patient is then given breathing instructions: to take a deep breath and very slowly let it out.

Local anesthesia is then administered using Lidocaine 15% sprayed over the soft palate, followed by an injection of 2 to 5 cc of 2% lidocaine with epinephrine bilaterally at the junction of the soft palate and uvula.
The CO_2 laser can be used in two ways: with the focused beam as a light knife' for performing haemostatic incisions of the velum, or with the Swiftlase for ablative char-free vaporization to debulk tissue. Neither sutures nor dressings are required.

At each of the planned sessions, about 5 to 8 mm of the velum is removed. Extending the incisions much higher into the soft palate will usually result in increased postoperative pain. The 'new-uvula' will gradually assume a more superior position, following each treatment until it reaches the level of Passavent's ridge.

A pharyngeal handpiece specifically designed for this procedure incorporates a 'backstop' and a smoke evacuator. The backstop shields and protects the lateral and posterior pharyngeal wall and the smoke evacuator maintains clear visibility in the operative field. The tongue base is depressed inferiorly with a wet wooden tongue depressor.
Subsequent sessions are basically the same as the first **(Kamami, 1996)**.

LAUP v's UPPP

The CO_2 laser has many advantages over a scalpel in surgery for snoring. Unlike the scalpel, the laser can coagulate, vaporize, or cut the velum, the posterior pillars and the uvula - as a sculptor! It also decreases postoperative oedema and pain, and allows a quicker and better cicatrisation. Because of the haemostatic action of the laser, the procedure can be performed under local anesthesia with minimal bleeding from the vascular tissue of the oral mucosa.

This ambulatory technique is a lesser operation with less morbidity than conventional UPPP. LAUP is better tolerated, allowing patients to return to work immediately following the procedure. There is less tissue swelling resulting in less pain, less scar tissue, and better healing than with the conventional surgical procedure. Bleeding is minimal because of the thermal effect of the laser.

LAUP allows more precise cutting, less tissue loss, and better overall control. It is more attractive to surgeons who have reservations about the traditional UPPP, because of its anesthetic risk and considerable postoperative pain. Limited palatal resection ensures success of operation and avoidance of nasal regurgitation of food or rhinolalia postoperatively.

LAUP may also be useful when UPPP has failed, due to obstruction of the hypopharynx from fatty and redundant tissue on the posterolateral pharyngeal walls.

It is also a good alternative for patients who present with major surgical and anesthesia-related risks. In all cases, healing of the laser-induced wound is faster than after a usual UPPP, the exception being in the cases of alcohol or tobacco abuse. In these cases, the duration of post-operative pain is prolonged **(Kamami, 1996)**.

The CO_2 laser also has some disadvantages: it is an expensive tool; it requires special training for its safe and effective use, some postoperative hemorrhage, local infection, throat dryness and tightness occurred in all patients immediately following surgery; Other complications included oral candidiasis, vasovagal episode, temporary palatal incompetence and temporary loss of taste But its advantages are multiple: LAUP is an ambulatory procedure, under local anesthetic, twice as fast as conventional UPPP **(Lee Jeung, 1994).**

The patient experiences less post-operative pain (i.e. the laser seals sensory nerves) and the laser affords great precision and more rapid healing.

The wound is sterile: no contact with instruments and the beam destroys bacteria as it cuts and vaporizes tissue.

The inflammatory response is delayed in laser surgery, but immediate with scalpel surgery.

CO_2 laser is a vaporizing or good-cutting instrument, rapid and safe, with minimal thermal damage to surrounding tissue, controlled by the duration of exposure (minimized by selecting high-power densities).

Its coagulation effect is limited and depth of penetration is controlled.

Incision, vaporization and coagulation become possible with the same instrument by altering the distance of the handpiece to the tissue.

Its haemostatic effect is confirmed in comparative studies of laser v's knife excision.

Most of the patients studied here felt better after this treatment. However, because subjective improvement is not a reliable indicator of surgical success, it is obvious that clinical improvement is very important for patient satisfaction.

Daytime somnolence is a significant subjective problem. It is present in 3-12% of all the population **(Kamami, 1994)**.

Twenty percent of people over 64 complain of this symptom'. The treatment of daytime somnolence may be very helpful in the prevention of traffic accidents.

Some patients admit to driving poorly or reacting slowly because of daytime somnolence. Driving efficiency is correlated to subjective assessment of vigilance.

After treatment of snoring, there is improvement in daytime somnolence and of the patient's driving.

As in cases of UPPP,snoring and OSAS may also recur after LAUP This is due to velopharyngeal hypotonia secondary to age, obesity, tobacco and alcohol use, excessive consumption of sedative-hypnotic drugs, tranquilizers or untreated hypothyroidism.

If symptoms of snoring or OSAS recur, a second treatment directed at the palate may induce cure. The CO_2 laser is preferred to the use of the Nd: YAG laser in this procedure, because of the low volume of absorption of the CO_2 laser beam in tissue. This property prevents excessive thermal necrosis of the target tissue. An additional advantage of the CO_2 laser is its use as a 'no-touch' technique, thereby eliminating contact with the palate and pharyngeal walls. This property reduces gagging, especially for the hypersensitive individual whose gagging occurs on a psychological basis despite having adequate anaesthesia at the surgical site. By regular and long-term follow-up, and by using LAUP, a significant majority of non apneic snorers can receive an efficient treatment **(Kamami, 1996**).

Conclusion

LAUP is an effective, simple, safe procedure, well tolerated by snorers. It will become a valuable alternative technique to conventional UPPP, with its great potential to reduce morbidity and cost to patients. Popularization of LAUP will require serious training of surgeons and further study, especially in OSAS surgery, which is more difficult to treat, because of the thickness of the soft palate in these patients, periodic polysomnographic controls are necessary to study long time results.

COBLATION RADIOFREQUENCY IN SNORING

There are several causes of snoring, including a blocked nose, large tonsils, floppy tongue or obesity. An ENT Surgeon or a dietician may correct these following assessments. In some patients, the snoring is due to excessive vibration of the soft part of the palate. In the past, these patients have been helped by operations, which have usually included removing a small part of the palate surgically, or with a laser. Unfortunately, these operations sometimes result in severe post-operative pain and a long recovery period. The technique of radiofrequency palatoplasty is associated with several important advantages, including much less post-operative pain, rapid return to work and the ability to perform the procedure under local anesthetic as an outpatient **(Laurence , 2003).**

Radiofrequency techniques

COBLATION RADIOFREQUENCY PALATOPLASTY

Radiofrequency technology is a relatively new method of precise tissue removal carried out at much lower temperatures than usual electro-diathermy or laser. The radiofrequency energy is applied with a narrow 'wand' to the center of the palate muscle and causes a small area of scar tissue, which shrinks and stiffens. This recently introduced procedure reduces the vibration of the palate in patients who have severe snoring.

Technique

The procedure is normally carried out under local anesthetic although a general anesthetic may be given if the patient has an extremely sensitive throat and a tendency to gag. The palate is numbed by a spray and local injection. The special electric probe is then inserted into the soft palate and the current activated for a few seconds. This causes a much-localized removal of tissue with minimal heat damage to adjacent structures. The resulting scar gradually contracts and stiffens the palate during the following weeks. The patient is monitored for a couple of hours after the procedure and then allowed home with a supply of simple pain killers **(Fig 38)**.

The majorities of patients have reported minimal pain following the procedure and have been able to return to normal activities including work within 3 days.

There is naturally a small amount of swelling of the palate shortly after the procedure and this may make the snoring temporarily worse for a few days. Normally the swelling settles quickly after sucking crushed ice.

Results so far have been promising with most patients reporting an improvement in the level of snoring within 8 to 12 weeks. A repeat procedure is sometimes recommended in order to gain maximum benefit.

The long-term results for this technique are obviously still unknown but many patients report benefit lasting for a year or more. The procedure can then be repeated as appropriate **(Laurence , 2003)**.

(Figure 37)

Patient selection Radiofrequency palatoplasty is not suitable for all patients with snoring. Patients usually need to have a video or tape recording of their snoring before undergoing the treatment. This is to help decide if the snoring noise is indeed caused by a floppy palate. Some patients require a detailed overnight sleep study to exclude problems such as 'Obstructive Sleep Apnea Syndrome', in which the patient often struggles to breathe and may hold their breath for a long time. Patients with blockage of the nose or enlarged tonsils will need to have this corrected before the radiofrequency palatoplasty operation can be effective.

Patients require referral from their General Practitioner prior to treatment. Private medical insurance companies will not reimburse the cost of treatment to the palate for snoring.

In general, Radiofrequency procedures of the palate are not covered by medical insurance since they are usually performed to reduce or eliminate snoring (snoring being a non-reimbursable disorder by most medical insurance companies.) Accordingly, it is necessary to pay in advance before having the procedure. We do use Radiofrequency procedures for the tongue, in patients with sleep

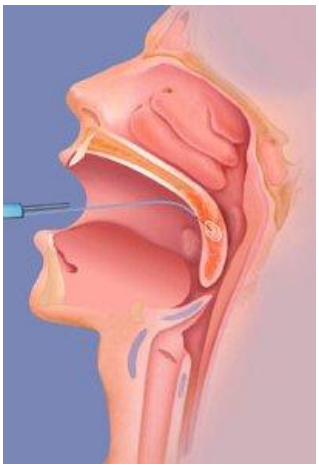
apnea, who have had the appropriate UPP procedure, and continue to have sleep apnea.

Somnoplasty

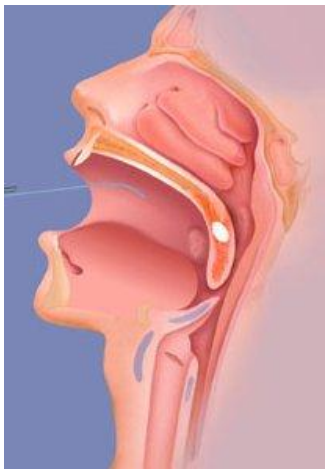
A similar technology is used in the technique known as somnoplasty. A fine electric probe is inserted in the soft palate and the radiofrequency current activated for two or three minutes to cause volumetric reduction in the bulk and stiffening of the soft tissue of the palate.

Coblation turbinate reduction

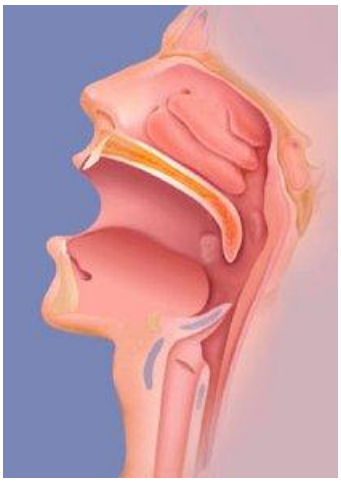
Radiofrequency techniques can also be applied to shrink excessive volume in enlarged nasal turbinates. Under local anesthetic or a light general anesthetic, the radiofrequency probe is inserted deep into the fleshy turbinate and the current activated. The resulting lesion contracts over the following 6 - 8 weeks and leads to a reduction in the turbinate volume with subsequent increase in nasal airflow **Troell (2003).**



Heating tissue with probe



Treated area in palate and uvula forming scar tissue



Palate and uvula decreased in bulk and tightened (8 weeks) Notice that the palate and uvula have less bulk, and the uvula is shorter than in the preceding picture.
(Figure 38)

TRANSPALATAL ADVANCEMENT *PHARYNGOPLASTY FOR OSA*

Indication for Transpalatal Advancement Pharyngoplasty

(1) UPPP failure.
(2) Severe OSA.
(3) Morbid obesity.
(4) Significant anatomic collapse on endoscopic examination of the retropalatal space extending above the margin of anticipated UPPP excision.
(O'Leary and Millman, 1991)

Surgical Technique

Step (1)

Uvulopalatopharyngoplasty is performed in a conservative manner, excising only tonsils, redundant mucosal tissues, and the free portion of the uvula. Care is taken with tonsillectomy to preserve normal pharyngeal structures, which provide blood supply to the palatal flap.

Step (2)

Palatal incision is outlined beginning at the central hard palate just posterior to the alveolus and carried posteriorly, in a curvilinear fashion, immediately medial to the greater palatine foramen. The incision is then flared laterally over the palpable process of the hamulus to the buccal mucosa.

A mucoperiosteal flap is elevated, exposing the hard palate and the proximal soft palate. Elevation is carried superficial to the tensor aponeurosis until the muscular palate is reached. Then, using electrocautery, the soft palate is separated from the hard palate and the nasopharynx is exposed.

Using a mastoid curette, the tensor tendon is exposed and carefully elevated off the hamulus. Using a heavy haemostat, that hamulus is then fractured. This allows mobilization of the central palate, exposing the posterior nasal septum. Removal of I- to 2cm of the margin of hard palate. Palatal drill holes are placed at a 4 5-degree angle to the palate, extending from the oral surface of the palate into the nasal cavity. A large tapered free needle is then used to pass two 2-0 proline sutures through the drill holes into the nasopharynx. Sutures are then grasped, separated, and withdrawn before being secured medially in the tensor aponeurosis and laterally in the tensor tendon.

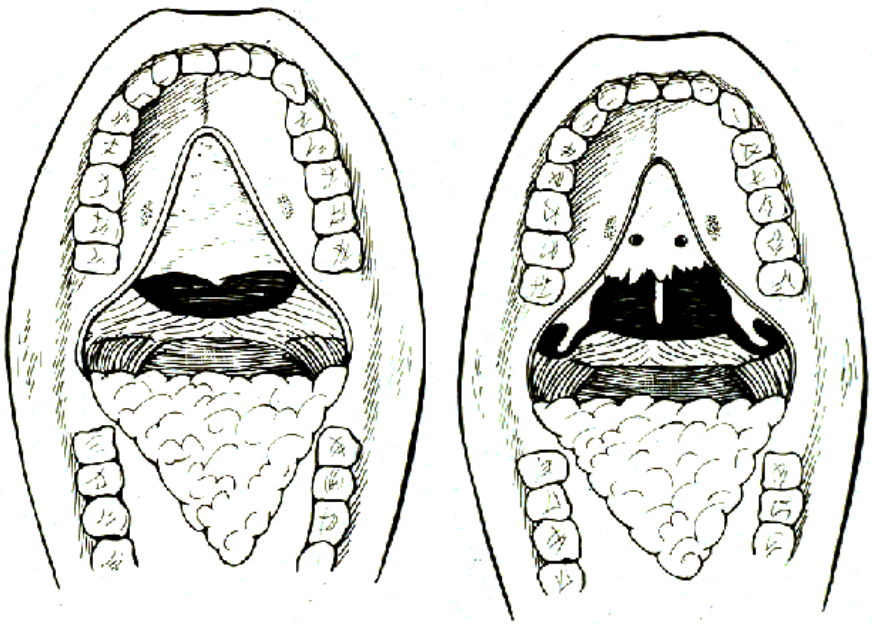
Step (3)

Palatal incision is continued laterally along 'the margin of the soft palate to just above the superior aspect of the palatoglossal folds. These incisions are carried down through mucosa and fibroadipose tissue, but are superficial to the muscular soft palate. A limiting undermining of the flap is performed.

The medial soft palate mucosa and muscle is then pulled forward until adequate position is obtained. A "corner"suture, using 3-0 vicryl, firmly secures this to the posterior alveolar periosteum **(Woodson and Toohill, 1993) (Figure 39).**

Complications

(1) Palatal fistula following partial flap necrosis secondary to early denture wearing.
(2) Serous otitis media.
(3) Oropharyngeal dysphasia with failure of complete pharyngeal clearing.
(4) Complications of UPPP.
(O'Leary and Millman, 1991)



A **B**

(Figure 39)

Figure 39a The gingivae is elevated off of the hard palate to expose hard palate.

Figure 39b Posterior aspect of hard palate is resected and drill holes are placed in hard palate in order to reattach soft palate.

MIDLINE LASER GLOSSECTOMY WITH LINGUOPLASTY

Multiple site-specific procedures have been proposed to treat OSAS. Midline glossectomy (MLG) with linguoplasty is a procedure that directly enlarges the hypopharyngeal airspace using the carbon dioxide laser. It is postulated that since the tongue is a primary determinate of hypopharyngeal collapse, a direct approach of reducing tongue-base size and shape could be successful. By excising a midline portion of the tongue base and removing excessive tissues that are often present, such as redundant lingual tonsils, respiratory function can be corrected with hypopharyngeal space widening **(Fujita *et al.*, 1991).**

Surgical Procedure

The procedure is performed end orally under general anaesthesia through temporary tracheostomy.

Sharplan's 1100 CO_2 laser unit attached to a Zeiss microscope is used, with power set at 35 watts for the tongue and 100 watts for the larynx.

The patient is placed in the tonsillectomy position, and a Crow-Davis mouth gag is applied to allow exposure of the tongue dorsum and base. Outlining the area to be excised with methylene blue. Vaporization of the tongue base is begun at the midpoint of the tongue with a width of 2.5cm and a length of 4 to 5 cm extending toward the tongue base in rectangular fashion. Vaporization of the tongue base until the vallecula is reached. The mouth gag is withdrawn and Dedo's laryngoscope is introduced to expose the tongue base.

The area to be removed at this stage is individualized and may include the remaining tongue base, hypertrophic lingual tonsils, redundant aryepiglottic folds, and collapsible epiglottis **(Parker, 1992).**

The original technique of MLG was improved by including additional steps to excise the remaining posteromedial aspect of the tongue base by wedge section approximating each edge of the wound of the tongue base (**Fujita *et al.*, 1991).**

HYOID ADVANCEMENT AND HYOID DISTRACTION

This new technique has been used to bring the hyoid bone forward in order to advance the hypopharyngeal tissues at the level of the inferior base of the tongue and vallecula. These procedures have been described by other authors using permanent suture or Mersilene ribbon to bring the hyoid to the mandible, as well as to bring the hyoid inferiorly down to the thyroid cartilage **(Ramirez, SG and Loube, 1996**).

This same screw technology is being investigated currently for application for these procedures as well. Using a simple midline stab incision below the mentum, the screw can be inserted easily into the posterior aspect of the mandible and a small incision made over the hyoid and then dissected down to the hyoid bone itself. Muscle attachments to the anterior, superior, and inferior portion of the hyoid then are separated and the suture is passed with the passing instrument subcutaneously down to the level of the hyoid bone. The suture then may be passed either around the hyoid bone and tightened, or it may be passed through the musculature superior to the hyoid bone and then tightened in the method of Krespi (Figures. 40 and 41).

A variation on this technique is the hyoid distraction that is being developed and investigated by Tucker Woodson. In this procedure the hyoid is split in the midline and sutures are placed on the posterior aspect of the mandible at I cm off midline. The suture then is passed subcutaneously in and around the segment of the hyoid on each side and then tied down. This will distract the hyoid not only anteriorly, but laterally as well, with the intention of not only opening the hypopharyngeal airway anteriorly, but also laterally (Figure 42).

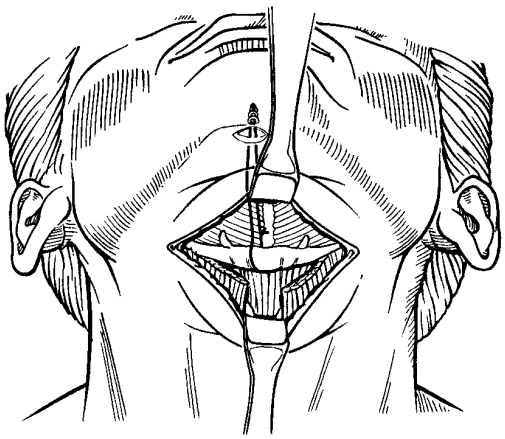


Figure 40 One method of suspending the hyoid is to pass the suture around the hyoid and pull it superiorly and anteriorly.

These procedures at this point are still under investigation. It is hoped that they will be successful in helping those patients with obstruction of the airway at the hypopharyngeal level.

As with most of these surgical procedures for sleep apnea, these will not replace the proven procedures but rather, one would hope, they will be important and acceptable alternative choices in properly selected patients. At this time, the authors' experience with these procedures has been very promising, and results in a very small number of patients have been equally

Successful compared to other standard procedures, but it is still early to stay whether or not these results will hold up over time

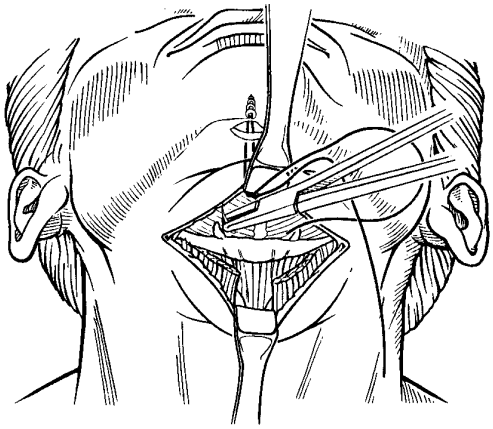


Figure 41 Krespi's method is to pass the suture through the musculature superior to the hyoid then tie the suture down the hyoid then tie the suture

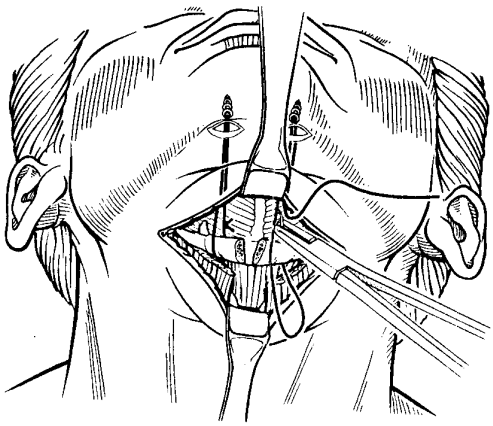


Figure 42 In the hyoid distraction procedure, the hyoid bone is split and two separate of suture are used to pull the bone not only anteriorly and superiorly but also laterally

PATIENTS AND METHODS

Our study was planned to study the relation of OSA and cardio-pulmonary disorders and the effect of management of OSA on this dysfunction.

Seventy (70) patients were selected from patients with OSAS and had any history cardiac manifestations, from ENT clinic of Benha university hospital and Shebien Al kom teaching- hospital during period from March 2001 to October 2004.
Patients were classified into two groups according to the site of obstruction.

*First group (**Nasal group**): Thirty patients all had nasal obstruction secondary to a deviated septum, nasal polyps, hypertrophy of turbinates and or nasal valve obstruction. No patient had narrowing of the lateral dimension of the oropharynx to 2 cm or less, as measured between the midpoints of each anterior tonsil pillar.

The type of operation done in the first nasal group as follow:

Submucous resection with partial inferior turbinectomy in 6 patients (20%). Septoplasty with partial inferior turbinectomy in 12 patients (40%).Nasal polypectomy with middle meatal antrostomy by endoscope in 8 patients (26.7%). Nasal polypectomy with septoplasty and adenoidectomy in 4 patients (13.3%).

*Second group (**Oropharyngeal group**): Forty patients with oropharyngeal obstruction (A long soft palate, narrow inlet to the nasopharynx, hypertrophic tonsils and redundant lateral pharyngeal mucosa.

In the second Oropharyngeal group, half patients (20 patients 50%) had done Coblation of the redundant soft palate and uvula with tonsillectomy if marked enlargement ,however with mild to moderate enlargement of tonsils, Coblation-Channeling Technique was done to reduce the size of tonsils **(subgroup- a-Coblation)** and another half (20 patients 50%) had traditional Uvulopalatopharyngoplasty **(subgroup- b-UPPP).**

*A control group: Other 10-nonapneic patients not complain of cardiopulmonary problems and with the same age and body mass index of our study.

The following studies were performed to every patient was subjected to the following: -

1. Medical History

A full medical history with special attention to the following symptoms:-

a. Snoring

When the patient was interviewed or his relatives, notation was made about consistency of snoring (every night, every position) and intensity of snoring (soft gurgle, blasts of heroic sounds).

Snoring was graded as follows **(Mayo, 2002):**

Grade 1: Heard only if you listen close to the face
Grade 2: Heard in the bedroom
Grade 3: Heard just outside the bedroom with the door open
Grade 4: Heard outside the bedroom with the door closed

b. Apnea

This is interruption of breathing (pauses, gasps and snorts).
The apneic periods lasts generally 10 to 60 seconds.
Apnea was grade as follows

Grade 0: No apnea noted.
Grade 1: occasional mild apnea or apneas occurring when sleep back only.
Grade II: Some episodes of apnea in any position.
Grade III: OSA in all positions.

C. Excessive Daytime Sleepiness (EDS)

A (yes - no) question about daytime sleepiness was often unrevealing because of the tendency of many males to deny or ignore its presence. More information is discovered by questions about feeling refreshed upon awakening, drowsiness on the job, or difficulty staying awake while driving a motor vehicle or watching a television show.

The Epworth sleepiness scale (ESS) is a self-administered questionnaire, which provides a measurement of the patients' general level of daytime sleepiness. ESS scores increase with severity of OSA and are more significantly correlated with the apnea index and respiratory disturbance index than the degree of hypoxaemia. Normal patients score between 2 and 10 whereas total ESS scores greater than 16 are found only in patients with OSA **(John, 1991).** Excessive daytime sleepiness is predominantly a subjective symptom. It may be used when the excessive daytime sleepiness suggests narcolepsy. The Epworth Sleepiness scale is a popular, quick and easy self-administered questionnaire that asks patients their likelihood of falling asleep in 8 situations ranged from 0 (would never doze) to 3 (high chance of

dozing). The numbers are then added together to give a global score between 0 and 24. A value of 10 or below is considered normal.

The eight situations are as follows:

1. Sitting and reading
2. Watching TV
3. Sitting inactive in a public place, e.g., theater
4. As a passenger in a car for one hour without a break
5. Lying down to rest in the afternoon when circumstances permit
6. Sitting and talking to someone
7. Sitting quietly after a lunch without alcohol
8. In a car, while stopped for a few minutes in traffic

Epworth Sleepiness Scale

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

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

Situation	Chance of dozing
Sitting and reading	_____
Watching TV	_____
Sitting inactive in a public place (eg, a theater or meeting)	_____
As passenger in a car for an hour without break	_____
Sitting and talking to someone	_____
Sitting quietly after lunch without alcohol	_____
In a car, while stopped for a few minutes in traffic	_____

(Redrawn from Johns, MW, Sleep 1991; 14:40.)

The Epworth sleepiness scale (ESS)

(Figure 43)

d. Headache

Especially in the morning headache which is usually due to fragmentation of sleep.

e. Personality Changes

The social effects of OSA can be devastating. Disruption of interpersonal relationships, marital sterility, and marriage dissolution can be occurred. Nervousness, irritability, Depression and decrease libido.

f. Ingestion of Sedatives

Ingestion of sedatives increases the rate of apnea and its duration.

g. Cardiovascular Symptoms

History of admission to the hospital due to cardiac disease, history of hypertension, chest pain, palpitation, dyspnea, cough and edema of the lower limbs may be indicator of serious cardiovascular problems.

2. General Examination

A full general examination were done for every patient and stressing on:-

a. Body weight

Overweight and obese patients were more susceptible for complications of OSA and cardiovascular problems.

Body mass index (BMI) calculated by weight in kg / height in meters 2, for men BMI should be less than 27.8 and for women BMI should be less than 27.3.
An increase of one standard deviation in any measure of body habitues is related to a threefold increase in the risk of having an apnea index of 5 or greater **(Young et al., 1993).**

b. Heart Rate

Examination of heart rate was done for every patient before and after surgery. Patient stays the night before surgery in the hospital and measuring of the pulse rate during sleep were done. After 8-12 weeks, heart rate was examined again.

c. Blood Pressure

Examination of blood pressure was very important to detect the hypertensive patients and their improvement after surgery. Pre- and post-operative (8-12 week) measurement of blood pressure was done during sleep for every patient. Hypertension was defined if patients were receiving anti-hypertensive medications without regard to the actual measurement of BP, or having a systolic BP >/= 140 mm Hg or a diastolic BP >/= 90 mm Hg (**Whelton, et al 2004).**

d. Cardiopulmonary Examination

Examination of the chest and the heart for every patient by specialized cardiologist occurred before and after surgery.

3. Otorhinolaryngological Examination

A complete otorhinolaryngological examination were done to all patients and stress on the anatomical abnormalities which obstruct the upper airway passages to classify the patients of OSA into two groups according to the site of obstruction.

***Nasal causes**	*** Velopharyngeal causes**
-Nasal deformity	- Enlarged adenoid
-Deviated Nasal septum	-Redundant oropharyngeal tissue
-Hypertrophy of turbinates	- Redundant lateral pharyngeal wall
-Nasal polyps	-Enlarged tonsil
-Others	-Others

a. Nose

Nasal abnormalities on physical examination were noted, nasal obstructive abnormalities might be one of the following: -

(a) Nasal septal deformity.
(b) Nasal turbinates hypertrophy.
(c) Nasal polyps.
(d) Nasal swellings.

b. Pharynx

The relative size of the oropharynx of the patients were used a measure for selection of UPPP candidates. Thus, a patient with large drooping soft palate, elongated floppy uvula, redundant lateral pharyngeal walls and large hypertrophy of tonsils was indeed to be a good candid. The pharyngeal examination often shows the following: -

i. Redundant oropharyngeal tissues

These may include the following: -

(a) Large edematous uvula
(b) Wide posterior pillar mucosa (web formation).
(c) Redundant mucosal folds of the lateral and posterior pharyngeal wall, which may extend from the nasopharynx to the hypopharynx

ii. A low palatal arch with low-hanging soft palate

In such cases, the free margin of the soft palate cannot be seen in the open mouth without using a tongue blade. It may be not seen even on phonation, which usually will lift the soft palate. This anatomical variant may contribute to the narrowing of the pharynx and facilitate airway collapse because a simultaneous hypotonia of the pharyngeal dilator muscle.

iii. Large tongue

The tongue may be large relative to the oral cavity, either the dorsum of the tongue, with a relatively patent lower supraglottic area or the base of the tongue. In the later case, the posterior positioned tongue may make it difficult or impossible to see the larynx with a mirror.

iv. Floppy epiglottis

An omega shaped floppy epiglottis with redundant aryepiglottic folds is occasionally seen.

v. Hypertrophic tonsils

Hypertrophic tonsils reduced the dimension of the pharynx.

vi. Redundant lateral pharyngeal walls

Redundant lateral pharyngeal walls secondary to excessive submucosal fat infiltration or hypertrophied musculature-

4. Fiberoptic nasopharyngoscopic examination

A Fiberoptic nasopharyngoscopic examination was performed to all patients during awake and sleeps

Technique

Nasopharyngoscope (during awake)

While the patient was sitting in an examining chair ،one of the nasal cavities was anaesthetized with topical application of 4% xylocaine. The pharyngeal mucosa was anaesthetized by 10% xylocaine oral spray solution. The pharyngoscope was passed through the nasal cavity into the pharynx and advanced into the hypopharynx. Its tip was then positioned at the lower oropharynx so that the entire hypopharynx could be visualized. The patient was instructed to close his mouth and vigorously inhale while the examiner occluded the nostrils (this procedure termed Muller's maneuver).

The degree of hypopharyngeal collapse was recorded as a percentage of the entire hypopharyngeal cross-section area .This maneuver was repeated several times, the endoscope was then retracted so that the tip would be positioned just cephaled and posterior to the free edge of the soft palate.

In this position, the velum as well as the entire oropharyngeal lumen is visible. The Muller maneuver was again repeated several times, and the degree of oropharyngeal collapse was noted and recorded.

The patients were classified according to **Fujita (1981)** into three groups or types (Table 2) based on upper airway anatomical findings: -

Type (1): in which the airway narrowing predominantly involved the oropharynx, but the palatal arch is in normal position. Patients in this group were characterized by large hypertrophic tonsils, enlarged uvula, and webbing of the pillars mucosa.

Type (II): in which the palatal arch is in a low position and this group was divided into two subgroups, depending on the level of predominant airway narrowing:

- ll a: predominantly involved the oropharynx only.
- ll b: involved both the Oro -and hypopharynx.

Type (III): The airway compromised was limited to the hypo pharynx only and the oropharynx is normal. This compromise include, a large tongue base, lateral wall bulge and hypertrophic lingual tonsils.

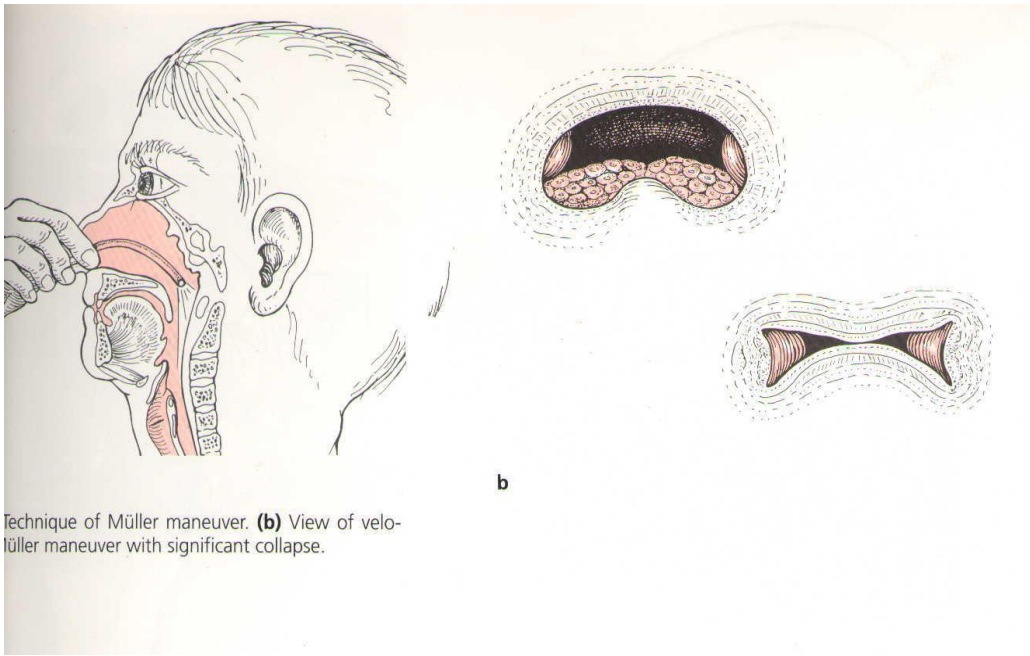
-

Type	Site of Obstruction
I	Oropharyngeal with normal palatal position
II ll.a ll.b	Low palatal position: Predominantly oropharyngeal Oro-hypopharyngeal
III	Normal oropharynx with hypopharyngeal obstruction

Table (2)

Upper airway anatomical classification by Muller's maneuver.

In this study, UPPP was done to those patients demonstrating only oropharyngeal obstruction that can be documented with oral inspection and fiberoptic nasopharyngoscopy.



Technique of Müller maneuver. **(b)** View of velo-
lüller maneuver with significant collapse.

Muller's maneuver
(Figure 44)

Nasopharyngoscope (during sleep)

Procedure

Start IV induction of sleep by infusion of propofol (diprivan) 2-2.5 mg/kg, and wait until full effects.**(Diprivan)** maintain UA reflexes, rapid induction and recovery. Premature induction may induce irritability and cardiac irregularities, so titration by snoring dose (sleeping) of the drug, then introduce the endoscope slowly to avoid bleeding.

Record data during introduction until the larynx is seen then withdraw the scope slowly and wait for 2-3 respiratory efforts

Stations

Visualize the epiglottis to check for supraglottic collapse.

Go above the level of tongue base and check the tongue base and lateral wall collapse.

Go at the level of the oropharynx and check for the anterior and lateral wall collapse.

Go in the nasopharynx and check for soft palate and lateral wall collapse

5. Laboratory investigations

Routine laboratory studies were done for all patients.
(CBC, Blood sugar, Liver and Kidney functions, ESR, Bleeding, Clotting times)
Investigations for hypothyroidism as T3, and T4, were done to exclude the effect of the thyroid hormones deficiency on sleep apnea in suspension patients.

6. X-ray lateral view for head and neck

Lateral x-ray for head and neck region was done.
The following findings may be present in the patients of OSA:-

(a) Their tongue and soft palate are significantly enlarged.
(b) The hyoid bone is displaced inferiorly.
(c) The mandible is normal in size and position (no micrognathia or malocclusion), but the face is elongated by an inferior displacement of the mandibular body.
d) The maxilla is retro positioned and the hard palate elongated.
(e) The nasopharynx is normal, but the oropharyngeal and hypopharyngeal airway is reduced in area by an average of 25%.

7. X-ray chest

X-ray chest was done to exclude lower obstructive airway lesions and cardiopulmonary diseases.

8. Arterial blood gases

Two samples of arterial blood has been obtained from all patients, the first sample before operation at night during sleeping and the other sample was taken after 8-12 weeks post-operatively. These samples were investigated for SaO_2, and $PaCO_2$.

Arterial Puncture

Getting arterial blood gases by a needle is painful and lead to iatrogenic changes in Pa CO_2, due to hyperventilation related to pain and to avoid this error an indwelling arterial catheter size 20 and 22 inserted in the patients under local anesthesia using xylocaine HCI 1% in order to get normal value when the patient was sleeping. Also this assisted **by pulse oximeter** to measure the nocturnal arterial oxygen saturation (SaO_2).

9. Electrocardiography (ECG)

ECG recording were done for all patients during sleep before the operation and 8-12 weeks after surgery. A separate ECG lead II was also continuously recorded to detect any arrhythmias or ectopics before and after surgery by 8-12 weeks.

10-Echocardiography Doppler examination

Doppler Echocardiography (DOP) was used to asses the cardiac functions by measuring these items:

LVEDD (left ventricle end diastolic dimension**)**
LVESD (left ventricle end systolic dimension)
EF% **(**ejection fraction**)**
FS% **(**fraction shortening**)**
R.V **(**Rt. ventricle diameter**)**
LA, AO **(**Lt. Atrium, Aortic root)
E/A Ratio **(**the ratio between the early peak of the transmitral flow velocity and the late peak atrial systolic velocity**)**
PASP (pulmonary artery systolic pressure)
Echocardiography done before and after surgery by 8-12 weeks

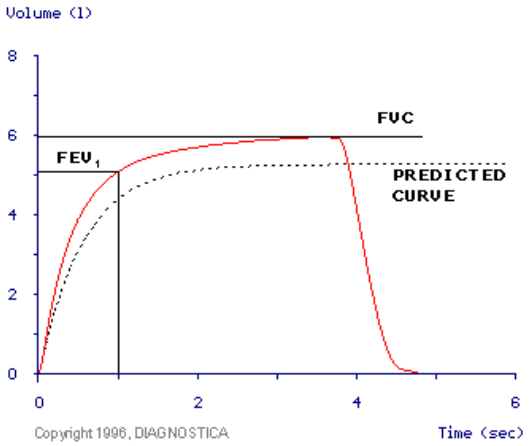
11. Pulmonary function test (Spirometry)

The measurement of forced expiratory flow rates by Spirometry is the most commonly used pulmonary function test and decreased as the OSAS severity increased. Spirometry is often performed in an office sitting. These items are measured in Spirometry

All parameters, except the VC, are based on a forced expiration and / or forced inspiration (Flow-Volume loop).

The following parameters describe the Volume-Time graph

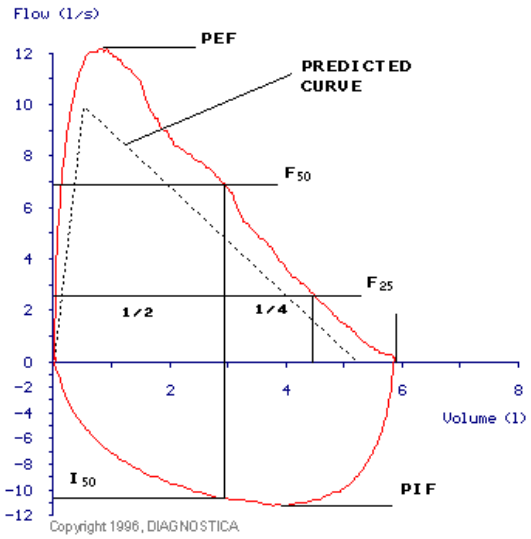
- **VC**
  Vital **C**apacity: The total volume (liter) of an unforced, but complete expiration following a full inspiration.
- **FVC**
  Forced **V**ital **C**apacity: The total volume (liter) of a forced expiration.
- **FEV_1**
  Forced **E**xpiratory **V**olume in the **1**st sec. of a forced expiration.
- **FEV_1%**
  Forced expiratory rate: The ratio FEV_1 / FVC in %.



(Figure 45)

These parameters describe the Flow-Volume graph

- **PEF**
  Peak **E**xpiratory **F**low (l/min or l/sec) during forced expiration.
- **F_{50}**
  Expiratory **F**low (l/sec) when **50**% of FVC remains. (Also called MEF_{50})
- **F_{25}**
  Expiratory **F**low (l/sec) when **25**% of FVC remains. (Also called MEF_{25})
- **MEF**
  Average expiratory flow (l/sec). (Also called $FEF_{25-75\%}$)
- **PIF**
  Peak **I**nspiratory **F**low (l/min and l/sec) during forced inspiration.
- **I_{50}**
  Inspiratory flow (l/sec) when **50**% of FVC is inspired.



(Figure 46)

(Spirometry) done before and after surgery by 8-12 weeks

12. Polysomnography

Overnight Polysomnography was performed to some patients in Sleep Research Unit; Ain shams University, Aldemardash hospital (Sleep lab department) using standard electro-oculogram (EOG), and submental electromyogram (EMG) monitory for sleep staging. Respiration was monitored using chest and abdominal impedance plethysmography and surface intercostal EMGs. Airflow was assessed with oral and nasal thermistors, and arterial oxygen saturation was thermistors, and arterial oxygen saturation was monitored using continuous pulse oximetry. Baseline arterial oxygen saturation measures while the patient was awake. Nadir arterial oxygen saturation levels were obtained from the polysomnogram. Heart rate and rhythm were monitored with continuous electrocardiogram (ECG) recording. Nocturnal myoclonus was monitored using bilateral tibialis EMG leads. Obstructive apneas were defined as a cessation of airflow for > 10s with continued chest and abdominal efforts. Obstructive hypopneas were defined as a decrease in airflow of at least 50% associated with at least a 4% decrease in O_2 sat. The apnea/hypopnea index (no. of apneas and hypopneas per hour) was used to diagnose OSA.

The study was considered negative if the apnea/hypopnea index (AHI) was < 5 and positive if the AHI was > 10:

Operative Technique

A-Traditional Uvulopalatopharyngoplasty is performed under general anesthesia with the patient in tonsillectomy position (The patient is placed supine with sandy bag under the shoulders and a horseshoe to support the head, which is extended). The oropharynx then exposed with a suitable self-retaining Boyle- Davis mouth gag.

The uvula was grasped with a non-toothed forceps and pulled antero-superiorly. The area on the oral surface of the soft palate produced by this maneuver was used for making a horizontal incision through the mucosa of the soft palate. The incision was extended laterally and downward to the base of the tongue, parallel to the free edge of the soft palate and palatoglossal arch. Submucous dissection with scissors and/or cutting diathermy was carried out towards the free edge of the soft palate.

The amount of the soft palate to be resected is determined (the attachment of the levator palatini makes a dimple in the palate that can be seen preoperatively and the resection line should be below this dimple and also can use preoperative nasendoscopy for estimating the amount of excess of the soft palate).In addition, in the anaesthetized patient the palate can be pushed posteriorly at its midsection until it meets the posterior pharyngeal wall.

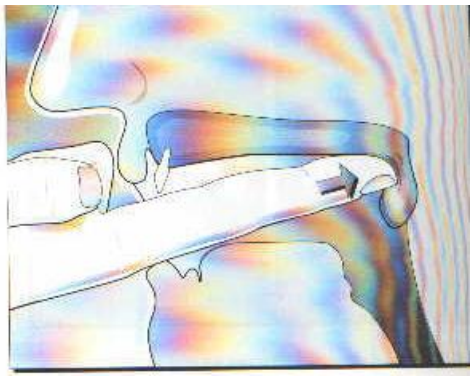
This point of contact with the pharyngeal wall is noted and the incision is started below this point and usually about (1-1.5 cm) at the midline, not including the length of the uvula.

The musculus uvula was transected and the palatine tonsils (if present) were dissected and removed together with the mucosa of the posterolateral pharyngeal wall as a part of the specimen.

Resection of the entire anterior tonsillar pillar, leaving just enough tissue for suturing later on, and bleeding is controlled with electro coagulation.

The remaining posterior pillar is then advanced forward to meet the resected edge of the anterior pillar. This stretching should flatten out the vertical fold redundancy of the posterior pharyngeal wall mucosa and smooth it out. In addition, if this does not occur, we resect more of the posterior pillar mucosa.

The pillars are then advanced and sutured together by 3/0 silk sutures.



Judging the amount of the soft palate to resects

(Figure 47)

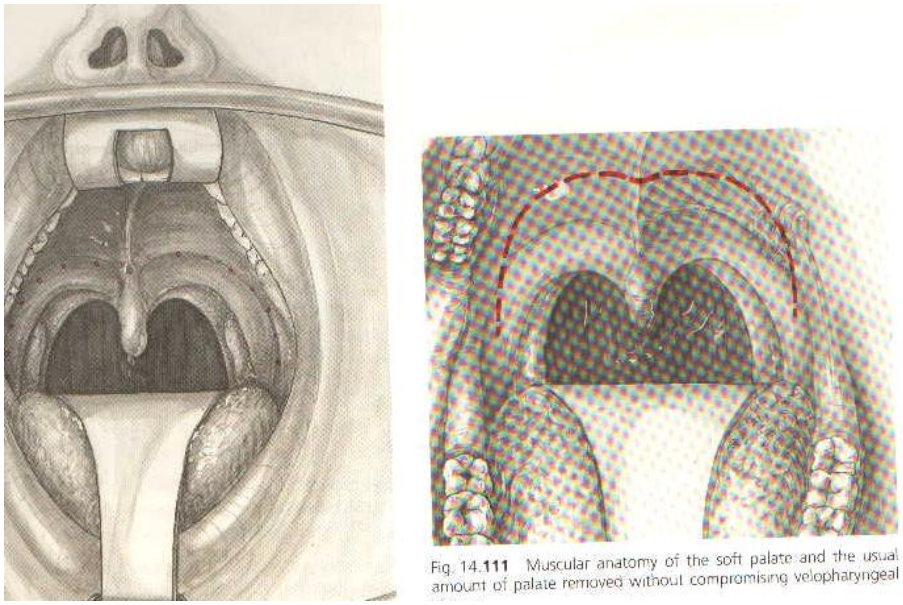
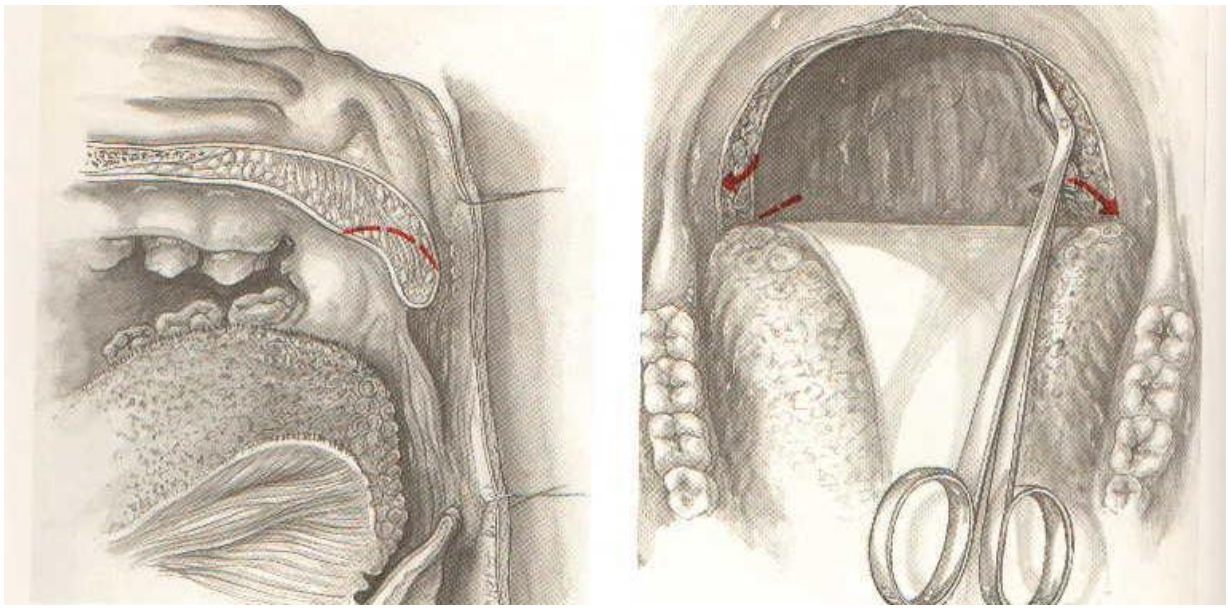
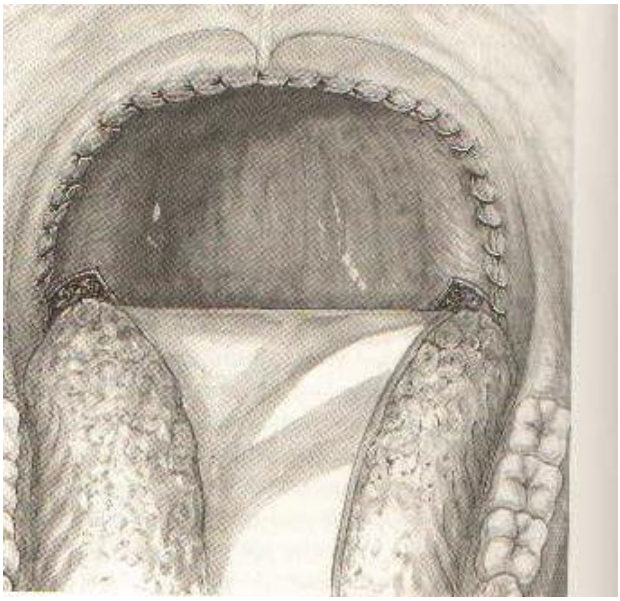


Fig. 14.111 Muscular anatomy of the soft palate and the usual amount of palate removed without compromising velopharyngeal

Making the landmark of incision with diathermy (Figure 48)

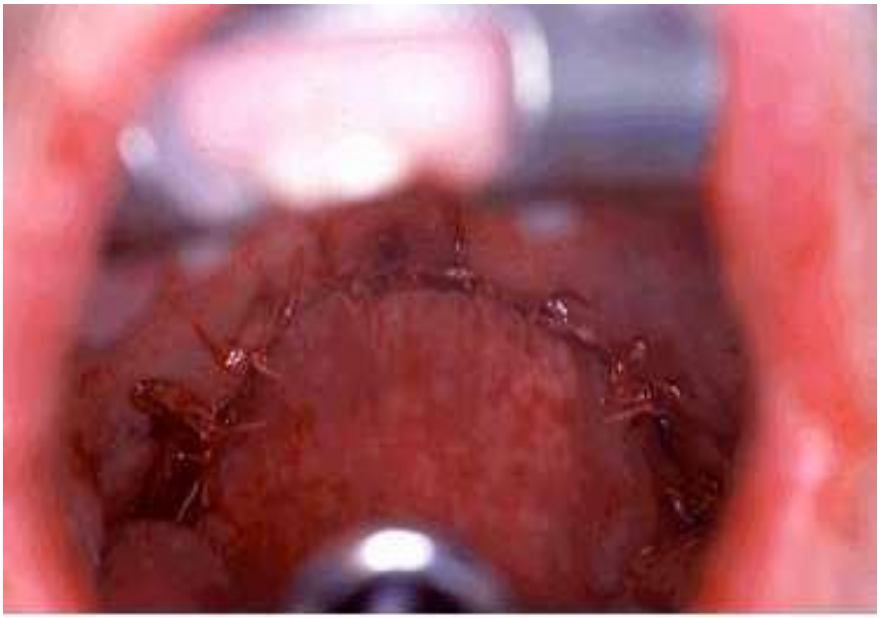


Beveling of incision to preserve nasopharyngeal mucosa
(Figure 49)

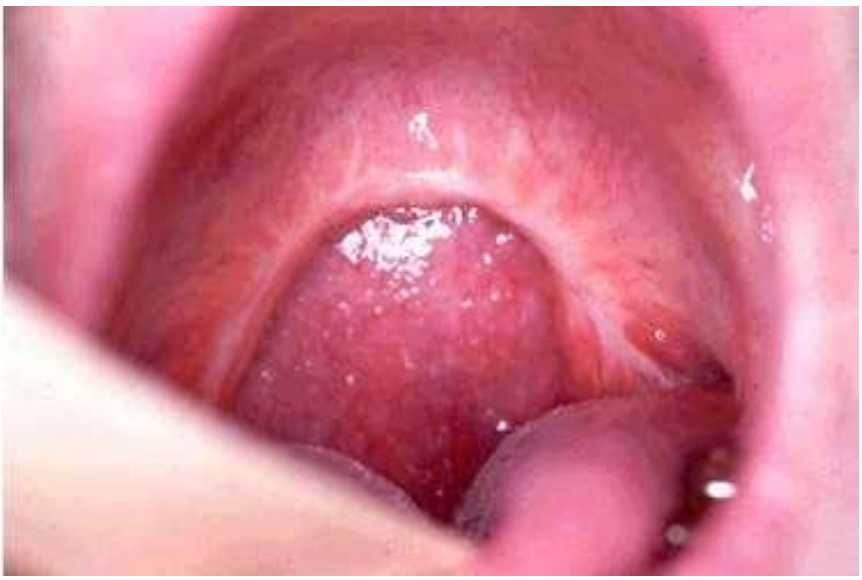


Back cut inferiorly on posterior pharyngeal wall mucosa allows rotation and advancement of mucosal flaps

(Figure 50)



(Figure 51a) Intraoperative photograph on completion of Uvulopalatopharyngoplasty (UPPP)



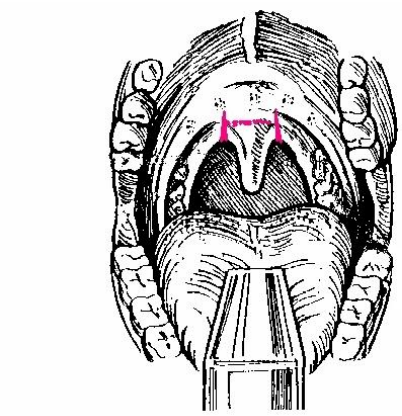
(Figure 51 b) Photograph of the Uvulopalatopharyngoplasty (UPPP) 3 months later.

(Figure 51)

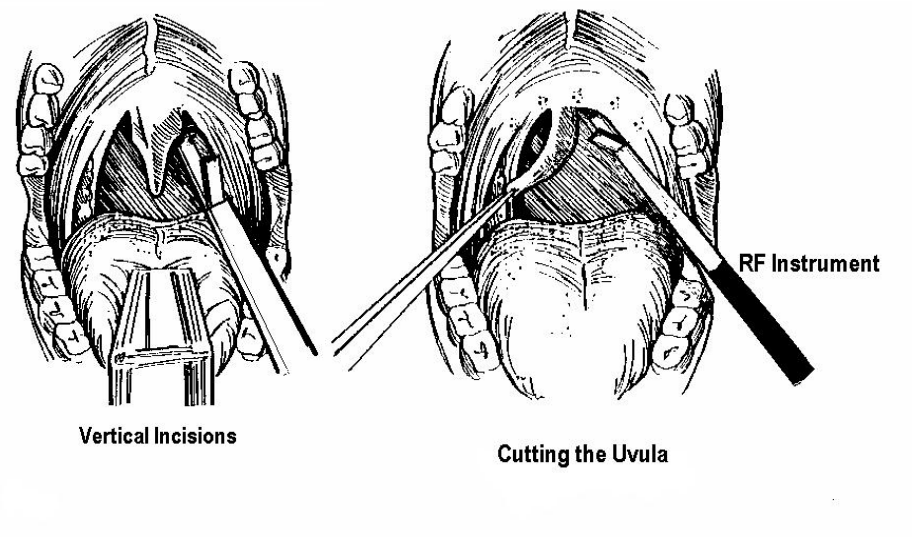
B-Coblation ablation technique

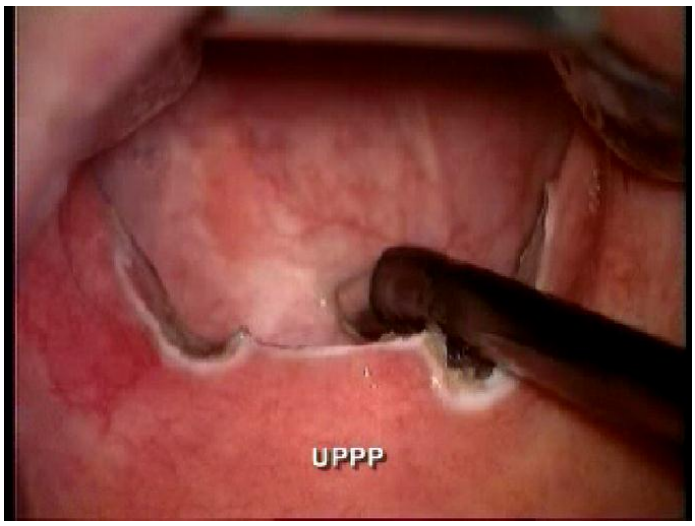
- **Bipolar Radiofrequency Ablation (Coblation):** by The ENTec Coblator **(Figure 52)**, this procedure produces an ionized saline layer that disrupts molecular bonds without using heat. As the energy is transferred to the tissue, ionic dissociation occurs. This mechanism can be used to remove all part of the tonsil with the uvula and the redundant soft palate. It is done under general anesthesia in the operating room .This causes removal of tissue with a thermal effect of 45-85 C°. The advantages of this technique are less pain, faster healing, and less post operative care**.** Special recently Evac probes were used for cutting and ablation of the redundant palate and uvula with tonsillectomy if marked enlarged. A special RF electrode is used to make two vertical cuts on either side of the uvula. These are joined by a horizontal cut and the uvula is removed with tonsillectomy **(Figure 53)**.
- However, with mild to moderate enlargement of tonsils Coblation-Channeling Technique was done. A special RF electrode (Reflex Ultra 55 Wand) is inserted through the incised area, 3 – 5 downward channels are made in each tonsil at a power setting of 5 or 6 for 10 - 12 seconds. It is important to note that the direction of the electrode should be strictly vertical, thus avoiding getting closer to the latero - posterior pharyngeal wall than 1/2 cm. This is necessary to avoid vessels and nerves. Channeling is performed as far down in the tonsils as can be performed in a controlled manner **(Figure 56)**.

The ENTec Coblator
(Figure 52)

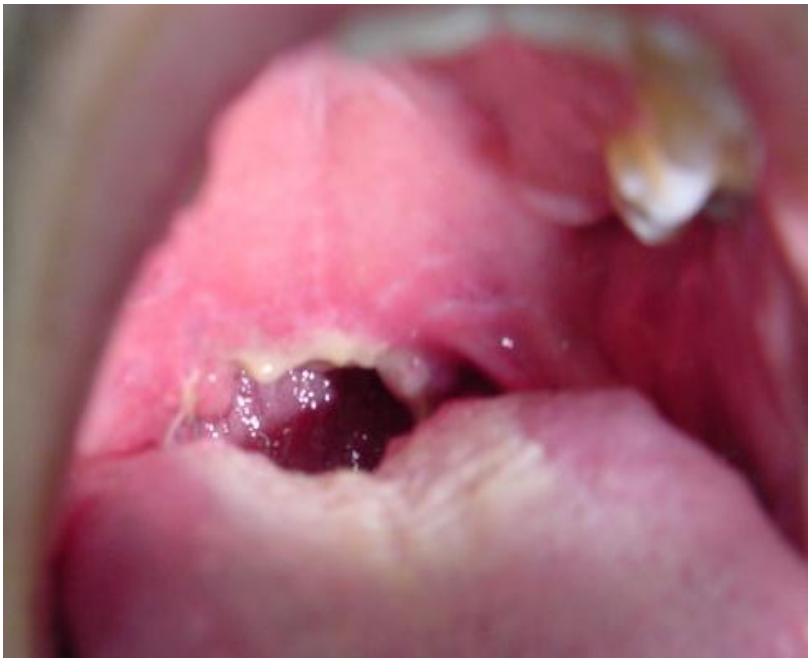


2 Vertical and 1 Horizontal Cuts

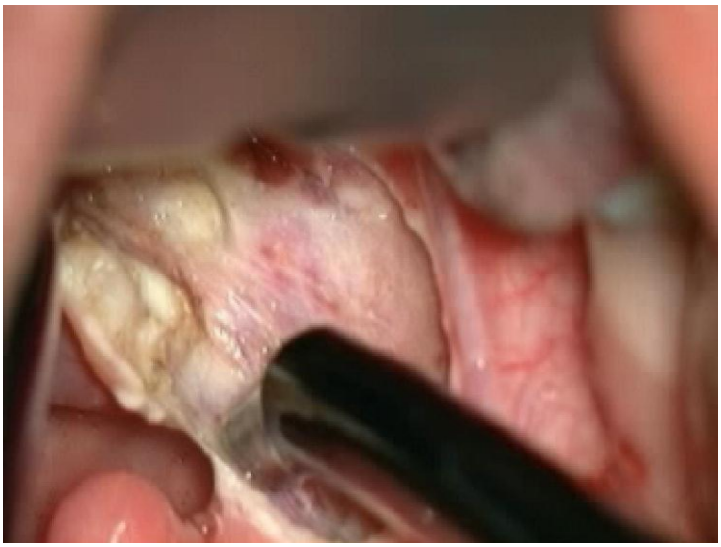




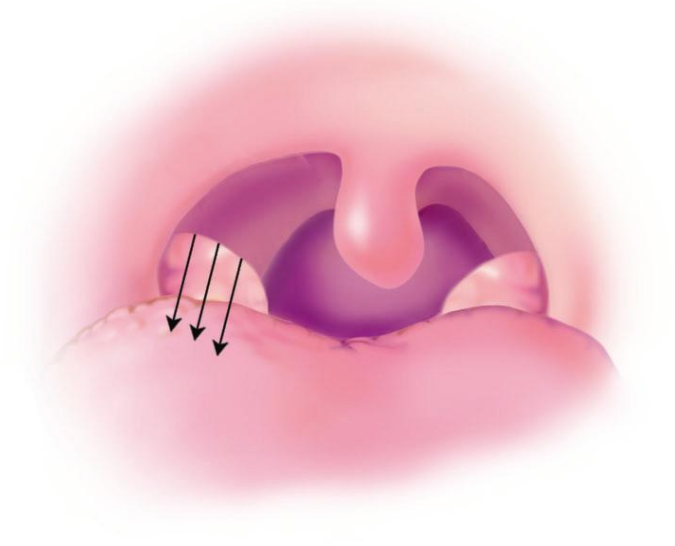
UPPP by Coblation (Figure 53)

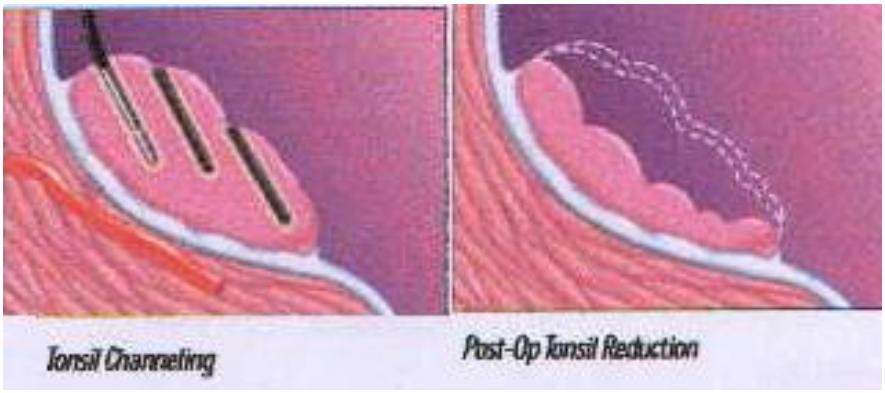


Radiofrequency- Uvulopalatopharyngoplasty (10 days post-op.)
(Figure 54)



Tonsillectomy by radioablation
(Figure 55)





Tonsil channeling (Figure 56)

Post-Operative Care

The post-operative care is like to that of a tonsillectomy. Intravenous or intramuscular antimicrobial prophylaxis is maintained for 5 days, and if swelling or edema begins to be apparent in the operating room or early in the post-operative period, a short course of steroids is given for 3 day. In OSA patients, pain medication is given more sparingly especially with Uvulopalatopharyngoplasty *i.e.* low-dose parenteral pethedine) with the recognition that apnea is aggravated by narcotics. The patients started with a soft diet, patients should be encouraged to eat and drink as soon as possible. Sleep apnea patients should spend at least the first postoperative night on a high dependency unit.

Discussion

Obstructive sleep apnea (OSA) is a complex neuromuscular syndrome with potentially serious respiratory and cardiovascular consequences **(Gilleminaut et al *1983).*** The obstructive sleep apnea-hypopnea syndrome (OSAHS) is characterized by repetitive upper airway obstructions during sleep, and it might cause cardiovascular complications such as myocardial infarction, arrhythmias, and systemic and pulmonary hypertension **(Dursunoglu et al 2005)**.

Marin et al (2005) found th**e** death from (myocardial infarction or stroke) and non-fatal cardiovascular events (non-fatal myocardial infarction, non-fatal stroke, coronary artery bypass surgery, and percutaneous transluminal coronary angiography).As follow: 264 healthy men, 377 simple snorers, 403 with untreated mild-moderate obstructive sleep apnoea-hypopnoea and 235 with untreated severe disease.

Nasal obstruction may contribute to sleep apnea. Several pathological conditions giving rise to nasal obstruction often require surgical treatment such as: septal deformities, nasal polyps, and choanal atresia, inferior turbinate hypertrophy, and sinusitis **(Friedman, et al 2000).**

Over the past years, UPPP has become the most common surgical treatment for Oropharyngeal OSA. The purpose of UPPP is to increase the space between the soft palate, tonsillar fossae and posterior pharyngeal wall in order to decrease upper airway resistance, particularly during sleep. The soft palate is displaced anteriorly and thus prevents occlusion of the airway **(Fairbanks, 1991)**.

Coblation is a new method using radiofrequency to reduce the volume of the enlarged tonsils and redundant palate. This treatment may be associated with a number of surgical and clinical advantages, including better operative results, reduced surgical time and less post-operative pain and bleeding. Radiofrequency treatment has been used in oral and nasal surgery where removing and shrinking tissue is required to correct and improve snoring, nasal congestion and even sleep apnea **(Friedman et al. 2004)**.

This study aims to evaluate the effectiveness of surgical correction of causes of OSAS on the cardiovascular complications that were associated with OSAS.

Seventy patients with OSAS associated with cardiovascular problems were evaluated during the period from March 2001 up to October 2004. Careful history, general examination, Otolaryngological examination and endoscopic examination of the upper airway were evaluated for every patient.

Both pre- and post-operative studies were done for heart rate, blood pressure, electrocardiography and arterial blood gases, pulmonary function test "Spirometry" and Doppler Echocardiography for every patient.

All surveys showed that OSAS was more common in men. **Croft, et al (1990)** comprised 50 patients with OSA, 41 of them were males and 9 females. In addition, **Shamszamman, et al (2002)** studied 22 patients with OSA, 18 males and 4 females.

Peker, et al (2002) found that OSA affect 24 % of middle aged men and 9% of women in the United States.

In this study "in Nasal group of OSA, there were 25 males (83.3%) and 5 females (16.7%), and also in Oropharyngeal group of OSA 29 were males (72.5%) while 11 females 27.5%).

The reasons for the gender difference are not well understood, however, both anatomical and functional correlates in the upper airway can be attributed to that difference. **Brown, et al (1986)** showed that upper airway resistance is greater in men than women are although pharyngeal dimension are also greater in men. Increased resistance in the upper airway required pressure that is more negative during inspiration, which facilitated the upper airway narrowing and the obstruction associated with snoring and apnea. In addition, the increased number of cigarettes smoking and alcohol consumption among men may explain this gender difference.

In addition **Mohsinin (2001)** Showed that OSA is more in males than females and found that airflow limitation in men is due to more complaint upper airway, more pronounced reduction in the pharyngeal caliber in supine position, and greater inspiratory pressure during sleep. Upper airway resistance in female patients changes little during sleep with no significant decrease in inspiratory intraluminal pressure, suggesting less obstructed airflow. Therefore, the pharyngeal size is an important determinant of OSA in men but not in women.

With respect to age, it was noted worthy that apnea prevalence was greatest in the fifth decade and declined in older age group, **(Norton and Dunn, 1985)**.

Craft et al (1990) studied 50 patients with OSA from 29 years to 70 years of age with average age 49 years. **Metes et al (1991)** studied 51 patients with OSA underwent UPPP, the main age was 45 years, S.D. was 10 with minimum age 27 years and maximum age was 67 years.

In addition **Robert, et al (2000)** studied 40 patients with OSA predominantly males (33 out of 40), averaged 46 years of age.

In our study, a similar pattern has been noted, the main age was 41.75 years, with minimum age 30 years and maximum age 55 years in nasal group of OSA patients, and the main age was 42.38 years in Oropharyngeal group of OSA with minimum age 33 years and maximum age 57 years.

Decline in the muscle tone of the pharyngeal dilator muscles as well as reduction in the excitability of respiratory center, which occurs by time, may contribute to such age incidence **(Lugaresi, et al 1988).**

Obesity is a strong risk factor for snoring and sleep apnea, **Pelausa and Tarshis (1989)** reported 84, 3% of their patients were overweight. **Lavie et al (1993)** also reported that obesity constitutes an independent risk factor for both hypertension and OSA.

In our study, statistical correlation analysis proved that there was a highly significant correlation between body weight and OSA. The main weight of patients with OSA in nasal group was 95.15 kg with minimum weight 80 kg, and maximum weight 108 kg. Also in Oropharyngeal group, the main weight of patients with OSA was 93.74 kg with minimum weight 85 kg and maximum weight was 110 kg. Obesity is thought to encourage OSA by a number of mechanisms:

* Excess weight on the abdomen and chest wall decrease lung compliance and lung volume.
* Fatty infiltration in the diaphragm and intercostals muscle decreased the strength of respiratory effort.
* Obesity associated with depression of the genioglossus muscle reflex **(Remmers, et al 1978).**

In addition, the obesity is related to fat deposition in the soft palate and pharyngeal walls has been confirmed by C. T scan **(Harman and Block 1986).**

Snoring, apneas as well as excessive daytime sleepiness (EDS) are the prime symptoms of OSAS, also morning headache, irritability and nervousness are common complaints **(Wetmore, et al 1986).**

The diagnosis of snoring and OSA and their severity are usually based on the subjective impression and complaints of the patients bed partner, family or friends.

Polysomnography is considered the "gold standard" for the diagnosis of sleep disorders, there are no studies assessing the validity of ploysomnography for making a diagnosis of OSA in adults **(Meyewr, et al 1993).** Furthermore, a negative polysomnogram does not conclusively exclude the diagnosis of OSA in adults if there is a high pretest clinical suspicion of the disease. In one report, for example, 11patients who were suspected of having OSA on the basis of obesity, hypertension, or a history of observed apneas during sleep, had a negative result on all-night

ploysomnography.Six of the 11 had a positive second sleep study, during which an increase in the mean AHI from 3.1+ l.0 to 19.8 +4.7 reflected a change from normal to clearly abnormal values. The investigators postulated that the degree of nasal patency, body position, or disruptive environmental factors, all may be important factors in producing this night-to-night variability **(Meyewr, et al 1993).**

However, the waiting lists of ploysomnography in many centers are prohibitory long, and the cost effectiveness sleep study for sleep apnea is questionable **(Pelause and Trashes, 1989).**

Zamarran, et al (1999) using oximeter spectral analysis in the diagnosis of OSA and considered nocturnal oximetry test results were abnormal (suspicion of OSA) if the peak in the spectrum between the period boundaries 30-70s was observed.

In our study selection of patients for polysomnographic, analysis based on nocturnal oximetric evaluation of arterial oxygen desaturation events in-patients of OSAS.

Accordingly, patients with multiple arterial O_2 desaturation events were sent for more sleep that is detailed study i.e. polysomnography in sleep unit in Ain shams university hospital "Al demardash Hospital" About **60** patients sent for sleep lab and diagnosed OSA (Mild to moderate and severe OSA).

After assessment of Seventy patients with OSA by Otolaryngological examination, Nasopharyngoscope and endoscopic evaluation during awake and sleep, we selected thirty patients of OSA had nasal problems to cause OSA.

In our study, Thirty patients with OSA had nasal problems, **18** patients **(60%)** had deviated nasal septum with hypertrophy of the inferior turbinates, **8** patients **(26.7%)** had allergic nasal Polyps with chronic maxillary sinusitis, **4** patients (**13.3%)** had allergic nasal polyps, deviated septum and adenoid hypertrophy.

David, et al (1985) studied 40 patients with OSA, 23 patients had nasal causes of OSA, deviated nasal septum and hypertrophy of inferior turbinates and no patients had narrowing of the lateral dimension of the oropharynx "Apnea index 44.2 ".

In addition, **Vijay, et al (1985)** studied seven patients with OSA and all had nasal causes OSA (4 patients had nasal valve area obstruction and one had marked deviation and another moderate deviation of the nasal septum posterior to the nasal valve area).

Also **Lofaso, et al (2000)** Studied 541 unselected consecutive snorers referred to sleep clinic over the course of two year, about 259 (48%) met this criterion of OSA and patients with OSA had significantly higher nasal resistance than those without OSA and in the same paper, **authors** noted that there was clinical evidence to support a role for nasal obstruction in OSA as septal deviation, nasal valve obstruction, allergic rhinitis and polyps.

In addition, **Thomas, et al (2002)** studied 26 patients with OSA from August 1996 to July 2000 and had impaired nasal breathing underwent attended Polysomnography in sleep lab.

So in our study after surgical management of nasal causes of OSA by: Submucous resection with partial inferior turbinectomy in **6** patients **(20%).** Septoplasty with partial inferior turbinectomy had done in **12** patients **(40%).** Nasal polypectomy with middle meatal antrostomy by endoscope in 8 patients **(26.7%)** and Nasal polypectomy with septoplasty with adenoidectomy in **4** patients **(13.3%).**

In all **30** patients, there was subjective improvement, as regard of snoring, **18** patients **(60%)** complete improvement, **6** patients (**20%)** partial improvement and **6** patients (**20%)** not improved. As regard of apnea, **10** patients (**33.3%)** complete improvement, **6** patients **(20%)** partial improvement and **14** patients (**46.7%)** no improvement. In excessive daytime sleepiness, there was **16** patients had EDS, after surgery, about **6** patients **(37.75%)** complete improvement, **3** patients **(12.25%)** partial improvement and **7** patients **(43.75%)** no improvement.

Also headache, there was **18** patients complaining of headache, after surgery, about **10** patients (**55.5%)** complete improvement, **3** patients (**16.7%)** partial improvement and **5** patients no improvement. In personality changes, irritability, and nervousness, about **4** patients were complaining. After surgery, **3** patients **(75%)** improved and one patient **(25%)** not improved.

Therefore, there was a highly significant subjective improvement in-patient with OSA after nasal surgery to correct OSA.

Vijay, et al (1985) studied six patients with OSA; they found a very good improvement in snoring and daytime somnolence in all six patients after nasal surgery.

Series, et al (1992) found a very good subjective improvement of 20 patients with OSA after nasal surgery.

Also **Friedman, et al (2000)** studied 50 patients with OSA, after nasal surgery,49 patients (98%) subjectively improved nasal breathing, whereas snoring disappeared in 17 patients (34%) , the remaining 33 (66%) patients did not notice any significant changes in their snoring, daytime excessive sleepiness improved in 39 patients (78%) and remaining 11 patients (22%) unchanged .

In addition, **Thomas verse, et al (2002)** found that daytime sleepiness improved significantly and arousal has decreased significantly in both apneics and simple snoring after nasal surgery in-patients with OSA.

In our study, objective assessment of nasal group OSA determined by sleep study, objective improvement occurred in **7** patients from **20** patients **(35%).**

Calderelli, et al (1985) found 35% objective improvement in his research after nasal surgery in-patients with nasal causes of OSA.

Deyal, et al (1985) reported that about 50% objective improvement in-patients with nasal causes of OSA after nasal surgery to correct OSA.

Heimer, et al (1983) reported 100% objective improvement in-patients with nasal causes of OSA after nasal surgery.

In addition, **Rubin, et al (1983)** reported about 55% objective improvement in patients with OSA after nasal surgery.

Series, et al (1992) studied 20 patients with OSA, after nasal surgery there was objective improvement in 8 patients (8/20) 40%.

Series, et al (1993) studied 14 patients that the apnea + Hypopnea index returned to normal (10 breathing abnormalities / hour in all patients). In addition, there was a decrease in the arousal index from 23.9 / hour to 10.6 / hour after nasal surgery.

Thomas, et al (2002) studied 26 patients (19 patients 73.1%) OSA and seven patients were simple snorers with an AHI below 10. The surgical response rates "defined as greater than or equal to 50% reduction in the postoperative AHI and postoperative AHI of less than 20," were 15.8% in the apneics after nasal surgery to correct OSA.

Tae Kim et al (2004) reviewed 21 patients who presented with nasal obstruction and snoring. Septal surgery with or without inferior turbinectomy was performed. Each patient was assessed pre- and postoperatively using PSG.

We measured the respiratory distress index (RDI), apnea index (AI), oxygen saturation index (OSI) and the duration of snoring. Selection criteria were an RDI of >15 as determined by PSG and clinical nasal obstruction and a deviated nasal septum as determined by physical examination. They found that nasal surgery had the following effects: RDI decreased from 39 to 29 (p=0.0001), AI decreased from 19 to 16 (p=0.0209), OSI decreased from 48 to 32 (p=0.0001) and the duration of snoring decreased from 44% to 39% (**p**=0.1595).

Friedman, et al (2000) found in his research that the respiratory distress index increased (though not significantly) from 32 / hr before surgery to 40 / hr after surgery, their results showed good improvement subjectively in the nasal breathing, snoring and increased energy after surgery. **The authors** suggested some explanation for these negative findings in terms of the deeper sleep experienced after surgery resulting in greater muscle relaxation and therefore greater airway collapse. In addition, repeated testing might have resulted in progressively deeper sleep.

Of special concern, here are the authors, finding that some OSA patients actually showed worsened OSA after surgery, a problem that comes up repeatedly, with varied explanation, the more convincing ones couched in terms of multiple sites of obstruction.

Nevertheless, I must accept that obstructed nasal breathing is a problem in and of itself, for which surgery may be indicated; regardless the poorly supported therapeutic effect on OSA.

Series, et al (1992) noted that there was a great variability in the effects of nasal surgery in sleep apnea patients between a very good improvement in subjective study and limited improvement as objective study. Therefore, we believed that the inconstant efficiency of nasal surgery observed in adult, was related to the presence of other oropharyngeal abnormality. Also due to chronic nasal airway obstruction, there may be other craniomandibular abnormalities. It can be hypothesized that these acquired abnormalities could by themselves interfere with the stability of upper airways and in some subjects' lead to pharyngeal airway obstruction in adults.

This is supported by the good polysomnographic results of surgical procedures correcting upper airway patience (Tonsillectomy, adenoidectomy, Nasal surgery) in OSA in children **(Brouillette, et al 1982).**

In our another group of OSA "Oropharyngeal group" after pre-operative assessment of the forty patients with OSA by Otolaryngological examination, Nasopharyngoscope during awake (Muller's maneuver's) and sleep endoscope to determine the most proper site of obstruction to select the cases for coblation of the palate, uvula and tonsils**(subgroup a)** compared with traditional surgery (UPPP) uvulopalatopharyngoplasty **(subgroup b)**.

The subjective response to surgical correction of OSAS in oropharyngeal group **(subgroup a – Coblation)** showed that 8 patients (40%) markedly improved from sleep apnea, 4 patients (20%) partially improved and 8 patients (40%) not improved (Totally there was a significant improvement, P-value=0.05).But in **(subgroup b-UPPP)** showed that 11 patients (55%) markedly improved from sleep apnea, 4 patients (20%) partially improved and 5 patients (25%) not improved (Totally there was a very highly significant improvement, P-value=0.001) . In addition, there was a good response in treatment of snoring in (subgroup a –Radioablation), 11 patients (55%) showed markedly improvement, and 4 patients (20%) showed partially improved and 5 patients (25%) not improved (Totally there was a very highly significant improvement, P-value=0.001). But in **(subgroup b-UPPP)** showed that as regard snoring 11 patients (55%) showed markedly improvement, and 7 patients (35%) showed partially improved and 2 patients (10%) not improved (Totally there was a very highly significant improvement, P-value=0.001). The subjective response in treatment of EDS reported by the patients showed that 8 patients (53.3%) became markedly improved, 3 patients (20%) became partially improved and 4 patients (26.4%) showed no improvement in **(subgroup a – Coblation)**, totally there was no significant improvement, P-value >0.05 .But in (subgroup b-UPPP) as regard of EDS showed that 12 patients (70.6%) markedly improved , 3 patients (17.6%) partially improved and 2 patients (11.8%) not improved (Totally there was a very highly significant improvement, P-value=0.001). The subjective response in treatment of headache showed that 4 patients (50%) became markedly improved, two patient (25%) partially improved and two patient (25%) had no response in **(subgroup a – Coblation).** However, in (subgroup b-UPPP) headache markedly improved in 6 patients (60%), two patients (20%) partially improved and two patients (20%) had no response (Totally there was a highly significant improvement, P-value=0.001). The subjective response in treatment of personality changes showed that 4 patients (66.6%) improved and two patients (33.4%) had no response in **(subgroup a – Coblation)**.But in **(subgroup b-UPPP)** 3 patients (50%) improved and 3 patient (50%) had no response

The results of objective assessment (Polysomnography) of oropharyngeal group of OSA **(subgroup a- Coblation)** 11 patients from 20 patients improved (55%) after Coblation of the palate and tonsils but in **(subgroup b-UPPP**) were 13 patients from 20 patients improved (65%) after Uvulopalatopharyngoplasty.

Powell , et al (1998) evaluate pain, swallowing, speech, edematous response, tissue shrinkage, sleep, snoring, and safety (energy limits and adverse effects) following radiofrequency (RF) treatment to the palate in 22 subjects with sleep-disordered breathing. Twenty-two healthy patients (18 men), with a mean age of 45.3+/-9.1 years, were enrolled. All were snorers seeking treatment and met predetermined criteria: a respiratory disturbance index < or = 15, oxygen saturation > or = 85%, and a complaint of daytime sleepiness, found that neither speech nor swallowing was adversely affected. Pain was of short duration (0 to 48 h) and was controlled with acetaminophen. There were no infections. Although there was documented edema at 24 to 48 h, there were no clinical airway compromises. Polysomnographic data showed improvement in esophageal pressure measurements of the mean nadir and the 95th percentile nadir (p=0.031, p=0.001) respectively, as well as the mean sleep efficiency index (p=0.002). Radiographic imaging showed a mean shrinkage of 5.5+/-3.7 mm (p< or =0.001). Subjective snoring scores fell by a mean of 77% (8.3+/-1.8 to 1.9+/-1.7, p=0.001) accompanied by improved mean Epworth sleepiness scores (8.5+/-4.4 to 5.2+/-3.3, p=0.001).

Fischer et al (2000) studied twenty-nine patients (26 male and 3 female) with obstructive snoring with apnea and socially disturbing character were followed over a ten week period. All patients were treated with radiofrequency volumetric tissue reduction (RFVTR) of the soft palate/Marked improvement of snoring was seen in 20 of 29 patients postoperatively and reduction of the respiratory-disturbance-index (RDI) for more than 10 was noticed in 7/29 patients polysomnographically

Fang et al (2003) studied a new surgical treatment, radiofrequency volumetric tissue reduction (RVTR) of the soft palate, was carried out for thirty-two patients received a single treatment of RVTR with a mean follow-up period of 4.5 months. All patients were assessed by a questionnaire using the Snore Outcomes Survey (SOS) and the Epworth Sleepiness Scale (ESS). Postoperative pain, speech and swallowing disturbances were also evaluated. The postoperative scores of SOS and ESS all significantly improved (p<0.05). Postoperative pain, speech and swallowing disturbances were all mild 1-3 days after treatment. With the success of treatment defined as a postoperative snoring index (SI) of <3 or a reduction of the SI by >5 points by the visual analogue scale, the success rate was 81.3% in patients with a respiratory disturbance index (RDI) of <20, and 50% in those with an RDI of >20.

Blumen et al (2002) studied twenty-nine patients with a respiratory disturbance index between 10 and 30 events per hour, body mass index equal to or less than 30 kg/m2, and obstruction at the level of the soft palate and evaluate prospectively the possible efficacy of temperature-controlled radiofrequency ablation applied to the soft palate in subjects with mild to moderate obstructive sleep apnea syndrome. and found the mean snoring level decreased significantly from 8.6 +/- 1.3 to 3.3 +/- 2.5 on a visual analogue scale (0-10). Daytime sleepiness decreased no significantly. Mean respiratory disturbance index decreased significantly from 19.0 +/- 6.1 events per hour to 9.8 +/- 8.6 events per hour. Mean lowest oxygen saturation value increased no significantly.

Friedman et al (2004) studied the goal of uvulopalatopharyngoplasty (UP3) in the treatment of obstructive sleep apnea-hypopnea syndrome (OSAHS) is to reduce obstruction by eliminating redundant tissue in three areas: the soft palate, tonsils, and pharynx. However, some OSAHS patients may present with tonsil hypertrophy and elongated soft palate without redundant pharyngeal folds. They treated this group of patients with tonsil reduction using radiofrequency coblation combined with uvulopalatoplasty (UP2) using a palatal flap technique without Pharyngoplasty. Morbidity and outcome was then compared with a group of patients who underwent classic UP3

METHODS: Patients were all staged according to the previously described Friedman staging system. Those with redundant pharyngeal folds were treated with UP3 (n = 33), and those without redundant pharyngeal folds were treated with tonsil coblation and UP2 (n = 30). Charts of patients undergoing UP2 and UP3 between July 1, 2001 and July 1, 2002 were reviewed. Thirty-three consecutive patients who underwent UP3 were selected for study as well as 30 consecutive patients who underwent UP2. Pre- and postoperative quality of life questionnaires and patient questionnaires focusing on diet, pain, and return to activity were reviewed to assess subjective morbidity and elimination of symptoms. Objective measurements include preoperative and postoperative (6-18 months) polysomnography (PSG). RESULTS: Symptom elimination and objective PSG results were compared. There was no statistical difference in results between the UP3 group and the UP2 group. Morbidity, however, was significantly more prominent, and recovery was more prolonged, in the UP3 group. Patients undergoing UP2 had fewer pain days, less narcotic use, quicker return to solid diet, and less long-term complaints of globus sensation.

In addition, my results in subgroup –b-UPPP are similar to the results of **Simmons, et al (1983**) who said that, the UPPP is a good operation for treatment of snoring, (100%) improvement in all patients with snoring.

Croft, et al (1990) studied 50 patients with OSA. Apnea was dramatically improved in 22 patients (44%), apneas episodes occasionally occurred in 10 patients (20%) and no improvement occurred in 18 patients (36%) after surgery (UPPP).

Maisel, et al (1992) recorded that 90 patients with moderate OSA, underwent UPPP, 40% of the patients became markedly improved, 24% of the patients became partially improved and 36% of the patients not improved after surgery.

Miljeteig, et al (1994) studied 69 patients with OSA, after UPPP, 78% of the patients reported reduction in snoring and 79% reported improvement in the quality of sleep.

Friberg, et al (1995) reported that 87% of their patients were improved and 13% of the snoring was much less improved after UPPP.

Doghramji, et al (1995) assessed the improvement of EDS by objective method. They selected 53 patients with OSA (45 men and 8 women) underwent UPPP; seventeen patients underwent diurnal multiple sleep latency testing (MSLT) pre and post operatively. They reported that UPPP led to improvement in sleep architectural quality and in daytime somnolence. In addition, subjective response to UPPP in treatment of headache reported by the patients (45.83%) markedly improved and (29.17%) partially improved and 25% of the patients with no improvement. In addition, subjective response to UPPP as regard the treatment of personal changes and nervousness reported by the patients (47.06%) were improved and (52.94%) were not responding to UPPP.

Janson, et al (1997) studied 25 patients with OSA ,after UPPP for 4-8 years Follow up with Polysomnography found that reduced prevalence of snoring and daytime sleepiness and reduction in AHI (50 % or more reduction in AHI and a post operative AHI of 10 or less) at follow up . Sixteen patients (64%) were responders after 6 months and 12 (48%) at long-term follow up.

Elasfour, et al (1998) studied 11 patients with OSA and after UPPP, reduction of AHI more than 50 % was found in 8 cases (72.7%) improvement.

Xiong, et al (2001) studied 70 patients with OSA, after UPPP, found about 55 (79%) improved for at least 6 months and 15 cases (21%) reported unchanged snoring and apnea and Hypopnea index.

Friedman, et al (2002) Studied 134 patients with OSA to correlate palate position and tonsil size to the success of the results of UPPP as based on postoperative Polysomnography results. They found success rate about 80.6% in stage I patients (was defined as having palate position 1 or 2 combined with tonsil size 3 or 4), in stage II patients (palate position 3 or 4 and tonsil size 3 or 4) had success rate of 37.9%.

Engstram, et al (2002) studied 95 male patients with mild to moderate OSA followed for 4 years, they found the results of UPPP about 53% succeed rate.

Przybylowski, et al (2002) Studied 43 men at the age 42.8 ± 6.8 years with OSA with different type of surgery they found 100% improvement after tonsillectomy, 41% improvement after UPPP, 42% improvement after nasal surgery.

Ntascha, et al (2003) evaluated subjectively and objectively 136 patients with snoring and OSA treated by UPPP (snoring 48 cases and OSA 88 cases), they found subjective improvement in 38 cases of snoring (79%) and 74 patients (84%) of the patient with OSA. Objectively dramatic improvement of 36 cases (40%) of the 88 patients with OSA and of 52 patients with a measurement after UPPP, a decrease in the apnea/Hypopnea index (AHI) was found in 38 cases (73%, median decrease: 48 %) and AHI dropped below 20 in 32 (62%). An overall positive result in the 88 patients with OSA was 71 cases (81%, positive subjective result and decrease in AHI).

Laser-assisted uvulopalatoplasty (LAUP) is an outpatient surgical procedure, which has been used as a treatment for snoring. LAUP has also been used as a treatment for sleep-related breathing disorders, including obstructive sleep apnea. The American Academy of Sleep Medicine Standards of Practice Committee concluded that "LAUP is not recommended for treatment of sleep-related breathing disorders **(Littner, et al., 2001),** and found in their reviewed literature that comparing LAUP vs. UPPP (either with or without tonsillectomy) as follow:

One study evaluated OSA **(Walker, et al 1997)** one study examined snoring and OSA **(Remacle, et al 1999**) and one study examined snoring and upper airway size **(Maw, 1997)**. Two of the three studies showed a decrease in AHI which because of sample size could not be compared for degree of efficacy to UPPP **(Remacle ,et al 1999 - Walker, et al 1997)**, the remaining study showed worsened postoperative upper airway anatomic characteristics by oral and nasopharyngoscopic examination for LAUP compared to UPPP patients **(Finkelstein, et al 1997**). Four studies reported subjective postoperative improvement in snoring levels with LAUP and no significant differences in levels of improvement between LAUP vs. UPPP (**Remacle , et al 1999- Carenfelt ,1991- Maw ,1997- Wennmo, et al 1992)**.

However, interpretation of the results of all of the above studies is difficult given the relative lack of detailed statistical analyses of the data.

As mentioned above, comparisons between studies are further limited by lack of standardization of the procedure.

Lastly, the long-term effectiveness of LAUP on treatment of snoring has not been convincingly established. Two separate found snoring improvement of 89.6% and 90%, in patients assessed between one and eight years and at five years following LAUP**(Hagert , et a ,1999- Coleman, 1998).** Less satisfactory results were found in a study that showed snoring improvement was reduced to 62.2% beyond two years **(Mckellson ,1999**). Another study found that 22% of patients had recurrence of snoring between 18 and 24 months following LAUP, with an overall success rate of 55% at 24 months,30 and a separate study found snoring improvement in 43% of patients, with 21% showing no improvement and 36% showed significant deterioration on sleep studies performed 3 to 24 (mean=7) months postoperatively **(Finkelstein ,1997).** Following an average post-LAUP duration of four years, another study found that 51.6% of patients reported that their snoring was eliminated (**Walker ,et al 1999).** As mentioned , the long-term efficacy on LAUP on OSA is not defined but should be considered problematic in view of the inconsistent findings on the long-term efficacy of LAUP on snoring.

There are data to suggest that the pain levels associated with LAUP may be comparable to those of UPPP. One study showed no difference between the average pain scores for the first (typically the most painful) LAUP stage and UPPP **(Maw, 1997).** However, the patients treated with UPPP remained in the hospital overnight and received parenteral analgesia. Another study showed similar maximum pain peaks and intensity for LAUP vs. UPPP, with comparable mean durations of the pain period of 13.76 and 11.80 days, respectively (**Remacle ,et al 1999)**. Similar results were reported in a separate study, which found comparable mean durations of the pain period for LAUP (13.8 days) vs. UPPP (14.3 days **(Troell , et al 2000**).

Besides pain, the most commonly reported side effects from LAUP appear to be transient velopharyngeal insufficiency, minor bleeding, local infection, globus sensation, and minor dysphonia and dysphagia **(Walker , et al 1996 - Pinczower ,1998**). Based on the literature review, the most common side effects with their reported frequency of occurrence are listed. In 27% of LAUP patients, either persistent dysphagia **(Esber, et al 1998)** or mild or moderate scar fibrosis have been observed **(Walker ,et al 1997)**.

Postoperative swelling may compromise an already marginal upper airway; use of narcotics or sedatives may further complicate this problem.

Alcohol should be avoided because of its adverse effects on upper airway muscle tone and closing pressures in snorers (**Issa , 1984**).
The smoke plume from lasers can create a bio-logical and chemical hazard for the patient and surgical team; however, an efficient smoke evacuator used during LAUP can obviate this hazard (**Baggish , 1987**).

There is also evidence to indicate that LAUP may result in a diminished velopharyngeal air space and decreased distensibility **(Finkelstein , 1997).** This study suggests that these structural modifications of the upper airway may decrease airway resistance, resulting in further narrowing during inspiration and collapse of the upper airway at the level of the tongue base, and consequent OSA. These results, from an anatomical perspective, indicate that LAUP may have a worse outcome than UPPP. A separate study examining LAUP patients between 48 and 72 hours after LAUP found worsening of the AHI, with a significant decrement in the cross-sectional area of the airway by videoendoscopy **(Terris ,et al 1996).** A study examining histopathologic changes of the soft palate after LAUP found extensive thermal-induced changes including diffuse fibrosis, oral epithelia ulceration, and a patchy inflammatory reaction, which the authors speculate may be responsible for worsening of OSA **(Berger, et al 1999).**
The selection process for candidates for LAUP or the anatomic, histopathologic, and physiologic effects of this procedure have not been well characterized, and there is a lack of under-standing of its consequences on pathologic respiration and its long-term effectiveness. In general, since insufficient data exists on the effectiveness and risks of LAUP, patients who elect to undergo this procedure as a treatment for snoring should have appropriate preoperative evaluation including screening for OSA, and should have close postoperative follow-up to monitor the patient for possible complications of this procedure.

Objective assessment to surgical correction of OSA in our study also including the assessment of the heart rate, blood pressure, arterial blood gases, ECG, pulmonary function test (spirometry) and Doppler Echocardiography pre and post operatively.

First as regard to the changes which occurred in the heart rate:
In our study, in nasal group of OSA, comparing the mean value of the heart rate during sleep before surgery was 81.96 beat / min and after surgery became 79.2 beat / min.
However, in oropharyngeal group **(subgroup a– Coblation) ,** the heart rate showed slight increase from 79.8 beat/min to 80.2 beat/min and in **(subgroup b-UPPP)** the heart rate showed slight increase from 77.96 beat/min to 82.16 beat/min, but it was of non-significant value This was due to other cardiovascular problems that affect the heart rate as bradycardia, tachycardia and ectopics.

Gislason, et al (1988) reported that there was no significant difference in the heart rate pre and post UPPP.

Murrysville, (2003) reported decrease in the heart rate from 68 to 64 beat/min in-patients with OSA that treated with CPAP and this means that the CPAP treated patients were able to achieve a higher output from the heart and lower heart rate and blood pressure is by expanding less energy.

As regard to blood pressure, comparing of the mean value of the diastolic blood pressure during sleep pre operatively in nasal group was 86.5 mm Hg with the mean value of the diastolic blood pressure postoperatively became (82.1mmhg). Also in oropharyngeal group the mean pre operative diastolic blood pressure in cases of **(subgroup a – Coblation)** was 86.8 mm Hg with the mean value of the diastolic blood pressure postoperatively became (82.23 mmg). In addition, in cases of **(subgroup b-UPPP**), the mean value of the diastolic blood pressure pre UPPP was 92.8 mm Hg and post UPPP became 85 mm Hg. It showed a highly significant improvement in the diastolic blood pressure post-operatively in both nasal and Oropharyngeal groups of OSA.

Also in systolic blood pressure, there was a highly significant improvement in the systolic blood pressure postoperatively as the mean systolic blood pressure pre-operatively in nasal group was (137 mm Hg) and post operatively became 125.6 mm Hg. Also in Oropharyngeal group, in cases of **(subgroup a – Coblation**) the mean systolic blood pressure pre-operatively was 139.16 mm Hg and postoperatively became (128.56 mmg).Also in cases of **(subgroup b-UPPP**) the mean systolic blood pressure pre-operatively was 138 mm Hg and post UPPP became 130.6 mm Hg.

Walker, et al (1989) reported 11 patients with OSA, 7 patients (64%) had hypertension. 4 of them (57%) showed a highly significant improvement after UPPP.

Mayer, et al (1991) studied the effect of nasal CPAP in 12 patients with apnea index from 58 to 2 and fall in arterial blood pressure from 147 /82 to 126/69 mm Hg, there changes occurred without a decrease in body weight.

In addition, **She W et al (2001**) found improvement of blood pressure significantly in cases with OSA treated with Uvulopalatopharyngoplasty.

In addition, **Justin, et al (2002)** noted the efficacy of nasal CPAP on reduction of blood pressure in-patients with OSA.

In addition, **Heinrich, et al (2003**) noted that the effect NCPAP treatment in-patients with moderate to severe OSA leads to reduction in both day and night arterial blood pressures.

Murrysville (2003) studied the effect of NCPAP on OSA patient with heart failure; found that the BP decreased from 126/62 to 116/59 mm Hg.

Sharabi, et al (2004) found in the early stages of sleep apnea are associated with high blood pressure and cardiovascular consequences. Despite our knowledge of the role of the sympathetic activation and vasoactive hormones, no specific antihypertensive therapy is superior, and the optimal way of controlling hypertension is to treat sleep apnea and associated obesity.

Dhillon et al (2005) studied in 180 clinical charts from 1995 to 2002 that patients were identified as hypertensive with sleep apnea and were reviewed after the use of CPAP. Of the patients diagnosed with sleep apnea, 32% were found to have hypertension (mean systolic BP: 164.4 +/- 20.3 mm Hg; mean diastolic BP: 96.9 +/- 5.3 mm Hg). The average use of CPAP was 12.1 +/- 22.4 months. The hypertensive group showed a significant reduction in BP with CPAP use: systolic BP dropped by an average of 11.2 mm Hg ($P < .001$) and diastolic BP dropped by an average of 5.9 mm Hg ($P < .001$).

In our study "in nasal group" pre-operative there was **9** patients **(30%)** had hypertension, after surgery **5** patients **(55,5%)** improved and four patients (**54,5%)** not improved, also in Oropharyngeal group, in cases of **(subgroup a – Coblation)** pre-operative there was **9** patients **(45%)** had hypertension, after **Coblation** , **5** patients (**55.5%)** improved and **4** patients (**45.5%)** not improved but in cases of **(subgroup b-UPPP**) pre-operatively there was **10** patients **(50%)** had hypertension, after UPPP, **6** patients **(60%)** improved and four patients (**40%)** not improved.

Fletcher (2000) said that, during an apneic episode, no airflow into the lung, these episodes might be long enough. So the patient will become hypoxic and hypercapnic. Hypoxia and hypercarbia associated with a rise in sympathetic output and catecholamine production resulting in peripheral vasoconstriction leading to transient pulmonary and systemic hypertension. Therefore, after correction of sleep apnea by surgery, the improvement of the blood pressure occurred due to improvement of the hypoxia and hypercarbia associated with sleep apnea.

In our study, as regard of assessment of arterial blood gases, first the O_2 saturation, the mean value before surgery was **90.2% in nasal group** and postoperative, there was a highly significant improvement, where the mean Sa O_2 became **96.05%.** While in **oropharyngeal group, (subgroup a- Coblation**) the mean pre operative SaO_2 was 89.45% and after **Coblation** there was a highly

significant improvement, where the mean Sa O_2 became **94.35%** and **in (subgroup b-UPPP)** the mean pre operative SaO_2 was 86.75% and after **UPPP** there was a highly significant improvement, where the mean Sa O_2 became **95.69%.**

The results as regard of PCO_2 showed a highly significant improvement after surgery as the mean value of PCO_2 pre-operatively in nasal group was **(41.98 mm Hg**) and after surgery became **34.1 mm Hg.** Also in Oropharyngeal, group before **Coblation** the mean value of (Pa Co_2) was **42.39** mm Hg and post-**Coblation** became **36.57 mm Hg,** in addition in cases of **(subgroup -b UPPP)** the mean value of (Pa Co_2) before surgery was **44.79** mm Hg and after UPPP became **36.57 mm Hg.**

The presence of hypoxaemia (decrease S_aO_2) and hypercapnia (increase $paCo_2$) in OSAS patients are due to upper airway obstruction which lead to the generation of high negative intra- thoracic pressure which is the sequel of respiratory system effort to overcome the upper airway obstruction **(Glassine and Zwrllich, 1992).**

The improvement of the hypoxaemia and hypercarbia after surgical correction of OSA was directly due to the improvement in the hyperventilation that occurred due to upper airway obstruction **(Wetmare, et al 1986).**

Zohar, et al (1991) reported that the main O_2 Sat in 23 patients with OSAS was 84.3 ± 41 become 87 ± 5.9 after UPPP, **Also Jung lu, et al (1995**) showed a highly significant improvement in O_2 Sat after UPPP.

GUQ and Zhang (2003) studied 18 patients with OSAS concomitant with nasal obstruction and palatopharyngeal obstruction were only operated with nasal surgery to improve ventilation, following up six months, They found very good improvement in O_2 Sat (improved from 75 ± 2.9% to 83.4 ± 2.3 % with no significant difference for body mass index).So conclude that nasal obstruction is an important pathogenic factor in OSA.

It is a necessary method to prevent OSAS by relieving nasal obstruction earlier and operation on nasal cavity is an effective method for management of OSAS concomitant with nasal obstruction.

As regard to cardiopulmonary complication of OSA, ECG done for all patients before and after surgery, also Doppler Echocardiography and Pulmonary function test (spirometry) for all patients before and after surgery by 8-12 weeks.

In our study the results showed that: preoperatively in nasal group patients with OSAS had multiple cardiac complications, **9** patients (30%) had sinus bradycardia rhythm, **5** patients (**16.7%)** had sins tachycardia rhythm, **5** patients (**16.6%)** had ventricular extrasystole, **2** patients (**6.7%)** had supraventricular tachycardia, **2** patients (**6.7%**) had 1st degree heart block, **6** patients (**20%)** had depressed S T segment (Ischemia) and **one** patient **(3.4%)** had peaked T wave (Ischemia) .

In oropharyngeal group **(subgroup a– Coblation)** hypertension were found in 9 patients (45%), sinus tachycardia in 3 patients (15 %), ventricular extrasystole 3 patients (15 %), sinus bradycardia 7 patients (35%), heart block in one patient (5 %), Ischemia in 6 patients (30%). Also **(subgroup b –UPPP)** hypertension were found in 10 patients (50%), sinus tachycardia in 3 patients (15%), ventricular extrasystole 3 patients (15%), sinus bradycardia 7 patients (35%), heart block one patient (5%),Ischemia in 6 patients (35%).

Zwillich, et al (1982) evaluated six patients with OSA, and demonstrated that bradycardia occurred during 95% of all apneas and became marked with increased apnea length and demonstrated a strong positive correlation between the level of arterial oxygen desaturation during an apneic event and degree of bradycardia.

They stated that every apnea is associated with some degree of bradycardia. The mechanism for this appears to be enhanced vagal tone, probability due to decrease in reflex respiratory inhibition of vagal tone.

Kryger et al (1992) documented that the patients with OSAS was shown to have sinus bradycardia as well as paroxysms of atrial fibrillation (AF) with a high degree of AV block.

The most frequent arrhythmias reported with OSA are sinus arrest, sinoatrial block, all of which may lead to ventricular asystole. The mechanism of these bradyarrhythmias is usually a reflex increase in vagal tone triggered by combination of apnea and hypoxaemia (**Robert, et al 2003).**

Koehler, et al (1993) found that the patients with OSA and nocturnal brady and tachyarrhythmias are considered to be patients at high risk for cardiac complication.

Also **Cutler, et al (2002)** found that the OSA can result in a multitude manifestation as hypertension, frequent brady and tachyarrhythmias that was due to airflow obstruction and resulting apnea, activate hypoxic and hypercarbic reflexes, which lead to profound elevation in sympathetic nerve activity and cyclical changes in parasympathetic nerve activity.

Olivier Milleron et al (2004) prospectively studied 54 patients (mean age 57.3 ± 10.1 years) with both CAD (⩾70% coronary artery stenosis) and OSA (apnoea–hypopnoea index ⩾15). In 25 patients, OSA was treated with continuous positive airway pressure (***n*=21**) or upper airway surgery **(*n*=4** turbinectomy in 4 patients, complented by ethmoidectomy in 1 patient and septoplasty in another); the remaining 29 patients declined treatment for their OSA. The median follow-up was 86.5 ± 39 months. The two groups were similar at baseline in age, body mass index, smoking history, hypertension, hypercholesterolemia, diabetes mellitus, number of diseased vessels, left ventricular ejection fraction, and CAD therapy. Treatment of risk factors other than OSA was similar in the two groups. The endpoint (a composite of cardiovascular death, acute coronary syndrome, hospitalization for heart failure, or need for coronary revascularization) was reached in 6 (6/25, 24%) and 17 (17/29, 58%) patients with and without OSA treatment, respectively (***P*** <0.01).

Chapman, et al (1989) observed that hypoxia which associated with OSAS increased the degree of sinus tachycardia.

Also **Goldman, et al (1993)** found that Ischemic changes ranging form 20 to 100% among patients with OSA and coexisting CAD (coronary artery disease) and ST segment depression are more frequent in those with more severe OSA or prior complaints of nocturnal angina.

Harald, et al (1997) found similar signs of myocardial Ischemia in 7 of 23 of patients with OSA (30%) not previously believed to have coronary heart disease and believed that the supply of blood oxygen to the heart is decreased by hypoxaemia, this may be one course of myocardial Ischemia, however, OSA also increase the demand for oxygen to the heart because of the reduction of the pressure within the chest as the obstructed patient attempts to breath. During REM sleep, when Ischemia and apneic events were most frequent, there is an increase in the heart rate with increased demand of blood to the heart, related to the autonomic activation typical of REM sleep.

In addition, **Shamsuzzaman, et al (2002)** found that C-reactive protein (CRP), a biomaker of systemic inflammation and an increased risk for coronary events, might also play an important role in atherogenesis. CRP is elevated in OSA, elevated plasma levels and cell expression of several adhesion molecules as well as evidence of increased oxidative stress have noted in OSA**.**

Also **Robert, et al (2003)** found that acute nocturnal Ischemia with ST segment depression occurred with OSA patients and often resistant to traditional therapy and several OSA related mechanisms such as, oxygen desaturation, high sympathetic activity, increased cardiac oxygen demand (due to tachycardia and increased systemic vascular resistance) and prothrombotic state, may contribute to the onset of these Ischemic episodes.

In our study, in nasal group of OSA, we recorded the following important objective postoperative evaluation of the effectiveness of surgical correction of OSA with cardiac problems, which revealed that sinus bradycardia which was present in **9** patients prior to the surgery was completely abolished in 5 patients (**55.5% improvement**) post-surgery. Also **5** patients with sinus tachycardia, **4** patients completely improved after surgery **(80% improvement), 5** patients with ventricular extrasystole, 4 patients improved after surgery (**80%), 2** patients with supraventricular tachycardia, completely improved after surgery (**100%), 2** patients of heart block, one patient improved and another patient not improved **(50%)** , **6** patients depressed ST segment (Ischemia), **2** patients improved (**33.3%),** also **one** patient had peaked T wave (Ischemia) improved after surgery.

In oropharyngeal group: -

There was improvement in arrhythmia after **Coblation (subgroup –a)** showed a very highly significant response (p-value<0.001) as regard of sinus tachycardia, all patients (100%) improved, ventricular extrasystole, all 3 patients improved (100%), sinus bradycardia, 4 patients improved from 7 patients (57 %) , one patient had heart block with good improvement (100%) , Ischemia, 2 patients improved from 6 patients (33.4%). Also there was improvement in arrhythmia after **UPPP (subgroup –b)** showed a very highly significant response (p-value<0.001) , as regard of sinus tachycardia, all patients improved (100%), ventricular extrasystole, all patients improved (100%) ,sinus bradycardia, 6 patients (85.7%)improved from 7 patients, one patient had heart block with good improvement (100%), Ischemia, 3 patients improved from 6 patients (50%), P-value<0.001.

Thomas, et al (**199**3) reported 10 patients with OSA with bradycardia; all patients restored normal cardiac rhythm in night after Nasal CPAP.

Randazzo, et al (1996) reported patients with OSA who developed episodes of supraventricular tachycardia during periods of apnea and oxygen desaturation. With the initiation of nasal CPAP during sleep, the arrhythmia was abolished.

The improvement of hypoxaemia and hypercarbia after surgical correction of OSA patients was direct due to upper airway obstruction so the cardiac arrhythmia improved.

Hanly, et al (1994) studied 23 patients with OSA; they found 7 patients (30%) had ST segment depression in ECG .All 7 patients initiated nasal CPAP. During sleep, they found that Nasal CPAP significantly reduces the duration of ST depression.

In our study, we study the effects of OSA on cardiopulmonary function using:

Doppler Echocardiography for assisting systolic and diastolic function of the left ventricle and pulmonary artery pressure.

In this study, we found the systolic function of the left ventricle (evaluated by LVESD – LVEDD, EF%, FS %,) as follow:

LVESD and LVEDD higher than control subjects in both groups of study (Nasal – Oropharyngeal) as compared with normal control but significant in LVESD 0.01. After surgery in nasal group LVEDD – LVESD became better than before surgery, but not significantly important 0.05.Also in both groups of oropharyngeal group LVEDD – LVESD became better as compared before surgery but significantly importance (0.01 and – 0.05 respectively) after **UPPP (subgroup-b)** only.

(Nasal group LVEDD, LVESD –4.84 ± 0.68, 3.10 ± 0.62).), (oropharyngeal group LVEDD – LVESD – 4.87 ± 0.38, 2.91 ± 0.51).

The left ventricular systolic function evaluated by LVEDD – LVESD, EF % and FS %

In cases of OSA (nasal and oropharyngeal group) these measurement were within high normal as compared with normal control. After surgery, there was improvement in some measurement as compared with normal control.

In oropharyngeal group (in subgroup –b) (LVEDD, LVESD, EF% and FS%, 4.87 ± 0.38, 2.91 ± 0.51, 0.66 ± 0.12 %, 0.38 ± 0.59 %). After UPPP, the results became (4.65 ± 0.21, 2.58 ± 0.69, 0.73 ± 0.13 %, and 0.44 ± 0.12%) with significant importance (<0.01, <0.05, <0.01,< 0 .01).

But in oropharyngeal group (in subgroup –a) and nasal group after surgery there were a little improvement in these measurement and within normal value LVEDD, LVESD, EF %, FS, 4.66±0.31,2.78±0.49,0.67±0.17,0.40± 0.12 in oropharyngeal group (in subgroup –b) and in nasal group 4.8 ± 0.68, 3.10 ± 0.52, 0.64 ± 0.11.306± 0.09, 3.05±0.48, 0.63 ± 0.08, 35 ± 0.07) with no significant importance, more than 0.05.

The RVD (Right ventricle diameter) in both groups (nasal – Oropharyngeal) was more than normal control subject without significant importance 0.05, but after surgery in nasal group became better without significant importance. (Before surgery 2.20 ± 0.42, after surgery 2.07 ± 0.31).But in both oropharyngeal groups RVD became better with significant importance 0.001 (Before surgery 2.23 ± 0.32, after surgery 2 ± 0.11 in subgroup –a- Coblation) and in (subgroup-b-UPPP) before surgery 2.06 ± 0.52, after surgery 1.83 ± 0.52).

The left ventricular diastolic function evaluated by E / A ratio.
The E/ A ratio in both group of OSA "Nasal – Oro pharyngeal" before surgery less than **1**, so there were left ventricular diastolic dysfunction as compared with normal control subject. (Normal control E /A ratio 1.16 ± 0.39). After surgery, the E / A ratio in both groups became well than before surgery with significant importance 0.05.

In nasal group E/ A ratio before surgery was 0.98 ± 0.12 and after surgery became 1.11 ± 0.18.

In oropharyngeal group (**subgroup –a- Coblation**) E /A ratio before Coblation was 0.94 ± 0.22, after Coblation became 1.2 ± 0.13) and in **(subgroup-b-UPPP)** before surgery was 0.93 ± 0.10, after UPPP became 1.10 ± 0.23). Therefore, after surgical correction of OSA, RVD improved and left ventricular diastolic function improved.

The Pulmonary artery pressures in both group of OSA (Nasal – Oro pharyngeal) were higher as compared with normal control.

As regard of nasal group, the mean value of PAP was 34.06 ± 11.2 mm hg) and the mean value of PAP in Oropharyngeal group **(subgroup –a- Coblation)** was 36.93± 8.98 and in **(subgroup-b-UPPP)** was 36.93 ± 9.68 mm Hg with highly significant importance 0.001.

In nasal group, there was pulmonary artery hypertension (PAH) in about 70% compared with 68 % (PAH) **in oropharyngeal group (subgroup –a-Coblation)** and 73% (PAH) **in oropharyngeal group(subgroup-b-UPPP).** After Surgery, there was significant decrease in pulmonary artery pressure in all groups.

In nasal group, the mean value of PAP became 18.14±7.14 mmHg instead of 34.06± 11.2 mmHg before surgery. Moreover, **in oropharyngeal groups** (subgroup-a – Coblation) became 23.24 ± 3.6mmHg instead of 36.93±8.98 mmHg before surgery and in (subgroup-b-UPPP) became 21.24 ± 4.6mmHg instead of 36.9 ± 9.68 mmHg before surgery.

Therefore, there was significant decrease in PAP in both groups of OSA after surgery .So there was a highly significant importance in correlation between the severity of OSA and the presence of PAH.

Tilkian, et al (1977) reported the effect of tracheostomy on patients with cardiac arrhythmia associated with OSAS. They had done their work on 15 Patients. Marked sinus arrhythmia was completely abolished in all patients immediately after tracheostomy (100% improvement). Asystole had been abolished in 3 from 5 patients (60 % improvement). Premature ventricular ectopics, which present in 10 patients with OSA patients, were abolished after tracheostomy (60% improvement). Extreme sinus tachycardia, A –V block and ventricular tachycardia were not detected after tracheostomy (100% improvement). Although with these good results occurring in management of the cardiac problems associated with OSAS by tracheostomy, when the tracheostomy site was temporarily occluded during sleep, pretracheostomy pattern of arrhythmia and ventricular ectopics were recurring promptly and apnea episodes were return back similar to the state before tracheostomy.

Also Zohar,Y et al (1992) found improvement in global and regional cardiac function of both ventricles was seen in 91% of patients with OSAS following Uvulopalatopharyngoplasty. (A trend toward significant elevation of left ventricular ejection fraction and a statistically significant increase in right ventricular ejection fraction were observed (45%+/- 9% to 50%+/- 7 .

Friedman et al (2004) studied the effect of UPPP with tongue base radiofrequency reduction on 8 patients with mild OSA and 22 patients with moderate to severe OSA and found all patients with mild to severe OSA had relative elevation of preoperative C-reactive protein (CRP) levels indicating increased risks of cardiovascular disease .Postoperative CRP levels were lower in most cases and interleukein-6(IL-6) levels were less reduced but still show a significant reduction even patients who did not achieve complete cure by classical polysomnography.

However**, Alchanatis, et al (2001)** studied the affect of nasal CPAP in the left ventricular function in- patients with OSA before and after NCPAP, they found that there was LV diastolic dysfunction but not systolic dysfunction compared with normal group. After NCPAP, there was a significant improvement in both ventricular function and systemic hypertension.

Peker, et al (2002) studied the incidence of cardiovascular disease in 182 middle-aged men with OSA and 7 years follow up. They found that the incidence of at least one CVD (CVD was classified as hypertension, CAD or Cardiovascular event “stroke or myocardial infarction”) was observed in 22 of 60 (36.7%) cases with OSA compared with 8 of 122 (6.6%) subjects without OSA and also found in cases with OSA, the incidence of CVD was observed in 21 of 37 (53.8%) in untreated cases completely, compared with 1 of 15 (6.7%) efficiently treated subjects.

They found that the initial result of UPPP was positive. The first follow up recordings was done 1-2 years after surgery. Within the first 4 years of observation period and demonstrated a 100% improvement effectiveness in the efficiently treated group of CVD. Assuming that some of these subjects develop OSA later than 4 years.

Kober, et al (1996) investigated 18 patients with OSA by Doppler Echocardiography to assess systolic and diastolic function of the heart, Rt. ventricle thickness and Pulmonary artery pressure. They found both systolic and diastolic ventricular dysfunction, right Ventricle hypertrophy and PAH.

Bady (2000) studied 44 patients with OSA and without COPD. They observed pulmonary artery hypertension (PAH) "defined as mean pulmonary artery pressure more than 20 mm Hg" in 12/44 (27%) patients with OSA and also the PAH+ patients had significantly lower values of vital capacity (VC) 87 (14%) predicted versus 105 (20%) predicted, (P 0.005 and forced expiratory volume in one second (FEV_1) was 82 (14) % predicted versus 101 (17 %) predicted, = 0.001.

Kang, et al (1997) studied six patients with OSAS with monitoring pulmonary artery pressure. All patients had pulmonary artery hypertension during periods of wakefulness and their mean pulmonary artery pressure was 31.1 ± 7.1 mm Hg.

Alchantis, et al (2001) studied 29 patients with OSA without any other cardiac or lung disease and 12 control subjects were studied. They found a significant higher mean (PA) was found in OSA patients as compared to control subjects (17.2 ± 5.2 versus 12 ± 11.9 mm Hg). Six patients out of 29 OSA patients had hypertension and to asses the effect of NCPAP treatment on pulmonary artery pressure. They found that CPAP treatment was effective in reducing mean pulmonary artery pressure and normotensive OSA patients.

Sajkov, et al (2003) measured pulmonary hemodynamics (by Doppler Echocardiography) in 20 patients with OSA before and after 1and 4 month of NCPAP treatment. The patients have normal lung function and no cardiac disease. Treatment resulted in a decrease in daytime pulmonary artery pressure (ppa 16.8 ± 1.2 mm Hg) before CPAP versus 11 ± 0.6 mm Hg after 4 months CPAP.

There is strong epidemiologic evidence showing an independent association between OSA and hypertension. High levels of AHI and sleep time below 90% oxygen saturation (**Lavie, et al 2000**).

Nieto, et al (2000) found there were associated with greater odds of hypertension in a dose-response fashion. LV afterload is increased by peripheral vasoconstriction because of recurrent arousals terminating the obstructive respiratory events and activating the sympathetic nervous system, **(Fletcher, et al 2000)** and activation of the arterial chemoreceptors by hypoxia and hypercapnia. Plasma levels of nitric oxide, a powerful vasodilator released from the endothelium, have been shown to be decreased in OSA patients but can be promptly reversed by nasal CPAP treatment.

Schulz, et al (2000) and **(Kraiczi, et al 2000)** showed in 20 subjects with OSA that worsening nocturnal hypoxemia (measured as minimum SpO_2 or percentage of sleep time with SpO_2 below 90%) was associated with a gradual deterioration of LV diastolic function (increased interventricular septum thickness, prolonged IVRT, and decreased E/A ratio), as well as reduced endothelium-dependent dilatory capacity of the brachial artery.

Other mechanisms that may result in LV dysfunction include increased preload by intermittent negative intrathoracic pressure during apnea, which may also increase the LV transmural pressure gradient and impair diastolic relaxation and LV filling **(Fletcher, et al 2000).**

Hall, et al (1989) showed that the increase in right ventricular volume, together with hypoxia-induced pulmonary hypertension, may displace the interventricular septum leftward during diastole and impair LV filling **(Scharf, et al 1992)**. Hypoxia and hypercapnia may also decrease myocardial contractility **(Sommers , et al 1989)**.

In this study, asymptomatic diastolic dysfunction was prevalent among OSA patients; more severe OSA, as reflected by the minimum SpO_2 and AHI40/h, was associated with worse diastolic parameters. It is possible that OSA, through hypoxia, hypertension, and other mechanisms discussed earlier, causes LV diastolic dysfunction that, in the end, may lead to symptomatic DHF.

It is important to keep a high index of suspicion for OSA when assessing patients with CHF. Not only can nasal CPAP or surgical correction of OSA effectively relieve disabling symptoms such as sleepiness, **(Jenkinson, et al 1999)** it may potentially improve LV systolic **(Malone, et al 1991)** and diastolic function, **(Alchanatis, et al 2000)** decrease activation of the sympathetic nervous system activity, **(Hedner, et al 1995**) and increase nitric oxide levels **(Schulz, et al 2000)** in patients with both OSA and cardiac problems. Nasal CPAP or surgical correction of OSA can reduce ventricular irritability, **(Javaheri, 2000**) improve LVEF, and reduce the combined mortality-cardiac transplantation rate in such patients (**Sin, et al 2000)**.

In our study, pulmonary function was assisted by spirometry by measuring these items (FEV1, FEV1 /FVC, FEF25-75, FEF75).

In nasal group spirometry parameter improved after surgical correction of OSA (P- value <0.05,< 0.01, 0.001,< 0.05,< 0.001 for FVC, FEV1, FEF25, FEF50, FEF75, respectively).

Also **in both groups of oropharyngeal group**, spirometry parameter improved after surgery (P-value <0.01, <0.001, <0.001, <0.001, <0.001, <0.001 for FVC, FEV1, FEV1/VC, FEF25, FEF50, FEF75, respectively.

Zekah- Lancner, et al (1997) evaluated pulmonary function abnormalities associated with sleep apnea syndrome (SAS) in 170 habitual snorers with moderately to severe OSA ad assessed by spirometry. They found that forced expiratory flows decreased as the OSAS severity (P 0.001, p <0.02, and p<0.05 for FEF 50, FEV1, and FEV1/ VC, respectively).

In our study, we found a strong correlation between the apnea-hypopnea index and the PCO2, with more severe OSAS being associated with higher PCO_2 values. Both Pa O_2 and SaO_2 decreased significantly when the AHI increased. Disturbances in gases exchange have been to be largely determined by alterations in ventilatory mechanics and to be related with diffuse airway obstruction.

Bradly et al (1986) found the presence of diffuse airway obstruction may be an important predisposing factor for the development of chronic CO2 retention in such patients. Also **Zekah- Lancner, et al (1997)** symptoms of chronic bronchitis ,a significant correlation between the severity of the apnea –hypopnea index and presence of lower and upper airway obstruction responsible for decreases in expiratory flow rates .Finally ,the surgical correction of OSAS is a good toll for reversal of cardiopulmonary complications associated with OSA to the normal values.

THE CONCLUSIONS

* The surgical correction of either nasal or oropharyngeal OSA significantly reduces both the systolic and diastolic blood pressure.

* The surgical correction of either nasal or oropharyngeal OSA significantly improves the left ventricular systolic and diastolic functions, left atrial dimension and pulmonary artery systolic pressure.However the right ventricle diameter is reduced in both groups of OSA but the reduction is significant in oropharyngeal group only.

* The number of patients with sinus tachycardia, sinus bradycardia, ventricular extrasystole, supraventricular tachycardia, complete heart block and/or ischemic ECG changes is significantly reduced by the surgical correction of OSA regardless of the etiology whether nasal or oropharyngeal.

* The arterial blood oxygen saturation and carbon dioxide pressure are significantly improved by the surgical correction in nasal and oropharyngeal OSA patients.

* The surgical correction of OSA patients with nasal or oropharyngeal causes shows significant improvement in respiratory function test (FVC, FEV_1 and FEF). However, the FEV% shows significant improvement only in patients with oropharyngeal disorders.

* The symptoms of OSA as snoring, excessive daytime sleepiness, apnea, as well as headache and apnea/hypopnea index assisted by polysomnography are significantly improved in both nasal and oropharyngeal OSA patients by the surgical correction.

* Recommendation

The surgical correction in patients with nasal or oropharyngeal causes of OSA is advised since it improves significantly the cardiopulmonary functions to the normal levels.

THE SUMMARY

Obstructive sleep apnea syndrome is a multisystem disorder and characterized by cessation of normal Oronasal airflow for 10 seconds or longer and repetitive closure of the upper airway during sleep resulting in sleep fragmentation and daytime hypersomnolence. OSA is associated with a range of cardiovascular sequelae as systemic and pulmonary hypertension, cardiac arrhythmias, angina pectoris, myocardial infarction, and decreased arterial blood oxygen saturation.

Seventy (70) patients were selected from patients with OSAS and had history of cardiac manifestations, from ENT clinic of Benha university hospital and Shebien Al kom teaching- hospital during period from March 2001 to October 2004.

Patients were classified into two groups according to the site of obstruction.

*First group (**Nasal group**) : Thirty patients all had nasal obstruction secondary to a deviated septum, nasal polyps, hypertrophy of turbinates, and nasal valve obstruction. No patient had narrowing of the lateral dimension of the oropharynx to 2 cm or less, as measured between the midpoints of each anterior tonsil pillar.

The type of operation done in the first nasal group as follow:

Submucous resection with partial inferior turbinectomy in 6 patients (20%). Septoplasty with partial inferior turbinectomy in 12 patients (40%).Nasal polypectomy with middle meatal antrostomy by endoscope in 8 patients (26.7%). Nasal polypectomy with septoplasty and adenoidectomy in 4 patients (13.3%).

*Second group (**Oropharyngeal group**): Forty patients with oropharyngeal obstruction (A long soft palate, narrow inlet to the nasopharynx, hypertrophic tonsils and redundant lateral pharyngeal mucosa.

In the second Oropharyngeal group, half patients (20 patients 50%) had done radioablation of the redundant soft palate and uvula with tonsillectomy if marked enlargement ,however with mild to moderate enlargement of tonsils, Coblation-Channeling Technique was done**(subgroup- a- Coblation)** and another half (20 patients 50%) had traditional Uvulopalatopharyngoplasty **(subgroup- b- UPPP).**

*A control group: Other 10-nonapneic patients free of cardiopulmonary problems and with the same age and body mass index of our study.

The results of our study in nasal group of OSA, most of the thirty patients had a good subjective improvement in snoring, apnea, excessive daytime sleepiness, headache and personality changes as irritability and nervousness.
In addition, as regard of objective assessment of nasal OSA group determined by sleep study, good improvement occurred in seven patients from 20 patients (35**%).**

The results of our study in Oropharyngeal group of OSA, most of the forty patients had a very good improvement as regard of snoring, apnea, excessive daytime sleepiness, headache and personality changes as irritability and nervousness.

Also as regard of objective assessment of patients with OSA **(Oropharyngeal group)**, the results of objective assessment (Polysomnography) of oropharyngeal group of OSA **(subgroup a- Coblation)** 11 patients from 20 patients improved (55%) after Coblation of the palate and tonsils but in **(subgroup b-UPPP**) were 13 patients from 20 patients improved (65%) after Uvulopalatopharyngoplasty

The surgical correction of OSA was an excellent procedure for improvement of arterial blood gases.

In our study, as regard of assessment of arterial blood gases, first the O_2 saturation, **in nasal group,** the mean value before surgery was **90.2%** preoperative and there was a highly significant improvement in O_2 saturation that became **96.05%** after surgery. Also in **Oropharyngeal group,** before UPPP, O_2 saturation was **86.75%** and Post-operative there was a highly significant improvement in Sa O_2 that became **95.69 %.**

In addition, the carbon dioxide tension (pa Co_2) showed a highly significant improvement after surgery in both groups of OSA.

The surgical correction of OSA is an excellent procedure for the reversal of the cardiovascular lesions associated with OSA.

As regard to blood pressure, comparing of the mean value of the diastolic blood pressure during sleep pre operatively in nasal group was 86.5 mm Hg with the mean value of the diastolic blood pressure postoperatively became (82.1mmhg). Also in oropharyngeal group the mean pre operative diastolic blood pressure in cases of **(subgroup a – Coblation)** was 86.8 mm Hg with the mean value of the diastolic blood pressure postoperatively became (82.23 mmHg). In addition, in cases of **(subgroup b-UPPP**), the mean value of the diastolic blood pressure pre UPPP was 92.8 mm Hg and post UPPP became 85 mm Hg. It showed a highly significant improvement in the diastolic blood pressure post-operatively in both nasal and oropharyngeal groups of OSA.

Also in systolic blood pressure, there was a highly significant improvement in the systolic blood pressure postoperatively as the mean systolic blood pressure pre-operatively in nasal group was (137 mm Hg) and post operatively became 125.6 mm Hg. Also in oropharyngeal group, in cases of **(subgroup a – Coblation**) the mean systolic blood pressure pre-operatively was 139.16 mm Hg and postoperatively became (128.56 mmHg).Also in cases of **(subgroup b-UPPP**) the mean systolic blood pressure pre-operatively was 138 mm Hg and post UPPP became 130.6 mm Hg.

In our study, in nasal group of OSA, we recorded the following important objective postoperative evaluation of the effectiveness of surgical correction of OSA with cardiac problems, which revealed that sinus bradycardia which was present in **9** patients prior to the surgery was completely abolished in 5 patients (**55.5% improvement**) post-surgery. Also **5** patients with sinus tachycardia, **4** patients completely improved after surgery **(80% improvement), 5** patients with ventricular extrasystole, 4 patients improved after surgery (**80%), 2** patients with supraventricular tachycardia, completely improved after surgery (**100%), 2** patients of heart block, one patient improved and another patient not improved **(50%)** , **6** patients depressed ST segment (Ischemia), **2** patients improved (**33.3%),** also **one** patient had peaked T wave (Ischemia) improved after surgery.

In oropharyngeal group: -

There was improvement in arrhythmia after **Coblation (subgroup –a)** showed a very highly significant response (p-value<0.001) as regard of sinus tachycardia, all patients (100%) improved, ventricular extrasystole, all 3 patients improved (100%), sinus bradycardia, 4 patients improved from 7 patients (57 %) , one patient had heart block with good improvement (100%) , Ischemia, 2 patients improved from 6 patients (33.4%). Also there was improvement in arrhythmia after **UPPP (subgroup –b)** showed a very highly significant response (p-value <0.001) , as regard of sinus tachycardia, all patients improved (100%), ventricular extrasystole, all patients improved (100%) ,sinus bradycardia, 6 patients (85.7%)improved from 7 patients, one patient had heart block with good improvement (100%), Ischemia, 3 patients improved from 6 patients (50%), P-value<0.001.

We study the effects of OSA on cardiopulmonary function using:
Doppler Echocardiography for assisting systolic and diastolic function of the left ventricle of the heart and pulmonary artery pressure.

In this study, we found a good improvement of both the systolic and diastolic ventricular function of the left ventricle but more significant in diastolic function in both groups of oropharyngeal group of OSA than nasal group , (The parameters of Echo were LVESD – LVEDD, EF%, FS%, RV and E/A Ratio) .

The Pulmonary artery pressure **(PAP)**: The Pulmonary artery pressure (PAP) in patients with OSA higher than control subjects with highly significantly importance (P –value <0.001**)**. In nasal group, there was pulmonary artery hypertension **(PAH)** in a about 70% compared with 68 % **(PAH)** in oropharyngeal group **(subgroup -a-Coblation)** and 73% **(PAH)** in oropharyngeal group **(Subgroup-b-UPPP)**. However, after surgical correction of OSA- there was significantly decease of pulmonary artery pressure in all groups of OSA (nasal, oropharyngeal group) P -value <0.001. Therefore, there was a significant correlation between the severity of OSAS and the presence of systolic and diastolic function of the heart.

Also pulmonary function of the patients of OSA assisted by spirometry that became better after surgery

In nasal and oropharyngeal groups of OSA, spirometry parameters (FVC, FEV1, FEF25, FEF50, and FEF75) were improved after surgical correction of OSA.

Therefore, the surgical correction of OSA is an excellent procedure for the reversal of the cardiovascular lesions associated with OSA.

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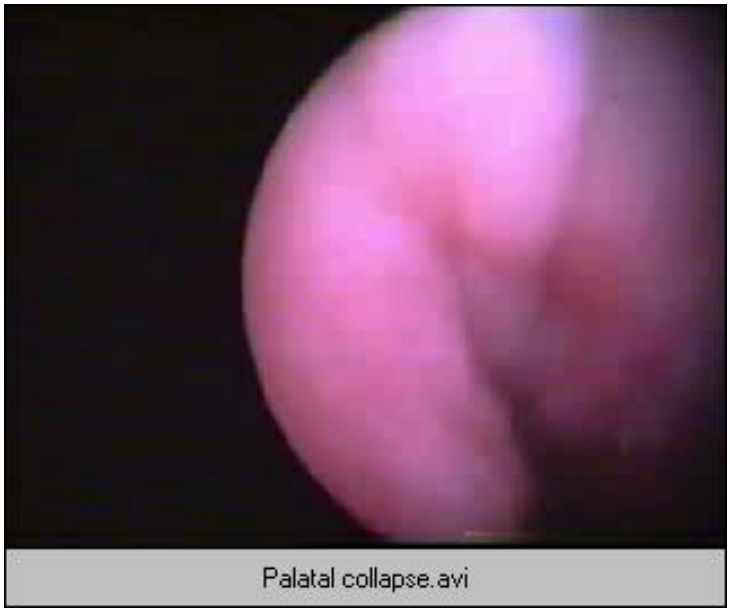
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