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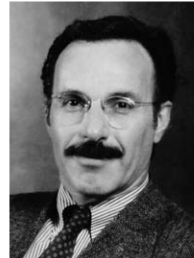
Preface
Sleep-related breathing disorders: new developments



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Guest Editors

Obstructive sleep apnea (OSA) is defined as recurrent episodes of airflow cessation during sleep despite persistence of respiratory effort. It is common in the general population—an estimated 15 million people in the United States are believed to suffer from the disorder. Furthermore, it is widely suspected that sleep-disordered breathing is underdiagnosed in both adults and children.

There are a variety of ways in which sleep-related breathing disorders are classified. In one simple schema, OSA may be considered the extreme end of a spectrum of repetitive sleep-related upper airway (UA) obstructions that includes, in order of severity, intermittent snoring, continuous snoring, UA resistance syndrome, asymptomatic hypopnea, and symptomatic apnea-hypopnea. An American Academy of Sleep Medicine Task Force Report published in 1999 defined four separate syndromes associated with abnormal respiratory events during sleep among adults, namely obstructive sleep apnea-hypopnea syndrome, central sleep apnea-hypopnea syndrome, Cheyne-Stokes breathing syndrome, and sleep hypoventilation syndrome. According to this classification, the UA resistance syndrome was not regarded as a distinct disease; rather, respiratory event related arousals (RERAs) were considered part of OSA.

Sleep state dependency is one of the most important and central features of OSA. During wakefulness, ventilation and oxygenation are generally normal, only to be disrupted during sleep by repetitive UA narrowing or obstruction. The diminished tone of the muscles maintaining UA patency is part of the generalized muscle hypotonia that occurs during sleep. Sleep apnea is terminated by an arousal accompanied by restoration of UA patency and airflow. Sleep state–dependent changes in UA biomechanics and neurophysiology may lead to alterations in the balance between inward forces that favor collapse of the airways and outward forces that counter the former. Not only do persons with OSA tend to have anatomically narrower and physiologically more collapsible UAs, they may also have decreased activity of the UA dilator muscles with which to compensate for the collapse.

Persons with OSA commonly have alternating episodes of loud snoring and periods of silence during sleep due to marked diminution or total absence of airflow. Blood oxygen saturation may drop during the apneic phase. Respiratory events typically recur throughout the evening, at times reaching numbers substantial enough to produce sleep fragmentation and subsequent daytime sleepiness. There is increasing recognition of the potential consequences of this disorder: neuropsychological impairment, adverse effects on quality of life, and seizure disorders, in addition to specific cardiovascular diseases such as hypertension, atherosclerosis, stroke, pulmonary hypertension, cardiac arrhythmia, and congestive heart failure.

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Technological innovations will likely transform the recognition and diagnosis of sleep-disordered breathing events. The overnight, attended, laboratory polysomnography is the generally accepted reference standard for diagnosis. Its limited availability and high costs have prompted the search for alternative sleep study protocols, such as portable sleep monitoring. Accurate monitoring of respiration during sleep, including measurements of airflow, respiratory effort, oxygenation, and ventilation, is indispensable in the identification of sleep-related respiratory events. In particular, measurement of respiratory effort using either esophageal pressure monitoring or surface diaphragmatic electromyography is vital in distinguishing central from obstructive apneas. Today, the sleep clinician has a wide variety of devices available to monitor oro-nasal airflow, including pneumotachometers, nasal pressure monitors, thermal or expired carbon sensors, strain gauges, and respiratory inductance plethysmography.

As we explore the indications for treatment and various options for managing persons with OSA, including behavioral modifications, pharmacological interventions, positive airway pressure devices, oral appliances, and surgery, the challenge is to provide a framework within which we can integrate basic research and clinical data with future therapies for this disorder.

Obesity is strongly correlated with the prevalence of sleep apnea, and weight reduction can be a highly effective short-term treatment. However, recurrence of sleep apnea appears to be common during long-term follow-up, either because of a failure to maintain weight loss or, for reasons that are unclear, despite successful maintenance of weight loss.

Positional modification, using a variety of devices such as posture alarms and wedge pillows to avoid the supine sleep position, appear to be most effective in persons with milder disease. Again, long-term data are sparse and dishearteningly variable.

The search for effective pharmacological targets continues. Currently, none of the agents that has been evaluated to reduce sleep-disordered breathing events is consistently effective to be considered as standard therapy. Identification of excitatory neurotransmitters of the UA dilator motoneurons is actively being pursued. Another area of research is pharmacological intervention using stimulant medications to attenuate residual daytime sleepiness that may persist despite regular use of positive airway pressure (PAP) therapy.

Since its first description in 1981, continuous positive airway pressure (CPAP) therapy has become the main therapy for OSA. It is highly effective, safe, and reliable. PAP therapy most likely acts primarily as a pneumatic splint; it may also decrease pharyngeal collapsibility by augmenting lung volume as well as increase UA length and tension. CPAP is typically titrated during a formal sleep study, determining the pressure at which it will effectively abolish all sleep-disordered breathing in the supine position and in REM sleep. Nevertheless, significant intra- and inter-night variability exists in the severity of sleep-disordered breathing and the corresponding corrective PAP settings. A new generation of PAP devices, referred to as *automated PAPs*, are capable of detecting signals serving as surrogates of UA obstruction (eg, snores, apneas, hypopneas, or airflow limitation) and, using model-specific diagnostic and therapeutic algorithms, responding to changes in airway resistance by either increasing or decreasing the pressures generated. Whether or not they are appropriate, automated-PAPs are being increasingly used to diagnose and treat OSA or to titrate pressures for conventional CPAP devices.

Oral devices, including tongue repositioning devices and mandibular repositioning appliances, are established therapies for primary snoring and milder forms of OSA. Some persons with more severe sleep-disordered breathing may also respond favorably to these devices. Oral appliances are becoming increasingly popular because of their ease of use, portability, and reversibility. Increased understanding of their mechanisms of actions (including effects of UA patency and muscle function), indications of therapy, predictors of treatment outcome, and complications will help clarify their roles in the management of patients.

Surgery remains an option for many patients, especially those who are either unwilling to try, or are intolerant of, positive pressure therapy. Advances in surgical techniques have significantly improved outcomes. Selection among the various surgical procedures is individualized, tailored primarily to the anatomical region of narrowing or obstruction. Thus, uvulopalatopharyngoplsty is commonly performed for oropharyngeal obstruction, whereas surgical alterations of the tongue, hyoid, and maxillomandibular complex are attempted for hypopharyngeal airway obstruction. The role of radiofrequency UA soft tissue ablation is still being debated.

Dionysius of Heracleia (born 360 BC) was described by Athenaeus as " . . . an unusually fat man . . . sleepy, difficult to arouse and had problems breathing . . .so [his] physicians prescribed . . . fine needles, long enough that they thrust through his ribs and belly when he happened to fall into a very deep sleep . . . " Could this be how OSA was treated then? If so, we would like to believe that over the past

2400 years there has been some progress in our understanding and management of this disorder.

The purpose of this issue of the *Clinics in Chest Medicine* is to provide a comprehensive discussion of the various aspects of OSA, focusing on new developments and controversies and emphasizing trends that may potentially offer a glimpse of the future of the science and practice of sleep medicine. We hope that readers find this issue to be clinically useful, and we welcome all feedback.

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