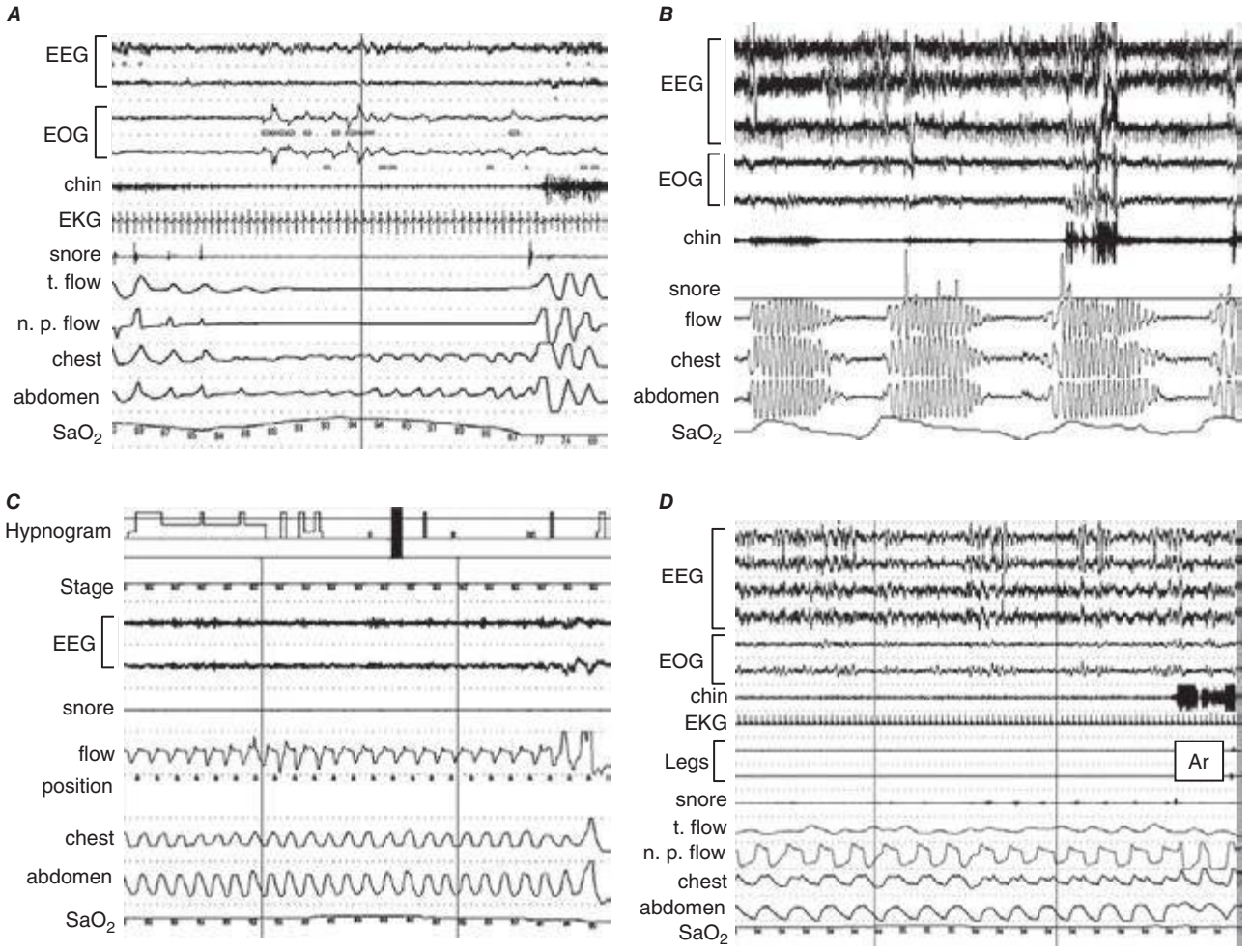

FIGURE 319-2 ***A.*** Obstructive apnea. There are 30 sec of no airflow, as shown in the nasal pressure (n. p. flow) and thermistor-measured flow (t. flow). Note the presence of chest-abdomen motion, indicating respiratory effort against an occluded airway. ***B.*** Central apnea in a patient with Cheyne-Stokes respiration due to congestive heart failure. The flat chest-abdomen tracings indicate the absence of inspiratory effort during the central apneas. ***C.*** Hypopnea. Partial obstruction of the pharyngeal airway can limit ventilation, leading to desaturation (a mild decrease in this patient, from 93% to 90%) and arousal. ***D.*** Respiratory effort–related arousal (RERA). Minimal flow reduction terminated by an arousal (Ar) without desaturation constitutes a RERA. EEG, electroencephalogram; EOG, electro-oculogram; EKG, electrocardiogram.

CARDIOVASCULAR, CEREBROVASCULAR, AND METABOLIC DISEASES Among the most serious health consequences of OSAHS is its impact on cardiac and metabolic functions. Strong epidemiologic evidence indicates that OSAHS significantly increases the risk of coronary artery disease, heart failure with and without reduced ejection fraction, atrial and ventricular arrhythmias, atherosclerosis and coronary artery disease, stroke, and diabetes. Treatment of OSAHS has been shown to reduce several markers of cardiovascular risk, improve insulin resistance, decrease the recurrence rate of atrial fibrillation, and improve various outcomes in patients with active cardiovascular disease. Large-scale trials are under way to evaluate the role of OSAHS treatment in reducing cardiac event rates and in prolonging the survival of patients with cardiac disease.

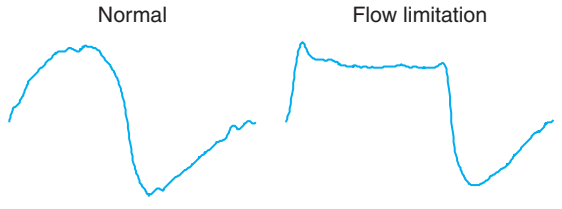

FIGURE 319-3 **Example of flow limitation.** The inspiratory flow pattern in a patent airway is rounded and peaks in the middle. In contrast, a partially obstructed airway exhibits an early peak followed by mid-inspiratory flattening, yielding a scooped-out appearance.

SLEEPINESS More than 50% of patients with moderate to severe OSAHS report daytime sleepiness. Patients with OSAHS symptoms have a twofold increased risk of occupational accidents. Individuals with elevated AHIs are involved in motor vehicle crashes as much as seven times more often than persons with normal AHIs. Randomized controlled trials have shown that treatment of OSAHS with nasal CPAP therapy alleviates sleepiness as measured by either questionnaire or objective testing. However, the degree of improvement varies widely. Residual sleepiness may be due to several factors, including suboptimal treatment adherence, insufficient sleep time, other sleep disorders, or prior hypoxic-mediated damage in brain areas involved in alertness. Visceral adipose tissue, whose amounts are increased in patients with OSAHS, releases somnogenic cytokines that may contribute to sleepiness. Thus, even after treatment, it is important to assess and monitor patients for residual sleepiness and to evaluate the necessity of optimizing treatment adherence, improving sleep patterns, and identifying other disorders contributing to sleepiness.

QUALITY OF LIFE AND MOOD Reductions in health-related quality of life are common in patients with OSAHS, with the largest decrements on the physical and vitality subscales. Treatment with CPAP often results in improvement in these patient-reported outcomes. Depression, in particular symptoms of somatic depression (irritability, fatigue, lack of energy) is commonly reported in OSAHS.