Occupational Rhinitis and Other Work-Related Upper Respiratory Tract Conditions

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KEYWORDS

• Occupational rhinitis • Occupational asthma • Sinusitis • Sinonasal cancer • Olfactory dysfunction • Vocal cord dysfunction • Sensory irritation • Allergy

KEY POINTS

- The upper airway serves as an air conditioner, filter, and sensory monitor.
- Irritants and allergens can impact the upper airway.
- According to the "unified airway" hypothesis, the development of occupational allergic rhinitis may herald the onset of occupational asthma, and airway irritant exposures may also contribute to both conditions.
- Other occupational upper airway conditions include sinusitis, nasal erosions, sinonasal cancer, olfactory dysfunction, and vocal cord dysfunction.

INTRODUCTION

The upper airway acts as a sentinel for the respiratory tract, alerting individuals to the physical and chemical qualities of inspired air. It also acts as a filter and air conditioner, and plays an important role in communication. Common occupational upper airway conditions include rhinitis, sinusitis, laryngitis, and vocal cord dysfunction (VCD). Less common are nasal erosions, sinonasal neoplasms, and chemically induced olfactory dysfunction. Etiologic agents range from those specific to occupational settings (eg, chromic acid in the case of nasal erosions) to more ubiquitous environmental agents, such as office dust, cold air, or second-hand tobacco smoke. The epidemiology, pathophysiology, diagnosis, and treatment of occupational upper airway conditions, in particular occupational rhinitis, are reviewed in this article.

ANATOMY OF THE UPPER AIRWAY

The upper airway refers to the airway above the vocal folds, including nasal cavities, nasopharynx, oropharynx, and hypopharynx. Along with the oral cavity, the oropharynx and hypopharynx (and glottis) are sometimes referred to as the "aerodigestive tract."[1] The cofunctionalities of breathing and swallowing dictate that the area be heavily innervated and endowed with a variety of reflex responses.

Anatomically, the lateral walls of the nasal cavity are invested with turbinates or concha (literally, "shells"), the functional consequence of which is to increase the surface area of contact between the mucosa and inspired air. The histology of the nasal cavity has evolved to meet the functional requirements of heat and humidity transfer; biochemical metabolism of inhaled substances; and mucociliary transport of particulate matter to the

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oropharynx (from which it is either swallowed or expectorated). Posterior to the resilient squamous and transitional epithelium of the anterior nares lies a pseudostratified columnar epithelium consisting of ciliated columnar, goblet, and basal cells, and submucous glands.[2]

PHYSIOLOGIC FUNCTIONS

Air Conditioning, Filtration, and Scrubbing

The nose serves as the main portal of entry for the respiratory tract, filtering, scrubbing, physically conditioning inspired air; signaling the quality of the surrounding atmosphere; and playing a role in communication (hearing and phonation). Under most climatic conditions, inspired air is heated and humidified in the upper airway, thereby reducing any thermal or osmotic stress on the tracheobronchial tree.[3]

Filtration of large particles is accomplished mechanically (by nasal vibrissae) and by the process of impaction (whereby particles collide with the turbinates, and are subsequently cleared by the mucociliary apparatus).[4] Finer particles, however, are more likely to evade this clearance system and reach the lower respiratory tract (**Fig. 1**). In the case of inhaled droplets carrying infectious agents, the mucosa produces specific and nonspecific defenses, the former including secretory IgA and the latter including lactoferrin and lysozyme.[5]

Water-soluble irritants, including such gases and vapors as ammonia, organic acids, aldehydes, and chlorine, readily dissolve in mucous membrane water, providing for immediate sensory impact and mass removal.[6] This effect (scrubbing) protects the lower respiratory tract during nasal breathing and incidentally reinforces the sensations of eye, nose, and throat irritation, which can serve as a warning to reduce exposure (**Fig. 2**).

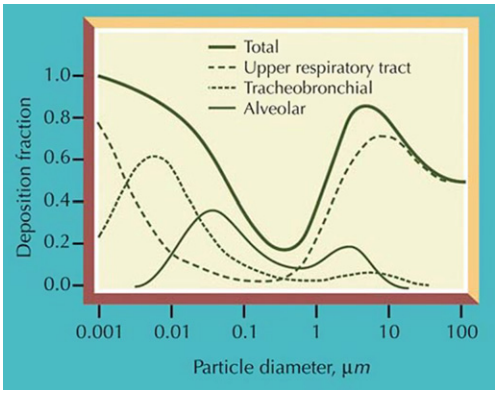

Fig. 1. Fractional deposition of particles in the upper respiratory tract, tracheobronchial tree, and alveolar region of the lung as a function of particle size. (*From* Shusterman D. Toxicology of nasal irritants. Curr Allergy Asthma Rep 2003;3(3):258–65; with permission.)

In contrast to the lower airway, patency in the upper airway is controlled through vascular engorgement rather than smooth muscle tone. Underlying this vasoactivity is an elaborate network of arterioles, capacitance vessels, and arteriovenous shunts located beneath the mucosal surface.[7] Controlling nasal patency (and secretory responses) is a variety of endogenous mediators derived from immune effector cells and mucosal nerves.[8,9]

Sensation and Reflexes

The sentinel function of the nose is achieved through the sense of smell and nasal irritant perception (chemesthesis). These senses are mediated by cranial nerve I (olfactory nerve) and cranial nerve V (trigeminal nerve), respectively (**Fig. 3**). Just as the appreciation of flavor involves a seamless combination of taste and smell, the appreciation of inhaled compounds involves smell and trigeminal stimulation. It is not unusual for an individual to describe “a pungent odor,” and in the process integrate information from two separate cranial nerves.[10]

Peripherally, the terminal branches of the trigeminal nerve include small diameter nociceptive neurons (C- and Aδ-fibers) invested with a variety of nociceptive (pain-perceiving) ion channels.[11] The C-fiber population also elaborates vasoactive neuropeptides, which in turn can be released as part of nociceptive reflexes.[12] Similar neurophysiology applies to the glossopharyngeal and vagal nerves (cranial nerves IX and X), which convey the sense of irritation for the hypopharynx and larynx. A recent development has been the identification of specialized receptor cells (solitary chemoreceptors cells) in the human nose, carrying transduction mechanisms for bitter taste and selected airborne irritants, further linking chemical exposures to airway inflammation.[13]

Reflexes in the upper airway include sneezing, secretion, and nasal obstruction. Upper respiratory tract nerves also participate in the laryngeal adductor reflex, cough, and bronchospasm.[14] Along with cold, dry air, chemical irritants can trigger upper respiratory tract symptoms that are virtually indistinguishable from those of allergic rhinitis, leading to inevitable diagnostic confusion (see later).

PATHOPHYSIOLOGY

Irritation

Upper airway irritation can be defined variously as stimulation of nociceptors (resulting in sensations

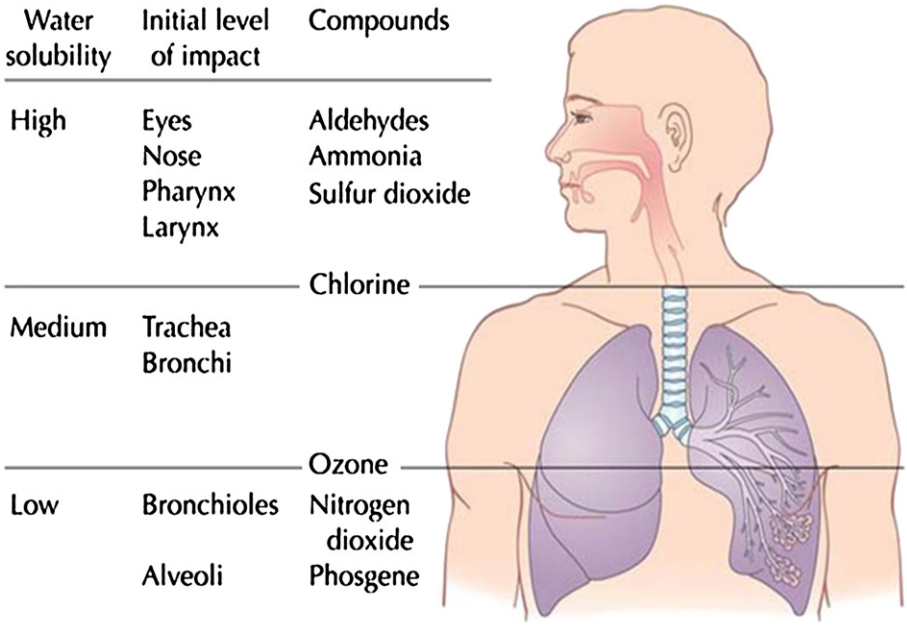

Fig. 2. Water solubility and site of initial impact of airborne irritants. (*From* Shusterman D. Toxicology of nasal irritants. Curr Allergy Asthma Rep 2003;3(3):258–65; with permission.)

of burning, stinging, or tingling); reflex vascular and secretory changes triggered by nerve stimulation; chemically induced tissue damage; or some combination of these.[15] Irritation of the combined mucosal distribution of the trigeminal nerve (eye, nose, and throat) has been termed "sensory irritation," which is also a principal constituent of nonspecific building-related illness (or sick building syndrome).[16] Because of their acute (and reversible) nature and the frequent lack of corresponding physical signs, sensory irritation complaints can be a source of frustration to clinicians and patients. Potential upper airway irritants commonly found in indoor environments include combustion products (from cigarette smoke); volatile organic compounds (from building materials, furnishings, cleaning products, or microbial overgrowth); and reactive chemicals found in household and commercial cleaning products (eg, chlorine and ammonia).[17]

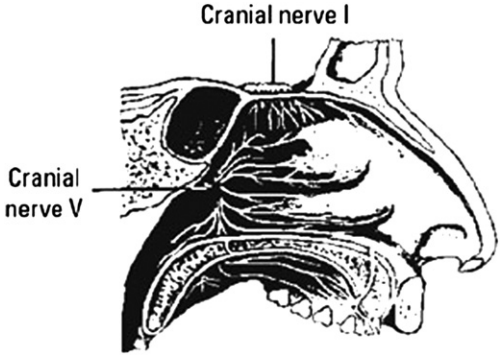

Fig. 3. Innervation of the nasal cavity. Cranial nerve I, olfactory nerve; cranial nerve V, trigeminal nerve. (*From* Shusterman D. The upper airway, including olfaction, as mediator of symptoms. Environ Health Perspect 2002;110(Suppl 4):649–53; with permission.)

Allergy

In contrast to nonspecific irritation, allergic reactions involve hypersensitivity to specific substances (allergens). Irritation can occur on first exposure, whereas hypersensitivity requires a period of asymptomatic exposure during which time cellular or humoral responses develop to the specific allergen. Immediate hypersensitivity refers to a range of IgE-mediated responses, including rhinitis, conjunctivitis, asthma, urticaria, angioedema, and anaphylaxis. Airborne allergens encountered in workplace settings include macromolecules (chiefly proteins) and low-molecular-weight chemical allergens (the mechanism of response to which is less fully understood than for high-molecular-weight allergens).

"Unified Airway" Hypothesis

IgE-mediated hypersensitivity can involve the upper airway (rhinitis) or the lower airway (asthma). The onset of occupational allergic rhinitis often precedes that of asthma in a given individual,

particularly if the sensitizer is a high-molecular-weight antigen.[18,19] Multiple epidemiologic, physiologic, and biochemical observations support a so-called unified airway hypothesis, in which rhinitis and asthma are pathophysiologically linked.[20–22] Furthermore, some investigators have linked skin exposure with airway sensitization, particularly in the case of diisocyanates (a component of polyurethanes).[23]

Interplay Between Irritation and Allergy

Augmentation of upper airway allergy has been demonstrated with several air pollutants. Priming (increased nasal response to allergen challenge after exposure to an irritant air pollutant) has been shown, for example, after ozone exposure.[24,25] Adjuvancy (or boosting of underlying sensitization) has also been shown with diesel exhaust particles and sidestream tobacco smoke.[26–28]

In terms of susceptibility to irritants, the presence of pre-existing allergic inflammation seems to confer greater upper airway sensitivity to air pollutants, including subjective irritation and objective airway obstruction.[29,30] Given the rising prevalence of atopy and allergies in the general population, and the ubiquitous nature of indoor and outdoor air pollutants, interactions between irritants, allergens, and atopy likely play an important role in promoting allergic and irritant rhinitis.

The pathophysiology of rhinitis explains the two main determinants of occupational rhinitis that have been identified in the epidemiology literature: exposure to the causative agents and a history of atopy. Atopy, typically defined as reactivity to common environmental allergens, increases the risk of occupational allergic rhinitis caused by large-molecular-weight allergens, but is not a risk factor for most low-molecular-weight allergens, such as isocyanates.[29] Smoking has not consistently been identified as a risk factor for occupational rhinitis.[29]

UPPER AIRWAY DISORDERS

Occupational Rhinitis

Occupational rhinitis has, until recently, lacked standardization in its clinical definition. In 2009, however, a task force of the European Academy of Allergy and Clinical Immunology proposed a working definition closely resembling that of occupational asthma:

> *Occupational rhinitis is an inflammatory disease of the nose, which is characterized by intermittent or persistent symptoms (ie, nasal congestion, sneezing, rhinorrea, itching), and/or variable nasal airflow limitation and/or hypersecretion due to causes and conditions attributable to a particular work environment and not to stimuli encountered outside the workplace.*[31]

The task force further specified:

> *Work-related rhinitis may be distinguished into: (1) occupational rhinitis that is due to causes and conditions attributable to a particular work environment (2) work exacerbated rhinitis that is pre-existing or concurrent rhinitis exacerbated by workplace exposures.*[31]

Irritant rhinitis

Several different industrial chemicals and manufacturing processes have been associated with irritant rhinitis or sinusitis in workers (**Table 1**). Among these are woodworking; spice grinding; exposure to fuel oil ash, nickel fumes, or dicumylperoxide in industry; and use of glutaraldehyde in medical sterilization.[32–37] Work processes that have occasionally proved problematic to office workers include use of photocopiers, laser printers, and carbonless copy paper.[38,39] Symptoms of irritant rhinitis can include nasal stinging or burning, rhinorrhea, congestion, postnasal drip, sinus headache, and epistaxis.

In polluted urban areas, outdoor workers may be more highly exposed to ambient air pollution than are indoor workers. Several studies have documented the effects of photochemical air pollutants on the upper respiratory tract. Two such studies were performed in a heavily polluted portion of Mexico City, where ozone levels are far in excess of US (and Mexican) standards. These studies compared urban residents with residents of an unpolluted locale, and examined visitors to the city who came from more rural areas. The results were dramatic: permanent residents showed squamous metaplasia, loss of normal cilia, vascular congestion, and glandular atrophy on nasal biopsy, whereas short-term visitors developed epithelial desquamation and neutrophilic inflammation that took more than 2 weeks to resolve after returning to their home towns.[40,41]

Irritant-induced rhinitis has been observed after one-time, high-level exposures to airborne irritants, similar to irritant-induced asthma (or reactive airways dysfunction syndrome).[42] Meggs[43] has coined the term “reactive upper airways dysfunction syndrome” to describe acute onset, irritant-induced rhinitis. Biopsies of the nasal mucosa among individuals acutely exposed to irritants reportedly have shown epithelial desquamation, defective epithelial cell junctions, and increased numbers of nerve fibers, although

Table 1
Selected occupations and associated irritants

Occupation	Irritant
Agricultural workers	Ammonia, nitrogen dioxide, hydrogen sulfide
Custodians	Ammonia, bleach (hypochlorite), chloramines, other cleaning products
Firefighters	Smoke, hazardous materials releases
Food service workers	Cooking vapors, cigarette smoke
Health professionals	Glutaraldehyde, formaldehyde
Laboratory workers	Solvent vapors, inorganic acid vapors or mists
Military personnel	Zinc chloride smoke
Power plant and oil refinery workers	Sulfur dioxide
Printers, painters	Solvent vapors
Pulp mill workers	Chlorine, chlorine dioxide, hydrogen sulfide
Railroad personnel, miners, truck drivers	Diesel exhaust
Refrigeration workers (commercial)	Ammonia
Roofers, pavers	Asphalt vapors, PAHs[a]
Swimming pool service workers	Chlorine, hydrogen chloride, nitrogen trichloride
Teachers and office workers	Cleaning products, printers, copiers
Waste water treatment workers	Chlorine, hydrogen sulfide
Welders	Metallic oxide fumes, nitrogen oxides, ozone
Woodworkers	Wood dust

[a] Polycyclic aromatic hydrocarbons (also skin and lung carcinogen).

patients and control subjects did not differ in staining for neuropeptides.[44]

Nasal septal perforation

Nasal septal perforation is an unusual outcome associated with protracted and high-level exposure to chromates (as in the electroplating industry).[45,46] Chromates (Cr^{6+} compounds) are also of concern with respect to carcinogenesis in the upper and lower respiratory tract (see later). Differential diagnostic considerations in such cases should include such nonoccupational causes as Wegener granulomatosis and recreational drug use (ie, cocaine).

Occupational allergic rhinitis

Allergens responsible for occupational allergic rhinitis are essentially the same as those seen in occupational asthma (ie, various high- and low-molecular-weight sensitizers; **Table 2**). The development of rhinitis may presage the development of asthma; hence, early recognition of occupational allergic rhinitis and timely removal from exposure may interrupt disease progression. Differentiating allergic from irritant rhinitis in the occupational setting may be challenging, however, for a variety of reasons: (1) presenting symptoms (eg, rhinorrhea and nasal obstruction) overlap between the two conditions; (2) there is a paucity of Food and Drug Administration–approved reagents for skin testing or serum immunoassays for specific

Table 2
Selected occupational allergens: rhinitis & asthma

Allergen	Occupation
High molecular weight	
Natural rubber latex	Healthcare workers
Psyllium	Pharmacists, nurses
Animal proteins	Animal handlers, veterinarians
α-Amylase, grain and flour dust	Bakers, grain workers
Insects and mites	Bakers, farm, animal workers
Gum arabic	Printers, food workers
Mold spores	Various
Pollens	Landscapers, florists
Fish, seafood proteins	Fish and seafood workers
Low molecular weight	
Abeitic acid (rosin, pine resin, colophony, solder)	Solderers, gluers
Plicatic acid (Western red cedar), other wood dusts	Wood workers, carpenters
Anhydrides	Plastics workers
Diisocyanates (MDI, TDI, HDI)	Car painters, boat builders, spray foam, construction and shipping workers

occupational allergens; and (3) some substances (eg, formaldehyde, glutaradehyde) can act as both sensitizers and irritants.[31]

Symptomatically, occupational allergic rhinitis commonly presents with nasal pruritus and sneezing, in addition to the less specific symptoms of hypersecretion and obstruction. Reflex secretion or nasal obstruction in response to nonspecific physical and chemical stimuli (termed "nasal hyperreactivity") can occur in the absence of allergy (ie, in nonallergic or vasomotor rhinitis), and is also observed in roughly 40% of allergic rhinitics.[47]

Sinusitis

Few studies have examined the endpoint of sinusitis and occupational exposures. Surveys of furriers, spice workers, vegetable picklers, hemp workers, and grain and flour workers all show increased prevalence rates for self-reported sinusitis.[48–52] Pathophysiologically, the causal sequence for an occupationally induced (or exacerbated) sinusitis may include initial allergic or irritant rhinitis; ciliastasis (with impaired clearance of pathogenic organisms); mucous membrane swelling (with occlusion of sinus ostia and impaired sinus drainage); and infection and mucosal remodeling.

Olfactory Dysfunction

Temporary and long-lasting alterations in olfactory function have been reported among workers exposed to a variety of industrial chemicals. Chemically induced olfactory dysfunction may include quantitative defects, including hyposmia (reduced odor acuity) and anosmia (absent odor perception); or qualitative defects, including olfactory agnosia (decreased ability to identify odors) and various dysosmias (distorted odor perception).

Occupational groups and exposures with which olfactory dysfunction has been associated include alkaline battery workers and braziers (cadmium or nickel exposure); tank cleaners (hydrocarbon exposure); paint formulators (solvent or acrylic acid exposure); and chemical plant workers (ammonia and sulfuric acid exposures).[53–55] In terms of specific olfactory toxicology, hydrogen sulfide produces acute and reversible olfactory paralysis with exposures in excess of roughly 50 parts per million.[56]

Of importance in the differential diagnosis of olfactory dysfunction, competing causes of olfactory impairment include head trauma; chronic nasal obstruction and inflammation caused by rhinitis; postinfectious inflammation; neurodegenerative and endocrine disorders; hepatic and renal disease; neoplasms; various drugs; ionizing radiation; congenital defects (eg, Kallmann syndrome); and selected psychiatric conditions.[57]

Sinonasal Cancer

A variety of occupations and imputed exposures have been linked with the development of malignant neoplasms of the paranasal sinuses. The strongest (and most consistent) associations include cigarette smoking (squamous cell carcinoma) and leather- and wood-dust-exposed workers (adenocarcinoma).[58,59] Workers engaged in nickel refining, chrome refining and plating, and selected aspects of textile and food processing have also been found to be at risk in some studies.[60–62] In addition, the potential of formaldehyde to produce nasopharyngeal cancer in humans is now widely recognized.[63–65]

Vocal Cord Dysfunction

VCD, also referred to as "paradoxic vocal fold motion," is a condition that is frequently confused with asthma. Overlapping symptoms includes episodic dyspnea, cough, and chest tightness. In contrast to asthma, VCD is characterized by inspiratory wheezing (stridor); hoarseness; and a pressure sensation in the throat (globus). VCD involves paradoxic adduction of the vocal cords (folds) during inspiration, as visualized on rhinolaryngoscopy. Alternatively, the condition can be diagnosed on the flow-volume loop with the finding of variable extrathoracic obstruction. Diagnosis is frequently hampered by a lack of reliable provocation maneuvers, although occasional patients with VCD react to inhaled methacholine. A subset of patients with VCD gives a history of initial onset of symptoms in relationship to a one-time, high-level irritant exposure. This diagnostic subgroup has been labeled "irritant-associated VCD."[66]

DIAGNOSIS

Occupational upper airway disorders are diagnosed based on history of exposure at work, physical examination, and for some conditions specialized diagnostic tests. Depending on their availability and degree of standardization, diagnostic techniques are classified here as research versus clinical methods (**Table 3**).[7,67–73]

Occupational and Exposure History

As with any occupational disorder, a careful medical, work, and exposure history is key to recognition and diagnosis. A history of allergies and asthma before the job in question should be clarified. The timing of the onset of symptoms

Table 3
Diagnostic tools for upper airway disorders

General	Specific	Research	Clinical Practice
Medical and exposure history	Occupational and environmental exposure history, temporal relationships between exposures and symptoms	X	X
Questionnaires	Symptom questionnaires	X	X
	Quality-of-life questionnaires	X	X
Direct visualization	Rhinolaryngoscopy	X	X
Allergy testing	In vitro (radioallergosorbent test or enzyme-linked immunosorbent assay)	X	X
	In vivo (skin prick testing)	X	X
Diagnostic radiology	Computerized axial tomography	X	X
Sensory testing	Odor identification (qualitative)	X	X
	Odor detection (quantitative)	X	
Nasal patency	Nasal peak flow	X	X
	Rhinomanometry	X	
	Acoustic rhinometry	X	
	Rhinostereometry	X	
Cytometry	Nasal cytology (curetting)	X	X
	Nasal lavage (cell counts)	X	
Biochemistry	Nasal lavage	X	
Mucociliary clearance	Saccharine transit test	X	

and association with exposures at work, such as improvement away from work, are important to inquire about, as are symptoms among coworkers. As rhinitis becomes more chronic, similar to asthma, patients tend to respond more nonspecifically to a wider array of exposures. Thus, one should inquire about work exposures when rhinitis symptoms first started or became exacerbated.

Questionnaires and Rating Scales

Specialized questionnaires have been developed to document the degree of interference with quality-of-life posed by upper airway allergies. These include the Rhinoconjunctivitis Quality of Life Questionnaire and the Sinonasal Outcome Test.[74,75] These tools can be used to assess symptoms and quality-of-life impairment longitudinally, including documenting the response to therapeutic and environmental interventions.

Physical Examination

Basic physical examination of the upper airway includes anterior rhinoscopy and percussion of the maxillary and frontal sinuses for "tap tenderness." Beyond this basic examination, rhinolaryngoscopy is an easily acquired skill and enables the practiced clinician to visualize the sinus ostia and to more completely evaluate patients for nasal polyposis. Flexible rhinolaryngoscopy also allows for superior visualization of the vocal cords for suspected cases of VCD.

Allergy Testing

The diagnosis of allergic rhinitis is supported by documenting reactivity to the suspect allergen (or mixture). Common practice in North America and in Europe involves either in vivo testing (epicutaneous skin prick) or in vitro serum immunoassays (radioallergosorbent test or enzyme-linked immunosorbent assay) for allergen-specific IgE. Local (nasal) allergen challenge is more commonly performed in Europe than in North America, and has incidentally resulted in the identification of a subset of individuals with positive local challenge but negative evidence for systemic sensitization. This diagnostic subset has given rise to the term "local allergic rhinitis." The implications of local mucosal allergy for occupational rhinitis remain largely unexplored at this time.[76]

Because irritant-associated symptoms, such as nasal congestion and rhinorrhea, may mimic an allergic response, the treating healthcare professional may be faced with a diagnostic challenge in determining responsible etiologic agents and pathophysiologic processes. In contrast to allergy, which typically occurs sporadically among

coworkers, a high prevalence rate of symptoms among coworkers favors a diagnosis of irritant rhinitis. In irritant rhinitis the laboratory work-up is characterized by a lack of systemic eosinophilia, the predominance of neutrophils on nasal smear, and when applicable a lack of in vivo or in vitro reactivity to identified workplace allergens. Air monitoring for airborne irritants may be of assistance in industrial settings, but is more often a source of frustration in the investigation of so-called problem buildings.

Diagnostic Radiology

In diagnostic radiology of the upper respiratory tract, computed tomography scanning has largely supplanted the use of plain radiographs. For clinically based research involving sinus computed tomography scans, it is common to use a standardized radiographic scoring system (ie, the Lund-Mackay Score).[77]

Miscellaneous Tests

Of the several tests documenting nasal patency, only nasal peak flow measurement is sufficiently standardized to recommend for routine clinical practice. Nasal peak flow measurements can also be obtained on an ambulatory basis to document the response to allergens (or irritants) encountered on the job. Nasal cytometry, although somewhat laborious, is also sufficiently straightforward to permit incorporation into the clinical work-up. In terms of chemosensory function, the University of Pennsylvania Smell Identification Test is portable and straightforward to administer.

MANAGEMENT

Primary Prevention

Occupational rhinitis and asthma are preventable conditions. In general, primary prevention should follow the so-called hierarchy of industrial hygiene controls: substitution of less hazardous materials; enclosure and ventilation; administrative controls (limited exposure time); and personal protective equipment (eg, gloves, respirator). This hierarchical approach can, in some instances, prevent incident cases of occupational rhinitis and asthma.[78]

Secondary Prevention

Secondary prevention involves the early detection of disease and interruption of disease progression. Early detection can be achieved by monitoring symptoms (ie, through the use of periodic questionnaires); by documenting physiologic alterations (eg, exaggerated decrements of pulmonary function); or by identifying biomarkers (eg, antigen-specific IgE). The value of surveillance is illustrated by longitudinal studies, which document a higher risk for developing occupational respiratory disorders in the first few years after entering a profession.[79–81] Medical surveillance programs (beginning with preplacement examination, following of workers through apprenticeship, and continuing thereafter) offer the potential for early detection and prevention of disease progression among susceptible individuals.

Tertiary Prevention

For established occupational irritant rhinitis, treatment consists of exposure reduction; nonspecific supportive measures (eg, saline nasal lavage); and occasionally topical steroids. Patients troubled by prominent reflex symptoms (eg, congestion and rhinorrhea) may benefit from the topical cholinergic blocker, ipratropium bromide. In atopic patients with irritant rhinitis, control of intercurrent allergic rhinitis (even if unrelated to the workplace) may also decrease reactivity to chemical irritants.

In occupational allergic rhinitis, timely removal from exposure is the most effective means of preventing disease progression. Effective pharmacotherapy includes topical steroids; selected topical antihistamines (with anti-inflammatory properties); and topical ipratropium bromide for symptomatic treatment of hypersecretion. Oral antihistamines, if used, should be limited to nonsedating varieties. Oral leukotriene receptor antagonists are an option that has been little studied in occupational settings. Nasal irrigation with saline remains a benign intervention that has been reported by some to be of benefit.

SUMMARY

Occupational upper airway disorders are common, and the development of rhinitis likely plays a role in the pathogenesis of lower airway disease. Primary prevention involves exposure controls for irritants and allergens. Secondary prevention (workplace surveillance and selective reassignment) can also help reduce the burden of disease. Tertiary prevention (treatment and disability management) may come into play if a strong sensitizer is involved, or if diagnosis has been delayed and disease progression has occurred.

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