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ثروت عبد الظاهر محمد عمارة
طبيب أسنان بوزارة الصحة

لقسم جراحة الفم والوجه والفكين
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تحت إشراف

الأستاذ الدكتور

محمد محمد السحيمي

أستاذ جراحة الفم والوجه والفكين

كلية طب الفم والأسنان - جامعة القاهرة

الأستاذ الدكتور

شريف جبر

أستاذ الأذن والأنف والحنجرة

كلية الطب - جامعة القاهرة

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Item	Page
Introduction	1
Review of literature	5
▶ Sleep disordered breathing	5
▶ Pathophysiology of OSA	7
▶ Risk factors of OSA	11
▶ Complications of OSA	16
▶ OSAS-related symptoms	19
▶ General characteristics of OSA	21
▶ Polysomnogram in OSA	27
▶ Role of Imaging of OSA	29
▶ Inferior Sagittal Mandibular Osteotomy and Genioglossal Advancement	36
▶ Hyoid Myotomy and Suspension	40
▶ Upper Airway Bypass Surgery for Obstructive Sleep Apnea Syndrome	43
Aim of the work	46
Patients and Methods	47
Results	71
Discussion	78
Summary	87
Conclusion & Recommendations	90
References	91
Protocol of Thesis	1
Arabic Summery	١

OBSTRUCTIVE SLEEP APNEA: SURGICAL CORRECTION

Thesis

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By
Tharwat Abd-El Zaher Omara
(B. D.S., M. Sc., Oral Surgery)

**Faculty of Oral & Dental Medicine
Cairo University
2008**

Supervised By

Prof. Dr. *Mohammad El-Sehímy*

Professor of Oral and Maxillofacial Surgery

Faculty of Oral & Dental Medicine

Cairo University

Prof. Dr. *Sherif Gabr*

Professor of ENT Surgery

Faculty of Medicine, Cairo University

Key Words

1-	O.S.A.	Opestructive sleep aprea
2-	S.D.S	Sleep disorder breathing
3-	GAHM	Gineoglossus muscle advancement and hyoid myolomy
4-	UARS	Upper airway resistance syndrome
5-	CPAP	Continuous positive airway pressure
6-	P crit	Pharyngeal critical pressure.
7-	REM	Rapid Eye movement
8-	BMI	Body mass index.
9-	GERD	Gastroesophageal reflex Diseases
10-	ANP	Atrial naturetio peplide.
11-	MMC	Modified mallampati classification
12-	PSG	Phosomonography
13-	MRI	Magnatic resenoning imaging
14-	C.T	Computerized tomography
15-	UPPP	Uveloplatopharyngeoplasty
16-	GA	Genioglossns advancement
17-	HM	Hyoid bone myotomy

INTRODUCTION

Apnea is defined as the cessation of airflow at the nostrils and mouth for at least 10 seconds. The apnea index is further defined as the number of apneas per hour. Three types of apneas have been described, obstructive sleep apnea (OSA) which is a cessation of air flow in the presence of continued inspiratory effort, central apnea which is the absence of inspiratory effort without air flow and a mixed apnea which is the combination of both components beginning as a central apnea followed by the onset of inspiratory effort without air flow **(1)**.

Sleep disordered breathing (S.D.B) is a broad term that encompasses obstructive sleep apnea, obstructive sleep hypopnea, snoring and upper airway resistance syndrome. Symptoms of (S.D.B) include snoring, restlessness, sleep disruption, choking during sleep, esophageal reflux, nocturia and heavy sweating. Its physical findings may include obesity, tonsillar hypertrophy, elongate uvula, redundant pharyngeal folds, thickened tongue and retro or micrognathia **(2-3)**.

Obstructive sleep apnea syndrome (OSAS) is the most common type of sleep disordered breathing condition. It is well documented that (OSAS) has a potentially serious physiological and psychological consequences. Patients not only can suffer from debilitating excessive daytime somnolence, but data accumulated from the Stanford Sleep Disorders Center showed that many patients have periods of depression 20%, cardiac arrhythmia (tachycardia, bradycardia, ventricular ectopy) during sleep 45%, and hypertension requiring medication 54% **(4-6)**. Obstructive

sleep apnea syndrome (OSAS) is characterized by repeated collapse of the upper airway producing hypopnea, apnea and ultimately oxygen desaturation of hemoglobin. The effects of repeated desaturation of hemoglobin significantly alter the normal cardiovascular and pulmonary functions resulting in pulmonary, systemic hypertension and arrhythmia which if not treated can lead to an early death **(7-10)**.

The hypopnic and apneic episodes with frequent arousal from sleep disrupt the normal sleep patterns. Ineffective sleep results in sleep fragmentation and excessive daytime sleepiness which can have a profound effect of social interaction and ability to work or to perform the normal activity such as driving a car **(11)**. Three variables are important in the development of collapse and obstruction of the upper airway in patients with (OSAS). The decreased activity of the muscles dilators of the pharyngeal airway, the relative vacuum generated in the upper air way during inspiration and the surgical anatomy of the upper airway passages **(12)**.

The occlusion typically begins in the oropharynx with the tongue contacting the soft palate and the posterior pharyngeal airway. In addition to the collapse in the anteroposterior direction there is a progressive collapse of the lateral oropharyngeal walls **(12-14)**.

Several authors reported on an incidence of obstructive sleep apnea syndrome (OSAS) between 1% and 3% of the population while others reported on 10% of population **(15-16)**.

In the past, tracheostomy, has been the most common surgical treatment of OSAs syndrome, however it is not an effective surgical treatment, therefore the surgeons try to make new techniques to correct the real causes not to overlap it (17).

Patton et al. have shown that the surgical expansion of the hyoid bone can result in expansion of the pharyngeal walls and enlargement of the pharyngeal space. It has been reported that the total mandibular advancement will advance the base of the tongue and expand the pharyngeal space and hence, improvement of OSA syndrome. However there are many problems with the total mandibular advancement techniques because intermaxillary fixation is required and orthodontic treatment is necessary to correct the altered occlusion. Also, many patients are over 50 or 60 years age and are neither tolerant of intermaxillary fixation nor accept orthodontic treatment (7).

This may lead to the development of a new technique called the inferior sagittal mandibular osteotomy. This method involves isolated advancement of the genial tubercles, genioglossus muscles and hyoid myotomy. Genioglossus muscles advancement is a safe, simple and a rapid method for improving symptomatic base of tongue obstruction in sleep disordered breathing. This technique will pull the tongue in a more anterior position through the genioglossus muscles leading to widening of the pharyngeal airway (17-19).

The base of the tongue and the deep pharyngeal walls are the common sites of obstruction in O.S.A syndrome, as there is an intimate relationship between the mandible, the base of the

tongue, the pharyngeal walls and the hyoid bone because of their muscular and ligaments attachments **(19-20)**. **Riley and Powell (1989) and Moghadai (2003)** recommended the use of inferior sagittal mandibular osteotomy for the treatment of (OSAS), they further recommended the use of a pre-surgical evaluation correctly to detect the actual cause of obstruction to perform this operation. Moreover they also recommended these surgical procedures for improvement of the state of apnea **(21-22)**.

Neruntaratc and Sher (2003) recommended the use of inferior mandibular sagittal osteotomy with gineoglossus muscles advancement and hyoid myotomy **(GAHM)** for surgical treatment of obstructive sleep apnea. They recommended this after pre-surgical evaluation of the site of the obstruction which is most commonly located at the hypopharynx, pharynx and the base of the tongue **(23-24)**.

Lewis (2003) stated that the location of the obstruction of the airway is very important to detect the surgical procedures selected. The surgical procedures available for the correction of the hypopharyngeal and the base of the tongue collapse include inferior sagittal mandibular osteotomy and hyoid myotomy which is an effective methods over the midline glossectomy and maxillo-mandibular advancement techniques **(25)**.

REVIEW OF LITERATURE

Sleep Disorder Breathing

Historical Aspects

In 1877, **Broadbent** described the sleeping characteristics of patients with apneas: "there will be perfect silence through two, three, or four respiratory periods, in which there are ineffectual chest movements; finally air enters with a loud snort, after which there are several compensatory deep inspirations...". He also described that obstructive apneas in snorers are distinguished from central apneas in Cheyne-Stokes respiration **(26)**.

In 1965, French and a German group published the first studies in which respiration was recorded during sleep in patients with "Pickwickian syndrome". When this new technique was developed, clinical sleep specialists began to investigate respiratory changes as a function of sleep. As a result of this, more patients were primarily investigated for sleep disorders rather than cardiorespiratory problems. At a later stage, some authors reported that sleep apnea not only occurred in patients with Pickwickian morphology but also in individuals without these characteristics **(28-29)**.

The **Swedish Medical Research Council at the State of the Art Conference** in 1994 stated a definition of Obstructive Sleep Apnea syndrome (OSAS) "as intermittent, complete or partial, upper airway obstruction during sleep causing mental and/or physical effects", together with a cut-off value for a laboratory finding of an apnea index of > 5 or an apnea/hypopnea index of > 10 **(4)**.

Sleep-related Breathing Disorders

There are three main types of sleep-related breathing disorders ranging from habitual snoring via increased upper airway resistance syndrome to sleep apnea syndrome. Snoring is an inspiratory noise caused by the vibrations of an enlarged and flabby soft palate. The vibrations are more intense in the supine position and also when pharyngeal muscular tone is reduced, caused by different factors such as the intake of alcohol and sedative medication **(30)**.

The Upper Airway Resistance Syndrome (UARS) is a sleep breathing disorder caused by an increase in breathing effort during periods of increased upper airway resistance but without apneas. Sleep fragmentation and daytime dysfunction are common among these patients. The definition of UARS is uncertain because no cut-off limit for what is pathological has been defined **(31)**. Sleep apnea can be divided into three categories: Obstructive due to occlusion of the pharyngeal airway, which results in increased respiratory effort, central due to a cessation of both respiratory efforts, airflow and mixture of these two respiratory patterns **(32)**.

The obstructive sleep apnea/hypopnea syndrome is characterized by recurring episodes of upper airway obstruction during sleep, leading to reduction of airflow through the nose and mouth. The underlying pathophysiology of obstructive sleep apnea is complex and not fully understood. However, it is generally accepted that the stability and patency of the upper airway are dependent upon the action of pharyngeal dilator muscles (e.g. the genioglossus muscle), which are normally activated in a rhythmical fashion during each respiration. In patients with obstructive apnea,

upper airway muscle activity decreases during sleep, particularly in the genioglossus muscle. This reduction in activity results in airway occlusion, which causes an increased respiratory effort during inspiration. The obstructive event is usually followed by an arousal, a short episode of awake brain activity caused by the increase in respiratory effort. This arousal restores upper airway dilating muscle tone by increased activity and the patient gasps, takes a few deep breaths and falls back to sleep, at which point the upper airway dilating muscles relax and the cycle begins again. During the apnea, there is an abrupt concomitant increase in blood pressure and a reduction in oxygen saturation, followed by a rapid change to normal levels after the onset of respiration **(32)**.

Central sleep apnea syndrome is characterized by apnea episodes during sleep with no associated inspiratory effort as a result of an absence of neural output from the respiratory centers. The apnea episodes commonly occur after central alveolar hypoventilation. Central sleep apnea is commonly seen in patients with congestive heart failure, older patients (> 65 years), in some patients after a stroke and in certain neurological disorders **(33)**.

Apnea is cessation of airflow for 10 or more seconds. Hypopnea differs from apnea in that during a hypopneic event, airflow is diminished but not absent **(34)**.

Pathophysiology of OSA

It is well recognized that airflow obstruction in obstructive sleep apnea is caused by collapse of the pharynx which is caused by a complex interaction of structural and neuromuscular factors. It is difficult to determine the precise mechanisms in which structural and neuromuscular factors cause pharyngeal airflow obstruction

(39). Investigators have examined the biomechanics of pharyngeal airflow obstruction. They have likened the development of pharyngeal airflow obstruction to that of flow obstruction in a simple collapsible tube such as a Penrose drain. The patency of a simple collapsible tube is largely determined by the gradient of pressure across its wall; therefore, the pharynx can be either patent or occluded, depending on the pressure exerted by tissues surrounding its lumen (40).

In practice, the pressure surrounding the pharyngeal airway can be measured in patients during routine nasal continuous positive airway pressure (CPAP) titration studies in a sleep laboratory. As nasal pressure is increased, tidal airflow is monitored, and the surrounding pressure is taken to be the level of nasal pressure at which flow first begins. This nasal pressure is called the Pharyngeal critical pressure (P_{crit}), and is a reflection of the overall collapsibility of the pharynx (41).

This method has been applied to measure pharyngeal critical pressures in normal individuals, snorers, and patients with obstructive hypopneas. From measurements of critical pressures in these groups, it is now recognized that quantitative differences in pharyngeal critical pressure can be discerned among groups across the spectrum of pharyngeal airflow obstruction during sleep from health to disease. From these observations, it is now clear that airflow obstruction in patients with obstructive sleep apnea is characterized by an increase in the pharyngeal critical pressure, indicating that the collapsibility of the pharynx is elevated in these patients during sleep (41).

Major question remains regarding observed differences in pharyngeal collapsibility (critical pressure) during sleep: what is the