Introduction:

Historical notes :

(OSA) described in the classic syndrome (obesity, hypersomnolence) in Charlis Dikens classic work the Pickwick papers. **(Koopmann and Moran., 1990).**

In 1889 William Hill also described this syndrome as his following statements illustrate:" The stupid looking lazy kid who frequently suffers from headaches at school, breathes through his mouth instead of his nose, snores and restless at night, and wakes up with a dry mouth in the morning, is well worthy of the solicitous attention of the school medical officer.

In 1957 a description of the electroencephalogram (EEG) in eye movement patterns of overnight sleep in humans using the term rapid eye movement (REM) sleep in describing the cyclic nature of REM and non REM sleep have been published **(Dement and Klietman., 1957).**

In 1959 Cole and Alexander described the relationship between obesity and chronic hypoventilation and pulmonary hypertension.

In 1965 Menashe et al described, corpulmonale secondary to obstructive tonsil and adenoid. In the same year Gastaut and his associates began to classify sleep induced apnea syndrome based upon the respiratory pattern seen during sleep.

In 1973, sleep apnea as a syndrome was first described by **(Guilleminault et al, 1980).**

Since 1980, sleep disorders (especially obstructive sleep apnea) have become well recognized. Numerous articles have been written in otolaryngology as well as in the pediatrics and internal medicine. Obstructive sleep apnea

Obstructive sleep apnea is a condition defined by repetitive episodes of cessation or reduction of airflow during sleep, associated with arousals from sleep due to complete obstruction of the upper airway, **(*Bahummam* AS et al., 1999).**

Obstructive sleep apnea (OSA) syndrome is a relatively common but fatal disorder affecting 24% percent of healthy middle aged men and 9% of healthy middle-aged women have evidence of obstructive sleep apnea. However 4% of the adult male and 2% of female are symptomatic and have the syndrome **(Bowden MT et al.,2005).**

The upper air way formed by the nose, mouth, the pharynx and the larynx each of these areas has different pathologies that can produce obstructive sleep apnea syndrome. **(Bachour 2004).**

Obstructive sleep apnea is a complex disease whose etiology is multifactorial. Any condition causing narrowing of the upper respiratory tract may lead to obstructive sleep apnea as nasal turbinate hypertrophy, nasopharyngeal adenoidal hypertrophy, elongated or thickened palate or uvula , macroglossia , laryngomalacia . Functional disorder as hypotonia of pharyngeal muscles by long use of sedatives, alcohol may also cause obstructive apnea during sleep (**Li et al., 2004).**

Two main stages of sleep, rapid eye movement (REM) sleep, characterized by the eye movements and an inert body, and non-rapid eye movement (NREM) sleep, which can be divided into four successive stages, the first two considered “light” sleep, or drowsy sleep, and the following two slow wave sleep (SWS), or deep sleep**. (Rinaldi et al., 2001)**

Detection of the sites of upper airway obstruction have been attempted by a variety of techniques including, acoustic reflection

(analyzing reflected sound waves from the respiratory system which provides a calculation of the upper airway area, Fluoroscopy (used to study upper airway closure during sleep in patients with sleep apnea) Cephalometry (a standardized lateral radiograph of the head and neck examining upper airway bony and soft tissue structure), Rhinometrics (evaluate the nasal passages , oropharynx , and vocal cords) , Pharyngomanometry (catheters positioned in the upper airway can measure pressure differences during an apnea to localize the sites of obstruction) , Computed tomography CT (provides excellent imaging of the airway , soft tissue , and bony structures from the nasopharynx to larynx) , Magnetic resonance imaging MRI (provides excellent upper airway and soft tissue resolution including adipose tissue) . **(Robinson et al., 2003).**

Obstructive sleep apnea (OSA) syndrome can affect the patient during sleep, by restless sleep, night mares, nocturnal enuresis and during day time by morning headache, impaired concentration, hyper-somnolence and this may cause cardiac arrhythmia, pulmonary hypertension, systemic hypertension, heart failure and even sudden death **(Fernjndez 2005)**.

Treatment aspects of obstructive sleep apnea divided into non-surgical aspect (conservative and medical) and surgical aspect. **(Watanabe et al., 2002).**

Conservative treatment include lifestyle modifications by identifying and attempting to correct lifestyle issues, **(Senior et al., 2000).**

Weight reduction particularly regional neck obesity is a major risk factor for (OSA) **(Dawson et al .,1997),** avoidance of alcohol and other

respiratory depressants , Smoking cessation as cigarettes smoking is a risk factor for (OSA) **(Weiss et al., 2000)** , Sleep position as the collapsibility of the upper airway during sleep in patient with (OSA) as measured by critical closing pressure is lower in the lateral than in the supine position **(Boudewyns et al., 2001).**

Pharmacological (medical) treatment have been tested, putative mechanisms of action include increased ventilatory drive (eg. acetazolamide), selective activation of upper airway dilator muscles (e.g. strychnine) many of these agents are limited by poor patient tolerance and there use is not supported by randomized controlled trials **(Smith et al ., 2002) .**

Continuous positive air way pressure (CPAP) oxygen supply improves overall oxygenation during sleep but increases apnea duration while reducing apnea frequency **(Eastwood et al. , 2002) .**

Surgical treatment of (OSA) predated the development of CPAP and oral appliances. the aim of surgical treatment is to bypass or remove the site of upper airway obstruction **(Polo-Kantola et al., 2003)** , tracheostomy achieves the goal of bypassing and was utilized as an effective treatment for OSA long before the tracheostomy as the primary treatment for the disease , but disfiguring nature and the attendant long term morbidity of tracheostomy have led to the development of other alternative surgical approaches as , resection of redundant soft tissue, (nasal surgery , uvuloplatopharyngoplasty (UPPP) , laser assisted UPPP and midline glossectomy , induction of scar tissue formation (cautery or radiofrequency ablation of soft palate , tongue or epiglottis) , displacement of bony and ligamentous attachments of upper airway soft

tissue structures , maxillary and mandibular osteotomies , tongue and hyoid suspension **. (Rama et al., 2002).**

Many combinations of surgical procedures coexist in aim to improve the success rates including; 50%success rate in UPPP and tongue –base radiofrequency, 42-59% in UPPP and midline glossectomy, 33-77% in UPPP and genioglossus advancement with or without hyoid myotomy and suspension. The most impressive results of 90% or more occur with combined UPPP, genioglossus advancement, and maxillary – mandibular advancement **(Watanabe et al., 2002).**