progesterone and estrogen can increase eosinophil migration into the nasal mucosa in contrast to testosterone, which decreases eosinophils in the nasal mucosa. This association of hormones with eosinophils may account for the greater prevalence and severity of rhinitis in females following puberty.[144] Rhinitis associated with pregnancy presents with congestion and while this may be secondary to an increase in estrogen and progesterone, the exact mechanism is not known.[145,146] Other endocrine diseases such as hypothyroidism and acromegaly also have been associated with nasal congestion.[72]

Drug-induced rhinitis

Drug-induced rhinitis can be classified based on proposed mechanism of action as local inflammatory, neurogenic, and idiopathic.[147] An acute inflammatory response may be induced following the ingestion of acetylsalicylic acid or other NSAIDs with isolated nasal symptoms or nasal symptoms as part of the NSAID-exacerbated respiratory disease with acute asthma symptoms and associated CRS with nasal polyposis. Disruption of the sympathetic and parasympathetic tone by alpha- and beta-adrenergic blockers produce rhinorrhea and nasal congestion through a neurogenic mechanism. The responsible pharmacological agents may be (1) centrally acting sympatholytic (eg, clonidine, reserpine, and methyldopa); (2) peripherally acting sympatholytic (eg, guanethidine and phentolamine); (3) ganglion-blocking (eg, trimethaphan); or (4) vasodilators, phosphodiesterase type-5 inhibitors (eg, sildenafil).[147] No mechanism has been clearly identified because there are many drugs that can produce nasal symptoms, such as calcium channel blockers, angiotensin-converting enzyme inhibitors, gabapentin, and psychotropics (eg, risperidone and chlorpromazine).[147,148] The effect of exogenous estrogens and oral contraceptives on nasal physiology is uncertain although it has been suggested that oral contraceptives may reduce allergen-provoked nasal congestion during ovulation but increase sneezing at the end of the menstrual cycle.[149-151] Overuse of topical decongestants can result in rhinitis medicamentosa, a form of drug-induced rhinitis, which is further discussed in the intranasal decongestants section.

Work-related rhinitis

Work-related rhinitis comprises (1) *de novo* occupational rhinitis (due to exposures from a particular occupational environment, not usually encountered outside the work environment) and (2) work-exacerbated rhinitis (preexisting or concurrent AR or NAR that is worsened by workplace exposures). Most occupational rhinitis is due to high molecular weight agents (>10 kDa) and is IgE- and T_H2 cell–driven. Low molecular weight (<10 kDa) occupational sensitizers may also induce occupational rhinitis symptoms through mechanisms without associated IgE.[72,152] Following specific inhalation challenge, when compared with low molecular weight agents, high molecular weight agents produced a significantly higher level of acute-phase reactant proteins, cell adhesion molecules, endothelial growth factors, and vitamin D binding proteins.[153] In work-exacerbated rhinitis, aggravation of rhinitis symptoms is often caused by nonallergic irritant triggers, such as from cold dry air, dust particles, smoke, chemicals, or strong odors. Rarely, when a single high-level exposure or multiple low-dose exposures to an irritant gas, vapor, dust, or smoke results in chronic rhinitis, this is referred to as reactive upper airways dysfunction syndrome. In nasal mucosa biopsies of individuals exposed to chlorine dioxide, pathological changes found include lymphocytic inflammation of the lamina propria, epithelial desquamation, and increased number of nerve fibers.[154] Analogous to irritant-induced asthma/reactive airways dysfunction syndrome,[155] the predominant basis for making the diagnosis of reactive upper airways dysfunction syndrome is based on occupational history.

Atrophic rhinitis

Atrophic rhinitis is a chronic nasal condition associated with atrophy of the nasal mucosa and paradoxically presents with nasal congestion due to a sensation of decreased airflow, likely a result of decreased airflow resistance. Atrophic rhinitis can be categorized as primary or secondary. While the pathophysiology of primary atrophic rhinitis is unknown, it is associated with mucosal colonization, predominantly with *Klebsiella ozaenae*, although other organisms have also been described. Primary atrophic rhinitis is more commonly seen in young to middle-aged adults in developing countries with dry climates, such as Saudi Arabia, China, Africa, and India, and is uncommon in the United States and Europe.[156] One US study of patients with atrophic rhinitis categorized approximately 19% of them as having primary atrophic rhinitis; the mean age of this primary atrophic rhinitis group was 52 years.[156] It is characterized by progressive atrophy of the nasal mucosa, resorption of underlying bone and turbinates, nasal dryness, and foul-smelling nasal crusts associated with a constant awareness of a bad smell. Biopsy findings consist of squamous metaplasia, glandular cell atrophy, and loss of pseudostratified epithelium. By definition, there is no history of nasal surgery or trauma in primary atrophic rhinitis as is often the case in secondary atrophic rhinitis.

Secondary atrophic rhinitis is more common in the United States and less severe than primary atrophic rhinitis. Secondary atrophic rhinitis often develops as a result of excessive nasal surgery, trauma, irradiation, or chronic granulomatous nasal infections. Therefore, patients with secondary atrophic rhinitis for which an iatrogenic cause has not been determined should be evaluated for an underlying inflammatory systemic disease (eg, leprosy, sarcoidosis, or syphilis). Repeated, and often radical, sinonasal surgeries for CRS, allergic fungal rhinosinusitis, and/or nasal sarcoidosis produce a widening of the nasal vault, referred to as an "empty nose syndrome."[157] The empty nose syndrome, as may occur after aggressive resection of the inferior and sometimes middle turbinates, is associated with the perception of severe nasal obstruction and inability to sense airflow through the nose. It is "paradoxical" because examination typically finds widely patent nasal cavities and nasal resistance as assessed by rhinomanometry is normal or low. Some patients sense profound dyspnea even though there is no pulmonary disease.[158,159]

Treatment has traditionally focused on reduction of crusting.[156,160] Conservative treatment can consist of nasal saline irrigation, glycerin-containing nose drops, nasal emollients, antibiotics, and vasodilators.[157] Surgical interventions attempt to decrease the size of the nasal cavities thereby promoting regeneration and increasing lubrication of the nasal mucosa and improving nasal vascularity. This can be achieved by surgically closing the nasal cavities (modified Young procedure) or implanting prostheses submucosally to decrease nasal cavity size.[157,161]