2.2 Physiology of the Nose

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As the initial upper part of the conductive respiratory system, the nose actively participates in airflow dynamics and conditioning of the 10,000 litres of air inhaled every day by an adult. As a specific organ, the nose contains the olfactory organ.

2.2.1 Airflow Dynamics

Air enters the nose through the nostrils. It takes a sharp turn and follows a curved course through the main nasal passage to exit the nasopharynx by the vertical choana; it then takes another sharp turn to the oropharynx. Inspiratory air is directed medially to course along the septum, where the main stream splits into different flows, arching between the inferior and superior turbinates. By comparison with the nasal cavities, airflow through the paranasal sinuses is inconsequential. The nose is responsible for 40–70% of the total airway resistance. The major portion of nasal resistance is confined to the nasal valve, e.g. the few millimetres posterior to the anterior edge of the upper lateral cartilage. The lumen of the nasal valve is regulated by lateral (head of the inferior turbinate) and medial (septum) erectile mucosa, and modulated by the tone of alar muscles and stabilised by bone and cartilage. At the entrance of the nasal cavity, airspeed is between 2 and 3 m s\(^{-1}\), at the nasal valve it reaches 12–18 m s\(^{-1}\) and beyond the nasal valve, due to the decrease of nasal resistance, it decelerates to 2–3 m s\(^{-1}\), promoting a disturbed pattern of respiratory airflow favourable for effective exchanges between air and mucosa.

The parameter most sensitive to change in nasal airway resistance is the size of the nasal cavities, which is proportional to the size of the turbinate mucosa. Several factors can affect the turbinate volume and thus the nasal resistance:
- Exercise usually decreases nasal resistance.
- Dust, smoke and alcohol usually increase nasal resistance.
- Pressure on one side of the body induces reflex nasal congestion on that side.

The nasal cycle is another factor affecting the volume of nasal turbinates: in healthy adults, each side of the nose alternatively congests and de congests every 3–7 h, leading to a spontaneous, resistive cycle phenomenon called the nasal cycle. The reason for the existence of this cycle remains unclear, but it occurs in approximately 80% of healthy adults. Although unilateral resistances can fluctuate between severe obstruction and optimum patency, the reciprocity between sides (as one side congests, the opposite side de congests) results in stable, combined resistance.

Sensory receptors of airflow have not been yet identified in nasal mucosa, but sensation of airflow could depend on thermal and/or sensitive stimulation of inhaled air. Moreover, several aromatic substances, notably L-menthol, enhance the sensations of chill, airflow and nasal patency, without modification of objective airflow indices. Thus, sensory interpretations of nasal airflow are influenced not only by ambient and pathophysiological conditions, but also by psychological factors.

The airflow distribution pattern through the nasal cavities and its characteristics are crucial to effective air conditioning.

2.2.2 Air Conditioning

In patients with tracheostomies, absence of nasal respiration induces squamous metaplasia of the respiratory epithelium. Moreover, in asthmatic subjects, oral breathing induces a higher degree of bronchoconstriction after exercise than nasal breathing does. Therefore, nose conditioning of inhaled air is essential to the lower airways, limiting aggression of the fragile structures of the alveoli. Essential in assuring the delicate function of air conditioning are the efficacy of the vascular network in the lamina propria, the contribution of watery secretion, the quantity of seromucous glands, the surface contact between inspired air and mucosa and the beating quality of the cilia.

The nose is responsible for thermal and hygrometric regulation of inhaled air, maintaining a 30°C mean temperature and 98% relative humidity, regardless of the variation of ambient humidity and temperature. The nose also participates in body thermoregulation via adjustment of the nasal mucosa blood flow. Arteriovenous
shunts within nasal mucosa allow elevation or decrease of blood flow when heat needs to be lost or retained, respectively. Moreover, in normal conditions, inhaled air is supplemented with nitric oxide (NO) synthesised in the paranasal sinuses. NO-induced broncho- and vasodilatation contribute to improve oxygenation as well as blood perfusion in ventilated alveolar areas, resulting in better function in the entire respiratory system.

During normal breathing, nasal air filtration is facilitated by the disturbed airflow pattern, allowing particles (microorganisms and noxious materials) to attach to the mucosa. The number of particles that will attach to the mucosal surface depends on several factors such as physical size, shape, density and hygroscopicity. Mucus film covering nasal epithelium is responsible for trapping particulates greater than 10 µm, which will be transported to the oropharynx and then swallowed and destroyed by gastric enzymes. Transport of particles depends on the mucociliary clearance, which is determined by the motion of the blanket of mucus from the front of the nose to the nasopharynx by the coordinated waves of cilia. The cilia are 0.3 µm in diameter and 7–10 µm long; there are about 100 cilia per cell. The rate of mucociliary transport is 1–2 mm h⁻¹ just behind the anterior portion of the inferior turbinate, and increases to 8–10 mm h⁻¹ on the posterior portion of the inferior turbinate. Factors affecting nasal mucociliary clearance are either primary abnormality of cilia (Kartagener’s syndrome, primary ciliary dyskinesia) and mucus (cystic fibrosis) or secondary to viruses, bacteria, chronic nasal inflammation (allergic rhinitis) and inhaled pollutants (tobacco smoke).

In addition to physical removal of particles by mucociliary clearance, the nose actively participates in immunological defence of the airways. Immunocompetent cells such as mast cells and lymphocytes T and B are found to migrate in nasal epithelium. These cells participate in antigenic-particle removal, immunological memory and release of preformed and granule-derived mediators of inflammation. Moreover, via HLA-DR and ICAM-1 expression, nasal epithelial cells could act as antigen-presenting cells to infiltrating lymphocytes. In addition, nasal NO and several nasal secretion constituents (peroxidases, interferon, lysozymes, lactoferrin, complement and immunoglobulins [A, G, M and E]) have immunological properties that may act nonspecifically to maintain the sterility of the lower airways. However, in patients with tracheostomies, despite colonisation of the lower airways with pathogenic bacteria, there is no increase in severe pulmonary infections.

2.2.3 Olfaction

Olfaction has a primary role in the regulation of food intake and in the perception of flavour. Olfaction has also an important protective function in the detection of irritating and toxic substances. During normal respiration, olfactory mucosa is sheltered from the inspiratory mainstream and, when sniffing, airflow is redistributed to the upper part of the nasal cavities to the olfactory organ. Humans can detect more than 10,000 different odours and discriminate between 5,000 of them. Age-related olfactory loss appears to begin at 60 years of age, and it becoming significantly worse after age 70. However, women consistently perform better than men do in smelling. The olfactory organ is functional at birth and is unique in the central nervous system, being the only part in direct contact with the environment and in its ability to regenerate damaged or lost neurons. In order to interact with the olfactory sensory neurons, hydrophilic odorants are dissolved in the olfactory mucus, and hydrophobic odorant molecules are bound and solubilised by odorant-binding proteins present in the nasal mucus. After interaction between the odorant molecules and receptor proteins, olfactory transduction (transformation of mechanical stimulation into electrical activity) probably involves an olfactory epithelium Gαolf protein–coupled cascade, with cAMP and/or inositol-phosphatidyl-3 as an intracellular second messenger, exciting an ion channel in the cilia, which depolarises the olfactory neuron. Depolarisation follows the olfactory axons that synapse in the olfactory bulbs, and then projections go to the amygdala, the prepyriform cortex, the anterior olfactory nucleus and the entorhinal cortex, as well as the hippocampus, hypothalamus and thalamus. The precise mechanism by which the vast number of smells is recognised and discriminated is unknown, but possible theories include specific odorants exciting specific receptors; differing solubilities of odorants, allowing a temporospatial distribution of the odorant across the olfactory mucosa or a response to the molecules’ vibration spectra. As three quarters of flavour is contributed to by olfaction, complaints of loss of taste are usually related to olfactory loss. There are three major classifications of olfactory disorders: transport (conductive), sensory and neural. Transport disorders interfere with the access of an odorant molecule to the olfactory receptor, sensory losses result from damage to the olfactory receptor, and neural losses result from interruptions in the peripheral or central nervous olfactory pathways.

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