PHYSICAL FINDINGS

Physical findings often reflect the etiologic factors for the disorder as well as comorbid conditions, particularly vascular disease. On examination, patients may exhibit hypertension and regional (central) obesity, as indicated by a large waist and neck circumference. The oropharynx may reveal a small orifice with crowding due to an enlarged tongue, a low-lying soft palate with a bulky uvula, large tonsils, a high arched palate, and/or micro/retrognathia. Since high-level nasal resistance can increase pharyngeal collapsibility, the nasal cavity should be inspected for polyps, septal deviation, and other signs of obstruction. Because patients with heart failure are at increased risk for both OSAHS and CSA, a careful cardiac examination should be conducted to detect possible left- or right-sided cardiac dysfunction. Evidence of cor pulmonale suggests severe OSAHS or a comorbid cardiopulmonary condition. A neurologic evaluation is needed to evaluate for conditions such as neuromuscular and cerebrovascular diseases, which increase OSAHS risk.

LABORATORY FINDINGS

Diagnostic Findings Since symptoms and signs do not accurately predict the severity of sleep-related breathing disturbances, specific diagnosis and categorization of OSAHS severity require objective measurement of breathing during the period of sleep. The gold standard for diagnosis of OSAHS is an overnight polysomnogram (PSG). A negative in-laboratory PSG rules out OSAHS except in unusual circumstances—e.g., with insufficient REM sleep or supine sleep. Home sleep tests that record only a few respiratory and cardiac channels commonly are used as a cost-effective means for diagnosing patients without significant comorbidity who have a high pretest probability of OSAHS. However, a home study may yield a false-negative result if sleep time is not accurately estimated, and further evaluation may therefore be required.

The key physiological information collected during a sleep study for OSAHS assessment includes measurement of breathing (changes in airflow, respiratory excursion), oxygenation (hemoglobin oxygen saturation), body position, and cardiac rhythm. In addition, PSGs and some home sleep studies measure sleep continuity and sleep stages (by electroencephalography, chin electromyography, and electro-oculography), limb movements (by leg sensors), and snoring intensity. This information is used to quantify the frequency and subtypes of abnormal respiratory events during sleep as well as associated changes in oxygen saturation, arousals, and sleep stage distributions. Tables 319-1 and 319-2 define the respiratory events scored and the severity guidelines employed during a sleep study. Figure 319-2 shows examples of sleep-related respiratory events. A typical sleep study report provides quantitative data such as the AHI and the profile of oxygen saturation over the night (mean, nadir, time at low levels). Reports may also include the respiratory disturbance index, which includes the number of respiratory effort–related arousals in addition to the number of apneas plus hypopneas. In-laboratory PSG also quantifies sleep latency (time from "lights off" to first sleep onset), sleep efficiency (percentage of time asleep relative to time in bed), arousal index (number of cortical arousals per hour of sleep), time in each sleep stage, and periodic limb movement index. OSAHS severity can be further characterized according to the degree of sleep fragmentation associated with respiratory disturbances. Relevant metrics include the frequency of cortical micro-arousals or awakenings per sleep hour, reduction in sleep continuity (low sleep efficiency), reduction of time in deeper stages of sleep (stage N3 and REM sleep) and increases in light sleep (stage N1). The detection of autonomic arousals, such as surges in blood pressure, changes in heart rate, and abnormalities in cardiac rhythm, also provides relevant information on OSAHS severity.

Other Laboratory Findings Various imaging studies, including cephalometric radiography, MRI, CT, and fiberoptic endoscopy, can be used to identify anatomic risk factors for OSAHS. Cardiac testing may yield evidence of impaired systolic or diastolic ventricular function or abnormal cardiac structure. Overnight blood pressure monitoring often displays a "non-dipping" pattern (absence of the typical 10-mmHg fall during sleep from blood pressure while awake). Arterial blood gas measurements made during wakefulness are usually normal. Waking hypoxemia or hypercarbia suggests coexisting lung disease or hypoventilation syndrome. Patients with severe nocturnal hypoxemia may have elevated hemoglobin values. A multiple sleep latency test or a maintenance of wakefulness test can be useful in quantifying sleepiness and helping to distinguish OSAHS from narcolepsy.

TABLE 319-1 RESPIRATORY EVENT DEFINITIONS

- *Apnea:* Cessation of airflow for ≥10 sec during sleep, accompanied by:
  - Persistent respiratory effort (obstructive apneas, Fig. 319-2*A*), or
  - Absence of respiratory effort (central apneas, Fig. 319-2*B*)
- *Hypopnea:* A ≥30% reduction in airflow for at least 10 sec during sleep that is accompanied by either a ≥3% desaturation or an arousal (Fig. 319-2*C*)
- *Respiratory effort–related arousal (RERA):* A partially obstructed breath that does not meet the criteria for hypopnea but provides evidence of increasing inspiratory effort (usually through pleural pressure monitoring) punctuated by an arousal (Fig. 319-2*D*)
- *Flow-limited breath:* A partially obstructed breath, typically within a hypopnea or RERA, identified by a flattened or "scooped-out" inspiratory flow shape **(Fig. 319-3)**

TABLE 319-2 OBSTRUCTIVE SLEEP APNEA/HYPOPNEA SYNDROME (OSAHS): QUANTIFICATION AND SEVERITY SCALE

- *Apnea-hypopnea index (AHI):*[a] Number of apneas plus hypopneas per hour of sleep
- *Respiratory disturbance index (RDI):* Number of apneas plus hypopneas plus RERAs per hour of sleep
- *Mild OSAHS:* AHI of 5–14 events/h
- *Moderate OSAHS:* AHI of 15–29 events/h
- *Severe OSAHS:* AHI of ≥30 events/h

[a]Each level of AHI can be further quantified by level of sleepiness and associated hypoxemia.

Health Consequences and Comorbidities OSAHS is a major contributor to cardiac, cerebrovascular, and metabolic disorders as well as to premature death. It is the most common medical cause of daytime sleepiness and negatively influences quality of life. This broad range of health effects is attributable to the impact of sleep fragmentation, cortical arousal, and intermittent hypoxemia on vascular, cardiac, metabolic, and neurologic functions. OSAHS-related respiratory events stimulate sympathetic overactivity, leading to acute blood pressure surges during sleep, endothelial damage, and nocturnal as well as daytime hypertension. OSAHS-related hypoxemia also stimulates release of acute-phase proteins and reactive oxygen species that exacerbate insulin resistance and lipolysis and cause an augmented prothrombotic and proinflammatory state. Inspiratory effort against an occluded airway causes large intrathoracic negative pressure swings, altering cardiac preload and afterload and resulting in cardiac remodeling and reduced cardiac function. Hypoxemia and sympathetic-parasympathetic imbalance also may cause electrical remodeling of the heart and myocyte injury.

HYPERTENSION OSAHS can raise blood pressure to prehypertensive and hypertensive ranges, increase the prevalence of a non-dipping overnight blood pressure pattern, and increase the risk of resistant hypertension. Elevations in blood pressure are due to augmented sympathetic nervous system activation as well as alterations in the rennin–angiotensin–aldosterone system and fluid balance. Treatment of OSAHS with nocturnal continuous positive airway pressure (CPAP) has been shown to reduce 24-h ambulatory blood pressure. Although the overall impact of CPAP on blood pressure levels is relatively modest (averaging 2–4 mmHg), larger improvements are observed among patients with high AHIs and sleepiness.