to ~20 mmHg. The ensuing onset of chest pain, breathlessness, paresthesia, or altered consciousness can be alarming. The resultant increase in minute volume to relieve these acute symptoms only serves to exacerbate symptoms that are often misattributed by the patient and health care workers to cardiopulmonary disorders. An unrevealing evaluation for causes of these symptoms often results in patients being anxious and fearful of additional attacks. It is important to note that **anxiety disorders and panic attacks are *not* synonymous with hyperventilation**. Anxiety disorders can be both an initiating and sustaining factor in the pathogenesis of chronic hyperventilation, but these are not necessary for the development of chronic hypocapnia.

DIAGNOSIS

Respiratory symptoms associated with acute hyperventilation can be the initial manifestation of systemic illnesses such as diabetic ketoacidosis. Causes of acute hyperventilation need to be excluded before a diagnosis of chronic hyperventilation is considered. Arterial blood gas sampling that demonstrates a compensated respiratory alkalosis with a near normal pH, low Pa_{CO_2}, and low calculated bicarbonate is necessary to confirm chronic hyperventilation. Other causes of respiratory alkalosis, such as mild asthma, need to be diagnosed and treated before chronic hyperventilation can be considered. A high index of suspicion is required because increased minute ventilation can be difficult to detect on physical examination. Once chronic hyperventilation is established, a sustained 10% increase in alveolar ventilation is enough to perpetuate hypocapnia. This increase can be accomplished with subtle changes in the respiratory pattern, such as occasional sigh breaths or yawning two to three times per minute.

TREATMENT HYPERVENTILATION

There are few well-controlled treatment studies of chronic hyperventilation due to its diverse features and the lack of a universally accepted diagnostic process. Clinicians often spend considerable time identifying initiating factors, excluding alternative diagnoses, and discussing the patient's concerns and fears. In some patients, reassurance and frank discussion about hyperventilation can be liberating. Identifying and eliminating habits that perpetuate hypocapnia, such as frequent yawning or sigh breathing, can be helpful. Some evidence suggests that breathing exercises and diaphragmatic retraining may be beneficial for some patients. The evidence for using medications to treat hyperventilation is scant. Beta blockers may be helpful in patients with sympathetically mediated symptoms such as palpitations and tremors.

Acknowledgment

We would like to acknowledge Eliot A. Phillipson for earlier versions of this chapter and Jan-Marino Ramirez for his careful critique and helpful suggestions.

319 Sleep Apnea

Andrew Wellman, Susan Redline

Obstructive sleep apnea/hypopnea syndrome (OSAHS) and central sleep apnea (CSA) are both classified as sleep-related breathing disorders. OSAHS and CSA share some risk factors and physiological bases but also have unique features. Each disorder is associated with impaired ventilation during sleep and disruption of sleep, and each diagnosis requires careful elicitation of the patient's history, physical examination, and physiological testing. OSAHS, the more common disorder, causes daytime sleepiness, impairs daily function, and is a major contributor to cardiovascular disease in adults and to behavioral problems in children. CSA is less common and may occur in combination with obstructive sleep apnea, as a primary condition, or secondary to a medical condition or medication. CSA impairs overnight gas exchange and may result in symptoms of either insomnia or excessive sleepiness.

OBSTRUCTIVE SLEEP APNEA/HYPOPNEA SYNDROME (OSAHS)

Definition OSAHS is defined on the basis of nocturnal and daytime symptoms as well as sleep study findings. Diagnosis requires the patient to have (1) either symptoms of nocturnal breathing disturbances (snoring, snorting, gasping, or breathing pauses during sleep) or daytime sleepiness or fatigue that occurs despite sufficient opportunities to sleep and is unexplained by other medical problems; and (2) five or more episodes of obstructive apnea or hypopnea per hour of sleep (the apnea-hypopnea index [AHI], calculated as the number of episodes divided by the number of hours of sleep) documented during a sleep study. OSAHS also may be diagnosed in the absence of symptoms if the AHI is above 15. Each episode of apnea or hypopnea represents a reduction in breathing for at least 10 sec. OSAHS is often identified when associated with a ≥3% drop in oxygen saturation and/or a brain cortical arousal. OSAHS severity is based on the frequency of breathing disturbances (AHI), the amount of oxygen desaturation with respiratory events, the duration of apneas and hypopneas, the degree of sleep fragmentation, and the level of daytime sleepiness.

Pathophysiology During inspiration, intraluminal pharyngeal pressure becomes increasingly negative, creating a "suctioning" force. Because the pharyngeal airway has no bone or cartilage, airway patency is dependent on the stabilizing influence of the pharyngeal dilator muscles. Although these muscles are continuously activated during wakefulness, neuromuscular output declines with sleep onset. In patients with a collapsible airway, the reduction in neuromuscular output results in transient episodes of pharyngeal collapse (manifesting as an "apnea") or near collapse (manifesting as a "hypopnea"). The episodes of collapse are terminated when ventilatory reflexes are activated and cause arousal, thus stimulating an increase in neuromuscular activity and opening of the airway. The airway may collapse at various levels: the soft palate (most common), tongue base, lateral pharyngeal walls, and/or epiglottis (Fig. 319-1). OSAHS may be most

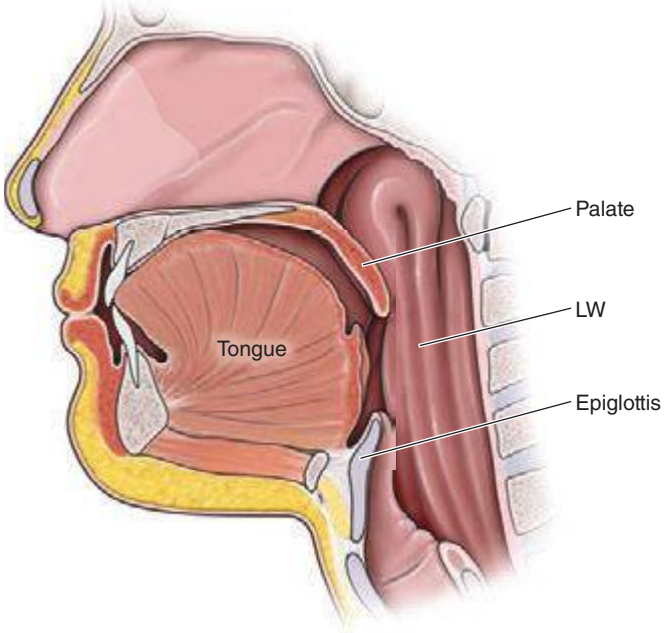

FIGURE 319-1 Common sites of airway collapse. For example, the palate, tongue, and/or epiglottis (Ep) can be posteriorly displaced, and the lateral pharyngeal walls (LW) can collapse.