NEW TRENDS IN SNORING SLEEP APNEA SYNDROME

ESSAY

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BY

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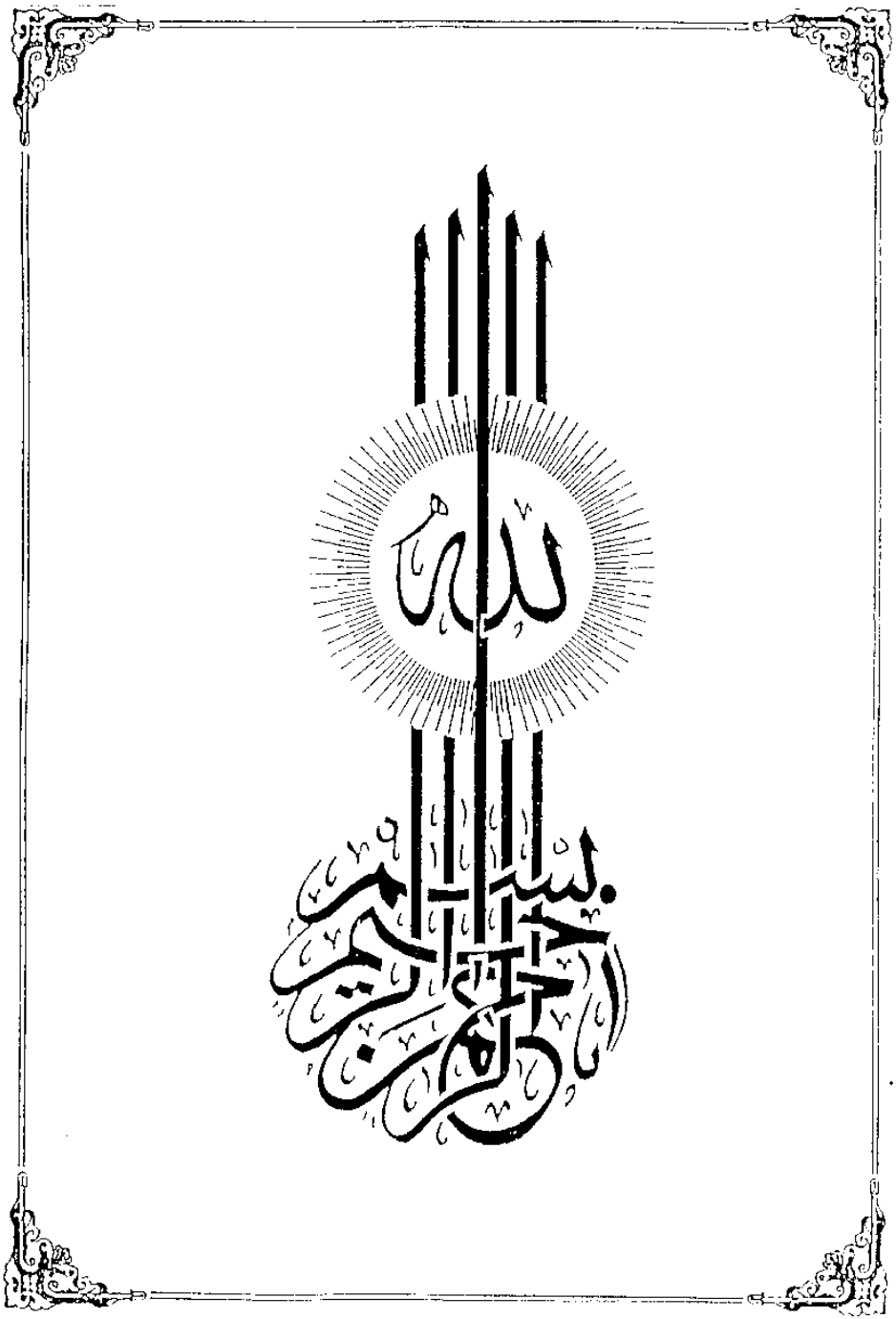
بسم الله الرحمن الرحيم

وأنزل الله عليك الكتاب والحكمة وعلمك ما لم تكن تعلم



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INTRODUCTION

Over the centuries, astute clinicians described syndromes "rediscovered" in the twentieth century. With each wave of investigators, the syndrome has been refined and knowledge of the physiopathology underlying the syndrome has grown. The sleep apnea syndrome offers an excellent example of this historical process. The British physician Caton in 1889 and his French colleague Lamacq in 1897 both observed that "narcoleptics" (i.e., subjects with excessive daytime sleepiness) may suffer from obstructed airways during sleep that lead to "periodic states of suffocation". In 1918, Sir William Osler coined the term "Pickwickian" referring to obese, hypersomnolent patients. In 1936, Kerr and Lagen noted that a significant cardiocirculatory problem could develop in such patients leading to cor pulmonale and complete cardiac failure. In 1956, Burwell et al. reviewed the principal clinical features of the Pickwickian syndrome, but Auchincloss and colleagues had, in 1955, already sparked interest in the possibility of various pathophysiologies leading to syndromes involving obesity, hypersomnolence, and polycythemia.

Between 1959 and 1962, Alexander and colleagues defined a "Joe" type of the Pickwickian syndrome, characterized only by obesity and hypersomnolence. In 1964 in french and 1965 in english, Gastaut and his colleagues reported on the presence of repetitive obstructive apnea during sleep in the obese "Pickwickian". Jung and Kuhlo in 1965 also presented a case report and, in passing, mentioned that the patient had had sleep apnea as long as 10 years before the Pickwickian syndrome appeared. Schwartz and Escande in 1967 performed the first cinematographic study of obstructive apnea in Pickwickians, demonstrating that an oropharyngeal collapse occurs. In 1972, Sadoul and Lugaresi organized the first international meeting on "hypersomnia with periodic breathing". In the following decade, clinicians began applying their knowledge of sleep physiology to the sleep apnea syndromes and tracing the various components underlying the syndromes.

The sleep apnea syndrome is characterized by more than 30 apneic episodes during 8 hours sleep. Apnea is defined as a cessation of airflow at the level of the nostrils and mouth lasting at least 10 seconds. Hypopnea is defined as fall in the tidal volume by more than 50%. The apnea plus hypopnea (partial obstruction) index (A+HI) is the number of abnormal breathing events per hour of sleep. The severity

index (SI) is the number of abnormal breathing events per hour of sleep resulting in oxygen saturation (SaO2) of less than 85%. Three major types of sleep apnea have been described; obstructive (upper airway) sleep apnea, central sleep apnea and mixed (both obstructive and central) sleep apnea. Obstructive sleep apnea (OSA) is characterized by the continuation of respiratory effort during apneic period, while central apnea is marked by loss of the respiratory effort. The fact that the majority of patients exhibit OSAS when studied polygraphically during sleep does not mean that the central sleep apnea syndrome does not exist. A central sleep apnea syndrome often develops into a mixed or obstructive syndrome during the natural evolution of a breathing problem. In fact, all three types of apnea -central, mixed and obstructive- are commonly recorded in a single sleeping patient.

Snoring is the most obvious manifestation of upper airway obstruction during sleep. It is now well recognized as a sign of the potentially lethal condition; OSAS. Since problems related to snoring and anatomic alteration of the upper airway have been considered their primary province, questions considering the diagnosis and treatment of OSAS are becoming frequently encountered by otolaryngologists. Now it is becoming more common for the otolaryngologist's

office to be the entry point into the medical system for patients with SAS, thus the practicing otolaryngologist has been thrust into the fore-front in the diagnosis and management of SAS.

The aim of this essay is to review the literature and the recent advances in the pathogenesis, diagnosis, differential diagnosis, and treatment of SAS, because an awareness of the spectrum of this syndrome, as well as its appropriate management, has become essential for the modern otolaryngologist.

PHYSIOLOGY
AND
ANATOMY

SLEEP AND WAKEFULNESS

Sleep is defined as a state of unconsciousness from which the person can be aroused by appropriate sensory or other stimuli. Therefore, the unconsciousness caused by deep anaesthesia, by total inactivity of reticular activating system in diseased states as coma and by excessive activity of reticular activating system as occurs in grand mal epilepsy would not be considered to be sleep (*Guyton, 1981*).

Normal Sleep and Arousal Mechanisms

The pattern of sleeping varies in the different epochs of life. A nocturnal predominance begins to appear after the first few weeks of postnatal life, resulting in the biphasic pattern of sleeping and waking which persists throughout adolescence and adulthood, unless altered by disease (*Martin, 1983*).

The reticular formation and the reticular activating system

The reticular formation is the phylogenetically old reticular core of the brain stem. It consists of an interlacing network of neurones. Many centres which regulate vegetative functions as respiration, blood pressure, heart rate are located within the reticular formation. It also contains descending tracts which regulate muscle tone and

vegetative functions and ascending tracts which are concerned with consciousness and modulation of sensory input. The reticular activating system is a complex polysynaptic system. Afferent collaterals converging onto it from ascending sensory tracts, trigeminal, auditory, visual, and olfactory systems. Because of the high degree of convergence and the complexity of neurones, the reticular activating system is a non specific system, i.e. the reticular neurones are stimulated equally by different sensory stimuli. This is opposite to the case with ascending sensory pathways which are specific, i.e. their neurones are stimulated by only one type of sensory stimulation. The reticular activating system is concerned with electric activity of the brain. It is also responsible for conscious and alert state that makes perception possible (*Ganong, 1985*).

The Dorsal Thalamus

It is a part of the thalamus that contains two groups of nuclei:

1. Non specific projection nuclei (Midline and interlaminar). They project diffusely to all neocortex. They receive impulses from reticular activating system and are responsible for consciousness, alertness or wakefulness.

2. Specific projection nuclei project to specific areas of the neocortex and limbic system and include nuclei with sensory function as medial geniculate body which relays auditory impulses to auditory cortex, lateral geniculate body which relays impulses to visual cortex, and ventrobasal nuclei which relay somatic sensations to sensory cortex. Also they include nuclei with motor function, limbic function, and integrative function (concerned with language)(*Ganong, 1985*).

Function of the reticular activating system in wakefulness

It is believed that the mesencephalic portion of the reticular activating system is basically responsible for the normal wakefulness of the brain. Electrical stimulation of the mesencephalic portion causes generalized activation of the entire brain, including activation of the cerebral cortex, thalamic nuclei, basal ganglia, hypothalamus, other portions of the brain stem and even the spinal cord. The thalamic portion relays most of the diffuse facilitatory signals from mesencephalon to all parts of the cerebral cortex to cause generalized activation of the cerebrum. Moreover, it causes selective activation of specific cortical areas and possibly or probably plays an important role in our ability to direct attention to certain parts of our medical activity (*Walton, 1983*).

Stimulation of the reticular activating system by cerebral cortex

The cerebral cortex can stimulate the reticular activating system because of large number of nerve fibres that pass from the motor regions of the cerebral cortex to the reticular formation . Motor activity is associated with a high degree of wakefulness, which partially explains the importance of movement to keep a person awake (*Guyton, 1981*).

Sleep Stages and Types

There are two types of sleep;

I. Slow wave sleep (non-rapid eye movement sleep, NREM).

II. Rapid eye movement sleep (REM).

Slow wave sleep is divided into 4 stages (Fig.1)

Stage 1: Low amplitude, fast frequency electroencephalography (EEG) pattern.

Stage 2: Appearance of sleep spindles (bursts of alpha like waves).

Stage 3: High amplitude, slow frequency EEG pattern.

Stage 4: Very high amplitude, very slow frequency EEG.