exposures (see discussion of RADS above) can lead to long-lasting or permanent symptoms.

Viral Infections Most asthmatics report that asthma exacerbations can be triggered by upper respiratory infections. The inflammation that occurs may be neutrophilic as well as eosinophilic. There is some evidence that IgE generation may reduce production of interferon, possibly predisposing to the effects of upper respiratory viruses. Increased airway reactivity after viral infections generally persists for 4–6 weeks but, in some cases, may be associated with permanent changes and impairment.

Exercise and Cold/Dry Air Exercise may be a trigger to asthmatic bronchoconstriction in patients with asthma. Hyperventilation that occurs with exercise dries the airway lining, changing the tonicity of lining cells and causing release of bronchoconstrictive mediators. This effect is more prominent the lower the moisture content of the air, and since cold air has a lower absolute moisture content, the lower the temperature of the inspired air, the less exercise is required to induce bronchoconstriction. In addition, cold air may produce airway edema during airway wall rewarming. At routine levels of exercise, these effects are short-lived.

Air Pollution Increased rates of exacerbations have been associated with increased ambient ozone, sulfur dioxide, and nitrogen dioxide, among other air pollutants.

Drugs Beta blockers may trigger bronchospasm even when used solely in ophthalmic preparations. While the more selective beta blockers are safe for most asthmatics, beta blocker use may be a cause of difficult-to-control asthma. Aspirin may precipitate bronchospasm in those with aspirin-exacerbated respiratory disease (see “Special Considerations”). Angiotensin-converting enzyme (ACE) inhibitors (and to a lesser extent angiotensin receptor blockers) may cause cough.

Occupational Exposures In addition to RADS (see above), episodic and/or recurrent exposures to workplace irritants and/or