A 37-year-old man with childhood asthma presents to his pulmonologist with complaint of increasing shortness of breath. On physical examination, blood pressure (BP) is 142/94 mm Hg. He is 300 pounds. The rest of the examination is noted for mild expiratory wheezing. He is prescribed a metered dose bronchodilator and is asked to return in 1 week for repeat examination. Upon follow-up, he is no longer short of breath, and the wheezing is significantly diminished. However, BP is 150/98 mm Hg. Antihypertensive therapy is initiated with diltiazem and hydrochlorothiazide. Nevertheless, there is no significant reduction in BP after several more visits with further titration of medication. Additional history, focusing on sleep patterns, is obtained. The patient and his family report heavy snoring, early morning headaches, dry mouth and hypersomnolence. Overnight polysomnography is performed, with a pertinent recording illustrated above.

The upper box (arrow) summarizes the total sleep pattern from midnight (0:00 h) to 9:00 AM (AWK = awake, REM = rapid eye movement, and sleep stages 1–4). The frequent spikes indicate a disruptive sleep pattern. The vertical line between 0:00 h and 3:00 AM is detailed in the figure (1:13
AM). It is represented in the **first five lines** by electroencephalogram (EEG) tracings, followed by the nasal/oral airflow tracing, the thorax and abdomen motion tracings and the oxygen saturation measurements in the **lower left corner** of the illustration. The nasal/oral airflow tracing shows significant absence of airflow in the **boxed region labeled B**. There is also paradoxical motion of the abdomen as compared to the motion of the thorax (illustrated in **box C**). These findings indicate obstructive airflow. Oxygen saturation decreases from 99.7% to 91.8%. Immediately after this period is a sudden increase in airway movement on the nasal/oral tracing with concomitant arousal signals on the EEG tracings (illustrated in the **box labeled A**). The abdominal and thoracic movement tracings are now synchronized. Over the course of this polysomnography, 68 of these apneic episodes were recorded.

Even minimal degree of sleep-disordered breathing can increase BP and may contribute to hypertension in 5% of hypertensive patients. Significant sleep-disordered breathing may cause an increase in BP in relatively lean individuals as well as those who are obese. Changes in oxygen concentration, carbon dioxide concentration, and blood pH cause an increase in catecholamine production. Furthermore, carotid chemoreceptors are stimulated producing a vasomotor center reflex, which with the catecholamines result in an increase in total peripheral resistance. The cyclic ventilatory pattern of sleep apnea also causes tachycardia and increased venous return, which result in an increase in cardiac output. The combination of an increase in total peripheral resistance and cardiac output cause hypertension. Nighttime continuous positive airway pressure (CPAP) has been shown to lower BP in these patients. After institution of CPAP, BP returned to normal, and this patient was able to stop taking antihypertensive medication.

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**References**