INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is well-defined syndrome that includes one or two of the following symptoms :

severe snoring, nocturnal respiratory arrest ,repeated nocturnal awakening, non-recuperative sleep, diurnal fatigue, and altered concentration, these clinical finding are related to the extent of hypoxemia and hypercapnia that develop as a result of disordered breathing (peled et al.,1998).

Obstructive sleep apnea is a common chronic respiratory disorder that occurs in approximately 4% of men and 2% of women of more than 30 years old. Increase in the ratio with age may depend on the role of OSA on the Complications of the disease (**Young et al.,2002**).

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).

Patients with OSAS have a morphological and functional abnormality of The upper respiratory tract, and the obesity of these patients is the main cause for OSAS. Obese patients with OSAS are closely associated with life-Style related diseases and metabolic syndromes. The circulatory system is Most strongly influenced by OSAS and hypertension cerebrovascular disease and cardiovascular disease are the prime complications of OSAS (Muramatsu et al.,2009).

Obstructive sleep apnea syndrome represents a highly prevalent disease and is recognized as a major public health burden. Substantial evidence shows that patients with OSAS have an increased incidence of hypertension compared with individuals without OSA and that OSAS a risk Factor for the development of hypertension (**Parish&somers**, 2009).

Moreover, several studies show that OSA may be implicated in stroke and transient ischemic attacks and may be associated with coronary heart disease, heart failure, and cardiac arrhythmias (comondore et al., 2009). Also, pulmonary hypertension may be associated with OSA, especially in patients with preexisting pulmonary disease, (Zamarron et al., 2008) and (Karkoulias et al., 2010).

The pathogenesis of cardiovascular complications in OSAS is not completely understood, but given the complexity of the disorder, a multifactorial etiology is likely (**Ryan &McNicholas,2009**).

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) and 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, hypersomnolence and this may cause cardiac arrhythmia, pulmonary hypertension, systemic hypertension, heart failure and even sudden death (Femjndez ,2005).

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

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**).

- O 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 (Swith et al.,2002).
- o Tracheostomy achieves the goal of bypassing and was utilized as an -3 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).
- o Continuous positive airway pressure (CPAP) is the primary treatment For OSAS, (Malhotra et al., 2000) since it eliminates upper airway collapse during sleep, and improves sleep fragmentation,

daytime symptoms, (Patel et al., 2003) and quality of life (D'Ambrosio et al., 1999).

Accumulative evidence supports that CPAP also reduces cardiovascular morbidity ,Peker et al.,(2002) and Milleron et al.,(2004) had already underlined the beneficial effect of CPAP treatment on reducing cardiovascular risk .Also, Marin et al.,(2005) reported a significant increase in ischemic cardiac events in patients with untreated severe OSAS compared to those receiving CPAP therapy over a long study period .