heightened airway reactivity in asthma. It is unlikely that asthma is caused by either a single cell or a single inflammatory mediator; rather, it appears that asthma results from complex interactions among inflammatory cells, mediators, and the cells and tissues present in the airways (Liu, Covar, Spahn, et al, 2016). However, recognition of the importance of inflammation has made the use of antiinflammatory agents a key component of asthma therapy.

Another important component of asthma is bronchospasm and airflow obstruction. The mechanisms responsible for the obstructive symptoms in asthma include (1) inflammatory response to stimuli; (2) airway edema and accumulation and secretion of mucus; (3) spasm of the smooth muscle of the bronchi and bronchioles, which decreases the caliber of the bronchioles; and (4) airway remodeling, which causes permanent cellular changes (Liu, Covar, Spahn, et al, 2016) (Fig. 21-7).

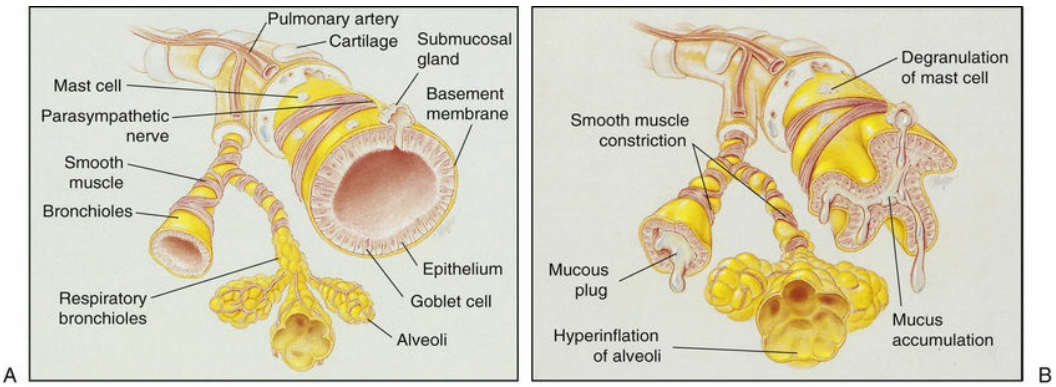

FIG 21-7 Airway obstruction caused by asthma. **A,** A normal lung. **B,** Bronchial asthma: Thick mucus, mucosal edema, and smooth muscle spasm causing obstruction of small airways; breathing becomes labored, and expiration is difficult. (Modified from Des Jardins T, Burton GG: *Clinical manifestations and assessment of respiratory disease,* ed 3, St Louis, 1995, Mosby.)

Airflow is determined by the size of the airway lumen, degree of bronchial wall edema, mucus production, smooth muscle contraction, and muscle hypertrophy. Bronchial constriction is a normal reaction to foreign stimuli; but with asthma, it is abnormally severe, producing impaired respiratory function. Because the bronchi normally dilate and elongate during inspiration and